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Acute cortisol administration modulates EEG alpha asymmetry in volunteers: relevance to depression

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Abstract

The acute effects of cortisol (35 mg) administration in 11 healthy male volunteers on resting frontal EEG asymmetry measured in the alpha band were investigated, using a within-subjects double-blind design. Results were indicative of a relative increase of right frontal activity with cortisol. This pattern of activity is similar to the deviant pattern that has been reported in patients suffering from depression, a condition often accompanied by elevated plasma cortisol levels. The significant effect on frontal asymmetry provides convergent support for our hypothesis, based upon previous results, that sustained (>30 minutes after stress termination) relative high levels of cortisol inhibit approach motivation.

Keywords: Cortisol; EEG; Frontal asymmetry; Approach; Avoidance; Depression risk factor

1. Introduction

Sustained (>30 minutes after stress termination), relatively high levels of the glucocorticoid cortisol (relative to e.g., (nor)epinephrine) are associated with uncontrollable stressors (Dickerson and Kemeny, 2004; Levine et al., 1989) and temperamental

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inhibition (Stansbury and Gunnar, 1994). In a meta-analysis of 208 laboratory studies of acute psychological stressors, conditions capable of eliciting cortisol responses were uncontrollable stress and social-evaluative stress, when methodological factors and other stressor characteristics were controlled for (Dickerson and Kemeny, 2004). Tasks containing both uncontrollable and social-evaluative elements were associated with the largest cortisol and adrenocorticotropin hormone changes and the longest times to recovery (al least 40 minutes longer compared to other types of tasks). These findings are consistent with the animal literature on the physiological effects of uncontrollable social threat and contradict the belief that cortisol is responsive to all types of stressors. From animal studies, glucocorticoids are known to facilitate inhibitory behaviors like passive avoidance learning and freezing, the animal analog to extreme behavioral inhibition in humans (see Korte, 2001). In contrast to the sustained cortisol response, quick, strong cortisol responses coupled with rapid recovery often are part of the mobilization process of active coping (Sapolsky et al., 2000).

Cortisol has been hypothesized to facilitate the functioning of (e.g., serotonergic, cholinergic) systems involved in the inhibition of approach and active coping both in the case of uncontrollability and temperamental inhibition (Laborit, 1976; Brown et al., 1996; Stansbury and Gunnar, 1994). This may be an adaptive mechanism in which sustained high levels of cortisol have a feedback function signaling situations in which approach may not be productive or even dangerous. This idea may be seen as an extension of the role of stress-level cortisol in counter-regulating acute bodily stress responses, preventing those responses from becoming harmful themselves (Munck et al., 1984). Extending this functionality to the behavioral domain, we suggested (Tops et al., 2003b) that sustained relatively high levels of cortisol may inhibit approach and acute active coping, preventing these behavioral responses from becoming harmful in situations of uncontrollability (see also Laborit, 1976).

It is interesting to note that, like elevated glucocorticoid levels, dopamine inhibition in the nucleus accumbens septi after the initial activation in response to acute stress, has been associated with uncontrollability (Cabib and Puglisi-Allegra, 1994). Some animal studies have suggested that stress-induced mesocortical dopamine activity proceeds from an initial left brain bias to a right brain bias as the stress is sufficiently prolonged and perceived as uncontrollable (Carlson et al., 1988; 1991; Sullivan and Szechtman, 1995). When rats are first exposed to an uncontrollable stressor (such as being restrained) they engage in vigorous behavior to control or ‘‘cope’’ with the stressor. This behavior is accompanied by a greater activity of dopamine (as indicated by increased metabolite levels) on the left side of the prefrontal cortex. However, at later time points, when coping behavior has ceased (e.g., when a restrained rat has stopped struggling), there is more dopamine metabolism on the right side of the prefrontal cortex (Carlson et al., 1991).

Glucocorticoids have been shown to increase dopamine activity in the animal brain in various experimental paradigms (reviewed by Schatzberg et al., 1985). However, animal research has also shown that glucocorticoids attenuate frontal dopaminergic function. Acute (Thomas et al., 1994) as well as chronic (Lindley et al., 1996) exogenously administered corticosterone has been found to reduce extra-cellular dopamine, respectively dopamine content, in the frontal cortex. Sustained hypercortisolism also diminishes dopamine release in the nucleus accumbens of rats, an important component of the