The Effects of Acute Stress Exposure on Cognitive Emotion Regulation: Psychophysiological Studies

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ACC:	anterior cingulate cortex
CPT:	cold pressure task
DA:	dopamine
dACC:	dorsal anterior cingulate cortex
dlPFC:	dorsolateral prefrontal cortex
dmPFC:	dorsomedial prefrontal cortex
EEG:	electroencephalography
EMG:	electromyography
ER:	cognitive emotion regulation
ERP:	event related potential
fMRI:	functional magnetic neuroimaging
HPA:	hypothalamic-pituitary-adrenal axis
LC:	locus coeruleus
LPP:	late positive potential
NA:	noradrenaline
PFC:	prefrontal cortex
SNS:	sympathetic nervous system
SECPT:	Socially Evaluated Cold Pressure Task
TSST:	Trier Social Stress Test
vlPFC:	ventrolateral prefrontal cortex
vmPFC:	ventromedial prefrontal cortex
WM:	working memory

Abstract

Cognitive emotion regulation (ER) is an effective way to adaptively alter emotional responses. However, in everyday life situations, we often fail to successfully regulate our emotional responses. ER relies on executive functioning and the involvement of the prefrontal cortex, which are known to be impaired by the extensive effects of stress. In this context, recent research has suggested that stress might also play a determinant role in ER failure. Nevertheless, the current state of studies has revealed heterogeneous results with respect to the effects of stress on ER. Therefore, two independent consecutive studies were conducted using psychophysiological measures in order to test whether and to what extent stress alters emotion responding and affects different strategies of ER.

In Study 1, 50 healthy participants were either exposed to a stressor (n=25) or to a control task (n=25). Subsequently, subjects either had to view or regulate (cognitive reappraisal) neutral and negative stimuli. The ER task was divided into an early (0-20 min) and a late stress phase (20-40 min) in order to investigate the temporal dynamics of the stress response. Study 2 aimed to compare different ER strategies in the face of acute stress exposure. 50 healthy subjects were also assigned to a stressor (n=25) or a control condition (n=25). Afterward, the participants either had to view or regulate (cognitive reappraisal and distraction) neutral and negative emotional stimuli. In both studies, an identical multimodal assessment was used, comprising subjective emotional self-reports, electroencephalography, and electromyography. Additionally, salivary cortisol, α-amylase, and negative affect were assessed as a marker of the stress response. Study 1 revealed a stress-related impairment of cognitive reappraisal in the late stress phase, while there was no increase in emotional reactivity. In Study 2, stress exposure led to an impairment of reappraisal, while distraction was not affected. As with Study 1, there was no increase in emotional reactivity due to acute stress exposure. In sum, the results indicate that acute stress exposure can lead to an impairment of cognitive reappraisal while distraction is not negatively affected, probably due to the impairment of mediating executive functions. Assumingly, reappraisal compared to distraction might be more vulnerable to effects of acute stress exposure as it requires higher cognitive costs. Altogether, the present findings provide a more detailed understanding of ER failure under acute stress and might contribute to explanatory approaches of stress-related mental disorders and psychopathological symptoms.

1. Introduction

Cognitive emotion regulation (ER) plays a crucial role in the development, maintenance, and therapy of a variety of mental disorders such as depression, anxiety disorders, substance- and alcohol-related disorders, eating disorders, somatoform disorders, and borderline personality disorder (e.g., Berking & Wupperman, 2012). While some types of ER have mental health promoting effects, others have not. For instance, suppression and avoidance have been linked to psychopathology, whereas cognitive reappraisal has been associated with long-term positive outcomes, such as mental and physical health (Aldao et al., 2010; Gross & John, 2003). To date, the majority of ER research has focused on the association of ER and psychopathology (e.g., Aldao et al., 2010; Sheppes, Suri, & Gross, 2015; Werner & Gross, 2010), the analyses of the effectiveness of different ER strategies (e.g., Webb, Miles, & Sheeran, 2012), neural correlates (e.g., Buhle et al., 2014; Goldin, McRae, Ramel, & Gross, 2008; Goldin et al., 2008; Taylor & Liberzon, 2007) and modes of action (e.g., Gross, 1998, 2015).

Often enough, we fail to successfully regulate our emotional responses to everyday life situations – especially in the face of pervasive stress. In line with this, negative affect, stress, and fatigue have been proposed to play a key role in self-regulation failure (Baumeister & Heatherton, 1996; Heatherton & Wagner, 2011). However, less is known about whether and to what extent stress leads to ER failure. Stress is known to have a severe impact on the functionality of the prefrontal cortex (PFC) and the amygdala (e.g., Arnsten, 2009). The PFC determines executive functions that play a fundamental role in successful ER, such as working memory (WM), while the amygdala plays a key role in emotional processing. In accordance, various mental disorders are worsened by stress exposure and are related to PFC impairment (Arnsten, 2009; Mazure, 1995). However, the research regarding the effects of stress on ER is in an early stage and has revealed inconsistent results so far. Thus, the association between stress and ER failure remains unclear and hypothetical.

1.1. Emotion regulation

1.1.1. Emotions and emotion regulation: basics and measurement methods

Emotions employ changes in subjective experience and peripheral physiology in order to initiate tendencies to behave in specific ways (Mauss et al., 2005). The emotion-related behavioral changes comprise modifications in facial expressions and posture as well as situation-specific instrumental behaviors (Ekman, 1971). Emotions are generated by the operation of valuation systems. According to Gross (2015), valuation systems are distinguished by internal or external states of the world and perceptions of those states.

During the valuation, each state receives a value based on benefits and costs, which leads to the computation of a discrepancy between current and desired states. If the discrepancy passes a certain threshold, a mental and/or physical action will be initiated to change the state of the world. The change leads to the activation of a second cycle and so on. Multiple valuation systems can be activated at the same time and be highly interactive with each other. ER refers to the interaction of two consecutive valuation systems, particularly, when the output of a first-level valuation system (emotion generation) is either negatively or positively evaluated by a second-level valuation system. The activation of the second-level system is intended to modify the output of the first-level system in order to reduce the discrepancy between positive and negative states (Figure 1a). The second-level valuation system can influence the first-level valuation system with five different ER processes (Figure 1b), which can be divided into *antecedent-focused* and *response-focused* strategies (Gross, 1998, 2015). Antecedent-focused regulation strategies occur early in the valuation cycle and emotion

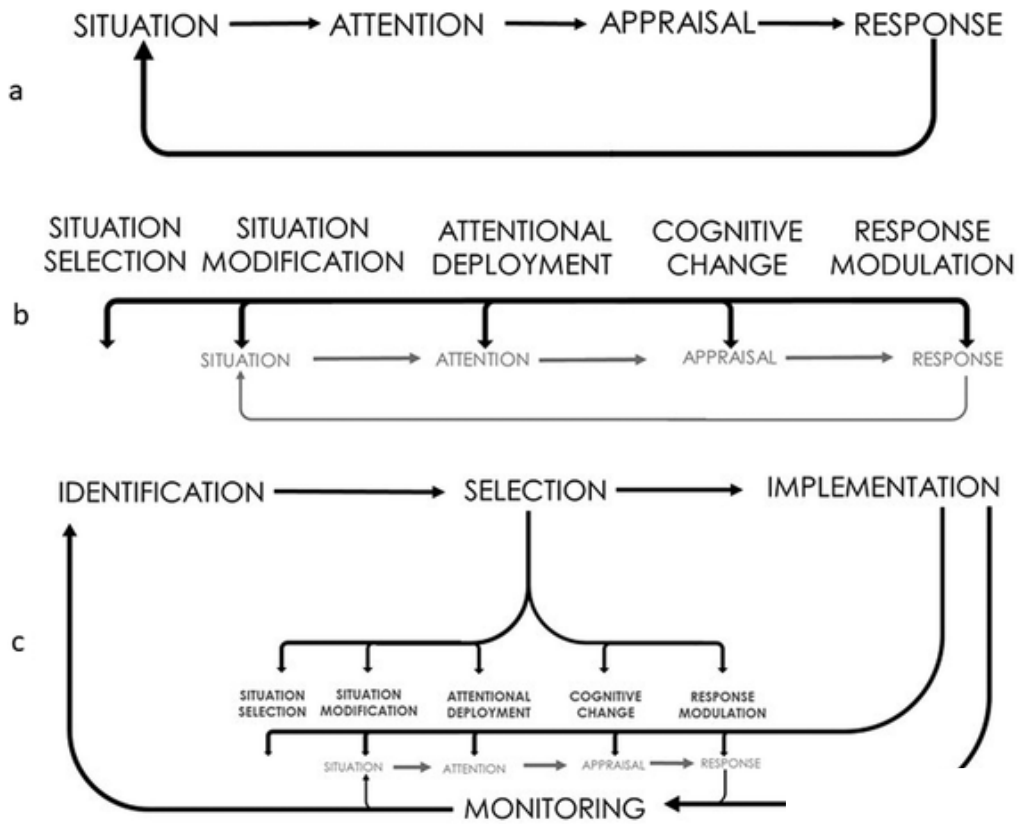

Figure 1. a) Sequential model of emotion generation. **b)** Families of ER strategies. **c)** Process model of ER. (Figure taken from McRae & Gross, 2020)

generation process. They comprise the strategies of situation selection (avoiding situations, stimuli, or people), situation modification (modifying a situation to change emotional impact), attentional deployment (focusing on different aspects in a situation), and cognitive change (modifying the cognitive evaluation of a stimulus or situation). Response-focused ER is initiated in a relatively later valuation cycle once an emotion is fully generated and includes strategies of response modulation (manipulation of response tendencies by reducing physiological, or behavioral response tendencies). Further, Gross (2015) defines three regulatory valuation systems: *identification, selection, and implementation* (Figure 1c). The identification valuation system includes the decision of whether to regulate the emotion or not. The selection stage valuation system defines the ER strategy (e.g., cognitive change, response modulation), while the implementation valuation system determines the specific regulatory strategy (e.g., reappraisal within cognitive change).

The ER strategies of distraction (attentional deployment) and cognitive reappraisal (cognitive change) have received the most attention in previous research (e.g., Kanske, Schönfelder, Forneck, & Wessa, 2015; Lois et al., 2017; McRae et al., 2010; Scheibe, Sheppes, & Staudinger, 2015; Schönfelder, Kanske, Heissler, & Wessa, 2014; Sheppes & Meiran, 2007; Strauss, Ossenfort, & Whearty, 2016; Thiruchselvam, Blechert, Sheppes, Rydstrom, & Gross, 2011). Distraction reflects the attempt to focus one's attention on more non-arousing and non-emotional parts of a situation. Cognitive reappraisal aims to modify the affective impact by changing its semantic meaning in form of an altered interpretation of an emotional stimulus or situation (Gross, 2013). Both strategies show great overlap in frontoparietal activation patterns, such as the medial and lateral PFC including the dorsal anterior cingulate cortex (dACC) and the inferior parietal lobe (Buhle et al., 2014; Kanske, Heissler, Schönfelder, Bongers, & Wessa, 2011; McRae et al., 2010). However, distraction is associated with more right prefrontal and parietal regions, whereas reappraisal depends on more medial, ventrolateral, and orbitofrontal PFC (Kanske et al., 2011; McRae et al., 2010). Both strategies proved to modulate bilateral amygdala activity, which is related to emotional responding (Buhle et al., 2014; Kanske et al., 2011; McRae et al., 2010). A meta-analysis by Webb, Miles, and Sheeran (2012) revealed that reappraisal has proven to be the most effective strategy in decreasing subjective negative emotion. Distraction leads to greater amygdala downregulation when compared to reappraisal.

There are several ways to measure emotional processing and ER, whereby subjective self-reports, electroencephalography (EEG), and facial expressivity have proved to represent valid and reliable markers. Subjective self-reports of the current emotion experience represent a reliable measure of emotional processing and ER (Mauss & Robinson, 2009).

However, self-reports have been criticized as subjective measures that can be affected by confounding variables (e.g., social desirability) and not all individuals are aware and/or capable of reporting emotional states e.g., in the case of alexithymia (Paulhus & John, 1998). EEG and facial expressivity represent more objective measures of emotional responding. In this regard, event-related potentials (ERPs) reflect cognitive and affective processes with exceptional temporal resolution but low spatial resolution. ERPs (e.g., P300; Late Positive Potential, LPP) have been used in a variety of studies and proved to be a valid marker for the measurement of emotional processing (e.g., Dennis & Hajcak, 2009; Foti & Hajcak, 2008; Hajcak et al., 2010; Schönfelder et al., 2014; Thiruchselvam et al., 2011). The P300 is linked to stimulus salience and attention, which emerges at around 300-500 ms post-stimulus presentation and is dominant at midline parietal sites (Hajcak et al., 2010). In this context, emotional compared to non-emotional stimuli lead to a greater P300, as emotional stimuli are assumingly automatically processed as task-relevant (Hajcak et al., 2010). Thus, the P300 represents a valid marker of emotional reactivity (Hajcak et al., 2012). Another neurophysiological marker of emotional processing is the LPP, which becomes evident at 300 ms post-stimulus presentation over posterior recording sites (Hajcak et al., 2009) and can last for the entirety of the stimulus presentation (e.g., Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Foti & Hajcak, 2008; Hajcak & Olvet, 2008). The LPP is associated with the emotional intensity of a stimulus and linked to emotional arousal (Hajcak et al., 2010; Schupp et al., 2006). Facial expressivity by means of the measurement of facial electromyography (EMG) represents a valid measure to assess emotional valence (Mauss & Robinson, 2009; vanOyen, Witvliet & Vrana, 1995). The main targeted muscle groups are the corrugator supercilii and the zygomatic major. The corrugator activity increases with the unpleasantness of affective stimuli while the corrugator activity decreases and vice versa (Bradley & Lang, 2000). EMG measurement of facial expressivity has already been used in a variety of ER studies (e.g., Jackson et al., 2000; Pedder et al., 2016; Ray et al., 2010; Schönfelder et al., 2014).

1.1.2. Emotion regulation, psychopathology, and mental health

ER is considered to have important effects on mental health and overall well-being (Gross & Muñoz, 1995; Rottenberg & Johnson, 2007; Werner & Gross, 2010)**.** On the other hand, deficits in ER skills can lead to difficulties in monitoring, evaluating, and modifying emotional reactions (Thompson, 1994) that are associated with a broad range of psychopathological symptoms (Berking & Wupperman, 2012; Cisler et al., 2010). Moreover, these deficits in ER are closely related to mental disorders, such as depression,

anxiety, or substance abuse disorders (Campbell-Sills & Barlow, 2007; Mennin et al., 2007; Sheppes et al., 2015).

A meta-analytic review by Aldao and colleagues (2010) examined the association between ER strategies (e.g., reappraisal, suppression, rumination, avoidance) and psychopathological symptoms (e.g., anxiety, depression). The authors report that the ER strategies of suppression, rumination, and avoidance were found to be positively correlated with psychopathological symptoms with medium to large effect sizes. On the other hand, reappraisal was found to be negatively associated with general psychopathology with small to medium effect sizes. On a more superior level, Sheppes and colleagues (2015) describe key points at which difficulties in ER can lead to various psychopathologies based on the extended process model of ER (see above, Section 1.1.1). Accordingly, ER failure can occur during all regulatory stages (identification, selection, implementation). Thus, different mental disorders or psychopathological symptoms can be provoked, depending on the stage or stages in the ER failure occurs**.** For instance, failure at the perception step during the identification-stage can lead to overrepresentation of emotional events which is featured in panic attacks and could develop into a panic disorder.

Taken together, previous research clearly indicates that the use of functional ER strategies (e.g., cognitive reappraisal) plays a crucial role in the maintenance of mental health and the absence of mental disorders, while dysfunctional ER strategy use (e.g., suppression or rumination) increases the probability for psychopathological symptoms. Although a lot of research has already focused on the association between different ER strategies, psychopathology symptoms, and mental disorders, less is known about which contextual conditions and situational factors might provoke ER failure, (i.e., the unsuccessful or impaired use of functional ER strategies). In this regard, previous studies suggest that the experience of stress plays a key role in the failure of self-regulation (i.e., the ability to make plans, choose from alternatives, control impulses, and regulate behavior) (Heatherton et al., 2015; Heatherton & Wagner, 2011). In this regard, due to the negative effects of stress on self-regulation, stress-related impairments of functional ER strategies (e.g., reappraisal) might represent a potential link between stressful life events and the development of psychopathology.

1.2. Stress

1.2.1. Neurobiology and basics of stress

Over the last decades, stress has been widely studied from various perspectives. Research has revealed that stress has significant detrimental effects on various physical and

psychological variables. In this regard, stress has been associated with mental disorders such as depression and posttraumatic stress disorder (PTSD) (Marin et al., 2011), risky health behaviors such as alcohol addiction, smoking, or illegal substance abuse (Herman, 2012; Kassel et al., 2003). Moreover, stress is related to all leading physical causes of death such as strokes or cancer (Cohen, Janicki-Deverts, & Miller, 2007). Stress can be divided into absolute or relative stress. Absolute stress refers to a real threat, such as being involved in a car accident or being attacked by a wild animal. Relative stress relates to an implied threat, meaning the interpretation of a situation (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). Nowadays, absolute compared to relative stress is seldom and the majority of experienced stressors are related to relative stress. For instance, daily struggles at work, financial problems, interpersonal conflicts or even simply giving a presentation in front of one's fellow students represent typical relative stress situations.

Stress can be triggered by situations that are interpreted as novel and/or uncontrollable and/or unpredictable and/or social-evaluative threatening (Dickerson & Kemeny, 2002; Mason, 1968). On a physiological level, stress is conceptualized as a two-component response in form of a fast activity system, the sympathetic nervous system (SNS), and a slow activity system, the hypothalamic-pituitary-adrenal axis (HPA). The two components are both adaptive systems to real or implied threats or challenges, defined as allostasis. The most common allostatic responses involve the SNS and the HPA axis and represent essential components in maintaining or achieving homeostasis (McEwen, 1998). For instance, both systems lead to an increase in physiological arousal, muscle tension, respiration, heart rate and glucose production in the liver, etc., in order to provide additional physiological resources to face current challenges.

On a neurobiological level, the amygdala triggers the hypothalamus in case of a threat. The SNS is immediately activated and innervates the adrenal medulla through preganglionic sympathetic fibers and leads to the secretion of acetylcholine, which activates the release of catecholamines, mainly adrenaline (epinephrine) and noradrenaline (norepinephrine) (Tsigos & Chrousos, 2002). Additionally, a stress response triggers the locus coeruleus (LC), a cluster of noradrenaline (NA) containing neurons located in the pons, which has extensively branched axons and leads to a norepinephrinergic innervation of e.g., the neocortex, the hippocampus, and the amygdala (Benarroch, 2009). The slow-acting system (HPA) is characterized by the secretion of vasopressin and corticotropin-releasing hormone by the paraventricular nucleus of the hypothalamus. Both hormones innervate the anterior pituitary gland and stimulate the secretion of adrenocorticotropic hormone. Adrenocorticotropic hormone activates the adrenal cortex, producing a hormonal cascade

that results in the secretion of corticosteroids (Herman & Cullinan, 1997). The release of both *corticosteroids* and *catecholamines* has marked effects on brain function:

Catecholamines comprise the neurotransmitter adrenaline, noradrenaline (NA), and dopamine (DA) and have significant effects on PFC functions. For instance, NA engages α-1-, α-2A- and β-adrenergic receptors in the PFC, while having a high affinity for α-2A-adrenergic and a low affinity for α-1- and β-1-adrenergic receptors (Arnsten, 2009, 2015). In non-stress situations, NA engages to α-2A-adrenergic receptors, which strengthens PFC connections, increases PFC activity, weakens amygdala activity, and reduces tonic firing of the LC. During a stressful situation, a high level of NA impairs PFC functioning by binding on lower-affinity α-1-receptors and β-1-receptors, strengthens amygdala activity, and maintains tonic firing of NA neurons in the LC (Arnsten, Paspalas, Gamo, Yang, & Wang, 2010; Arnsten, Wang, & Paspalas, 2012; Arnsten, 2009). The effects of NA follow an inverted-U-shape, meaning too high or too low NA levels result in cognitive impairments, whereas a moderate NA level can lead to improvements (Chamberlain et al., 2006). Moreover, DA exerts similar modulatory actions like NA through the stimulation of D1-receptors in PFC (Arnsten, 2009, 2015; Berridge & Arnsten, 2015). Even mild forms of stress can induce DA release and can have an impact on cognitive functioning. DA also follows an inverted U-shape pattern (Berridge & Arnsten, 2015; Cools & D'Esposito, 2011), whereby either too high or too low DA level can lead to cognitive impairments (Butts et al., 2011; Robbins & Arnsten, 2009; Vijayraghavan et al., 2007; G. Williams & Castner, 2006).

Corticosteroids are lipophilic and thus can easily pass the blood-brain barrier and get access to the brain where they exert a broad range of molecular, structural, and functional effects through their binding on high-affinity mineralocorticoid receptors (MR) and low-affinity glucocorticoid receptors (GR) (Lupien & McEwen, 1997). MR are mainly distributed in the limbic system whereas GR are represented in both, limbic structure and PFC (Lupien et al., 2007). MR and GR regulate specific gene transcriptions which influence cell metabolism, cell structure, and synaptic transmission (de Kloet, Joëls, & Holsboer, 2005) leading to rather rapid non-genomic or slow-acting genomic effects of corticosteroids on brain functionality (Hermans et al., 2014). The MR mediate rapid non-genomic effects which activates limbic cells and promotes behavioral choices in order to quickly respond to threats, leading to an increase in hippocampal and amygdala activity (Joëls et al., 2013). MR are predominantly important for the appraisal of sensory information and the maintenance of stress-related neural-circuits (de Kloet et al., 2005). Comparable to catecholamines, the relationship between corticosteroid levels and cognitive performance during the rapid non-genomic phase is similar to an inverted-U-shaped pattern (de Kloet, Oitzl, & Joëls, 1999;

Lupien et al., 2007). In addition, corticosteroids interact with catecholaminergic activity (Joëls & Baram, 2009). For instance, corticosteroids can increase the NA levels in the amygdala (McReynolds et al., 2010) and may potentiate the effects of NA on PFC and amygdala function (Barsegyan et al., 2010; Robbins & Arnsten, 2009; van Stegeren et al., 2010). Moreover, the DA release is enhanced by the circulation of cortisol levels (Marinelli & Piazza, 2002; Pruessner et al., 2004; Saal et al., 2003). The slow genomic corticosteroid effects are mediated by GR, which are mainly responsible for the consolidation and normalization of homeostasis (Joëls et al., 2013), resulting in a downregulation of hippocampal activity and an enhanced PFC function, assumingly providing a mechanism that actively reverses the rapid corticosteroid effects (Hermans et al., 2014).

1.2.2. Temporal dynamics of the stress response

The effects of corticosteroids and catecholamines in the brain play a vital role in the case of absolute stress or in short-term survival in terms of allostasis. In this context, acute stress initiates responses that affect diverse cognitive and affective domains. In this regard, the effects of corticosteroids and catecholamines lead to a dynamic shift in network balance in order to enable the individual to reallocate neural resources according to their cognitive demands (Hermans et al., 2014). Particularly, the salience network and the executive control network appear to be crucial neurocognitive systems that are targeted by these effects (Hermans et al., 2014). The executive control network incorporates the frontoparietal network including the dlPFC, dmPFC, frontal eye-fields, and dorsal posterior parietal areas (e.g., Vincent et al., 2008) and supports executive functions, such as WM, cognitive flexibility, or decision making (e.g., Niendam et al., 2012). The salience processing network involves the amygdala, anterior insula, thalamus, inferotemporal/temporoparietal regions, striatum, brainstem/midbrain nuclei (Hermans et al., 2014) and is associated with the facilitation of vigilance and thus improves the reorientation of attention to threats or the quick adaptation of an unpremeditated action (Uddin, 2016).

Acute stress leads to a dynamic shift in network balance towards the salience processing network at the expense of the executive control network (Hermans et al., 2014; van Oort et al., 2017; Young et al., 2017) (see Figure *2*). This shift in neutral network functioning is greatest immediately after stressor exposure due to the interactions of catecholamines and non-genomic corticosteroid effects. After approximately 60 minutes relative to the onset of stress, the effects reverse due to the activation of the slow-acting genomic corticosteroid effects and the absence of catecholaminergic activation. The reallocation of neural resources towards the salience network leads to the facilitation of

vigilance and an increased emotional reactivity while PFC-related cognitive functions are downregulated. In line with this, several studies report cognitive impairments of WM, cognitive flexibility, and rational decision making after acute stress exposure (cf. Shields et al., 2016) (The negative effects of acute stress exposure on executive functions as well as emotional reactivity are discussed in more detail in Section 1.3.). In the aftermath of stress, higher-order cognitive functioning recovers, and emotional reactivity normalizes. Thus, the effects of acute stress on affective and cognitive functions can differ depending on the timing relative to stress exposure.

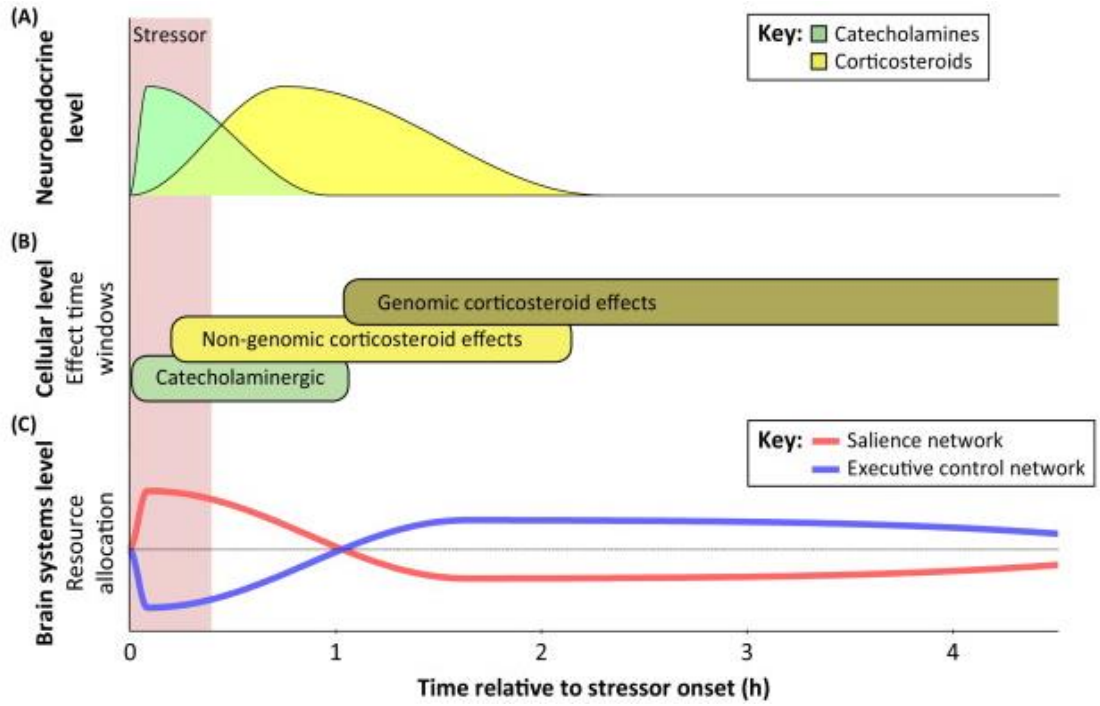

Figure 2. Biphasic-reciprocal model of reallocation of neural resources in response to stress. (Figure taken from Hermans et al., 2014).

1.3. How stress might affect emotion regulation

1.3.1. The mediating role of executive functions

Executive functions are neurocognitive top-down processes that are involved in conscious, goal-directed control of thoughts, actions, and emotions (Zelazo & Carlson, 2012). Executive functions comprise three major elementary processes: WM, inhibitory control, and cognitive flexibility (Diamond, 2013). WM involves the maintenance and manipulation of information as well as the incorporation of new information, referred to as *updating* (Baddeley, 1992). Inhibitory control represents the ability to suppress goal-irrelevant impulses and natural, habitual, or behavioral responses (Tiego et al., 2018). Cognitive flexibility (or shifting) enables the individual to flexibly shift back and forth between

multiple tasks or mental sets and to adjust changed demands or priorities (Diamond, 2013). The three core functions represent the basis for higher-order cognitive processes, such as reasoning, problem-solving, and planning (Collins & Koechlin, 2012). Executive functions are associated with a widespread bilateral activation in the frontoparietal network (also central executive network) comprising, e.g., the dlPFC, dmPFC, the posterior parietal cortex, and intraparietal sulcus (Buchsbaum et al., 2005; Niendam et al., 2012; Rottschy et al., 2012; Yuan & Raz, 2014).

Executive functions and ER show great overlap with respect to the underlying neuronal structures of the frontoparietal-network. Moreover, various studies emphasize the important role of executive functioning on ER, as the cognitive regulation of emotion is generally a complex and goal-directed process (Compas, 2006; Gyurak et al., 2011; Hofmann et al., 2012; Ochsner et al., 2012; Zelazo & Carlson, 2012). For instance, cognitive reappraisal requires the maintenance of explicit regulatory goals and representations, the inhibition of disrupting external or internal interference, and the manipulation of declarative memory knowledge (Ochsner et al., 2012; Okon-Singer, Hendler, Pessoa, & Shackman, 2015; Raio & Phelps, 2015). In this context, the role of WM in cognitive reappraisal is supported by research by Schweizer, Grahn, Hampshire, Mobbs, and Dalgleish (2013), who report an improvement of cognitive reappraisal after intense WM training.

Critically, stress exposure is known to have significant effects on PFC functioning and the associated executive processing network via corticosteroids and catecholamines (for details see Section 1.2.). In this regard, a variety of studies have found that high levels of acute stress have detrimental effects executive functions (see Shields et al., 2016). While studies consistently found that acute stress leads to a decrease in WM performance (Duncko et al., 2009; Luethi, 2008; Porcelli et al., 2008; Schoofs et al., 2008, 2008) and an impairment of cognitive flexibility (Alexander et al., 2007; Beversdorf et al., 1999; Plessow et al., 2011, 2012), the effects on inhibitory control are less clear. While some studies indicated an impairment of inhibitory control (e.g., Sänger, Bechtold, Schoofs, Blaszkewicz, & Wascher, 2014; Taverniers, Van Ruysseveldt, Smeets, & von Grumbkow, 2010) other studies report enhancements (e.g., Chajut & Algom, 2003; Schwabe, Höffken, Tegenthoff, & Wolf, 2013; Shields, Bonner, & Moons, 2015). A meta-analysis by Shields and colleagues (2016) indicated that stress leads to no impairments in overall inhibitory control but rather affects subordinated processes by impairing cognitive inhibition (i.e. the suppression of competing cognitive processing) and enhancing response inhibition (i.e. the suppression of a prepotent response to perform another response).

Integrating the above-mentioned findings, acute stress might have detrimental effects on cognitive ER as a result of an impairment of underlying executive functions. Regarding this, there are various ways in which impaired executive functions could affect different ER strategies. For instance, an impairment in WM performance results in an impaired manipulation of information, thus representing a key principle of cognitive reappraisal which is mainly based on reframing/re-interpretation of stimulus information. Further, a decrease in WM can disturb the maintenance of current regulation goals and leads to a loss of relevant information during the regulation process. Further, impaired cognitive flexibility might have substantial effects on ER in the form of a maladaptive and rigid selection between different ER strategies. In accordance, Cheng and Cheung (2005) report that participants who were less flexible in their ER choice tended to use higher monitoring on threat-related situational characteristics. As different ER strategies rely on different neural structures, it can be assumed that stress might have a greater impact on some ER strategies than others. For instance, distraction compared to cognitive reappraisal involves greater activation of parietal regions (Kanske et al., 2011; Ray et al., 2010) and is assumed to require fewer executive functions and cognitive costs (Shafir et al., 2015; Sheppes et al., 2009; Sheppes & Meiran, 2008; Strauss et al., 2016).

1.3.2. Alterations in emotional processing

Acute stress promotes the activation of a salience processing network due to the effects of corticosteroids and catecholamines on the PFC and the amygdala (cf. Section 1.2.). The activation of the salience processing network facilitates vigilance in order to improve the reorientation of attention towards a potential threat or salient stimulus (Uddin, 2016). In line with this, studies showed that acute stress exposure can lead to an increase in emotional reactivity to environmental cues. For instance, an EEG study by Alomari and colleagues (2015) revealed no LPP differentiation between non-emotional and emotional stimuli after acute stress exposure, indicating that neutral stimuli are similarly processed as emotional stimuli. Further, an EEG study by (Weymar et al., 2012) found greater LPPs for unpleasant pictures after acute stress exposure when compared to a control condition, indicating that stress sensitizes the brain for increased processing of potentially alarming cues or stimuli. Additionally, an fMRI study carried out by van Marle et al. (2009) revealed that the amygdala showed similar activation towards the presentation of happy, fearful, and angry faces under stressful conditions, while there was significantly less amygdala activation for happy faces than for angry or fearful faces in the control condition. The results indicate an increase of emotional reactivity in form of heightened sensitivity and lower specificity of the

amygdala under stressful compared to non-stressful conditions.

Thus, another potential way of how acute stress exposure might affect ER is by an increase in emotional reactivity that leads to generally greater emotional intensity towards a stimulus or situation, making it less disposed to regulation, as the regulation process requires greater effort and cognitive costs. In compliance with this, findings already suggest that the emotional intensity of a stimulus has a significant influence on ER success. For example, the strategy of cognitive reappraisal is applied less often and is less effective in situations with high emotional intensity (Murphy & Young, 2017; Shafir et al., 2015; Sheppes et al., 2011, 2014; Sheppes & Levin, 2013). Moreover, participants tend to use ER strategies that require less cognitive demand and regulation effort (e.g., distraction as compared to reappraisal) in situations of high emotional intensity, suggesting that some strategies might be more suitable than others depending on the degree of emotional intensity.

1.4. The current state of research: effects of acute stress exposure on emotion regulation

Only a small number of studies have systematically investigated the influence of stress on ER, using different approaches and revealing inconsistent results. In an early study by Raio and colleagues (2013), participants underwent a classical fear-conditioning paradigm on the first day and were subsequently trained to use an ER strategy, based on the principles of cognitive reappraisal. On the second day, participants were either assigned to a physiological stress group (Cold Pressure Test, CPT) or a control group, respectively. After completing the stress or control task the participants repeated the fear-conditioning paradigm with the explicit instruction to make use of the previously trained ER strategy to control the conditioned fear responses. In comparison to the control group, the stressed participants showed significant impairments in the regulation of physiological and self-reported fear responses. In addition, Raio et al. (2013) reported a significant correlation between salivary α-amylase and fear response while no association with salivary cortisol was found, relating the impairment of ER to the release of catecholamines and the SNS. A further study by Shermohammed and colleagues (2017) investigated the influence of acute stress exposure on cognitive reappraisal using functional magnetic resonance imaging (fMRI). In the study, a psychosocial stress task or control task was alternated with an ER task, during which participants either had to view or regulate (reappraisal) negative and neutral images. There were no significant effects of acute stress exposure on the ability to successfully regulate the emotional responses, while a positive relationship between self-reported stress and negative affect in response to negative images was found. A study conducted by Kinner, Het, and Wolf (2014) investigated the influence of acute stress on the reappraisal and distraction ER

strategies. Participants underwent a socially evaluated cold pressure task (SECPT) or control task, respectively. Subsequently, the participants performed an ER paradigm with negative and positive pictures. Kinner et al. (2014) report higher subjective arousal ratings in the distraction condition in the stress compared to the control group, while there were no stress-related effects regarding subjective valence ratings. In contradiction to Raio et al. (2013), reappraisal was not negatively affected by stress, rather, stressed women showed slight facilitations in reappraisal. A recent study by Langer et al. (2020) investigated the effect of acute stress (TSST) on the strategies of reappraisal and distraction while using affective ratings and pupil dilation as a measure for ER. The authors report improvements in cognitive reappraisal after acute stress exposure in men, but not in women, reflected by reduced arousal ratings as well as enhanced valence ratings. Moreover, men showed stronger pupil dilations during reappraisal, suggesting greater cognitive engagement.

In sum, the current research regarding the influence of acute stress on ER has revealed rather inconsistent results: while some studies showed detrimental effects of stress on ER (Raio et al., 2013), others showed no clear effects (Kinner et al., 2014; Shermohammed et al., 2017) or even selective improvements (Langer et al., 2020). Most likely, the heterogeneous results occur due to the different methodological approaches and operationalizations of the studies. For instance, all studies varied with respect to the applied stress protocols (CPT, SECPT, speech induction and math problems, or TSST) and dependent variables (e.g., subjective self-ratings, neural response, electrodermal activity, heart rate, pupil dilation). Moreover, as described above (see Section 1.2.2.), the temporal dynamics of a stress response play a crucial role in cognitive and affective functioning due to dynamic shifts in neural networks (e.g., Hermans et al., 2014). In this regard, the studies considerably varied with respect to the timing of the ER task in relation to the stress tasks. Specifically, while Shermohammed et al. (2017) alternated the tasks of the ER paradigm and the stress protocol, other studies implemented the ER task with a delay of 10 min (Raio et al., 2013), 20 min (Langer et al., 2020), or 25 min (Kinner et al., 2014) after stressor off-set. Considering the temporal dynamics of a stress response, the different time windows might have had a substantial influence on the subject's task performance. Thus, further research is necessary in order to extend the previous findings by taking potential temporal dynamics into account as well as integrating further well-established measures of emotional processing and ER.

1.5. Summary and clinical implications

ER represents an individual's ability to regulate their responses to emotional stimuli or emotional situations through a variety of different strategies. Hence, ER plays a crucial role in the maintenance of mental health and the development of mental disorders (cf. Section 1.1.2). Emotional processing and ER are based on the interaction of limbic and frontoparietal structures. In this regard, stress might have significant effects on ER due to the specific effects of corticosteroids and catecholamines on the PFC and amygdala functioning, resulting in substantial alterations in cognitive and affective processing (cf. Sections 1.2.1, 1.2.2.). Particularly, two potential mechanisms might determine deleterious effects of acute stress exposure on cognitive ER: 1) the impairment of PFC-related executive functions which mediates ER, and 2) the increase in emotional reactivity resulting in an enhanced emotional intensity requiring greater ER effort (cf. Sections 1.3.1 & 1.3.2). As some ER strategies rely more on PFC structures and require greater cognitive costs (e.g., cognitive reappraisal) they are probably more vulnerable to stress-related effects than others (e.g., distraction). However, only a few studies have investigated the effects of stress on ER, revealing mixed results (cf. Section 1.4.). Assumingly, the different results can be explained by varying study operationalization (e.g., stress induction, measures of dependent variables) and potential effects of temporal dynamics of the stress response (cf. Section 1.2.2) which have not been systematically considered in previous studies.

Stress has been recognized as an important contributor to the development of psychopathology and mental disorder, thus, a variety of models have featured stress as a determinant of disordered functioning (e.g., diathesis-stress models) (Ingram & Luxton, 2005). For instance, a link between stressful events and the onset of depression has been shown in a variety of studies (e.g., Kendler et al., 2010; Kessler, 1997). While diathesis-stress models provide a link between stressful life events and mental disorders, less is known about the potential underlying mechanisms. In this regard, stress-related impairments in the use of functional ER strategies might represent a mediating variable that could help to understand the association between stress experience and the development of psychopathological symptoms and mental disorders.

2. Aims of the present dissertation

As outlined above, there are only a small number of studies that have investigated the influence of acute stress exposure on ER, revealing heterogeneous and inconsistent results. As research has just begun to focus on this topic, the present dissertation aims to extend previous results by implementing emotional self-reports and neurophysiological methods (EEG, EMG), covering both emotional valence and emotional arousal by subjective and

objective measures. Moreover, as previous literature suggests substantial influences of the timing of a cognitive task in relation to the stressor, the present thesis also aims to investigate the potential effects of the temporal dynamics of the stress response. Furthermore, as previous studies mainly emphasized the effects of acute stress exposure on cognitive reappraisal, the thesis aims to identify more stress-resistant strategies by comparing different ER strategies. In this regard, the goal of the first study (Study 1) was to investigate the dynamic interplay between the neuroendocrine stress systems and brain networks (Hermans et al., 2014) and its potential contribution to cognitive reappraisal and emotional processing. Based on the findings of Study 1, a follow-up study was conducted in order to identify more stress-resilient ER strategies by comparing reappraisal and distraction in an ER paradigm after acute stress exposure (Study 2).

As stress is known to play a crucial role in the development of mental disorders and psychopathological symptoms (e.g., diathesis-stress-models), the results of the conducted studies could provide a potential explanation of determining modes of action. Particularly, the impairment of functional ER strategies (e.g., cognitive reappraisal) due to stress exposure could represent an important mediating variable with regard to an increased vulnerability to specific mental disorders (e.g., depression). In this context, the present dissertation aims to contribute to fundamental research in this field in order to initiate and encourage future studies in more naturalistic and clinical settings.

The studies of the dissertation were conducted as follows:

Study 1 aimed to investigate the influence of acute stress exposure on cognitive reappraisal using multimodal measures of ER, including ERPs, EMG, and subjective self-reports. The study hypotheses are based on a model by Hermans et al. (2014) that claims that acute stress exposure reallocates neural resources to a salience network that promotes vigilance and fear, at the cost of an executive control network. After the effects of stress subside, neural resources are reallocated to the executive control network, normalizing emotional reactivity and cognitive functionality. Hence, it was hypothesized that cognitive reappraisal would be impaired by two processes due to the dynamic neural network shift. First, it was expected that the activation of the salience processing network leads to an increase in emotional reactivity. In this regard, cognitive reappraisal could be impaired because emotional responses under acute stress are more intense and less amenable to cognitive reappraisal. Second, PFC regions mediating successful cognitive reappraisal and corresponding executive functions could be directly affected by the neuroendocrine stress response. As a consequence, PFC resources could be less retrievable and therefore dampening cognitive

reappraisal capacities. In order to test the hypotheses, participants were subjected to the Trier Social Stress Task (TSST) or a control condition, after which they completed an ER task. The ER task was divided into an early and a late stress phase in order to cover the predominant activation of the stress systems and the temporal dynamics of the stress response.

Study 2 was intended to extend the results of Study 1 by comparing the effects of acute stress exposure on two different ER strategies (reappraisal and distraction) using ERPs, EMG, and subjective self-reports. Potential effects of the temporal dynamics of the stress response were not specifically considered in Study 2, as the hypotheses of Study 1 could not be substantiated. Nevertheless, as the results of Study 1 indicated an impairment of cognitive reappraisal, it was concluded that the impairment was probably based on an impairment of PFC-related executive functions (e.g., WM) which are assumed to be crucial for successful cognitive reappraisal. Hence, it was expected that ER strategies that require fewer cognitive costs should be less impaired by the detrimental effects of acute stress exposure. Besides cognitive reappraisal, distraction represents a highly effective ER strategy (Webb et al., 2012). However, compared to reappraisal, distraction requires less cognitive capacity (e.g., Shafir et al., 2016) and is associated with fewer prefrontal regions (e.g., Kanske et al., 2011). Hence, we hypothesized distraction to be less impaired by acute stress exposure than reappraisal. To test the assumptions, participants underwent a social-evaluative stressor or a control condition (cf. Akdeniz et al., 2014), respectively, after which they completed an ER paradigm, requiring them to regulate their emotions using reappraisal and distraction.

3. Study 1:

The Influence of Acute Stress Exposure on Cognitive Emotion Regulation: a Psychophysiological Study

Abstract

Successful and efficient ER is a key mechanism for the maintenance of mental health. However, acute stress may impact the efficacy of ER due to its detrimental effects on prefrontal functionality. To investigate such adverse effects of stress on ER we investigated 50 young healthy adults that were either exposed to the Trier Social Stress Test (N = 25) or a control condition (N = 25). Afterward, subjects conducted an ER task during which they were instructed to either regulate (cognitive reappraisal) or passively view neutral and negative visual stimuli. The ER task was divided into an early (0-20 minutes) and a late post-stress phase (20-40 minutes), in order to investigate temporal dynamic effects of stress on ER. Self-reported emotional state, corrugator electromyographic activity, and the mean activity of the P300 and LPP were used as indices of emotion reactivity and regulation. Salivary cortisol was assessed as a marker of the neuroendocrine stress response. Our results suggest no increase in emotional reactivity, but an impairment of ER in the late post-stress phase. The results provide a more detailed understanding of potential determinants of ER failures under acute stress.

3.1. Introduction

Despite its important role in mental health and the significant contribution of stress to cognitive impairments, little is known about the effects of stress on ER processes. ER represents an active regulatory mechanism that adaptively and flexibly tunes reactions to emotional stimuli and/or situations (Gross & John, 2003). Among several ER strategies, cognitive reappraisal is very effective (Webb et al., 2012) and has been linked to long-term positive health outcomes (e.g. Aldao, Nolen-Hoeksema, & Schweizer, 2010; Gross & John, 2003). Cognitive reappraisal incorporates conscious reinterpretations of emotion-eliciting situations by modifying their meaning (situation-focused reappraisal). Individuals who use reappraisal strategies in daily life were more likely to experience generally more positive emotions and less anger, show more adaptive physiological responding to threats, report greater overall well-being, and generally fewer psychopathological symptoms (Feder et al., 2009; Kalisch et al., 2015; Troy & Mauss, 2011). Contrarily, the use of maladaptive ER strategies has been associated with mental disorders, such as anxiety, affective disorders, and borderline personality disorder (e.g. Berking and Wupperman, 2012; Campbell-Sills and

Barlow, 2007; Kanske et al., 2015, 2012). Considering the role of cognitive reappraisal in the etiology and the prevention of stress-related psychopathologies as well as the beneficial effects against stress-related dysfunctions, it appears highly relevant to investigate the effects of stress exposure on ER functioning. Two mechanisms could potentially determine stress-related effects on ER and, therefore, deserve further investigation.

First, ER is mediated by prefrontal brain regions, such as the dmPFC as well as dlPFC and vlPFC. Importantly, these prefrontal regions could be directly affected by neuroendocrine stress effects. The endocrine stress response is characterized by two major systems: (1) the rapid-acting sympathetic nervous system (SNS), resulting in the release of catecholamines (norepinephrine, epinephrine, dopamine), followed by (2) the slow-going HPA axis, leading to the release of corticosteroids (mainly cortisol). NE increases immediately in the face of a stressor and normalizes within ~15 minutes of stressor offset (Hermans et al., 2014). NE has significant effects on brain function where it engages α-1-, α-2A- and β-1-adrenergic receptors in the amygdala and PFC (Arnsten, 2015, 2009). Cortisol usually peaks 15-25 minutes after stressor offset (Hermans et al., 2014) and exerts a broad range of molecular, structural, and functional effects in the brain by binding with mineralocorticoid and glucocorticoid receptors in the amygdala, PFC, and the hippocampus (Herman et al., 2005; Lupien and McEwen, 1997). With regards to cognitive functioning, both cortisol and NE follow an inverted-U-shape, indicating that too high or too low levels can result in cognitive impairments (Dedovic et al., 2009; Lupien et al., 2007). On a neural level, Hermans and colleagues (2014) proposed that relatively shortly after acute stress exposure, the executive control network (encompassing the vlPFC, dlPFC, dmPFC, and parietal cortex) is downregulated, and resources are allocated to the salience processing network (encompassing the dorsal anterior cingulate cortex, anterior insula, amygdala, hypothalamus, thalamus, putamen, and substantia nigra/ventral tegmental area). This leads to limited selective allocation of processing resources in order to facilitate vigilance and increase emotional reactivity, thus improving the reorientation of attention to threats or a quick adaptation of an unpremeditated action. After approximately 60 minutes, the cortisol release decreases while genomic effects of glucocorticoids increase. As a consequence, activity in the salience processing network is actively downregulated while executive network activity increases. Hence, a gradual switch in neural networks occurs (Hermans et al., 2014) This dynamic interplay between endocrine stress responses and activation in different brain networks suggests that network switches after acute stress exposure may contribute to impairments in prefrontal-based cognitive functioning, including cognitive reappraisal. Second, as mentioned above, an increase in activity of the salience processing

network leads to increased emotional reactivity. In accordance, acute stress exposure has been shown to increase the emotional intensity of presented stimuli (Alomari et al., 2015; van Marle et al., 2009; Weymar et al., 2012). In this regard, recent findings revealed that reappraisal is applied less often and is less effective in situations with high emotional intensity (Murphy & Young, 2017; Shafir et al., 2015; Sheppes et al., 2011, 2014; Sheppes & Levin, 2013), indicating an indirect impairment of reappraisal through an increase in emotional reactivity.

So far, few studies have systematically investigated the effects of acute stress on ER success. Raio and colleagues (2013) investigated participants' regulation (reappraisal) of fear-conditioned stimuli after acute stress exposure. In this study, stress exposure increased self-reported fear and non-stressed participants showed a robust fear reduction, while stressed participants showed no reduction. In a further study, Kinner and colleagues (2014) investigated distraction and reappraisal after acute stress induction by using a picture-based paradigm and observed higher subjective arousal in the distraction condition in the stress compared to the control group. However, no other stress-related effects on reappraisal were observed. Finally, in the most recent functional magnetic resonance imaging (fMRI) study by Shermohammed and colleagues (2017), a psychosocial stress task was alternated with an ER task, revealing a positive relationship between self-reported stress and negative affect in response to negative images while no impairment of cognitive reappraisal was found. In contrast to the previous results, a recent study by Langer et al. (2020) revealed an improvement of reappraisal in men but not in women in form of reduced arousal and more positive valence ratings after acute stress exposure. Moreover, the reappraisal success in men was positively correlated with cortisol secretion. Importantly, these studies vary with respect to different methodological aspects (i.e., the assessed dependent variables, the operationalization of ER, and the used stress protocol) [cold pressure task (CPT; Raio et al., 2013); socially-evaluated CPT (Kinner et al., 2014); TSST-like procedure (Langer et al., 2020; Shermohammed et al., 2017)]. The latter aspect also implies that the studies differ in the time relation between the stressor and the ER paradigm. While Shermohammed and colleagues (2017) alternated the ER task with the stress protocol, Raio et al. (2013), Kinner et al. (2014) and Langer et al. (2020) presented the ER paradigm subsequently to the stress protocol within different time frames (Kinner et al., 2014: ~25 min after stressor offset; Langer et al., 2020: ~20 min after stressor offset; Raio et al., 2013: ~10 min after stressor offset). These timing aspects could be crucial due to the above-discussed dynamic shift in the executive control and salience processing networks (Hermans et al., 2014) and thus for the dynamic state of the stress response in which the ER task is presented.

The present study thus aimed to specifically address the potential influence of the temporal dynamics of stress on ER. Therefore, participants were exposed to the TSST or a control condition and subsequently tested in an ER paradigm including two different time windows. We aimed at covering the effects of the rapid-acting SNS by implementing an early stress phase (~0-20 min after stressor offset). Further, a late stress phase (~20-40 min after stressor offset) was applied in order to detect potential effects of the slow-going HPA axis. In order to assess ER and emotional reactivity, we included a set of neurophysiological, peripheral, and subjective measures that reflect the three components of emotional responses (Barrett et al., 2007) to assess ER success after stress exposure: (1) subjective emotional state ratings to reflect subjective emotional experience, (2) EMG of the corrugator supercilii as a measure of emotional expression (Ray et al., 2010; Schönfelder et al., 2014) and (3) event-related potentials (i.e., the P300 and the LPP), as an indicator of physiological responding to emotional stimuli. The P300, mainly located at midline parietal sites (Hajcak et al., 2009) has been associated with the relatively fast allocation of attention towards salient stimuli and thus represents a valid neural marker of emotional reactivity (Hajcak et al., 2012). The LPP, typically starting at 500 ms post-stimulus onset over posterior recording sites (Hajcak et al., 2009), has been linked to facilitated attention to emotional stimuli and emotion processing, particularly emotional arousal (Hajcak et al., 2010), and has already been used in a variety of ER studies (e.g., Foti and Hajcak, 2008; Hajcak et al., 2010; Schönfelder et al., 2014). Based on the discussed results of previous research, two major hypotheses were tested in the present study. First, we hypothesized an increase in emotional reactivity in the stress group, as indicated by more negative emotional state ratings, higher P300, and LPP magnitudes, and greater corrugator supercilii activity to negative stimuli in the stress group as compared to the control group. This effect is supposed to be greater in the early compared to the late stress phase due to the activation of the salience processing network. Second, we hypothesized a stress-related impairment of ER, indicated by increased subjective emotional state ratings, LPP magnitudes, and Corrugator supercilii activity in the stress group compared to the control group. Again, we expect this stress-related impairment in ER to be greater in the early compared to the late stress phase, primarily due to the downregulation of the executive control network.

3.2. Methods

3.2.1. Participants

Fifty medication-free volunteers were recruited for study participation at the University of Mainz, Germany. For descriptive data of the sample and statistical indices of the respective

group comparisons, see Table 1. Subjects were randomly assigned to either a stress group (n=25) or a control group (n=25). All participants were nonsmokers. All female volunteers were not pregnant, did not use hormonal oral contraceptives, and were tested during the luteal phase of their menstrual cycle. Exclusion criteria included: any acute or chronic physical disease (e.g., cardiovascular disease, diabetes, endocrine disease, epilepsy), previous experience with the stress protocol or the ER paradigm, and illegal drug consumption. These exclusion criteria were verified by telephone and/or personal structured interviews which were carried out by trained psychologists. Subjects were instructed to refrain from eating and drinking anything but water one hour prior to the experiment and to abstain from alcohol and exercise on the day of assessment, which took place between 2:00 p.m. and 6:00 p.m. All participants gave written informed consent before study participation. The study has was carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) and was approved by the ethics committee of the University of Mainz, Institute of Psychology. All participants received course credit or were paid €40 for their participation. Four participants had to be excluded from EMG data analysis ($n_{control}$=2; n_{stress}=2) and one participant from EEG analysis (control group) due to excessive data artifacts.

3.2.2. Experimental Design and Procedure

A week prior to experiment day in the laboratory, the participants completed several questionnaires with respect to ER (ERQ; Gross & John, 2003), anxiety (STAI; Spielberger et al., 1970), and depression (BDI-II; Beck et al., 1996). Upon arrival at the laboratory, subjects rested for about 60 minutes while EEG and EMG preparation took place. Afterward, participants received detailed instructions for the experimental procedure and the ER strategy. Cognitive reappraisal was practiced with the study participants in order to verify that the participants applied the strategy correctly. Afterward, either the TSST or the control condition was carried out. Saliva sampling took place prior to the TSST or placebo protocol (T0, baseline), at the end of the stress protocol prior to the ER paradigm (T1, +20 minutes), between the early and late post-stress phase (T2, +40 minutes) of the ER paradigm, after completion of the experiment (T3, +60 minutes) and after the complete testing (T4, +90 minutes). In addition, negative affect was measured using the Positive and Negative Affect Scale (PANAS; Watson et al., 1988) before (T0, baseline) and after the stress induction (T1, +20 minutes) as well as after the ER paradigm (T2, +60 minutes). After completion of the ER paradigm, the participants completed a short questionnaire measuring the individual's effort regarding the ER task. The study procedure is displayed in Figure 3A.

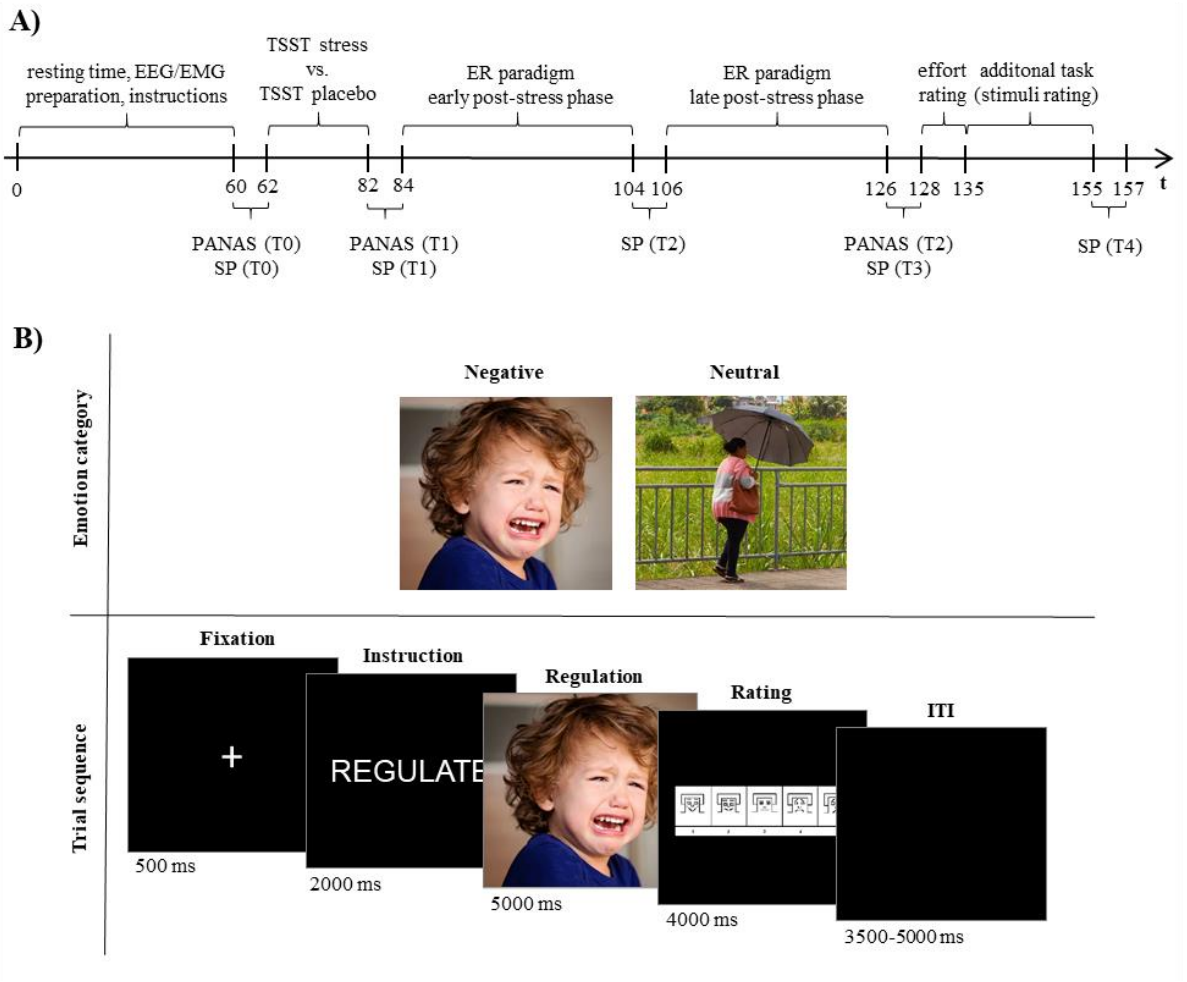

Figure 3. A) Study procedure on the laboratory day. **B)** Trial Sequence. SP = Saliva Sample

3.2.3. Stress protocol (Stress vs. Placebo)

For stress induction, we used the Trier Social Stress Test (TSST), a widely used and valid procedure to induce stress in the laboratory. The TSST was carried out according to Kirschbaum and colleagues (1993). Participants were instructed to prepare a free speech of five minutes during which they presented themselves as perfect applicants for a vacant position ("dream job") in front of a committee composed of one male and one female evaluator dressed in white coats. The committee was asked to maintain a neutral face during the whole experiment. Additionally, the speech was recorded by a video camera placed in front of the participant. Preparation time was 10 minutes (for which the committee left the room). After the speech, a mental arithmetic task (counting backward in steps of 17 from 2043) was performed for five minutes. In case of an error, the participants had to start again from 2043. For the control condition, we followed the procedure proposed by (Het et al., 2009). In the beginning, the participant was accompanied into an empty room with the instruction to talk for five minutes about a favorite topic with a preparation time of 10 minutes. After preparation, the experimenter entered the room and asked the participant to

stand up and take a position somewhere in the room. Subsequently, the experimenter left the room and the participant talked aloud about his chosen topic. After five minutes, the participant was told to add the number 15 starting at 0 for 5 minutes. The placebo condition took place in the same room as the TSST, however, no camera, microphone, or committee was present. At the end of the study, each participant received a full debriefing with respect to the study aims.

3.2.4. Emotion Regulation Paradigm

We employed a modified version of a previously used and validated ER paradigm (Schönfelder et al., 2014). In the present study, negative and neutral pictures, all depicting human characters, or at least parts of the human body, were used. The stimuli were selected from the International Affective Picture System (Lang et al., 2008) and Emotional Picture Set (Wessa et al., 2010). Normative ratings of neutral stimuli and negative stimuli differed significantly for valence (neutral: M_v=4.98, SD_v=.20; negative: M_v=2.09, SD_v=.42; t(158)=55.02, p<.001) and arousal (neutral: M_a=2.92, SD_a=.28; negative: M_a=6.59, SD_a=.50; t(158)=57.46, p<.001). In the present study, two within-subject task conditions were presented. In the "view" condition, participants were instructed to draw their attention to the presented stimulus and perceive the stimulus as naturally as possible without altering their emotional reaction. In the "reappraisal" condition, participants were instructed to reinterpret the depicted situation in the picture (situation-focused reappraisal) in a way that reduces the negative emotion evoked by the stimulus (e.g., by imagining a rather positive outcome of the situation). The condition x task combinations were balanced with respect to effective norm values provided by the authors of the picture sets (valence, arousal; Lang et al., 2008; Wessa et al., 2010). In order to investigate the temporal dynamics of the stress response, stimuli were presented in a randomized block design with two blocks, an early post-stress phase and a late post-stress phase, each lasting for 20 minutes. Each block consisted of 80 trials (20 view-neutral; 20 view-negative; 20 reappraisal-neutral; 20 reappraisal-negative), The picture-instruction combinations were presented in a pseudo-randomized order, meaning that neither the condition nor the task was repeated more than three times in a row.

Each trial of the ER task started with a fixation cross (500 ms) followed by a single-word instruction ("View" vs. "Reappraise") (2000 ms), displayed as white text on a black background. Subsequently, the stimulus was presented (5000 ms) followed by a 9-point scale (Self-Assessment-Manikin Scale, SAM, for valence; (Lang et al., 2008)) to rate the individual emotional state (4000 ms). Participants were instructed to indicate their emotional

state by marking one of the faces or between them with a slider, leading to a scale ranging from 1 (negative valence) to 9 (positive valence). Finally, a variable inter-trial interval (ITI) of 3500–5000 ms was presented. The trial structure of the ER task is displayed in Figure 3B. The complete ER paradigm lasted about 40 minutes with a pause of approximately two minutes between the early and late post-stress phase.

3.2.5. Neurophysiological data (EEG, EMG)

EEG was recorded using Ag/AgCl-electrodes from 32 scalp positions according to the international 10–20 system. Online EEG signals were referenced to the right mastoid. Four additional electrodes were placed around the orbital regions of the face to monitor vertical and horizontal eye movement (EOG). Electrode impedances were kept <10kV before starting the experimental paradigm. EEG and EOG data were registered with a sampling rate of 1 kHz and 16-bit A/D conversion using BrainAMP amplifiers (Brain Products, Inc., Munich, Germany). Brain Vision Analyzer II software (Brain Products GmbH, Munich, Germany) was used for offline analysis. EEG data were down-sampled to 250 Hz, re-referenced to the averaged mastoid, and filtered with a 0.1 to 40 Hz (24 dB/oct) bandpass (Picton et al., 2000). An independent component analysis algorithm was applied to correct stereotypic artifacts (eyeblinks) using the internal algorithm provided by the Brain Vision Analyzer II software. ERP epochs were extracted from -500 to 5000 ms relative to stimulus presentation and segments were baseline corrected to 250 ms pre-stimulus onset. Artifacts were semiautomatically rejected with the following criteria: peak-to-peak differences > 300 µV, voltage steps of 50 µV between sampling points, and a maximum difference of less than .50 µV within 100 ms intervals. Additional artifacts were identified and rejected based on visual inspection. Stimulus-locked ERPs were constructed using EEG signals of a parietal cluster (P1-, Pz- and P2-electrode) by separately averaging trials for each condition and participant. The P300 covered the time range of 300 to 500 ms, whereas the LPP comprised the time range of 500 to 5000 ms.

In order to measure EMG activity related to negative valence of the stimuli, two 4mm Ag/AgCl electrodes were placed over the Corrugator supercilii (Fridlund & Cacioppo, 1986). EMG was amplified using a BrainAmp ExG amplifier (Brain Products GmbH, Munich, Germany) and registered with a sampling frequency of 1 kHz. Raw EMG signals were filtered using a 30 Hz low cut-off, a 500 Hz high cut-off, and a 50 Hz notch filter. EMG signals were full-wave rectified and smoothed with a moving average of over 125 ms. EMG scores were calculated as activity change relative to the baseline period of 1000 ms prior to

stimulus onset (Blumenthal et al., 2005). The time window was defined by averaging the segment of 0 ms to 5000 ms spanning the whole 5s of stimulus presentation.

3.2.6. Measurement of Salivary Cortisol and α-Amylase

Psychosocial stress in humans leads to an increase in cortisol levels via the activation of the HPA axis (Kirschbaum and Hellhammer, 1993) and an increase in salivary α-amylase (sAA) activity, as an indirect measure of the SNS (Nater & Rohleder, 2009). Therefore, Salivette sampling devices (Sarstedt, Nümbrecht, Germany) were used to assess free salivary cortisol levels as well as sAA levels as a measure of HPA and SNS activity, respectively. Saliva samples were stored at -20°C immediately after the completion of the experiment. The devices were assayed by Dresden LabService GmbH (Dresden, Germany) using chemiluminescence immunoassay kits.

3.2.7. Statistical Analyses

In order to minimize Type I error, Bonferroni Holm correction was applied for post-hoc analyses (Holm's alpha). The statistical significance level was set to $p<0.05$ and effect sizes are reported using partial eta square (η^2p) and Cohen's d. Statistical data analyses were performed with SPSS23 (IBM, Chicago IL).

Effective emotion induction (i.e., more aversive evaluation of negative as compared to neutral pictures), and effective task manipulation (i.e., a significant ER effect through reappraisal), were verified by a 2x2x2x2 rmANOVA including the within-subject factors 'task' (view, reappraisal), 'valence' (neutral, negative) and 'stress phase' (early, late), and the between-subject factor 'group' (stress, control) for each dependent variable. Manipulation checks were considered successful if significant main effects of the within-subject factors 'valence' (neutral, negative) and 'task' (view, reappraisal) were observed. Cortisol and sAA were analyzed using two 5x2 rmANOVAs (within-subject factor 'sampling time': T0, T1, T2, T3, T4; between-subject factor 'group': stress, control). To evaluate the subjective experience of negative affect induced by the stress protocol, a 3x2 rmANOVA (within-subject factor 'time': T0, T1, T2; between-subject factor 'group': stress, control) was performed. In order to examine a stress-related increase in emotional reactivity (Hypothesis 1), 2x2x2 rmANOVAs with the within-subject factors 'valence' (negative, neutral) and 'stress phase' (early, late) and the between-subject factor 'group' (stress, control) were performed for each dependent variable. To test the hypothesis of a stress-related impairment of cognitive reappraisal (Hypothesis 2), we performed 2x2x2 rmANOVAs including the within-subject factors 'task' (view, regulate) and 'stress phase' (early, late) and the between-subject factor 'group' (stress, control) for the negative stimuli

condition for each dependent variable. For the full sets of ANOVA results regarding Hypothesis 1 and 2, see Appendices.

3.3. Results

3.3.1. Manipulation checks

Emotion induction and task manipulation: The analyses revealed significant main effects of 'valence' regarding the subjective emotional ratings, $F(1,48)=197.89$, $p<.001$, $\eta^2p=.81$, EMG magnitudes, $F(1,44)=36.53$, $p<.001$, $\eta^2p=.45$, and the LPP magnitudes, $F(1,47)=245.69$, $p<.001$, $\eta^2p=.84$. The main effects consistently indicated more negative processing of aversive (higher scores on SAM rating, lower EMG and LPP magnitudes) compared to neutral pictures, reflecting successful negative emotion induction.

Moreover, the analyses revealed significant main effects of 'task' regarding the subjective emotional ratings, $F(1,48)=234.81$, $p<.001$, $\eta^2p=.83$, EMG magnitudes, $F(1,44)=17.09$, $p<.001$, $\eta^2p=.28$, and the LPP magnitudes, $F(1,47)=106.47$, $p<.001$, $\eta^2p=.69$. The results consistently revealed more positive processing of neutral and negative pictures (higher scores on SAM rating, lower EMG, and LPP magnitudes) in the reappraisal as compared to the viewing condition, indicating successful downregulation of negative emotions by cognitive reappraisal.

Neuroendocrine stress response: A significant effect of group, $F(1,45)=13.81$, $p<.01$, $\eta^2p=.24$, and time x group interaction, $F(2.28,102.72)=11.33$, $\varepsilon=.57$, $p<.001$, $\eta^2p=.20$, indicated a stress-related increase in salivary cortisol. With respect to sAA, no significant main effect of group, $F(1,45)=1.81$, $p=.19$, $\eta^2p=.04$, nor a significant time x group interaction, $F(1.69,75.96)=1.96$, $\varepsilon=.42$, $p=.15$, $\eta^2p=.04$, was observed, indicating no stress-related effects. Follow-up t-tests for cortisol and sAA are presented in Table 1. Neuroendocrine and affective stress responses are illustrated in Figure 4.

Affective stress response: A significant time x group interaction, $F(1.581,75.91)=6.62$, $\varepsilon=.79$, $p<.01$, $\eta^2p=.12$ indicated a stress-related increase in negative affect after acute stress exposure (corresponding post-hoc t-tests are displayed in Table 1).

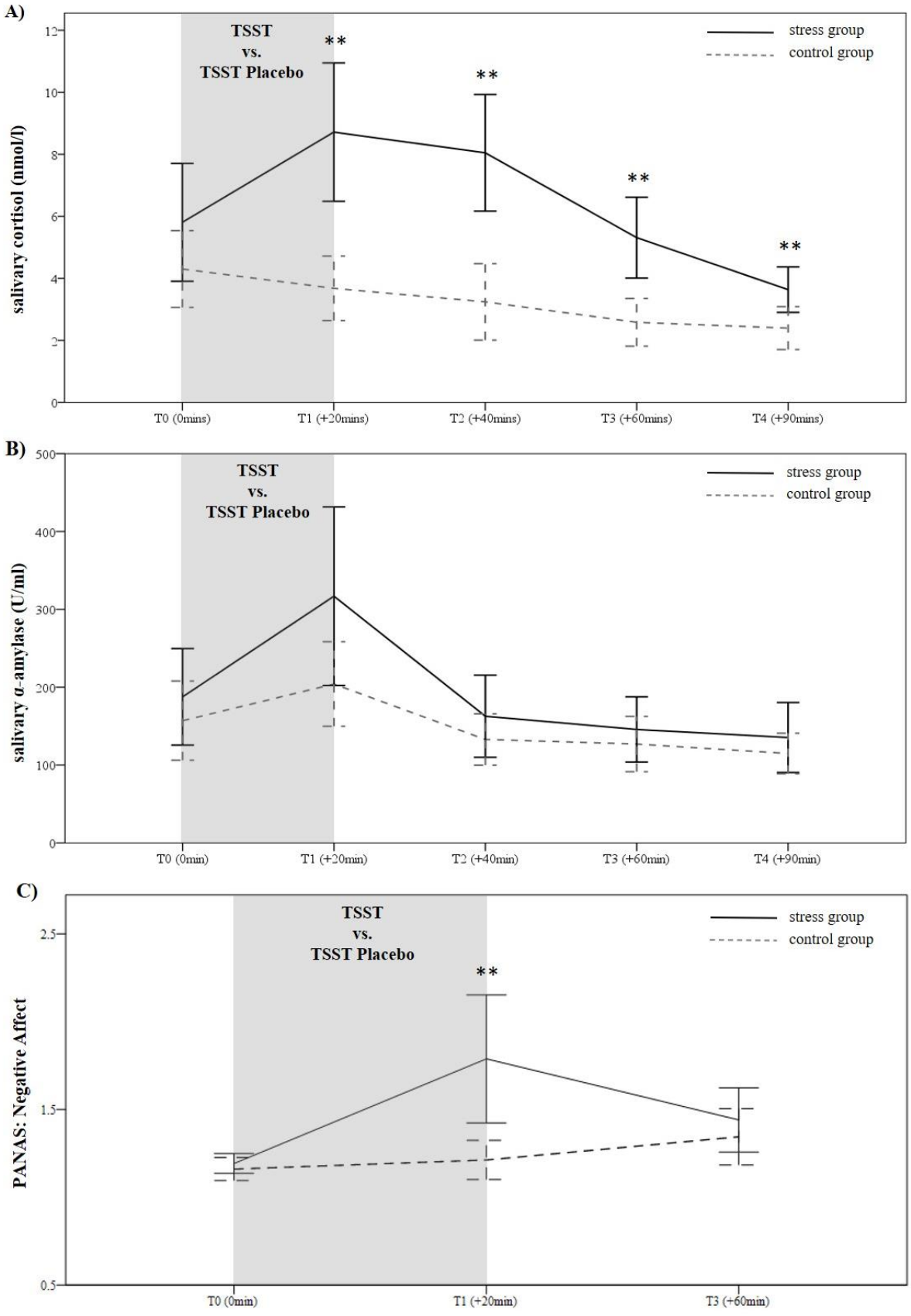

Figure 4. A) Salivary cortisol in (nmol/l) and **B)** salivary α-amylase (U/ml) measured at five points during the experiment. Negative affect was measured at three time points **C)**. The shaded area represents the Trier Social Stress Test (TSST) or the placebo condition, respectively. Error bars represent +/- 2 standard error of the mean, ** $\alpha < 0.01$, t-tests

Table 1. Descriptive data and statistical indices for sample characteristics as well as stress induction measures (salivary cortisol, salivary α-amylase, and negative affect)

Variable	**Stress group** Mean (SD)	**Control group** Mean (SD)	**Statistical indices** (X^2/t-values)	**P-value** (Holm's alpha)
Sample characteristics				
Sex (*N* female / male)	13 / 12	13 / 12	$X^2(1,N=50) < .01$	.61
Age in years	22.20 (2.99)	22.84 (3.09)	t(48) = -.75	.46
BMI	21.56 (2.18)	21.04 (2.17)	t(48) = .85	.40
BDI-II	27.20 (5.28)	25.60 (4.74)	t(48) = 1.13	.27
STAI (trait anxiety)	39.96 (8.38)	36.16 (9.4)	t(48) = 1.50	.14
ERQ - Reappraisal	4.97 (.89)	4.52 (1.17)	t(48) = 1.18	.25
ERQ - Suppression	3.84 (1.35)	3.59 (1.22)	t(48) = .69	.49
Salivary cortisol				
T0: baseline	5.71 (4.59)	4.67 (3.14)	t(48) = .928	.36
T1: +20 minutes	8.69 (5.35)	3.93 (2.57)	t(38.22) = 3.94	< .001 (.017)
T2: +40 minutes	8.52 (5.10)	3.39 (2.92)	t(38.23) = 4.89	< .001 (.010)
T3: +60 minutes	5.31 (3.20)	2.67 (1.83)	t(48) = 3.94	.001 (.013)
T4: +90 minutes	3.68 (1.77)	2.39 (1.66)	t(48) = 3.10	.013 (.025)
Salivary α-amylase				
T0: baseline	182.05 (151.15)	161.72 (120.82)	t(48) = .525	.60
T1: +20 minutes	306.80 (279.58)	203.07 (126.98)	t(33.50) = 1.69	.10
T2: +40 minutes	158.46 (128.34)	136.05 (76.78)	t(48) = .169	.46
T3: +60 minutes	145.65 (102.54)	130.19 (82.94)	t(48) = .582	.56
T4: +90 minutes	132.44 (108.85)	114.82 (62.44)	t(48) = .680	.50
Negative affect (PANAS)				
T0: baseline	1.19 (.14)	1.16 (.17)	t(48) = .735	.47
T1: +20 minutes	1.79 (.91)	1.21 (.28)	t(28.45) = 3.02	.004 (.013)
T2: +40 minutes	1.44 (.46)	1.34 (.40)	t(48) = .790	.43

Note: BMI = Body Mass Index (kg/m²); BDI-II = Beck Depression Inventory-II; ERQ = Emotion regulation questionnaire; STAI = State-Trait Anxiety Inventory; PANAS = Positive and Negative Affect Scale

3.3.2. Hypothesis 1: Stress-related increase of emotional reactivity

Subjective emotional state ratings (SAM): There was neither a significant main effect of group, $F(1,48)=.92$, $p=.34$, $\eta^2p=.02$, nor significant interaction effects of stress phase x group, $F(1,48)=2.49$, $p=.12$, $\eta^2p=.05$, valence x group, $F(1,48)=0.04$, $p=.84$, $\eta^2p<.01$, or stress phase x valence x group, $F(1,48)=1.20$, $p=.28$, $\eta^2p=.02$, indicating that the stress induction had no effect on the emotional reactivity to neutral or negative pictures reflected by SAM ratings.

EMG: We observed no significant main effect of group, $F(1,44)=.61$, $p=.44$, $\eta^2p=.01$, but a significant valence x stress phase x group interaction, $F(1,44)=5.37$, $p=.03$, $\eta^2p=.11$. However, post-hoc 2x2 rmANOVAs (within-subject factor 'valence; between-subject factor 'group') for each stress phase (early, late) revealed no significant main effects for group (early stress phase: $F(1,44)=1.21$, $p=.28$, $\eta^2p=.03$; late stress phase: $F(1,44)=.11$, $p=.74$, $\eta^2p<.01$) nor significant valence x group interaction effects (early stress phase: $F(1,44)=2.23$, $p=.14$, $\eta^2p=.05$; late stress phase: $F(1,44)=.10$, $p=.75$, $\eta^2p<.01$), indicating no effects of stress exposure on emotional reactivity reflected by EMG magnitudes.

P300: We observed no main effects for group, $F(1,47)=2.23$, $p=.14$, $\eta^2p=.05$, and no significant interaction effects of stress phase x group, $F(1,47)=.02$, $p=.89$, $\eta^2p<.01$, valence x group, $F(1,47)=.51$, $p=.48$, $\eta^2p=.01$, or stress phase x valence x group, $F(1,47)=.35$, $p=.56$, $\eta^2p<.01$. The results indicate that stress exposure had no significant impact on emotional reactivity reflected by the P300.

LPP: Analyses revealed no significant main effect for group, $F(1,47)=.56$, $p=.46$, $\eta^2p=.01$. The stress phase x group interaction, $F(1,47)=1.34$, $p=.25$, $\eta^2p=.03$, the valence x group interaction, $F(1,47)=.39$, $p=.54$, $\eta^2p=.01$, and stress phase x valence x group interaction, $F(1,47)=.45$, $p=.51$, $\eta^2p<.01$ were not significant. The results revealed no effects of stress induction on emotional reactivity reflected by the LPP.

3.3.3. Hypothesis 2: Stress-related impairment of cognitive reappraisal

Subjective emotional state ratings: We observed a significant three-way task x stress phase x group interaction, $F(1,48)=5.62$, $p=.02$, $\eta^2p=.11$. Two follow-up 2x2 rmANOVAs (within-subject factor 'task'; between-subject factor 'group') were calculated for each stress phase (early, late). The results revealed a significant task x group interaction in the late stress phase, $F(1,48)=4.08$, $p=.049$, $\eta^2p=.01$, but not in the early stress phase, $F(1,48)=.04$, $p=.85$, $\eta^2p<.01$, indicating a decreased success in reappraisal in the stress compared to the control group in the late stress phase. Figure 5 displays the subjective emotional ratings.

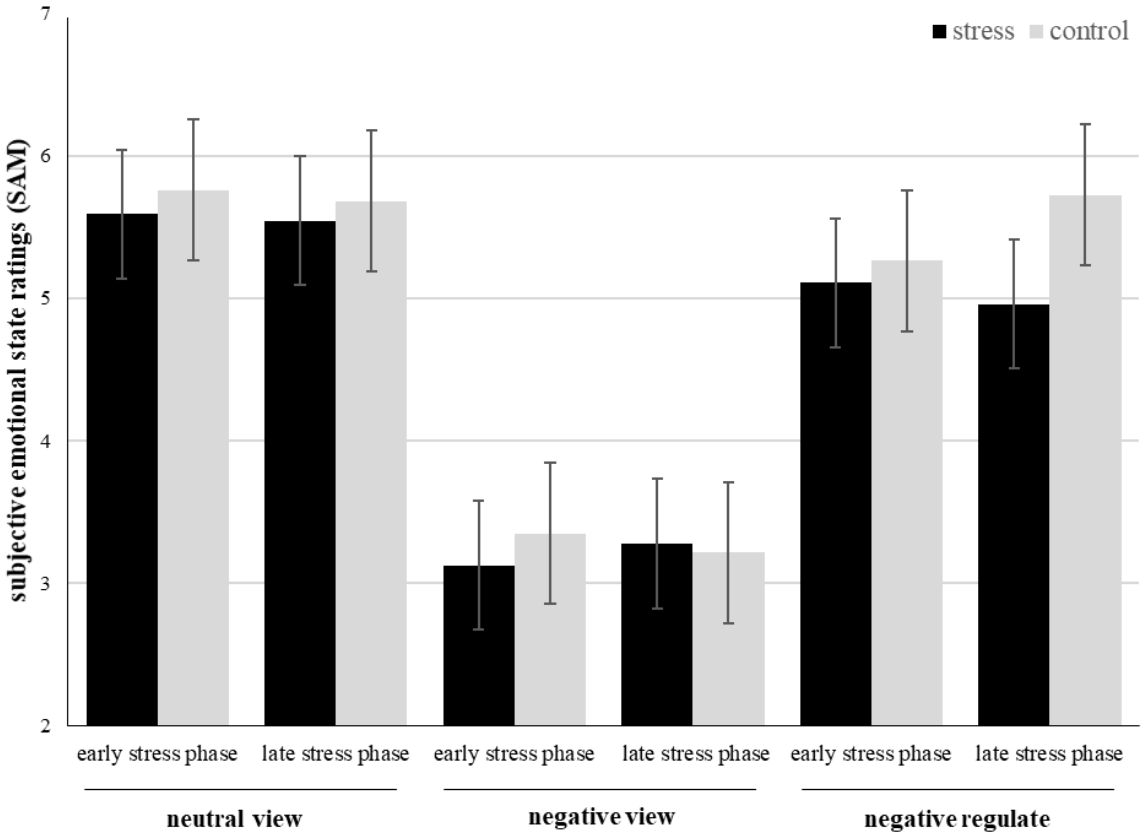

Figure 5. Subjective emotional state ratings of the stress and the control group. Error bars represent +/- 1 standard error of the mean.

EMG: We observed a significant three-way task x stress phase x group interaction, $F(1, 44)=5.64$, $p=.02$, $\eta^2p=.14$. Two follow-up rmANOVAs (within-subject factor 'task'; between-subject factor 'group') were conducted for each stress phase (early, late). The analysis revealed a significant task x group interaction in the late stress phase, $F(1,44)=8.13$, $p=.007$, $\eta^2p=.16$, but not in the early stress phase, $F(1,44)=.27$, $p=.61$, $\eta^2p<.01$. Paired-sample t-tests in each group for the late stress phase revealed that EMG magnitudes control group EMG magnitudes were significantly lower during regulation as compared to view trials, $t(22)=3.29$, $p=.003$, d=.62. In stressed individuals, EMG magnitudes during the two tasks did not differ, $t(22)=-.26$, $p=.80$, d=.03, indicating an impairment of cognitive reappraisal in the late stress phase. The EMG data are depicted in Figure 6.

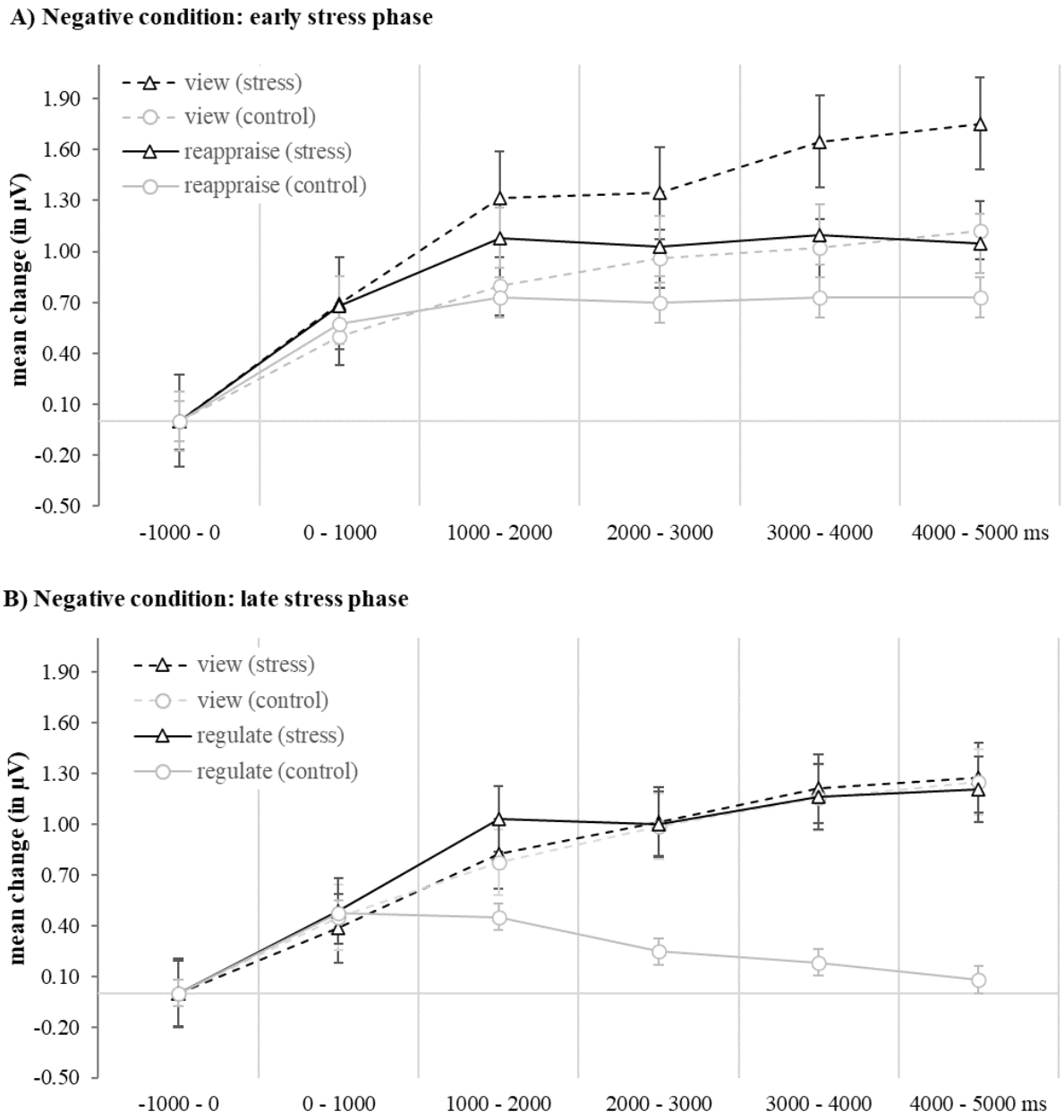

Figure 6. Corrugator supercilii - EMG activity of the **A)** early (~0-20 minutes) and **B)** late (~20-40 minutes) stress phase of the negative stimulus condition. Error bars represent +/- 1 standard error of the mean.

LPP: The analysis revealed a significant three-way task x stress phase x group interaction, $F(1,47)=5.64$, $p=.02$, $\eta^2p=.14$. To further elucidate the interaction, we conducted two follow-up 2x2 rmANOVAs (within-subject factor 'task'; between-subject factor 'group') for each stress phase (early, late). We observed no significant task x group interaction in the early stress phase, $F(1,47)=1.41$, $p=.24$, but in the late stress phase, $F(1,47)=5.27$, $p=.03$, $\eta^2p=.10$. Paired-sample t-tests in each group for the late stress phase showed a task differentiation of LPP magnitudes in the control group, $t(23)=4.44$, $p<.001$, d=.59, but not in the stress group,

t(24)=.85, *p*=.41, d=.15, indicating impaired ER in the stress compared to the control group in the late stress phase. EEG data are presented in Figure 7.

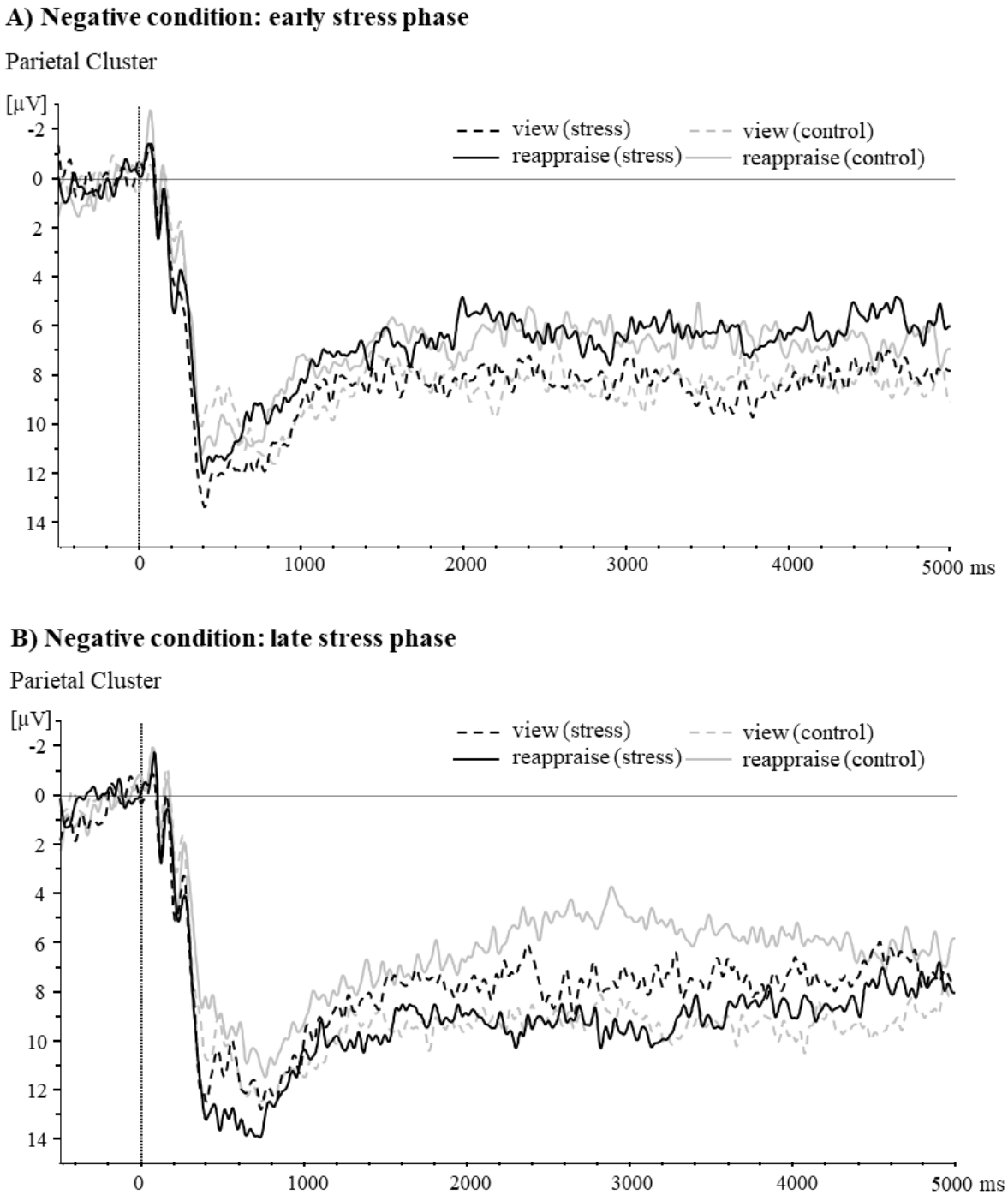

Figure 7. ERPs of the parietal cluster of the **A)** early (0-20 minutes) and **B)** late (20-40 minutes) stress phases of the negative stimulus condition.

3.4. Discussion

To the best of our knowledge, this is the first study that investigated the influence of acute social stress (TSST) on ER processes using multimodal measures of ER, i.e., ERPs, facial

expressivity, and self-reports. Contrary to our first hypothesis, we did not observe a stress-related increase in emotional reactivity, neither for negative nor for neutral pictures, and neither for the early nor for the late stress phase. We partly confirmed our second hypothesis that acute stress exposure impaired cognitive reappraisal, as indicated by a diminished decrease in subjective emotional ratings, LPP, and EMG magnitudes in the stress compared to the control group. Contrary to our hypothesis, this impairment was observed in the late, not in the early stress phase, suggesting a selective impairment of reappraisal in the stress group at a later stage of the experiment.

The lack of increased emotional reactivity in the stress group is in contrast to previous studies reporting altered self-reported or physiological responses to emotional or neutral stimuli after acute stress induction (Alomari et al., 2015; van Marle et al., 2009). Both Alomari et al. (2015) and van Marle et al. (2009) observed a stress-related loss of differentiation between negative and neutral or negative and positive pictures, respectively, when cortisol concentration was at its peak. Only recently, Shermohammed et al. (2017) showed different results. The authors observed no direct increase in emotional reactivity in the stress as compared to the control group, but treating stress as a continuous variable analysis revealed that greater self-reported stress was related to more negative evaluations of negative pictures than neutral pictures. Timing effects possibly account for the different results in previous studies and our own study. In contrast to our study, Alomari et al. (2015), van Marle et al. (2009) observed increased emotional reactivity at time points, where cortisol concentration was highest, whereas we observed the peak of cortisol concentration immediately after stressor offset and an already declining cortisol response during presentation of the ER tasks blocks (see Figure 4A). We therefore could have missed the increase in emotional reactivity due to timing issues.

With respect to ER, we confirmed the hypothesis that stress negatively affects the individual's ability to regulate negative emotions via reappraisal on multiple response levels. It has previously been shown that glucocorticoids and catecholamines affect the function of the PFC (Arnsten, 2009; Hermans et al., 2014), parts of which play a key role in successful cognitive reappraisal (Buhle et al., 2014). Further, previous research has demonstrated that acute stress impairs PFC-related executive functions, such as WM, cognitive flexibility, and cognitive inhibition (i.e., the ability to inhibit thoughts or internal distractors) (Shields et al., 2016 for meta-analysis), which are assumed to be crucial for successful ER (Compas, 2006; Hofmann et al., 2012; Ochsner et al., 2012). Thus, diminished allocation of neural resources to these executive functions may also result in less successful cognitive reappraisal. This proposition, however, has to be further investigated by using fMRI in a similar study design

than ours.

Our hypothesis of a stress-related impairment of reappraisal was only partly confirmed, as we did not observe the expected effects in the early stress phase. Our assumptions were based on a model proposed by Hermans and colleagues (2014) which stated a dynamic shift of network balance towards a salience processing network at the expense of executive functioning after stress exposure. This shift in neural network functioning is greatest during stressor exposure and reverses after approximately 60 minutes relative to stressor onset. Thus, we expected greater ER impairment in the first compared to the late stress phase. However, we observed the reverse pattern (i.e., stress-related impairments in cognitive reappraisal in the late stress phase). Different explanations could account for that result:

First, the stress compared to the placebo task might have led to greater mental fatigue, particularly in the second half of the task. Indeed, mental fatigue has previously been shown to negatively affect ER capacities (Grillon et al., 2015). In line with this, a potential increase in mental fatigue might have affected memory and training processes. In this regard, inspection of the subjective emotional ratings revealed that participants from the control group (but not from the stress group) showed significantly greater regulation effects in the late than the early time window. These effects were not only driven by an impairment of reappraisal in the stress group but by an improvement in reappraisal in controls (see Figure 5). Such improvement could be explained by learning and training effects from the early to the late phase of the experiment in the control group, whereas in the stress group, stress-related mental fatigue might have disturbed such training processes. Moreover, stress is known to directly impair and affect learning and memory processes (see Shields et al., 2017; Wolf et al., 2016) and might additionally have impaired an improvement in the use of cognitive reappraisal in the stress group.

Second, the model by Hermans and colleagues (2014) provides suggestions on stress-related alterations in resting-state networks, and thus likely cannot be transferred to active task networks (here ER task), where highly interactive emotional and cognitive processes must be integrated. Future studies are therefore needed to directly address the neural and endocrine stress response in relation to ER and its underlying brain networks. Interestingly, a recent study by Jentsch, Merz, and Wolf (2019) showed that the administration of 30mg cortisol 90 minutes prior to an ER task facilitates ER processes. Together with our findings, this result suggests that the dynamics of stress hormone responses in relation to ER are of particular importance.

Considering previous research, our results are in line with the study of Raio and

colleagues (2013), who report an impairment of the regulation of conditioned fear responses using cognitive reappraisal after acute stress exposure. However, the overall results of studies regarding the influence of acute stress on ER provided very heterogeneous results. While Kinner et al. (2014) and Shermohammed et al. (2017) reported no or rather inconsistent effects, Langer et al. (2020) found contrary results to our study in the form of an improvement of reappraisal in men while using a comparable methodological approach. Particularly, in the study of Langer and colleagues (2020) the ER task was applied 15 min after stressor offset, which corresponds to the late time window of our study. The differences in results could be explained by the chosen stimuli material as both studies differed with respect to the normative ratings regarding valence and arousal. In contrast to the study of Langer ($M_{valence}$ = 3.55, $M_{arousal}$ = 4.35) we used highly aversive stimuli pictures ($M_{valence}$ = 2.09; $M_{arousal}$ = 6.59) which might have significantly increased the task difficulty and therefore might provoke reappraisal failure. In this regard, previous studies already suggested that reappraisal is less effective for highly intensive emotional situations (Murphy & Young, 2017; Shafir et al., 2015; Sheppes et al., 2011, 2014; Sheppes & Levin, 2013). Probably, potential dysfunctional effects of acute stress exposure on ER are crucial only during high task demands that require more prefrontal-based cognitive resources. Future studies could answer this question by comparing high vs. low aversive stimuli material in an ER task under stressful conditions. Moreover, as the effects of glucocorticoids and catecholamines follow an inverted U-shape dose-response curve, the differing results of the studies also could be due to the release of different amounts of these hormones or neurotransmitters, respectively. In this regard, moderate stress levels may induce an enhancement while high levels could lead to an impairment of cognitive reappraisal. Future research should address this question by varying different levels of glucocorticoids and/or catecholamines (i.e., using different dosages of hydrocortisone).The results of this latter and the present study are also relevant to mental health prevention training as well as psychotherapeutic interventions including ER techniques. Although cognitive reappraisal is one of the most effective emotional regulation strategies, it is prone to interfering processes and disruptive contextual conditions, such as the high emotional intensity of stimuli and additional stressful situations. Our results suggest that other ER strategies that are less reliant on prefrontal regions may be better suited to change the emotional response after acute stress exposure as well as that the timing of using different ER strategies may be crucial. For instance, Jentsch et al.'s findings (2019) suggest using cognitive reappraisal preferably 90 min after stress than too shortly after stress. In this regard, future research should apply different ER approaches in order to identify more stress-resilient strategies.

The results of the present study should be considered within the scope of a few limitations. There are numerous ways of measuring ER and cognitive reappraisal and each measuring method has advantages and disadvantages. In the present study, we decided to implement the task cue prior to stimulus presentation to assure a clear stimulus onset and to avoid preceding habituation processes. Therefore, we used a well-established method that has already been used in previous neuroscientific experimental studies (e.g., Langer et al., 2020; Ochsner et al., 2004; Schönfelder et al., 2014; Thiruchselvam et al., 2011). However, a disadvantage of our paradigm is that the timing of the task cue prior to the stimulus could provoke confounding anticipation processes, such as affective cueing (e.g., Liu et al., 2016; Shafir & Sheppes, 2018). For instance, an ERP study reported increased LPP response after an informative cue predicted an emotional compared to a neutral stimulus (Liu et al., 2016). Thus, we cannot exclude the possibility that stress might have affected such early anticipation processes. In this regard, future studies investigating the influence of stress on cognitive reappraisal should take this into consideration (e.g., by implementing an induction phase prior to the task condition). Further, we did not discriminate between different types of negative emotions, such as fear, anger, or sadness. Due to time limits, we were not able to include enough stimuli per category to analyze them separately, particularly with respect to event-related potentials. Stress may alter the reactivity to or the regulation of some emotions while others remain rather unaffected. Future studies could take this aspect into account as this may also be crucial for training programs or clinical interventions.

In summary, we found that stress significantly affects subjective and psychophysiological measures of ER with stress exposure hampering cognitive reappraisal predominantly during the late stress phase. Together with recent findings from other groups, the present study suggests that stress-related impairments of ER are critically dependent on the timing of applying ER strategies in relation to stress exposure.

4. Study 2:

Acute Stress Exposure Impairs Reappraisal but not Distraction

Abstract

Distraction and reappraisal are the two most studied ER strategies. Recent studies have suggested an impairment of reappraisal following stress exposure. The present study aimed to extend previous findings by investigating stress-related effects on reappraisal compared to distraction. Fifty healthy men and women were either exposed to social-evaluative stress (N=25) or a control condition (N=25). Subsequently, the participants either had to view, distract, or reappraise aversive and neutral pictures. Subjective emotional self-ratings, EMG, and EEG data were obtained to measure ER. Salivary and α-amylase samples were assessed in order to reflect neuroendocrine stress responses. Stress led to a temporary increase in salivary cortisol and negative affect. The results of EMG and emotional state ratings revealed a stress-related impairment of reappraisal while distraction was not affected. The analysis of the EEG data indicated no stress-related impairments. Glucocorticoids and catecholamines affect regions of the PFC which are related to ER. In this regard, acute stress exposure might have impaired reappraisal but not distraction, as reappraisal relies more on PFC and is related to higher cognitive costs. These findings could contribute to improving the development of therapeutic interventions regarding stress-related mental disorders.

4.1. Introduction

The ability to successfully regulate one's maladaptive emotions represents a core mechanism in the maintenance of mental health or illness (e.g., Berking and Wupperman, 2012). These mechanisms are summarized under the concept of ER. According to the process model of ER by Gross (1998), emotional processing is composed of successive stages that can be targeted by different families of ER strategies. These strategies are divided into antecedent-focused and response-focused strategies (Gross, 1998). Antecedent-focused strategies occur early in the emotion generation process and include strategies such as attentional deployment and cognitive change. Response-focused strategies are initiated once an emotion is already generated and include strategies of response modulation. Particularly ER strategies of attentional deployment (e.g., distraction) and cognitive change (e.g., cognitive reappraisal) have received the most attention in previous research (e.g., Kanske et al., 2015, 2015; Lois et al., 2017; McRae et al., 2010; Scheibe et al., 2015; Schönfelder et al., 2014; Sheppes and Meiran, 2007; Strauss et al., 2016; Thiruchselvam et al., 2011). Distraction redirects attention away from emotion-triggering aspects of a stimulus or situation (Gross, 2013),

leading to a decrease in emotional responding, whereas cognitive reappraisal yields an altered interpretation of an emotional stimulus or situation in order to change its affective impact. Distraction and reappraisal have both been shown to be highly effective, however, reappraisal results in greater decreases of negative emotion and is the most effective way to down-regulate self-reported emotions (e.g., Webb et al., 2012).

The use of maladaptive ER strategies is related to symptoms of psychopathology and clinical disorders such as anxiety disorders, borderline personality disorder, depression, or posttraumatic stress disorder (Aldao et al., 2010; Eftekhari et al., 2009; Sheppes et al., 2015). Particularly, stress may play a key role in ER failure, as it is known to have detrimental effects on brain regions mediating executive functioning, emotional processing, and ER, such as the PFC and the amygdala (Arnsten, 2009, 2015; Lupien et al., 2009; McEwen & Morrison, 2013; Shields et al., 2016). Previous studies that addressed the influence of acute stress on ER revealed heterogenous results: While Shermohammed et al. (2017) found no effects of acute stress exposure on cognitive reappraisal, Raio and colleagues (2013) reported a stress-related impairment of cognitive reappraisal of previously fear-conditioned stimuli. Further, Kinner and colleagues (2014) reported an increase in emotional arousal during distraction after acute stress exposure, while reappraisal was not affected. A study by Langer et al. (2020) reported an improvement of cognitive reappraisal in men after acute stress exposure, but no effects on distraction. Recently, we investigated the role of the temporal dynamics of a stress response in a neurophysiological study (Study 1). Participants either had to reappraise and view aversive and neutral pictures during an early (0-20 min) and a late (20-40 min) stress phase after stressor offset. We found an impairment of reappraisal in the stress compared to the control group only in the late stress phase, indicating that stress-related effects on ER may depend on the timing of applying the ER strategies in relation to the stress exposure. As corticosteroids and catecholamines directly affect the function of the PFC (e.g., Arnsten, 2009; Hermans et al., 2014), the impairment of reappraisal may be based on an impaired allocation of neural resources and underlying executive functions (e.g., WM) which determine reappraisal. Different explanations may account for the varying results regarding acute stress exposure on ER. So far, the previous studies that have investigated the effect of acute stress on ER have applied different methodological approaches and operationalizations, such as the implementation of different stress protocols, stimuli material, and dependent variables. Moreover, as the effects of corticosteroids and catecholamines follow an inverted U-shaped dose-response pattern (e.g., Arnsten, 2009; Dominique et al., 2009), varying stress intensity between the studies might have affected the results.

Regarding our previous findings that reported an impairment of reappraisal after acute stress exposure, it appears important to identify strategies that could potentially be more effective under stressful conditions. A promising approach could be the ER strategy of distraction. In this regard, recent studies of ER choice have revealed that participants preferred distraction over reappraisal in contexts of high emotional intensity (Murphy and Young, 2017; Shafir et al., 2015; Sheppes et al., 2014, 2011; Sheppes and Levin, 2013). It is assumed that the tendency to prefer distraction in situations with high emotional intensity can be explained by the amount of cognitive resources required for the regulation process, as reappraisal requires greater cognitive costs (Shafir et al., 2015, 2016; Sheppes et al., 2009; Sheppes & Meiran, 2008). Thus, the advantages of distraction over reappraisal may also applicable to stressful conditions, as acute stress exposure can lead to an increase in emotional intensity (Alomari et al., 2015a; van Marle et al., 2009) and impair executive functioning (e.g., Shields et al., 2016). Moreover, reappraisal engages at a later stage compared to distraction. Particularly in highly intense emotional situations (such as acute stress exposure), relatively early attentional selection processes are assumed to be more efficient, as they block emotional information processing (e.g., Hay et al., 2015; Shafir et al., 2015; Sheppes et al., 2014).

Neurophysiological and peripheral measurements represent a valid and objective measurement of ER. ERPs map cognitive and affective processes with a high temporal resolution. The LPP is associated with emotional processing and is modulated by ER (see Hajcak et al., 2010). As the LPP is typically increased for positive and negative compared to neutral stimuli, the LPP is assumed to reflect emotional arousal (Hajcak et al., 2010a; Schupp et al., 2006). Facial EMG represents a valid method to measure emotional valence (van Oyen Witvliet & Vrana, 1995) and has been implemented in a variety of ER studies (Jackson et al., 2000; Pedder et al., 2016; Ray et al., 2010; Schönfelder et al., 2014, Study 1).

The goal of the present study was to replicate and extend our previous findings (Study 1) by directly comparing reappraisal and distraction after acute stress exposure using neurophysiological methods (EMG, LPP) and subjective self-reports. We suggested an impairment of reappraisal as well as distraction due to dysfunctional effects of corticosteroids and catecholamines on PFC-related executive functions. However, as stress exposure leads to an impaired allocation of neural resources which in turn mediates ER (via PFC functioning) (e.g., Arnsten, 2009), we assumed a significantly greater impairment of reappraisal compared to distraction, due to greater cognitive costs (e.g., Shafir et al., 2015; Sheppes et al., 2009; Sheppes and Meiran, 2008).

4.2. Methods

4.2.1. Participants

Fifty subjects, aged 18-58 years, were recruited for study participation via social media (e.g., Facebook, eBay) and at the Freie Universität Berlin, Berlin, Germany. All subjects were German native speakers, had normal or corrected-to-normal vision, and a body mass index (BMI; kg/m²) ranging between 19 and 29. Subjects were randomly assigned to either a stress (n=25, n_{female}=17) or control group (n=25, n_{female}=17). Forty-one percent of female participants used oral contraceptives (n_{stress}=9, $n_{control}$=5). Exclusion criteria were: any acute or chronic physical disease (e.g., cardiovascular disease, diabetes, irritable colon, endocrine disease, neuropathy, epilepsy), pregnancy or breastfeeding period, specific medication (psychotropic drugs, anxiolytics, beta-blockers, drugs containing glucocorticoids), current infection, and psychiatric disorder (borderline or antisocial personality disorder, schizophrenia, psychosis, delusional disorders, bipolar disorder, suicidal tendency, alcohol/illegal drug abuse/addiction). The exclusion criteria were verified by an online screening implemented during the recruitment process (unipark.com, QuestBack GmbH). Subjects were instructed to refrain from eating and drinking anything except water one hour prior to the experiment and to abstain from alcohol, smoking (smoking > 5 cigarettes/day was an exclusion criterion) and exercising. In order to control for the diurnal cycle of cortisol, the assessment took place between 12:00 p.m. and 4:00 p.m. The study was approved by the ethics committee of Freie Universität Berlin. The participants either received course credits or €40 reimbursement for their participation. Due to strong artifacts (e.g., blinks, movements) as well as technical difficulties, nine participants had to be excluded from EMG data analysis ($n_{control}$=7; n_{stress}=2) and four participants from EEG analysis ($n_{control}$=4). The data of one participant of the control group had to be excluded from the analysis of the salivary samples.

4.2.2. Experimental design and procedure

During the online recruitment process, questionnaires measuring depressive symptoms (BDI-II; Beck et al., 1996), ER (CERQ; Garnefski et al., 2001), and clinically relevant symptoms (BSI; Derogatis and Melisaratos, 1983) were administered. At the beginning of the experiment, subjects gave their written informed consent. Afterward, subjects rested for about 60 minutes while the EEG and EMG setups were prepared. Following this, the participants were given detailed instructions on the experimental procedure. To verify the correct use of distraction and cognitive reappraisal, both strategies were practiced before the ER started. Subsequently, the first salivary sample (T0, baseline) was collected using a

salivette sampling device (Sarstedt, Nümbrecht, Germany). Afterward, the participants underwent the stress induction or placebo protocol. After stressor offset, second (T1, +12 min) and third (T2, +17 min) saliva samples were collected. Further, mood state ratings measuring negative affect with the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988) were assessed simultaneously to the salivary samples to T0 and T1. Afterward, subjects underwent the ER paradigm. Fourth (T3, +30min) and fifth salivary samples (T4, +45 min) were collected halfway through and at the end of the ER paradigm, respectively. Finally, the participants were debriefed. Saliva samples were stored at -20°C before the devices were assayed by Dresden LabService GmbH (Dresden, Germany) using chemiluminescence immunoassay kits.

4.2.3. Stress protocol (stress vs. control)

For stress induction, a well-established stress paradigm (e.g., Akdeniz et al., 2014; Dahm et al., 2017; Lederbogen et al., 2011) was adapted. This stress task represents a valid procedure to induce stress under fMRI conditions by transferring basic principles of the TSST (Kirschbaum et al., 1993) into a neurophysiological setting. The participants were randomly assigned to a stress group (n=25) or a control group (n=25). The timing was kept the same across the stress and control conditions. Two different task types were presented during the stress and the control paradigms: mental rotation and mental arithmetic (see below). For each task type, the trials were presented in a block, each block lasting for 60 sec, followed by a break of 20 sec. The task types were presented in an alternating sequence (arithmetic, rotation, arithmetic, etc.). In total, five blocks per task type were presented, resulting in a total of 10 blocks. Both the stress and control paradigms lasted for about 13 min.

In the stress condition, one male and one female confederate researcher wearing laboratory coats entered the room and were introduced by the experimenter. The participant was told that the confederates will give individual feedback on the participant's task performance while observing them via live video transmission. Accordingly, a webcam was positioned in front of the participant. Afterward, the experimenter and investigators left the room and observed the participant from a separate room. Both investigators were presented on the participant's screen via live video stream. The stress paradigm consisted of two different task types. During the arithmetic task, subjects had to continuously subtract 13 from a four-digit number in a number-matching task. In the mental rotation task, participants were asked to match a three-dimensional object with an identical but rotated target. During the task, negative written feedback was presented on the participant's screen depending on the participant's performance ('wrong', 'too slow'). In addition, participants were pressed for

time. Furthermore, after ~8min the experiment (6/10 blocks) was paused and an investigator of the same sex entered the room, urging the participant to improve their performance, otherwise, the obtained data could not be considered for the study. During breaks, participants saw that the task performance is monitored by the investigators.

With respect to the control condition, there were no investigators, time pressure, or notifications, so as to minimize stress-inducing factors. The participant saw the experimenter turning his or her back towards them via live video stream. In the control arithmetic and rotation task, participants simply had to match two identical numbers or objects, respectively. During the break, the participants saw the information "break".

4.2.4. Emotion regulation paradigm

The ER paradigm is a well-established and valid paradigm that has been successfully used in previous neurophysiological studies (Schönfelder et al., 2014; Thiruchselvam et al., 2011; Study 1). Pictures depicting either neutral or aversive scenes with human characters were selected as stimuli from the IAPS (International Affective Picture System; Lang et al., 1997) and the EmoPics (Emotional Picture Set, Wessa et al., 2010). Subjective ratings of neutral stimuli and negative stimuli differed significantly for valence (neutral: M_v=4.97, SD_v=.17; negative: M_v=2.10, SD_v=.43; t(104.62)=51.75, p<.001) and arousal (neutral: M_a=2.85, SD_a=.23; negative: M_a=6.58, SD_a=.51; t(110.85)=55.79, p<.001). Three task conditions were implemented. During the 'view' condition, participants draw their attention to the presented stimulus but did not manipulate the emotional response. In the 'reappraisal' condition, participants hat to decrease the emotional response by reinterpreting the situation (e.g., to imagine a rather positive outcome of the situation (situation-focused reappraisal)). During the 'distraction' condition, participants had to solve mathematical equations, which were presented crossfading over the image. The participants had to indicate whether the displayed solution was correct or incorrect by pressing a button. Each condition x task combination (view-negative, view-neutral, reappraise-negative, distract-negative, distract-neutral) contained 25 stimuli (125 stimuli in total), which were presented counterbalanced across subjects and implemented in a randomized block design. A pseudo-randomized order was chosen for the picture-instruction combination, which means that neither the stimuli condition nor the task was repeated more than three times in a row. A trial consisted of the presentation of a fixation cross (500 ms), followed by a cue ('view', 'reappraise', 'calculate') (2000 ms). Subsequently, the stimulus was presented for 5000 ms. Subjective emotional state ratings were measured on a 9-point self-assessment manikin scale (SAM) after stimulus presentation, followed by a variable inter-trial interval of 3500-5000 ms. The ER task was

paused after ~13 min for about two min to allow for a break. The complete ER paradigm lasted ~25 min.

4.2.5. Neurophysiological data (EEG, EMG)

EEG data were recorded from a 32-channel Ag/AgCl electrode system with an online reference to the right mastoid in accordance with the international 10–20 system. Vertical and horizontal eye movements were monitored with four additional electrodes (EOG) placed above and below the eyes. The impedances of the electrodes were kept <10 kΩ. EEG and EOG data were registered with a sampling rate of 1kHz and 16-bit A/D conversion with the BrainAmp amplifier (Brain Products, Inc., Munich, Germany). Brain Vision Analyzer II (Brain Products GmbH, Munich, Germany) was used for offline analyses. EEG data was down-sampled to 250 Hz, re-referenced to the averaged mastoid, and filtered with a 0.1 to 4 Hz (24 dB/oct) bandpass. An independent component analysis logarithm was applied for artifact correction (eye blinks and eye movements). Artifacts were semiautomatically rejected with the following criteria: peak-to-peak differences > 300 µV, voltage steps of 50 µV between sampling points, and a maximum difference of less than .50 µV within 100 ms intervals. Additional artifacts were identified and rejected based on visual inspection. ERP epochs were extracted from -500 to 5000 ms relative to stimulus presentation, including a 250 ms pre-stimulus baseline. Stimulus-locked ERPs were constructed by separately averaging trials for each condition and participant. The LPP was quantified as the mean level of activity at a centro-parietal electrode cluster (Cz, P1, Pz, P2) between 500 to 5000 ms during stimulus presentation.

Concerning EMG activity, two 4 mm Ag/AgCl electrodes were placed over corrugator supercilii (Fridlund & Cacioppo, 1986). EMG was amplified using a BrainAmp ExG amplifier (Brain Products GmbH, Munich, Germany) and registered with a sampling frequency of 1 kHz. EMG signals were full-wave rectified and smoothed with a moving average over 125 ms filtered using a 30 Hz low cut-off, a 500 Hz high cut-off, and a 50 Hz notch filter. EMG scores were calculated as activity change relative to the baseline period of 1000 ms prior to stimulus onset (Blumenthal et al., 2005). The time window was defined by averaging the mean activity change relative to the baseline period from 0 to 5000 ms.

4.2.6. Statistical analyses

Statistical data analyses were performed with SPSS23 (IBM, Chicago IL). Effect sizes are reported using partial eta square (η^2p) and Cohen's d, and the statistical significance level was set to $p<0.05$. In order to minimize Type I error, Bonferroni Holm correction was applied for post-hoc analyses (Holm's alpha) if necessary. Neuroendocrine stress induction

was analyzed separately for α-amylase and cortisol data using 5x2 rmANCOVAs with the within-subject factor ‘time’ (T0, T1, T2, T3, T4) and the between-subject factor ‘group’ (control, stress) and the covariate ‘intake of oral contraceptives’. Further, a 2x2 rmANOVAs including the within-subject factor ‘time’ (T0; T1) and the between-subject factor ‘group’ (stress; control) was performed to test for affective stress response (negative affect). To test successful task manipulation (i.e., significant ER effects through distraction and reappraisal), 3x2 rmANOVAs were performed for the negative stimuli condition including the within-subject factor ‘task’ (view, reappraise, distraction) and the within-subject factor ‘group’ (control, stress) for each dependent variable. To test valid emotion induction, i.e., more aversive processing of negative as compared to neutral stimuli, 2x2x rmANOVAs were conducted with the within-subject factor ‘valence’ (neutral, negative) and the between-subject factor ‘group’ (control, stress) of the view task conditions for each dependent variable. To test for stress-related impairments on reappraisal and distraction, 3x2 rmANOVAs were calculated for each dependent variable including the within-subject factor ‘task’ (negative: view, reappraise, distraction) and the between-subject factor ‘group’ (control, stress).

4.3. Results

4.3.1. Sample characteristics

The stress group (M_{age}=27.00, SD_{age}=8.03; M_{BMI}=21.99, SD_{BMI}=2.11) did not differ from the control group (M_{age}=32.00, SD_{age}=10.61; $M_{BMI=}$22.63, SD_{BMI}=2.84) with respect to age, t(44.68)=1.88, p=.07, d=.53; BMI, t(48)=.911, p=.37, d=.26, depressive symptoms, t(48)=.22, p=.83, d=.06; use of ER strategies (all t’s<1.65, all p’s>.10), and clinically relevant symptoms, t(48)=.65, p=.52, d=.18. There was no difference between the stress and the control group with respect to sex ratio, X^2(1, N=50)<.001, p=1.

4.3.2. Manipulation checks

Neuroendocrine stress response: With respect to the salivary cortisol response, the analysis revealed a significant time x group interaction, F(2.19,100.91)=3.30, ε=.55, p=.04, η^2p=.07. The covariate *intake of oral contraceptives* was significantly related to the participant’s salivary cortisol level as shown by a significant interaction of time x oral contraceptives, F(2.19,100.91)=3.72, ε=.55 p=.02, η^2p=.08. Independent sample t-tests indicated significant greater salivary cortisol in the stress compared to the control group to T2, t(1,41.22)=2.21, p=.03, d=.63, while all other time points (T0, T1, T3, T4) revealed no significant group differences (all t’s≤1.21, all p’s≥.23). Cortisol time curves are depicted in Figure 8. With

respect to salivary **α-amylase,** there was no significant time x group interaction, $F(4,184)=.54$, $p=.71$, $\eta^2p=.01$, indicating no stress-related response of the SNS.

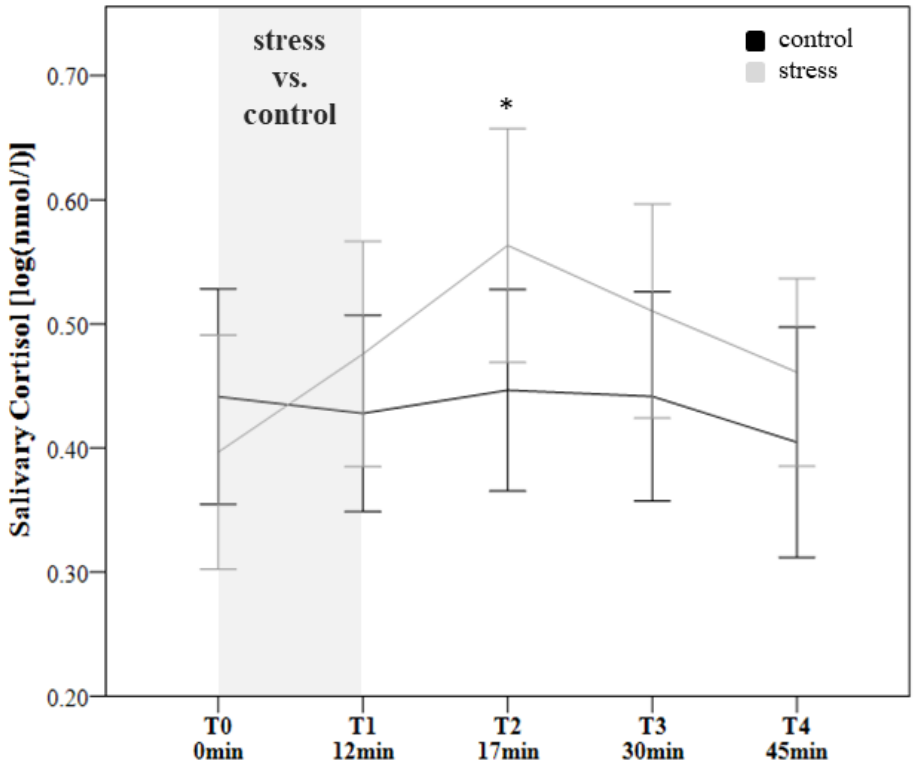

Figure 8. Stress Response. Salivary cortisol measured at five points. The shaded area represents the stress or the control condition, respectively. For better graphical representation, data has been logarithmized. * p < 0.05, t-tests

Affective stress response: A significant time x group interaction, $F(1,48)=17.93$, $p<.001$, $\eta^2p=.27$, indicated a stress-related increase in negative affect. Independent sample t-tests showed significant higher negative affect in the stress compared to the control group after stressor offset (T1), $t(32.11)=3.44$, $p<.01$, $d=.97$, while there was no group difference before stressor onset (T0), $t(48)=.76$, $p=.45$, $d=.22$. The affective stress response is displayed in Figure 9.

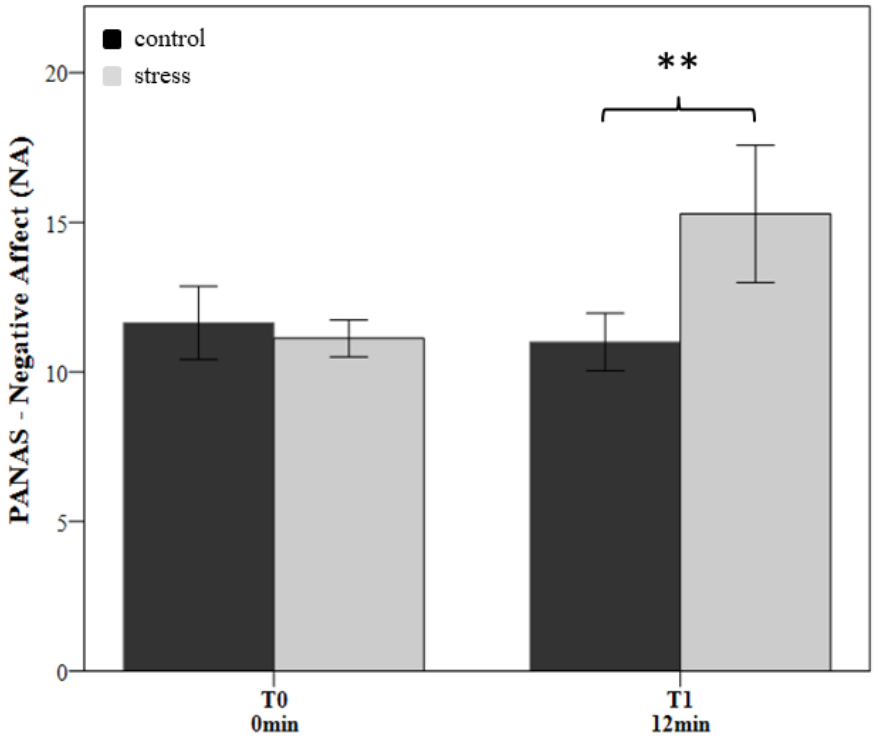

Figure 9. Pre- and post stress effects on negative affect. Error bars represent +/- 1 standard error of the mean. ** p < 0.01, t-tests

Emotion induction: The analysis revealed successful emotion induction reflected by significant main effects of valence for subjective emotional ratings, $F(1,48)=205.20$, $p<.001$, $\eta^2p=.81$, EMG magnitudes, $F(1,39)=7.76$, $p=<.01$, $\eta^2p=.17$, and the LPP, $F(1,44)=84.71$, $p<.001$, $\eta^2p=.66$, indicating more aversive processing of negative as compared to neutral stimuli (lower scores on SAM ratings, higher EMG and LPP magnitudes).

Task manipulation: The analyses revealed significant main effects of the task with respect to subjective emotional ratings, $F(2,96)=75.94$, $p<.001$, $\eta^2p=.61$, EMG magnitudes, $F(2,78)=15.08$ $p<.001$, $\eta^2p=.17$, and the LPP, $F(2, 88)=40.20$, $p>.001$, $\eta^2p=.48$. The results consistently indicated more aversive processing of non-regulated as compared to regulated stimuli (lower scores on SAM rating, higher EMG and LPP magnitudes).

4.3.3. Stress-related impairments of cognitive reappraisal and distraction

Subjective emotional state ratings (SAM): With respect to stress-related impairments in ER, the analysis revealed a significant task x group interaction, $F(2,96)=4.08$, $p=.02$, $\eta^2p=.08$. Independent sample t-tests revealed significantly higher negative emotional state ratings in the stress compared to the control group in the reappraisal condition, $t(48)=-2.19$, $p=.03$, $d=0.62$, but not in the distraction, $t(39.78)=.38$, $p=.70$, $d=.11$, or view condition,

t(48)=43, *p*=.67, *d*=.12. The results indicated a stress-related impairment of reappraisal but not of distraction (see Figure 10).

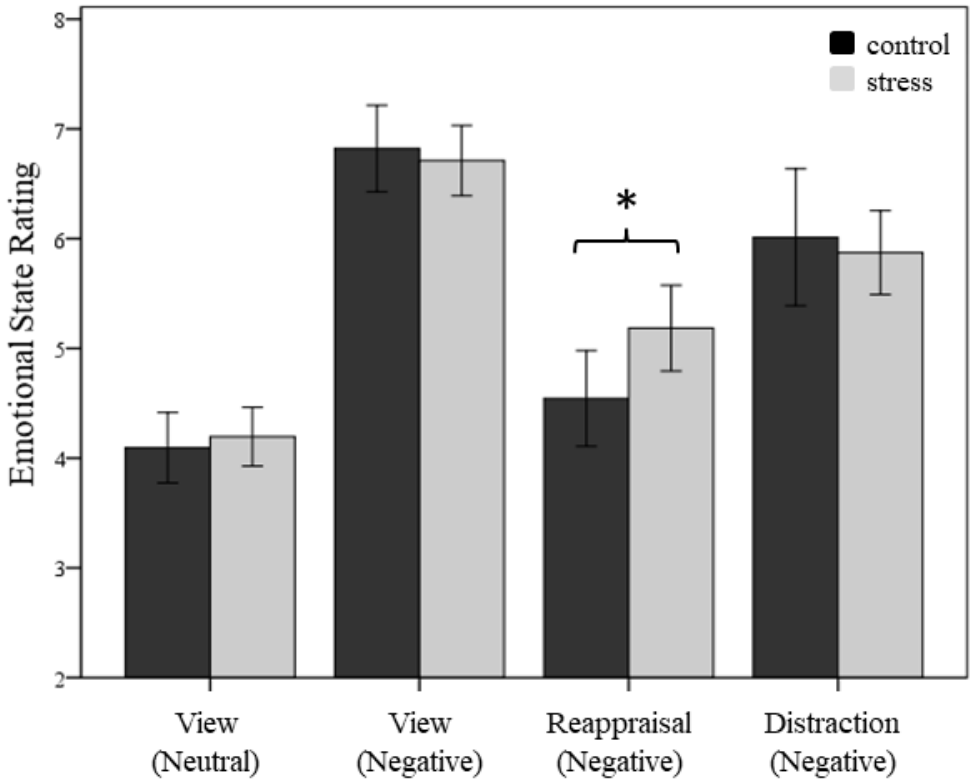

Figure 10. Subjective emotional state ratings. Error bars represent +/- 1 standard error of the mean. * p < 0.05, t-tests

EMG: The analysis revealed a significant task x group interaction, *F*(2,76)=3.64, *p*=.03, η^2p=.09. Independent sample t-tests revealed significantly higher corrugator activity in the stress compared to the control group for reappraisal, *t*(37.60)=2.85, *p*<.01, *d*=.58, but not for distraction, *t*(39)=.02, *p*=.99, *d*=.06, or view, *t*(39)=1.08, *p*=.29, *d*=.31. Regarding corrugator activity, the effects indicated a stress-related impairment of cognitive reappraisal, but not distraction. EMG graphs are depicted in Figure 11.

LPP: The analysis revealed no significant task x group interaction, *F*(2,88)=2.50, *p*=.09, η^2p=.05, indicating no stress-related effect on cognitive reappraisal or distraction (see Figure 12).

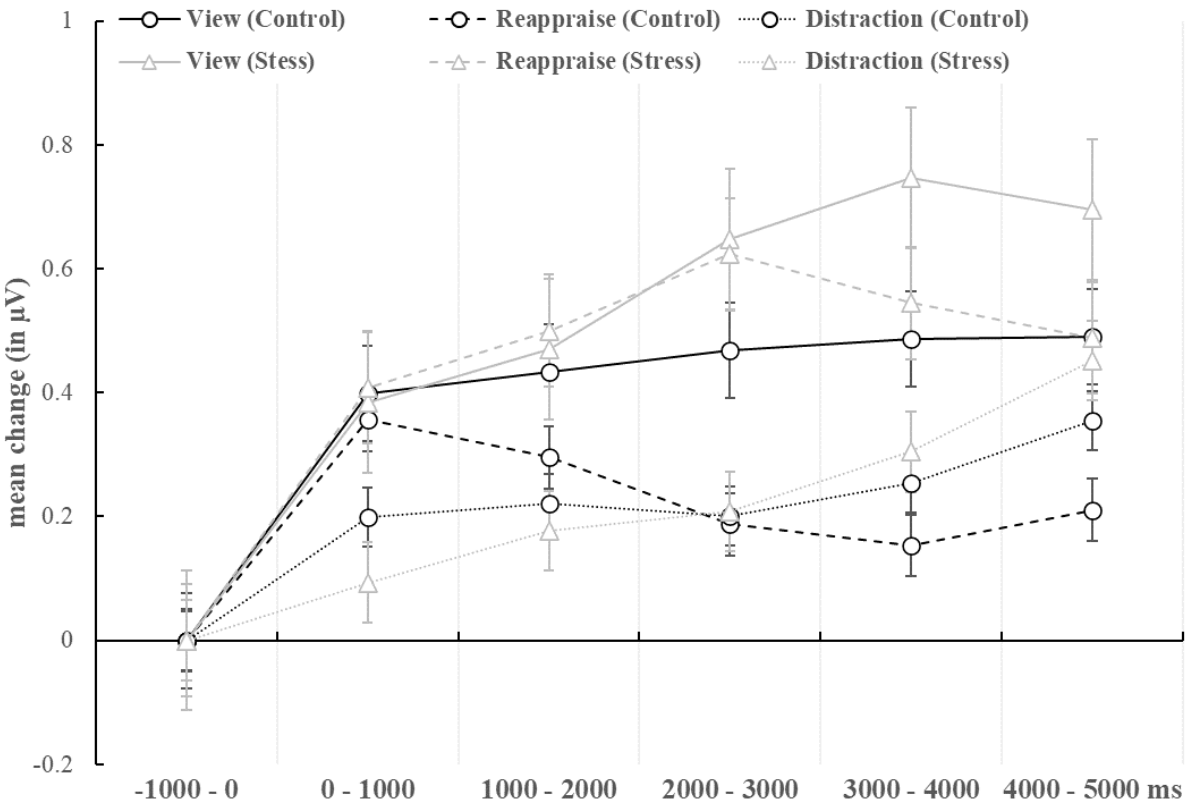

Figure 11. Corrugator supercilii - EMG activity of the negative stimulus condition. Error bars represent +/- 1 standard error of the mean.

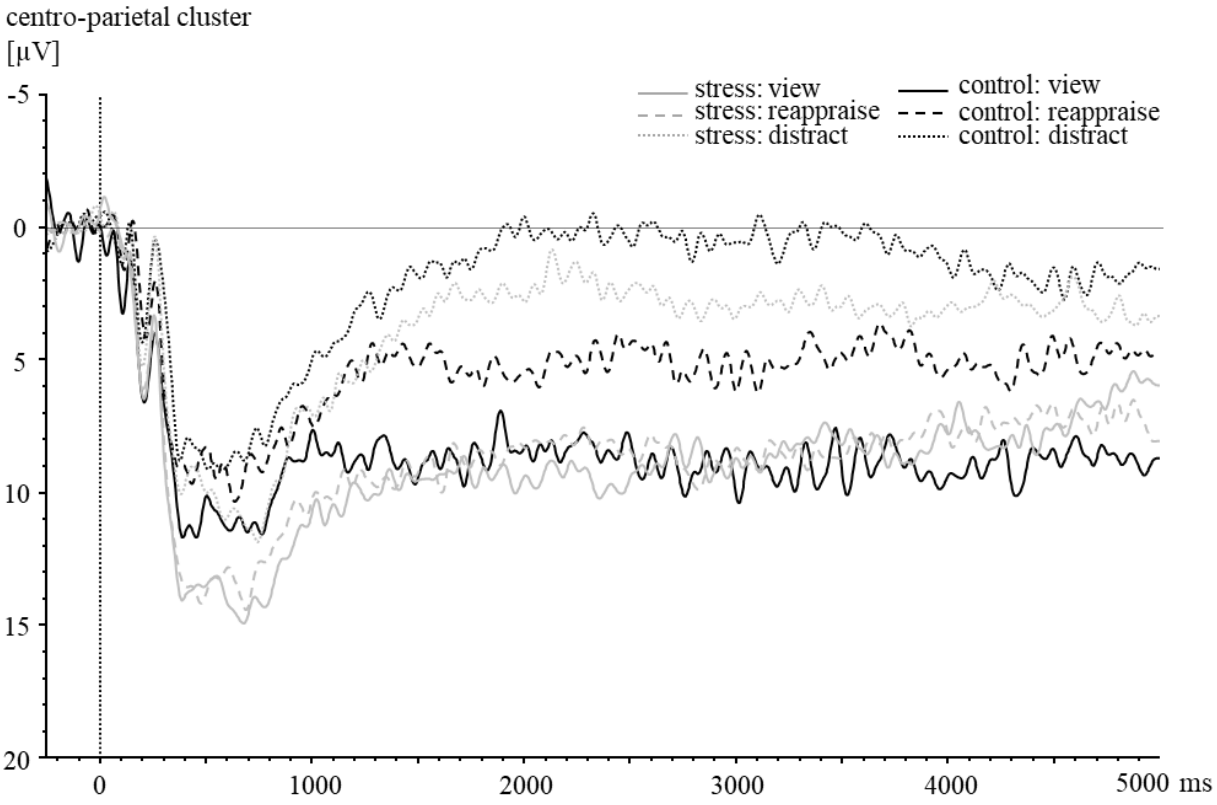

Figure 12. ERPs of the centro-parietal cluster of the negative stimulus condition.

4.4. Discussion

The present study aimed to investigate the influence of acute stress exposure on cognitive reappraisal and distraction using neurophysiological measures, including the LPP, Corrugator electromyographic activity, and subjective emotional state ratings. As previous studies had found an impairment of reappraisal (Raio et al., 2013; Study 1), the present study aimed to identify a more stress-resilient ER strategy by contrasting cognitive reappraisal to distraction. We hypothesized that both ER strategies would be impaired after acute stress exposure due to dysfunctional effects of corticosteroids and catecholamines on PFC-related executive functioning. However, we assumed greater impairments for reappraisal than distraction due to higher cognitive costs of reappraisal (Shafir et al., 2015; Sheppes et al., 2009; Sheppes & Meiran, 2008) as distraction compared to reappraisal relies on more parietal regions (Kanske et al., 2011; Ray et al., 2010), which are less affected by corticosteroids and catecholamines. As a stress paradigm, we implemented an adapted version of a well-established stress protocol (Akdeniz et al., 2014; Dahm et al., 2017; Lederbogen et al., 2011).

Stress-induction: Results indicated that we were partially able to induce a valid stress response. Participants in the stress compared to the control condition displayed a higher cortisol level around ~17 minutes after stress offset and an increase in negative affect. However, the stress exposure did not lead to a sustained increased cortisol level. Moreover, we could not detect stress-related changes in the α-amylase level reflecting the activation of the SNS.

Emotional regulation: Results of all assessment levels (subjective emotional state ratings, EMG Corrugator activity, LPP) indicated successful induction of negative emotion using aversive vs. neutral pictures and successful ER using either reappraisal or distraction as opposed to the view task condition.

Stress-related effects on ER*:* We found that psychosocial stress exposure led to an impairment of reappraisal. This effect seems to be consistent as it was reflected by medium effect sizes regarding the subjective emotional state ratings and EMG Corrugator activity. However, the LPP did not indicate a stress-related impairment of ER. Moreover, contrary to our expectation, distraction was not affected by acute stress exposure at all.

In sum, the results are in line with the initial assumptions of a stress-related impairment of reappraisal and a greater stress resistance of distraction. Reappraisal is known to be the most effective strategy to reduce emotional aversive states (Webb et al., 2012). However, the present findings suggest that the efficacy of reappraisal may depend on mediating factors, such as stress exposure. As a result, under stressful conditions, other

strategies (e.g., distraction) might be better suited to regulate aversive emotional states. From a clinical perspective, behavioral dialectic therapy (DBT) has already taken advantage of the stress-resistant effects of distraction by using 'skills' in the treatment of borderline personality disorder. In situations in which distress is very high, patients are asked to use 'TIPP skills' (e.g., splashing the face with very cold water, intensive sports), leading to a distraction away from the initial highly intensive emotional stimuli. Afterward, after a decrease of high emotional distress or at low emotional intensity, reappraisal-like 'skills' are applied (Linehan, 1993). The involvement of higher cognitive processes during cognitive reappraisal, including WM and cognitive flexibility, may lead to a greater vulnerability towards interfering processes and disruptive factors, such as the effects of corticosteroids and catecholamines. This assumption is supported by studies of ER choice, which indicated that under high compared to low intensive emotional situations, participants tend to use distraction over reappraisal (Murphy & Young, 2017; Shafir et al., 2015; Sheppes et al., 2011, 2014; Sheppes & Levin, 2013). Thus, the present results fit well into previous literature considering ER choice and may help to improve upon as well as explain already existing therapeutic interventions in the context of a highly intense emotional situation (i.e., 'skills' in DBT). Future studies regarding ER and stress should include clinical populations, such as patients with borderline personality disorder or depression, in order to develop and improve more stress-resistant therapeutic interventions and strategies.

The sensitivity of cognitive reappraisal towards acute stress exposure has profound effects. The main advantage compared to distraction is that reappraisal enables changes in the form of an affective adaptation due to altered cognitive evaluations on the basis of higher-order mental processes (Wilson & Gilbert, 2008). In line with this, stimuli that were previously distracted compared to those that were reappraised demonstrate a rebound effect in form of an enhanced LPP during re-exposure (MacNamara et al., 2010; Thiruchselvam et al., 2011). On the contrary, reappraisal accords to long-term goals as it attends to and explains emotional information. As a consequence, it provides permanent alterations with respect to the emotional response to a stimulus or situation. Moreover, cognitive behavioral therapy (CBT) involves implementing reappraisal-like strategies in the treatment of mental disorders such as depression (e.g., Beck, 1979) in order to achieve long-term behavioral changes. The present findings suggest an impairment of this fundamental ability, which could in the long term (i.e., chronic stress exposure) facilitate the development of psychopathological symptoms or mental disorders. In this regard, stress has been linked to detrimental effects on physical and psychological variables (e.g., Cohen et al., 2007; Herman, 2012; Kassel, 2003) and a variety of models already feature the role of stress as a

determinant of disordered functioning (e.g., diathesis-stress models) (Ingram & Luxton, 2005).

We hypothesized that reappraisal and distraction should be negatively affected by acute stress exposure as both rely on frontoparietal regions (Buhle et al., 2014; Kanske et al., 2011; McRae et al., 2010), that are also targeted by corticosteroids and catecholamines. However, although both strategies show great overlap in frontoparietal patterns, distraction depends on more parietal regions (Kanske et al., 2011; McRae et al., 2010), while reappraisal is related to greater PFC activation (Golkar et al., 2012; McRae et al., 2010; Olsson & Ochsner, 2008). Thus, reappraisal was probably more targeted by the effects of corticosteroids and catecholamines. Additionally, reappraisal is related to higher cognitive costs (Shafir et al., 2015; Sheppes et al., 2009; Sheppes & Meiran, 2008; Strauss et al., 2016), based on a complex interplay of executive functioning, comprising WM, inhibitory control, and cognitive flexibility (Compas, 2006; Hofmann et al., 2012; Ochsner et al., 2012) that can be particularly impaired under acute stress exposure (cf. Shields et al., 2016). In contrast, the processes which underlie distraction are mainly linked to selective attention and inhibitory control, which are known to be less impaired when compared to WM (Shields et al., 2016) or even improved under acute stress exposure (Chajut & Algom, 2003; Schwabe & Wolf, 2013; Shields et al., 2015).

Limitations*:* A substantial limitation of the present study is the temporary effect of the stress paradigm on the cortisol level, as we observed a significant increase in salivary cortisol only at one time point at the beginning of the ER paradigm. One possible reason is that the cortisol response was diminished due to the high proportion of women using hormonal contraceptives, particularly in the stress group. Women using hormonal contraceptives show smaller cortisol responses than naturally cycling women in response to a psychosocial stressor (Crewther et al., 2015; Kirschbaum et al., 1999; Nielsen et al., 2013, 2014). Moreover, the specifications of the stress protocol could be accountable for the limited and temporary stress effect. Compared to the TSST, the present stress protocol is less demanding as it has a shorter duration and the participant does not perform directly in front of committee members. Hence, the distance to the observers due to the video transmission possibly reduced the awareness of a social evaluative threat, which plays a crucial role in stress protocols. In a follow-up interview, subjects also reported that the intention of the stress protocol was relatively transparent, which could have led to decreased perception of social-evaluative threat. Therefore, the present stress protocol might have not been as effective as other protocols. Hypothetically, a more intense stress response could have led to different results in terms of potential stress-related impairment of distraction. Future studies

should take this into consideration and implement more intense stress protocols in order to investigate the effects of a more intense stress response.

As the significant stress effect only appeared temporary during the experiment, we cannot exclude that other factors might also have determined the impairment of cognitive reappraisal. As the stress paradigm, compared to the control task, was cognitively more demanding, it might have resulted in greater mental fatigue which could have negatively affected the performance of the subsequent ER paradigm. As cognitive reappraisal requires more cognitive resources than distraction, it is conceivably more vulnerable to the detrimental effects of mental fatigue. In this regard, Grillon and colleagues (2015) have already shown that mental fatigue can lead to an impairment of ER. Moreover, previous research showed that the performance of a cognitively demanding task can impede the efficiency of preforming a subsequent task (Schmeichel, 2007).

Moreover, in contrast to the EMG corrugator activity and the subjective emotional state ratings, the LPP did not reflect a stress-related impairment of cognitive reappraisal. While EMG corrugator activity and the emotional state ratings represent emotional valence, the LPP reflects processes of emotional arousal (Hajcak et al., 2010; Schupp et al., 2006; van Oyen Witvliet & Vrana, 1995). Thus, the stress protocol probably only affected emotional valence but not arousal. Nevertheless, as the EEG data suggest a trend with respect to an impairment of reappraisal, a more effective stress protocol (e.g., TSST) might have also consistently induced an impairment of reappraisal on the level of emotional arousal. In this regard, in our previous study (Study 1) we found a stress-related impairment of reappraisal reflected by the LPP as we were able to induce a more effective and valid cortisol stress response.

Taken together, the findings suggest a stress-related impairment of cognitive reappraisal on neurophysiological and subjective measures of cognitive reappraisal, whereas distraction was not affected. The results could contribute to further development of therapeutic interventions regarding the use of appropriate ER strategies in the context of high emotional intensity.

5. General Discussion

5.1. Summary of study findings

The present thesis aimed to investigate the influence of acute stress exposure on ER. For this purpose, two independent studies were conducted:

Study 1 aimed to investigate the influence of acute stress exposure on cognitive reappraisal while also taken the temporal dynamics of the stress response into consideration. Consequently, an ER task was divided into an early (0-20 min) and a late (20-40 min) post-stress phase in order to reflect the predominant activation of the SNS or the HPA axis, respectively. It was expected that acute stress exposure (TSST) impairs cognitive reappraisal and increases emotional reactivity, particularly in the early stress phase due to the dominant activation of a salience-processing network and downregulation of the executive control network (Hermans et al., 2014). The hypotheses could only be partly confirmed as an impairment of reappraisal was only observed in the late post-stress phase on all measures (LPP, corrugator EMG activity, self-reported emotional ratings). Moreover, there was no stress-related increase in emotional reactivity, neither in the early nor late post-stress phases. Further, the neuroendocrine data suggest that the division in an early and late post-stress phases did not properly differentiate between the activation of the SNS or HPA axis, respectively.

Study 2 intended to extend the previous study results by comparing different ER strategies after acute stress exposure. For this purpose, two of the most studied ER strategies (cognitive reappraisal vs. distraction) were compared, using a similar ER paradigm to Study 1. Further, an adapted version of a well-established stress paradigm used in the fMRI context was applied (cf. Akdeniz et al., 2014). It was expected that both ER strategies would be impaired due to acute stress exposure. Moreover, it was assumed that acute stress would lead to greater impairments of reappraisal as compared to distraction as reappraisal requires greater cognitive costs (e.g., Shafir et al., 2016). The results revealed that the stress paradigm induced only a temporary increase in cortisol level. However, comparable to the findings of Study 1, corrugator EMG activity and self-reported emotional ratings indicated an impairment of cognitive reappraisal in the stress compared to the control group, while the impairment was not reflected by the LPP. Acute stress exposure did not lead to an impairment of distraction, confirming the hypotheses of a greater stress-resistance of distraction compared to reappraisal. Similar to Study 1, stress exposure did not lead to alterations in emotional reactivity.

5.2. Main findings and integration of the study results

One of the main findings of both studies in the present thesis is the impairment of cognitive reappraisal in the stress group compared to the control group. The effects were almost consistently found in both studies on all assessment levels, representing stress-related changes in emotional arousal (LPP) and valence (EMG, subjective self-reports) with small to medium effect sizes. In contrast to the initial assumptions, an impairment of reappraisal was only observed in the late phase of the stress response. Moreover, stress-related increases in emotional reactivity were not observed. Thus, the assumptions regarding the effects of temporal dynamics of the stress response on reappraisal as well as the hypothesis of a stress-related heightened emotional intensity impeding the regulation process cannot be confirmed by the present results. Moreover, another key finding is the resistance of distraction towards the effects of acute stress exposure, as no stress-related alterations to the effectiveness of distraction were observed on any assessment level. The subsequent sections review the main findings, discuss potential underlying mechanisms, and point out future research perspectives.

5.2.1. Impairment of cognitive reappraisal

In the context of the current state of research, the present thesis comprises the first studies that investigated the effects of acute stress exposure using neurophysiological measures (EEG, EMG), extending previous findings by integrating new measures of emotional processing. The present results are consistent with the findings of a previous study by Raio et al. (2013) that indicate an impairment in the regulation of conditioned fear responses via cognitive reappraisal after acute stress exposure. Moreover, the results of Study 1 indicate that reappraisal is not impaired by acute stress exposure if applied promptly after stressor off-set. This corresponds with the results of the study conducted by Shermohammed et al. (2017) who report no stress-related effects on reappraisal when alternating the ER and the stress tasks. In contrast to the initial expectations, the results of Study 1 and the findings of Shermohammed et al. (2017) indicate that cognitive reappraisal may be relatively robust to the effects of the early stages of acute stress exposure. Nevertheless, at least at a later stage of the response, acute stress exposure consistently leads to an impairment of cognitive reappraisal as indicated by the results of Study 1, Study 2, and Raio et al. (2013). In this regard, the explicit reasons for the delayed impairment remain unclear. An early network shift in form of an immediate downregulation of the executive control network due to the activation of the SNS as proposed by the model Hermans and colleagues (2014) does not reconcile with the present data. Probably, the neural resources which are required for the

cognitive reappraisal of the stimuli are preserved for at least a short period of time. Whether the impairment of reappraisal is based on the effects of the SNS, the HPA axis, or the interaction of both cannot sufficiently be addressed by the present studies. As no increases in α-amylase were observed in Studies 1 and 2, the present studies at least emphasize the role of the HPA axis and not the SNS. Moreover, a further explanation to address the delayed impairment of reappraisal is to also take stress-independent factors into consideration. For instance, a previous study indicated that mental fatigue can lead to an impairment of ER (Grillon et al., 2015). In this regard, the stress protocol quite possibly demanded a greater cognitive load, which could have had a significant impact on the subsequent ER task, especially at the later stage of the experiment after already processing the ER task for ~20 min. However, the possible causes and underlying mechanisms for the time-delayed impairment of reappraisal remains unclear and cannot sufficiently be answered by the present studies due to methodological limitations. In this regard, future studies should include pharmacological approaches (e.g., via hydrocortisone and catecholamine administration) in order to gain a better understanding of the contribution of the two stress systems, the temporal dynamics, and to exclude other mediating factors, such as mental fatigue. The potential underlying mechanisms (and stress resistance of distraction) are discussed in more detail below (see Section 5.2.4.).

The results are in contrast with previous studies that report either no impairments or even improvements in cognitive reappraisal after acute stress exposure (Kinner et al., 2014; Langer et al., 2020). In this regard, multiple explanatory approaches are worth to be considered in order to understand the divergent results. The study of Kinner and colleagues (2014) found somewhat mixed results, revealing stress-induced impairments in form of greater arousal ratings during distraction and facilitation of reappraisal only in female participants. However, the results of Kinner et al. (2014) need to be interpreted with reservation due to some methodological limitations. First, only subjective ratings of valence and arousal have been measured as a dependent variable for ER. In this regard, the interpretation of subjective ratings can be error-prone (e.g., Paulhus & John, 1998) and should at best be extended by more objective dependent variables, such as neurophysiological approaches. Second, as pointed out by the authors, the findings are also limited due to an unfavorable chosen between-subject design, resulting in a small number of participants per condition. Moreover, a significant stress increase was only observed in men. At last, the stimuli intensity of the negative stimuli condition has differed quite significantly from the stimuli selection of the present studies, which included pictures with substantially more negative valence and higher arousal ratings. This increase in emotional intensity might

have additionally impeded the regulation process (see below for more detailed discussion).

Langer et al. (2020) observed that acute stress exposure increases the effectivity in cognitive reappraisal in men but not in women in form of lower arousal ratings, increased valence ratings, and increased subjective ER success ratings in stress compared to a control group. The comparison of the present study findings with the results of Langer and colleagues appears relevant as their study used quite comparable operationalizations to Study 1 and 2. For instance, Langer presented the ER task ~20 min after stressor-offset, which is comparable to the time window of Study 2 and the late post-stress phase of Study 1. Moreover, the ER paradigm of Langer et al. (2020) was equivalent to the ER task used in the present studies. In this regard, the divergent results are surprising due to the methodological similarities. However, as already mentioned with respect to the study of Kinner et al. (2014), a significant difference between the studies was the selection of the stimuli material. In Study 1 and 2, pictures with extremely negative valence and high arousal ratings in the negative stimuli condition were chosen while Langer and colleagues used more positive valence and lower arousal ratings. Thus, the stimuli material in the present studies compared to Langer et al. (2020) might have been more intense. In this context, the fear-conditioned stimuli via electric wrist-shocks in the study by Raio et al. (2013) is also possibly of higher emotional intensity as compared to the stimuli of Langer et al. (2020). In this regard, studies already showed that stimuli with high emotional intensity are less malleable by reappraisal compared to low-intensity stimuli (e.g., Sheppes et al., 2011; Sheppes & Levin, 2013). Hence, probably the high emotional intensity of the stimuli material played an important role in ER failure by additionally impeding the reappraisal process. Another reason for the divergent effects of the studies may be due to the U-shape response pattern of corticosteroids and catecholamines, meaning that either too high or too low level can result in impairments of higher cognitive function that mediates cognitive reappraisal. Thus, differences in the intensity of the stress protocols could have either led to impairments or even improvements of cognitive reappraisal, depending on the amount of corticosteroid or catecholamine secretion. However, this assumption cannot sufficiently be answered by the present results and needs to be addressed by future studies using different dosages of hydrocortisone or varying the intensity of stress protocols.

Taken together, the results of the present thesis significantly extend previous findings with respect to the influence of acute stress exposure on cognitive reappraisal. Particularly, the results of the conducted studies in addition to the findings of Raio et al. (2013) clearly suggest an impairment of cognitive reappraisal due to acute stress exposure. Nevertheless, this assumption needs to be considered under the influence of multiple determining factors.

First, although the results suggest stress-related impairments of reappraisal, the findings of Study 1 and the study conducted by Shermoahammed et al. (2017) indicate that reappraisal may be preserved and be relatively robust during an early stage of the stress response. In this regard, future work could provide further insights by covering even more time windows, e.g., already during the stressor-presentation (e.g., during an alternation of the ER and the stress task to reliably cover the activation of the SNS) and later time windows after stress-offset. Moreover, contrasting the present results to the study of Langer et al. (2020), the findings suggest that an impairment of reappraisal is likely dependent on the emotional intensity of the stimulus or situation. In this regard, the results suggest that the presence of a stimulus or situation which is already difficult to regulate (i.e., high emotional intensity) may interact with the detrimental effects of acute stress exposure, leading to an impairment of the reappraisal process, while the process may be preserved in the context of less intense stimuli or situations. This is of particular importance, as the comparison of the present studies to Langer et al. (2020) suggests that in the context of less intense emotional situations acute stress exposure may even facilitate reappraisal processes. Thus, further understanding of the effectiveness of reappraisal under acute stress in the context of low and high intense emotional situations could have significant clinical implications. For instance, more flexible use of different ER strategies in the context of stressful situations could improve ER success and as a consequence preventing the development of psychopathological symptoms. Nevertheless, whether the emotional intensity of the stimuli determined the different effects needs to be addressed by future studies. In this regard, studies should systematically vary the emotional intensity of the stimuli during an ER task after acute stress exposure in order to answer this research question.

All in all, the present studies show that acute stress exposure can lead to an impairment of cognitive reappraisal. In this regard, the present results revealed new research prospects particularly by emphasizing the relevance of time-dependent effects. Moreover, the comparison of the present and previous studies points out the role of further potential mediating factors, such as the emotional intensity of the stimuli or the role of the U-shaped dose-response curve between corticosteroids and catecholamines. As described above, these assumptions remain hypothetical for the moment and need to be addressed by future studies.

5.2.2. Stress resistance of distraction

Study 2 focused on the influence of acute stress exposure on reappraisal compared to distraction. While we found a consistent impairment of reappraisal (see Section 5.2.1.), distraction was not negatively affected by acute stress exposure. To the best of the author's

knowledge, there are two studies that also compared the effects of acute stress exposure on distraction and reappraisal which have already been discussed above in the context of stress effects on reappraisal (Kinner et al., 2014, Langer et al., 2020, see Section 5.2.1). The study of Kinner and colleagues (2014) revealed higher subjective emotional arousal ratings in the stress compared to the control group. Although the study to some extent indicates detrimental effects of stress on distraction, the results are substantially limited due to multiple methodological restrictions (see Section 5.2.1). The study by Langer et al. (2020) compared distraction and reappraisal after acute stress exposure using a comparable study design to Study 2. Similar to the results of Study 2, the study by Langer and colleagues revealed no effects of acute stress exposure on distraction.

Presumably, the ER strategy of distraction is not be influenced by the effects of acute stress exposure as it relies on more parietal regions (Kanske et al., 2011; Ray et al., 2010) which are less targeted by corticosteroids and/or catecholamines (Arnsten, 2009; Berridge & Arnsten, 2015). Moreover, distraction compared to reappraisal is related to lower cognitive costs (Shafir et al., 2015; Sheppes et al., 2009; Sheppes & Meiran, 2008) and may therefore be more resistant towards a stress-related impairment of executive functions. Distraction is mainly linked to inhibitory control, which is known to be less impaired or even improved under acute stress exposure (cf. Shields et al., 2016), whereas reappraisal is based on a more complex interplay between multiple higher cognitive processes, such as WM, declarative memory, and cognitive flexibility. Thus, in line with the hypotheses, the impairment of PFC-related cognitive processes due to acute stress exposure may have a greater impact on reappraisal than distraction. In order to explain the findings, it may also be helpful to consider evolutionary aspects, as stress aims to facilitate the adaptation to threatening situations and/or stimuli by shaping cognition and behavior. In the context of a real threat (e.g., the attack of a wild animal), cognitive processes unrelated to the threat (e.g., processes that require WM or cognitive flexibility) are suppressed in order to reduce interference and ambiguity. This ability to suppress potential internal and external interference and to focus on the threatening stimulus is predominantly determined by inhibitory control. In other words, stress leads to a shift from a ‘cognitively flexible’ to a more ‘rigid habit’ system, promoting salience processing (Hermans et al., 2014; Schwabe & Wolf, 2013). Thus, the neural resources and cognitive costs of distraction might have been preserved under stress in order to provide neural resources that promote adaptive behavior regarding survival.

The complete resistance of distraction towards the effects of acute stress exposure is not fully consistent with the initial assumptions. As both distraction and reappraisal rely on

structures of the frontoparietal network, it was hypothesized that acute stress exposure may also lead to an impairment of distraction, although to a lesser extent as compared to reappraisal. Probably, a more intense stress protocol would have also led to an impairment of distraction. In order to answer this research question, future studies need to systematically vary the stress intensity, for instance, in the form of pharmacology studies including the exogenous application of cortisol and catecholamine at different dosages.

In sum, Study 2 revealed that the effects of acute stress exposure can have different effects on different ER strategies. In this regard, the results suggest that distraction reflects a stress-resistant strategy while reappraisal seems to be more stress-susceptible. This is of particular importance, as under non-stressful conditions reappraisal is known to be the most effective strategy in reducing negative emotional states (Webb et al., 2012). The results indicate that in case of experiencing stressful conditions, other ER strategies can be more successful in decreasing negative emotion. This may play a determinant role in the context of clinical interventions. In this regard, techniques of behavioral dialectic therapy are already based on the use of 'skills' in the context of highly stressful and arousing situations (Linehan, 1993). For instance, some of the so-called TIPP-Skills (Temperature, Intense exercise, Paced breathing, Pared muscle relaxation) lead to a distraction from the initial highly intensive emotional stimulus towards sensory perceptions (e.g., intensive sports, cold water on the face). Moreover, due to the alteration of the semantic meaning, reappraisal compared to distraction provides long-term changes of the emotional experience of a stimulus or situation (MacNamara et al., 2010; Thiruchselvam et al., 2011). Thus, under stressful conditions, we could be less able to make adequate learning experiences in order to cope with similar situations in the future. At this point, future studies are needed in order to investigate to which extent these long-term alterations of reappraisal are affected and whether reappraisal remains more effective compared to other ER strategies, such as distraction.

5.2.3. Stress-related alterations on emotional reactivity

In contrast to our expectations, neither Study 1 nor Study 2 revealed a consistent increase in emotional reactivity in the stress compared to the control group. The hypothesis of an increase in emotional reactivity was based on previous findings indicating that acute stress exposure leads to the activation of the salience processing network due to the effects of corticosteroids and catecholamine (e.g., in form of strengthened function of the amygdala). As the amygdala plays a central role in emotional processing (e.g., fear and anxiety) (Phelps & LeDoux, 2005), a strengthened function leads to the facilitation of attentional processes towards salient stimuli (van Marle et al., 2009). To this end, previous studies already

demonstrated that acute stress exposure leads to an increase in emotional reactivity in form of heightened sensitivity and lower specificity of the amygdala (Alomari et al., 2015; van Marle et al., 2009; Weymar et al., 2012). It was expected that one potential cause of ER failure in the context of acute stress exposure is the stress-related increase of the emotional intensity of the stimulus, making it less inclined to being regulated due to greater regulation efforts. However, the results of the present studies suggest that an increase in emotional activity did not play a decisive role in the impairment of reappraisal. Moreover, previous studies that investigated the influence of stress on did not report a consistent increase in emotional reactivity, either (Kinner et al., 2014; Langer et al., 2020; Shermohammed et al., 2017). The reasons for the contradictory findings remain unclear. Shermohammed and colleagues (2017) argue that at least a moderate level of stress induced in laboratory stress tasks may not be sufficient to significantly increase emotional reactivity. In line with this, mild to moderate forms of stress do not necessarily strengthen the emotional associations and motor habits by the amygdala (Arnsten, 2009). However, the studies which investigated stress-related increases in emotional reactivity (Alomari et al., 2015; van Marle et al., 2009; Weymar et al., 2012) and effects on ER (Kinner et al., 2014; Langer et al., 2020; Shermohammed et al., 2017, Study 1, Study 2) used comparable psychosocial stress protocols, yet revealed different results. Nevertheless, a possible approach to investigate the influence of the stress level on emotional reactivity could again be addressed by pharmacological approaches (e.g., by varying the amount of hydrocortisone, or by considering investigations in more naturalistic settings). Thus, based on the heterogeneous results, the effects of stress on emotional reactivity remain unclear and require more studies. In sum, the present findings suggest that the impairment of reappraisal is not further affected by alterations of emotional processing in the form of an increase in emotional sensitivity towards the stimuli.

5.2.4. Potential role of PFC-related executive functions

Although the data of the present studies do not allow to conclude the precise neural mechanisms underlying the impairment of reappraisal, the non-genomic effects of corticosteroids and catecholamines on the PFC and amygdala seem to be of particular relevance in explaining the impairment of cognitive reappraisal. Prefrontal-related executive functions are downregulated in favor of a salience processing network that is mainly associated with limbic structures (Hermans et al., 2014). As a result, stress leads to substantial alterations in frontoparietal regions (Arnsten, 2009, 2015) that mediate cognitive reappraisal, such as the dmPFC, dlPFC, and vlPFC (Kanske et al., 2011; McRae et al., 2010).

A large body of literature has revealed that acute stress exposure impairs higher cognitive processes (e.g., WM and cognitive flexibility and to a lesser degree, inhibitory control, due to its detrimental effects on PFC functioning) (Shields et al., 2015, 2016). As ER is a very complex, goal-directed process that depends on the interplay of multiple cognitive functions, (Compas, 2006; Gyurak et al., 2011; Hofmann et al., 2012; Ochsner et al., 2012; Zelazo & Carlson, 2012), an impairment of PFC-related executive functions might have mediated the impairment of cognitive reappraisal. Specifically, cognitive reappraisal is known to substantially rely on WM, which facilitates the online maintenance and manipulation of information needed to change the initial evaluation of a stimulus (Aldao et al., 2015; Compas, 2006; Cunningham & Zelazo, 2007; Sheppes & Levin, 2013). Moreover, not only does WM and cognitive reappraisal show high overlap in PFC areas (Buhle et al., 2014; Kanske et al., 2012; Ochsner et al., 2012; Ray et al., 2010), but a previous study by Schweizer and colleagues (2013) revealed that WM training can improve cognitive reappraisal performance. Thus, the stress induction might have affected WM performance, leading to an impairment of associated updating and integration processes crucial for the online manipulation of stimulus information, reflecting a key principle of reappraisal.

The specific mechanism behind the stress-related impairment of reappraisal remains unclear and can only be hypothesized in the framework of the present thesis. Future studies could address this issue by clarifying the contribution of different higher cognitive processes to ER. In this regard, neuroimaging methods such as fMRI could provide important insights with respect to the neural bases of the effects, which could not be addressed by the present study procedures.

5.3. Synthesis model, research perspectives, and clinical implications

Based on previous findings and the present results, a preliminary multi-stage model of the interaction of stress and ER is outlined (Figure 13). The first part of the model (Figure 13a) focuses on the influences of acute stress exposure on ER based on the findings and implications of Study 1 and 2 as well as previous studies regarding the influence of stress on emotional processes and cognitive functioning. The second part of the model (Figure 13b, c) needs to be considered as mainly theoretical with the intention to stimulate further research and bridge the gap to clinical implications. Accordingly, the model proposes simplified hypotheses based on previous studies about a reciprocal interaction of impaired ER and stress emphasizing potential implications and perspectives regarding ER failure, psychopathological symptoms, and mental disorders.

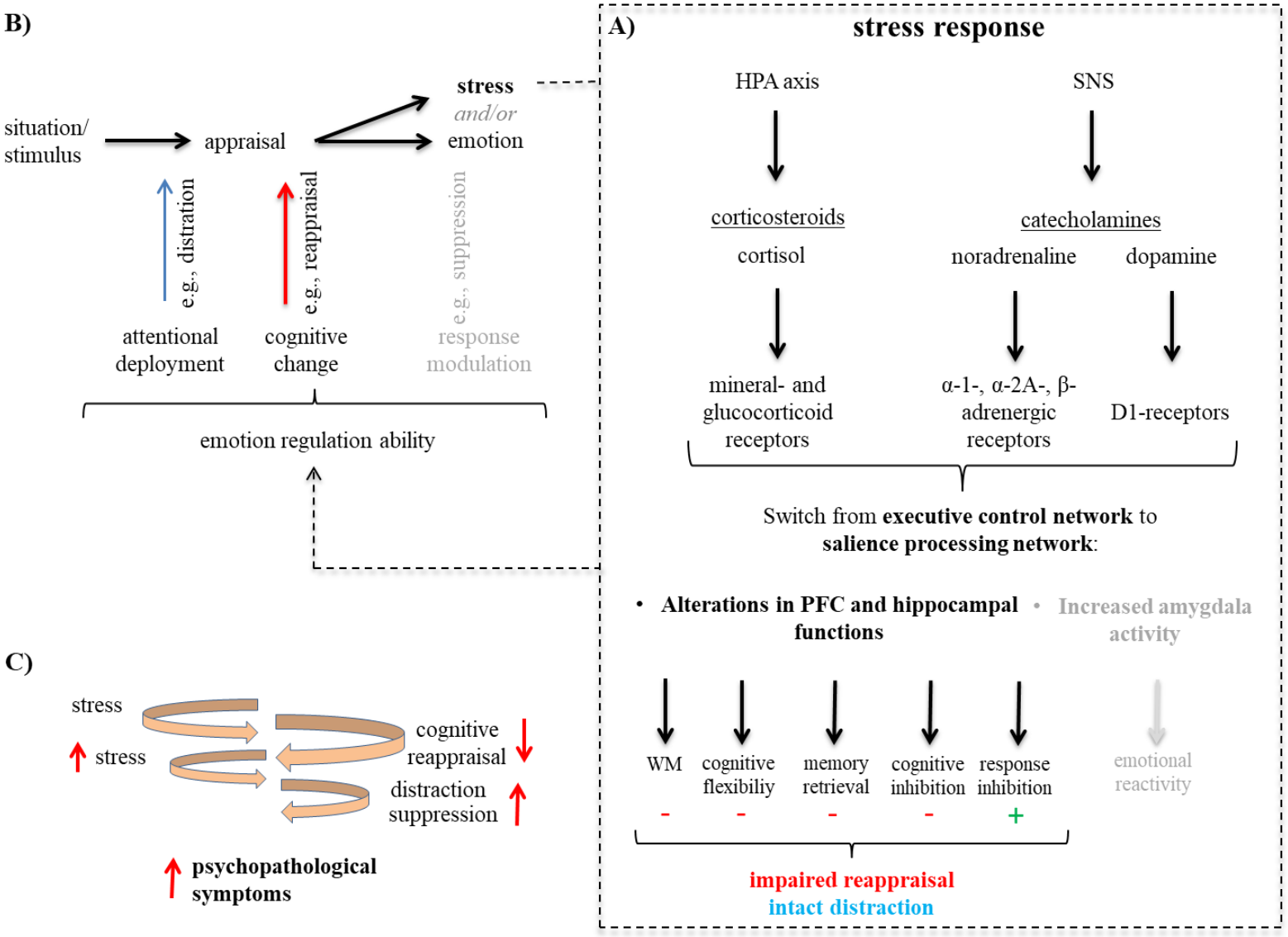

Figure 11. Synthesis model; **A)** Schematic representation of stress-related effects on cognitive reappraisal and distraction (cf. Section 5.3.1.); **B)** Assumption of a negative interaction of ER impairment and stress coping (cf. Section 5.3.2.); **C)** Assumption of the impact of a prolonged reciprocal interaction of stress and ER on the development on psychopathological symptoms and mental disorders (cf. Section 5.3.2.)

5.3.1. Stress-related effects on reappraisal and distraction (Figure 13a)

As illustrated in Figure 13a, a stress response is characterized by the activation of the HPA axis and the SNS. As a consequence, corticosteroids (mainly cortisol) and catecholamines (noradrenaline and dopamine) are released (e.g., Tsigos & Chrousos, 2002). Each of the hormones or neurotransmitters binds to specific receptor types in limbic structures (e.g., amygdala, hippocampus) and the PFC. Particularly, cortisol binds to mineral- and glucocorticoid receptors, noradrenaline binds to α-1-, α-2A-, β-adrenergic receptors and dopamine to D1-receptors (e.g., Arnsten, 2009; Berridge & Arnsten, 2015). Thus, separately and/or synergistically (e.g., Joëls & Baram, 2009), the neuroendocrine and neurochemical changes shift brain function from an executive control network to a salience processing network (Hermans et al., 2014; van Oort et al., 2017; Young et al., 2017). As a consequence, higher cognitive processes are limited in order to provide neural resources that promote adaptive behavior regarding survival. In this regard, recent studies revealed that acute stress exposure impairs WM, cognitive flexibility, cognitive inhibition (Shields, 2016), as well as declarative memory (Lupien et al., 2007) while enhancing response inhibition (Shields, 2016). The higher cognitive functions form fundamental and indispensable processes for effective ER, such as reappraisal and distraction (Compas, 2006; Gyurak et al., 2011; Hofmann et al., 2012; Ochsner et al., 2012; Zelazo & Carlson, 2012). The involvement of higher executive functioning differs between the ER strategies. For instance, reappraisal, as compared to distraction, is related to greater cognitive costs and to demand greater regulation effort (Shafir et al., 2015, 2016; Sheppes et al., 2009; Sheppes & Meiran, 2008) and relies on a complex interplay of WM, declarative memory, inhibitory control, and cognitive flexibility (Compas, 2006; Hofmann et al., 2012; Ochsner et al., 2012). As a consequence, acute stress exposure probably leads mainly to impairment of strategies that are substantially based on more complex cognitive functions, such as reappraisal (Raio et al., 2013; Study 1; Study 2), while less demanding strategies (e.g., distraction) may remain unaffected (Study 2).

Whether an increase in emotional reactivity due to increased amygdala activation also affects the processes of reappraisal or distraction is less clear. While some findings indicate a stress-related increase in emotional reactivity (Alomari et al., 2015; van Marle et al., 2009; Weymar et al., 2012), studies in the context of stress and ER do not reveal an increase in emotional reactivity (Kinner et al., 2014; Langer et al., 2020; Shermohammed et al., 2017). Thus, based on the current state of research, stress-related emotional reactivity alterations seem to have no substantial effect on ER.

5.3.2. Reciprocal interaction of ER impairment and stress (Figure 13b, c)

This section mainly aims to provide future research perspectives regarding potential interactions of ER failure, psychopathology, and stress exposure. In the short term, stress promotes adaptation to challenges of daily life ('allostasis'). However, in case of chronic stress in form of an 'allostatic load' (e.g., financial worries, relationship problems, increasing professional requirements, difficulties in combining work and care activities), stress can cause 'wear and tear' of the body and brain (McEwen, 2004), leading to the development of psychopathological symptoms up to mental disorders such as depression or anxiety disorders (Caparros-Gonzalez et al., 2017; March-Llanes et al., 2017). In this regard, the model aims to provide a potential mediating variable by hypothesizing that the individual's ability to cope with a lasting stressor is impaired as fewer capabilities and resources remain to successfully regulate emotions via cognitive reappraisal. In particular, because of the reduced cognitive capacities due to stress exposure, an individual may tend to use less efficient and less demanding strategies, such as suppression or distraction (e.g., Sheppes et al., 2011), while more effective and long-lasting functional strategies (e.g., reappraisal) are impaired (Raio et al., 2013; Study 1; Study 2). As a result, stress-related impairments of reappraisal may generally result in a decreased ability to regulate further emotions and stressors (Figure 1b). The model claims that this effect may particularly play a role in face of prolonged and multiple stressors as these processes could facilitate negative reciprocal processes between stress and ER in form of a downward spiral (Figure 1c). In a long term, this effect may lead to a cascade of reciprocal interactions in the form of the continuous use of dysfunctional and less effective ER strategies, leading to the development of psychopathological symptoms. In this regard, previous studies already showed that cognitive reappraisal can positively modify stress experience (Jamieson et al., 2012, 2013, 2016; Lewis et al., 2018), while other strategies (e.g., suppression, avoidance, rumination) lead to enhanced stress parameters and unchanged or even increased negative emotional responses (Butler et al., 2006; Gross, 1998; Gross & Levenson, 1993, 1997; Moore et al., 2008; Ruscio et al., 2015). Moreover, Kross and Ayduk (2008) found that distraction can decrease depressive mood immediately after emotion induction. However, in follow-up sessions, one and seven days after emotion induction, the levels of self-reported depressed mood increased significantly compared to other strategies. Contrary, reappraisal led to enduring positive effects on subjective and neural response that causes lasting changes in neural representations (Denny et al., 2015; MacNamara et al., 2010; Morawetz et al., 2017). The author expects that these potential reciprocal effects may result in a high allostatic load, increasing the individual's vulnerability for the development of mental disorders.

In line with this, previous studies revealed a relation between (chronic) stress and mental disorders such as depression, anxiety disorders, or substance abuse (Bradford et al., 2015; Brady & Sinha, 2005; Herman, 2012; Kendler et al., 1999; Marin et al., 2011; Pine et al., 2002). Moreover, in clinical psychology and psychotherapy, the common diathesis-stress model already emphasizes the triggering factor of stress (and especially the accumulation of multiple stressors) on a variety of mental disorders (cf. Ingram & Luxton, 2005), while in addition more than 75% of diagnostic categories of the DSM IV are characterized by emotion dysregulation or problems with ER (Barlow, 2000). In this regard, the detrimental effects of stress on cognitive reappraisal may represent an important mediating variable that fills the gap between stress exposure and the development of mental disorders and complements current diathesis-stress models of mental disorders.

5.3.3. Limitations of the model

As described above, the synthesis model is largely simplified and mainly aims to stimulate future research by demonstrating potential relationships between stress exposure, ER failure, and psychopathological symptoms. Although the concepts are based on previous findings, more research is necessary in order to confirm the mainly hypothetical assumptions. The studies regarding the influence of acute stress exposure on ER are still in early stages and have revealed quite heterogeneous results (cf. Sections 5.2.). Regarding this, a variety of potential mediating variables may make an impact that is not additionally considered in the model, such as time-dependent effects of stress on ER, the potential role of stimuli intensity, or the U-shaped dose-response curve between corticosteroids, catecholamines, and executive functions (cf. Section 5.2.1.). Moreover, beyond the described mechanisms, a number of further stress-related variables possibly influence the development of psychopathological symptoms and mental disorders, such as genetic disposition or other psychological variables (e.g., self-efficacy beliefs). In this regard, the model rather claims that the impairment of reappraisal represents one mediating variable of many, demonstrating how stress exposure may lead to the development of mental disorders and psychopathological symptoms.

In this regard, it is important to pass from hypothetical frameworks to more concrete concepts. Therefore, future research is necessary in order to close the gap between chronic stress, ER failure, and psychopathology. Particularly the systematic analysis and repetitive assessment of ER strategies and ER choice in longitudinal studies in high-risk populations (e.g., foreign assignment of soldiers, firefighters) would be most appropriate for investigation, as they are frequently exposed to stressful situations. An additional assessment

of cortisol and catecholamines could further allow the analysis of potential associations of detrimental effects of prolonged stress exposure and changes in ER. Moreover, in order to prove the assumptions in a more naturalistic setting, future studies could apply ambulatory assessments measuring the habitual use of ER strategies and stress experience in daily life. This could be of particular importance in clinical populations (e.g., depression, borderline personality disorder) in order to test potential interactions of stress experience and ER failure on the development of psychopathological symptoms or even the recurrence of mental disorders.

5.4. General limitations and perspectives

The results of the present thesis need to be interpreted in the scope of some limitations. First, there are limitations regarding the stress protocols in both studies. Study 1 revealed an increase in cortisol concentration in the stress group compared to the control group already in the early stress phase immediately after stressor-offset, while no increase in α-amylase was observed at all. A possible explanation for this early cortisol responsiveness could be that anticipation processes at the beginning of the TSST had already provoked a substantial stress response early on. As a result, the HPA axis had already been activated shortly after stressor-offset. Thus, in contrast to our expectations, the methodological approach of a division into an early and late post-stress phase did not properly differentiate between a predominant activation of the SNS and the HPA axis.

Moreover, Study 2 revealed only a temporally significant increase in salivary cortisol. One potential cause might have been an insufficient sample selection with respect to specific characteristics (e.g., sex ratio), which could have reduced salivary cortisol level. Specifically, most of the participants were female, which might have been a confounding factor, as young men generally show higher cortisol responses compared to women after exposure to real-life stress or controlled laboratory tasks (Kudielka & Kirschbaum, 2005), while women in the follicular and luteal phase of the menstrual cycle, and women using oral contraceptives show significant lower salivary cortisol response (Kirschbaum et al., 1999). Moreover, participants indicated a high face validity of the stress protocol in follow-up interviews, which possibly led to a decreased perception of a social evaluative threat, which is a substantial determinant in the development of a stress response (Dickerson & Kemeny, 2002; Mason, 1968). Future studies need to take these confounding variables into consideration by an optimized standardization of the sample with respect to sex ratio, hormonal contraceptives, menstrual cycle phase, and BMI.

Another limitation is that both studies focused on aspects of explicit ER, whereas

research increasingly emphasizes the role of implicit ER (Gyurak et al., 2011; Koole et al., 2015; Van Dongen et al., 2016). It has been argued that implicit ER as a form of automatic processing does not require additional cognitive resources (Mauss et al., 2007; Williams et al., 2009). Therefore, it can be assumed that implicit ER may be preserved under acute stress exposure, as no (additional) executive functions are required. Hence, future studies could focus on the impact of stress exposure on implicit ER strategies. For instance, extensive training programs and practice of specific ER strategies (e.g., reappraisal) could lead to more automated processing, probably resulting in higher stress-resilience of the strategy. As the recruitment of ER strategies becomes more habitual, less top-down cognitive control and executive functioning are required that are compromised by corticosteroids and catecholamines.

Further, the present studies investigated ER under acute laboratory-induced stress, and thus, it remains unclear whether the results can be transferred to real-life situations. Future studies could address this issue by using techniques of ambulatory assessment (e.g., Trull & Ebner-Priemer, 2013), especially in high-risk populations (e.g., soldiers, firefighters, policemen). Moreover, the present studies focused on the ER strategies of reappraisal and/or distraction. Future studies could compare further strategies, which require less top-down cognitive control (e.g., acceptance) in order to identify more stress-resilient strategies.

At last, the neuroexperimental setup used in the present studies does not allow conclusions about the neural correlates of the reported effects. Although a decreased PFC functioning with a concurrent increased amygdala activity was expected, these assumptions can only remain hypothetical on the basis of the present studies. Therefore, future studies need to implement imaging methods such as fMRI or PET in order to gain further insight. Particularly, the emphasis should lie on the alteration of neural connectivity patterns (e.g., executive control network, salience processing network) during and after acute stress exposure. Neuroimaging approaches could provide clarification with respect to timing effects of stress on a reappraisal of Study 1, or rather, provide further information with respect to potentially divergent neural activation patterns of reappraisal and distraction under acute stress.

5.5. General conclusion

The findings of the present thesis reveal evidence for the detrimental effects of acute stress exposure on cognitive reappraisal while indicating that distraction may represent a more stress-resilient ER strategy. The effects were probably determined by substantial alterations of corticosteroids and catecholamines on the PFC-related executive functions mediating ER. The negative effects of stress exposure on reappraisal may reflect one potential mediating

variable in the relationship between stress exposure and the development of psychopathological symptoms and mental disorders. Nevertheless, there is still a great deal of research to be done. In this regard, the present findings provide a wide range of new research perspectives in order to understand the relationship and underlying mechanisms between stress, ER failure, and psychopathology.

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Appendices

Study 1:

Manipulation Check: Emotion induction and emotion regulation effects

Hypothesis 1: Stress-related increase of emotional reactivity

1) Subjective emotional state ratings (SAM):

2x2x2 rmANOVA: withing subject-factors 'valence' (negative, neutral) and 'stress phase' (early, late); between-subject factor 'group' (stress, control)

	df	MS	F	*P* value	η^2p
valence	1	286.86	252.50	0.00	0.84
valence x group	1	0.04	0.04	0.84	0.00
error(valence)	48	1.14			
stressphase	1	0.04	0.29	0.59	0.01
stressphase x group	1	0.36	2.49	0.12	0.05
error(stressphase)	48	0.14			
valence x stressphase	1	0.09	0.62	0.44	0.01
valence x stressphase x group	1	0.17	1.20	0.28	0.02
error(valence x stressphase)	48	0.14			
group	1	0.64	0.92	0.34	0.02
error(group)	48	0.69			

2) EMG:

4.1) 2x2x2 rmANOVA: withing subject-factors 'valence' (negative, neutral) and 'stress phase' (early, late); between-subject factor 'group' (stress, control)

	df	MS	F	*P* value	η^2p
valence	1	762.46	38.94	0.00	0.47
valence x group	1	6.52	0.33	0.57	0.01
error(valence)	44	19.58			
stressphase	1	28.35	6.22	0.02	0.12
stressphase x group	1	12.36	2.71	0.11	0.06
error(stressphase)	44	4.56			
valence x stressphase	1	0.72	0.23	0.64	0.01
valence x stressphase x group	1	17.21	5.37	0.03	0.11
error(valence x stressphase)	44	3.21			
group	1	33.03	0.61	0.44	0.01
error(group)	44	53.82			

3) P300:

2x2x2 rmANOVA: withing subject-factors ‘valence’ (negative, neutral) and ‘stress phase’ (early, late); between-subject factor ‘group’ (stress, control)

	df	MS	F	*P* value	η^2p
valence	1	1577.47	169.70	0.00	0.78
valence x group	1	4.75	0.51	0.48	0.01
error(valence)	47	9.30			
stressphase	1	14.67	1.32	0.26	0.03
stressphase x group	1	0.21	0.02	0.89	0.00
error(stressphase)	47	11.15			
valence x stressphase	1	1.86	0.21	0.65	0.00
valence x stressphase x group	1	3.09	0.35	0.56	0.01
error(valence x stressphase)	47	8.80			
group	1	253.90	2.23	0.14	0.05
error(group)	47	113.87			

4) LPP:

4.1.) 2x2x2 rmANOVA: withing subject-factors ‘valence’ (negative, neutral) and ‘stress phase’ (early, late); between-subject factor ‘group’ (stress, control)

	df	MS	F	*P* value	η^2p
valence	1	30348.00	121.38	0.00	0.72
valence x group	1	97.28	0.39	0.54	0.01
error(valence)	47	250.02			
stressphase	1	1523.28	7.64	0.01	0.14
stressphase x group	1	267.33	1.34	0.25	0.03
error(stressphase)	47	199.47			
valence x stressphase	1	41.95	0.33	0.57	0.01
valence x stressphase x group	1	57.70	0.45	0.51	0.01
error(valence x stressphase)	47	129.12			
group	1	397.29	.56	.46	.01
error(group)	47	715.55			

4.2.) post-hoc 2x2 rmANOVAs: within-subject factor ‘valence; between-subject factor ‘group’

a) early stress phase

	df	MS	F	*P* value	η^2p
valence	1	405.05	40.31	0.00	0.48
valence x group	1	22.45	2.23	0.14	0.05
error(valence)	44	10.05			
group	1	42.89	1.21	0.28	0.03
error(group)	44	35.33			

b) late stress phase

	df	MS	F	*P* value	η^2p
valence	1	358.13	28.12	0.00	0.39
valence x group	1	1.27	0.10	0.75	0.00
error(valence)	44	12.74			
group	1	2.49	0.11	0.74	<.01
error(group)	44	23.05			

Hypothesis 2: Stress-related impairment of cognitive reappraisal (negative stimuli condition)

1) Subjective emotional state ratings (SAM):

1.1) 2x2x2 rmANOVA: within-subject factors ‘task’ (view, regulate) and ‘stress phase’ (early, late); between-subject factor ‘group’ (stress, control)

	df	MS	F	*P* value	η^2p
task	1	202.15	153.45	0.00	0.76
task x group	1	1.62	1.23	0.27	0.03
error(task)	48	1.32			
stressphase	1	0.28	1.28	0.26	0.03
stressphase x group	1	0.25	1.15	0.29	0.02
error(stressphase)	48	0.22			
task x stressphase	1	0.20	0.49	0.49	0.01
task x stressphase x group	1	2.28	5.62	0.02	0.11
error(task x stressphase)	48	0.41			
group	1	3.46	1.20	0.28	0.02
error	48	2.89			

1.2.) post-hoc 2x2 rmANOVAs: within-subject factor ‘task’; between-subject factor ‘group’

a) early stress phase

	df	MS	F	*P* value	η^2p
task	1	94.86	122.35	0.00	0.72
task x group	1	0.03	0.04	0.85	0.00
error(task)	48	0.78			
group	1	0.92	0.59	0.45	0.01
error(group)	48	1.57			

b) late stress phase

	df	MS	F	*P* value	η^2p
task	1	107.49	113.34	0.00	0.70
task x group	1	3.87	4.08	0.05	0.08
error(task)	48	0.95			
group	1	2.79	1.82	0.18	0.04
error(group)	48	1.54			

2) LPP

2.1.) 2x2x2 rmANOVA: within-subject factors ‘task’ (view, regulate) and ‘stress phase’ (early, late); between-subject factor ‘group’ (stress, control)

	df	MS	F	*P* value	η^2p
task	1	2075.83	15.92	0.00	0.25
task x group	1	73.51	0.56	0.46	0.01
error(task)	47	130.40			
stressphase	1	1298.47	6.46	0.01	0.12
stressphase x group	1	1269.71	6.32	0.02	0.12
error(stressphase)	47	200.99			
task x stressphase	1	89.72	0.84	0.37	0.02
task x stressphase x group	1	722.47	6.72	0.01	0.13
error(task x stressphase)	47	107.49			
group	1	347.55	0.32	0.57	0.01
error	47	1084.55			

2.2.) post-hoc 2x2 rmANOVAs: within-subject factor ‘task’; between-subject factor ‘group’

a) early stress phase

	df	MS	F	*P* value	η^2p
task	1	651.22	5.49	0.02	0.11
task x group	1	167.54	1.41	0.24	0.03
error(task)	47	118.53			
group	1	144.34	0.24	0.63	0.01
error(group)	47	601.12			

b) late stress phase

	df	MS	F	*P* value	η^2p
task	1	1514.33	12.69	0.00	0.21
task x group	1	628.44	5.27	0.03	0.10
error(task)	47	119.36			
group	1	1472.92	2.15	0.15	0.04
error(group)	47	684.43			

3) EMG

3.1) 2x2x2 rmANOVA: within-subject factors ‘task’ (view, regulate) and ‘stress phase’ (early, late); between-subject factor ‘group’ (stress, control)

	df	MS	F	*P* value	η^2p
task	1	151.00	12.85	0.00	0.23
task x group	1	45.00	3.83	0.06	0.08
error(task)	44	11.75			
stressphase	1	16.92	2.97	0.09	0.06
stressphase x group	1	8.91	1.56	0.22	0.03
error(stressphase)	44	5.70			
task x stressphase	1	4.25	1.09	0.30	0.02
task x stressphase x group	1	21.89	5.64	0.02	0.11
error(task x stressphase)	44	3.88			
group	1	225.22	2.41	0.13	0.05
error(group)	44	93.62			

3.2.) post-hoc 2x2 rmANOVAs: within-subject factor ‘task’; between-subject factor ‘group’

a) early stress phase

	df	MS	F	*P* value	η^2p
task	1	102.94	13.43	0.00	0.23
task x group	1	2.06	0.27	0.61	0.01
error(task)	44	7.66			
group	1	161.87	2.73	0.11	0.06
error(group)	44	59.37			

b) late stress phase

	df	MS	F	*P* value	η^2p
task	1	52.30	6.56	0.01	0.13
task x group	1	64.83	8.13	0.01	0.16
error(task)	44	7.97			
group	1	72.27	1.81	0.19	0.04
error(group)	44	39.95			

Erklärung

gemäß § 6 Absatz 2 g) und gemäß § 6 Absatz 2 h) der Promotionsordnung der Fachbereiche 02, 05, 06, 07, 09 und 10 vom 04. April 2016

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Hiermit erkläre ich, dass ich die eingereichte Dissertation selbständig, ohne fremde Hilfe verfasst und mit keinen anderen als den darin angegebenen Hilfsmitteln angefertigt habe, dass die wörtlichen oder dem Inhalt nach aus fremden Arbeiten entnommenen Stellen, Zeichnungen, Skizzen, bildlichen Darstellungen und dergleichen als solche genau kenntlich gemacht sind.

Von der Ordnung zur Sicherung guter wissenschaftlicher Praxis in Forschung und Lehre und zum Verfahren zum Umgang mit wissenschaftlichem Fehlverhalten habe ich Kenntnis genommen.

Darüber hinaus erkläre ich, dass ich keine Hilfe von kommerziellen Promotionsberatern in Anspruch genommen habe.

Berlin, den 15.02.2021 _____

(Unterschrift Jérôme Rimpel)

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