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Youth offspring of mothers with posttraumatic stress disorder have altered stress reactivity in response to a laboratory stressor

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Summary Parental Posttraumatic Stress Disorder (PTSD), particularly maternal PTSD, confers risk for stress-related psychopathology among offspring. Altered hypothalamic—pituitary—adrenal (HPA) axis functioning is one mechanism proposed to explain transmission of this intergenerational risk. Investigation of this mechanism has been largely limited to general stress response (e.g., diurnal cortisol), rather than reactivity in response to an acute stressor. We examined cortisol reactivity in response to a laboratory stressor among offspring of mothers with a lifetime diagnosis of PTSD ($n = 36$) and age- and gender- matched control offspring of mothers without PTSD ($n = 36$). Youth (67% girls; mean age = 11.4, SD = 2.6) participated in a developmentally sensitive laboratory stressor and had salivary cortisol assessed five times (one pre-stress, one immediate post-stress, and three recovery measures, spaced 15 min apart). Results were consistent with the hypothesis that offspring of mothers with PTSD would exhibit a dysregulated, blunted cortisol reactivity profile, and control offspring would display the expected adaptive peak in cortisol response to challenge profile. Findings were maintained after controlling for youth traumatic event history, physical anxiety symptoms, and depression, as well as maternal depression. This finding contributes to the existing literature indicating that attenuated HPA axis functioning, inclusive of hyposecretion of cortisol

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in response to acute stress, is robust among youth of mothers with PTSD. Future research is warranted in elucidating cortisol reactivity as a link between maternal PTSD and stress-related psychopathology vulnerability among offspring.

1. Introduction

Posttraumatic Stress Disorder (PTSD) is a prevalent, costly, and debilitating disorder among adults (Kessler et al., 2005) that can impact multiple areas of an affected person's day-to-day functioning, including one's family (Galovski and Lyons, 2004). Offspring in particular are often impacted by parental PTSD (Samuelson and Cashman, 2008; Chemtob et al., 2010; Weems and Scheeringa, 2013), which may involve ongoing exposure to the parent's symptoms of intrusion (e.g., traumatic nightmares), avoidance (e.g., of people or places that serve as trauma reminders), negative core thoughts and beliefs (e.g., distorted self-blame), and hyperarousal (e.g., exaggerated startle reflex) (APA, American Psychiatric Association, 2013). Increased rates of PTSD and other stress-related psychopathology are found among offspring of parents with PTSD (e.g., Scheeringa and Zeanah, 2001; Yehuda et al., 2008), which is partially attributed to shared trauma exposure or increased likelihood of trauma exposure (e.g., Lehmann, 2000). Roberts et al. (2012) found that children's trauma exposure accounted for 74% of the increased risk of PTSD in their population-based sample of children of mothers with PTSD. Nonetheless, even in the absence of an offspring's exposure to a parent's index traumatic event, the literature supports well-established associations between parents with PTSD and related psychopathology among their offspring (Yehuda et al., 2001a; Leen-Feldner et al., 2013).

As such, the intergenerational transmission of offspring stress-related psychopathology from parents has been of keen empirical and clinical interest. Research that has specifically examined the role of parent gender in the intergenerational transmission of psychopathology from parental PTSD to offspring psychopathology has indicated that effects are particularly robust for mothers (Leen-Feldner et al., 2013); although links between paternal PTSD and offspring depression (Yehuda et al., 2008), as well as the notion of 'dose effects' (i.e., associations with offspring psychopathology are stronger when both parents have PTSD) (Yehuda et al., 2001a; Al-Turkait and Ohaeri, 2008), are supported by this small literature base. That maternal PTSD has been shown to lead to significantly elevated risk for offspring PTSD (Yehuda et al., 1998), as well as a range of other psychopathology outcomes (i.e., multifinality; Cicchetti and Toth, 2009), such as anxiety symptoms and externalizing problems (e.g., Linares et al., 2001; Graham-Bermann et al., 2009), lends itself to questions regarding the mechanisms underlying this intergenerational transmission process. Among the biological (e.g., epigenetics; Yehuda and Bierer, 2009; see Schmidt et al., 2011 for a review), psychological, and environmental mechanisms that have been proposed (Leen-Feldner et al., 2013; Rijlaarsdam et al., 2014), a growing body of neuroendocrine research supports the postulation that altered hypothalamic–pituitary–adrenal (HPA) axis functioning plays a role in this transmission (Yehuda et al., 2002). The main aim of this study is to extend this literature by examining the cortisol reactivity of child offspring of mothers with PTSD.

Yehuda and colleagues have examined altered HPA axis functioning in the link between maternal PTSD and offspring vulnerability to psychopathology among offspring of Holocaust-exposed parents (Yehuda et al., 2000, 2001b, 2007a,b) and among a sample of a sample of mothers exposed to 9/11 during their pregnancy (Yehuda et al., 2005). These studies have focused on general biological stress response (e.g., basal cortisol levels, diurnal HPA rhythms). This research team has also examined glucocorticoid sensitivity via dexamethasone suppression tests (Yehuda et al., 2007a), and more recently, via lysozyme suppression tests (Lehrner et al., 2014) among offspring of Holocaust survivors, supporting its association with parental PTSD—and maternal PTSD in particular (Lehrner et al., 2014). In general, the results of these seminal investigations show that the offspring of mothers with PTSD have an attenuated diurnal cortisol response and greater glucocorticoid sensitivity. Similarly, in one of the only studies to date examining HPA axis functioning among school-aged, traumatically-injured offspring of parents with posttraumatic stress, Nugent et al. (2007) found that children with low (initial) cortisol levels and with parents with high levels of posttraumatic stress were at highest risk for the later development of posttraumatic stress. With the exception of this body of literature, relatively few studies have focused specifically on mothers diagnosed with PTSD. There is a broader range of studies that have examined HPA axis functioning through the measurement of cortisol stress response involving offspring whose mothers had experienced stress while pregnant (see Glover et al., 2010 for a review) or who were depressed (Halligan et al., 2007; see Guerry and Hastings, 2011 for a review). However, given the literature that has emphasized specific links between maternal PTSD and later problems in offspring functioning (see Leen-Feldner et al., 2013 for review), beyond exposure to traumatic events (Li et al., 2010) and other psychiatric disorders (Yehuda et al., 1998), children of mothers with PTSD are an important population to study in unveiling stress-related psychopathology transmission mechanisms.

The relatively consistent finding across offspring-maternal PTSD studies to date indicating a blunted effect for cortisol suggests that the mechanism transmitted across generations is related to the tendency for low cortisol levels (Yehuda et al., 2002). Indeed, hyposecretion of cortisol, which represents insufficient activation of the HPA-axis in response to stress, has been found to be a risk factor for PTSD among adults (Mason et al., 1986; Yehuda et al., 1993; Mouthaan et al., 2014) and youth (Nugent et al., 2007).