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Research report

Stress improves task processing efficiency in dual-tasks

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HIGHLIGHTS

- Dual-task performance becomes better under stress.
- Improvements emerge as a consequence of increased processing efficiency.
- Improvements are predictable on the basis of salivary cortisol concentrations.

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ABSTRACT

Psychological stress has attracted much interest as a potential modulator of response control processes. However, especially in dual-task situations, the effect of psychological stress is less understood. In the current study we investigated these effects. "Thirty six" healthy young male participants were exposed to stress applying the socially evaluated cold pressor task (SECPT) or a control condition. Afterwards they participated in a psychological refractory period (PRP) paradigm comprising two tasks (a "tone task" and a "letter task"). With the PRP task, four different stimulus onset asynchronies (SOA) were realized separating the tone from the letter task. The results show that stress improves task processing efficiency in dual-tasks. Stressed participants showed a reduced PRP effect (i.e., shorter response times), which was especially prominent in the short SOAs conditions (16 and 133 ms). The analysis of the response times suggests that stress increases dual-tasking performance by modulating the efficiency to process the different tasks and not because 'cognitive flexibility' and switching between task components at the bottleneck is altered. Increases in processing efficiency in dual-tasks were predictable by means of individual salivary cortisol levels.

1. Introduction

Response selection and control processes play a pivotal role in daily life and have been examined in relation to a plenty of factors. Out of these, psychological stress has attracted interest as a potential modulator of response control processes, but the results are contradictive: some results suggest that stress improves selective attention processes, necessary for efficient unfolding of response control functions [1] and it has also been shown that stress facilitates performance in task-switching and stroop paradigms [2]. However, with respect to task switching other findings account for opposite effects, i.e., task-switching is impaired under stress [3,4]. These results are well in line with findings showing that stress increases shielding of action goals and thereby leads to reduced cognitive flexibility [5].

However, only recently, the effects of stress have been examined in relation to multi- and dual-task performance. While stress-induced increases in task shielding are evident in single-tasks, it has been shown that acute stress leads to reduced shielding of task goals, when one task is prioritized over the other in dual-task situations [6]. However, current evidence [6] on the effects of stress on dual-task processing focused on processes related to a priorization of the first task. Hence, little is known about the effects of stress on the succeeding task, when both tasks are of equal importance. However, this is of importance, since especially task 2 (T2) processing is subject to processing restrictions when the two tasks in two different streams of information are processed simultaneously or quasi-simultaneously [7]. This phenomenon is known as the psychological refractory period (PRP) effect [8,9]. The psychological refractory period (PRP) paradigm is a classical paradigm to examine dual-task interference [10,11]. In this paradigm two tasks are presented in close succession and participants are asked to respond as quickly as possible to each task. The typical finding is that responses on the second task are slower when the second task was presented shortly after the first task (=PRP effect) [8,9]. With increasing time (stimulus onset asynchrony, SOA) between the

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tasks, the interruption of T2 processing (i.e., the PRP) that becomes smaller [10]. Even though the precise nature of this processing limitation is still open to debate [10–13] it is widely agreed that the limitation is due to processing limitations at the response selection stage [14]. As flexible behavioural adaption have long been suggested to promote dual-tasking performance [8,15] it may be hypothesized that stress impairs dual-tasking abilities due to its known negative effect on cognitive flexibility [3,5].

However, psychological stress has been suggested to affect catecholaminergic signalling [16] and dopamine D2 receptor related neural transmission in particular [e.g., 17,18]. Response control processes, as examined using the PRP, have been shown to depend on dopaminergic neural transmission and fronto-striatal networks [e.g., 19–21]. Very recent results indicate that dual-task performance is rendered more efficient under conditions, where punishment feedback is provided in case of slow reaction times [22]. Yildiz et al. [22] showed that reaction times on the second task are faster, when punishment is provided in case of slow reactions. Opposed to this they showed that rewards were not able to speed up responses on the second task, but rather led to a slowing of responses. As reward and punishments are mediated via different dopaminergic receptors, these results suggest that dual-tasking is differentially modulated by dopaminergic subsystems.

The effects of punishments on dual-tasking are of relevance for the modulator "stress". Punishments have been shown to be mediated via the dopamine D2 receptor system and hence a receptor system that plays an important role in the mediation of stress effects on cognitive functions [16–18]. Against this background it is therefore more likely that stress increases dual-tasking performance, i.e., leads to faster reaction times on task two in short SOAs. However, the recent study by Yildiz et al. [22] suggest that modulatory effects were only evident in an experimental condition, where the task order was unpredictable. In the current study we therefore examine the effect of stress in two blocks, where task order is either predictable or unpredictable. If the effects of stress are similar to the effects of punishments [cf. 22] we expect that stress modulated reaction times in short SOAs in the unpredictable task block, only.

2. Materials and methods

2.1. Participants

A sample of 36 healthy, right-handed male participants were recruited and randomly assigned to the experimental ($N = 18$) and the control group ($N = 18$). Participants had normal or corrected-to-normal vision. The participants received course credits or financial compensation for their participation. The study was approved by the Ethics committee of the Ruhr-University of Bochum. Each subject gave written informed consent in addition that experiments were carried out in accordance with the Declaration of Helsinki. Smokers were excluded from participation because nicotine changes the neuroendocrine stress response [23].

2.2. Induction and quantification of stress

Participants in the stress condition ($N = 18$) were exposed to the socially evaluated cold pressor test (SECPT) [cf. 24]. Briefly, they put their left or right foot for 3 min (or until they could no longer tolerate it) into ice water (0–2 °C). Deviating from the usual SECPT protocol we did not use the hand in order to avoid that manual response times (RTs) are unaffected. During this phase, they were videotaped and monitored by an unfamiliar person. Participants in the control condition put their foot into warm water (35–37 °C) for 3 min. They were neither videotaped nor monitored by an unfamiliar person. To assess whether the stress induction by the SECPT was successful, subjective stress ratings, blood pressure, and salivary cortisol were measured: Immediately after the SECPT or control condition, subjects indicated on a scale from 0 ("not at all") to 100 ("very much") how stressful, painful, and unpleasant they had experienced the previous situation. Blood pressure and pulse frequency was measured 5 min before, during, and again for 5, 20 and 50 min after the stress or control condition with the cuff placed on the left upper arm. Participants collected saliva samples before as well as 5, 20, and 50 min after the SECPT or control condition with a Salivette collection device (Sarstedt, Nuembrecht, Germany). Saliva samples were kept at −20 °C until analysis. Free cortisol concentrations were measured using an immunoassay (IBL, Hamburg, Germany). Interassay and intra-assay coefficients of variance were below 10%.

2.3. Experimental paradigm

We used a PRP paradigm that is identical to the paradigm used in a previous study by our group investigating the effects of rewards on dual-task performance [22]. The paradigm comprised of two tasks: a "tone task" (task 1) and a "letter task" (task 2). In the "letter task", white letters ("H" or "O"; 1.8° × 2.3° visual angle) are presented on a dark blue screen and subjects had to indicate, whether an "H", or and "O" was presented on the screen (task 2). In the "tone task", sine wave tones were presented with a pitch of 300 or 900 Hz (task 1). Each stimulus lasted for 200 ms. Each trial consists of both of these tasks and begins with the presentation of a central fixation cross at the centre of the screen. After one second the stimulus S1 (tone) was presented, followed by the presentation of the S2 stimulus (letter) in a predefined stimulus onset asynchrony (SOA) of either 16, 133, 500 or 1000 ms. Participants responded with their left hands to the tone stimulus and with their right hands to the letter stimulus and subjects were instructed to place equal emphasis on both tasks. For the tone stimulus, the button underlying the left middle finger had to be pressed for low tones (300 Hz) and the button underlying the left index finger had to be pressed for the high tone (900 Hz). For the letters, subjects pressed with their right index finger on an "H" and with their right middle finger for an "O" [cf. 13]. Participants had to respond as quickly and accurately as possible to each stimulus and were told to place equal emphasis on both tasks.

Subjects were required to respond to the second stimulus within 2000 ms. Trials exceeding this deadline were defined as misses. In case of misses the next trial was started within 1500 ms jittered between 500 and 2500 ms. For trials, in which responses were given within 2000 ms, the next trial was started after a response-stimulus interval (RSI) jittering between 1000 and 4000 ms. In one block of the PRP task the letter stimulus always follows the tone stimulus. In another experimental block there was no fixed, but a random order of the T1 and the T2 stimulus; i.e., it was impossible to predict, which of the two tasks comes first, and which comes second. There were two fixed and two random blocks presented in counterbalanced order across participants (ABAB or BABA). Each block consisted of 320 trials, summing to 1280 trials for the whole experiment.

For the RT data analysis across SOAs the data was screened for trials in which the difference in RT between task 1 and task 2 was 100 ms or less, to account for possible effects of 'response grouping'. Subjects were requested to respond first to the first stimulus appearing (irrespective of the task). This means that in the random task order RT1 comprises responses to tones and letters. The same is true for T2. For the statistical analysis of the data, data was not grouped with respect to the modality of the stimulus, but for its occurrence (i.e., first or second stimulus). Hence, data was pooled across the different modalities in the random block. In the statistical analysis the modality of the T1 and T2 stimulus is therefore discarded in the random block. This procedure is reasonable, since the PRP effect is determined by the temporal order and proximity of stimuli.

2.4. Statistical analysis

Behavioural data were analyzed using mixed effects ANOVAs. Greenhouse–Geisser correction was applied where necessary and post hoc tests were Bonferroni-corrected. Before testing, Kolmogorov–Smirnov tests were carried to test normal distribution. All variables included were normal distributed ($p > .4$).

3. Results

3.1. Physiological effects of stress induction

Salivary cortisol levels were analyzed in a mixed effects ANOVA with time point of salivary cortisol probe sampling at within-subject factor and "stress" as between-subject factor. The ANOVA revealed an interaction "time point × stress" ($F(3, 105) = 5.2$; $p = .002$; $\eta^2 = .931$). Subsequent bonferroni-corrected post hoc t-tests revealed that there was no difference in salivary cortisol concentrations prior to stress induction ($t(35) = 0.26$; $p > .45$). At all other time points, the salivary cortisol levels were higher in the stressed group, compared to the control group (all $t(35) = -2.14$; $p = .014$) (refer Fig. 1A) ($p < .001$).

As peripheral physiological measures systolic and diastolic blood pressure (refer Fig. 1B) as well as pulse frequency (refer Fig. 1C) was analyzed. The mixed effects ANOVA on the systolic blood pressure there was an interaction "time point × stress" ($F(3, 140) = 3.01$; $p = .20$; $\eta^2 = .079$). The same was found for the diastolic blood pressure ($F(3, 140) = 12.26$; $p < .001$; $\eta^2 = .259$). For the diastolic blood pressure, bonferroni-corrected post hoc t-tests revealed differences between the stressed and the control group at the time point of stressing ($t(35) = -2.43$; $p = .01$), but not at the

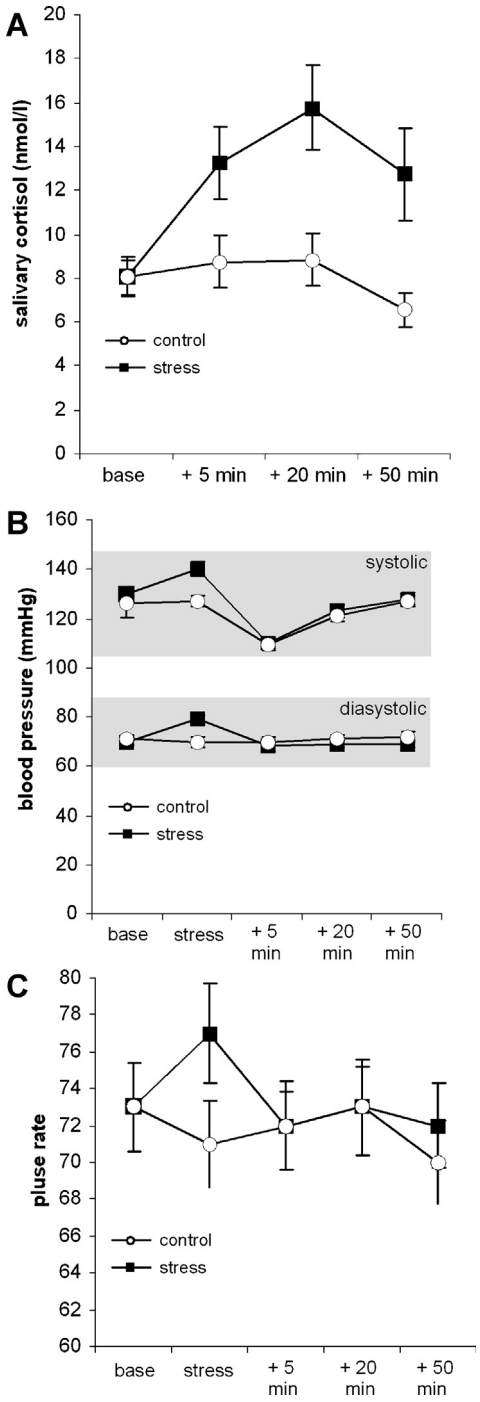

Fig. 1. (A) Salivary cortisol level concentrations (nmol/l) the stressed and the control group before, and *at* various time points after stress induction (5, 20 and 50 min). (B) Systolic and diastolic blood pressure in the stressed and the control group prior stressing, during stress exposure and after stress exposure (5, 20 and 50 min). (C) Pulse rate in controls and the stressed *group* prior, during and after induction of stress (5, 20 and 50 min).

other time points (all $t(35) < .1.19$; $p > .15$). The same was evident for the systolic blood pressure, which was higher in the stressed group at the time point of stressing ($t(35) = -3.18$; $p = .003$, but at all other time points ($t(35) = -.51$; $p > .3$). When analyzing the pulse rate and interaction "time point × stress" was also evident ($F(4, 140) = 7.24$; $p < .001$; $\eta^2 = .171$). Here, the stressed group revealed an increase in pulse rate during stress exposure ($F(4, 72) = 6.63$; $p < .001$; $\eta^2 = .269$), no changes were observed in the control group.

3.2. Effects of stress on dual-task performance

The reaction time (RT in ms) data on for T1 and T2 responses are given in Fig. 2. Data across the fixed and the random block is given in Fig. 2A, data separated for the fixed and the random block is given in Fig. 2B and C, respectively.

Analyzing the T2 responses in a mixed ANOVAs including "block (fixed vs. random order)", "SOA length" as within-subject factors and "stress" as between subject factor, revealed a main effect "SOA length" ($F(3, 102) = 459.20$; $p < .001$; $\eta^2 = .931$), showing a usual PRP-effect in which RTs decreased with increasing SOA length. There was a difference in RTs between all SOAs, as indicated by Bonferroni-corrected pair-wise comparisons ($p < .001$). Importantly, there was an interaction "SOA length × stress" ($F(3, 102) = 7.03$; $p = .002$; $\eta^2 = .171$) (Fig. 2), which was not further modulated by the factor "block" ($F(3, 102) = 0.83$; $p > .4$; $\eta^2 = .024$). Bonferroni-corrected post hoc tests comparing RTs at each SOA across groups revealed that there was a trend towards a shorter RTs in the stressed group, compared to controls at SOA 1000 ($t(23.53) = 1.35$; $p = .120$) and SOA 500 ($t(22.21) = 1.68$; $p = .06$), but there were differences with strong effect sizes at SOA 133 ($t(21.48) = 2.29$; $p = .014$; $d = 0.79$) and SOA 16 ($t(24.76) = 3.43$; $p = .001$; $d = 1.18$). The main effect "stress" indicated that RTs in the stressed group are shorter, compared to the control group ($F(1, 34) = 5.71$; $p = .022$; $\eta^2 = .141$). There was a main effect "block" ($F(1, 34) = 105.75$; $p < .001$; $\eta^2 = .757$) showing that RTs were shorter in the fixed (509 ± 11), compared to the random block (621 ± 17). SOA length modulated this effect ($F(3, 102) = 21.42$; $p < .001$; $\eta^2 = .387$) showing that the difference in RTs between the random and the fixed block was stronger at shorter SOA, compared to longer SOAs. All other interaction effects were not significant (all $F < 0.31$; $p > .5$). Concerning RTs on the T1 stimulus, there was necessarily a main effect of "block" ($F(1, 34) = 25.50$; $p < .001$; $\eta^2 = .438$) showing that RTs on the T1 stimulus were shorter in the fixed (555 ± 12), compared to the random block (628 ± 17). There were no further main, or interaction effects (all $F < 1.2$; $p > .2$) (refer Fig. 2). However, it may be argued that parts of the interaction observed for the RT2s (i.e., RT2 reduction at short SOAs) are due to an RT1 speeding effect [25]. To account for this we subjected the mean reaction time on the T1 stimulus as a covariate in the mixed effects ANOVA. This did not change the model: The interaction "SOA length × stress" was still significant ($F(3, 102) = 5.33$; $p = .003$; $\eta^2 = .151$) and also the other effects were not changed.

It may further be argued that pooling the data in the random task blocks is not appropriate, since processing of the T1 and T2 tone and letter stimuli may be different and may therefore be also differentially modulated by stress. We therefore repeated the data analysis and used only trials in the random block that were of similar order of tone and letter stimuli as it is the case in the fixed block. The results were identical. There was an interaction SOA length × stress" ($F(3, 102) = 5.55$; $p = .007$; $\eta^2 = .131$) (Fig. 2D), which was again not further modulated by the factor "block" ($F(3, 102) = 1.01$; $p > .2$; $\eta^2 = .009$). Post hoc tests revealed that no differences in RTs in the SOA 1000 and the SOA 500 trials, but there were differences in the SOA 133 ($t(22.46) = 2.21$; $p = .012$) and SOA 16 condition ($t(23.55) = 3.25$; $p = .002$) (refer Fig. 2D).

Previously, Miller et al. [13] suggested that the total reaction time (TRT; TRT = RT1 + RT2) resembles a well-suited measure for dual-task improvement in terms of dual-task efficiency. On the basis of this measure, a mixed effects ANOVA revealed a main effect SOA ($F(3, 102) = 196.47$; $p < .001$; $\eta^2 = .438$), showing that TRT increased from the SOA 1000 (1017 ± 23), to the SOA 500 (1072 ± 27), to the SOA 133 (1217 ± 32) to the SOA 16 condition (1321 ± 25). All conditions differed from each other ($p < .001$). There was also a main effect "block" ($F(1, 34) = 59.98$; $p < .001$; $\eta^2 = .634$) showing that TRT is lower in the fixed (1065 ± 23),

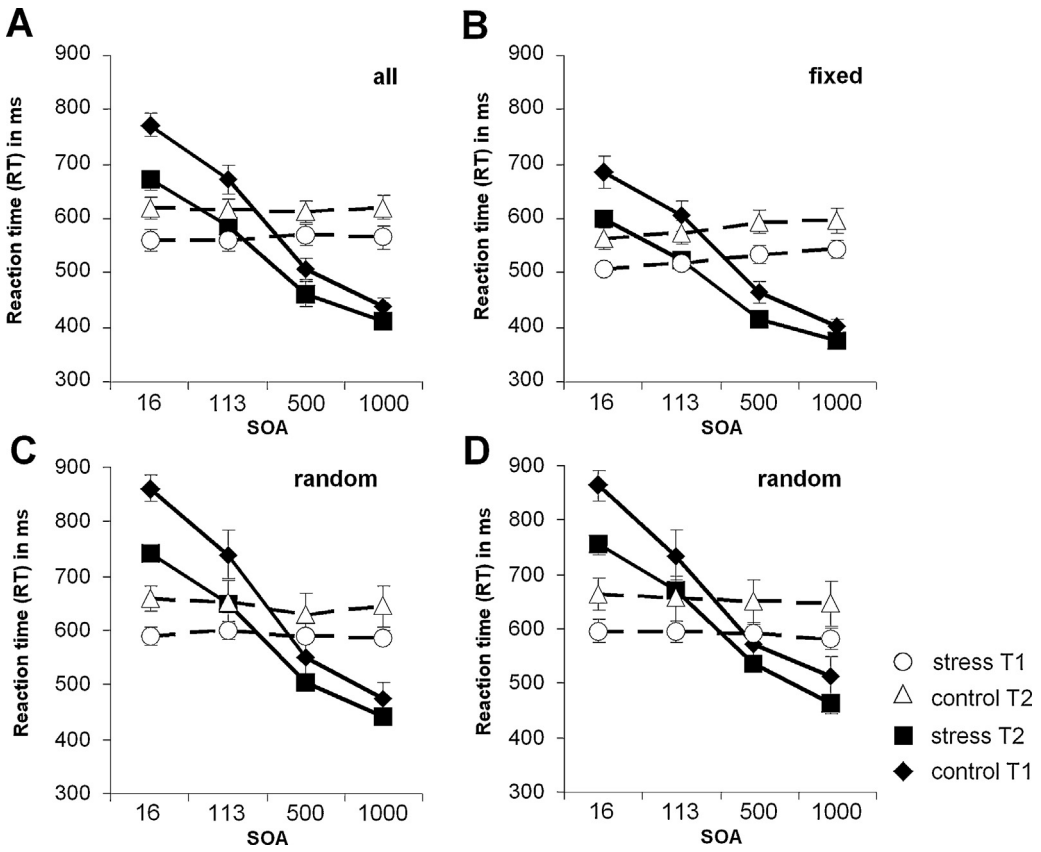

Fig. 2. Mean reaction times (RTs) (±SEM) dependent on SOA length, separated for the stress and control group and the T1 and T2 responses. The dashed, white triangle line denotes T1 responses in the control group; the dashed circle line denotes T1 responses in the stressed group. The solid, black diamond line denotes RTs on T2 in the control group; the solid black squared line denotes RTs on T2 in the stressed group. The plot (A) denotes RTs in T1 and T2 collapsed over the fixed and the random block. The plot (B) denotes these RTs in the fixed block. The plot (C) denotes these RTs in the random block and the plot (D) denotes RTs in the random block only for trials in which the order of the tone and letter stimulus was the same as in the fixed condition.

compared to the random block (1245 ± 33). Moreover, there was a main effect "group" ($F(1,34) = 5.61$; $p = .024$; $\eta^2 = .142$) showing that the stressed group revealed a lower TRT (1095 ± 36, than the control group (1219 ± 36). Importantly, there was an interaction "SOA × group" ($F(3, 102) = 4.30$; $p = .007$; $\eta^2 = .112$). Subsequent post hoc tests revealed no group differences in TRT in the SOA 1000 and SOA 500 condition ($p > .2$). However, for the SOA 133 the TRT was lower in the stressed group (1024 ± 21), compared to the control group (1147 ± 41) ($p = .021$). The same was evident for the TRT in the SOA 16 condition (stress group: 1234 ± 21; control group: 1408 ± 46) ($p = .002$).

The error rates for RT1 and RT2 are given in Table 1.

When analyzing the error rates on the T2 stimulus, there was only a main effect SOA ($F(3, 102) = 12.86$; $p < .001$; $\eta^2 = .275$) showing that error rates were higher in the SOA 133 and SOA 16 condition, compared with the SOA 1000 and the SOA 500 condition ($p < .004$). Between the SOA 16 and 133 conditions, as well as between the SOA 1000 and 500 condition there was no difference in error rates ($p > .5$). Importantly, there were no interactions with the factor "stress" (all $F < 0.46$; $p > .7$). With respect to the shorter RTs in the stressed group, the results are therefore not biased due to a speed-accuracy trade off. Analyzing the error rates on the T1 stimulus did not reveal any significant main of interaction effects (all $F < 0.5$; $p > .6$).

Table 1
Error rates (collapsed across the fixed and the random block) for task 1 and task 2 for the control group and the stressed group separated for each SOA condition.

	SOA	T1	T2
Control group	16	3.6 (0.5)	5.1 (1.5)
	133	3.2 (0.8)	4.9 (0.8)
	500	2.1 (0.5)	3.8 (1.2)
	1000	1.5 (0.3)	3.1 (1.6)
Stress group	16	3.4 (0.7)	6.1 (1.8)
	133	3.1 (0.5)	5.3 (1.9)
	500	2.0 (0.7)	4.8 (1.3)
	1000	1.4 (0.2)	4.7 (1.1)

3.3. Correlational analyses

In order to investigate a potential direct relation between stress-induced reductions in the degree of the PRP effect and markers of the hypothalamus–pituitary–adrenal axis we calculated correlation analyses. For this we first calculated the magnitude of the PRP effect. Doing so, we calculated the difference in RT2s between the SOA 1000 and the SOA 16 condition. Moreover, we calculated the mean salivary cortisol level by averaging salivary cortisol concentration obtained for the time points 5, 20 and 50 min after the SECPT. The correlation analysis shows that there was an inverse correlation of the magnitude of the PRP effect and salivary cortisol level for random ($r = -.482$; $R^2 = 0.23$; $p = .021$) and the fixed block ($r = -.618$; $R^2 = 0.37$; $p = .003$) (refer Fig. 3A).

The analysis on the total response times (TRT) suggest that psychological stress leads to reductions in TRT in the SOA 133 and the SOA 16 condition. Therefore we performed similar regression analyses with the TRT in these conditions. The results are shown in Fig. 3B. The results reveal an inverse correlation between TRT

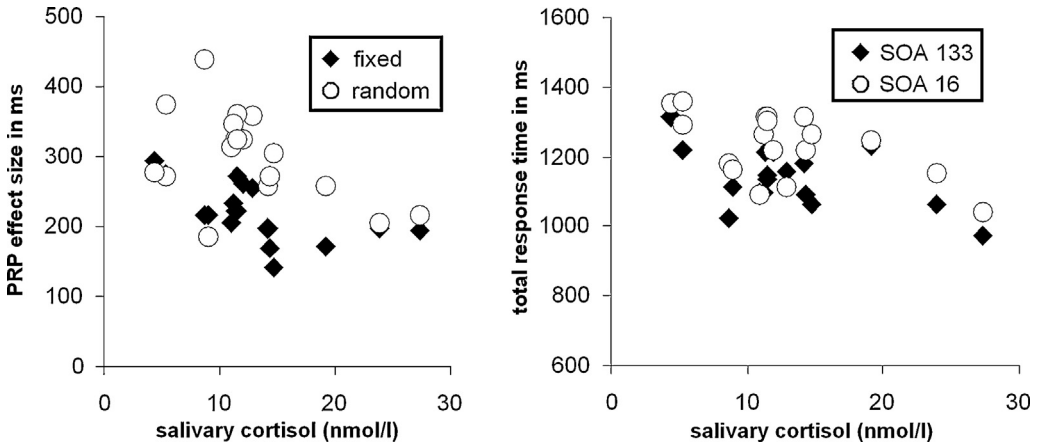

Fig. 3. (A) Scatterplot denoting the correlation between the PRP effect size in ms ($RT2_{SOA1000} - RT2_{SOA16}$) and the salivary cortisol levels in the fixed (black diamonds) and the random block (white circles). (B) Scatterplot denoting the correlation between total response time (TRT) in the SOA 133 and SOA 16 conditions (collapsed across the random and the fixed block) and salivary cortisol levels.

in the SOA 133 ($r = -.596$; $R^2 = 0.35$; $p = .005$) and SOA 16 condition ($r = -.577$; $R^2 = 0.32$; $p = .006$) with salivary cortisol levels (Fig. 3B).

4. Discussion

In the current study we examined the effects of acute psychosocial stress on dual-task performance in healthy male subjects. Only male subjects were enrolled into the study, to avoid complex interactions of cortisol with other hormones. Overall, the results show that acute stress improves performance in the PRP paradigm, a paradigm that examines dual-task interference [7]. In short SOAs, stress leads to reductions in RTs on task 2, compared to controls. In longer SOAs there was virtually no effect of the stress manipulation. There was also a speeding of T1 responses in the stressed group. However, when controlling for this effect the SOA-dependent effect of stress on T2 responses was still evident. Further analyses suggest that the effects of stress on RT2 were still the same, when controlling for the order on the tone and the letter task in the random task block. As the PRP effect is partly determined by the duration of processing in pre-bottleneck and bottleneck stages in T1, these results further underline that the results observed for T2 processing are unbiased with respect to T1 processing.

These results are corroborated by the analysis of the total reaction time (TRT), which has been suggested to reflect a well-suited index of for dual-task improvement in terms of dual-task efficiency [13]. This measure suggests that stress leads to an improvement in dual-task performance in conditions where two tasks are presented in close succession. Exposure to the laboratory stressor increase salivary cortisol concentrations as well as measures of sympathetic nervous system activity (heart rate and blood pressure). There was a linear correlation between stress-induced increases in salivary cortisol levels and reduction of PRP effect size under stress. A limitation of the study is that no single task condition was examined.

Previous studies have shown that stress leads to improvements in attentional selection processes [26,27]. However, the PRP effect has been suggested to reflect processing capacity limitations at the response selection level, and not at the level of stimulus processing [14]. The results therefore reveal a novel aspect of the modulation of stress on cognitive functions.

It has been suggested previously that flexible shifting and switching between responses promotes performance dual-task interference [7,15,28]. In the current experiment these processes were manipulated by introducing a task block where the order of the two tasks was not predictable. The effects of stress were not different for this block. This suggests that stress does not improve dual-task performance by influencing switching between task component processing at the bottleneck [29]. If a switching component, or 'cognitive flexibility' were influenced by stress one would have expected decreases in dual-task performance, since several results indicate that task switching performance is compromised under stress [3,4]. Yet, the opposite was the case. It is more likely that elevated dual-task performance emerges as a consequence of increased processing efficiency. In this regard it has been suggested that total reaction time (TRT; TRT = RT1 + RT2) resembles a well-suited measure for dual-task improvement in terms of dual-task efficiency [cf. 13]. TRT was generally lower in the stressed group than in the control group and especially in the SOA 133 and SOA 16 condition. RTs on T1 were faster under stress, which shows that not only the T2 component, but also the T1 component of the PRP effect is altered. This may indeed suggest that stress increases task component processing efficiency and to a lesser extend dual-task efficiency. However, the data shows that the effects of stress were most pronounced in the short SOA conditions (i.e., SOA 133 and SOA 16 condition). These conditions are the most critical conditions in dual-task processing. Therefore, the data also shows that dual-task processing is altered when demands are high. This is all the more the case, since the effect of stress on RT2 was evident, when controlling for the speeding of RT1. However, it is possible that this may subsequently lead to efficiency increases in dual task performance, when dual-tasking demands are high as it is the case in the SOA 133 and SOA 16 conditions. The results suggest that dual-tasking becomes better, because stress positively affects efficiency to process the different tasks and not because "cognitive flexibility" and switching between task components at the bottleneck is altered.

One can only speculate about the neural mechanisms leading to these effects: The regression analysis shows that task performance (i.e., *PRP effect size and TRT as a measure for processing efficiency*) in the stressed group is predictable on the basis of salivary cortisol levels. Cortisol induces rapid changes in neuronal information processing [30] by acting on receptors at the neuronal membrane [31]. It has been shown previously that increases in dual-task performance similar to those observed in the current study are evident when punishments are provided in case of slow reaction times [22]. Punishments have been shown to exert their effects via the dopamine D2 receptor system [32] in the nucleus accumbens [e.g., 33]. Similarly, stress has been shown to predominantly affect the dopamine D2 receptor system in the nucleus accumbens [17,18],

which is at least partly mediated by activation of glucocorticoid receptors by cortisol [18]. It is therefore possible that the modulatory effects of stress on dual-task processing efficiency may be mediated via the effect of glucocorticoids on dopamine D2 receptors.

In summary, our findings show that dual-tasking performance is enhanced under acute psychosocial stress. The results suggest that elevated dual-task performance emerges as a consequence of increased processing efficacy, but not due to increased switching between task components at the bottleneck. Increases in task performance were predictable on the basis of individual salivary cortisol levels. These effects of stress on dual-task performance may become manifest via modulatory effects of stress (glucocorticoids) on dopamine neurotransmission.

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References

[1] Chajut E, Algom D. Selective attention improves under stress: implications for theories of social cognition. Journal of Personality and Social Psychology 2003;85:231–48.
[2] Kofman O, Meiran N, Greenberg E, Balas M, Cohen H. Enhanced performance on executive functions associated with examination stress: evidence from task-switching and Stroop paradigms. Cognition and Emotion 2006;20:577–95.
[3] Steinhauser M, Maier M, Hübner R. Cognitive control under stress: how stress affects strategies of task-set reconfiguration. Psychological Science 2007;18:540–5.
[4] Plessow F, Kiesel A, Kirschbaum C. The stressed prefrontal cortex and goal-directed behaviour: acute psychosocial stress impairs the flexible implementation of task goals. Experimental Brain Research 2012;216:397–408.
[5] Plessow F, Fischer R, Kirschbaum C, Goschke T. Inflexibility focused under stress: acute psychosocial stress increases shielding of action goals at the expense of reduced cognitive flexibility with increasing time lag to the stressor. Journal of Cognitive Neuroscience 2011;23:3218–27.
[6] Plessow F, Schade S, Kirschbaum C, Fischer R. Better not to deal with two tasks at the same time when stressed? Acute psychosocial stress reduces task shielding in dual-task performance. Cognitive Affective and Behavioral Neuroscience 2012;12:557–70.
[7] Sigman M, Dehaene S. Dynamics of the central bottleneck: dual-task and task uncertainty. PLoS Biology 2006;4:e220.
[8] Pashler HE. Dual-task interference in simple tasks: data and theory. Psychological Bulletin 1994;116:220–44.
[9] The Welford AT. Psychological refractory period and the timing of high-speed performance—a review and a theory. British Journal of Psychology 1952;43:2–19.
[10] Wu C, Liu Y. Queing network modeling of the psychological refractory period (PRP). Psychological Reviews 2008;115:913–54.
[11] Meyer DE, Kieras DE. A computational theory of executive cognitive processes and multiple-task performance: part 1. Basic mechanisms. Psychological Reviews 1997;104:3–65.
[12] Oberauer K, Kliegl R. Simultaneous cognitive operations in working memory after dual-task practice. Journal of Experimental Psychology Human Perception and Performance 2004;30:689–707.
[13] Miller J, Ulrich R, Rolke B. On the optimality of serial and parallel processing in the psychological refractory period paradigm: effects of the distribution of stimulus onset asynchronies. Cognitive Psychology 2009;58:273–310.
[14] Sigman M, Dehaene S. Brain mechanisms of serial and parallel processing during dual-task performance. Journal of Neuroscience 2008;28:7585–98.
[15] Jentzsch I, Leuthold H, Ulrich R. Decomposing sources of response slowing in the PRP paradigm. Journal of Experimental Psychology Human Perception and Performance 2007;33:610–26.
[16] Arnsten AF. Stress signalling pathways that impair prefrontal cortex structure and function. Nature Reviews Neuroscience 2009;10:410–22.
[17] Cabib S, Puglisi-Allegra S. The mesoaccumbens dopamine in coping with stress. Neuroscience and Biobehavioral Reviews 2012;36:79–89.
[18] Pascucci T, Ventura R, Latagliata EC, Cabib S, Puglisi-Allegra S. The medial prefrontal cortex determines the accumbens dopamine response to stress through the opposing influences of norepinephrine and dopamine. Cerebral Cortex 2007;17:2796–804.
[19] Beste C, Ness V, Lukas C, Hoffmann R, Stüwe S, Falkenstein M, et al. Mechanisms mediating parallel action monitoring in fronto-striatal circuits. Neuroimage 2012;62:137–46.
[20] Humphries MD, Stewart RD, Gurney KN. A physiologically plausible model of action selection and oscillatory activity in the basal ganglia. Journal of Neuroscience 2006;26:12921–21242.
[21] Redgrave P, Rodriguez M, Smith Y, Rodriguez-Oroz MC, Lehericy S, Bergman H, et al. Goal-directed and habitual control in the basal ganglia: implications for Parkinson's disease. Nature Reviews Neuroscience 2010;11:760–72.
[22] Yildiz A, Chmielewski W, Beste C. Dual-task performance is differentially modulated by rewards and punishments. Behavioral Brain Research 2013 [in press].
[23] Rohleder N, Kirschbaum C. The hypothalamic–pituitary–adrenal (HPA) axis in habitual smokers. International Journal of Psychophysiology 2006;59:236–43.
[24] Schwabe L, Haddad L, Schachinger H. HPA axis activation by a socially evaluated cold-pressor test. Psychoneuroendocrinology 2008;33:890–5.
[25] Fischer R, Hommel B. Deep thinking increases task-set shielding and reduces shifting flexibility in dual-task performance. Cognition 2012;123:303–7.
[26] Elling L, Schupp H, Bayer J, Bröckelmann AK, Steinberg C, Dobel C, et al. he impact of acute psychosocial stress on magnetencephalographic correlates of emotional attention and exogenous visual attention. PLoS ONE 2012:e:35767.
[27] Schwabe L, Wolf OT. Emotional modulation of the attentional blink: is there an effect of stress. Emotion 2010;10:283–8.
[28] Lien MC, Proctor RW. Stimulus-response compatiblity and psychological refractory period effects: implications for response selection. Psychonomic Bulletin and Reviews 2012;9:212–38.
[29] Band GPH, Jolicœur P, Akyurek EG, Memelink J. Integrative views on dual task costs. European Journal of Cognitive Psychology 2006;18:481–92.
[30] Strelzyk F, Hermes M, Naumann E, Oitzl M, Walter C, Busch HP, et al. Tune it down to live it up? Rapid, nongenomic effects of cortisol on the human brain. Journal of Neuroscience 2012;32:616–25.
[31] Groneweg FL, Karst H, de Kloet ER, Joëls M. Mineralocorticoid and glucocorticoid receptors at the neuronal membrane, regulators of nongenomic corticosteroid signalling. Molecular and Cellular Endocrinology 2012;350:299–309.
[32] Kravitz AV, Tye LD, Kreitzer AC. Distinct roles for direct and indirect pathway striatal neurons in reinforcement. Nature 2012;15:816–8.
[33] Shigemune Y, Tsukiura T, Kambara T, Kawashima R. Remembering with gains and losses: effects of monetary reward and punishment on successful encoding activation of source memories. Cerebral Cortex 2013 [Epub ahead of print].