Behavioural Brain Research 326 (2017) 272–280

Contents lists available at ScienceDirect

Behavioural Brain Research

journal homepage: www.elsevier.com/locate/bbr

Research report

Memory for objects and startle responsivity in the immediate aftermath of exposure to the Trier Social Stress Test☆

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HIGHLIGHTS

- The study investigated the effects of stress exposure on memory and startle.
- After the TSST participants remembered more central objects from the stressor.
- Startle responsivity was overall only descriptively enhanced.
- Startle responsivity to an odour ambient during the stressor tended to be enhanced.
- Commonalities and differences between immediate and delayed stress effects are discussed.

ARTICLE INFO

Article history:
Received 12 November 2016
Received in revised form 22 February 2017
Available online 14 March 2017

Keywords:
Eye blink
Cortisol
Olfaction
Consolidation
Recognition
Free recall

ABSTRACT

Previously, we observed enhanced long-term memory for objects used (central objects) by committee members in the Trier Social Stress Test (TSST) on the next day. In addition, startle responsivity was increased. However, response specificity to an odour involved in the stressful episode was lacking and recognition memory for the odour was poor. In the current experiments, immediate effects of the stressor on memory and startle responsivity were investigated. We hypothesised memory for central objects of the stressful episode and startle response specificity to an odour ambient during the TSST to be enhanced shortly after it, in contrast to the control condition (friendly TSST). Further, memory for this odour was also assumed to be increased in the stress group. We tested 70 male (35) and female participants using the TSST involving objects and an ambient odour. After stress induction, a startle paradigm including olfactory and visual stimuli was conducted. Indeed, memory for central objects was significantly enhanced in immediate aftermath of the stressor. Startle responsivity increased at a trend level, particularly with regard to the odour involved in the stressful episode. Moreover, the stress group descriptively tended towards a better recognition of the odour involved. The study shows that stress enhances memory for central aspects of a stressful situation before consolidation processes come into play. In addition, results preliminarily suggest that the impact of stress on startle responsivity increases in strength but decreases in specificity during the first 24 h after stress exposure.

1. Introduction

Stressful events can lead to long-lasting memories due to adaptive response mechanisms activated in a stressful situation. Stress induces rapid activation of the sympathetic nervous system (SNS) associated with increased vigilance and reduced executive control [1]. With a slight delay, the stress hormone cortisol is released through activation of the hypothalamus-pituitary-adrenal (HPA) axis. Cortisol initially potentiates the arousal-induced enhancement of basolateral amygdala activation via rapid non-genomic mechanisms, thereby enhancing memory consolidation [2]. Subsequently, cortisol reduces neuronal excitability via genomic mechanisms, causing a return to homeostasis [3]. This latter process reduces interference and further promotes long-term memory consolidation [4,5]. Particularly stress occurring within the learning context boosts memory for this event [5] and for potentially relevant items involved in the stressful situation [6].

☆ This work was supported by the German Research Foundation (DFG) as project B4 of the Collaborative Research Centre (SFB) 874 "Integration and Representation of Sensory Processes" awarded to Oliver T. Wolf.

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http://dx.doi.org/10.1016/j.bbr.2017.03.002
0166-4328/

In humans, few studies have tested memory for experimentally induced stressful events [6,7]. While some studies resulted in impaired memory for stimuli experienced during stress [8] or reported general stress related memory impairments [9], others observed enhanced memories, especially for stressor-related stimuli [6,10,11] or pictures encoded during the stressful experience [12]. All of these studies tested delayed memory in a longer temporal distance to the acute stressor, when consolidation processes come into play and HPA activity restored to baseline. Memory for items related to the stressful situation has not yet been tested in the direct aftermath of the acute stress phase.

We recently compared memory performance of participants exposed to a psychosocial stressor (*Trier Social Stress Test*) [13] to that of participants exposed to a similar but non-stressful control condition (*friendly TSST*) [14]. In two independent studies, we could demonstrate that participants exposed to the stressful TSST, holding a free speech in front of a committee in a simulated job interview, performed better recognising central items (e.g. a rubber or a pen) which had been used by the members of the committee in a standardised fashion [6,14], on the next day. In contrast, there were no differences in recognition memory between the groups for peripheral objects (e.g. a mug or a ruler) which were also present on the table the committee was sitting at, but had not been used. As in the other studies mentioned before [6,10–12], memory was tested on the next day. It thus remained open whether this effect developed over time or might have been already detectable immediately after stress exposure.

In the acute stress phase, vigilance is increased to enhance attention for potentially relevant stimuli and to promote fast responses to the situation at hand [3]. This state leads to stronger responses particularly to negative stimuli in any sensory modality, e.g. a loud noise or a flash. Besides the immediate effects of stress on increased vigilance, long-term effects of stress on vigilance in terms of startle responsivity could be observed in the past. In a previous study, participants stressed with the TSST in a room with a previously neutral and unknown odour showed an enhanced startle response even 24 h after the stressful experience [15]. By applying a white noise burst via headphones, a startle eye blink reflex can be provoked whose amplitude or magnitude can be measured via electrodes attached to the eye muscle [16,17]. Being a well-established method for assessing implicit affective states, the fear-potentiated startle is often used to study the effects of valent stimuli or emotional conditions. Since negative stimuli presented during the startle procedure enhance the startle response [18–20], initially neutral stimuli associated with a stressful situation and the correspondent aversive state should have the potency to enhance the startle response like stimuli innately negative would do. The increased startle responsivity found in our previous study, however, was not specific to stimuli presented during the TSST (e.g. the ambient odour or the faces from the committee members), but was rather general and unspecific, indicative of increased anxiety [15]. This stronger responsivity at the expense of specificity was hypothesised to represent a functional shift in amygdaloidal activity [21]. Thus, a one-time laboratory stress experience apparently had the potency to activate an amygdaloidal pathway which was consolidated during a 24 h time course and could pre-attentively be re-accessed by the startle procedure on the following day.

In contrast to this, in a previous study inducing stress with the Cold Pressor Test (CPT), diminished startle responsivity in the immediate aftermath of the stress induction was found [22]. The application of different stressors (CPT vs. TSST) might be responsible for the different results. Alternatively, acute stress might initially cause diminished startle responsivity, which, over a time course of 24 h, increases and eventually results in significantly enhanced startle responsivity. Moreover, it is unclear whether the response specificity towards stimuli experienced in the stressful context is increased in the immediate aftermath of the stressor.

Taken together, the described previous experiment from our laboratory is in line with the notion of enhanced memory consolidation and increased fearfulness after a single stress exposure, manifesting 24 h after the stressful event [15]. It is yet unclear whether this effect is indeed owed to a consolidation period or might also be observed in a startle paradigm administered directly subsequent to stress exposure. Causing a vigilant processing mode, stress should lead to an instant improvement of memory through its enhancing effects on memory encoding [1,23]. In contrast, the impact of stress on memory consolidation [24–27] suggests an enhancing long-term effect on the next day. Similarly, for effects of vigilance on the startle response, it is unclear whether startle specificity in response to stress-associated stimuli is increased in the immediate aftermath of the stressor, in contrast to 24 h later.

Given the rapid effects of stress on vigilance and attention, as well as previous findings of these effects and cortisol on startle responsivity [22,28], we intended to investigate short-term effects on the startle response during cortisol peak and on memory for stimuli experienced during the stressor in terms of visual object memory as well as olfactory memory immediately after the acute stress phase. Similar to our previous work [6,15], memory for the stressful episode and startle responsivity were assessed, this time focussed on effects after stress exposure at times of elevated cortisol concentrations. Memory retrieval took place approximately 40 min after termination of the stressor when stress impairs retrieval of stressor-unrelated material, such as peripheral objects in the TSST, whereas stressor-related memory contents are often unaffected [5,23,29]. Stress-induced physiological and psychological alternations (e.g. increased cortisol and increased negative affect) however still persist at that time after the stressor. We hypothesised that stressed participants would show an enhanced startle response specifically to the target odour and thus exhibit more specificity than 24 h later. Additionally, we presumed a better memory for the target odour in stressed participants. Moreover, stress is beneficial for memory when experienced during the learning episode (within the learning context [5,30,31]), which is the case in our study. Hence, for object memory it was hypothesised that enhanced vigilance caused by acute stress would be beneficial for free recall and recognition memory in the aftermath of the stressor, in particular for central objects.

2. Methods

2.1. Participants

We tested 70 non-smoking male ($n = 35$) and female students from the Ruhr-University Bochum without any mental and physiological diseases and regular medication use. Females were only tested when having a regular menstrual cycle. Pregnant women, women during their menses, and those taking hormonal contraceptives were excluded [32,33].

The participants' age ranged from 18 to 33 years ($M = 23.59$, $SD = 3.62$) and their Body Mass Index from 18.07 to 28.99 ($M = 22.69$, $SD = 2.63$). For their participation, psychology students received course credits, and all others were paid an expense allowance of 20 €. The study was approved by the local ethic committee of the Faculty of Psychology at the Ruhr-University Bochum, and the Declaration of Helsinki was followed.

2.2. Experimental session

Randomly assigned to either a stress or a control condition, participants underwent the Trier Social Stress Test (TSST) [13] or

the friendly TSST (f-TSST) [14], respectively. After they had signed informed consent, participants filled in the Social Interaction Anxiety Scale (SIAS) [34]. Then, they rated their current affect by means of the *Positive and Negative Affect Scale* (*PANAS*) [35] while delivering the first saliva sample using Salivettes® (Sarstedt, Nümbrecht, Germany). In the following, participants were brought to a room with either an evaluation or a friendly committee, without knowing which condition they had been assigned to. There, a fixed amount of an odour concentration, methyl benzoate (Sigma-Aldrich Corp., St. Louis, MO, USA), was dispersed during the participants' speech [36] using a special compact ventilation device (TaoMaus, TAOASIS GmbH, Detmold, Germany). After the participants had been brought back to the testing room, they delivered another saliva sample (+1 min) and again filled in the PANAS and the Edinburgh Handedness Inventory. Then, a third saliva sample (+10 min) was collected before the startle paradigm was initiated. Afterwards, the last saliva sample (+35 min) was delivered. Participants then were asked to recall as many objects present during the TSST/f-TSST as possible, before they engaged in an object recognition task where they had to rate on a 6-point scale how sure they were to have seen the objects displayed before, during speech or social interaction, respectively.

Finally, participants rated the pleasantness of six odours on a 4-point scale (1 = "very pleasant", 2 = "pleasant", 3 = "unpleasant", 4 = "very unpleasant") and were asked to decide which one was the odour present during TSST/f-TSST. The odours were the target odour methyl benzoate, two unknown distractor odours presented before via the olfactometer (bornyl acetate and linalool), as well as the unknown odour damascenone and the known odours lemon and lavender [37].

2.3. Material

2.3.1. Trait questionnaire

The Social Interaction Anxiety Scale (*SIAS*) [34] includes 20 items to be rated on a 5-point scale ranging from 1 = 'not at all' to 5 = 'extremely'. With a Cronbach's α of .90 the German version of the SIAS exhibits a good reliability [38].

2.3.2. Stress procedure and control condition

2.3.2.1. Trier Social Stress Test (TSST). Stress was elicited using a modified version of the TSST [13] activating the HPA axis which leads to cortisol release [39]. The core factors are socially evaluative threat, caused by holding a free speech in front of a cold and reserved evaluation committee while being videotaped, and uncontrollability. The mental arithmetic task of the original version of the TSST was replaced by additional five minutes of speech, extending it to 10 min [6]. Introduced as trained behavioural psychologists analysing the participants' behaviour, one male and one female form the committee, displaying neutral and reserved behaviour. Simulating a job interview, participants are instructed to only refer to personal qualifications for the desired job position. Before the speech, a five minutes preparation time is permitted.

2.3.2.2. Friendly TSST (f-TSST). A comparable control condition not activating the HPA axis and increasing negative affect is the friendly TSST (f-TSST) [14]. The committee is introduced by their names and the participant is allowed to choose from suggested topics resembling the contents of a job interview. The friendly committee engages in an informal interaction with the participant without videotaping. Apart from this, the f-TSST is identical to the stress procedure.

2.3.3. Physiological stress measures

2.3.3.1. Salivary cortisol. Participants were to refrain from any drug intake as well as from physical exercise 24 h before testing. One hour before the appointed test time, they were instructed to drink nothing but water and not to brush their teeth. Salivettes® of the four collected saliva samples per participant were deep-frozen at −18 °C. Analysis was done at the local laboratory of the Ruhr-University Bochum with the *DEMEDITECs Cortisol Free in Saliva enzyme-linked immunosorbent assay (ELISA) Kit*. Intra- and inter-assay coefficients of variation (CV) were below 10%. Since cortisol release follows a circadian rhythm, we had two fixed time slots for testing, participants were assigned to equally with regard to sex and condition, in a pseudo-random manner. Time slots lasted from 10 am to approximately 12 pm and 12 pm to approximately 2 pm.

2.3.3.2. Salivary alpha amylase. The enzyme alpha-amylase (sAA) was analysed from the saliva samples to measure the response of the sympathetic nervous system [40]. CNP-G3 was the substrate for the measurement of the enzymatic action of sAA at 405 nm. Intra- and inter-assay CV were both below 8%.

2.3.4. Affect measurement

We assessed the participants' affect using the Positive and Negative Affect Scale (PANAS) [35]. It features 10 positive and 10 negative emotional items to be rated on a 5-point scale ranging from 1 = 'very slightly or not at all' to 5 = 'extremely'. Results are calculated as a positive (PA) and a negative affect (NA) score.

2.4. Startle procedure

2.4.1. Startle evocation

A startle stimulus consisting of a 100 dB white noise with 50 ms duration and an instantaneous rise time was randomly presented via 80 Ω headphones (DT770 M, beyerdynamic GmbH & Co. KG, Heilbronn, Germany). The stimulus occurred between 0.5 and 2.5 s after presentation onset of odour or picture, respectively. The auditory stimulus was combined with any picture or odour, but only for 50% of the presentations, which were two for each visual or olfactory stimulus. Startle stimuli were accompanied by pictures and odours intermittently. The inter-stimulus interval (ISI) lasted for 8.5 s with 0 to 2 randomly applied startle stimuli. The startle block started with a 30 s habituation phase including 7 startle stimuli.

2.4.2. Data recording

Two bio potential electrodes (EASYCAP GmbH, Herrsching, Germany) were attached to the orbicularis oculi muscle of the left eye for the electromyography recordings [16,17,41]. A disposable ground electrode (GOLMED GmbH, Weddel, Germany) was attached to the forehead. The MP150 data acquisition device (BIOPAC Systems Inc., Essen, Germany) with filter settings 10 to 500 Hz was used as an amplifier and transmitter. The programme was written and executed with MatLab (version R2012a, MathWorks Inc., Ismaning, Germany).

2.4.3. Startle stimuli

2.4.3.1. Odours. Three odorants were dissolved in 50 ml scentless paraffinum liquidum (methyl benzoate: 60 µl, bornyl acetate: 850 µl, linalool: 100 µl; *Sigma–Aldrich Co*., Munich, Germany) and were delivered via the olfactometer. They were used in the experiment because they had previously been rated as unfamiliar and neutral [37]. The different odour concentrations assured comparable odour intensity [36]. The 6-channel constant-flow (50 ml/s) olfactometer was in-house built as described [42]. The odour mixtures were delivered via oxygen masks (*ROESER Medical GmbH*, Essen, Germany) covering nose and mouth. The activation of the odour channels was adjusted to the olfactometer's mean latency (447.5 ms for onset, 608.5 ms for offset) for constant and maximum intensity of odour delivery. Additionally, one channel containing a

non-odorant cotton pad was activated five times for comparison with startle responses to the three odours.

In line with our previous study [15], we did not implement a breathing belt, as participants comply well with breathing instructions, producing reliable respiration patterns [43,44]. Additionally, a countdown from 3 to 0 served as an instruction for the participants to inhale when 0 is displayed, which was synchronised with the olfactometer's latency to the point in time of odour onset. Pseudo-randomly, each odour was presented seven times, never twice in a row.

2.4.3.2. Pictures. Visual stimuli have proven to influence the startle response, thus pictures served as variable for comparison. Photographs of the committee members' faces and unfamiliar faces were included for comparison of modality effects in relation to stress exposure. The committee pictures showed their faces only, while other features such as hair and background were masked. These pictures were presented four times each, as they were limited to the two faces of the committee members. Furthermore, 6 positive (landscapes) and 6 negative (attacks) pictures of the International Affective Picture System (IAPS) [45] were matched for arousal within each category. Their main purpose was to provide values for comparison to guarantee validity of the startle responses to the odours. Previous studies resulted in robust effects on the fear-potentiated startle response [46,15]. Same as the odours, each picture was only once combined with a startle stimulus, but presented twice. It was a 15″ × 12″ presentation screen with a resolution of 1280 × 1024 pixels and brightness of 100. During the ISI, a 20 × 20 pixels fixation cross was displayed at the centre of the screen. The chair participants were seated in had an approximate 45 cm distance from the screen.

2.4.4. Startle data processing

By a semiautomatic mechanism of the software BrainVision Analyzer 2.0 (Brain Products GmbH, Gilching, Germany) valid startle trials were identified. A time frame ranging from 50 ms to 225 ms from startle onset was selected for peak detection of the startle response. We applied a 50 Hz notch filter and a baseline correction mechanism (0–50 ms). Before peak detection, the data were rectified. The final verification and revision was done manually. Of the overall startle responses, 3.2% were rejected in total due to reactions outside the usual time scope for startle responses (0.0613%) or non-responsiveness (amplitude did not exceed largest baseline amplitude by factor 2; 3.13%) [43].

2.4.5. Data analysis

For each stimulus (three odours, four picture types) and sensory modality (olfactory, visual), mean values were calculated. A repeated-measures ANOVA with within-subjects factors modality (odours, pictures) and between-subjects factors condition (stress vs. control) and sex (male, female) was conducted. It was repeated for the two different modalities separately, with ODOURS (4) × CONDITION (2) × SEX (2) or PICTURES (4) × CONDITION (2) × SEX (2), respectively. SPSS 20 was used for statistical analyses.

3. Results

3.1. Participants

Due to software failures, 2 of the 70 participants were excluded. Further exclusions concerned 3 participants who were non-responding to the startle stimulus and one who expressed an enlarged startle amplitude of more than three standard deviations (SD) from the mean, another 5 due to unusually high baseline cortisol levels (more than three SD from the mean), as well as 4 due to a cortisol decrease in the stress group, and one due technical issues with the electrodes. Of the 54 participants remaining, 26 were in the stress and 28 in the control group. According to self-reports, 10 of the 26 female participants were in their luteal, 8 in their follicular phase and 8 were ovulating. To check for menstrual cycle phase differences between the groups, the distribution of the different cycle phases in female participants were compared using the Chi square test. No significant differences between the groups in menstrual cycle phase were found ($\chi^2(2) = .248$, $p = .884$).

Table 1
Mean (*SD*) negative (NA) and positive affect (PA) scores from the PANAS for stress and control group before (pre) and after (post) the TSST or TSST, respectively.

	Stress	Control
NA pre	12.77 (3.80)	12.14 (1.78)
NA post	18.23 (6.59)	10.68 (0.91)
PA pre	29.50 (4.49)	28.54 (7.00)
PA post	29.38 (5.93)	33.39 (7.54)

3.2. Social interaction anxiety

There were no differences between stress and control group in social interaction anxiety as measured by the SIAS ($F(1,52) = 2.704$, $p > .10$).

3.3. Stress induction

3.3.1. Affect ratings

Participants of the stress group reported lower positive affect (PA) and higher negative affect (NA) compared to the control group after the experimental manipulation, as expected (Table 1). A repeated measures ANOVA was conducted separately for the two affect scales (PA, NA). Participants of the stress and the control group did not differ in their affect ratings before the experimental manipulation (NA: $t(52) = -.785$, $p = .436$; PA: $t(52) = -.597$, $p = .553$).

A significant within-subjects effect of TIME ($F(1,50) = 8.918$, $p = .004$), a significant interaction of TIME × STRESS ($F(1,50) = 26.490$, $p < .001$), with lower NA post compared to pre scores in the control and the opposite pattern in the stress group, and a significant main effect of STRESS ($F(1,50) = 25.743$, $p < .001$) for the NA was found. Participants of the stress group had significantly higher scores in the post assessments of NA than the control group ($t(52) = -6.005$, $p < .001$).

For PA, we found a significant within-subjects effect of TIME ($F(1,50) = 12.712$, $p = .001$). Further, a significant TIME × STRESS interaction was revealed ($F(1,50) = 14.583$, $p < .001$). In the post assessment, control participants expressed a significantly higher PA than the stress participants ($t(52) = 2.160$, $p = .035$).

3.3.2. Alpha amylase

The sAA data lacked normal distribution, thus they were log-transformed. Since a violation of sphericity was shown by Mauchly's Test ($\chi^2(5) = 6.178$, $p < .001$), Greenhouse Geisser corrected p-values ($\varepsilon = .789$) are reported. A significant within subject effect of TIME ($F(2.37,104.1) = 40.456$, $p < .001$) was found, with a peak of sAA release one minute after the intervention, declining steadily afterwards in both groups (Fig. 1A). No significant main effect of STRESS ($F(1,44) = .024$, $p = .878$) and no TIME × STRESS interaction ($F(2.37,104.1) = 1.188$, $p = .317$) were found, indicating a similar time course of sAA release in participants of both groups. Additionally, a significant between-subjects STRESS × SEX interaction was shown ($F(1,44) = 4.126$, $p = .048$), as in females, the sAA level of the control participants was slightly above the sAA level of the stress participants, while in males it was the other way round.

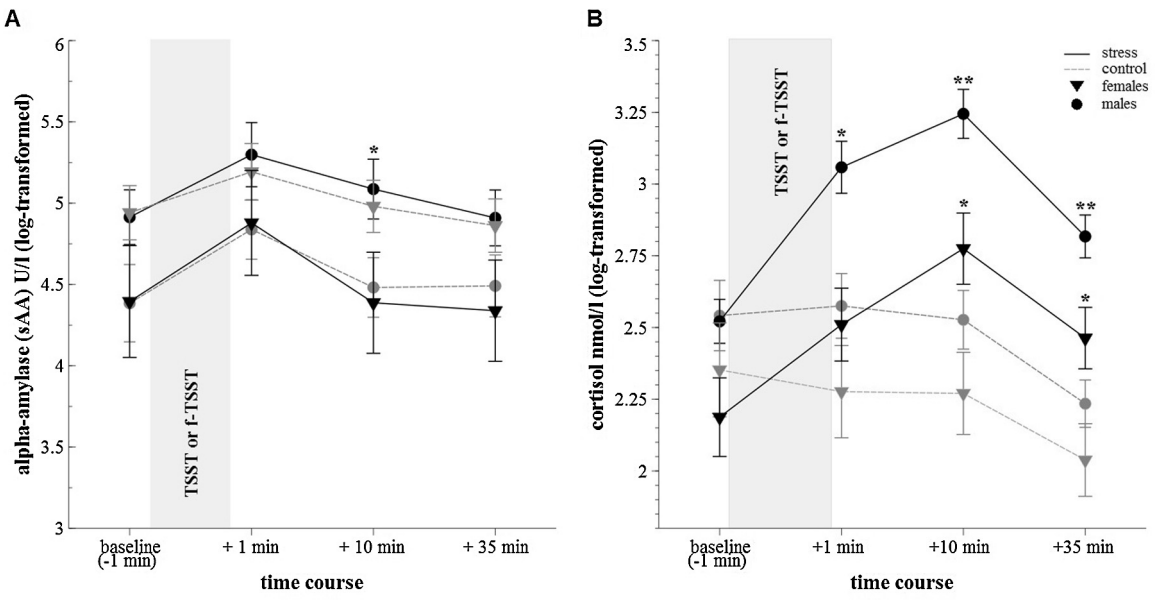

Fig. 1. (A) Mean α-amylase (sAA), (B) mean cortisol responses in the stress and control group at the different time points of assessment. *Differences between stress and control group significant at a .05 level. **Differences between stress and control group significant at a .01 level.

3.3.3. Cortisol

Since the cortisol data lacked normal distribution, they were log-transformed. Mauchly's Test revealed a violation of sphericity ($\chi^2(5) = 44.269$, $p < .001$), hence Greenhouse Geisser corrected p-values ($\varepsilon = .645$) are reported. The ANOVA showed that stress induction was successful, with participants of the stress group expressing a rise in cortisol concentration, reflected in a significant TIME × STRESS interaction ($F(1.94,85.13) = 33.129$, $p < .001$). As expected, salivary cortisol levels at time points +1 ($t(51) = -2.666$, $p = .010$), +10 ($t(50) = -4.938$, $p < .001$), and +35 ($t(51) = -4.919$, $p < .001$) differed significantly between stress and control group, with maximum difference occurring at time point +10 (Fig. 1B). Further, a significant main within-subjects effect of TIME ($F(1.94,85.13) = 29.297$, $p < .001$) and significant main between-subjects effects of STRESS ($F(1,44) = 7.979$, $p = .007$) as well as SEX ($F(1,44) = 8.512$, $p = .006$) were detected, with women displaying generally slightly lower cortisol levels than men.

3.4. Memory performance

3.4.1. Free recall

Results of the free recall task showed a significant STRESS × OBJECT interaction ($F(1,50) = 4.937$, $p = .031$, $\eta^2 = .093$) and main effect of STRESS ($F(1,50) = 9.265$, $p = .004$, $\eta^2 = .156$), as participants of the stress group demonstrated a generally better memory performance than those of the control group, which was especially pronounced for central objects (Fig. 2A). Further, we found a significant within-subjects effect of OBJECT TYPE ($F(1,50) = 121.082$, $p < .001$, $\eta^2 = .708$), with better performance for central compared to peripheral objects in both groups.

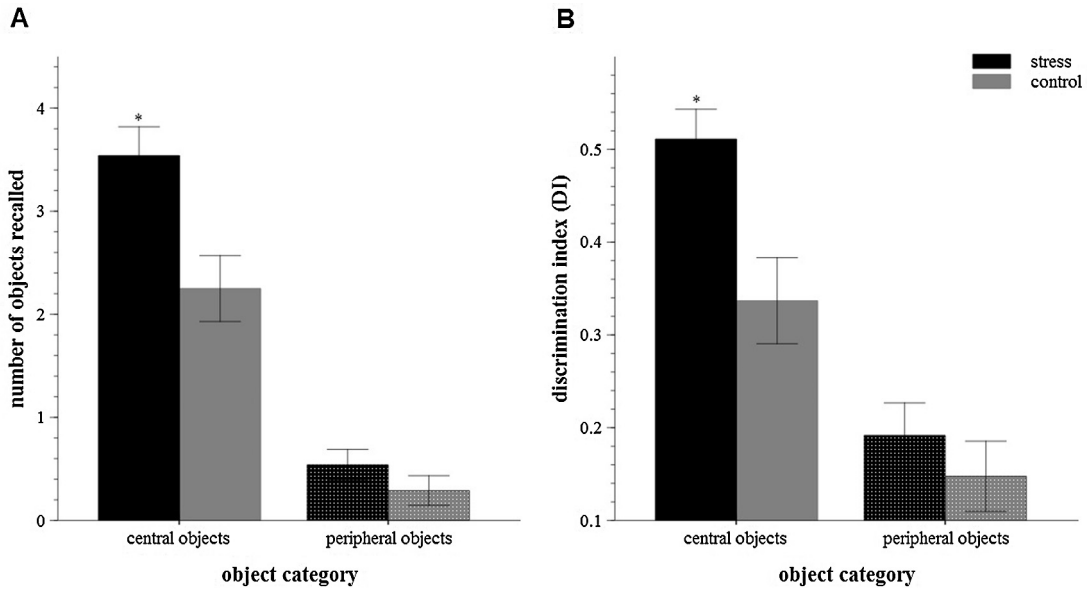

Fig. 2. (A) Mean number of central and peripheral objects freely recalled, (B) discrimination index for both object categories; *$p < .01$ compared to the control condition.

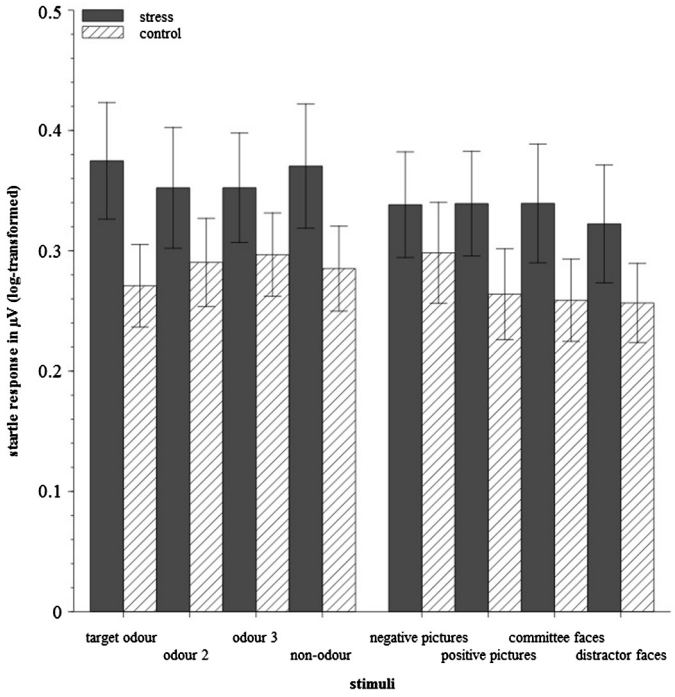

Fig. 3. Mean startle responses to odours and pictures for stress and control group.

3.4.2. Discrimination index

For the DI we again found a significant main effect of STRESS, revealing a better memory performance of stressed participants in the object recognition task compared to control participants ($F(1,48) = 5.193$, $p = .027$, $\eta^2 = .098$). A significant STRESS × OBJECT TYPE interaction ($F(1,48) = 5.532$, $p = .023$, $\eta^2 = .103$) showed that this effect was especially pronounced for central compared to peripheral objects (Fig. 2B). Again, in both groups memory for central objects was significantly better than for peripheral objects, reflected by a significant effect of OBJECT TYPE ($F(1,48) = 84.507$, $p < .001$, $\eta^2 = .638$).

3.5. Startle responses

As the startle data were not normally distributed, they were log-transformed for further analyses. Since Mauchly's Test resulted in a violation of sphericity ($\chi^2(27) = 53.600$, $p = .002$), Greenhouse–Geisser corrected p-values ($\varepsilon = .785$) are reported. An ANOVA including the four factors MODALITY (odours, pictures), STIMULUS (4 odours, 4 pictures), STRESS (stress, control), and SEX (male, female) resulted in a significant main within-subjects effect of MODALITY ($F(1,50) = 8.305$, $p = .006$) with an overall higher startle responsivity for odours in comparison to pictures ($M = .323$, $SD = .21$; $M = .300$, $SD = .21$). No significant main effect of STRESS was found ($F(1,50) = 1.682$, $p = .201$), and no STRESS × STIMULUS interaction ($F(5.49,274.66) = 1.209$, $p = .303$). A threefold MODALITY × STIMULUS × STRESS interaction tended to become significant ($F(3,150) = 2.413$, $p = .069$). An independent-samples t-test comparing startle responses to the different odours between the groups revealed a trend towards higher startle responsivity in the stress group for the target odour ($t(52) = -1.768$, $p = .083$) (Fig. 3).

3.6. Subjective odour ratings and odour recognition

Odour recognition data resulted in 53.8% (14 out of 26) of the participants in the stress group identifying the target odour out of 6 odours in the forced choice trial, but only 25% (7 out of 28) of the control participants. This descriptive difference was not significant ($\chi^2(1) = 2.645$, $p = .104$).

In order to test for differences in affective quality of the target odour between stress and control group, the subjective ratings of the three odours were compared in a repeated-measures ANOVA with the factors STRESS × SEX × ODOUR. No significant STRESS × ODOUR interaction ($F(3,150) = .372$, $p = .773$), nor significant main effect of STRESS ($F(1,50) = .255$, $p = .616$) occurred.

4. Discussion

We hypothesised increased vigilance in stressed participants to cause rapid effects of memory enhancement for the objects involved in the stressful episode after a short delay, in particular regarding central items. We further aimed at demonstrating acute startle response specificity to be enhanced in stressed participants when re-exposed to an odour ambient during the stressful episode. In accordance with this, we predicted a better memory for this odour in stressed compared to control participants. The results of the study are being compared to our previous results showing an enhancement of object memory [47] and startle responsivity, 24 h after stress exposure [6,15,36].

Cortisol results as well as affect ratings show a successful stress induction. Both cortisol response and negative affect are significantly increased in the stress compared to the control group. The increase in positive affect in the control group after the f-TSST can be attributed to insecurity on whether being assigned to the stress condition, diminishing the positive affect before while increasing it during the control condition. Moreover, the social interaction during the f-TSST is likely to contribute to an increase in positive affect, as shown in a similar study in our department [47]. The time course of the physiological stress response in terms of cortisol and sAA is of the same characteristics as we have previously shown with this paradigm [14,48].

The memory results for the objects show that the effects we have found 24 h after stress induction in our previous studies [6,36] are already present in the immediate aftermath of the stress experience. Thus, they seem not to be primarily dependent on consolidation processes. Interactions of glucocorticoids with the noradrenergic system of the basolateral amygdala (BLA) cause the enhancing effects of stress on memory consolidation [49]. However, rapidly proceeding non-genomic effects on immediate attentional and mnemonic processes during the acute stress phase [3] are apparently crucial for the enhanced memory performance observed in our paradigm. Activation of the basolateral amygdala during memory encoding plays a pivotal role in modification of memory contents under stress [2,27]. Being equipped with corticosteroid receptors, the amygdala additionally is directly influenced by stress [50] and emotional information is thus privileged to be encoded [51–53]. Hereby, noradrenaline, its action on amygdala and hippocampus, and the interaction with glucocorticoids create an optimal state for neuronal plasticity [12], such that the encoded objects can be optimally stored. Particularly for highly arousing and stressor-related stimuli, item-emotion bindings are encoded [54,55]. This requires attentional selectivity which is enhanced with stress onset [56]. Attention is thus drawn towards potentially relevant, stress-related items [57], and rapid memory effects operate. This seems to be the main effect our findings are based on, showing significant short-term effects of acute stress on memory. The rapid effects observed in the current study might conceivably be further enhanced by consolidation processes stabilising the most important – mainly dependent on emotional aspects – memory contents [58]. Our results are in line with findings of increased vigilance under stress leading to a vantage point in processing stressor-related information of relevance [1,59]. Similar to previous studies

[6,54,60,61], the effect of enhanced memory in stressed participants was apparent particularly for central compared to peripheral objects. During a stressful experience, attention and memory for stimuli which might become of relevance for similar situations in the future is particularly promoted [3]. Objects that are moved and being used are likely bound to the particular situation which potentially reoccurs in the future and are thus remembered better. Additionally, the initially neutral office items gained emotional valence by being associated with the main stressor, the committee. A previous study has already shown that, by association with an emotionally laden context, memory for initially neutral elements is promoted [62]. Besides, cortisol increases the item-context binding [63] and thus strengthens the association of the specific central item with the stressful context. These mechanisms may have contributed to memory enhancement for central items in our studies.

Our startle data descriptively show enhanced startle amplitudes in stressed compared to control participants, but the difference between the groups is not significant. In contrast, one day after the stressor startle responses in stress and control group differ significantly, as shown in our previous study [15]. However, a more pronounced startle response in the control group in the direct aftermath of the social interaction during the f-TSST leads to less prominent group differences. As already shown in similar studies featuring the f-TSST as a control condition [6,14], we found an increase of sAA in both stress and control group. Apparently, the SNS was similarly activated in stressed and control participants, presumably due to the social interaction enhancing arousal. The startle response has been shown to be highly sensitive to arousal [18], which might have caused enhanced startle responsivity immediately after the control procedure in our current study. In contrast, in our previous study, SNS activity on the second day had ceased, causing more pronounced differences between stress and control group.

The hypothesis that participants stressed would show enhanced response specificity to the odour ambient during the stressful episode could not be confirmed. Instead, they exhibit only a trend towards a stronger startle response to the target odour compared to the control participants. This non-significant finding might reflect a lack of power of the current study. The startle results have to be considered preliminary and are in need of replication.

In contrast to our previous finding (after a 24 h delay) of enhanced responsivity being accompanied by lack of specificity [15], the results show a trend to enhanced specificity as well as enhanced general responsivity. The closer proximity to odour exposure might mediate this relation, that is the odour had only recently been experienced such that implicit as well as explicit memory for it is stronger (as discussed in short), leading to a slightly more pronounced responsivity to the target odour.

In our previous study, participants had been wearing the oxygen mask for odour delivery during the olfactory startle block only [15]. To rule out the possibility of the oxygen mask having been responsible for enhanced startle responsivity to odours in contrast to pictures, in the current study participants were wearing the oxygen mask during both odour and picture presentation. The startle response to olfactory cues was still significantly stronger than the response to visual cues, in line with a previous study comparing these two modalities [43]. The strong connectivity of the olfactory system with the cortical nucleus of the amygdala might be responsible for that [64]. Rapid activation of areas in the amygdala by odours might have caused increased vigilance for an amygdaloidal response cascade initiated by the loud noise, leading to more pronounced startle responsivity to odours than to pictures.

The target odour was correctly identified by 53.8% of the stressed, but only 25% of control participants in comparison to 10% of correct identifications in both groups one day later, in our former study [15]. We found a descriptive trend towards a difference between stress and control group, whereas in the former study an equally small amount of participants (two) correctly identified the target odour in each group. Whereas acute stress effects seem to act in favour of an increased memory for the ambient odour, shown by the descriptive difference between the groups, the affective rating of this odour remains uninfluenced. However, 24 h later, explicit memory for this odour apparently declines while the affective response to it seems to have consolidated, causing a more aversive rating [15]. These dissociative effects might reflect different forgetting rates of explicit and implicit memory contents. Evidence shows that explicit and implicit memory processes can act independently of each other [65–68]. It is thus conceivable that explicit memory decreases more rapidly than implicit memory, represented in the more aversive rating of an odour which is not anymore consciously associated with the stressful experience on the previous day.

As already stated above, the startle and olfactory memory results should, however, be interpreted with caution since the group differences are based on trends and thus are in need of replication. Even more so due to the relatively large amount of exclusions reducing the sample to a size probably not big enough to detect mean stress effects on startle responsivity and/or specificity.

Taken together, our results show that enhancing stress effects on memory are detectable in the immediate aftermath of psychosocial stress, before consolidation processes then transfer salient enough stimuli into long-term memory. Furthermore, acute psychosocial stress enhances the auditory fear-potentiated startle response at a trend level, in particular in response to the odour ambient during the stressful episode. Compared to previous results of our two-days study [15], this effect seems to slightly increase over a time-course of 24 h after stress experience. In contrast, startle response specificity seems to decrease within the same time span.

Acknowledgements

We thank Tobias Otto for his immense help and technical support before and during this study. Furthermore, we are grateful to all student assistants involved in testing, especially as committee members of the TSST/f-TSST.

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