

The stressed prefrontal cortex and goal-directed behaviour: acute psychosocial stress impairs the flexible implementation of task goals

Franziska Plessow · Andrea Kiesel ·
Clemens Kirschbaum

Received: 20 August 2011 / Accepted: 4 November 2011
© Springer-Verlag 2011

Abstract Goals are often at the basis of human actions. As an essential mechanism of behavioural adaptation, individuals need to be able to flexibly implement new task goals so as to alter their actions (switch tasks) in response to contextual changes. The present study investigated the effect of acute psychosocial stress on cognitive control processes of flexible task-goal implementation with temporal focus on the occurrence interval of the hypothalamus–pituitary–adrenal (HPA) stress response. For this, forty-eight healthy volunteers were either challenged with a standardised stress-induction protocol (the Trier Social Stress Test) or underwent a standardised control situation. Subsequently, they were exposed to a task-switching procedure with two tasks alternating in random order. Participants of the stress group displayed increased salivary α -amylase activity immediately after stress exposure as well as elevations of salivary cortisol from 10 min after stress cessation, reflecting the typical stress-related activity increases in the sympathetic nervous system and the HPA axis, respectively. At the time interval of elevated cortisol levels, stressed individuals persistently showed larger performance differences between task switches and task repetitions (switch costs) than controls. This effect was reliably evident when tested 5–20 min as well as 25–40 min following treatment cessation. These results indicate that acute psychosocial stress impairs cognitive

control processes of flexible task-goal implementation essential for voluntary goal-directed behaviour.

Keywords Executive functions · Cognitive control · Task switching · Acute psychosocial stress · Trier Social Stress Test · Cortisol

Introduction

Human actions typically orient towards a particular goal or intention. Any goal-directed behaviour relies on so-called executive functions or cognitive control processes, that is, a variety of processes that ensure successful goal attainment by incorporating both intentions and context conditions at all times (e.g., Miller and Cohen 2001; Norman and Shallice 1986). First and foremost, goal-directed behaviour requires maintaining and shielding the goal or intention in question. Simultaneously, however, agents need to remain flexible in terms of abandoning a current task goal in favour of a new one whenever significant changes within their external environment or internal context require behavioural adaptation (e.g., Goschke 2000; Mayr and Keele 2000). Thus, the flexible implementation of task goals represents a pivotal process of efficient and successful action control.

Neuroanatomically, task-goal implementation primarily relies on (dorsolateral) prefrontal cortex (PFC) functioning (e.g., MacDonald et al. 2000; Miller and Cohen 2001) embedded in widespread neural networks comprising closest interactions with other frontal cortical areas (e.g., Brass and von Cramon 2002) as well as subcortical brain regions via a prefrontal-basal ganglia-thalamocortical circuit (e.g., Cools et al. 2001). For the experimental investigation of the cognitive control processes of task-goal

F. Plessow (✉) · C. Kirschbaum
Department of Psychology, Technische Universität Dresden,
01062 Dresden, Germany
e-mail: plessow@biopsych.tu-dresden.de

A. Kiesel
Department of Psychology, Julius-Maximilians-Universität
Würzburg, Röntgenring 11, 97070 Würzburg, Germany

implementation, the task-switching paradigm (e.g., Allport et al. 1994; Rogers and Monsell 1995) has been frequently employed in cognitive psychology studies. In a task-switching setting, participants perform two different tasks (i.e., respond to stimuli based on univocal stimulus–response mappings) in an alternating sequence resulting in trials in which the current task matches the previous one (task repetition) and trials in which the current task differs from the former one (task switch). A robust finding is that responses are slower and less accurate when the task switches compared to task repetition. This performance difference is referred to as switch costs and is assumed to reflect cognitive control processes that serve to implement new task goals when required (for a recent review, see Kiesel et al. 2010).

It has been well established that some neurological and psychiatric conditions go along with impairments in flexible task-goal implementation, for example, Parkinson's disease (Cools et al. 2001), attention-deficit/hyperactivity disorder (Cepeda et al. 2000), and anorexia nervosa (Friederich and Herzog 2011). In contrast, within healthy individuals, the integrity of these cognitive control processes has rarely been questioned. Only recent studies suggest that the ability to flexibly implement task goals also depends on the individual's current state since, for example, acute total sleep loss resulted in impaired task switching (Heuer et al. 2004; Bratzke et al. 2009). Even less extreme and more naturalistic sleep disturbances proved to affect task-switching ability. Minor but long-term reductions of nighttime-sleep duration in new parents were found to be associated with significant impairments of task-goal implementation (Plessow et al. 2011b). Furthermore, Dreisbach and Goschke (2004) showed that mild positive affect improves the flexible implementation of task goals. As a conclusion, the ability to flexibly implement task goals may not be unrestrainedly viable under all conditions and thus less reliable than hitherto assumed. Basic research is required to identify the factors affecting the flexible task-goal implementation for two reasons: First, accumulating evidence of modulators will gain a deeper understanding of the addressed cognitive control processes and stimulate theoretical work in the field. Second, links for applied psychological science indicating intervention needs will be provided.

As one of the most important factors affecting daily life, stress has become ubiquitous in both work and private modern life with significant effects on health and psychological functioning. Surprisingly, only few studies have addressed the link between stress and task-goal implementation with rather inconclusive results. Steinhauser et al. (2007) exposed participants to a task-switching procedure immediately after providing them with either low-stress or high-stress intelligence-test tasks. In contrast to

the group that received the low-stress tasks, participants who had previously performed the high-stress tasks did not show the typical reduction of switch costs when sufficient time for endogenous processes of task reconfiguration was available suggesting that the applied stress impaired cognitive control processes underlying flexible task-goal implementation. Kofman et al., on the contrary, tested students before and during an examination period and found reduced switch costs indicating an improvement of cognitive control processes of task-goal implementation during the more stressful examination period (Kofman et al. 2006; but see Liston et al. 2009). Whether these conflicting results are associated with differences in the physiological stress response or not remains elusive, since neither study assessed stress markers.

As a first step towards the inclusion of stress-related changes on the physiological level, the present study added the assessment of the physiological stress response via biological stress markers to the hitherto assessed levels of cognitive performance and subjective stress experience. The present study thus aimed to extend previous research by addressing the effect of acute psychosocial stress on cognitive control processes of task-goal implementation by building both reasoning and methodological implementation upon a biological stress concept. From a biological perspective, an effect of acute stress on PFC-dependent cognitive control processes is quite conceivable. When exposed to stress, two major physiological response pathways are activated. First, stress triggers an immediate increase of sympathetic nervous-system (SNS) activity that is associated with increased catecholamine release. The consequential high levels of catecholamines were repeatedly shown to decrease firing of PFC neurons (e.g., noradrenaline via $\alpha 1$ and $\beta 1$ receptors, cf. Ramos and Arnsten 2007; dopamine via D1 receptors, e.g., Vijayraghavan et al. 2007). This rapid stress response dissolves shortly after stress cessation. Second, a more prolonged stress-induced increase of the hypothalamus–pituitary–adrenal (HPA) axis activity leads to the synthesis and release of glucocorticoids (mainly cortisol) into the bloodstream (e.g., de Kloet et al. 2005). Glucocorticoids pass the blood–brain barrier and bind to mineralocorticoid and glucocorticoid receptors. In addition to their well established involvement in genomic stress effects, there is now accumulating evidence for non-genomic signalling via membrane-localised mineralocorticoid and glucocorticoid receptors (for a review, see Groeneweg et al. 2011). Under normal conditions, glucocorticoids primarily bind to mineralocorticoid receptors. Under conditions of high glucocorticoid levels (e.g., stress) and thus occupied mineralocorticoid receptors, however, glucocorticoids mainly bind to glucocorticoid receptors that are (among other brain regions) evident in large numbers in the PFC (e.g., Perlman et al. 2007; Sanchez et al. 2000). As shown in neuroimaging

studies, stress-induced increased glucocorticoid levels are associated with alterations in prefrontal brain activity (e.g., Qin et al. 2009; Kern et al. 2008).

While SNS-mediated stress response and subjective stress experience (e.g., increased arousal, worse mood) are temporally linked, peak glucocorticoid levels are reached when subjective stress levels are already back to baseline levels (e.g., Plessow et al. 2011a). Because any performance impairment can be considered most critical when the individual concerned is not expecting it (e.g., in the absence of both stressor and subjective stress experience), we focused on the effect of a previously experienced acute stressor on task-goal implementation during the occurrence and continuation of the HPA-stress response after stress cessation.

For the induction of acute psychosocial stress, we applied the Trier Social Stress Test (TSST; Kirschbaum et al. 1993), which proved to be the most powerful way for reliably triggering both major stress responses in human volunteers within a laboratory setting (cf. Dickerson and Kemeny 2004). Half of our participants were challenged with the TSST protocol, whereas the other half underwent a standardised control situation (Het et al. 2009). Stress-induction success was validated by analysing salivary α -amylase (sAA) and salivary cortisol as markers of SNS and HPA-axis activity, respectively (Kirschbaum and Hellhammer 1994; Nater and Rohleder 2009). Subjective stress levels were assessed using a standardised mood questionnaire (Steyer et al. 1997) comprising the three dimensions mood, arousal, and fatigue.

In order to explain stress effects on cognition, many authors claim that stress depletes the available resources. This assumption was proposed for both the cognitive process level where stress-related information processing and coping mechanisms bind resources (e.g., Hockey 1997) and the neuroanatomical level where local brain activity as well as functional connectivity between brain regions was found to be reduced under stress (e.g., Qin et al. 2009; Liston et al. 2009). In a theoretical model, Arnsten (2009) claims that acute and uncontrollable stress induces a shift from controlled top-down processes towards a higher impact of automatic bottom-up processes based on phylogenetically older brain areas. If less resources are available under stress, the ability to flexibly implement task goals will be impaired, particularly considering that these cognitive control processes are highly capacity-demanding (Liefvooghe et al. 2008). Therefore, the resource-depletion account predicts larger switch costs under stress than under non-stressful conditions.

In order to gain a more in-depth understanding of a potential effect of stress on task-goal implementation, we used a task-switching version that allows the simultaneous measurement of a second cognitive control function,

namely stimulus-based interference control and thus examine the impact of stress on that further aspect of cognitive control. For this, we used the same target stimuli and responses in both tasks while stimulus–response mappings partially differed. As a consequence, targets that require the same response in both tasks (congruent targets) were easier to respond to (i.e., faster and more accurately) than targets requiring different responses in the two tasks (incongruent targets). This performance difference is referred to as target-congruency effect (e.g., Meiran 1996) and is interpreted as a measure of stimulus-based interference (and therefore interference control) from the currently irrelevant task on the conductance of the indicated task (cf. Kiesel et al. 2007). Furthermore, we varied the preparation time for the upcoming task by manipulating the time interval between the cue that informed about the task to be performed and the target stimulus (cue-target interval, CTI). This served to examine whether a potential stress influence occurs on all cognitive control processes involved in task-goal implementation or whether the potential stress influence addresses preparation-dependent processes only (e.g., Steinhäuser et al. 2007).

Finally, since we recently observed that acute stress effects on cognitive control processes are closely linked to the HPA-stress response time course (Plessow et al. 2011a), we chose to implement the task-switching procedure in two parts. Thereby, part length was determined by the minimum trial number required for reliable measurement of all manifest variables and their interactions. Between the two parts, neuroendocrine as well as subjective stress measures were assessed. Each part covered a different time interval of the prolonged HPA-stress response, thus allowing to additionally examine the time line of potential stress-related behavioural changes.

Methods

Participants

Forty-eight volunteers (24 male, 18–29 years; mean age \pm SD, 22.17 ± 2.72 years) participated in the study. They were all healthy, medication-free, normally weighted (as indicated by the body-mass index, BMI, $17 < \text{BMI} < 28$; mean BMI \pm SD, 21.79 ± 2.09) with normal or corrected-to-normal vision. None of the participants reported any acute or chronic stress. Since attenuated physiological stress responses (i.e., lower stress-related increase in free cortisol levels analysed from saliva samples) were found for both habitual smoking (e.g., Rohleder and Kirschbaum 2006) and oral-contraceptive intake (e.g., Kirschbaum et al. 1999), all participants were non-smokers and female volunteers did not use hormone-based birth

control. The volunteers gave their written informed consent prior to their inclusion in the study and received financial compensation or course credits, respectively. The study was approved by the local ethics committee and conducted in accordance to ethical standards of the 1964 Declaration of Helsinki.

Stress induction and stress validation

Volunteers were randomly assigned to a stress group or a control group (12 men and 12 women per group). Participants of the stress group were challenged with the TSST (Kirschbaum et al. 1993), a standardised stress-induction protocol consisting of a public speaking and a mental arithmetic task in front of a committee preceded by an anticipatory period (total time: 15 min). Participants of the control group were exposed to a standardised control situation featuring maximum similarity with the TSST while lacking stress-inducing features (for details regarding the procedure, see Het et al. 2009).

Saliva samples were collected with Salivette-sampling devices (Sarstedt, Nümbrecht, Germany) at eight measurement time-points (15, 5, and 1 min before and 1, 10, 20, 30, and 40 min after end of either treatment) for later determination of sAA activity and cortisol levels. For sAA analysis, we applied a quantitative enzyme-kinetic method (cf. Rohleder and Nater 2009). Free cortisol levels were obtained using a chemiluminescence immunoassay (IBL International, Hamburg, Germany). Subjective stress levels were assessed with the German “Mehrdimensionaler Befindlichkeitsfragebogen” (multidimensional mental-state questionnaire, MDBF; Steyer et al. 1997) that measures the current mental state on three dimensions: good mood versus bad mood, calmness versus restlessness, and alertness versus fatigue. The MDBF was given five times throughout the session (15 and 1 min before and 1, 20, and 40 min after the respective treatment). In contrast to all other measurement time-points at which collecting the saliva sample and completing the MDBF took place simultaneously, at measurement time-point -1 min, participants first provided a saliva sample and subsequently received the instruction for the upcoming treatment. Immediately afterwards, they were asked to answer the MDBF. Thus, they reported their current mental state at a time point at which they already anticipated the upcoming treatment situation (i.e., TSST or standardised control situation).

Task switching

In an explicit-cuing version of the task-switching paradigm (e.g., Meiran 1996), participants switched between the two tasks to categorise single-digit numbers (1–9, except 5) as smaller or larger than five or as odd or even, respectively.

Random task order led to two types of task transition, that is, trials in which the task was identical to the former one (repetition trials) and trials in which the task differed from the previous one (switch trials), respectively. A cue (i.e., square or diamond) displayed at the beginning of each trial indicated which task was to be performed. A square instructed participants to categorise the target’s magnitude, whereas a diamond indicated to categorise its parity. After a CTI of either 200 ms (short CTI) or 1,000 ms (long CTI), the target stimulus appeared. Participants responded left if the target was smaller than five or odd and right if the target was larger than five or even as fast and accurate as possible. Using bivalent target stimuli and responses (i.e., the same targets and responses in both tasks while stimulus–response mappings only partially concurred) allowed for analysis of target-congruency effects. Whereas 1 and 3 required a left response in both tasks, and 6 and 8 were consistently linked to the right response (congruent targets), 2, 4, 7, and 9 required different responses in the magnitude and the parity task (incongruent targets).

Task cue and target stimulus were displayed until a response occurred (3,600 ms maximum). Subsequently, a feedback was provided for 300 ms. A correct response was followed by a blank screen, an erroneous response by the word “falsch” (false) together with an acoustic signal (sinus tone) through headphones, a missing response by the feedback “zu langsam” (too slow) combined with the tone. Time lag between feedback offset and task-cue onset was reversed to the subsequently following CTI providing a constant response-target interval of 1,500 ms (cf. Meiran 1996).

Stimuli were centrally displayed white against black on a 17-in. monitor connected to an IBM-compatible personal computer. With a viewing distance of approximately 60 cm, task cues (i.e., square and diamond) had a side length of 2.48° visual angle, and target stimuli (1–9, except 5) extended 0.29° to 0.57° horizontally and 0.86° vertically. Participants responded by pressing the ‘Alt’ and ‘AltGr’ key of a standard QWERTZ keyboard with the left and right index finger, respectively. Presentation software (version 0.71; Neurobehavioral Systems, Inc., Albany, CA, USA) provided the basis for both stimulus presentation and data recording.

Procedure

At first, a cognitive training with a total of 160 trials introduced participants to the task-switching procedure and aimed to minimise the occurrence of practice and learning effects during cognitive testing after treatment. At 20 min after arrival, participants underwent the treatment (i.e., TSST or standardised control situation). After a waiting period of 5 min, the first part of cognitive testing followed

(six blocks with 64 trials each = 384 trials). 25 min after end of treatment, the second part of cognitive testing started (six blocks with 64 trials each = 384 trials; total number of test trials: 768). This ensured both an interim of at least 5 min between both task-switching parts and a standardised time lag between treatment offset and beginning of the second cognitive test part for all participants (total session duration: 90 min).

Testing took place between noon and 7 p.m. to minimise variance due to circadian variations in stress-hormone levels. Since previous studies revealed effects of instantaneous food and caffeine intake on the cortisol response to stress when measured in saliva (Gonzalez-Bono et al. 2002; Lovallo et al. 2006), participants were to refrain from eating as well as consuming sugar- or caffeine-containing drinks 2 h prior to testing. Because acute glucose availability represents a condition precedent for the stress-induced increase of HPA-axis activity (Kirschbaum et al. 1997), we aimed to converge blood-glucose levels by providing all participants with 200 ml grape juice at session start.

Data analysis

One participant of the stress group was excluded from the analysis because she did not follow the task-switching instruction, that is, she constantly categorised target stimuli with regard to their magnitude independent of the cue presented. For the remaining 47 participants, repeated-measures analyses of variance (ANOVAs) with the within-subjects factor measurement time-point (eight or five levels, respectively) and the between-subjects factor stress (stress vs. no stress) were conducted on sAA, cortisol, and total scores of the three MDBF dimensions, respectively to analyse changes in physiological and subjective stress levels over the session course. Both sAA and cortisol data were previously logarithmised to base 10 to ascertain that they meet the requirements of general linear model-based statistics in terms of Gaussian distribution. Regarding task-switching performance, the within-subjects factors task transition (switch vs. repetition), CTI (short vs. long), target congruency (incongruent vs. congruent), and part (first vs. second), and the between-subjects factor stress (stress vs. no stress) were entered into repeated-measures ANOVAs on error rates and mean response times (RTs), respectively. Thereby, all trials without a correct answer, that is, trials with an incorrect or missing response, were considered as errors. The first trial of each block as well as post-error trials (6.23%) were excluded since it is unclear whether they should be considered as switch trials or repetition trials. For RT analysis only, both error trials (4.30% = 4.16% incorrect + 0.14% missing responses) and RTs differing more than 2.5 standard deviations from

mean RT of each participant and condition (2.57%) were additionally excluded. Greenhouse-Geisser correction was applied where appropriate.

Results

Stress response

Neuroendocrine measures

As expected, the time course of salivary cortisol levels differed between stress group and control group, $F(7, 315) = 16.42$, $P < .001$, $\eta_p^2 = .27$. Post hoc t tests revealed higher cortisol levels in the stress group from 10 min after stress cessation throughout the remainder of the session, all P s $< .01$. Although the overall time course of sAA did not differ between groups, $F(7, 315) = 1.53$, $P = .20$, $\eta_p^2 = .03$, separate analyses of group differences showed higher sAA levels for stressed participants than controls immediately after treatment only (1 min), $t(45) = 2.31$, $P < .05$, all other P s $\geq .29$ (Fig. 1, top).

Mental state

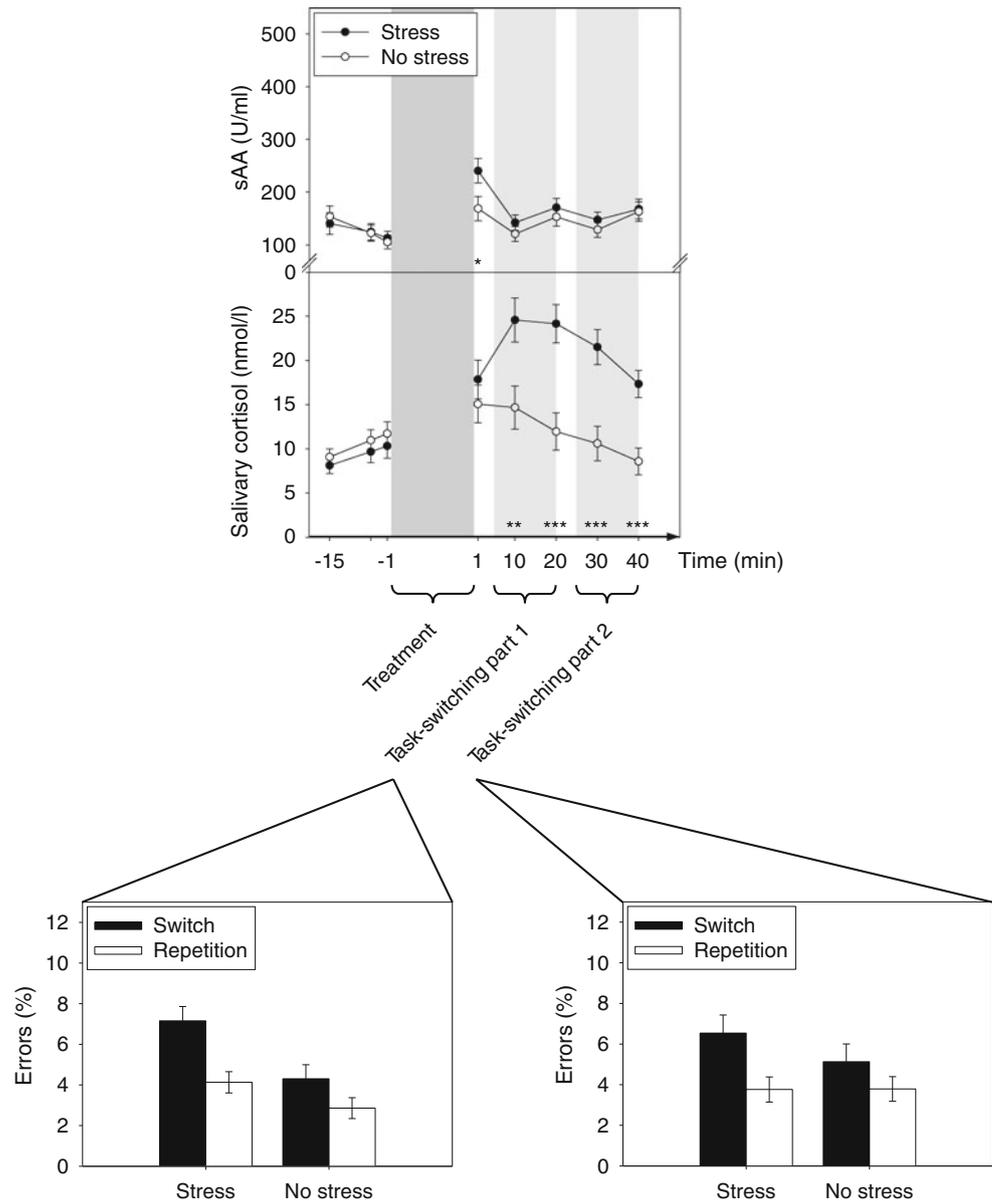
Time courses of MDBF scores differed significantly between stress group and control group for the two dimensions good mood versus bad mood and calmness versus restlessness, $F(4, 180) = 5.65$, $P < .01$, $\eta_p^2 = .11$ and $F(4, 180) = 7.77$, $P < .001$, $\eta_p^2 = .15$. Stressed participants were in a worse mood immediately after stressor offset only (1 min), $t(45) = 3.74$, $P < .01$, all other P s $\geq .09$. Furthermore, they reported being more restless after receiving instructions for the upcoming treatment during the anticipatory period, $t(45) = 3.17$, $P < .01$, as well as directly after stress cessation (1 min), $t(45) = 3.29$, $P < .01$, all other P s $\geq .12$. In addition, fatigue increase emerged over time, $F(4, 180) = 14.78$, $P < .001$, $\eta_p^2 = .25$, with similar changes and mean fatigue level in both groups, $F(4, 180) = 1.11$, $P = .34$, $\eta_p^2 = .02$ and $F < 1$ (Fig. 2).

Cognitive performance

Error rates

Task-switching performance The ANOVA revealed significant main effects of task transition (switch: 5.78%, repetition: 3.64%), $F(1, 45) = 66.41$, $P < .001$, $\eta_p^2 = .60$, and target congruency (incongruent: 7.84%, congruent: 1.58%), $F(1, 45) = 84.42$, $P < .001$, $\eta_p^2 = .65$, as well as a marginally significant effect of CTI (short: 4.95%, long:

Fig. 1 Mean levels of salivary α -amylase (sAA) and salivary cortisol as a function of time (minutes before or after treatment) for the stress group and the control group, respectively (*top*). Error rates as a function of task transition (switch vs. repetition) for the stress group and the control group and for task-switching part 1 and 2, respectively (*bottom*). Error bars represent standard errors of the mean. * $P < .05$; ** $P < .01$; *** $P < .001$



4.47%), $F(1, 45) = 3.16$, $P = .08$, $\eta_p^2 = .07$. Task transition was further modulated by CTI, $F(1, 45) = 9.83$, $P < .01$, $\eta_p^2 = .18$, target congruency, $F(1, 45) = 28.66$, $P < .001$, $\eta_p^2 = .39$, and the combination of both, $F(1, 45) = 4.42$, $P < .05$, $\eta_p^2 = .09$, respectively. More precisely, switch costs (i.e., error rates in switch trials minus error rates in repetition trials) were larger in trials with short CTI (2.90%) than in trials with long CTI (1.39%) as well as for incongruent targets (3.64%) compared to congruent targets (0.65%). Moreover, the switch-costs difference between responses to incongruent targets and responses to congruent targets was larger for trials with short CTI (4.15%) compared to trials with long CTI (1.84%). CTI and target congruency did not interact, $F < 1$.

Effects of acute psychosocial stress on task-switching performance There was a significant interaction between task transition and stress, $F(1, 45) = 8.21$, $P < .01$, $\eta_p^2 = .15$, with increased switch costs in the stress group (2.90% vs. 1.39% in the control group). This effect primarily originated from error-rate differences in switch trials (6.85% in stressed participants vs. 4.72% in controls), $t(45) = 2.03$, $P < .05$, rather than being driven by group-dependent performance differences in repetition trials, $t(45) = 1.03$, $P = .31$. The interactions target congruency \times stress, $F(1, 45) = 2.55$, $P = .118$, $\eta_p^2 = .05$, and task transition \times target congruency \times stress, $F(1, 45) = 3.71$, $P = .06$, $\eta_p^2 = .08$, did not reach significance level. Moreover, stressed participants showed numerically larger mean error rates than participants of the control group

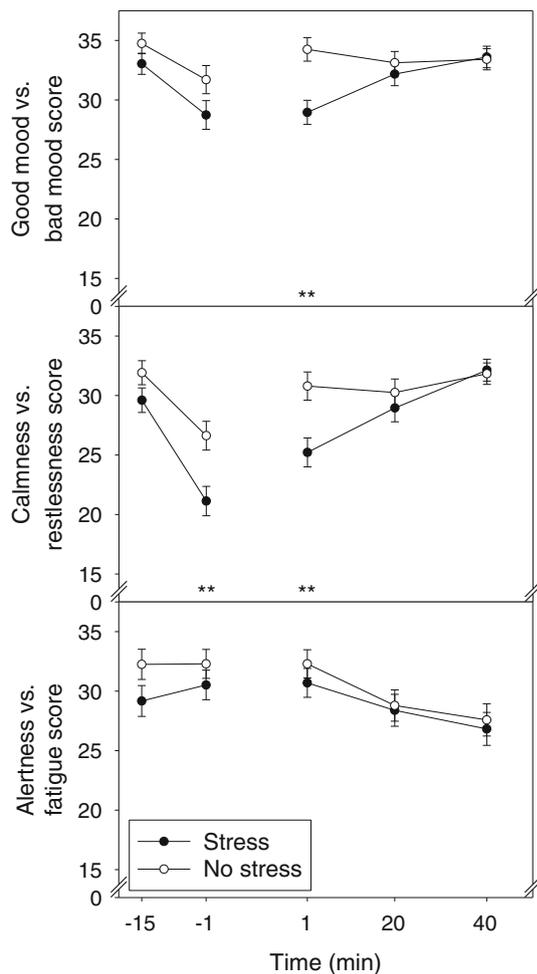


Fig. 2 Mental state on the three dimensions good mood versus bad mood, calmness versus restlessness, and alertness versus fatigue assessed with the German mood questionnaire “Mehrdimensionaler Befindlichkeitsfragebogen” (multidimensional mental-state questionnaire, MDBF; Steyer et al. 1997) as a function of time (minutes before or after treatment) for the stress group and the control group, respectively. The mental-state measurement -1 took place directly after instruction for the subsequent treatment (i.e., Trier Social Stress Test or standardised control situation) within the anticipatory period. Error bars represent standard errors of the mean. ** $P < .01$

(5.40% vs. 4.02%). This main effect of stress on error rates, however, failed to reach significance, $F(1, 45) = 2.57$, $P = .116$, $\eta_p^2 = .05$.

To additionally explore whether group differences in task-switching performance result from the at least numerically observed differences in mean error rates between stressed individuals and controls, we conducted an analysis of covariance (ANCOVA) comprising all factors of the original ANOVA plus individual mean error rate as covariate. Most importantly, the outcome confirmed the significant interaction between task transition and stress, $F(1, 44) = 5.24$, $P < .05$, $\eta_p^2 = .11$. At the same time, however, for both target congruency \times stress and task

transition \times target congruency \times stress, no statistical trends remained, $F < 1$ and $F(1, 44) = 1.11$, $P = .30$, $\eta_p^2 = .03$, suggesting that the former trends towards significant thresholds may rather reflect a consequence of initial-level dependency (Wilder 1962). Within the original analysis, no further interaction of stress with one or more of the task switching-related factors reached significance, $P_s \geq .13$.

Comparison of the two parts Overall task-switching performance (i.e., without considering the factor stress) did not differ between first and second cognitive testing part, all $P_s \geq .14$. There was only a weak tendency for an interaction between target congruency and part, $F(1, 45) = 2.79$, $P = .10$, $\eta_p^2 = .06$, reflecting an increase of the target-congruency effect from the first to the second part. Although the stress effect on overall error rates was larger in the first cognitive testing part (stress: 5.64%, no stress: 3.58%) than in the second part (stress: 5.15%, no stress: 4.46%), $F(1, 45) = 4.05$, $P = .05$, $\eta_p^2 = .08$, the effect of stress on switch costs remained stable over time, $F < 1$.

To further elaborate these findings, we repeated the main analysis for the two parts separately. We again found a significant interaction between task transition and stress in both the first and the second cognitive testing part, $F(1, 45) = 7.36$, $P < .01$, $\eta_p^2 = .14$ and $F(1, 45) = 4.17$, $P < .05$, $\eta_p^2 = .09$ (Fig. 1, bottom). At the same time, ANOVAs showed a significant impact of stress on mean error rates in the first part, $F(1, 45) = 6.32$, $P < .05$, $\eta_p^2 = .12$, while no evidence for such an influence was found for the second part, $F < 1$. No further interactions were revealed, $P_s \geq .12$. Mean error rates and standard errors of the mean for all combinations of task transition, CTI, target congruency overall as well as separately for the two task-switching parts for the stress group and the control group, respectively, are presented in Table 1.

RTs

Task-switching performance RT analysis showed significant main effects of task transition (switch: 864 ms, repetition: 740 ms), $F(1, 45) = 84.10$, $P < .001$, $\eta_p^2 = .65$, CTI (short: 856 ms, long: 748 ms), $F(1, 45) = 116.12$, $P < .001$, $\eta_p^2 = .72$, and target congruency (incongruent: 853 ms, congruent: 751 ms), $F(1, 45) = 173.79$, $P < .001$, $\eta_p^2 = .79$. Switch costs were larger in trials with short CTI (152 ms) compared to trials with long CTI (96 ms), $F(1, 45) = 28.21$, $P < .001$, $\eta_p^2 = .39$, as well as for incongruent targets (135 ms) compared to congruent targets (113 ms), $F(1, 45) = 5.87$, $P < .05$, $\eta_p^2 = .12$. The target-congruency effect was larger in trials with short CTI (115 ms) than in trials with long CTI (91 ms), $F(1, 45) = 5.83$, $P < .05$, $\eta_p^2 = .12$, while the three-way

Table 1 Error rates (%) for all combinations of task transition, cue-target interval (CTI), and target congruency for the stress group and the control group overall as well as separately for task-switching part 1 and 2

Task transition	CTI	Target congruency	Stress ($n = 23$)			No stress ($n = 24$)		
			Overall	Task-switching part 1	Task-switching part 2	Overall	Task-switching part 1	Task-switching part 2
Switch	Short	Incongruent	12.31 (1.63)	12.80 (1.60)	11.82 (1.92)	9.00 (1.60)	7.85 (1.56)	10.14 (1.88)
		Congruent	2.44 (0.44)	2.91 (0.53)	1.97 (0.54)	1.83 (0.43)	2.12 (0.52)	1.55 (0.53)
	Long	Incongruent	10.76 (1.33)	10.97 (1.35)	10.54 (1.52)	6.56 (1.30)	6.09 (1.33)	7.04 (1.48)
		Congruent	1.88 (0.42)	1.94 (0.43)	1.82 (0.59)	1.48 (0.41)	1.16 (0.42)	1.79 (0.58)
Repetition	Short	Incongruent	5.74 (0.76)	5.69 (0.86)	5.79 (1.00)	5.62 (0.75)	4.39 (0.84)	6.86 (0.98)
		Congruent	1.23 (0.36)	1.39 (0.39)	1.07 (0.50)	1.41 (0.36)	1.38 (0.39)	1.43 (0.49)
	Long	Incongruent	7.45 (1.00)	7.74 (1.10)	7.17 (1.23)	5.24 (0.98)	4.65 (1.08)	5.83 (1.21)
		Congruent	1.37 (0.28)	1.71 (0.36)	1.03 (0.36)	1.04 (0.28)	1.03 (0.35)	1.06 (0.35)

$N = 47$

Values are given as means (standard errors of the mean)

interaction between task transition, CTI, and target congruency was not significant, $F < 1$.

Effects of acute psychosocial stress on task-switching performance Mean RTs as well as switch costs did not differ between stressed participants and controls, both $F_s < 1$. The ANOVA only showed a tendency for a smaller CTI effect in the stress group (89 ms) compared to the control group (125 ms), $F(1, 45) = 3.22$, $P = .08$, $\eta_p^2 = .07$. This tendency seemed to arise from trials with short CTI in which the stressed participants responded faster (840 ms vs. 871 ms for controls) rather than trials with long CTI (stress: 751 ms, no stress: 746 ms) as indicated by numerical inspection. Importantly, this tendency did not affect the interaction task transition by stress, $F < 1$. No further interaction between stress and one or more of the task switching-related factors reached significance, all $P_s \geq .23$.

Comparison of the two parts Analysis revealed a decrease from the first task-switching part to the second part in both mean RTs (first part: 825 ms, second part: 779 ms), $F(1, 45) = 27.26$, $P < .001$, $\eta_p^2 = .38$, and switch costs (first part: 137 ms, second part: 111 ms), $F(1, 45) = 12.37$, $P < .01$, $\eta_p^2 = .22$. RT difference between trials with short CTI and trials with long CTI was larger in the first task-switching part (125 ms) than in the second one (89 ms), $F(1, 45) = 17.08$, $P < .001$, $\eta_p^2 = .28$. No further factors or factorial combinations (inclusive of all interactions comprising the factor stress) were modulated by part, $P_s > .05$. Stress did not differentially affect mean RT level in the two task-switching parts, $F < 1$. Mean RTs and standard errors of the mean for all combinations of task transition, CTI, target congruency overall as well as separately for the two task-switching parts for the stress group and the control group, respectively, are provided in Table 2.

Table 2 Response times (ms) for all combinations of task transition, cue-target interval (CTI), and target congruency for the stress group and the control group overall as well as separately for task-switching part 1 and 2

Task transition	CTI	Target congruency	Stress ($n = 23$)			No stress ($n = 24$)		
			Overall	Task-switching part 1	Task-switching part 2	Overall	Task-switching part 1	Task-switching part 2
Switch	Short	Incongruent	983 (47)	1,009 (49)	956 (47)	1,012 (46)	1,051 (48)	973 (46)
		Congruent	843 (41)	887 (47)	799 (38)	889 (40)	942 (46)	836 (37)
	Long	Incongruent	841 (45)	855 (48)	827 (44)	848 (44)	868 (47)	829 (43)
		Congruent	747 (38)	778 (42)	715 (38)	749 (37)	756 (41)	742 (37)
Repetition	Short	Incongruent	812 (34)	832 (38)	793 (33)	846 (34)	875 (38)	818 (32)
		Congruent	723 (33)	740 (37)	706 (31)	737 (33)	764 (37)	711 (30)
	Long	Incongruent	752 (37)	767 (39)	737 (37)	733 (36)	739 (38)	727 (36)
		Congruent	663 (30)	672 (33)	655 (28)	653 (29)	663 (32)	644 (27)

$N = 47$

Values are given as means (standard errors of the mean)

Discussion

The current study exposed healthy participants to acute psychosocial stress (TSST; Kirschbaum et al. 1993) before performing in a task-switching experiment (e.g., Meiran 1996) addressing the question of how acute psychosocial stress affects cognitive control processes of flexible task-goal implementation. As expected, the stress group displayed increased sAA activity immediately after stress exposure and elevated cortisol levels from 10 min after stress cessation throughout the remaining session. This reflects the typical kinetic of immediate SNS-related and prolonged HPA-mediated stress responses indicating successful stress induction.

We here report evidence for a significant alteration of task-switching performance after stress: The stress group displayed larger error-related switch costs indicating less efficiency of the cognitive control processes involved. In other words, acute psychosocial stress impaired cognitive control processes underlying the flexible implementation of task goals as requirement for successful task switching. This interpretation is further supported by the observation that the switch-cost increase under stress primarily originated from stress-related performance changes for task switches but not for task repetitions.

The larger switch costs in stressed individuals compared to controls are unlikely to be explained in terms of a stress-induced performance-criterion shift from favouring accurate responses towards faster responses (i.e., a speed-accuracy trade-off). If stress resulted in premature responding, stressed individuals compared to controls should display (a) smaller mean RTs, (b) increased error rates not only for task switches but also for task repetitions, and importantly, (c) a higher susceptibility (i.e., higher error rates) to automatic conflicting response activations as particularly present in incongruent trials, while showing unchanged or even decreased error rates in conditions of corresponding response activations (i.e., in congruent trials). Since none of these three predictions derived from a performance-criterion shift-account was confirmed within the present study, such an (explicit or implicit) strategic performance change as the basis of the observed performance pattern seems to be unlikely.

On the contrary, the findings rather point towards a specific effect of acute stress on task performance. Stressed individuals displayed larger switch costs suggesting a stress-related impairment of a central cognitive control function (i.e., flexible task-goal implementation). Such cognitive control impairment under stress seems in line with assumptions derived from the account of resource depletion in conditions of stress (e.g., Arnsten 2009; Liston et al. 2009) that postulates an action-control shift from higher-order cognitive control functions to more habitual stimulus-based control.

At the same time, however, stimulus-based interference control, as indexed by the target-congruency effect, did only numerically but not significantly differ between stress and control group, and this difference was no longer visible when controlling for mean error rates (see also Plessow et al. 2011a; Wolf et al. 2001). Although interpretations of none-effects have to be taken with caution and we cannot exclude that an impact of acute stress on target congruency could eventually become evident when operating with considerably larger sample sizes, in the present study and with a sample size sufficient to detect a substantial negative impact of acute stress on flexible task-goal implementation, no reliable modulation of stimulus-based interference control by stress could be revealed. Such a finding suggests interesting theoretical implications: An insensitivity of stimulus-based interference control to stress would be difficult to concur with the assumption of an unspecific general decrease in PFC regulation in favour of reflexive bottom-up processes primarily based on subcortical structures (cf. Arnsten 2009). If stress negatively affected PFC functioning, all PFC-dependent functions should be impaired under conditions of stress. Thus, a differential effect of acute stress on cognitive control functioning would be more in line with the idea of priority-dependent resource allocation under stress (e.g., Hockey 1997) stating that some cognitive processes may be deteriorated under stress while others may not (depending on the amount of available resources as well as the current internal and external context).

Pushing this hypothesis even further, it might be helpful to consider characteristics of the two relevant outcome measures (manifest variables) and related cognitive control functions (according latent variables) assessed with the applied cognitive task. Task-goal implementation and interference control substantially differ in two aspects: (a) the level of abstraction on which a conflict occurs and needs to be resolved and (b) the processing period in relation to stimulus presentation and behavioural response. Interference control serves to reduce erroneous responses resulting from stimulus-based automatic activation of the corresponding response within the currently irrelevant task (i.e., a particular stimulus-response link, e.g., Wendt and Kiesel 2008). Task-goal implementation, however, aims at prioritising the correct task set (i.e., the task-specific whole sample of stimulus-response links). Hence, interference control exerts its effect on the stimulus level, whereas the implementation of a new task goal operates on the more abstract task level. Clearly, at the current stage of research, although exciting we can only speculate about the specificity of acute stress effects on distinct cognitive control functions, and subsequent research is now required to directly test this hypothesis.

In addition, it is interesting to note that although stressed participants were subjectively stressed by the TSST as

indicated by worse mood and more restlessness immediately after stress exposure, they did not differ from controls in current mental state during the cognitive testing. Stress-related performance decrements thus occurred in the absence of subjective mood changes (see also Plessow et al. 2011a). Moreover, since both mean fatigue level and course of fatigue over time were similar in both groups, psychological and physical exertion can also not account for the revealed stress-related cognitive impairments. Likewise, the observed stress effects were not gender-specific indicating a universal impact of stress on cognitive control processes of task-goal implementation.¹

The increased switch costs in stressed individuals compared to controls equally occurred in conditions of short and long preparation time. This speaks against a stress-induced impairment of preparation-dependent cognitive control processes serving task-goal implementation. This finding does not correspond with results of Steinhilber et al. (2007) who showed an impact of acute stress (previous processing of a high-stress intelligence-test tasks) on task-switching performance specifically for conditions of sufficient preparation time. However, a direct comparison of both studies has to be handled with caution, because, first, stress induction substantially differed in both studies and, second, RT measures and error rates in task switching may not reflect the same underlying mechanisms (Altmann and Gray 2008).

A further open question addresses the time course of acute stress effects on PFC-dependent cognitive functions (e.g., cognitive control). In the current study, the stress induction led to an impairment of cognitive control processes of task-goal implementation not only during testing from 5 to 20 min after stressor offset but also at 25–40 min subsequent to stress cessation. Thus, the observed effect is rather persistent than transient and also occurred during time intervals of elevated cortisol levels indicating increased HPA-axis activity while stress-related increases in SNS activity were already back to normal. Plessow et al. (2011a) even demonstrated an effect of acute psychosocial stress on cognitive control processes that only developed with increasing time lag to the stressor. However, different studies observed diverse time courses of acute stress effects on PFC-dependent cognitive functions (e.g., Elzinga and Roelofs 2005; Schoofs et al. 2008). Schoofs et al. (2008), for example, investigated the impact of acute psychosocial stress on PFC-dependent working-memory functions from 10 min after stress cessation and showed impaired cognitive performance in the first two out of eight experimental

blocks only. Whereas in both studies the potential relevance of the HPA-stress response for the development of the stress-related behavioural effects is highlighted (supported by correlations between treatment-related cortisol increase and performance-outcome measures), a substantial performance difference between stressed individuals and controls in Plessow et al.'s study was only observed during a later time period at which the effects of Schoofs and colleagues were no longer evident.

Besides fundamental differences in the investigated cognitive parameters (i.e., measures of working-memory and cognitive control functions, respectively), a critical difference between the Schoofs et al. and the present findings is the control for task practice. Whereas our participants received extensive task training prior to treatment in order to enable learning and confident application of task rules in stress-free conditions (Plessow et al. 2011a), in the study by Schoofs et al., participants engaged with the task only after stress induction. Therefore, it is conceivable that acute stress did not only affect the intended mechanism of interest but also targeted practice-related mechanisms involved in first task encounters (e.g., task-rule learning).

However, to date, research addressing the link between acute stress and PFC-dependent cognitive functions (e.g., cognitive control) is in its early stage. Accordingly, the current goal is to accumulate empirical evidence in order to define the nature of this relationship (e.g., linear, inverted U-shaped) as well as to gain a deeper understanding in the highly complex neuro-modulation of multiple stress-sensitive physiological systems that likely play part in the mediation of the discussed effects (for recent reviews, see Arnsten 2009; Joels and Baram 2009). Thereby, the current study protocol may serve as a well suited tool to study the underlying biological mechanisms in more detail by employing, for example, pharmacological or invasive methods.

Acknowledgments We thank Susann Schade and Moritz Walser for assistance in data acquisition.

Conflict of interest The authors declare that they have no conflict of interest.

References

- Allport DA, Styles EA, Hsieh S (1994) Shifting intentional set: exploring the dynamic control of tasks. In: Umiltà C, Moscovitch M (eds) *Conscious and nonconscious information processing: attention and performance XV*. MIT Press, Cambridge, pp 421–452
- Altmann EM, Gray WD (2008) An integrated model of cognitive control in task switching. *Psychol Rev* 115(3):602–639
- Arnsten AFT (2009) Stress signalling pathways that impair prefrontal cortex structure and function. *Nat Rev Neurosci* 10(6):410–422
- Brass M, von Cramon DY (2002) The role of the frontal cortex in task preparation. *Cereb Cortex* 12(9):908–914

¹ This was indicated by a separate repeated-measures ANOVA on error rates with the additional between-subjects factor gender that showed no modulation of the interaction between task transition and stress by gender, $F < 1$. Likewise, none of the other interactions comprising stress as well as gender reached significance, all $P_s \geq .23$.

- Bratzke D, Rolke B, Steinborn MB, Ulrich R (2009) The effect of 40 h constant wakefulness on task-switching efficiency. *J Sleep Res* 18(2):167–172
- Cepeda NJ, Cepeda ML, Kramer AF (2000) Task switching and attention deficit hyperactivity disorder. *J Abnorm Child Psychol* 28(3):213–226
- Cools R, Barker RA, Sahakian BJ, Robbins TW (2001) Mechanisms of cognitive set flexibility in Parkinson's disease. *Brain* 124 (Pt 12):2503–2512
- de Kloet ER, Joels M, Holsboer F (2005) Stress and the brain: from adaptation to disease. *Nat Rev Neurosci* 6(6):463–475
- Dickerson SS, Kemeny ME (2004) Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychol Bull* 130(3):355–391
- Dreisbach G, Goschke T (2004) How positive affect modulates cognitive control: reduced perseverance at the cost of increased distractibility. *J Exp Psychol Learn Mem Cogn* 30(2):343–353
- Elzinga BM, Roelofs K (2005) Cortisol-induced impairments of working memory require acute sympathetic activation. *Behav Neurosci* 119(1):98–103
- Friederich HC, Herzog W (2011) Cognitive-behavioral flexibility in anorexia nervosa. *Curr Top Behav Neurosci* 6:111–123
- Gonzalez-Bono E, Rohleder N, Hellhammer DH, Salvador A, Kirschbaum C (2002) Glucose but not protein or fat load amplifies the cortisol response to psychosocial stress. *Horm Behav* 41(3):328–333
- Goschke T (2000) Intentional reconfiguration and involuntary persistence in task set switching. In: Monsell S, Driver J (eds) *Attention and performance XVIII: control of cognitive processes*. MIT Press, Cambridge, pp 331–355
- Greeneweg FL, Karst H, de Kloet ER, Joels M (2011) Mineralocorticoid and glucocorticoid receptors at the neuronal membrane, regulators of nongenomic corticosteroid signalling. *Mol Cell Endocrinol*. doi:10.1016/j.mce.2011.06.020
- Het S, Rohleder N, Schoofs D, Kirschbaum C, Wolf OT (2009) Neuroendocrine and psychometric evaluation of a placebo version of the 'Trier Social Stress Test'. *Psychoneuroendocrinology* 34(7):1075–1086
- Heuer H, Kleinsorge T, Klein W, Kohlisch O (2004) Total sleep deprivation increases the costs of shifting between simple cognitive tasks. *Acta Psychol (Amst)* 117(1):29–64
- Hockey GR (1997) Compensatory control in the regulation of human performance under stress and high workload: a cognitive-energetical framework. *Biol Psychol* 45(1–3):73–93
- Joels M, Baram TZ (2009) The neuro-symphony of stress. *Nat Rev Neurosci* 10(6):459–466
- Kern S, Oakes TR, Stone CK, McAuliff EM, Kirschbaum C, Davidson RJ (2008) Glucose metabolic changes in the prefrontal cortex are associated with HPA axis response to a psychosocial stressor. *Psychoneuroendocrinology* 33(4):517–529
- Kiesel A, Wendt M, Peters A (2007) Task switching: on the origin of response congruency effects. *Psychol Res* 71(2):117–125
- Kiesel A, Steinhauser M, Wendt M, Falkenstein M, Jost K, Philipp AM, Koch I (2010) Control and interference in task switching—a review. *Psychol Bull* 136(5):849–874
- Kirschbaum C, Pirke KM, Hellhammer DH (1993) The 'Trier Social Stress Test'—a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology* 28(1–2):76–81
- Kirschbaum C, Hellhammer DH (1994) Salivary cortisol in psychoneuroendocrine research: recent developments and applications. *Psychoneuroendocrinology* 19(4):313–333
- Kirschbaum C, Gonzalez Bono E, Rohleder N, Gessner C, Pirke KM, Salvador A, Hellhammer DH (1997) Effects of fasting and glucose load on free cortisol responses to stress and nicotine. *J Clin Endocrinol Metab* 82(4):1101–1105
- Kirschbaum C, Kudielka BM, Gaab J, Schommer NC, Hellhammer DH (1999) Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosom Med* 61(2):154–162
- Kofman O, Meiran N, Greenberg E, Balas M, Cohen H (2006) Enhanced performance on executive functions associated with examination stress: evidence from task-switching and Stroop paradigms. *Cogn Emot* 20(5):577–595
- Liefooghe B, Barrouillet P, Vandierendonck A, Camos V (2008) Working memory costs of task switching. *J Exp Psychol Learn Mem Cogn* 34(3):478–494
- Liston C, McEwen BS, Casey BJ (2009) Psychosocial stress reversibly disrupts prefrontal processing and attentional control. *Proc Natl Acad Sci USA* 106(3):912–917
- Lovallo WR, Farag NH, Vincent AS, Thomas TL, Wilson MF (2006) Cortisol responses to mental stress, exercise, and meals following caffeine intake in men and women. *Pharmacol Biochem Behav* 83(3):441–447
- MacDonald AW 3rd, Cohen JD, Stenger VA, Carter CS (2000) Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science* 288(5472):1835–1838
- Mayr U, Keele SW (2000) Changing internal constraints on action: the role of backward inhibition. *J Exp Psychol Gen* 129(1):4–26
- Meiran N (1996) Reconfiguration of processing mode prior to task performance. *J Exp Psychol Learn Mem Cogn* 22:1423–1442
- Miller EK, Cohen JD (2001) An integrative theory of prefrontal cortex function. *Annu Rev Neurosci* 24:167–202
- Nater UM, Rohleder N (2009) Salivary alpha-amylase as a non-invasive biomarker for the sympathetic nervous system: current state of research. *Psychoneuroendocrinology* 34(4):486–496
- Norman DA, Shallice T (1986) Attention to action: willed and automatic control of behavior. In: Davidson RJ, Schwartz GE, Shapiro D (eds) *Consciousness and self-regulation: advances in research*, vol 4. Plenum Press, New York, pp 1–18
- Perlman WR, Webster MJ, Herman MM, Kleinman JE, Weickert CS (2007) Age-related differences in glucocorticoid receptor mRNA levels in the human brain. *Neurobiol Aging* 28(3):447–458
- Plessow F, Fischer R, Kirschbaum C, Goschke T (2011a) Inflexibly 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. *J Cogn Neurosci* 23(11):3218–3227
- Plessow F, Kiesel A, Petzold A, Kirschbaum C (2011b) Chronic sleep curtailment impairs the flexible implementation of task goals in new parents. *J Sleep Res* 20(2):279–287
- Qin S, Hermans EJ, van Marle HJ, Luo J, Fernandez G (2009) Acute psychological stress reduces working memory-related activity in the dorsolateral prefrontal cortex. *Biol Psychiatry* 66(1):25–32
- Ramos BP, Arnsten AF (2007) Adrenergic pharmacology and cognition: focus on the prefrontal cortex. *Pharmacol Ther* 113(3):523–536
- Rogers RD, Monsell S (1995) Costs of a predictable switch between simple cognitive tasks. *J Exp Psychol Gen* 124:207–231
- Rohleder N, Kirschbaum C (2006) The hypothalamic-pituitary-adrenal (HPA) axis in habitual smokers. *Int J Psychophysiol* 59(3):236–243
- Rohleder N, Nater UM (2009) Determinants of salivary alpha-amylase in humans and methodological considerations. *Psychoneuroendocrinology* 34(4):469–485
- Sanchez MM, Young LJ, Plotsky PM, Insel TR (2000) Distribution of corticosteroid receptors in the rhesus brain: relative absence of glucocorticoid receptors in the hippocampal formation. *J Neurosci* 20(12):4657–4668
- Schoofs D, Preuss D, Wolf OT (2008) Psychosocial stress induces working memory impairments in an n-back paradigm. *Psychoneuroendocrinology* 33(5):643–653

- Steinhauser M, Maier M, Hübner R (2007) Cognitive control under stress: how stress affects strategies of task-set reconfiguration. *Psychol Sci* 18(6):540–545
- Steyer R, Schwenkmezger P, Notz P, Eid M (1997) Der mehrdimensionale befindlichkeitsfragebogen (MDBF) [the multidimensional mental-state questionnaire (MDBF)]. Hogrefe, Göttingen
- Vijayraghavan S, Wang M, Birnbaum SG, Williams GV, Arnsten AF (2007) Inverted-U dopamine D1 receptor actions on prefrontal neurons engaged in working memory. *Nat Neurosci* 10(3):376–384
- Wendt M, Kiesel A (2008) The impact of stimulus-specific practice and task instructions on response congruency effects between tasks. *Psychol Res* 72(4):425–432
- Wilder J (1962) Basimetric approach (law of initial value) to biological rhythms. *Ann N Y Acad Sci* 98:1211–1220
- Wolf OT, Convit A, McHugh PF, Kandil E, Thorn EL, De Santi S, McEwen BS, de Leon MJ (2001) Cortisol differentially affects memory in young and elderly men. *Behav Neurosci* 115(5):1002–1011