Psychoneuroendocrinology 89 (2018) 69–77

Contents lists available at ScienceDirect

Psychoneuroendocrinology

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

Stressful life events, relationship stressors, and cortisol reactivity: The moderating role of suppression

Lydia G. Roos[a], Sara M. Levens[a,b], Jeanette M. Bennett[a,b,*]

[a] Health Psychology PhD Program, USA
[b] Department of Psychological Science, The University of North Carolina at Charlotte, USA

ARTICLE INFO

Keywords:
Acute stress
Suppression
Cortisol
Relationship stress
Emotion regulation
HPA axis

ABSTRACT

Stressful life events (SLEs) are exceedingly common and have been associated with a range of psychological disorders, perhaps through dysregulation in the hypothalamic-pituitary-adrenal (HPA) axis. The use of certain emotion regulation strategies in response to stress, such as expressive suppression and cognitive reappraisal, has additionally been linked to heightened HPA axis reactivity to acute stress. However, it is unclear how emotion regulation may interact with SLEs to affect HPA axis reactivity, particularly concerning relationship stressors (RSs). Using cross-sectional data from 117 men and 85 women aged 18–55 years old (M = 39.9 ± 10.7), we investigated whether trait use of suppression or reappraisal interacted with recent negatively perceived SLEs and relationship stressors to impact HPA axis response to an acute stressor. Separate area under the curve and linear mixed models revealed that trait suppression interacted with SLEs and RSs to predict cortisol response to stress, while reappraisal did not. Findings indicate higher trait expressive suppression may influence the cortisol response to acute stress after exposure to more recent stressful events, particularly when those stressful events include relationship stress.

1. Introduction

Stressful life events (SLEs) are exceedingly common and have been associated with a range of mental health disorders, including increased risk of depression, posttraumatic stress disorder (PTSD), and anxiety disorders (Cameron et al., 2010; Francis et al., 2012; Kessler, 1997; Moitra et al., 2011). These disorders are also linked to dysregulated hypothalamic-pituitary-adrenal (HPA) axis reactivity to stress (Jacobson, 2014; Palazidou, 2012; Sherin and Nemeroff, 2011), suggesting that dysregulated HPA axis functioning may be a pathway by which SLEs give rise to psychopathology (Holsboer, 2001; Pariante and Lightman, 2008). Nonetheless, few studies have examined moderators of the relationship between SLEs and HPA axis reactivity, and no research to date has investigated the impact of relationship stressors on HPA axis reactivity to acute stress.

1.1. Stressful life events and neuroendocrine dysregulation

Exposure to traumatic SLEs is linked with dysregulated HPA axis stress reactivity (Carpenter et al., 2007; Heim et al., 2002; Jacobson, 2014; Palazidou, 2012; Pariante and Lightman, 2008; Sherin and Nemeroff, 2011). While it is challenging to determine whether dysregulated HPA axis reactivity or psychological disorders come first, exaggerated HPA axis reactivity to stress can occur both prior to and during depressive episodes (Ehlert et al., 2001; Holsboer, 2001; Pariante and Lightman, 2008), suggesting changes in HPA axis functioning may precipitate the development of psychopathology. As such, dysregulated HPA axis functioning, including reactivity to stress, following recent SLEs may be a neurobiological risk factor for the development of mental health disorders. While research exists concerning the effects of traumatic SLEs on the HPA axis, no known research has explored the possible impact of stressful interpersonal events, and specifically those that involve relationships, on HPA axis reactivity to acute stress.

Social relationships can facilitate better quality and quantity of social support, as well as reduced perceived stress, enhanced immune function, and improved mental and physical health (Cohen, 2004; Fagundes et al., 2011; Kiecolt-Glaser et al., 1997). Notably, SLEs that include threat to social relationships can deeply impact psychological wellbeing. Relationship stress and loss are associated with increased anxiety, lower levels of life satisfaction and higher rates of mental illness (Aseltine and Kessler, 1993; Knopfli et al., 2016; Simon and

* Corresponding author at: Department of Psychological Science, The University of North Carolina at Charlotte 9201 University City Boulevard, 4018 Colvard, Charlotte, NC 28223, USA.
E-mail address: jbenne70@uncc.edu (J.M. Bennett).

https://doi.org/10.1016/j.psyneuen.2017.12.026
Received 11 May 2017; Received in revised form 28 December 2017; Accepted 28 December 2017

Barrett, 2010; Rhoades et al., 2011). Furthermore, stressful events that involve relationship stressors (RSs) may encompass not only the stressor itself, but also the possible stress of a sudden loss of social support and sense of belonging (Cohen and Wills, 1985; Williamson and Schulz, 1992). The importance of relationships for wellbeing (Fagundes et al., 2011) and a threat to such relationships may be one reason for the disparate responses to interpersonal and noninterpersonal traumas.

Interpersonal events (e.g., physical attack, sexual assault) typically lead to greater psychological distress and higher rates of PTSD than events caused by accident or nature (e.g., automobile accident, natural disaster; Green et al., 2000). Further, interpersonal traumas that are perpetrated by someone that is close to and trusted by the victim are more distressing than those caused by strangers (Freyd, 1996; Goldsmith et al., 2012). Together, the importance of good relationships to health, the negative impacts of relationship loss, and the additive effects of relationships on traumatic responses suggests that when considering SLEs, stressors that include relationships may be perceived as especially stressful and could have a particularly potent effect on HPA axis stress reactivity; however, no research to date has specifically examined this association.

While SLEs and RSs are common, not everyone who experiences them develops dysregulated HPA axis functioning or poorer health. In fact, the majority of individuals exposed to stressful or traumatic events exhibit resilience (Bonanno et al., 2011). Continued healthy functioning for most people suggests other individual factors, such as emotion regulation, may influence vulnerability to the negative psychological and physiological effects of SLEs and RSs.

1.2. Emotion regulation strategies

Emotion regulation strategies are ways individuals exhibit control over their emotions, when they have them, and how they are expressed (Gross, 1998). Two common emotion regulation strategies are expressive suppression and cognitive reappraisal. *Expressive suppression* involves withholding emotional expression despite internal arousal (Gross, 1998; Gross and John, 2003). It is related to less social sharing, heightened sympathetic nervous system activation (Gross and Levenson, 1993, 1997), less positive emotion, and greater negative emotion (Gross and John, 2003), higher rumination and depressive symptoms (Gross and John, 2003; John and Gross, 2004) and its frequent use is generally considered to be maladaptive (John and Gross, 2004).

In comparison, *cognitive reappraisal* involves re-interpreting an emotion-eliciting event or situation to alter its emotional impact (Gross and John, 2003). Reappraisal is typically regarded as an adaptive emotion regulation strategy; increased reappraisal use is associated with healthier patterns of affect, social functioning, and well-being than suppression (Gross and John, 2003). Increased reappraisal ability is also related to fewer depressive symptoms (Troy et al., 2010) and is protective against the negative impact of increased stress on body mass index and type II diabetes (Sagui and Levens, 2016). Additionally, reappraisal is associated with an increased ability to recover psychologically from negative emotionally arousing situations both on a day-to-day basis as well as in response to experimentally-induced negative stimuli (Augustine and Hemenover, 2009; Gross and John, 2003; Meyer et al., 2012).

1.3. Emotion regulation and cortisol reactivity

Although suppression and reappraisal are different emotion regulation strategies, habitual use of both is linked with greater HPA axis reactivity to acute stress (Lam et al., 2009). Further, when participants were instructed to use reappraisal during an acute stressor, they exhibited a greater cortisol response than their counterparts who were given no instruction (Denson et al., 2014). As an elevated HPA axis response is typically viewed as detrimental to health (e.g., Kirschbaum et al., 1995; Morris and Rao, 2014; Puig-Perez et al., 2016), these findings raise the question of how an adaptive emotion regulation strategy, such as reappraisal, and a maladaptive strategy, such as suppression, could both give rise to elevated HPA axis stress reactivity.

One potential explanation may be in the short- versus long-term effects of reappraisal. Reappraisal, an approach-oriented strategy, requires individuals to exert cognitive effort to engage with and process negative emotion, identify ways in which negative content may be framed more positively, and then cognitively reinterpret the situation (Sheppes et al., 2011). Consequently, reappraisal in the short-term may increase negative affect and stress as the individual engages more cognitive resources to process and reframe the negative stimuli. When exposed to more stressors, however, habitual use of an adaptive strategy such as reappraisal may increase ability, ease of reappraisal, and meaning finding, possibly resulting in greater psychological and physiological habituation to stress and lower reactivity in the future. Although reappraisal is effortful in the short-term (Shafir et al., 2015), it is associated with long-term reduction of negative affect and adaptive outcomes following traumatic events and stressors (Denson et al., 2014; Moore et al., 2008; Troy et al., 2010). Hence, reappraisal may exacerbate HPA axis activity in the short-term in response to stress for those with less stressor experience, but result in a well-regulated system in the long-term for those with higher reappraisal, leading to a habituated stress response.

Conversely, expressive suppression is cognitively and physiologically taxing because it involves the behavioral inhibition of ongoing emotion expression during emotional arousal, but it does not change the subjective experience, which increases negative affect and risk for cardiovascular disease when used habitually (Gross, 1998; Gross and John, 2003; Hu et al., 2014; Mauss and Gross, 2004). As suppression contributes to greater negative affect and is physiologically demanding due to the increased cognitive and behavioral effort required to suppress expression, habitual suppression may additively interact with the experience of recent stressful events to uniquely heighten HPA axis reactivity. Despite the psychological effects of reappraisal and expressive suppression in response to stressors as well as their association with HPA axis reactivity, no research thus far has investigated the possible interaction between these SLEs and emotion regulation strategies on HPA axis reactivity to stress.

1.4. Current study

The aim of this study was to determine whether reappraisal or suppression modulates HPA axis reactivity to acute stress within the context of recent SLEs and, in particular, RSs. We hypothesized that consistent with prior research, individuals higher in reappraisal and suppression would have exaggerated cortisol reactivity to an acute stressor. We also predicted that suppression would interact with SLEs and RSs exposure such that as an individual experiences more SLEs, those who habitually suppress would have exaggerated HPA axis reactivity to stress as their SLEs and RSs increase. Furthermore, we expected higher reappraisal would buffer the effects of SLEs and RSs exposure on HPA axis stress reactivity as long-term or well-practiced reappraisal skills would dampen HPA axis stress reactivity among those with greater reappraisal compared to lower levels of reappraisal.

2. Methods

Secondary data were obtained from the Pittsburgh Cold Study 3 (PCS 3) within the Common Cold Project. The data were collected by the Laboratory for the Study of Stress, Immunity, and Disease at Carnegie Mellon University under the directorship of Sheldon Cohen, PhD; and were accessed via the Common Cold Project website (www.commoncoldproject.com; grant number NCCIH AT006694). The PCS 3 was originally a viral challenge study conducted between 2007 and 2011; including an acute stressor laboratory session before and after the