



## Emotion regulation as a predictor of the endocrine, autonomic, affective, and symptomatic stress response and recovery



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### ABSTRACT

Stress is associated with the development of mental disorders such as depression and psychosis. The ability to regulate emotions is likely to influence how individuals respond to and recover from acute stress, and may thus be relevant to symptom development. To test this, we investigated whether self-reported emotion regulation predicts the endocrine, autonomic, affective, and symptomatic response to and recovery from a stressor. Social-evaluative stress was induced by the Trier Social Stress Test (TSST) in  $N = 67$  healthy individuals (53.7% female,  $M_{age} = 29.9$ ). Self-reported habitual emotion regulation skills were assessed at baseline. We measured salivary cortisol, heart rate, negative affect, state depression and state paranoia at three time points: pre-TSST, post-TSST, and after a 10 min recovery phase. Repeated-measures ANOVA showed all indicators to significantly increase in response to the stressor ( $p < .001$ ) and decrease during the recovery phase ( $p < .001$ ), except for salivary cortisol, which showed a linear increase ( $p < .001$ ). The habitual use of maladaptive emotion regulation (e.g., rumination, catastrophizing) significantly predicted an increased affective and reduced cortisol response. Adaptive emotion regulation (e.g., acceptance, reappraisal) was not predictive of the stress response for any of the indicators. Neither type of emotion regulation predicted response during the stress recovery phase. Individuals who habitually resort to maladaptive emotion regulation strategies show a stronger affective and a blunted endocrine stress response, which may make them vulnerable to mental health problems. However, further research is needed to identify the full scope of skills required for effective stress-regulation before this knowledge can be used to develop effective prevention programs.

### 1. Introduction

Vulnerability-stress-models of psychopathology propose that vulnerable individuals show an increased sensitivity to acute stressors that is reflected in stronger affective, endocrine, and autonomic response, possibly accompanied by a “symptomatic” response. Dating back to the 1980’s and earlier, this model has been used to explain the development of mental disorders, such as depression (e.g., Beck, 1987; Bebbington, 1987) and psychosis (e.g., Zubin and Spring, 1977), which has inspired research on the phenomenon of stress-sensitivity in the context of these mental disorders.

In order to investigate the stress-sensitivity, researchers have applied different methods ranging from experimental designs, where the stress is induced in the laboratory, to the experience sampling method (ESM), where data is collected via electronic diaries in everyday life. In one ESM study, Myin-Germeys et al. (2003) found that both individuals with psychosis and those with depression exhibit elevated emotional sensitivity to daily stressors. Similarly, individuals at risk for psychosis

show an increased emotional response to stressors in everyday life (Lataster et al., 2009) and an increased endocrine response to daily stress (Collip et al., 2011). Experimental studies have also found both emotions and symptoms to increase in response to stressors in individuals with psychosis (e.g., Ellett et al., 2008; Freeman et al., 2015; Lincoln et al., 2015b; Veling et al., 2016). Hence, there is solid empirical evidence that psychosis and depression are associated with stronger emotional responses to stress, which tend to translate into symptoms.

This raises the question by which specific vulnerability factors the emotional stress response is being driven. It seems intuitive to expect that one such vulnerability factor could be impaired emotion regulation (ER). In some regards ER is a similar concept to coping (Compas et al., 2014; Wang and Saudino, 2011). However, in contrast to coping, which has been defined as “constantly changing cognitive and behavioral efforts to manage specific external and/or internal demands that are appraised as taxing or exceeding the resources of the person” (Lazarus and Folkman, 1984, p. 141), the concept of ER specifically refers to “the

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process by which individuals influence which emotions they have, when they have them, and how they experience these emotions” (Gross, 1998, p. 275). Thus, ER is more narrowly defined than coping and is likely to be the more suitable concept when it comes to understanding the emotional stress response that precedes psychotic symptoms.

In healthy individuals, different ER strategies were found to be important in relation to different aspects of subjective stress responses. In particular, the habitual use of rumination (i.e., focusing attention on the situation and the emotion) was associated with increased negative mood-congruent thinking and with increased endocrine reactivity or delayed recovery (e.g., Thomsen et al., 2003; Zoccola and Dickerson, 2012; Zoccola et al., 2008). Studies also demonstrate negative effects on well-being when negative emotions are suppressed (i.e., inhibiting behaviors associated with emotional responding; e.g., Butler et al., 2003; Gross and John, 2003), whereas accepting emotions was found to have beneficial effects on mental health (Bond and Bunce, 2003). Empirical results on habitual reappraisal are mixed, with some studies reporting a negative association with self-reported negative affect and physiological responses (e.g., Carlson et al., 2012; Memedovic et al., 2010; Mauss et al., 2007) suggesting a resilience function of reappraisal. In contrast, other studies found no effect of reappraisal on self-reported stress and a positive association with physiological responses (e.g., Denson et al., 2014; Lam et al., 2009). Although previous work indicates that specific ER strategies such as rumination or reappraisal are relevant to stress responses and well-being in healthy individuals, it also suggests that different strategies tend to load on the same latent factor rather than representing separate constructs (Aldao and Nolen-Hoeksema, 2010; Garnefski et al., 2001). As a consequence, different strategies are often subsumed into “maladaptive” strategies if their habitual use is considered to be less advantageous (e.g., rumination, suppression), and into “adaptive” strategies if their habitual use is considered as more advantageous (e.g., reappraisal, acceptance). It has been shown that the habitual use of maladaptive strategies is related to negative outcomes such as psychopathology, whereas the habitual use of adaptive strategies has been found to protect against psychopathology (Aldao et al., 2010).

Specifically, increased habitual use of maladaptive strategies has been found to be associated with depression (Aldao et al., 2010) and psychosis (O’Driscoll et al., 2014). Furthermore, several studies point to the lack of habitual use of adaptive strategies as a risk factor for psychosis and depression (e.g., Joormann and Stanton, 2016; Kimhy et al., 2012; Lincoln et al., 2015a,b; Perry et al., 2011; van der Meer et al., 2009). Furthermore, in an at-risk sample lower levels of habitual use of adaptive and higher levels of maladaptive ER accounted for the increase in paranoid beliefs after social exclusion (Lincoln et al., 2017b). Thus, increased habitual use of maladaptive and limited habitual use of adaptive ER could alter the stress response and therefore represent a risk factor for the development of depression and psychosis in the longer term.

However, there are two pitfalls of the available research. One is that a stress response includes various indicators, such as an endocrine, autonomic, affective, and symptomatic response. So far, however, most studies have focused on either subjective stress indicators or cortisol responses alone. Few studies have concurrently assessed numerous stress indicators and those that did have found some indicators (e.g., subjective stress) to be more responsive to experimentally induced stress than others (e.g., cortisol) in people with psychosis or depression (e.g., Lincoln et al., 2015a,b). This aligns with studies pointing to a dysregulated hypothalamic-pituitary-adrenal (HPA) axis in these disorders (Chaumette et al., 2016; Shah and Malla, 2015; Stetler and Miller, 2011). Other studies point to compensatory patterns of different stress indicators (Ali et al., 2017). This indicates that a pathological stress response could be characterized by a complex pattern of hyper- and hyposensitivity of different stress indicators, which would require a comprehensive, concurrent measurement of various stress indicators in order to be detected.

The other pitfall is that although the definition of emotion regulation also includes influencing the *duration* of the emotion, research has mostly focused on the acute stress response. This is problematic, as a prolonged recovery from a stressor could represent an additional burden for individuals. The few studies that considered this aspect have been summarized in a review by Zoccola and Dickerson (2012) who discuss the mixed findings on whether or not the increased rumination is associated with a delayed cortisol decrease after the cessation of a stressor in healthy individuals. Furthermore, Lewis et al. (2017) found that in healthy individuals, the adaptive ER strategy reappraisal was associated with greater cortisol recovery after a stressor. Thus, there is some indication that ER strategies could have an impact on the recovery from a stressor, but this evidence is limited to the endocrine stress recovery.

The present study thus extends on the existing research in this field by testing the hypothesis that higher scores in habitual maladaptive and lower scores in habitual adaptive ER skills will predict 1) a stronger acute stress response and 2) a weaker recovery from a social evaluative stressor, which will be evident across a broad spectrum of stress indicators, including salivary cortisol, heart rate, affect, and symptoms.

## 2. Methods

### 2.1. Participants

Participants were recruited by print adverts in the facilities of the Universität Hamburg, churches, job centers, residential homes in Hamburg, and via several internet postings (e.g., Facebook, Stellenwerk, Ebay-Kleinanzeigen). To be included, the participants needed to be 18 years or older, have sufficient command of the German language, have no mental disorder and be able to provide informed consent. Additional exclusion criteria involved factors that could possibly influence the physiological stress response: any kind of medication, hormonal contraception, diagnosed cardiac or thyroid disorders, smoking, drug use, as well as eating, caffeine intake or sports within two hours prior to testing.

The final sample consisted of 67 participants (53.7% female,  $M_{age} = 29.94$ ,  $SD_{age} = 12.25$ ). About one third of the participants (29.9%) reported working full-time and 58.2% were students. The majority of the participants reported having a university degree (36%) or a general qualification for university entrance (A-level equivalent, 35%), 18% reported having a lower school degree and 11% reported having a professional training degree. Participants’ mean body mass index (BMI) was  $M_{bmi} = 23.18$  ( $SD_{bmi} = 3.61$ ).

### 2.2. Design and procedure

The procedure is depicted in Fig. 1. The first part of the study consisted of a questionnaire battery that the participants completed online. After this, they were invited to participate in the second part of the study that took place at the Universität Hamburg. All appointments took place between 10AM and 7PM. After arriving at the lab, the participants were informed about the procedure and signed an informed consent, after which an ambulatory electrocardiogram (ECG) was attached to the participants’ chest, pre-TSST heart rate was measured (HRT1; see Fig. 1) and the first (i.e., pre-TSST) assessment of salivary cortisol, negative affect, state depression and paranoia took place (t1; see Fig. 1). Following this, the participants completed cognitive assessment tests (ca. 30 min). Next, all participants underwent the Trier Social Stress Test (TSST; Kirschbaum et al., 1993). The test consisted of an anticipation period (3 min) where the participants were asked to prepare a speech and a test period where the participants had to deliver a speech (5 min) and then perform mental arithmetic tasks (5 min) in front of an audience. During the speech task, the second measurement of heart rate took place (HRT2; see Fig. 1). Immediately after the stress manipulation, the second assessment of salivary cortisol, negative

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