Psychoneuroendocrinology xxx (xxxx) xxx–xxx

Contents lists available at ScienceDirect

Psychoneuroendocrinology

journal homepage: www.elsevier.com/locate/psyneuen

The cortisol reactivity threshold model: Direction of trait rumination and cortisol reactivity association varies with stressor severity

Suzanne Vrshek-Schallhorn*, Bradley M. Avery, Maria Ditcheva, Vaibhav R. Sapuram

The University of North Carolina at Greensboro, Department of Psychology, USA

ARTICLE INFO

Keywords:
Stress
Cortisol
Rumination
Cortisol reactivity threshold model
Depression

ABSTRACT

Various internalizing risk factors predict, in separate studies, both augmented and reduced cortisol responding to lab-induced stress. Stressor severity appears key: We tested whether heightened trait-like internalizing risk (here, trait rumination) predicts heightened cortisol reactivity under modest objective stress, but conversely predicts reduced reactivity under more robust objective stress. Thus, we hypothesized that trait rumination would interact with a curvilinear (quadratic) function of stress severity to predict cortisol reactivity. Evidence comes from 85 currently non-depressed emerging adults who completed either a non-stressful control protocol (n = 29), an intermediate difficulty Trier Social Stress Test (TSST; n = 26), or a robustly stressful negative evaluative TSST (n = 30). Latent growth curve models evaluated relationships between trait rumination and linear and quadratic effects of stressor severity on the change in cortisol and negative affect over time. Among other findings, a significant Trait Rumination x Quadratic Stress Severity interaction effect for cortisol's Quadratic Trend of Time (i.e., reactivity, B = .125, p = .017) supported the hypothesis. Rumination predicted greater cortisol reactivity to intermediate stress (r_p = .400, p = .043), but *blunted* reactivity to more robust negative evaluative stress (r_p = −0.379, p = 0.039). Contrasting hypotheses, negative affective reactivity increased independently of rumination as stressor severity increased (B = .453, p = 0.044). The direction of the relationship between an internalizing risk factor (trait rumination) and cortisol reactivity varies as a function of stressor severity. We propose the Cortisol Reactivity Threshold Model, which may help reconcile several divergent reactivity literatures and has implications for internalizing psychopathology, particularly depression.

1. Introduction

Dysregulation in hypothalamic-pituitary-adrenal (HPA) axis stress responding is associated with risk for and concurrent experience of internalizing psychopathology—depression (Doane et al., 2013; Halligan et al., 2007; Vrshek-Schallhorn et al., 2013) and anxiety disorders (Adam et al., 2014). However, research examining how numerous trait-like internalizing psychopathology risk factors (such as trait rumination, neuroticism, and low extraversion) predict lab-based reactivity in cortisol provides diverging results. Some results indicate that trait-like risk factors predict *greater* cortisol reactivity to lab-based stress (Wirtz et al., 2007; Zoccola et al., 2010), while others indicate these same risk factors predict relatively *blunted* cortisol reactivity (Bibbey et al., 2013; Oswald et al., 2006; Vrshek-Schallhorn et al., under review; Zoccola et al., 2008).[1] Similarly, a genetic predictor of cortisol reactivity suspected of being a risk factor for depression was linked first with heightened cortisol responding in two studies (Brummett et al., 2014; Brummett et al., 2012), but later with blunted responding across three samples (Avery and Vrshek-Schallhorn, 2016; Way et al., 2016). The present study examines a novel model that may help reconcile divergent findings and offer novel predictions about HPA functioning in internalizing psychopathology—predominantly depression.

2. The cortisol reactivity threshold model

Examination of some studies' methods suggests a striking pattern: In those yielding positive risk-reactivity associations, manipulations appear milder (e.g., reading a statement instead of giving a speech; Brummett et al., 2012) compared to those yielding negative associations (e.g., receiving negative evaluative non-verbal feedback instead of neutral feedback; Avery and Vrshek-Schallhorn, 2016). This suggests a

* Correspondence at: Department of Psychology, University of North Carolina at Greensboro, 296 Eberhart Building, P.O. Box 26170, Greensboro, NC, 27402-6170, USA.
E-mail address: Suzanne.Schallhorn@uncg.edu (S. Vrshek-Schallhorn).

[1] Papers that dichotomized rumination (Young and Nolen-Hoeksema, 2001) and extraversion and neuroticism (Schommer et al., 1999) predicting cortisol reactivity obtained non-significant results. Though consistent with then-common ANOVA-based approaches, dichotomizing continuous predictors is now accepted to contribute to false negative findings, Type II error (MacCallum et al., 2002).

https://doi.org/10.1016/j.psyneuen.2017.11.002
Received 13 April 2017; Received in revised form 10 October 2017; Accepted 7 November 2017

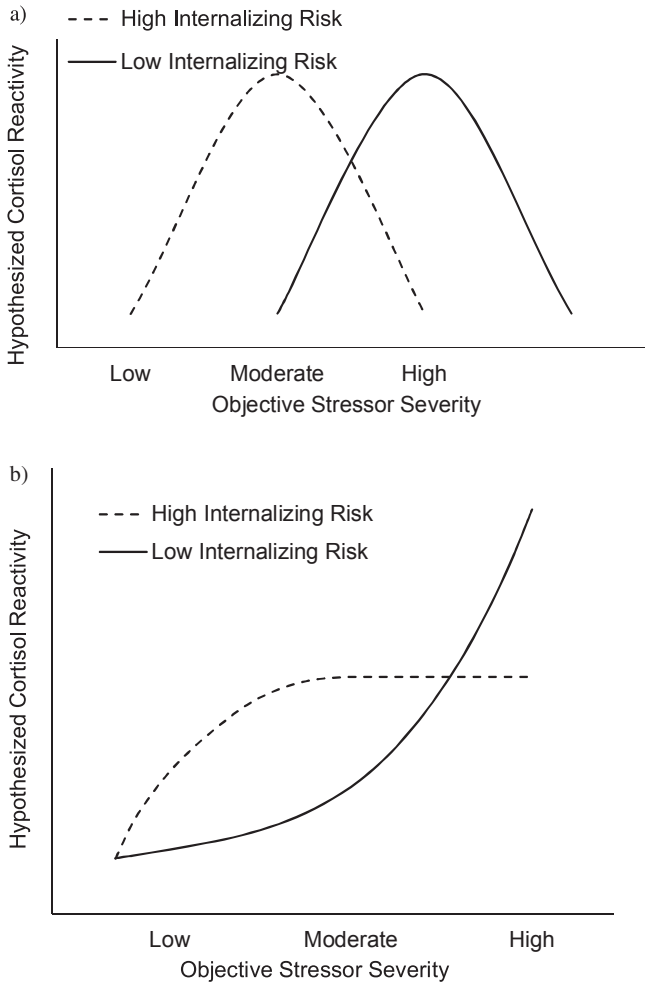

Fig. 1. Hypothesized relationship between internalizing risk factors and cortisol reactivity as a function of stressor severity level. Dashed line represents elevated internalizing risk; solid line represents relatively low internalizing risk. (a) The inverted-U curve variant. (b) The inflexibility variant.

model in which (a) individuals systematically differ in the level of objective stressor severity that provokes their peak cortisol reactivity, (b) internalizing risk contributes to this individual difference, and (c) the relationship between internalizing risk factors and cortisol reactivity will vary nonlinearly as objective stressor severity increases, such that (d) internalizing risk predicts relatively greater reactivity to modest threats, but (e) relatively less reactivity to robust threats. In addition to observations about the potential role of stressor severity, this model relies on evidence that cortisol functions in part as a resource-mobilizing hormone (for a review, see Sapolsky et al., 2000), and that internalizing risk is associated with biased perception of threat (e.g., Conway et al., 2016). Such biases might lead to mobilizing resources more readily under modest threat, but also to giving up more readily when threats are more robust (i.e., anhedonic stress responding; Pizzagalli, 2014).

In an initial conceptualization, informed by Yerkes-Dodson theory (for a review, see Teigen, 1994; Yerkes and Dodson, 1908), risk predicts achieving peak cortisol responses at a lower threshold of objective stressor severity but declining in reactivity as stressor severity increases (Fig. 1a, the "inverted-U variant"). A slightly different pattern would yield similar observations. In the "inflexibility variant" (Fig. 1b), higher risk individuals reach peak reactivity at a lower severity threshold, but have a flatter slope of reactivity change between moderate and robust stressors than their lower risk counterparts.

3. Influence of explicit negative evaluation on cortisol reactivity

A related question is whether explicitly negative evaluative stress inductions result in greater cortisol reactivity than those without explicit negative evaluation. Studies without *explicit* negative evaluation have been described by some as neutral, and by others as provoking (implicit) negative evaluation due to ambiguity. For example, standard TSST judges are, "trained to communicate with the subject in an unresponsive neutral manner…[without] any facial or verbal feedback," behaviors that are not intended to, "resemble harassment or evoke anger in participants," (Kudielka et al., 2007). Others characterize such methods as negative evaluative because of the potential for participants to infer negative evaluation, e.g., "confederates provided negative, non-verbal feedback by maintaining stoic expressions and eye contact," (Zoccola and Dickerson, 2015). Thus, although a number of studies report using negative evaluation, typically this characterizes ambiguous, neutral responses, rather than explicit negative evaluation. Critically, methods in which confederates are explicitly instructed to display non-verbal negative evaluative behavior (e.g., Taylor et al., 2010) primarily emerged after a rigorous meta-analysis showed that uncontrollability and social evaluation uniquely contribute to reactivity (Dickerson and Kemeny, 2004). No test yet compares cortisol reactivity between ambiguous, neutral manipulations and explicitly negative evaluative ones.

4. Negative affect under stress

Diathesis-stress models predict that trait-like internalizing risk factors will interact with objective stress to predict augmented or prolonged negative affect (e.g., Monroe and Simons, 1991; Nolen-Hoeksema et al., 2008). Evidence includes that trait rumination interacted with self-reported negative events to predict greater negative affect in an experience sampling study (Moberly and Watkins, 2008). Similarly, neuroticism interacted with interview-assessed stressful events to predict depression onset, consistent with both heightened and prolonged negative affect (Kendler et al., 2004), and engagement in rumination mediated self-reported stressful events' effect on increased internalizing symptoms (Michl et al., 2013). These findings suggest that internalizing risk factors may amplify the effect of increasingly stressful experiences on negative affect, thus differing from the curvilinear pattern anticipated for cortisol.

5. The present study

The present study examined non-depressed emerging adults and employed latent growth curve modeling to test how one transdiagnostic internalizing risk factor, trait rumination (dwelling on the causes or consequences of depressed mood; Treynor et al., 2003), predicts cortisol and negative affect reactivity to three levels of lab-induced stress: a non-stressful control, an intermediate severity-level TSST, and a negative evaluative TSST. We selected trait rumination to extend our previous work in which there was an association of trait rumination with blunted cortisol reactivity in a negative evaluative TSST compared to a non-stressful control (Vrshek-Schallhorn et al., under review).

We tested six hypotheses—five pertaining to cortisol and one pertaining to negative affect. We predicted that the relationship between trait rumination and cortisol reactivity would vary as a nonlinear function of stressor severity, an interaction between rumination and quadratic stressor severity (Hypothesis 1), such that trait rumination would predict greater cortisol reactivity to an intermediate stressor (Hypothesis 2), but would inversely predict reactivity in a more robust stressor (Hypothesis 3). We did not hypothesize an association in the non-stressful control. We predicted that increasing stressor severity would predict on-average greater cortisol reactivity (Hypothesis 4; Dickerson and Kemeny, 2004) and that an explicitly negative evaluative TSST would yield greater cortisol reactivity than a neutral/ambiguous