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The impairing effect of acute stress on suppression-induced forgetting of future fears and its moderation by working memory capacity

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ABSTRACT

Unwanted imaginations of future fears can, to some extent, be avoided. This is achieved by control mechanisms similar to those engaged to suppress and forget unwanted memories. Suppression-induced forgetting relies on the executive control network, whose functioning is impaired after exposure to acute stress. This study investigates whether acute stress affects the ability to intentionally control future fears and, furthermore, whether individual differences in executive control predict a susceptibility to these effects. The study ran over two consecutive days. On day 1, the working memory capacity of one hundred participants was assessed. Thereafter, participants provided descriptions and details of fearful episodes that they imagined might happen in their future. On day 2, participants were exposed to either the stress or no-stress version of the Maastricht Acute Stress Test, after which participants performed the Imagine/No-Imagine task. Here, participants repeatedly imagined some future fears and suppressed imaginings of others. Results demonstrated that, in unstressed participants, suppression successfully induced forgetting of the episodes' details compared to a baseline condition. However, anxiety toward these events did not differ. Acute stress was found to selectively impair suppression-induced forgetting and, further, this effect was moderated by working memory capacity. Specifically, lower working memory predicted a susceptibility to these detrimental effects. These findings provide novel insights into conditions under which our capacity to actively control future fears is reduced, which may have considerable implications for understanding stress-related psychopathologies and symptomatologies characterized by unwanted apprehensive thoughts.

1. Introduction

Imaginations of our future are based on thoughts of past events (Schacter et al., 2017). However, not all imaginations are desirable. To some extent, we have the ability to choose which future thoughts we keep revisiting and which we forget (Benoit et al., 2016). These attempts of intentional control serve as an adaptive emotion regulation strategy, fostering the retrieval of positive experiences and inducing the forgetting of other experiences that pose a threat to our integrity and well-being (e.g., fear-related thoughts; Nørby, 2018). The inability to control fear-related memories plays a key role in the development and maintenance of stress-related psychopathology, observable as intrusive and worrying thoughts in anxiety and mood disorders (Hertel and Gerstle, 2003; Joormann et al., 2005; Mary et al., 2020). Despite these far-reaching implications, the factors that influence our capacity to intentionally control our future fears are largely unknown.

Intentional control of future thoughts or past memories comprises two distinct processes: intentional retrieval (positive control) and suppression (negative control), which subsequently leads to the enhancement or impairment of memory, respectively (Anderson and Green, 2001; for meta-analyses, see Anderson and Huddleston, 2012; Stramaccia et al., 2019). Suppression can be achieved via inhibitory control, an executive control function that can be engaged to stop memory retrieval (Anderson and Huddleston, 2012). Individual differences in executive control, therefore, have been suggested to account for variation in the ability to suppress unwanted memories (Anderson and Green, 2001; Levy and Anderson, 2008). Executive control is also a critical element of working memory, guiding attention to relevant and inhibiting irrelevant information (Kane et al., 2001; Marsh and Hicks, 1998). Importantly, higher working memory capacity has been found to predict an increased ability to intentionally suppress thoughts (Brewin and Beaton, 2002).

The ability to intentionally control future thoughts can be enabled through control mechanisms similar to those engaged when we recall

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our past memories (Benoit et al., 2016). Suppression of future fear imaginings relies on an executive control network, guided by the right dorsolateral prefrontal cortex (dlPFC), which downregulates activity in the ventromedial PFC and hippocampus (Benoit et al., 2016). These regions support the retrieval of past episodes and the simulation of future events (Addis et al., 2007; Benoit and Schacter, 2015; Hassabis et al., 2007; Schacter et al., 2017). Activity in the ventromedial PFC can be more strongly engaged when imaginations are situated in more familiar contexts (Benoit et al., 2014). Familiar contexts increase the vividness of our imaginations (Szpunar and McDermott, 2008), making them harder to forget (Hirst and Phelps, 2016). In turn, stronger downregulation of these regions has been linked to successful suppression and subsequent forgetting (Benoit and Anderson, 2012; Gagnepain et al., 2014).

Acute stress alters, amongst others, functioning of frontal and temporal brain areas implicated in intentional control (Hermans et al., 2014). The dlPFC is a key neural substrate of the executive control network and activity in this region is found to reduce after acute stress exposure (McEwen and Morrison, 2013). In large-scale brain networks, the combined autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA) acute stress response systems prompt the reallocation of resources to the salience network, promoting a hypervigilant state at the expense of the executive control network (Hermans et al., 2011, 2014). In addition, increased levels of cortisol can result in impairments of dlPFC associated higher cognitive functions such as working memory (Oei et al., 2006; Schoofs et al., 2009; for meta-analysis, see Shields et al., 2015). These impairing effects peak during a period in which both the rapidly acting ANS and slower HPA stress response systems are activated (Elzinga and Roelofs, 2005). Furthermore, increased activation of the sympathetic stress response has been related to lower baseline working memory (Hernaus et al., 2018).

Based on these findings, the aim of the current study was two-fold. First, to investigate the influence of acute stress on the suppression of future fears. In the absence of acute stress, suppressing imaginings of future fears causes forgetting of details that are associated with the feared event and, further, attenuates anxiety toward that event (Benoit et al., 2016). In addition to replicating these original findings within the control (unstressed) condition, we hypothesized that acute stress would negatively affect the ability to suppress imaginations of future feared events, preventing suppression-induced forgetting and attenuation of anxiety. Second, to explore whether individual differences in working memory capacity influence the efficacy of suppression after exposure to an acute stressor.

Acute stress was elicited using the 'Maastricht Acute Stress Test' (MAST), a potent and reliable procedure to elicit subjective, autonomic and glucocorticoid stress responses (Quaedflieg et al., 2017; Smeets et al., 2012). To assess the intentional control of fear imaginings, the current study employed an adapted version of the 'Imagine/No-Imagine' task, tailored to the retrieval and suppression of future feared events (Benoit et al., 2016). Working memory capacity was assessed using the digit span task as a measure of executive control (McCabe et al., 2010).

2. Materials and methods

2.1. Participants

The *a-priori* power calculation with G* Power ($\alpha = 0.05$, $1\text{-}\beta = 0.85$; Faul et al., 2007), based on the suppression effect reported in Benoit et al. (2016), indicated a required sample of 90 participants. In total, one hundred healthy participants were recruited via university subject pools and online study advertisement. All participants were aged between 18–35 and were screened for the following inclusion criteria: BMI between 17.5–28; drink less than 10 alcoholic drinks per week; smoke less than 10 cigarettes per week; no use of medication in the previous week; use drugs less than twice per month and have no history of mental illness within the past five years. All females were using hormonal contraception, to control for the known influence on the cortisol stress response (Kirschbaum et al., 1999; Strahler et al., 2017). Eight participants were excluded from the analysis due to missing data as a result of measurement error. The final sample consisted of 92 participants (23 male). All participants provided written informed consent and were reimbursed with University credits or monetary compensation. The test protocols were approved by the ethics committee of the Faculty of Psychology and Neuroscience, Maastricht University.

2.2. Design

Participants were randomly allocated into one of two conditions (stress: n = 50; control: n = 50). The study ran over two consecutive test days for a duration of approximately 1.5 and 2.5 h, respectively. Day 1 testing ran between 08:30am-12:00pm. To avoid fluctuations in the circadian rhythm of cortisol, day 2 testing ran between 12:30 − 18:30pm. Prior to each session, participants were instructed to refrain from eating, smoking, strenuous exercise or drinking anything but non-sparkling water for 2 h before testing.

2.3. Imagine/No-Imagine paradigm

Intentional control of future fears was assessed using an adapted version of the Imagine/No-Imagine paradigm (I/NI; see Benoit et al., 2016), presented using E-Prime (Version 2.0, Psychology Software Tools, Pittsburgh, PA). The original paradigm consists of three phases, designed to be completed over one session. The current study divided the three phases over two days (see Fig. 1). To account for the 24 h delay between sessions, an additional 'Reminder Task' was added and is described in the procedure.

2.3.1. Phase 1

On day 1, participants generated descriptions of 18 future feared events. The descriptions generated were then summarised by participants, by a 15-word limit. Participants then generated corresponding reminder words, which act as a cue for the associated fear. Each fear had to fulfil six criteria: the event must be negative; imaginable through their own eyes; possibly occur within approximately the next two years; be a specific episode that occurs over a short time; and, importantly, it must be an event they tend to think about. Participants also provided subjective ratings for each fear on a 5-point Likert scale (1 = not at all; 5 = extremely) based on: vividness; emotional intensity; anxiety; likelihood of occurrence; frequency of thought; and distance in the future. These subjective ratings were used to equally and randomly divide fear items into one of three stimuli categories: Imagine; No-Imagine; or baseline.

On day 2, after the reminder task, participants provided a typical detail for each of the 18 fears they had listed on day 1. These details were supposed to relate to a specific element envisioned in the participants' imagination (see Fig. 2). It could not be a word from the description, nor could it be too similar to the reminder cue.

2.3.2. Phase 2

The Imagine/No-Imagine manipulation tasked participants to actively imagine some of their future fears and to suppress imaginations of others. During this phase, we presented all six reminder cues of the Imagine and of the No-Imagine items. Baseline items were not cued during this phase. Each reminder cue was repeatedly presented (12 times each) in a random order, with the restriction that no more than three reminder cues of the same condition (i.e., Imagine, No-Imagine) could be shown consecutively. During Imagine trials, participants were instructed to vividly imagine the associated fear. With each repetition of the same reminder cue, participants were instructed to build on what they had previously imagined and develop a vivid, detailed episode. For

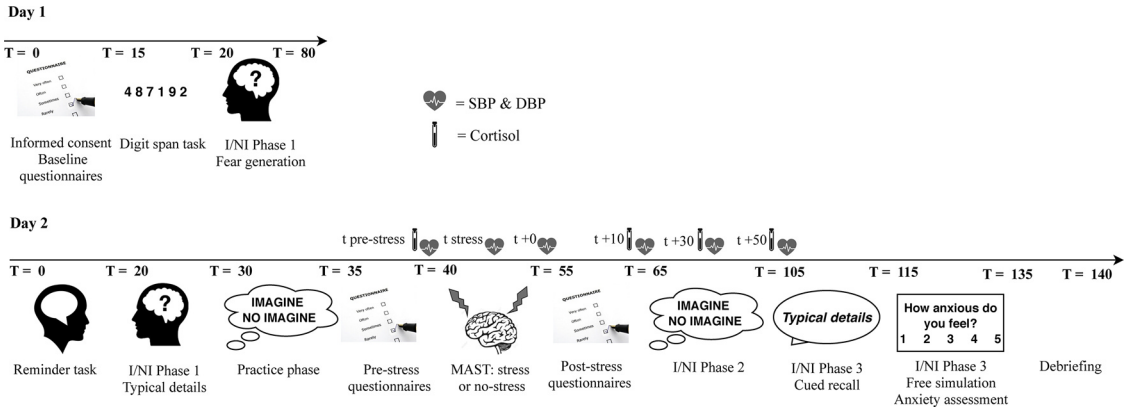

Fig. 1. Outline of the three phases of the Imagine/No-Imagine paradigm with example fears. Phase 1: participants generated descriptions, reminder cues, typical details and subjective ratings of future feared events. Phase 2: in response to reminder cues, participants intentionally retrieved (Imagine items) or suppressed (No-Imagine items) imaginations of the associated fear. At the end of each trial, participants reported how often they had imagined the fear (1 = never; 2 = sometimes; 3 = always). Phase 3: cued recall and anxiety assessment. In response to reminder cues, participants recalled typical details for all Imagine, No-Imagine and baseline items. Hereafter, the reminder cues were presented once more and participants freely described the associated fear before providing a final anxiety rating with respect to that event.

No-Imagine trials, participants had to block out all imagination of the event until the reminder cue disappeared from the screen and do so without using distraction tactics, such as replacing the imagination with an alternative thought. Further, they were instructed that if any thoughts about the event did enter their mind, they should actively push the event out of mind. Participants completed the task in silence and maintained their focus on the reminder cues. Each trial began with a centred fixation cross with a random duration of 2–2.5 seconds, followed by either an Imagine or No-Imagine cue (1.5 s), followed by a corresponding reminder cue (4 s; see Fig. 2). Imagine trials appeared in green text and No-Imagine trials appeared in red text. At the end of each trial, participants responded to the question: "How often did you imagine the future fear?" (1 = never; 2 = sometimes; 3 = always).

Prior to the main task, participants completed the practice phase, in which they were provided with the descriptions and reminder cues of 3 example fears to familiarise themselves with the Imagine/No-Imagine procedure. Two reminder cues were presented in the No-Imagine condition and one reminder cue was presented in the Imagine condition.

2.3.3. Phase 3

The effects of the Imagine/No-Imagine manipulation were assessed in the cued recall phase. Here, participants recalled all typical details for each event of the Imagine, No-Imagine and baseline conditions in response to their respective reminder cue (presented in black font). All 18 reminder cues were presented in a random order for 4 s each, during which participants had to recall the corresponding typical detail aloud. The experimenter recorded the percentage of correctly recalled typical details in the Imagine, No-Imagine and baseline conditions.

The effects of the manipulation on anxiety were assessed in the final task. Participants were presented with all 18 reminder cues in a random order for 1 min each. During this time, participants vividly described the future feared event aloud. At the end of each trial, participants rated their current feeling of anxiety toward the event on a 5-point Likert scale (1 = not at all; 5 = extremely).

2.4. The Maastricht Acute Stress Test

The Maastricht Acute Stress Test (MAST) was used to activate the human stress system (see Smeets et al., 2012). The MAST consists of a 5 min instruction phase, followed by a 10 min acute stress phase alternating between two trial types: exposure to ice-cold water (4 °C) and challenging mental arithmetic, in which participants count backwards in increments of 17 as fast and as accurately as possible, starting at 2043. The experimenter only provided negative feedback, addressing mistakes and speed of the arithmetic. Participants were told that they would be videotaped throughout in order to later analyse their facial expressions during the task. Unbeknownst to participants, the video camera did not record any footage and the data were not used at any point.

The no-stress control version aims to not elicit a stress response. Participants were required to immerse their hand in lukewarm water (between 35 − 37 °C) and count continuously from 1 to 25 for mental arithmetic trials. Participants were not videotaped, nor provided with negative feedback.

2.5. Physiological stress measures

Systolic and diastolic blood pressure (SBP; DBP) were measured from the right arm at 6 time points during day 2 ($t_{pre\text{-}stress}$, t_{stress}, t_{+0}, t_{+10}, t_{+30}, t_{+50}). Blood pressure measurements were recorded using an Omron 705IT (HEM-759-E; Omron Healthcare Europe BV).

Salivary cortisol samples were obtained at 4 time points ($t_{pre\text{-}stress}$, t_{+10}, t_{+30}, t_{+50}) via synthetic Salivettes (Sarstedt1, Etten- Leur, The Netherlands). Saliva samples were stored at −20 °C after collection

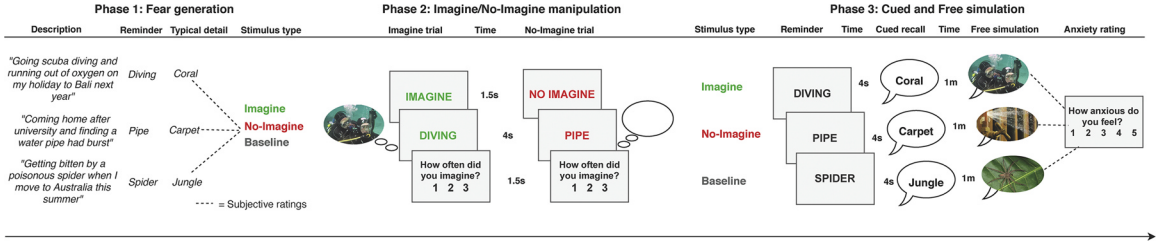

Fig. 2. Overview of the study procedure. Approximate timings (T) are denoted in minutes.

until cortisol concentrations were determined by a commercially available chemiluminescence immunoassay with high sensitivity (IBL International, Hamburg, Germany). The intra and interassay coefficients were below 9%.

2.6. Subjective stress

Participants self-reported how stressful, how painful and how unpleasant they had perceived the MAST via three 100 mm Visual Analog Scales (VASs; anchors: 0 = not at all; 100 = extremely). Subjective stress was determined via the mean score of the three VASs for each participant.

Mood ratings were recorded pre and post stress induction through completion of the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988). The PANAS includes two 10-item mood scales relating to positive affect (PANAS-P) or negative affect (PANAS-N). Each item is rated on a 5-point Likert scale (1 = not at all; 5 = extremely) in response to how the participant feels at the present moment.

2.7. Anxiety

Stress-induced changes in state anxiety were measured via the State-Trait Anxiety Inventory (STAI-S; Spielberger et al., 1983). The STAI-S consists of 20 self-report items, with scores recorded on a 4-point Likert scale in response to how anxious the participant feels at the present moment (1 = not at all; 4 = very much so). This was administered at two time points (pre-stress and post-stress).

2.8. Working memory

The Digit Span Task from the Wechsler Adult Intelligence Scale-Revised (Wechsler, 1981) was used to assess working memory capacity. Participants were read aloud a sequence of digits and asked to repeat the digits back either in the same order (forward condition) or the reverse order (backward condition). After two sequences of the same digit span length were completed successfully, the participant would proceed to the next. Each sequence increased in increments of 1 digit. If two errors were made on one sequence, the task was stopped. Scores obtained from the backward condition ($WM_{Backward}$) were used as a measure of working memory capacity for later analysis. Performance on the backward condition has been found to correlate with activity in the right dlPFC, a key neural substrate of intentional memory control (Hoshi et al., 2000).

2.9. Procedure

On test day 1 (see Fig. 1), participants were first given information regarding the experiment and thereafter provided informed consent. Next, participants completed baseline questionnaires and subsequently performed the digit span task. For the remainder of test day 1, participants generated 18 fear descriptions and corresponding reminder cues (I/NI, phase 1).

On test day 2 (see Fig. 1), participants first completed the reminder task. During this task, all 18 reminder cues that were created on day 1 were presented for up to 1.5 min. During that time, participants vividly described all imaginations of the fears aloud. After each trial, the corresponding fear description was presented for 10 s as feedback. Participants then completed the remainder of phase 1, generating typical details for each fear description. Following this, participants completed the practice phase of the I/NI task. Pre-stress measures of negative affect, state anxiety, blood pressure and saliva were then recorded ($t_{pre-stress}$). This was immediately followed by exposure to either the stress or no-stress version of the MAST. Blood pressure was measured once during the task (t_{stress}) and immediately after (t_{+0}). Following this, participants completed subjective stress, negative affect and state anxiety questionnaires. Ten minutes after completion of the MAST, blood pressure and the second saliva sample were taken (t_{+10}). Participants then continued to the first block of the Imagine/No-Imagine task (phase 2). At the end of block one, blood pressure and saliva samples were measured (t_{+30}) and then participants proceeded to block two under the same instructions. EEG data were recorded of each participant during phase 2. However, these results are not reported in this paper. After completion, the final blood pressure and saliva samples were measured (t_{+50}). This was followed by a cued-recall task to test memories for the typical details and a subsequent free simulation task designed to provide a final anxiety assessment (I/NI, phase 3). Participants were then debriefed, thanked and compensated for their time.

2.10. Behavioural & statistical analysis

For the Imagine/No-Imagine paradigm, intrusions were determined based on the subjective ratings recorded during No-Imagine trials in phase 2. Responses of 2 or 3 were classified as intrusions (following Levy and Anderson, 2012; Benoit and Schacter, 2015), indicating that the participant had been unsuccessful in fully suppressing the imaginations of the associated future fear.

In line with previous studies (Hellerstedt et al., 2016; Kuhl et al., 2007), we calculated a subject-specific measure of forgetting relative to baseline memory performance. The suppression-induced forgetting (SIF) index was calculated by subtracting the recall of No-Imagine items from Baseline items and then dividing by Baseline items ((Baseline – No-Imagine) / Baseline). Higher positive index scores indicate increased forgetting. This index calculation was also applied to intentional retrieval, in order to account for individual variation in baseline performance ((Baseline – Imagine) / Baseline).

Analyses of anxiety changes following the Imagine/No-Imagine manipulation were based on the final ratings (phase 3). Single value index scores were calculated for suppressed and retrieved items, respectively (suppression: ((Baseline anxiety – No-Imagine anxiety) / Baseline anxiety); retrieval: ((Baseline anxiety – Imagine anxiety) / Baseline anxiety)). We then multiplied these measures by -1 to render positive instead of negative values, thus higher positive index scores indicate increased anxiety.

Statistical analysis were performed using SPSS Statistics for Windows, version 25 (SPSS Inc., Chicago, Ill., USA). All data were checked for normality using Q-Q plots and Shapiro-Wilk tests. A log-transformation was performed to account for skewed cortisol data, the values of which were used in subsequent analyses. Eight participants were identified as outliers due to recall and anxiety index scores of more than 2.5 SDs below the mean. These participants were excluded from further analyses. Therefore, the final sample size was 84 (stress: n = 43 (10 male); control: n = 41 (11 male)).

Independent *t*-tests, one-sample *t*-tests and repeated-measures ANOVA's were performed. In cases of violated normality or sphericity, adjusted Welch's F ratios and Greenhouse-Geisser corrected values are reported, respectively. Two-tailed *p*-values are reported for interaction analyses for the stress manipulation and the moderation analysis of working memory. One-tailed *p*-values are reported for planned directed comparisons for the stress and Imagine/No-Imagine manipulations. ANOVA results are supplemented with Partial Eta Squared values (η_p^2) as a measure of effect size (η_p^2 of 0.01 indicate small effects, η_p^2 of 0.06 medium effects, and η_p^2 of 0.14 large effects; Fritz et al., 2012). Results from *t*-tests are supplemented with Cohen's *d* as a measure of effect size, calculated by the mean difference score as the numerator and the average standard deviation of both groups as the denominator (Cohen's *d* of 0.20 indicate small effects, 0.50 medium effects and 0.80 large effects; Cohen, 1988).

3. Results

3.1. Successful acute stress induction via the MAST

3.1.1. Physiological stress responses

Significant increases in blood pressure and cortisol confirmed the success of the acute stress induction. One participant had missing data for blood pressure. To assess the difference in noradrenergic response between groups before, during and repeatedly after the MAST, a repeated-measures ANOVA was performed on blood pressure recordings (SBP and DBP) with Time (6 levels: $t_{pre\text{-}stress}$, t_{stress}, t_{+0}, t_{+10}, t_{+30}, t_{+50}) as the within-subjects (WS) factor and Condition (2 levels: stress vs. control) as the between-subject (BS) factor. Results revealed that systolic and diastolic blood pressure were elevated in response to the stress-version of the MAST, but not in response to the control manipulation (Time*Condition; SBP: $F_{(3.49,\ 282.82)} = 34.00$, $p < .001$, $\eta_p^2 = 0.30$; DBP: $F_{(2.57,\ 207.95)} = 43.13$, $p < .001$, $\eta_p^2 = 0.35$; see Fig. 3). Follow-up tests demonstrated that, prior to the MAST, the conditions did not differ significantly in blood pressure (both p's > .16). Commencing the MAST, the conditions differed significantly at t_{stress} (SBP: $t_{(81)} = -6.16$, $p < .001$, $d = 1.36$; DBP: $t_{(81)} = -7.03$, $p < .001$, $d = 1.56$), t_{+0} (SBP: $t_{(81)} = -6.15$, $p < .001$, $d = 1.36$; DBP: $t_{(81)} = -6.82$, $p < .001$, $d = 1.50$) and t_{+10} (SBP: $t_{(81)} = -2.28$, $p = .013$, $d = 0.39$; DBP: $t_{(82)} = -3.28$, $p = .001$, $d = 0.23$) and t_{+30} (SBP: $t_{(81)} = -1.90$, $p = .031$, $d = 0.42$). No significant differences were observed at t_{+30} for DBP and t_{+50} for both SBP and DBP (all p's > .09).

To assess the effects of the MAST on the neuroendocrine response, we performed a repeated-measures ANOVA on cortisol concentrations at different time points (WS; Time, 4 levels: $t_{pre\text{-}stress}$, t_{+10}, t_{+30}, t_{+50}) for each condition (BS; 2 levels: stress vs. control). The analysis revealed elevated concentrations in stressed participants compared to controls (Time*Condition: $F_{(2.18,\ 178.36)} = 14.78$, $p < .001$, $\eta_p^2 = 0.15$; see Fig. 3). Follow-up tests identified differences between groups at $t_{pre\text{-}stress}$ ($t_{(82)} = -2.67$, $p = .005$, $d = 0.58$), t_{+10} ($t_{(82)} = -7.68$, $p < .001$, $d = 1.68$), t_{+30} ($t_{(82)} = -6.93$, $p < .001$, $d = 1.52$) and t_{+50} ($t_{(82)} = -3.74$, $p < .001$, $d = 0.82$). Pairwise comparisons with the Holm-Bonferroni correction (Holm, 1979) were performed to assess the change in cortisol levels between the time-points within each condition. In stressed participants, cortisol levels increased significantly between $t_{pre\text{-}stress}$ and t_{+10} ($p = .003$) and subsequently decreased between t_{+10} and t_{+30}, and between t_{+30} and t_{+50} (both p's = .003). Contrastingly, in unstressed participants, cortisol levels decreased significantly between $t_{pre\text{-}stress}$ and t_{+10} ($p = .003$). Cortisol was not found to differ between the subsequent time-points (all p's > .98).

3.1.2. Subjective stress responses

Participants in the stress condition reported higher subjective stress compared to controls. The independent samples t-test revealed that participants in the stress condition experienced the experimental manipulation as significantly more stressful than participants in the control condition (VAS: $t_{(82)} = -22.27$, $p < .001$, $d = 4.90$; see Table 1). Furthermore, negative affect and state anxiety increased significantly in stressed participants compared to unstressed participants (PANAS-N; Time*Condition: $F_{(1,82)} = 45.41$, $p < .001$, $\eta_p^2 = 0.36$; STAI-S: Time*Condition: $F_{(1,82)} = 92.03$, $p < .001$, $\eta_p^2 = 0.53$). Follow up tests revealed no significant difference between the conditions at the pre-stress measure (both p's > .19) and a significant difference at the post-stress measure (PANAS-N: $t_{(82)} = -7.19$, $p < .001$, $d = 1.55$; STAI-S: $t_{(82)} = -9.91$, $p < .001$, $d = 2.15$).

3.2. Acute stress impairs suppression-induced forgetting of future fear details

In order to investigate whether acute stress influenced the success of suppression during No-Imagine trials, the total amount of intrusions reported by stressed and unstressed participants were compared using an independent samples t-test. Due to measurement error, one participant had missing data and was excluded from this part of the analysis only. Stressed participants reported a significantly greater amount of intrusions compared to unstressed participants ($t_{(81)} = -2.63$, $p = .005$, $d = 0.58$; see Fig. 4A), demonstrating that acute stress impaired the ability to suppress imaginations of future fear details.

The effect of acute stress on suppression-induced forgetting was assessed using an independent samples t-test with Condition (2 levels: stress vs. control) as the between subjects variable. Acute stress impaired the ability to forget, as reflected by a lower SIF index in stressed participants compared to unstressed participants ($t_{(82)} = 2.22$, $p = .015$, $d = 0.49$; see Fig. 4B and Table 1)[1] . Follow up tests were performed using one sample t-tests for each condition. The SIF index of stressed participants was not significantly different from zero ($t_{(40)} = -1.43$, $p = .081$, $d = 0.21$), indicating an impairment in suppression-induced forgetting. In contrast, SIF for unstressed participants was significant ($t_{(40)} = 1.81$, $p = .039$, $d = 0.28$). We thus replicated the original finding by Benoit et al. (2016) that suppression of future fear details leads to subsequent forgetting.

For anxiety toward suppressed future fear details, an independent samples t-test was performed with condition (2 levels: stress vs. control) on the suppression index. Although numerically higher indices were observed in stressed participants, indicating increased anxiety, this did not differ significantly from unstressed participants ($t_{(82)} = 0.72$, $p = .24$, $d = 0.16$; see Fig. 4C). Unlike Benoit et al. (2016), we did not find evidence for attenuated anxiety within the control condition ($t_{(40)} = 0.11$, $p = .46$, $d = 0.01$).

3.3. Acute stress does not influence the intentional retrieval of future fear details

The effect of acute stress on intentional retrieval of future fear details was assessed using an independent samples t-test with Condition (2 levels: stress vs. control) as the between-subjects variable (see Fig. 5A). The retrieval index was not found to differ between stressed and unstressed participants ($t_{(82)} = 1.18$, $p = .12$, $d = 0.26$; see Fig. 5A), suggesting that acute stress did not influence the intentional retrieval of fear details. Within the control condition, one-sample t-tests showed that the retrieval index did not differ significantly from zero ($t_{(40)} = 1.41$, $p = .084$, $d = 0.22$), consistent with Benoit et al. (2016).

For anxiety toward retrieved fears, an independent samples t-test was performed with condition (2 levels: stress vs. control) and the retrieval index. No significant difference was observed between conditions ($t_{(82)} = 0.52$, $p = .30$, $d = 0.11$; see Fig. 5B), suggesting that acute stress did not influence anxiety toward imagined future fear details. Within the control condition, one sample t-tests showed that the retrieval index differed significantly from zero ($t_{(40)} = 2.83$, $p = .004$, $d = 0.44$), suggesting that retrieval of future fears increased anxiety.

3.4. Moderation of the effect of acute stress on suppression-induced forgetting by working memory capacity

To investigate the moderating effect of working memory on the stress-induced impairment in suppression induced forgetting, a multiple regression analysis using the PROCESS tool for SPSS (Model 1 with 1000 bootstrapping) was performed with scores from the backward condition ($WM_{Backward}$) of the digit span task as a moderator.

[1] To investigate whether acute stress had influenced recall in general, and therefore excluding items that were cued in the Imagine/No-Imagine manipulation (phase 2), we performed an exploratory independent samples t-test on the recall scores for baseline items between stressed and unstressed participants. No significant group difference was observed ($t_{(82)} = 1.18$, $p = .12$, $d = 0.26$), showing that stress more selectively impaired the suppression of fear details.

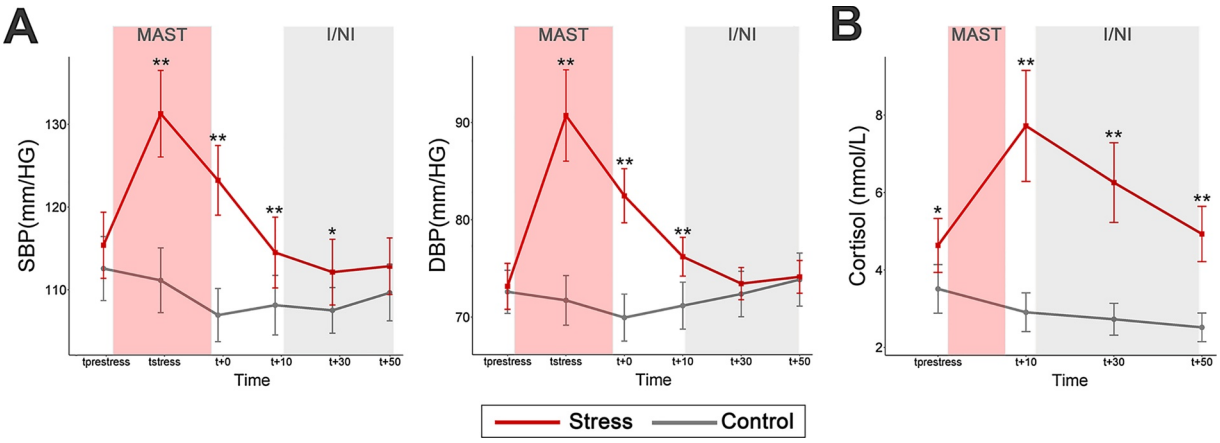

Fig. 3. Physiological and neuroendocrine stress response. Systolic and diastolic blood pressure (SBP; DBP; Panel A) and untransformed salivary cortisol concentrations (Panel B) in response to the MAST and throughout the Imagine/No-Imagine (I/NI) task. Significant differences between groups are indicated (** $p < .001$; * $p < .05$). Graphs display means and errors bars indicate 95 % confidence intervals.

Table 1
Means (± SE) of subjective stress, negative affect and state anxiety scores before and after the MAST, recalled fear details and anxiety ratings for each stimulus type in the stress and control conditions.

	Subjective stress				
	VAS	PANAS-N		STAI-S	
		Pre	Post	Pre	Post
Stress	71.68 (2.61)	7.21 (0.42)	10.02 (0.61)	37.60 (1.46)	49.74 (1.54)
Control	6.07 (1.26)	6.73 (0.33)	5.49 (0.16)	36.07 (1.39)	31.56 (1.00)

	Fear detail recall (%)		
	Imagine	No-Imagine	Baseline
Stress	63.47 (3.89)	67.05 (3.11)	66.28 (3.35)
Control	63.81 (3.68)	62.19 (3.64)	71.54 (2.92)

	Anxiety ratings		
	Imagine	No-Imagine	Baseline
Stress	3.54 (0.10)	3.42 (0.11)	3.38 (0.11)
Control	3.55 (0.11)	3.32 (0.11)	3.35 (0.11)

Furthermore, a significant interaction between condition and working memory was observed ($b = 0.21$, $t_{(80)} = 2.92$, $p = .005$, CI [0.07, 0.35], see Fig. 6). To explore this effect, further simple slope analyses on the conditional effects of condition were tested at two levels of working memory: low (-1SD = 4.04) and high (+1SD = 6.49). A lower working memory capacity significantly contributed to the effect of acute stress on suppression-induced forgetting ($WM_{Backward(low)}$: $b = -0.45$, $t_{(80)} = -3.68$, $p < .001$, CI [-0.69, -0.21]). A higher working memory capacity did not contribute significantly ($p = .63$). This indicates that suppression-induced forgetting of lower capacity individuals was influenced by acute stress; in contrast, this effect was absent in higher capacity individuals.

The overall model using the retrieval index did not prove significant ($p = .67$); therefore, there was no evidence that working memory capacity moderated effects of the intentional retrieval of fear details. Further, working memory was not found to moderate anxiety toward suppressed or retrieved fears ($p = .51$ and $p = .88$ respectively).

4. Discussion

The current study investigated the effects of acute stress on the in-

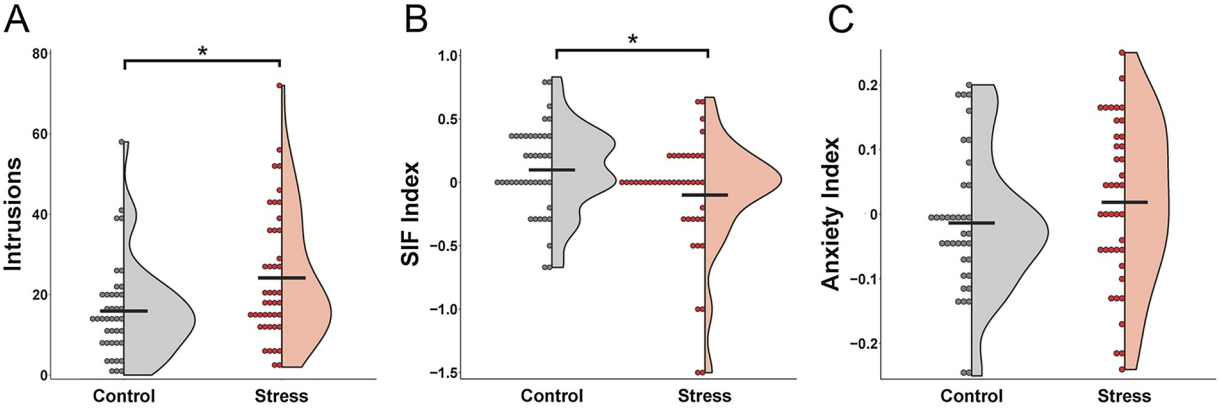

Fig. 4. Suppression Indices: Total number of intrusions (Panel A), suppression-induced forgetting index (Panel B) and index for anxiety of suppressed items (Panel C) for each condition. Positive SIF index values indicate increased forgetting and positive anxiety index values indicate increased anxiety. Significant differences between groups are indicated (* $p < .05$). Split violin plots display the distribution of the data, group means (indicated by the black bar) and individual data points.

The overall model including condition was significant ($WM_{Backward}$: $F_{(3,80)} = 4.68$, $p = .005$, $R^2 = 0.15$). In support of our prior *t*-test result, condition significantly predicted differences in suppression-induced forgetting ($b = -1.29$, $t_{(80)} = -3.36$, $p = .001$, CI [-2.05, -0.53]). tentional control of future fears. Further, we examined whether individual differences in executive control would moderate these effects. The effect of acute stress and its moderation by working memory capacity was selective for the suppression of future fears, as opposed to

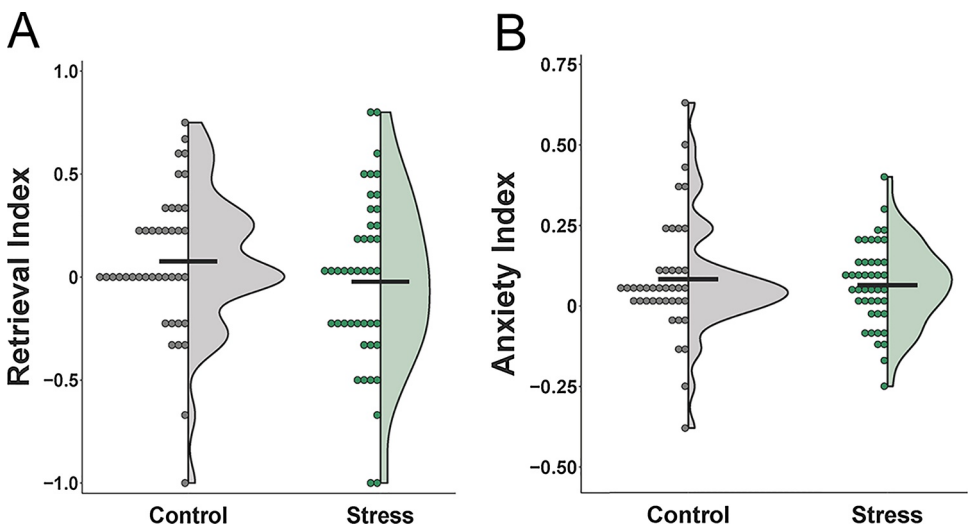

Fig. 5. Retrieval Indices: Intentional retrieval index (Panel A) and index for anxiety of retrieved items (Panel B) for each condition. Positive SIF index values indicate increased forgetting and positive anxiety index values indicate increased anxiety. Split violin plots display the distribution of the data, group means (indicated by the black bar) and individual data points.

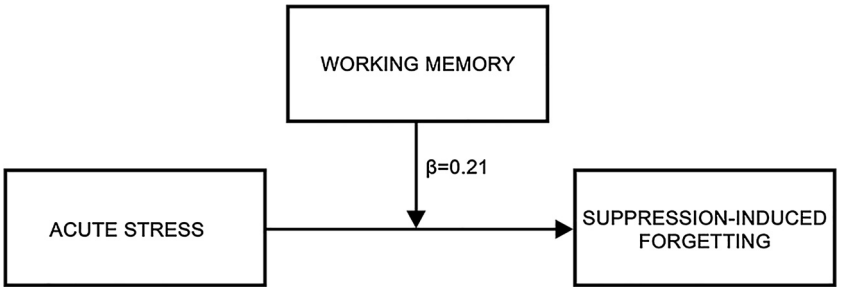

Fig. 6. Moderation by working memory: A model for the effect of acute stress on suppression-induced forgetting, as moderated by working memory. The β coefficient for the significant interaction between condition and working memory is denoted.

retrieval.

Due to the known impairing effects of stress on executive control, we predicted that exposure to an acute stressor would impair suppression of future fear details. This should thus hinder suppression-induced forgetting. Interestingly, acute stress significantly increased the number of intrusions reported by participants during attempted suppression. Consequently, stressed participants demonstrated a significantly lower SIF index compared to unstressed participants, corroborating the recent findings that stress impairs suppression-induced forgetting of unwanted memories (Quaedflieg et al., in press). We did not find evidence that stress influenced anxiety toward suppressed fears. Previous research has found that, in the absence of noradrenergic activation, (genomic) glucocorticoid actions interfere with inhibitory processing (Shields et al., 2015). This concurs with present findings, supporting the notion that inhibitory control is critical for suppressing unwanted thoughts or memories (Benoit et al., 2016; Levy and Anderson, 2008). Furthermore, the fact that our data showed an effect that was specific to No-Imagine items speaks against the view that our findings represent another example for the well-known stress-induced retrieval changes (de Quervain et al., 2000; Gagnon and Wagner, 2016). If acute stress had merely affected retrieval *per se*, one would have expected that memory for baseline and Imagine fear details would also have been affected. This was, however, not the case.

The dlPFC facilitates successful suppression-induced forgetting of future fears by downregulating activity in the vmPFC and hippocampus (Benoit et al., 2016). On the one hand, forebrain circuits are involved in regulating the HPA axis response and stress integrative functions (Ulrich-Lai and Herman, 2009). On the other hand, non-genomic glucocorticoids switch the network balance in the brain, reducing activity in the executive network, including the dlPFC (Hermans et al., 2011, 2014; Qin et al., 2009). Moreover, individual differences in cortisol levels during stress recovery have been found to moderate the dlPFC–amygdala connectivity during rest (Quaedflieg et al., 2015). A stress-induced down-regulation of the dlPFC may thus prevent the top-down inhibitory control signal to these regions (McEwen et al., 2016).

Results further revealed that individuals with lower working memory capacity were more susceptible to the negative effects of acute stress on suppression-induced forgetting. In contrast, individuals with higher working memory were unaffected and their ability to forget did not differ from their counterparts in the control condition. This emphasizes the critical role executive control serves for suppressing imaginations of future fears under acute stress. Levy and Anderson (2008) suggested that individual differences in executive control are able to predict the success of intentional memory control. They further suggest that, as such, factors that influence the ability to engage efficient executive control should therefore also affect the latter. When observing intentional memory control effects in the absence of further manipulation, baseline working memory capacity has not been found to predict the outcome of suppression-induced forgetting (Waldhauser et al., 2011). However, when the demand of working memory is manipulated to a high load during memory control, suppression-induced forgetting has been found to decrease (Noreen and de Fockert, 2017). Furthermore, individuals with low working memory capacity have been found to be more susceptible to the detrimental effects of stress on other forms of higher cognitive functioning, such as goal-directed behaviour (Otto et al., 2013; Quaedflieg et al., 2019). Combined, these past and current findings suggest that a reduced working memory capacity negatively affects suppression-induced forgetting and, furthermore, creates a vulnerability to the detrimental effects of stress.

The physiological and subjective data provide evidence that the MAST successfully induced acute stress. In line with previous studies (e.g. Quaedflieg et al., 2017; Smeets et al., 2012), participants in the

stress condition showed an increase in subjective stress, systolic and diastolic blood pressure and cortisol concentrations when compared to control. Expanding on findings from previous studies, participants in the stress condition reported increased state anxiety following the MAST. It has previously been shown that individuals with higher trait anxiety are less able to reduce feelings of anxiety toward future fears via suppression (Benoit et al., 2016). As such, an increase in state anxiety could, in part, influence the subsequent outcome of attempts to suppress imaginings of future fears.

It should be noted that the sample consisted of predominately female participants (n = 63), all of which were using hormonal contraception. Hormonal alterations throughout the menstrual cycle have been related to the variability in cortisol responses after acute stress in women (Kudielka et al., 2009). The use of hormonal contraceptives has also been found to alter the learning and memory of emotional content under response to stress (Nielsen et al., 2014). As such, these findings may not generalize to naturally cycling women. It is also important to note that, unexpectedly, stressed participants demonstrated increased cortisol concentrations at baseline compared to unstressed participants. Although, relative to the baseline measure, stressed participants demonstrated an increase in cortisol after the MAST, whereas unstressed participants demonstrated a decrease. Participants were randomly allocated to each group and other factors that could account for variation in cortisol were controlled for (such as time of testing, age, weight, alcohol and drug intake; see Strahler et al., 2017). As such, we cannot offer conclusive reasoning to explain the initial difference at baseline. As a speculative explanation: it could be that the researchers' approach toward participants differed unintentionally during interactions prior to the MAST, if they were aware that they would imminently have to induce acute stress. Future research may preclude this possibility by adopting a double-blind design in which an independent researcher performs the MAST across both conditions.

Accumulating evidence points to the fascinating possibility that we can, to some degree, intentionally control our fears and thoughts by actively retrieving and imagining some experiences while suppressing others. The current study expands on previous findings by showing how suppression of fear imaginings can be deficient under acute stress and that this is moderated by individual differences in executive control. Specifically, a lower working memory capacity seems to predict a susceptibility to the detrimental effects of acute stress. Working memory can, to some extent, be improved. Working memory training has shown promising results, reducing symptomatology of anxiety and depression in vulnerable individuals (Beloe and Derakshan, 2019; Sari et al., 2016). However, it has been argued that, despite the benefits of working memory training, these learned skills do not transfer to other tasks (Gathercole et al., 2019). It would be of interest for future research to explore whether working memory training could be developed specifically to enhance suppression-induced forgetting.

In the presence of stress-related psychopathology, it has been shown that the efficacy in suppressing unwanted memories is reduced (Hertel and Gerstle, 2003; Joormann et al., 2005; Mary et al., 2020; Nørby, 2018). Moreover, meta-analytical evidence indicates, more generally, that individuals with problems in controlling intrusive thoughts are deficient in suppressing memories (Stramaccia et al., 2019). Here, we demonstrate that exposure to acute stress negatively affects suppression by increasing intrusions and impairing the ability to forget. Furthermore, individual differences in working memory moderate this effect. The current findings serve as an insightful step toward understanding the causes of failure in the intentional control of future fear imaginings.

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