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Social evaluative threat with verbal performance feedback alters neuroendocrine response to stress

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ABSTRACT

Laboratory stress tasks such as the Trier Social Stress Test (TSST) have provided a key piece to the puzzle for how psychosocial stress impacts the hypothalamic-pituitary-adrenal axis, other stress-responsive biomarkers, and ultimately wellbeing. These tasks are thought to work through biopsychosocial processes, specifically social evaluative threat and the uncontrollability heighten situational demands. The present study integrated an experimental modification to the design of the TSST to probe whether additional social evaluative threat, via negative verbal feedback about speech performance, can further alter stress reactivity in 63 men and women. This TSST study confirmed previous findings related to stress reactivity and stress recovery but extended this literature in several ways. First, we showed that additional social evaluative threat components, mid-task following the speech portion of the TSST, were still capable of enhancing the psychosocial stressor. Second, we considered stress-reactive hormones beyond cortisol to include dehydroepiandrosterone (DHEA) and testosterone, and found these hormones were also stress-responsive, and their release was coupled with one another. Third, we explored whether gain- and loss-framing incentive instructions, meant to influence performance motivation by enhancing the personal relevance of task performance, impacted hormonal reactivity. Results showed that each hormone was stress reactive and further had different responses to the modified TSST compared to the original TSST. Beyond the utility of showing how the TSST can be modified with heightened social evaluative threat and incentive-framing instructions, this study informs about how these three stress-responsive hormones have differential responses to the demands of a challenge and a stressor.

1. Introduction

When encountering a stressor, an individual engages in physiological preparedness, which starts with the perception of a threat or a challenge to the organism. A biopsychosocial model is useful for understanding how and why a stressor impacts biological measures (Blascovich, 2008). Often in the laboratory, a stressor's efficacy is inferred according to whether the context elicits neuroendocrine acute stress response, typically cortisol release (Dickerson and Kemeny, 2004). The Trier Social Stress Test (TSST) developed by Kirschbaum et al. (1993), involves delivering a speech and mental arithmetic in front of live, white-coated judges and a video camera (Campbell and Ehlert, 2012; Kudielka et al., 2007) and is putatively the most common laboratory stressor. While effective, it was several years after its design that researchers systematically recognized that the TSST's efficacy relied on social evaluative threat and uncontrollability (Dickerson and Kemeny, 2004). Social evaluative threat occurs when an interchange of social interactions is perceived as a threat or social judgment, and the organism must engage the stressor to protect the self. Uncontrollability also enhances reactivity by increasing the stressor's demands on the organism. This paradigm has allowed for a burgeoning of our understanding of the timing and mechanisms of the human stress response system and most recently, has extended to systematic TSST alterations to better understand uncontrollability and social evaluative threat. To our knowledge, it is relatively novel that our study explored whether an experimental manipulation of the TSST mid-way through the TSST alters the physiological stress response.

1.1. Biopsychosocial stress responsive biomarkers

Changes in cortisol can indicate that the individual is experiencing a stressor at a physiological level. Central reward pathways (Fuchs and

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Flügge, 2003; Esch and Stefano, 2010) as well as limbic neurocircuitry (e.g., amygdala, orbitofrontal cortex) are activated (Dedovic et al., 2009) by stress. The stressor begins the hormone cascade when corticotrophin-releasing hormone (CRH) is released from the hypothalamus (see details in Sapolsky et al., 2000) and ends when steroid hormones are released from target organs, such as the adrenal gland, including the glucocorticoid cortisol within 15–25 min following stress exposure (Dickerson and Kemeny, 2004). Cortisol alters lipid and glucose metabolism and influences neural functioning by binding to glucocorticoid receptors (GRs) and mineralocorticoid receptors (MRs) at differential binding affinity (Groeneweg et al., 2012). Cortisol binding to GRs in the hypothalamus largely explains negative feedback as occupied GRs suppress subsequent HPA axis activation. Consequently, different neural preparative and reactive processes are involved in accordance to the level and timecourse of a stressor (Sapolsky et al., 2000; De Kloet et al., 2008; Kinner et al., 2016).

Although cortisol is the quintessential stress hormone, it is not the only stress reactive hormone. Dehydroepiandrosterone (DHEA) is also released from the adrenal gland (as well as other glands such as the gonads), in response to adrenocorticotropic hormone (ACTH) and in response to stressors (Shirtcliff et al., 2007), including the TSST (Lennartsson et al., 2012; Shirotsuki et al., 2009). DHEA remains largely understudied as a stress-responsive hormone (Stárka et al., 2015), despite this abundant hormone's neuroprotective and anti-glucocorticoid activities within emotion-related neurocircuitry (Maninger et al., 2009). DHEA serves a role as a biosynthetic precursor to neurosteroids and androgens like testosterone and thus this hormone connects both glucocorticoid and androgen hormones (Carlström et al., 1988).

Like DHEA, testosterone is underappreciated as a stress-responsive hormone and more often is examined as an end-product of the hypothalamic-pituitary-gonadal (HPG) axis. Testosterone is involved in development of male secondary sexual characteristics, such as increased muscle mass (Mazur and Booth, 1998). Testosterone also has been shown to play an important role in adult social behavior (Booth et al., 2006; Bos et al., 2012a), such as competitive drive (Archer, 2006; Casto and Edwards, 2016). Both genders produce testosterone, yet there are gender differences. Compared to males, females release less testosterone (Booth et al., 2006). Gonadal testosterone follows somewhat different metabolic pathways (Handa and Weiser, 2014), and testosterone in females is largely of adrenal origin, which further bolsters the idea of testosterone can be a stress-responsive hormone (Drury et al., 2014). Few studies have examined testosterone reactivity to the TSST (see Schoofs and Wolf, 2011 for an exception), but a parallel literature illustrates that testosterone acutely rises when an individual faces a challenge or competition (see Archer, 2006 for a review). If testosterone changes during a stressor, it is unknown if testosterone is reactive through enhanced responsiveness to social evaluative threat (like cortisol or, presumably, DHEA) or through a more testosterone-relevant mechanism, such as competition, challenge, or reward.

In addition to examining whether testosterone and DHEA are reactive under social evaluative threat, an emerging literature is demonstrating dual-axis activation within the individual as evidence of crosstalk between the HPA and HPG axes (Shirtcliff et al., 2015; Viau, 2002). Prior theories suggested inhibitory cross-talk (Viau, 2002), yet a series of studies find consistent positive within-individual associations, known as 'coupling,' of androgens and cortisol (Dismukes et al., 2014; Han et al., 2015), including during stressful contexts (Marceau et al., 2014). This dual-axis view is providing important mechanistic insights into when and why these hormones may work together to influence behavior, such as under conditions of challenge (Mehta et al., 2008; Mehta and Josephs, 2010). Initially, 'coupling' was theorized to be observed primarily in adolescents who may need to maintain capacity to activate androgens even under stress (Ruttle et al., 2015; Susman et al., 2017), yet positive coupling has also been observed in adults (Bobadilla et al., 2015; Harden et al., 2016). Marceau et al. (2014) examined coupling in response to three stressors and other research has examined multiple stress biomarkers (Bedgood et al., 2014; Chatterton et al., 1997; Eatough et al., 2009; Knight and Mehta, 2017; Turan et al., 2015). Yet to our knowledge, this is the first study to examine coupling of cortisol, testosterone, and DHEA during the TSST.

1.2. Enhanced social evaluative threat through verbal performance feedback and incentivized performance

Modified versions of the TSST are increasingly frequent (Campbell and Ehlert, 2012; Wadiwalla et al., 2010). For example, the TSST has been modified in order to fit the constraints of experimental protocols for use in children (Buske-Kirschbaum et al., 1997), groups (Von Dawans et al., 2011), for neuroimaging (Kern et al., 2008), and with virtual audiences (Kelly et al., 2007). Other studies have modified the TSST in order to better understand the psychological and social components that make the TSST work as a biological stressor (Andrews et al., 2007). These studies can be framed in terms of a biopsychosocial model (Seery, 2011; Tomaka et al., 1997; Blascovich and Tomaka, 1996) in which a motivated performance situation, like the TSST, relies on psychological processes within the individual (task engagement, evaluation of resources and situational demands).

Situational demands shift according to the level of social evaluative threat or uncontrollability. For instance, the confederate audience changes social evaluative threat (Dickerson et al., 2008; Wadiwalla et al., 2010), such that speech tasks without social judgment or with positive social cues from confederates do not stimulate neuroendocrine responsivity (Het et al., 2009; Taylor et al., 2010; Wiemers et al., 2013; Gruenewald et al., 2004). The original TSST provides no direct verbal feedback about speech performance, but Dedovic et al. (2005) found negative verbal and nonverbal feedback about math performance enhanced cortisol reactivity during the Montreal Imaging Stress Task (Dedovic et al., 2005).

The biopsychosocial model (Seery, 2011) also postulates that motivated performance is necessary to elicit increased stress responsivity across multiple physiological systems (see Campbell and Ehlert, 2012 for a comprehensive review). Some studies have altered performance motivation by changing the speech topic to be personal, based on the idea that greater ego involvement and self-referential components should enhance the TSST (Wadiwalla et al., 2010; Andrews et al., 2007). It is possible that social stimuli may enhance performance motivation as individuals are motivated to help or impress others. Dedovic et al. (2005) told participants that data would not be used if performance did not improve. Unfortunately, it is unclear whether the usefulness of the data for the researcher is motivating to the participant. A more direct method of motivating performance may be through use of incentives (Seery et al., 2009), given the role of cortisol in punishment and reward sensitivity (Van Honk et al., 2004) and the emerging literature that androgens like testosterone are sensitive to reward or challenge (Mehta et al., 2008; Mehta and Josephs, 2006; Bos et al., 2012b). Several studies attached the descriptor "motivated performance" to the TSST without describing whether (and how) incentives were delivered and performance during the TSST is not typically tied to compensation. Thus, incentives may motivate participants to be in a research study, but not necessarily to perform well during the stressor. Lastly, there is some evidence that multiple TSST modifications best impact reactivity. Wadiwalla et al. (2010) found that the effect of ego involvement or divided attention were only observed under conditions of enhanced social evaluative threat, suggesting that the biopsychosocial processes of motivated performance and situational demands are not mutually exclusive.

1.3. The present study aims and hypotheses

We used a multi-pronged experimental modification to the TSST that targeted both social evaluative threat and performance motivation. This was accomplished through Verbal Evaluation of Speech