



Anger responses to psychosocial stress predict heart rate and cortisol stress responses in men but not women



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Abstract While previous research has suggested that anger and fear responses to stress are linked to distinct sympathetic nervous system (SNS) stress responses, little is known about how these emotions predict hypothalamus-pituitary-adrenal (HPA) axis reactivity. Further, earlier research primarily relied on retrospective self-report of emotion. The current study aimed at addressing both issues in male and female individuals by assessing the role of anger and fear in predicting heart rate and cortisol stress responses using both self-report and facial coding analysis to assess emotion responses.

We exposed 32 healthy students (18 female; 19.6 ± 1.7 yr) to an acute psychosocial stress paradigm (TSST) and measured heart rate and salivary cortisol levels throughout the protocol. Anger and fear before and after stress exposure was assessed by self-report, and video recordings of the TSST were assessed by a certified facial coder to determine emotion expression (FACS).

Self-reported emotions and emotion expressions did not correlate (all $p > .23$). Increases in self-reported fear predicted blunted cortisol responses in men ($\beta = 0.41$, $p = .04$). Also for men, longer durations of anger expression predicted exaggerated cortisol responses ($\beta = 0.67$, $p = .004$), and more anger incidences predicted exaggerated cortisol and heart rate responses ($\beta = 0.51$, $p = .033$; $\beta = 0.46$, $p = .066$, resp.). Anger and fear did not predict SNS or HPA activity for females (all $p > .23$).

The current differential self-report and facial coding findings support the use of multiple modes of emotion assessment. Particularly, FACS but not self-report revealed a robust anger-stress association that could have important downstream health effects for men. For women, future research may clarify the role of other emotions, such as self-conscious expressions of shame, for physiological stress responses. A better understanding of the emotion-stress link may contribute to behavioral interventions targeting health-promoting ways of responding emotionally to stress.

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1. Introduction

Stress may elicit a range of emotions, including feelings of anger and fear. Although recent studies have examined the situational and cognitive predictors and moderators of acute stress (Denson et al., 2009; Dickerson and Kemeny, 2004; Gaab et al., 2003), less is known about the role of emotion. Research is particularly lacking on how specific emotions, such as anger and fear, may predict hypothalamic–pituitary–adrenal (HPA) axis activation as measured by cortisol responses to stress. Unraveling potentially specific associations between anger, fear, and cortisol stress responses could be helpful in differentiating harmful from more productive stress responses. Furthermore, it could lead to interventions that introduce better ways of coping with the emotions that stress may elicit.

Acute stress activates a coordinated set of physiological responses that prepares the body to deal with an immediate threat. A rapid “first wave” response dictated by the Sympathetic Nervous System (SNS) increases respiration and heart rate as oxygen and glucose speed to the skeletal muscles and heart (Sapolsky, 2000). A second response dictated by the hypothalamic–pituitary–adrenal (HPA) axis stimulates the production and release of stress hormones including cortisol. In addition to prolonging the cardiovascular effects of the SNS, cortisol protects the body from the harmful effects of an overactive immune system by selectively suppressing those functions that are no longer needed once the stressor has ended (Besedovsky et al., 1985). Though adaptive when activated in the short term, repeated or chronic activation of the HPA axis is believed to cause wear and tear on the body, and has consistently been linked to negative health outcomes (McEwen, 1998; McEwen and Seeman, 1999; Tsigos and Chrousos, 2002).

According to appraisal theory, it is not the stressor itself, but rather what one thinks about the situation, that will determine the physiological effects that result from the experience (Lazarus and Folkman, 1984). These subjective appraisals of stressful situations are also thought to give rise to emotions. That is, if a person interprets a situation as beneficial, positive emotions result; if the situation is appraised as potentially harmful, negative emotions result (Lazarus, 1982; Smith and Lazarus, 1990). It is not yet clear, however, if and how positive or negative emotions are linked to acute biological stress responses, since prior studies failed to find such associations (Clark et al., 2001; Dickerson and Kemeny, 2004; Denson et al., 2009). Instead, some evidence suggests that within these broad valence categorizations for positive and negative emotions, individual emotions may be linked to acute stress responses. For example, a line of research has found links between self-conscious emotions such as shame and strength of cortisol stress responses (Dickerson et al., 2004, 2008).

Anger and fear, two other negative-valence emotions, have also been linked to stress responses, particularly with regards to the Sympathetic Nervous System. Both emotions have been found to be positively correlated with heart rate increases (Ekman et al., 1983; Ray et al., 2008). Compared with fear, anger seems to be more strongly associated with increased diastolic blood pressure, arterial pressure (Schwartz et al., 1981), and finger temperature (Ekman

et al., 1983). Current literature also suggests possible gender differences in the emotion-SNS link. For example, in one particular study, young women showed stronger heart rate reactivity than young men in both anger- and fear-induction conditions (Labouvie-Vief et al., 2003). Taken as a whole, this research suggests that anger and fear may differentially predict specific SNS responses in the context of stress and, further, that these relationships may be moderated by gender.

With regards to the second wave of acute stress responses, studies linking emotion to HPA stress responses are few and often report conflicting results. For instance, one study found that self-reported anger after stress predicted stronger cortisol stress responses, while self-reported fear predicted decreases in cortisol levels (Moons et al., 2010). Yet, another recent study found that participants who felt more fear during a stressor actually showed higher cortisol increases (Lerner et al., 2007). The methodological differences between the studies could help explain the conflicting findings; for example, the study by Moons et al. (2010) utilized a psychosocial stress paradigm (Trier Social Stress Test: TSST) and emotions were self-reported after completion of the task, while the latter used mathematical stress tasks and facial coding analyses of emotion expressions shown throughout the stressor. It may be that the emotion-cortisol link differs based on the type of stress test given, as well as the method of emotion assessment (self-report vs. facial coding), but this has not yet been investigated. Notably, neither of the above studies reported gender differences in the emotion-HPA link. Clearly, more research is needed to tease apart the effects that anger and fear may have on the HPA axis for both men and women.

To our knowledge the study by Lerner et al. (2007) is the first to use facial coding to assess emotion in the context of stress. This is a notable development, since most studies rely on self-report to assess emotions. This method is not without its weaknesses; namely, due to the nature of many laboratory stress tests, questionnaires are only given before and after the stressor, and so may not capture the more immediate emotional reactions that occur during the actual stress test. According to Scherer’s components processing model of emotion, emotion consists of a combination of five different elements: cognitive appraisal, bodily symptoms, action tendencies, feeling, and expression (Scherer, 1987, 2005). While self-report may address subjective experience through feelings and cognitive appraisals of a situation, facial coding may shed light on another component- that of expression. The ‘online’ way of measuring emotion versus the ‘retrospective and processed’ way of self-report could account for some of the differences currently found in the literature linking emotions and cortisol responses. Thus, for the current study in addition to self-report, we added facial coding to assess emotion expressions throughout stress exposure. The Facial Action Coding System (Ekman and Friesen, 1978; Ekman et al., 2002) is one widely-used method to categorize expression of emotion through movement of individual facial muscles. This analysis not only allows for assessment of emotion expressions as they occur, but also can detect emotions that the participant may not self-report accurately, either purposefully or unknowingly (Cronbach, 1970; Derakshan and Eysenck, 1999; Myers and

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