



Differences in HPA axis reactivity to intimacy in women with and without histories of sexual trauma



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ABSTRACT

Background: Sexual trauma can lead to longstanding effects on individuals' intimacy functioning. The current study aimed to assess hypothalamic pituitary adrenal (HPA) axis functioning (i.e., cortisol reactivity) prior to (−5 min), during (+15, +30, +45 min), and following (+60 min) an experimental manipulation of emotional closeness in a sample of women survivors of sexual trauma with varying levels of posttraumatic stress disorder (PTSD) symptomatology versus controls.

Methods: Participants included 50 women, which were divided into 2 groups on the basis of a structured clinical interview: 26 women with a history of sexual trauma with and without PTSD (sexual trauma group), and 24 women without a history of sexual trauma or PTSD (controls). Participants came into the lab and participated in a 45 min emotional closeness exercise with a male confederate and completed self-report questionnaires of closeness, state anxiety/depression, and cortisol assays at the aforementioned time points.

Results: Women with a history of sexual trauma exhibited a blunted cortisol response and greater anxious mood in reaction to the intimacy induction task compared to controls. Results also demonstrated that, unexpectedly, PTSD symptom severity scores among sexual trauma survivors were not associated with differential cortisol responding to the task compared to controls.

Conclusions: Adaptive responses to stress are characterized by a relatively rapid cortisol increase followed by a steady decline. The results of this study demonstrated that women with a history of sexual trauma, in contrast, displayed a blunted cortisol response to an intimacy induction task. Both controls and women with a history of sexual trauma reported increased feelings of closeness to the male confederate in response to the intimacy induction task, suggesting that survivors were able to achieve similar adaptive feelings of intimacy when provided with the right conditions.

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1. Introduction

Previous research indicates that having a history of sexual trauma, including childhood sexual abuse (CSA) and/or adult sexual assault (ASA), can lead to difficulty forming and maintaining intimate bonds in adulthood (e.g., Pistorello and Follette, 1998; Fleming et al., 1999; Davis and Petretic-Jackson, 2000; DiLillo, 2001; Connop and Petrak, 2004; Mills and Turnbull, 2004; Peleikis and Dahl, 2005; Katz et al., 2012). The specific mechanisms that contribute to this relationship, however, remain unclear. One possible explanation involves dysregulation of the body's stress response system, the hypothalamic-pituitary-adrenal (HPA) axis. Indeed,

HPA axis dysregulation is common among sexual trauma survivors, and prior research has found stress levels in the general population to be inversely related to intimacy behaviors (e.g., Floyd and Riforgiate, 2008). Research investigating whether HPA axis dysregulation occurs in response to intimacy among women sexual trauma survivors is needed to help further clarify the relationship between sexual trauma and the intimacy issues that commonly follow.

1.1. The impact of sexual trauma on intimacy

There is no consensus in the literature regarding the definition of intimacy, though intimacy has previously been defined as the quality of being close, self-disclosing, and affectionate with another person (e.g., see Levitz-Jones and Orlofsky, 1985; Laurenceau et al., 1998; Mosier, 2006). Previous research has revealed several nega-

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tive long-term consequences for emotional intimacy among sexual trauma survivors, including but not limited to: increased relationship problems, inappropriate sexualized behaviors, distorted ideas about sexuality, lower self-reported satisfaction with past interpersonal relationships, greater fear of intimacy, less comfort with closeness, and either an excess or lack of control in relationships (e.g., DiLillo and Long, 1999; Leonard and Follette, 2002; Katz et al., 2012). Survivors of sexual trauma also tend to have difficulty with achieving romantic intimacy, emotional communication, tolerating emotional closeness, trust, and confiding in and discussing personal concerns with partners (e.g., Mullen et al., 1994; Pistorello and Follette, 1998; Rumstein-McKean and Hunsley, 2001; Davis et al., 2001; Lipman, 2002; Peleikis and Dahl, 2005; Blake and Weinberger, 2006). The established relationship between sexual trauma history and interpersonal problems in adulthood highlight the importance of further study on this topic.

1.2. The role of HPA axis functioning

In addition to intimacy problems, sexual trauma has been associated with many other mental and physical health effects (e.g., Goodwin and Stein, 2004; Green and Kimerling, 2004; Hulme, 2004; Kendall-Tackett, 2012). Researchers have proposed that such difficulties can, at least in part, be explained by the body's stress response system: the HPA axis. It is well established that stress, in its various forms, activates the HPA axis which serves to produce cortisol. Typically, adaptive responses to stress are characterized by a relatively rapid cortisol increase followed by a steady decline. Under such conditions, cortisol helps to provide necessary energy resources when faced with stressors while also helping to modulate and contain other physiological stress response components and thus the ability to affect physiological changes encompassing most of the main organ systems (Adam and Kumari, 2009). Sporadic, short-term HPA activations are necessary and evolutionarily adaptive for survival, though excessive or chronic activation in general has been associated with various detrimental health outcomes with research implementing the neurotoxic effects of cortisol (e.g., Sapolsky et al., 1986; Sapolsky et al., 1986; Schnurr and Green, 2004).

However, because the HPA axis is very sensitive to psychological, environmental, and biological factors, this adaptive response can become dysregulated. Indeed, different reactivity phenotype patterns have emerged in different studies, with a number of studies showing exaggerated HPA axis and sympathetic nervous system (SNS) responses (e.g., De Bellis et al., 1999), and other studies showing blunted cortisol responses (e.g., King et al., 2001; Yehuda, 2004; Bremner et al., 2007; De Bellis et al., 2011; Bremner and Vaccarino, 2015).

To account for the disparity in findings in the cortisol literature, Hellhammer and Wade (1993) hypothesized that after trauma, the HPA axis is initially hyperactive corresponding with the acute stress response, but corticotropin-releasing hormone (CRH) receptors begin to down-regulate over time. At this point, a normalization of CRH results in diminished adrenocorticotropic hormone (ACTH) secretion, and ultimately produces cortisol levels below the normal baseline. Importantly, this model corresponds with studies that have demonstrated age at traumatization to be an important factor for cortisol reactivity (e.g., Bosch et al., 2012; Voellmin et al., 2015).

Due to the potential damaging effects of too much or too little cortisol production, researchers have posited HPA axis dysfunction as one potential cause of the observed intimacy symptoms among sexual trauma survivors. This is based in the finding that among healthy individuals, stress levels appear to be negatively associated with aspects of intimacy (Floyd and Riforgiate, 2008). Given the issues surrounding intimacy among sexual trauma survivors, therefore, it is possible that intimacy may serve as a potential

stressor for such individuals, thereby triggering HPA axis activation (i.e., positive correlation). Accordingly, emotional or interpersonal closeness can provide a context for the occurrence and consequences of psychosocial stress that activates the HPA axis system. Ineffective physiological regulation may be both driven by and be a contributor to intimacy distress (e.g., Laurent and Powers, 2007), and lead to more intimacy problems in this population.

1.3. The role of PTSD

Sexual victimization is often related to the development of PTSD (e.g., Bremner et al., 2004; Tolin and Foa, 2006), and symptoms associated with PTSD have been shown to impact a survivor's ability to form and maintain healthy bonds and relationships (e.g., Mills and Turnbull, 2004). PTSD symptoms including blunted emotionality (e.g., lack of affective responses, impairment in ability to experience emotions) and relatedness (e.g., difficulty in initiating and maintaining relationships), for example, have been shown to negatively impact relationships (e.g., Mills and Turnbull, 2004). Other PTSD symptoms such as intrusive memories, recurrent flashbacks, hyperarousal, and cognitive disturbances also have the propensity to negatively impact the quality of survivors' relationships (Bremner et al., 2004; Cobia et al., 2004; Mills and Turnbull, 2004). According to Mills and Turnbull (2004), the aforementioned symptoms can directly impair an individual's intrapsychic intimacy (intimacy within the individual), which thereby interferes with interpersonal intimacy (intimacy between two individuals). In addition, some researchers have proposed that neuroendocrine changes, such as lower levels of cortisol, may influence the formation and processing of traumatic memories and may be associated with the underlying pathology of PTSD (Yehuda, 2004; Daskalakis et al., 2013). Taken together, it is important to investigate the impact of intimacy-related stressors among sexual trauma survivors at varying levels of PTSD symptomatology. Surprisingly, no research to date has directly investigated the relationship between HPA axis functioning and intimacy among women with and without histories of sexual trauma, and the extent to which PTSD symptoms contribute to this relationship.

1.4. The current study

The purpose of the current study was to assess for the role of HPA axis functioning (i.e., cortisol reactivity) across an experimental manipulation of emotional closeness in a sample of women survivors of sexual trauma and a comparison group of women without a sexual trauma history (i.e., controls). In addition, the impact of varying levels of PTSD symptomatology associated with sexual trauma and resultant intimacy issues was examined. Because no research to date has directly investigated the relationship between HPA axis functioning, PTSD symptom severity, and intimacy within a sexual trauma population, we hope the current results will contribute to a greater understanding of the relationship between these factors and the physiological contributions to intimacy problems. Specific hypotheses included:

1. HPA axis (i.e., cortisol) reactivity to an emotional closeness induction task is expected to be suppressed (i.e., blunted) in women with a history of sexual trauma compared to women without such histories.
2. PTSD symptom severity is expected to be inversely correlated with cortisol reactivity.
3. Women with histories of sexual trauma are expected to report greater psychological distress in response to the emotional closeness induction task compared to controls.

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