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Effects of mood and rumination on cortisol levels in daily life: An ambulatory assessment study in remitted depressed patients and healthy controls

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Summary The influence of naturally occurring emotional and cognitive experiences on hypothalamic—pituitary—adrenal axis (HPAA) activity is still underinvestigated, particularly in clinical populations. The present study examined effects of mood and rumination on cortisol levels in daily life in remitted depressed patients with recurrent episodes or a chronic precourse (n = 31) and healthy controls (n = 32). Ambulatory assessment of subjective variables (valence, calmness, energetic-arousal, ruminative self-focus), daily stressors, and saliva cortisol samples was performed five times a day on two consecutive workdays, whereby cortisol was collected 20 min after the subjective assessments. In addition, depressive symptoms and trait rumination (brooding, reflection) were measured retrospectively. Multilevel models revealed that remitted depressed patients showed lower cortisol activity compared to healthy controls. Depressive symptoms and trait rumination did not predict HPAA activity, whereas, by controlling for daily stressors, higher daily means of ruminative self-focus and lower daily means of valence, energetic arousal and calmness were associated with higher daily cortisol levels. Separate analyses per group revealed that mean daily ruminative self-focus predicted higher cortisol in both samples. In contrast, lower daily means of calmness, but also of valence and energetic arousal, were significantly linked to higher cortisol output only in healthy controls, but not in the patient sample. These findings indicate that naturally occurring rumination and low mood are associated with increased

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activation of the HPAA in daily life. Moreover, our data revealed a potentially reduced mood—cortisol coupling in remitted recurrent depression, possibly indicating that during the course of recurrent depression HPAA activation might become less responsive toward subtle emotional experiences in natural contexts.

1. Introduction

Major depression as a stress-related disorder has been linked to alterations of the hypothalamic—pituitary—adrenal axis (HPAA). In acute depressive states, a substantial subgroup of patients shows hyperactivation of the HPAA with elevated basal cortisol (Stetler and Miller, 2011). However, cortisol downregulation has also been observed, indicating the existence of different depression pathophysiologies (Fries et al., 2005; Lamers et al., 2012). Furthermore, consistent with the notion that HPAA abnormalities are not just state-dependent but linked to depression vulnerability (Pariante and Lightman, 2008), there is evidence that after remission of acute depression HPAA activity remains dysregulated. Here, empirical findings involve both reduced and increased basal cortisol levels. Young et al. (2000) used repeated assessments of morning (8 am) and evening (before bedtime) saliva cortisol samples over 14 days and identified a trend for higher mean cortisol levels in monozygotic twins with a history of depression compared to never-depressed twins. Similarly, Lok et al. (2012), by using two consecutive morning (8 am) and one single evening (10 pm) sample showed that highly recurrent remitted depressed patients displayed higher mean cortisol concentrations than nondepressed individuals. In contrast, Ahrens et al. (2008) collected individual morning (8 am) and afternoon (4 pm) samples over seven days and reported reduced morning cortisol in women with remitted depression compared to healthy women, whereas afternoon cortisol levels did not differ between groups. Furthermore, lower cortisol levels have been linked to indicators of a more severe previous depression course. Gex-Fabry et al. (2012) examined average cortisol exposure over the day by collecting seven saliva cortisol probes per day at six occasions during a 14 months period and found that a longer illness duration predicted lower cortisol output (AUC). Similarly, in the study by Lok et al. (2012), more previous depressive episodes predicted lower mean cortisol output in remitted depressed patients. Finally, Bockting et al. (2012) found that lower morning cortisol (sampled at 8 am on two consecutive days) predicted earlier recurrence of depression. To explain hypocortisolism in recurrent depression, it has been suggested that lower cortisol levels might occur after prolonged hyperactivation of the HPAA due to its eventual downregulation (Ahrens et al., 2008). While HPAA downregulation can have beneficial effects by protecting against allostatic overload (Fries et al., 2005; Miller et al., 2007), hypocortisolism also points to adrenal exhaustion and can imply detrimental effects, for example through overactive immune responses and inflammation (Raison and Miller, 2003).

Very little is known about factors that influence HPAA functioning in daily life. However, the identification of such factors might help to explain the large inter- and intraindividual variability in cortisol activity and to dissolve related diverging findings. While laboratory stressors have been rather consistently identified to elicit cortisol increases (Kudielka and Wüst, 2010), few studies have investigated possible associations of naturally occurring emotional and cognitive processes with cortisol activity in real life. Recently, Kudielka et al. (2012) have stressed the importance to transfer research on HPAA regulation from the lab to ecologically valid contexts, and there is evidence that cortisol responses in the lab do not necessarily converge with cortisol responses in natural settings (Wolfram et al., 2012). To study HPAA regulation in daily life, ambulatory assessment (AA) methods can be applied that capture real-time, real-world experiences, preferably with electronic devices (Trull and Ebner-Priemer, 2013). Using AA, it becomes thus possible to repeatedly measure both subjective experiences and cortisol activity over the day in real life which enhances reliability and generalizability of identified associations.

A small number of previous AA studies have addressed possible influences of emotional experiences on cortisol activity during normal daily life. These studies found that higher momentary negative affect was linked to higher cortisol in community samples (Smyth et al., 1998; Hanson et al., 2000; Adam, 2006; Jacobs et al., 2007; Matias et al., 2011), in pregnant women (Giesbrecht et al., 2012), and in siblings of psychotic patients (Collip et al., 2011). To our knowledge, there is only one clinical study that compared associations between affect and cortisol in daily life in acutely depressed patients and healthy controls (Peeters et al., 2003). This study identified similar cortisol levels over the day in the two groups. Moreover, negative affect predicted higher cortisol levels in both groups; however, this effect was marginally weaker in patients compared to controls. Furthermore, patients also showed reduced cortisol reactivity to daily events compared to controls in this study. Thus, this study suggests that in depressed individuals HPAA activity in response to daily experiences is reduced. Similar findings of reduced stress—cortisol coupling in daily life have been reported in highly recurrent bipolar patients (Havermans et al., 2011). Thus, specific alterations of psychoneuroendocrine regulation within the flow of subjective experiences in daily life might represent an important pathophysiologic marker of depressive and bipolar disorders. However, systematic research on this topic is still missing.

In addition to emotional experiences, cognitive processes, specifically rumination, might also influence cortisol activity. Rumination has been defined as repeatedly thinking about one's negative mood and its possible causes and consequences, and has been postulated as an important cognitive vulnerability factor for depression (Nolen-Hoeksema et al., 2008). Longitudinal studies indicate that rumination predicts later depression particularly in nonclinical samples (Nolen-Hoeksema et al., 2008; Huffziger et al., 2009). According to the perseverative cognition hypothesis by Brosschot et al. (2005), rumination entails an ongoing mental representation of a stressor, which should be associated with prolonged