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# Cognitive emotion regulation withstands the stress test: An fMRI study on the effect of acute stress on distraction and reappraisal

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ABSTRACT

Cognitive emotion regulation is a key mechanism for the maintenance of mental health, but may fail, when individuals are exposed to acute stress. To date, it is not well understood whether and to what extent acute stress effects contribute to impairments in emotion regulation capacities as the sparse existing studies have yielded heterogeneous results, indicating that stress timing might be a crucial factor.

In the present study, 81 healthy participants underwent either an acute stress task (Scan*STRESS*-C; $n = 40$) or a control condition ($n = 41$) while lying in the MRI scanner. In the subsequent Cognitive Emotion Regulation Task (CERT), participants were confronted with neutral or negative pictures and instructed to either view them, or regulate their upcoming emotions using either distraction or reappraisal. Subjective ratings of affective state as well as functional brain imaging data served to indicate emotion regulation.

The results showed a successful stress manipulation as indicated by group differences in subjective wellbeing, saliva cortisol concentrations, heart rate, and functional brain activity in regions implicated in stress processing. With respect to emotion regulation, CERT data revealed a significant regulation effect at the neural and behavioral level (less negative emotional ratings after reappraisal and distraction trials compared to view trials) in both groups. However, no significant group differences were observed, neither in BOLD responses to the CERT, nor in behavioral ratings.

Contrary to previous studies, our study did not reveal further evidence of stress-related effects on emotion regulation, potentially being related to differences between studies in experimental setting, timing, and procedures. This study therefore underlines the need of future studies that disentangle the complex interplay of stress and emotion regulation and identify different factors influencing their bidirectional relationship.

## 1. Introduction

The ability to deliberately regulate our emotions is of crucial importance to adequate psychosocial functioning and the maintenance of mental health, especially when facing acute stressors in life (McRae and Gross, 2020). However, little is known about how emotion regulation abilities change in acute stress situations, where these abilities are probably needed the most.

In general, cognitive emotion regulation (ER) constitutes an effective way to cope with emotions that are either too intense or poorly matched to situational demands. In the last decades, a growing body of research identified and investigated different strategies of ER ranging from attentional deployment to cognitive change (Gross, 1998; Webb et al., 2012). Attentional deployment involves the redirection of attention away from emotion-triggering information (*distraction*). Cognitive change incorporates the *reappraisal* of a given stimulus or situation with the aim to change its emotional impact. Both strategies proved effective in altering emotions on multiple response levels: self-reported affective state (Song et al., 2019; Webb et al., 2012; Wu et al., 2019), peripheral physiological markers (Denson et al., 2011; Ray et al., 2010; Schönfelder et al., 2014), and neural measures of emotions (Kanske et al., 2011; Morawetz et al., 2017; Ochsner et al., 2012; Shahane et al., 2019). In general, successful ER has often been linked to long-term mental health outcomes (Aldao et al., 2010; Boyes et al., 2016; Cludius et al., 2020).

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Reversely, a deficit in cognitive ER is common to various mental disorders, i.e. anxiety disorder, depression, and borderline personality disorder (Berking and Wupperman, 2012; Joormann and Gotlib, 2010; Kanske et al., 2012, 2015) and is often the subject of cognitive behavioral therapy. Importantly, the cognitive regulation of emotions can be considered a complex interplay of multiple higher-order cognitive functions, such as attention, cognitive flexibility, and working memory (Hofmann et al., 2012; Ochsner et al., 2012; Papousek et al., 2017). Results from previous imaging studies indicate that ER relies heavily on prefrontal functioning, recruiting a network of ventrolateral (vlPCF) and dorsolateral (dlPFC) prefrontal and parietal regions usually implicated in cognitive control processes (Buhle et al., 2014). Connectivity studies suggest that these prefrontal regions exert a top-down regulation on limbic structures, i.e. the amygdala, thereby contributing decisively to the regulation of emotional responses (Buhle et al., 2014; Kanske et al., 2011).

Given the crucial role of prefrontal brain structures in cognitive ER, the effective implementation of the respective ER strategies may well be challenged in the face of acute stress as the secretion of stress hormones leads to activation changes in cortical and subcortical brain structures (Arnsten, 2009). More precisely, acute stress leads to an immediate increase in (nor)adrenalin triggered by the sympathetic nervous system (SNS), followed by a slower increase in cortisol, as an end-product of the multistage hypothalamus-pituitary-adrenal (HPA) cascade (De Kloet et al., 2005). These neuroendocrine interactions contribute to a systematic re-allocation of cognitive resources in the face of acute stress (Hermans et al., 2014): thus, activity increases in structures of the salience network, i.e. the anterior insula and the dorso-anterior cingulate cortex (dACC), to enhance alertness and enable the organism to react rapidly and adequately to a changing environment (Seeley et al., 2007). In parallel, mediated by the neuroendocrine substrates, acute stress is usually associated with diminished activity in higher-order prefrontal structures (Arnsten, 2009), possibly limiting higher-order cognitive functioning. In line with this, acute stress was associated with a shift in brain activation towards processing of emotionally significant stimuli at the cost of working memory performance (Oei et al., 2012). This indicates that a second burden to ER under stress may refer to the stress-related increase of emotional sensitivity and intensity (van Marle et al., 2009; Weymar et al., 2012), which particularly impedes the implementation of higher-order cognitive functions as ER (Murphy and Young, 2018; Shafir et al., 2015; Webb et al., 2012).

In line with these considerations, previous studies investigating the relationship of acute stress exposure and ER reported a stress-related impairment in the cognitive regulation of previously fear-conditioned stimuli (Raio et al., 2013). Zhan et al. (2017) found reappraisal to be less effective in reducing anger in participants that have previously been stressed, compared to a control group. Kinner et al. (2014) explicitly targeted different ER strategies following an acute stress task and reported significant stress-related impairments in distraction, (but not reappraisal), as indicated by higher self-reported arousal after distraction in stressed compared to non-stressed participants. In contrast to these studies indicating detrimental stress effects on cognitive ER, there is recent evidence that ER might actually benefit from stress exposure (Langer et al., 2020), especially when tested about 90min after laboratory stress induction (Langer et al., 2021) or the administration of external cortisol (Jentsch et al., 2019). These latter findings provide first evidence for a delayed cortisol-induced facilitation of cognitive processes in the longer aftermath of stressful events via slow genomic glucocorticoid activity (see Hermans et al., 2014).

Hence, the previous studies on stress and ER show significant inconsistencies in the results, which are further underlined by a recent neuroimaging study (Shermohammed et al., 2017). Here, the authors report no stress effect at all, neither on emotional reactivity, nor on reappraisal success and neither in subjective ratings, nor in brain activity. A possible interpretation of these conflicting results may be related to the experimental *timing* of the studies since stress effects underlie a fine-tuned dynamic interplay of the early SNS and the delayed HPA axis response. Shermohammed and colleagues confronted their participants with interleaved blocks of stress induction (i.e. challenging mental arithmetic) and ER while lying in the magnetic resonance imaging (MRI) scanner. Considering the fine-tuned dynamics of the stress response, this interleaved block design with multiple stress onsets might have resulted in repetitive baseline shifts of the endocrine stress systems, bearing the risk for adaptation or habituation effects. Hence, it may well be that ER was assessed at a point in time when (nor)adrenalin and cortisol did not yet exert their full effects on the brain and the body. To avoid this problem, the present study employed a stress protocol with one distinct stress onset, short stress duration, and a strict separation from the ER task.

Taken together, the heterogeneous findings of previous studies do not provide clear evidence as to what extend and under what circumstances stress affects subsequent ER and if this stress effect differs between ER strategies. These inconsistencies in findings may be related to differences in experimental *timing* and associated dynamics of the neuroendocrine stress response (see also discussion in section 4), further underlying the importance of advanced methodological considerations. To complement and extend previous studies in this field, the aim of the present study was to investigate acute stress effects on ER using fMRI methodology and a between-subject design comparing a stress and a control group. We used the Scan*STRESS*-C for stress induction, which is the compact version of the Scan*STRESS*, an established stress paradigm in fMRI research (Streit et al., 2014) and which has proven effective in eliciting significant multidimensional stress responses in an fMRI setting (Sandner et al., 2020). The Scan*STRESS*-C consists of one control and one stress phase of only 6 min each and thereby provides a short protocol with one distinct stress onset (and offset) to investigate stress effects on subsequent processes, here cognitive ER (for details, see section 2.2). The ER paradigm started 20 min (and lasted until 40 min) after stress onset, when cortisol concentration is usually at its peak (Kirschbaum and Hellhammer, 1989; Kudielka et al., 2009). To assess ER abilities, we used the Cognitive Emotion Regulation Task (CERT; Kanske et al., 2011), in which participants were instructed to view neutral and negative pictures and respond naturally to them, or to *reappraise* the content of these pictures to decrease upcoming negative emotions, or to *distract* themselves by solving a math equation presented on the picture as overlay (see section 2.5 for details). We hypothesized that a significant stress response elicited by the Scan*STRESS*-C would affect subsequent cognitive ER, manifested as group differences in both outcome variables of the CERT, i.e. subjective emotional state ratings as well as brain activity during ER. We expect stress-related impairments in ER as our stress protocol enabled distinct testing of ER at a time-window after stress, when cortisol secretion is suggested to peak. In detail, we expect more negative emotional ratings and less amygdala reduction during ER in the stress compared to the control group. When comparing both ER strategies, we expect distraction to be more impaired by stress than reappraisal in accordance with Kinner et al. (2014).

## 2. Methods

### 2.1. Participants

Eighty-one participants (40 women; 78 right-handed) at the age of 18–42 years ($M = 24.47$, $SD = 4.49$) were recruited for participation via flyer and postings at the university and university medical center Mainz, Germany. Sample size was determined a priori via power analysis using G*Power 3.1 (Faul et al., 2009), based on a small effect size $f = 0.16$ (in accordance with Langer et al., 2020 and Jentsch et al., 2019), an assumed correlation of $r = 0.70$ for repeated measurements, and an alpha error set to 0.05, revealing a minimum sample size of 79 participants in order to achieve a power of $1\text{-}\beta \geq 0.95$ to detect a significant 3-way interaction of task*group*sex. The selected sample size complies with recent guidelines regarding fMRI studies (Yeung, 2018). Subjects

were randomly assigned to either a stress group (SG; $n = 40$) or control group (CG; $n = 41$), which did not differ in age or Body Mass Index (BMI), see Table 1. All participants underwent a telephone screening to preclude acute or chronic diseases, a history of and current mental disorders, past or ongoing psychotherapy treatment, a history of neurological, cardiovascular, or endocrine diseases, use of steroid-based lotions or asthma sprays, and smoking behavior or use of opioids or cannabis. Participants with a BMI ($kg/m^2$) below 18 and over 26 were excluded. To reduce variability in cortisol responses related to hormonal alterations throughout the menstrual cycle phase, the intake of oral contraceptives was an additional and mandatory inclusion criterion for female participants. The study was approved by the local ethics committee of the Psychological Institute of the Johannes-Gutenberg University Mainz according to the declaration of Helsinki. Participants were compensated for their time with 60 Euros or received course credits.

### 2.2. Procedure

The experimental procedure lasted approx. 2.5 h (see Fig. 1A). All sessions took place between 1 p.m. and 5 p.m. to control for diurnal cortisol variations. We provided a cover story informing participant of the alleged study aim (i.e. the investigation of neural activity patterns in performance situations) to ensure the authenticity of the experimental stress paradigm. Participants subsequently completed a training session of the MRI-tasks (Scan*STRESS*-C and CERT). Hereafter, they watched a relaxing movie for approx. 30 min before entering the MR scanner. The MRI session started with a localizer (for details on fMRI acquisition and analysis, see section 2.6), followed by the Scan*STRESS*-C (see section 2.3). After approx. 6 min of anatomical measures, participants started the CERT (see section 2.4), which then took place 20–40 min after stress onset. After participants left the scanner, they were debriefed in detail. During the experimental session, participants indicated the current state of their well-being at four timepoints (see Fig. 1A), using the german mood questionnaire *Mehrdimensionaler Befindlichkeitsfragebogen* (MDBF, see section 2.4). In addition, they provided six saliva samples (S1–S6) using a Salivette® device (Sarstedt AG & Co, Nümbrecht, Germany). In-MR samples were taken by moving the bench outside the scanner only far enough for the experimenter to place a Salivette in the participants' mouth for 2 min, but the bench was never removed from the scanner completely (see Dedovic et al., 2005).

**Table 1**
Group comparisons with means (standard deviations) and statistical parameters for all relevant variables.

| | SG | | CG | | *t* (*df*) | *p* |
|---|---|---|---|---|---|---|
| | *N* | *M (SD)* | *N* | *M (SD)* | | |
| **Sample characteristics** | | | | | | |
| Age | 40 | 23.85 (4.07) | 41 | 25.07 (4.89) | 1.22 (79) | .225 |
| BMI | 40 | 22.30 (1.79) | 40 | 22.08 (2.02) | 0.53 (78) | .599 |
| **Stress reactivity** | | | | | | |
| S1 (−37min) | 38 | 3.61 (3.21) | 36 | 3.20 (1.93) | 0.68 (72) | .500 |
| S2 (−10min) | 38 | 2.85 (1.59) | 36 | 2.89 (1.51) | −0.28 (72) | .782 |
| S3 (+6min) | 38 | 3.67 (2.28) | 36 | 2.86 (1.29) | 1.78 (72) | .078 |
| S4 (+16min) | 38 | 5.12 (4.61) | 36 | 2.74 (1.02) | 3.09 (72) | .004** |
| S5 (+52min) | 38 | 4.68 (4.28) | 36 | 2.70 (1.28) | 2.69 (72) | .009** |
| S6 (+62min) | 38 | 4.38 (5.15) | 36 | 2.58 (1.49) | 2.03 (72) | .044* |
| MDBF 1 (−40min) | 38 | 12.04 (1.58) | 37 | 12.36 (1.50) | 0.74 (73) | .464 |
| MDBF 2 (−10min) | 38 | 11.72 (1.72) | 37 | 11.86 (1.47) | 0.28 (73) | .781 |
| MDBF 3 (+6min) | 38 | 10.56 (2.09) | 37 | 12.04 (1.58) | 3.30 (73) | .001** |
| MDBF 4 (+50min) | 38 | 11.70 (1.90) | 37 | 12.14 (1.47) | 1.45 (73) | .150 |
| HR phase 1 | 30 | 73.38 (15.08) | 36 | 72.09 (14.69) | 0.35 (64) | .727 |
| HR phase 2 | 30 | 81.66 (16.93) | 36 | 70.14 (13.89) | 3.04 (64) | .003** |
| **CERT SAM ratings** | | | | | | |
| View_neutral | 38 | 6.06 (0.78) | 38 | 6.01 (0.76) | −0.29 (74) | .777 |
| Distract_neutral | 38 | 5.75 (0.86) | 38 | 5.80 (0.78) | 0.28 (74) | .782 |
| View_negative | 38 | 3.63 (0.92) | 38 | 3.73 (1.02) | 0.47 (74) | .640 |
| Distract_negative | 38 | 3.79 (0.96) | 38 | 4.08 (1.24) | 1.14 (74) | .260 |
| Reappraise_negative | 38 | 4.70 (0.90) | 38 | 4.74 (1.12) | 0.16 (74) | .877 |

*Note.* CERT = Cognitive Emotion Regulation Questionnaire; CG = Control Group; BMI = Body Mass Index; HR = Heart Rate in bpm; MDBF = German Mood Questionnaire, higher scores indicate higher well-being; S1–S6 = saliva cortisol samples in nmol/l; SAM = Self-Assessment Manikins; SG = Stress Group. $^*p < .05$, $^{**}p < .01$.

### 2.3. Stress paradigm: ScanSTRESS-C

For in-MR stress induction, we used the Scan*STRESS*-C (Sandner et al., 2020), which is the compact version of the Scan*STRESS*, an established stress paradigm in fMRI research (Streit et al., 2014). The paradigm consists of two phases: an initial control phase and a subsequent stress phase of approx. six minutes each. Each phase contains six blocks of task performance lasting 40 s per block, interleaved with 20 s pauses (see Fig. 1B). During the stress blocks, participants had to perform two types of cognitive challenging tasks, mental rotation and arithmetic subtraction, within a given time frame as indicated by a countdown bar. Task speed and difficulty were preprogrammed to adjust to the participant's performance to increase the likelihood of failure. While performing these tasks, participants were shown a live video of a jury (two lab members in white coats), sitting in front of the scanner and observing the participant's performance to further induce social-evaluative stress. In case of slow or incorrect answers, the jury used a red buzzer to give negative feedback in terms of short written instructions (e.g. "Error!"). In between the stress phase, the jury additionally gave standardized verbal feedback via speakers, indicating that the participant's performance so far was below average, and that maximum effort is needed for the sake of good data quality. Thus, the Scan*STRESS*-C contains both, uncontrollable mental challenge as well as social-evaluative elements – a combination which was identified to result in largest neuroendocrine stress responses (Dickerson and Kemeny, 2004) and to appeal to both male and female participants (Stroud et al., 2002). During control blocks, participants performed simple figure- and number-matching tasks in the absence of visual and verbal jury feedback and time-pressure. In this case, the jury in the video stream remained passive, did not look into the camera, and the video picture was overlaid by a grey diagonal cross to signal the absence of active monitoring (see Fig. 1B). While the SG passed through an initial control and a subsequent stress phase as described above, participants of the CG underwent two control phases instead (see Fig. 1A).

### 2.4. Acute stress reactivity measures

Stress responses to the Scan*STRESS*-C, were assessed by (1) subjective well-being ratings at four time points using the german mood questionnaire, *Mehrdimensionaler Befindlichkeitsfragebogen* (MDBF; Steyer et al., 1997). The MDBF consists of 24 adjectives reflecting positive or negative emotional states. The participants' ratings on a five-point Likert scale can be summed up to a total score of subjective well-being where higher scores reflect a more positive emotional state. Sufficient reliability (Cronbach's alpha $\alpha = .80$ to .92) and validity of the

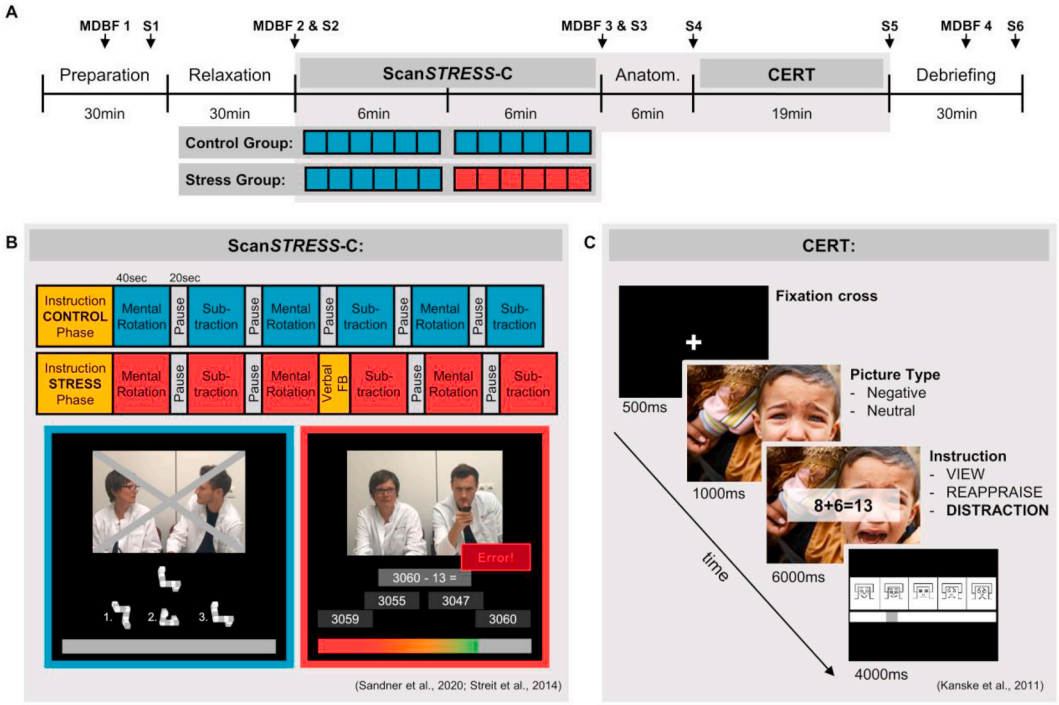


**Fig. 1. Overview of experimental procedure. A.** Schematic of the complete experimental session, including the MRI part (light grey) and the experimental tasks (dark grey), as well as saliva sampling and MDBF rating. B. Details on the Scan*STRESS*-C procedure (Sandner et al., 2020), consisting of a control phase and a stress phase of six blocks each, including easy or difficult tasks of mental rotation and subtraction (see section 2.3). C. Sequence of events in a distraction trial of the CERT paradigm (Kanske et al., 2011, see section 2.5). FB = verbal feedback of the jury in the middle of the stress phase; MDBF = German Mood Questionnaire; S1–S6 = saliva cortisol samples. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

MDBF was confirmed in several studies (Buckert et al., 2014; Klinkenberg et al., 2016; Plessow et al., 2011). In the present study, Cronbach's alpha of .91 indicated excellent internal consistency. (2) We collected six saliva samples to ensure frequent monitoring of the cortisol stress response. All saliva samples were stored at −20 °C and send to the Institute of Biopsychology at the Technical University Dresden, Germany, for analysis. Salivary concentrations were measured using commercially available chemiluminescence immunoassay with high sensitivity (IBL International, Hamburg, Germany). The intra- and interassay coefficients were below 8%. (3) We recorded heart rate (HR) data with an MR-compatible pulse-oximeter with an infrared emitter placed on the left index finger (50 Hz sampling) for the total duration of both the control and the stress phase each. And (4), we analyzed BOLD responses during the Scan*STRESS*-C phases as well as during the ER task (see section 2.6).

### 2.5. Emotion regulation paradigm: CERT

To assess ER in the aftermath of acute stress exposure, we used the Cognitive Emotion Regulation Task (CERT), previously described and validated in several studies (Heissler et al., 2014; Kanske et al, 2011, 2012, 2015). During the CERT (see Fig. 1C), participants are presented with images of either neutral or negative content. All pictures were taken from the EmoPicS stimulus database (Wessa et al., 2010). They were landscape in orientation and matched for content and complexity. After 1 s of stimulus presentation (intended to elicit an initial emotional response to the picture), participants are given one of three different instructions (1 s, transparent overlay): (1) they are instructed to just *view* the image and respond naturally to it; (2) they are asked to indicate as fast as possible, if the given math equation is correct or incorrect via button press (*distraction*); (3) they are asked to *reappraise* the content of the image with the aim to decrease their upcoming negative emotional reaction to it. For the *reappraisal* condition, participants were reminded not to distract themselves by thinking of e.g. the next trip to the supermarket, but to stay in the displayed scene of the given image and find another interpretation for it, e.g. a positive ending of the situation. Each trial ended with a rating of the participants' current emotional state on a 9-point scale using the Self-Assessment Manikins (SAM, Bradley and Lang, 1994) ranging from unpleasant to pleasant with higher values indicating more positive emotions. The inter-trial interval (ITI) was jittered from 3 s to 5 s. In our study, the CERT consisted of 75 trials of approx. 14.5 s each, 15 trials each for the following conditions: view_negative, view_neutral, distract_negative, distract_neutral, and reappraise_negative. In total, the CERT lasted approx. 19 min.

### 2.6. fMRI acquisition and analysis

We acquired imaging data on a 3T scanner (Siemens Trio, Erlangen, Germany) with a 32-channel head coil. The following parameters were used for anatomical images: slice thickness = 1 mm; FoV = 250 mm; voxel size 1mm isotropic; TR = 1900 ms; TE = 2.52 ms; flip angle = 9°. Functional images during the Scan*STRESS*-C and CERT were acquired using identical echo planar imaging (EPI) multiband sequences with the following scanning-parameters: slice thickness = 2.5mm; FoV = 210 mm; voxel size: 2.5mm isotropic; TR = 1000 ms; TE = 29 ms; flip angle = 56°; multiband acceleration factor = 4. Preprocessing and statistical analysis were conducted using Statistical Parametric Mapping (SPM12, http://www.fil.ion.ucl.ac.uk/spm). To account for tissue reaching a steady state of radiofrequency excitation, we discarded the first four images of each sequence. Images were realigned to the first functional image by a 6-parameter rigid body transformation, then co-registered to the anatomical T1 scan, non-linear normalized to the Montreal Neurological Institute (MNI) EPI reference space (voxel size: 2mm isotropic) and smoothed with a 6mm full-width at half-maximum Gaussian filter.

#### 2.6.1. Analysis of ScanSTRESS-C fMRI-data

As a manipulation check for successful stress induction, individual subjects' data of the Scan*STRESS*-C were analyzed within a general linear model framework including one regressor for the 6 min control phase modelling the onsets and duration of the 40 s control blocks, and (in case of SG participants) two stress regressors modelling the 40 s stress blocks, as the stress phase was split into two sequences due to the verbal feedback. The 20 s pauses in between the active control or stress blocks served as an implicit baseline. In addition, we included six motion regressors as covariates of no interest to control for residual motion artefacts after reorientation. First level analysis was performed for the SG and CG separately. We computed contrast images of [stress vs. control] for each participant of the SG or [control1 vs. control2] for the CG to investigate the general effect of task (stress induction). For second level analysis, we used a two-sample *t*-test to examine group differences in the general effect of acute stress, i.e. [stress vs. control] for the SG and [control1 vs. control2] for the CG. Imaging results of the main task effects were corrected via family-wise error (FWE) for multiple comparisons at a significance level of $p$whole_brain<0.05.

#### 2.6.2. Analysis of CERT fMRI-data

To investigate neural correlates of ER, individual subjects' data of the CERT were analyzed within a general linear model framework including the following regressors: view_neutral, view_negative, distract_negative, reappraise_negative, induction phase, rating phase, as well as the six movement parameters from the realignment step. Similar to previous studies on ER (Jentsch et al., 2019; Kanske et al., 2011), first level analyses focused on (A) the emotional reactivity contrast (view_negative vs. view_neutral), (B) the distraction effect (distract_negative vs. view_negative), (C) the reappraisal effect (reappraise_negative vs. view_negative), and (D) direct comparison of ER strategies (distract_negative vs. reappraise_negative). The t-contrast images from the first level analysis were subjected to second-level models where we performed one-sample *t*-test in a first step to determine the main effect of "task" (A-D) on brain activation independent of group. In addition, two-sample *t*-tests (SG vs. CG) were performed to examine "group*task" interaction effects. Finally, a full factorial model was also implemented to investigate potential "sex*task", "sex*group", or "sex*task*group" interaction effects. To correct for multiple comparisons, imaging results were corrected via family-wise error (FWE) at a significance level of $p$whole_brain<.05. Note, that we additionally performed a region of interest (ROI) analyses using MNI coordinates from the meta-analysis by Buhle et al. (2014) to compare ROI activity between groups, but results did not differ from the whole brain analysis (see Supplement 1).

### 2.7. Statistical analyses of affective, endocrine, physiological, and CERT data

Statistical analysis of affective, endocrine, and heart rate data as well as analysis of subjective CERT data was carried out using SPSS 22 (SPSS Inc., Chicago, IL, USA). For all analyses of variance (ANOVAs), which will be described below, statistical effects were evaluated using the Greenhouse–Geisser correction when appropriate. As a manipulation check, we analyzed group differences in acute stress reactivity to the Scan*STRESS*-C: (1) Regarding changes in subjective well-being, we analyzed MDBF data using a three-way mixed ANOVA with "time" (4 levels, within-subject factor), "group" (2 levels), and "sex" (2 levels) as between-subject factors. Note that we included "sex" as a between-subject factor in analyses as some studies report sex differences in acute stress reactivity (Kudielka and Kirschbaum, 2005). (2) Cortisol data was logarithmized to base 10 to reduce typical data skewness. Changes in saliva cortisol within both groups were compared using a three-way mixed ANOVA with "time" (6 levels) as within-subject factor, "group" (2 levels), and "sex" (2 levels) as between-subject factors. (3) For HR data, the average heart rate in beats-per-minute (bpm) for each subject was computed for the control and the stress phase separately. We conducted a three-way mixed ANOVA with "time" (2 levels, within-subject factor), "group" (2 levels) and "sex (2 levels) as between-subject factors.

To test for group differences in ER, we conducted a three-way ANOVA on CERT ratings with "task" (5 levels for the 5 task conditions, i.e. view_negative, view_neutral, distract_negative, distract_neutral, and reappraise_negative) as a within-subject factor and "group" (2 levels) and "sex" (2 levels) as between-subject factor.

## 3. Results

### 3.1. Manipulation check: stress responses to the ScanSTRESS-C

To verify successful stress induction by the Scan*STRESS*-C, we analyzed group differences in temporal fluctuations of (1) subjective well-being, (2) saliva cortisol concentrations, (3) heart rate, and (4) BOLD-responses. Table 1 displays means and standard deviations for each group for all except BOLD-response data. Note that analyses were restricted to $N = 80$ as one participant had to be excluded due to anatomical abnormalities in brain data, see (4).

(1) MDBF data from five participants were missing due to technical problems. The three-way ANOVA on MDBF mean scores resulted in a significant main effect "time", $F(3, 213) = 14.17$, $p < .001$, partial $\eta 2 = 0.17$, as well as a significant interaction effect "time*group", $F(3, 213) = 8.64$, $p < .001$, partial $\eta 2 = 0.10$. Post-hoc *t*-tests revealed a significant difference between the SG and the CG at MDBF3, indicating less well-being in the SG after the Scan*STRESS*-C compared to the CG, see Table 1 and Fig. 2A. In addition, we found a significant "time*sex" interaction, $F(3, 213) = 4.95$, $p = .005$, partial $\eta 2 = 0.07$ (Greenhouse-Geisser corrected, $\varepsilon = 0.80$). Post-hoc analyses revealed a significant difference in MDBF1 scores, indicating higher well-being scores in female ($M = 12.56$, $SD = 1.34$) compared to male ($M = 11.82$, $SD = 1.67$) participants at the beginning of our experiment, $t(75) = 2.16$, $p = .034$. See Supplement 3 for additional sex comparisons in stress reactivity.

(2) For endocrine analyses, we excluded four participants due to missing cortisol values and another three participants with cortisol values > 3 *SD* of the group mean. The three-way ANOVA revealed a significant main effect "time", $F(5, 345) = 3.09$, $p = .010$, partial $\eta 2 = 0.04$, as well as a significant interaction effect "time*group", $F(5, 345) = 2.64$, $p = .023$, partial $\eta 2 = 0.04$ (Greenhouse-Geisser corrected, $\varepsilon = 0.59$). Post-hoc *t*-tests revealed significant differences between the SG and the CG at sampling point S4, S5, and S6, with higher cortisol values in the SG, see Table 1 and Fig. 2B.

(3) For HR analyses, we had to exclude 14 participants due to recording issues, resulting in subsamples of $n_{stress} = 30$ and $n_{control} = 36$. The three-way ANOVA revealed a significant main effect "time", $F(1, 62) = 12.41$, $p = .001$, partial $\eta 2 = 0.17$, as well as a significant "time*group" interaction effect, $F(1, 62) = 31.60$, $p < .001$, partial $\eta 2 = 0.34$. Post-hoc t-tests indicated a significant difference in mean HR between the SG and the CG after stress with higher values in the SG (see Table 1 and Fig. 2C).

(4) For MRI analyses, one participant had to be excluded due to anatomical abnormalities. We analyzed significant group differences in activations and deactivations, contrasting both experimental phases of the Scan*STRESS*-C, i.e. [stress vs. control] for the SG and [control1 vs. control2] for the CG. Compared to the CG, participants of the SG showed significant activity increases in structures of the salience network, i.e. the bilateral anterior insula, the SMA, the dACC, and the brainstem (all $ps < .001$, whole-brain FWE-corrected). Further activations were found in the parietal, and frontal inferior cortex and the cerebellum. Furthermore, we found strong deactivations in the SG compared

to the CG in medial regions of the prefrontal cortex, the posterior cingulate cortex, temporal regions, as well as the posterior insula cortices (see Supplementary Table 2 and Supplementary Fig. 1 for further details on significant local maxima with MNI coordinates and *Z* values).

### 3.2. Emotion regulation (CERT) results

#### 3.2.1. Behavioral CERT results

For the subjective CERT ratings, we had to exclude two participants due to missing data and another two with mean values >3SD of the group mean. The three-way ANOVA resulted in a significant main effect of "task", $F(4, 288) = 157.61$, $p < .001$, partial $\eta 2 = 0.69$ (Greenhouse-Geisser corrected, $\varepsilon = 0.74$). Post-hoc *t*-tests comparing the different trial conditions revealed that ratings to the view_negative trials, $M = 3.67$, $SD = 0.96$, were significantly more negative than to (A) view_neutral trials, $M = 6.10$, $SD = 0.85$, ($t(76) = 18.63$, $p$corr $< .001$), indicating emotional reactivity to negative pictures, (B) to distract_negative trials, $M = 3.98$, $SD = 1.16$, ($t(76) = 2.10$, $p$corr $= .039$), indicating a distraction effect, and (C) to reappraise_negative trials, $M = 4.71$, $SD = 0.85$, ($t(76) = 8.41$, $p$corr $< .001$), indicating a reappraisal effect, see Fig. 3. When directly compared, mean ratings to the reappraisal-negative condition were significantly more positive than those to distraction trials ($t(76) = 5.78$, $p$corr $< .001$). There was no significant interaction effect of "task*group" in the CERT ratings, revealing no differences between experimental groups in emotional reactivity or ER, see Fig. 3.

#### 3.2.2. Imaging CERT results

Analyses of the (A) emotional reactivity contrast (view_negative vs. view_neutral) revealed increased activity in the occipital cortex, the thalamus, and the brainstem. In addition, we found an extensive network of lateral and medial prefrontal, parietal, and lateral temporal regions showing stronger activity during (B) distraction (distract_negative vs. view_negative) and (C) reappraisal (reappraise_negative vs. view_negative) of negative images compared to viewing negative images (see Table 2). When (D) directly comparing both strategies (distract_negative vs. reappraise_negative), we found stronger deactivation during distraction in the anterior cingulate cortex, the hippocampus and the amygdala, see Table 2.

Importantly: no interaction effect with neither "group" nor "sex" were found, neither for (A) emotional reactivity, nor (B) for distraction, (C) reappraisal, or (D) their direct comparison. In fact, the brain activation pattern was very similar across the two groups, see Fig. 4. To avoid over-interpretation of this null-finding, we used Bayesian hypothesis testing as recently suggested by Keysers et al. (2020). We aimed at elaborating if this null-finding indicates just the absence of evidence (i.e. the data are not informative to draw conclusions in favor of $H_1$ or $H_0$) or rather evidence for absence (i.e. the data provides support in favor $H_0$). Results indicate moderate evidence for the absence of an effect, see Supplement 1.

## 4. Discussion

The aim of the present study was to investigate the potentially detrimental effects of acute stress exposure on the cognitive regulation of negative emotions. Although we were successful in inducing stress by means of the in-MR procedure Scan*STRESS*-C, we did not confirm our hypothesis of impaired cognitive ER in the face of acute stress neither with respect to *distraction* nor *reappraisal*. Notwithstanding, we found a significant ER effect for both distraction and reappraisal in the entire sample.

### 4.1. Manipulation check: General stress effects

As the present study was set out to investigate stress-related impairments of cognitive ER, we first checked for significant changes in dependent variables measuring stress effects. As in our validation study (Sandner et al., 2020), the Scan*STRESS*-C successfully induced stress in the SG as compared to the CG, as indicated by more negative affect state, higher saliva cortisol secretion, and increased heart rate after stress in the SG (see Fig. 2). Further, individuals of the SG showed increased BOLD responses in structures of the salience network in stress as compared to control blocks. These acute stress effects found in the SG are in line with previous fMRI studies using the original version of the Scan*STRESS* (Dahm et al., 2017; Streit et al., 2014) as well as other established in-MR stress protocols, e.g. the Montreal Imaging Stress Task (MIST; Dedovic et al., 2005) or the imaging Maastricht Acute Stress Task (iMAST; Quaedflieg et al., 2013). Furthermore, our validation study of the Scan*STRESS*-C (Sandner et al., 2020) yielded cortisol responder rates of almost 74% which stands up to comparison with to the most prominent behavioral (non-imaging) stress protocols, i.e. the Trier Social Stress Test (TSST; Kirschbaum et al., 1993) or the Cold Pressor Task (CPT; Lovallo, 1975).

### 4.2. General emotion regulation effects

General analyses of the CERT data, independent of the experimental group, revealed a significant ER effect in the entire sample: distraction as well as reappraisal of negative pictures resulted in reduced negative emotional state ratings and increased cognitive control network activity compared to passive viewing of negative pictures. Interestingly, when

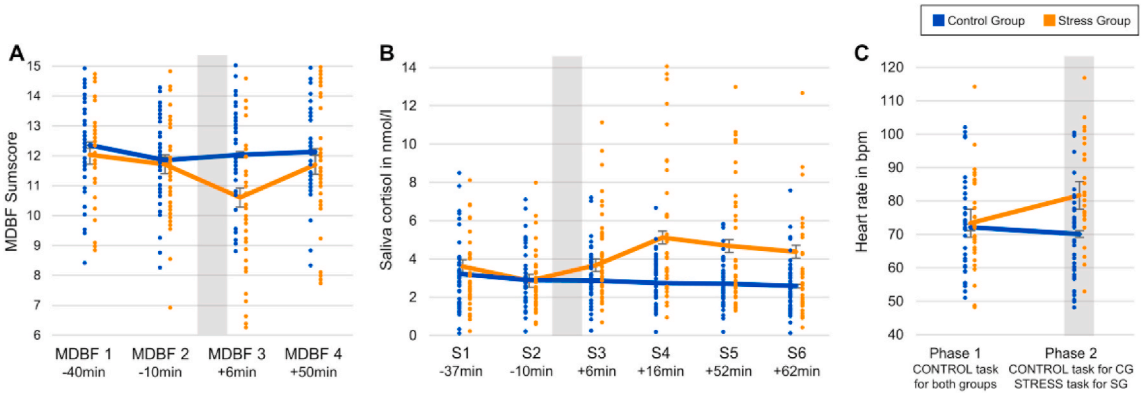


**Fig. 2.** Acute stress reactivity on multiple response levels: (A) Mood ratings and (B) saliva cortisol level at respective time points, as well as (C) heart rate values during the two phases of the Scan*STRESS*-C. Error bars represent *SEM*. Grey bars indicate stress induction. MDBF = German Mood Questionnaire, higher scores indicate higher well-being; S1–S6 = saliva cortisol levels (untransformed). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

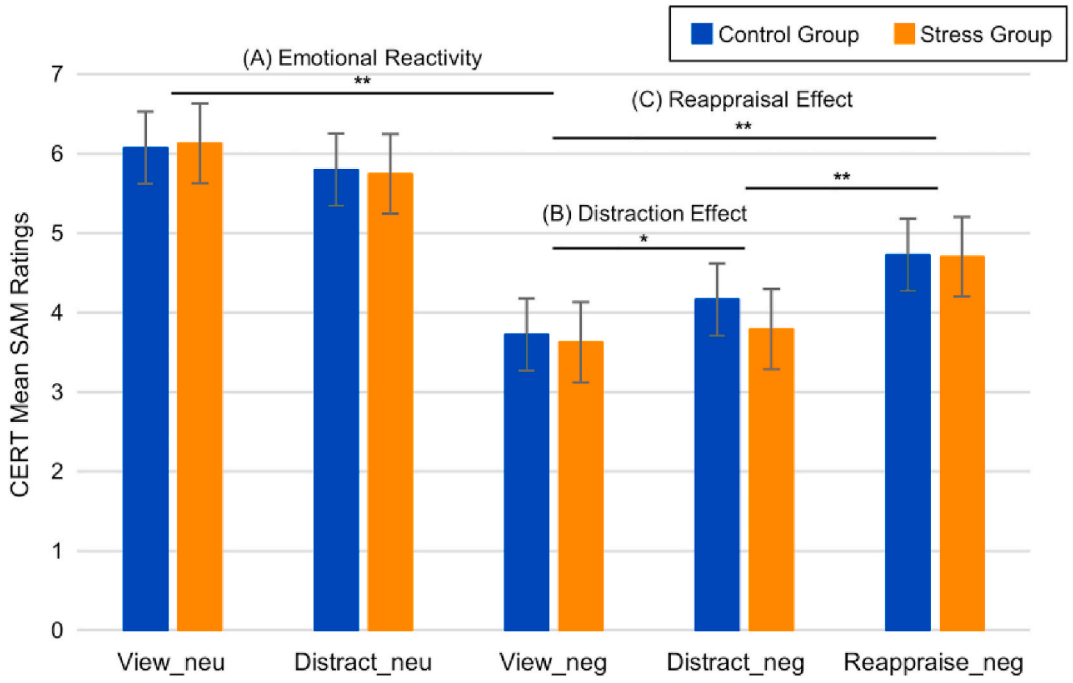


**Fig. 3.** Mean SAM ratings to the CERT task conditions, indicating (A) general emotional reactivity, (B) a significant distraction effect, and (C) a significant reappraisal effect. Groups do not differ in any CERT Score. Error bars represent *SEM*. CERT = Cognitive Emotion Regulation Task; neg = negative; neu = neutral; SAM = Self-Assessment Manikin Scale; *$p < .05$, **$p < .01$. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

contrasted directly, amygdala deactivation was stronger for distraction than reappraisal (see Table 2). This is surprising, as in the subjective ratings, reappraisal resulted in a stronger reduction of negative emotions compared to distraction (see Fig. 3). However, this pattern is in line with various other studies reporting stronger amygdala downregulation for distraction but more pronounced decreases in self-reported negative affect for reappraisal (Jentsch et al., 2019; Kanske et al., 2011; McRae et al., 2010). Since the amygdala is known to be involved in the detection and processing of negative affective stimuli (Phelps and LeDoux, 2005), the authors suggest that reappraisal, in contrast to distraction, leads to a more elaborate processing of the negative content of the picture. Hence, amygdala activity may be maintained during reappraisal, whereas distraction involves a shift of attention away from the emotion triggering information, resulting in stronger reduction of BOLD responses in the amygdala (Jentsch et al., 2019).

### 4.3. *Stress effects on emotion regulation – integrating relevant findings*

Our main hypothesis, i.e. a stress-related impairment in cognitive ER, was not confirmed by the present study. Although we found a significant stress response in the SG on multiple response levels, these stress effects did not affect subjective ratings and brain activation during ER. There were no group differences, neither in emotional reactivity (i.e. view_neutral - view_negative), nor in cognitive ER via distraction (i.e. distract_negative - view_negative), or reappraisal (i.e. reappraise_negative - view_negative). The lack of group differences was present for all dependent variables, (i.e., subjective affect ratings and BOLD responses in the cognitive control and emotion processing networks). Furthermore, Bayesian hypotheses testing revealed moderate evidence for the *absence* of a significant group difference in most ER ROIs, see Supplement 1.

This is surprising since Kinner et al. (2014) reported a significant stress-related impairment at least in distraction. Furthermore, acute stress has been shown to impair anger regulation (Zhan et al., 2017) as well as to undermine an ER training of previously fear conditioned stimuli (Raio et al., 2013). Our results are in line with Shermohammed et al. (2017), the only neuroimaging study investigating the effects of psychosocial stress on ER so far. This study also failed to find a stress effect on emotional reactivity or ER in an experimental set-up where the ER task was interleaved with stressful mental calculation. In contrast to their study design, our stress task provided one distinct stress onset and offset with a cognitive ER task following 20–40min after stressor onset, i.e. when cortisol secretion is suggested to peak. Yet, despite these differences in study design, our study also failed to show stress-related impairments in ER on a subjective as well as neural level. Given that both studies used fMRI methodology, we point out here that the narrow and noisy MRI environment may constitute a constant challenge that possibly interferes with both, the stress induction effects as well as its interaction with ER processes, limiting explanatory power (see limitations). Interestingly, a recent line of studies provides first evidence for a delayed cortisol-induced improvement in ER outcomes when tested 90min after either laboratory stress induction (Langer et al., 2021), or after administration of 30mg external cortisol (Jentsch et al., 2019). Taken together, current literature on stress and ER is characterized by inconsistencies in study results, ranging from ER impairments (Kinner et al., 2014; Raio et al., 2013; Zhan et al., 2017), to no stress effect on ER (this study; Shermohammed et al., 2017), to even ER improvements after stress (Jentsch et al., 2019; Langer et al., 2020, 2021). In the following, we discuss possible explanations for these inconsistencies.

### 4.4. *Relative predominance of stress systems*

When disentangling stress effects on ER, a closer look at the neuroendocrine stress systems was recently suggested (Langer et al., 2020). It has been argued that the upregulation of the salience network and downregulation of prefrontal brain regions in the face of stress are mainly driven by early SNS activity, while the late (genomic) effects of cortisol rather contribute to the restoration of homeostasis and a normalization of brain network activity (De Kloet et al., 2005; Hermans et al., 2014). Supporting evidence comes from Raio et al. (2013) who reported that the impairment in fear regulation after stress was correlated with $\alpha$-Amylase, a biomarker for SNS activity (Nater and Rohleder,

**Table 2**
MNI coordinates of peak voxels and corresponding T and p_FWE values of activation clusters that show significant activation when contrasting the experimental conditions of the CERT in the whole sample.

| Brain structure | | MNI coordinates | | | Statistical values | | |
|---|---|---|---|---|---|---|---|
| | | *x* | y | z | *K* | Mean *T* | $p_{\_FWE}$ |
| **(A) Emotional Reactivity contrast** | | | | | | | |
| **[view_negative vs. view_neutral]** | | | | | | | |
| Middle occipital gyrus | R | 46 | −74 | 6 | 16,468 | 15.49 | <.001 |
| | L | −34 | −86 | 0 | | | |
| Cerebellum | L | −6 | −76 | −34 | 209 | 8.46 | <.001 |
| Thalamus | R | 22 | −28 | 0 | 46 | 7.65 | <.001 |
| Cerebellum | R | 8 | −74 | −34 | 35 | 7.33 | <.001 |
| Inferior parietal gyrus | R | 32 | −50 | 56 | 131 | 7.27 | <.001 |
| Brainstem | L | −6 | −28 | −6 | 18 | 6.75 | .001 |
| Thalamus | L | −20 | −30 | 2 | 36 | 6.02 | <.001 |
| **[view_neutral vs. view_negative]** | | | | | | | |
| No suprathreshhold clusters | | | | | | | |
| **(B) Distraction contrast** | | | | | | | |
| **[distract_negative vs. view_negative]** | | | | | | | |
| Inferior parietal gyrus | L | −42 | −42 | 50 | 24,768 | 19.91 | <.001 |
| | R | 42 | −40 | 46 | | | |
| Middle frontal gyrus | L | −24 | 2 | 56 | | | |
| Anterior insula cortex | L | −30 | 18 | 6 | | | |
| Supplementary motor area | M | 2 | 12 | 48 | | | |
| Cerebellum | R | 28 | −60 | −26 | 4115 | 18.37 | <.001 |
| Inferior temporal gyrus | L | −52 | −56 | −10 | 682 | 15.28 | <.001 |
| Anterior insula cortex | R | 32 | 22 | 4 | 961 | 14.94 | <.001 |
| Inferior frontal gyrus | R | 44 | 38 | 26 | 1738 | 12.57 | <.001 |
| Cerebellum | L | −30 | −56 | −34 | 1211 | 12.40 | <.001 |
| Middle frontal gyrus orbital | R | 26 | 50 | −12 | 319 | 9.43 | <.001 |
| Inferior temporal gyrus | R | 58 | −50 | −12 | 188 | 8.51 | <.001 |
| Middle frontal gyrus orbital | L | −22 | 42 | −14 | 36 | 7.79 | <.001 |
| Inferior occipital gyrus | L | −24 | −96 | −10 | 122 | 7.44 | <.001 |
| Inferior frontal gyrus orbital | R | 52 | 8 | 22 | 193 | 7.38 | <.001 |
| **[view_negative vs. distract_negative]** | | | | | | | |
| Amygdala | R | 22 | −8 | −14 | 24,946 | 18.45 | <.001 |
| | L | −20 | −12 | −18 | | | |
| Superior occipital gyrus | L | −12 | −98 | 24 | | | |
| Rectus gyrus | L | −2 | 40 | −20 | 5974 | 14.42 | <.001 |
| | R | 4 | 48 | −14 | | | |
| Middle frontal gyrus | L | −4 | 52 | −12 | | | |
| Angular gyrus | L | −52 | −70 | 28 | 1073 | 11.13 | <.001 |
| Inferior frontal gyrus orbital | R | 38 | 34 | −14 | 655 | 10.89 | <.001 |
| | L | −52 | 26 | 4 | 109 | 9.43 | <.001 |
| Cerebellum | R | 30 | −80 | −34 | 229 | 9.18 | <.001 |
| | L | −20 | −82 | −36 | 188 | 6.44 | <.001 |
| **(C) Reappraisal contrast** | | | | | | | |
| **[reappraise_negative vs. view_negative]** | | | | | | | |
| Supplementary motor area | L | −6 | 8 | 66 | 18,825 | 14.07 | <.001 |
| Middle temporal gyrus | L | −52 | −34 | −2 | | | |
| Middle cingulate cortex | L | −4 | 16 | 42 | | | |
| Middle frontal gyrus | L | −40 | 4 | 51 | | | |
| Cerebellum | R | 38 | −60 | −28 | 3361 | 10.97 | <.001 |
| Superior temporal gyrus | R | 50 | 18 | −22 | 3396 | 10.22 | <.001 |
| Orbito frontal gyrus | R | 50 | 34 | −8 | | | |
| Fusiform gyrus | L | −30 | −60 | −10 | 4564 | 9.48 | <.001 |
| Middle occipital gyrus | L | −36 | −88 | 8 | | | |
| Middle temporal gyrus | R | 48 | −34 | −2 | 712 | 9.33 | <.001 |
| Caudate | L | −14 | 4 | 16 | 724 | 8.42 | <.001 |
| Inferior parietal gyrus | R | 54 | −54 | 32 | 483 | 8.13 | <.001 |
| Caudate | R | 16 | 12 | 10 | 502 | 8.01 | <.001 |
| Posterior cingulate cortex | L | −10 | −46 | 34 | 477 | 7.31 | <.001 |
| Precuneus | L | −8 | −54 | 35 | | | |
| Superior occipital gyrus | R | 24 | −74 | 42 | 76 | 6.65 | <.001 |
| Superior parietal gyrus | R | 26 | −60 | 62 | 28 | 6.15 | <.001 |
| **[view_negative vs. reappraise_negative]** | | | | | | | |
| Superior temporal gyrus | L | −48 | −8 | 0 | 50 | 6.92 | <.001 |
| Rolandic operculum | L | −40 | 2 | 14 | 39 | 6.75 | <.001 |
| Posterior insula cortex | L | −38 | −18 | 12 | | | |
| Lingual gyrus | R | 14 | −76 | −2 | 64 | 6.16 | <.001 |
| **(D) Comparing Strategies** | | | | | | | |
| **[distract_negative vs. reappraise_negative]** | | | | | | | |
| Inferior parietal gyrus | L | −40 | −42 | 46 | 20,670 | 19.04 | <.001 |
| | R | 40 | −40 | 44 | | | |
| Cerebellum | R | 26 | −60 | −25 | 1862 | 11.17 | <.001 |
| Inferior temporal gyrus | R | 58 | −50 | −12 | 86 | 9.10 | <.001 |
| Cerebellum | L | −26 | −60 | −30 | 415 | 8.49 | <.001 |
| Inferior frontal gyrus | R | 44 | 38 | 26 | 613 | 7.96 | <.001 |
| Inferior occipital gyrus | L | −22 | −98 | −6 | 26 | 6.46 | <.001 |
| Inferior parietal gyrus | L | −40 | −42 | 46 | 20,670 | 19.04 | <.001 |
| **[reappraise_negative vs distract_negative]** | | | | | | | |
| Anterior cingulate cortex | R | 24 | −8 | −14 | 31,672 | 17.35 | <.001 |
| Hippocampus | L | −20 | −12 | −18 | | | |
| Amygdala | R | 30 | 2 | −19 | | | |
| | L | −23 | −5 | −16 | | | |
| Superior frontal gyrus | L | −8 | 54 | 36 | 9951 | 16.56 | <.001 |
| | R | 2 | 54 | 24 | | | |
| Cerebellum | M | 6 | −52 | −40 | 454 | 12.99 | <.001 |
| | R | 28 | −78 | −32 | 512 | 12.59 | <.001 |
| | L | −24 | −82 | −36 | 446 | 10.52 | <.001 |
| Caudate | L | −14 | 12 | 10 | 245 | 10.11 | <.001 |

*Note.* L = left hemisphere; R = right hemisphere; M = medial; *k* = cluster size in voxels; MNI = Montreal Neurological Institute.

2009), while no correlation was found for cortisol concentrations. Given these fine-tuned and partly opposing effects of the SNS and the HPA axis, the relative predominance of one stress system over the other may distinctively determine the intensity (or presence) and direction of a stress effect on ER. Furthermore, a deeper understanding of the factors that influence this balance of the stress systems may help to understand the inconsistencies in previous results: while *timing* certainly constitutes an influencing factor, with SNS predominance in the acute face of stress and cortisol effects predominating the longer aftermath of a stressor (Hermans et al., 2014), it only partly explains the heterogeneity in previous results. On closer examination, it becomes obvious that study designs also differ in the *type of stressor*: Those studies reporting ER impairments after stress used a physical stressor, i.e. the CPT (Raio et al., 2013), or its social-evaluative version (SECPT; Schwabe et al., 2008;

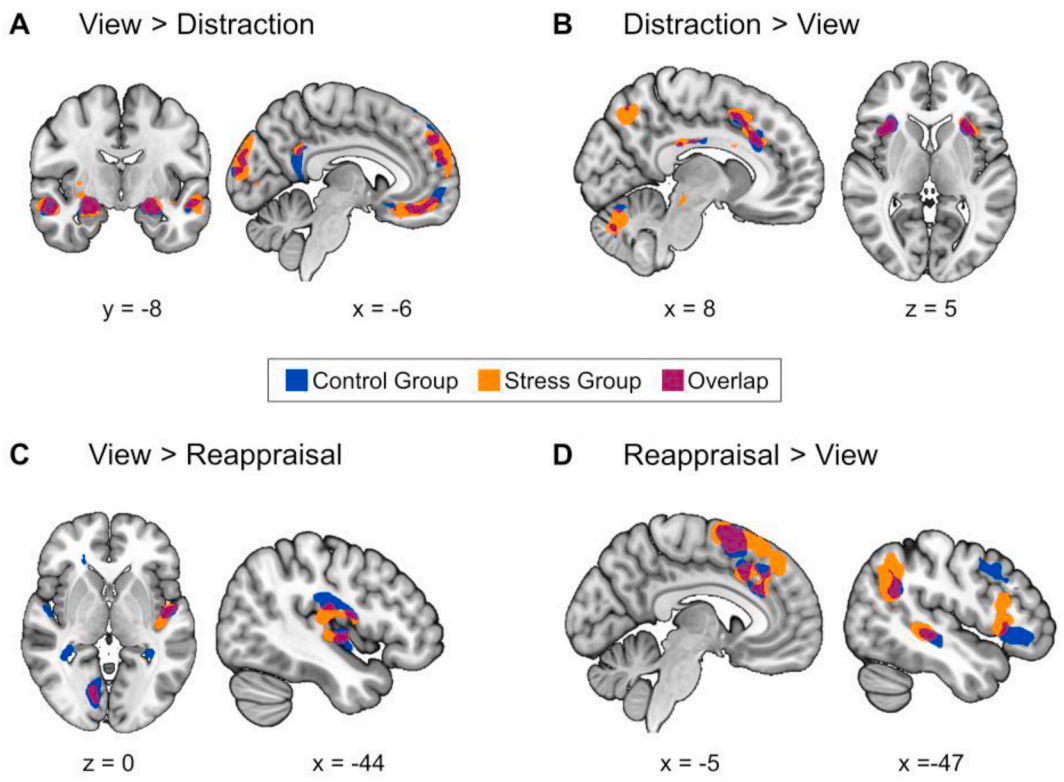


**Fig. 4.** Whole brain fMRI analyses examining activations and deactivations of the distraction contrast (A, B) and the reappraisal contrast (C, D). Images are $p < .05$, whole-brain FWE-corrected, and have been further thresholded at z = 7 (distraction contrast) and z = 4 (reappraisal contrast) for visualization purposes. For graphical display, MRIcroN (https://www.nitrc.org/projects/mricron) was used with the MNI template brain. FWE = family-wise error corrected for multiple comparisons; MNI = Montreal Neurological Institute. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

Kinner et al., 2014; Zhan et al., 2017). Physical stressors resemble a threat to the goal of physical integrity or self-preservation (Dickerson and Kemeny, 2004) which usually implies a rapid and intense activation of the SNS to mobilize energy resources and enable unpremeditated actions to overcome danger and challenges (fight-or-flight). Thus, these physical stressors might have led to a relative predominance of the SNS, involving detriments in prefrontal-based functions, in this case ER. In contrast, the other studies reporting no or an enhancing effect of stress on ER used a psychosocial stressor, such as the TSST (Langer et al., 2020, 2021) or the Scan*STRESS*-C (this study), or a comparable stress protocol (Shermohammed et al., 2017). These psychosocial stressors, while still being effective in eliciting a significant response of both stress systems (Dickerson and Kemeny, 2004), might be less prone to the rapid dynamics of the SNS response compared to intense physical pain, as used in the aforementioned CPT studies. Thus, a relative predominance of the HPA axis over the SNS might explain the missing impairments (Shermohammed et al., 2017, this study) or even improvements (Langer et al., 2020, 2021) in ER after stress. In summary, the relative predominance of one stress system over the other certainly needs further systematic investigation in future studies as it may be of high explanatory power and practical relevance in the context of interventions aimed at improving stress resilience and coping.

### 4.5. Limitations

Beside the rather homogenous study sample of mainly young and healthy students limiting the generalizability of our results, three methodological limitations of the present study should be considered: First, the nature of our between-group study design does limit explanatory power of our results regarding our research questions, because we cannot preclude group differences in baseline ER abilities and habits (see also *Outlook and further directions*). In addition, as we unfortunately did not assess subjective well-being directly before the CERT, reflecting a pre-CERT baseline level (see Fig. 1), we cannot disentangle whether and to what extent the groups differed in their subjective stress level during the actual CERT. This is especially important given the challenging environment of the MRI scanner as well as the more distant participant-jury-interaction, which (as described above) might have interfered with the stress induction as well as its interaction with ER processes. Second, we assessed emotional responses to the CERT pictures only as subjective ratings of valence on a scale from *unhappy* to *happy*. Future studies might include measures of arousal as well as psychophysiological markers, considering that the impairment in distraction after stress reported by Kinner et al. (2014) was only found in arousal ratings, not valence. Third, since the aversive pictures used in the CERT mainly depict scenes of war or crying or injured persons, ecological validity of these stimuli is rather low. This might explain the lack of a stress-related increase in general emotional *reactivity* in our study (see Fig. 3), which in turn may -at least partly-account for the missing stress effect on emotion *regulation* as there is no stress-related increase in emotional responses to regulate. Another indicator of the lacking emotional *reactivity* to the negative pictures is the missing BOLD response in limbic regions when contrasting negative and neutral pictures during the viewing condition (see Table 2). Other studies using the CERT typically report a strong BOLD response of e.g. the amygdala in this contrast (Kanske et al., 2011; Ochsner et al., 2012), indicating the stronger processing of arousing stimuli when viewing negative as compared to neutral pictures. In our study however, the negative and neutral pictures were chosen carefully to minimize differences in complexity and content, (i.e. both mainly depicting human social content). Further, we deliberately chose negative pictures of only moderate intensity to increase the opportunity of generating alternative

interpretations (i.e. reappraisal) of the depicted scenes. Thus, the pictures were quite similar with respect to arousal or threat detection, possibly resulting in comparable limbic responding, emotional *reactivity* and emotion *regulation* possibilities might have been limited. Hence, future studies investigating ER might consider using stimulus material of higher (or at least systematically varying) emotional intensity and higher personal relevance to the participants.

### 4.6. Outlook and further directions

When discussing the present and previous studies, it is important to note that stress and ER are close constructs that are based on a bidirectional relationship: Acute stress might affect ER, but recent research suggests that ER abilities also affect the response to acute stress. Several studies report a main effect of the *habitual use of reappraisal* on acute cortisol reactivity (Raymond et al., 2019), recovery (Lewis et al., 2018), or HPA axis habituation (Roos et al., 2019) to an acute stressor. Moreover, reappraising the acute stressor itself significantly improved cardiovascular recovery while this effect critically depended on the habitual reappraisal use (Jentsch and Wolf, 2020). The habitual use of maladaptive ER strategies was shown to predict increased affective stress responses (Krkovic et al., 2018) and altered corticolimbic recovery from stress (Murray et al., 2021). In addition, stress is known to generally lead to more habit-like behaviour (Schwabe and Wolf, 2009, 2013; Wirz et al., 2018). Hence, within our stress group, participants with high habitual use of reappraisal or distraction may have benefit from the stress induction, because it promoted the use of their habitual ER strategies during the CERT, thereby increasing ER performance after stress. Reversely, for participants who habitually use other ER strategies (e.g. rumination, catastrophizing), these strategies might have interfered with our CERT task when promoted by the stress induction. To further verify these assumptions, future studies would benefit from assessing habitual ER in addition to instructed ER strategies after stress.

Another individual factor possibly affecting the bidirectional relationship of stress and ER might be *sex*. Besides numerous studies observing sex-differences in both, stress reactivity (Kirschbaum et al., 1999; Liu et al., 2017) as well as ER effectivity (McRae et al., 2008) and flexibility (Goubet and Chrysikou, 2019), a very recent study additionally discovered that the effect of stress on ER differed decisively according to differences in sex hormone concentrations: Langer et al. (2020) investigated ER in the face of stress using a similar study design as the present study, with the CERT following approx. 25–50min after stress onset. Interestingly, the authors report an improvement in ER outcomes after stress in only male participants, but no such stress-effect in women, suggesting a complex interplay of sex-specific hormones, (i.e. estrogens, gestagens, and androgenes) and stress-related neuroendocrine activity (for more information see e.g. McEwen et al., 2016; Ter Horst et al., 2012). In the present study however, we did not find sex differences in stress reactivity (see Supplement 3), nor a sex*group interaction effect for the ER conditions. Given the importance of interindividual differences (such as *habitual reappraisal* or *sex*) in the complex bidirectional interaction of stress and ER, future studies might benefit from a within-subject design, testing ER before and after stress to tease out interindividual variance.

## 5. Conclusion

In the present study, we investigated cognitive ER abilities in the face of stress to complement and extend previous studies in this field. Results of our study indicate that there might be no effect of an acute psychosocial laboratory stressor on ER – at least when tested in a between-group design and in this specific time window of 20–40 min after stress onset. The relationship of stress and ER seems rather complex and may be influenced by several co-varying contextual and individual factors. Hence, when investigating ER in the face of stress in future studies, careful methodological considerations of the experimental design, i.e. the timing and characteristics of the paradigms used, seem warranted.

## Declaration of competing interest

None.

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## Appendix A. Supplementary data

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## Author contributions

Magdalena Sandner: Methodology, Software, Formal analysis, Investigation, Writing – original draft, Writing – review & editing, Visualisation. Peter Zeier: Investigation, Writing – review & editing. Giannis Lois: Software, Formal analysis, Writing – review & editing, Visualisation. Michèle Wessa: Conceptualisation, Methodology, Writing – review & editing, Supervision, Project administration, Funding acquisition

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