



Person-centered examination of salivary cortisol and alpha-amylase responses to psychosocial stress: Links to preadolescent behavioral functioning and coping

Jason José Bendezú*, Martha E. Wadsworth

Department of Psychology, The Pennsylvania State University, University Park, PA 16802, United States

ARTICLE INFO

Keywords:

Coping
Preadolescence
Cortisol
Alpha-amylase
Trajectories

ABSTRACT

This study adopted a person-centered approach to identify preadolescent salivary cortisol (sC) and alpha-amylase (sAA) co-activation response patterns and examine links to behavioral functioning and coping. Children ($N = 151$, 51.7% male) were exposed to the Trier Social Stress Test (TSST) and one of two randomly-assigned, post-TSST coping conditions: distraction or avoidance. Multi-trajectory modeling yielded four child subgroups. Child internalizing and externalizing positively predicted High sC–High sAA relative to Low sC–Low sAA and Low sC–High sAA relative to High sC–Low sAA subgroup membership, respectively. Low sC–Low sAA children demonstrated more efficient sC recovery when primed with distraction and more protracted sC recovery when primed with avoidance. For High sC–High sAA, internalizing children, the opposite was true. Findings illustrate adjustment-linked variability in preadolescent sC–sAA co-activation response patterns that further articulates for whom effortful coping works to effectively manage stressor-induced neuroendocrine activation.

1. Introduction

Hypothalamic-pituitary-adrenal axis (HPA) and sympathetic-adrenomedullary system (SAM) co-activation has been implicated in the development of psychopathology (Bauer, Quas, & Boyce, 2002; Hastings et al., 2011), leading to the need to understand individual differences in HPA–SAM co-activation in childhood and adolescence. Theorists have posited that both symmetrical (e.g., high HPA–high SAM, low HPA–low SAM) and asymmetrical (e.g., low HPA–high SAM, high HPA–low SAM) activity is potentially reflective of neuroendocrine response dysregulation linked to children's internalizing and externalizing behavior (Bauer et al., 2002). When assessed via HPA (salivary cortisol, sC) and SAM (salivary alpha-amylase, sAA) indices in response to laboratory stressor paradigms, early adolescent studies have found mixed support for each proposition. Thus, there remains a lack of consensus on what constitutes well-functioning as well as dysregulated HPA–SAM co-activation response patterns.

The evidence supporting these views is based on variable-centered approaches that assume preadolescents exhibit more or less the same pattern of HPA–SAM co-activation. However, theory suggests that preadolescence is a period of substantive reformation to the HPA and SAM, when children are exposed to novel psychosocial stressors that shape their development (Spear, 2000; Steinberg, 2014). If so, then

variable-centered approaches may overlook the possibility that certain subgroups of children evidence qualitatively distinct patterns of HPA–SAM co-activation that may be uniquely linked to their behavioral adjustment. Person-centered approaches that incorporate multiple, theory-driven neuroendocrine response indices (e.g., sC, sAA) have the potential to identify these subgroups. Such an approach may also provide a more precise approximation of theoretical cross-system activation patterns (Bauer et al., 2002) that index healthy functioning or signal risk for psychopathological development (Rutter, 2007).

Children's coping, defined as effortful emotional, cognitive, and behavioral attempts to manage a stressor or children's emotional/cognitive/behavioral reactions to it, is thought to buffer against such psychopathological development by mitigating against one of the mechanisms of risk; neuroendocrine response dysregulation (Wadsworth, 2015). Recent evidence suggests that children's coping does in fact get “underneath the skin” (Foland-Ross, Kircanski, & Gotlib, 2014; Sladek, Doane, & Stroud, 2017), supporting quick, efficient recovery of stressor-induced neuroendocrine activation (Stewart, Mazurka, Bond, Wynne-Edwards, & Harkness, 2013). This research points to putative mechanisms of change to be leveraged in the design of coping interventions (Davidson & McEwen, 2012). However, reliance on variable-centered designs has limited understanding about *for whom* certain types of coping combat the potentially damaging effects of stress. If

* Corresponding author.

E-mail address: jjb490@psu.edu (J.J. Bendezú).

subgroups of children with distinct, adjustment-linked HPA-SAM co-activation patterns exist whose post-stressor neuroendocrine recovery is differentially influenced by specific forms of coping, then variable-centered approaches may obscure these differences (von Eye & Bogat, 2006). Person-centered analysis of this sort may bolster inference about patient-centered means of tailoring intervention content towards children's coping strengths and weaknesses. Thus, using a community sample of preadolescent boys and girls exposed to the Trier Social Stress Test (TSST-C; Buske-Kirschbaum et al., 1997), the current study adopted a novel person-centered approach to identify HPA-SAM co-activation profiles based children's sC-sAA response patterns, examine links to children's internalizing and externalizing behavior, and test if two coping skills differentially contributed to sC-sAA recovery efficiency for the identified subgroups.

1.1. Models of HPA-SAM co-activation and links to behavioral functioning

Current theory posits that the sympathetic nervous system (SNS), of which the SAM is a part, and HPA respond in a coordinated fashion to support adaptation both during and following a stressor (Bauer et al., 2002; Hastings et al., 2011). Initial changes that occur in support of this adaptation take place in the SNS and involve a taxing mobilization of cardiovascular, immunologic, and central nervous system resources to quickly neutralize immediate threat. A positive feedback loop between the SNS and HPA stimulates subsequent glucocorticoid production, mobilizing longer-term resources to support adaptation to more prolonged threat exposure (Chrousos & Gold, 1992). Cortisol produced by the SNS-innervated HPA helps suppress the initial SNS response, protecting the body from damage that may result from protracted, energy-depleting SNS activity. Well-functioning HPA-SNS co-activation might involve a brief SNS response to quickly neutralize threat that does not necessitate a more dramatic HPA response or stimulates cortisol production in the event that immediate threat is not neutralized (Sapolsky, Romero, & Munck, 2000). Alternatively, HPA-SNS dysregulation might involve elevated SNS activity that fails to innervate the HPA or elevated cortisol mobilization that fails to terminate SNS activity (e.g., Koss et al., 2014).

Consensus on what constitutes neuroendocrine response dysregulation as it pertains to HPA-SNS co-activation has yet to be reached. Bauer et al. (2002) proposed two models of HPA and SNS (in particular the SAM system) co-activation that may manifest as symptoms of psychopathology. The *Additive-Symmetry Model* predicts that symmetric activation of the HPA and SNS would indicate risk and be associated with symptomatic functioning. The alternative *Interactive-Asymmetry Model* proposes that asymmetric activation would indicate risk for poor behavioral functioning. The available early adolescent findings lack coherence to support either model as each study has used varied methods for examining HPA-SAM co-activation in both non-clinical and at-risk children. Support for the *Additive-Symmetry* model has been found when examining sC-sAA basal levels to internalizing associations (El-Sheikh et al., 2008) and combinations of sC-sAA basal and reactivity level (e.g., area-under-curve with respect to ground; AUCg) to externalizing links (Gordis et al., 2006). Support for the *Interactive-Asymmetry* model has also been found when examining sC-sAA basal level to externalizing associations (Chen, Raine, & Granger, 2015) and sC-sAA percent increase to problem behavior links (Allwood et al., 2011). Still others have found support for both models within the same study, noting specific directions of symmetry (high HPA-high SAM) and asymmetry (low HPA-high SAM) in predicting children's behavioral functioning (Koss et al., 2014).

To better understand individual differences in HPA-SAM co-activation to behavioral functioning linkages, three issues must be addressed. First, the inconsistent use of basal levels (e.g., pre-TSST levels, average of pre- and post-TSST levels) or reactivity indices (e.g., area-under-the-curve increase, AUCi; percent increase) within and across the HPA and SAM has contributed to discrepant operationalizations of

“high” and “low” neuroendocrine activity, limiting consensus about how HPA-SAM co-activation relates to behavior. As noted by Quas et al. (2014), there is a need to model various aspects of the full cross-system response, as both well-functioning and dysregulated neuroendocrine activation involve basal levels and response organization; i.e., change patterns. Second, studies that have modeled these aspects often utilize variable-centered methods (e.g., growth curves), which are critical to understanding normative HPA-SAM co-activation while also restricted in their articulation of diverse neuroendocrine response organization. As theory posits multiple well-functioning and dysregulated neuroendocrine response profiles that may bear little semblance to one another (Bauer et al., 2002; Del Giudice, Ellis, & Shirtcliff, 2011; Quas et al., 2014), person-centered methods may more appropriately capture heterogeneity in HPA-SAM co-activation, especially for studies where variability is implicit to the developmental period examined. Third, person-centered studies have usually not modeled HPA and SAM activity concurrently (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Ji, Negri, Kim, & Susman, 2016) or used modeling of both level and concurrent change (i.e., response trajectories) in each system (Del Giudice, Benjamin Hinnant, Ellis, & El-Sheikh, 2012; Quas et al., 2014).

1.2. Regulatory interference and fit processes

Bauer et al. (2002) also proposed that our understanding of HPA-SAM co-activation and related behavioral functioning may be aided by attending to children's coping behaviors in response to stress. Stressor-induced neuroendocrine activation is functional in that it mobilizes resources to help children meet the demands of a stressful experience.¹ Likewise, and consistent with Responses to Stress (RTS) theory (Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000), children's effortful coping ensues following a stressful experience, helping them modify the source of stress or modulate their reactions to it. In so doing, coping should also contribute to *recovery efficiency*, or the swift termination of stressor-induced neuroendocrine activation that, left unchecked, might otherwise contribute to peripheral biological “wear and tear” and behavioral maladjustment (Brosschot & Thayer, 1998; Javaras et al., 2012). To test this proposition, immediately following the TSST-C, children were experimentally primed with either behavioral distraction or cognitive avoidance coping. Following acute stress exposure, distraction is believed to combat the potentially damaging effects of stress (i.e., terminate neuroendocrine activation, *efficient recovery*) by helping children consciously reengage their attention to productive or soothing activities while avoidance is thought to exacerbate these effects (i.e., prolong neuroendocrine activation, *protracted recovery*) by inadvertently refocusing children's attention to the stressor. Nevertheless, different coping skills may work for different children, such that there are no universals to what constitutes *effective coping* (Wadsworth, 2015).

As alterations to neuroendocrine activation processes (e.g., protracted recovery) increase children's risk for development of psychopathology (Javaras et al., 2012), it is critical to understand *for whom* specific forms of coping buffer against such alterations following exposure to an acute stressor. Children with higher internalizing and externalizing behaviors present with increased attention to threat, worry-related cognitions, anger-related information processing biases, perceived uncontrollability, and impulsive action (Connor-Smith et al., 2000; Grant et al., 2003). Thus, distraction for children with dysregulated, internalizing/externalizing-linked HPA-SAM profiles may contribute to protracted recovery, given that these prepotent response tendencies are thought to consume executive resources needed for

¹ Stressor-induced neuroendocrine activation does not need to be reduced for optimal well-being. Some research in adults suggests that inadequate cortisol (Duncko, Makatsori, Fickova, Selko, & Jezova, 2006) and alpha-amylase (Hlavacova, Solarikova, Marko, Brezina, & Jezova, 2017) responses are evident in persons with high trait anxiety.

Download English Version:

<https://daneshyari.com/en/article/7278205>

Download Persian Version:

<https://daneshyari.com/article/7278205>

[Daneshyari.com](https://daneshyari.com)