Brief cognitive intervention can modulate neuroendocrine stress responses to the Trier Social Stress Test: Buffering effects of a compassionate goal orientation

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Summary
Background: The hypothalamic–pituitary–adrenal (HPA) axis is a critical mediator linking stress to health. Understanding how to modulate its reactivity could potentially help reduce the detrimental health effects of HPA axis activation. Social evaluative threat is a potent activator of this system. Access to control and coping responses can reduce its reactivity to pharmacological activation. Compassionate or affiliative behaviors may also moderate stress reactivity. Impact of these moderators on social evaluative threat is unknown. Here, we tested the hypotheses that interventions to increase control, coping, or compassionate (versus competitive) goals could reduce HPA-axis response to social evaluative threat.

Methods: Healthy participants (n = 54) were exposed to social evaluative threat using the Trier Social Stress Test (TSST). They were randomly assigned to receive one of four different instructions prior to the stressor: Standard TSST instructions (SI), standard instructions with access to “control” (SI Control), or one of two cognitive interventions (CI) that (1) increased familiarity and helped participants prepare coping strategies (CI Coping), or (2) shifted goal orientation from self-promotion to helping others (CI Compassionate Goals). ACTH and cortisol were obtained before and after stress exposure via intravenous catheter.

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1. Introduction

The hypothalamic–pituitary adrenal (HPA) axis and its end product, cortisol, critically mediate the negative impact of "stress" on health, impacting the onset, course and pathophysiology of medical (Chrousos and Gold, 1998; McEwen, 1998) and psychiatric disorders (Carroll et al., 1981; Young et al., 2003). They link early life experience to lifelong vulnerability to biobehavioral disturbances that undermine adaptive functioning (Ladd et al., 1999). Understanding HPA psychobiology can help reduce stress-related illness and psychiatric disorders and promote healthy development and adaptive resilience. Despite decades of research, however, we still have not clearly defined what specific aspects of "stress" activate this system or the factors that moderate its activity when it is stimulated. Identification of specific factors that activate and modulate the HPA axis can guide efforts to reduce exposure to the type of stress that damages health and can shape interventions to reduce the deleterious impact of predictable but unavoidable stressors.

Links between psychosocial stress, the HPA axis, and health outcomes have been extensively explored epidemiologically. Cortisol levels are influenced by socioeconomic status, race/ethnicity, and complex psychosocial constructs like overload, job strain, or burnout (Adam and Kumari, 2009). Cortisol is also linked to onset or course of autoimmune, allergic, infectious and neoplastic diseases (Elenkov et al., 1999), as well as vascular disease (Rosmond et al., 1998), heart disease, diabetes, and stroke (Rosmond and Bjorntorp, 2000). Epidemiological work has the power needed to detect associations and control for covariates (Adam and Kumari, 2009), but it cannot precisely identify key factors and causal pathways that mediate the impact of social and psychological experience on the acute biological responses that are relevant to longer-term health. For this, experimental studies are needed. A meta-analysis of laboratory-based psychosocial stress studies has demonstrated that cortisol reactivity is not a function of negative affect or subjective distress, but is linked most closely to social evaluative threat or lack of control over perceived stressors (Dickerson and Kemeny, 2004). These two factors may be linked, in that social evaluative threat may be a particularly potent HPA activator because it is inherently uncontrollable. Enhancing sense of control may be one way to modulate system activation, and perhaps enhance health and resilience.

Another moderator of HPA axis activity is social support (Levine, 2000; Uchino et al., 1996), which may moderate general stress effects (Cosley et al., 2010; Kirschbaum et al., 1995) through affiliation-induced release of oxytocin, which can inhibit HPA axis activation (Heinrichs et al., 2003). Affiliation is also experienced through giving social support (Brown et al., 2008; Konrath and Brown, 2012) and giving to others out of concern for their wellbeing (operationalized in constructs like compassion, loving-kindness, altruism, helping, and volunteerism) is attracting scientific interest as a stress reducer and health enhancer (e.g., Hofmann et al., 2011). Endorsing compassionate goals can prospectively enhance perceptions of available social support and reduce focus on self-other competition, and is associated with enhanced emotional well-being (Crocker and Canavello, 2008; Crocker et al., 2010). Giving to others (e.g., volunteering, caregiving) reduces self-focus and can yield psychological and health benefits for the giver (Konrath and Brown, 2012). Cultivating a motivational state related to helping others may activate a neurobehavioral “caregiving system” that promotes social bonds and perhaps enhances health by dampening stress reactivity (Davidson and McEwen, 2012; Konrath and Brown, 2012; Taylor et al., 2000; Wang, 2005). The key psychosocial factors at work may involve shifting focus from self-protection or self-promotion to core values that emphasize concern for the well-being of others and contributing to the greater good (Crocker and Canavello, 2012). Biological mechanisms through which compassionate or altruistic goals and giving to others can enhance emotional well-being and improve health may involve HPA axis modulation (Konrath and Brown, 2012). However, efforts to directly demonstrate modulation of cortisol release in the laboratory by compassion meditation (Pace et al., 2009) or providing support to a stressed other (Smith et al., 2009) have not been successful. A more direct counter to social evaluative threat (Dickerson and Kemeny, 2004), by shifting focus from self-image goals toward compassionate goals (Crocker and Canavello, 2012), may be more effective.

Our laboratory has explored psychosocial modulation of HPA axis activity using specific psychological manipulations and an externally activated system. Initial studies used direct pharmacological activators to provide a “cleaner” test of psychological modulators. Access to control and/or cognitive coping tools reduced cortisol or corticotropin (ACTH) output even when the system was activated by direct pituitary stimulation (Abelson et al., 2005, 2008, 2010), suggesting

Results: Control alone had no effect. CI Compassionate Goals significantly reduced ACTH and cortisol responses to the TSST; CI Coping raised baseline levels. Compassionate Goals reduced hormonal responses without reducing subjective anxiety, stress or fear, while increasing expression of pro-social intentions and focus on helping others.

Conclusions: Brief intervention to shift focus from competitive self-promotion to a goal orientation of helping-others can reduce HPA-axis activation to a potent psychosocial stressor. This supports the potential for developing brief interventions as inoculation tools to reduce the impact of predictable stressors and lends support to growing evidence that compassion and altruistic goals can moderate the effects of stress.

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