

Patterns of cortisol reactivity to laboratory stress

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Abstract

Cortisol responses to a laboratory stress protocol were investigated in 82 male firefighters. Saliva samples were collected during an adaptation period beginning between 9 and 10 am, and then at the end of each of six 10-min trials (a mental arithmetic task, an inter-task recovery period, a speech task, and three recovery periods). Individual differences in the mean cortisol response to the stress tasks were characterized by variation in the direction of the response, as well as the size of the response. Neither pre-stress cortisol levels nor responses were correlated with cardiovascular and mood responses. Cortisol levels before stress task presentation were negatively correlated with recent stress severity. Larger mean cortisol responses were associated with lower reports of recent stress exposure, lower negative affect scores, and a coping style characterized less experience of anger, more control over anger expression, and a tendency to screen out threatening information in stressful situations. Thus, increased cortisol activity was associated with less recent stress exposure and a more adaptive behavioral style than for those whose cortisol levels fell or were largely unchanged in response to a laboratory stressor.

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Introduction

Increased cortisol activity has been demonstrated in response to laboratory and daily life stress (e.g., Brantley et al., 1988; Deinzer et al., 1997; Kirschbaum et al., 1992). Generally, the more salient and intense a stimulus, the larger the response, thus parachute jumping elicits far larger responses than public mental arithmetic or speech task performance (Deinzer et al., 1997 vs. Kirschbaum et al., 1992). However, there are some inconsistencies with a simple linear stress–response relationship. Cortisol decreases during stress in some individuals (Heim et al., 2000), and lower cortisol levels with higher recent stress exposure (Roy et al., 2003). Differences in the pattern of cortisol responses also introduce significant methodological problems (e.g., Deinzer et al., 1997), and there are inconsistencies concerning cortisol's association with other reactivity measures and measures of affect and coping.

Cortisol regulation and stress

Individual differences in part reflect the state of hypothalamic pituitary adrenal (HPA) regulation (Keller-Wood and Dallman, 1984), in turn modulated by antecedent events. However, cortisol is ubiquitous, and thus regulation serves diverse roles, for example, as an immune regulator (McEwen et al., 1997), as a promoter of energy mobilization (Sapolsky, 1992), and a potentiator of vascular catecholamine action (Walker and Williams (1992)). Recent evidence has shown that cortisol responding is not unidirectional (Heim et al., 2000), and hypocortisolism describes a profile where there is a reduced mean output, hyporeactivity, enhanced negative feedback, or reduced receptor sensitivity. Munck et al. (1984) suggested that cortisol promotes equilibrium during stress (an allostatic agent), preventing an overshoot in the defense reaction and conserving resources. But one caveat is that serving one homeostatic goal may ultimately compromise another (e.g., potentiating catecholamine vasoconstriction), and thus generate allostatic load (McEwen and Seeman, 1999). Hypocortisolism as a component of the PTSD response profile (Yehuda et al.,

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1993), may be functionally analogous to the inadequate response allostatic load profile described by McEwen and Seeman (1999).

Variation in the patterns of cortisol responses represents an interesting challenge for the analysis of individual differences. Firstly, change scores are insensitive to changes that would have occurred simply as a function of diurnal variation, a problem also for response algorithms such as area under the curve. As the diurnal pattern is absent or unstable in almost 50% of individuals (Smyth et al., 1997), sampling on a control day is not reliable. Variation in the timing of responses also presents a problem (Deinzer et al., 1997; Richter et al., 1996), as does variation in the direction of the cortisol change (Heim et al., 2000; Rose and Fogg, 1993). Lastly, Kirschbaum et al. (1995) found that reactivity across five repeated daily stress sessions did not habituate in only 35%.

Generalization of responses across stress axes

Some studies have found that high cardiovascular reactors exhibit larger adrenocortical responses (Cacioppo et al., 1995; Sgoutas Emch et al., 1994). However, Lovallo et al. (1990) found this was limited to aversive stressors. al'Absi et al. (1994) found that borderline hypertensives showed larger cortisol responses than normotensives to continuous but not intermittent stress tasks. Thus, greater autonomic reactivity appeared to be associated with greater cortisol reactivity, but only for certain stress task characteristics.

Affect, coping, and recent stress exposure, and their association with cortisol responses

Various factors may contribute to inconsistent findings, including operationalization of cortisol activity (e.g., level, reactivity), moderating factors (e.g., coping styles or recent stress exposure history). Ellenbogen et al. (2001) found depression was differentially associated with stress response change scores (negatively correlated) and the recovery level (positively correlated). Roy et al. (2001) found cortisol recovery (recovery-stress) was not associated with depression or anxiety, and Young and Nolen-Hoeksema (2001) found no association between cortisol responses and tendency to ruminate (passive and repetitive focus on distress). Pruessner et al. (1997) raise a methodological issue, finding reliable associations only when cortisol responses were averaged over multiple sessions. Scarpa and Luscher (2002) found self-esteem differentially modulated the association with depression, a cortisol fall predicting low self-esteem depression, an increase predicting high self-esteem depression, and the highest depression scores predicted by a cortisol fall and low self-esteem. A number of studies have reported a negative association between measures of recent stress exposure and current cortisol activity, particularly where there is extreme varia-

tion in exposure to stress (e.g., PTSD, Yehuda et al., 1993; combat Bourne et al., 1967), but also in response to relatively common fluctuations in daily life stress exposure (e.g., Roy et al., 2003). Coping (disengagement) has also been implicated in the hypocortisolism response to stress exposure described in PTSD (Mason et al., 2001).

The primary aim of this paper was to explore sources of individual difference in the laboratory stress salivary cortisol response. The presumption that cortisol levels increase under conditions of stress seems to be violated by results from several studies. It is hypothesized that laboratory stress will be associated with an increase in cortisol in some participants, little change in some, and a reduction in the cortisol level in others. Analyses explore whether cortisol responses were associated with cortisol levels before the stress challenge. The study investigated whether changes in cortisol activity were associated with changes in cardiovascular and mood responses. Due to the inconclusive nature of previous studies, it was hypothesized that there would be no generalized reactivity pattern across measures (cortisol, cardiovascular, and mood responses) and that change scores would be uncorrelated. Finally, the study investigated the association between cortisol activity and measures of affect, coping, and recent stress exposure.

Methods

Participants

Ninety male probationary firefighters (age range, 19–32) took part in the study soon after completing basic training, although the analyses in this paper are based upon 82 participants, eight being removed due to incomplete data. Female probationary firefighters were not included in the study as they represented less than 1% of the training cohort at the time of recruitment.

Measures

Cortisol

Unlike electrophysiological response measures (e.g., ECG), which are relatively immediate, and which can be evaluated in real time, the time course for cortisol responses is in minutes, and as sampling is intermittent, this means that sampling must be conducted with sufficient frequency, and over a sufficient time, so as to capture responses being generated at different rates. In the present study, samples were collected at 10-min intervals across the protocol for 1 h following the onset of the first stress task.

A detailed description of sample collection, handling, and cortisol determination can be found in Roy et al. (1994). Briefly, saliva samples were collected using cotton dental rolls (Salivettes, Sarstedt). Shirtcliff et al. (2001) reported that cotton dental rolls artificially inflated levels of some biomarkers (testosterone, DHEA, progesterone, and estra-

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