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

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Abstract

■ Dynamically adjusting the right amount of goal shielding to varying situational demands is associated with the flexibility of cognitive control, typically linked with pFC functioning. Although stress hormones are found to also bind to prefrontal receptors, the link between stress and cognitive control remains elusive. Based on that, we aimed at investigating effects of acute psychosocial stress on dynamic control adjustments. Forty-eight healthy volunteers were exposed to either a well-established stress induction protocol (the Trier Social Stress Test, TSST) or a standardized control situation before a selective attention (Simon) task involving response conflicts. The individual physiological stress response was monitored by analyzing levels of free cortisol and α-amylase activity in saliva samples showing that the TSST reliably induced an increase of endogenous stress hormone levels. Acute stress did not inevitably impair cognitive functioning, however, as stressed participants showed tonically increased goal shielding (to reduce interference) at the expense of decreased cognitive flexibility. Importantly, as a novel finding in humans, stress effects on cognitive functions were not present immediately after the stress experience but developed gradually over time and, therefore, paralleled the time course of the hypothalamus–pituitary–adrenal (HPA) stress response. In addition, the total increase of individual cortisol levels reflecting HPA activity, but not the total changes in α-amylase activity associated with sympathetic activity, was reversely related to the amount of cognitive flexibility in the final block of testing. Our study provides evidence for a stress-induced time-dependent decrease of cognitive flexibility that might be related to changes in cortisol levels. ■

INTRODUCTION

An important feature of efficient action control is the high degree of flexibility with which behavior is constantly adjusted to current environmental properties. This flexibility is based on mechanisms of cognitive control that enable us to shield current action goals from competing distracting influences while simultaneously monitoring for potential action-relevant information (Miller & Cohen, 2001; Goschke, 2000). Efficient action control is, thus, reflected in the adaptive regulation of the amount of goal shielding in response to changing situational demands, because too much or too little goal shielding can both result in dysfunctional behavior such as increased perseveration or distractibility, respectively (Goschke & Dreisbach, 2008; Muller et al., 2007; Braver & Cohen, 1999).

In the present study, we aimed at investigating the ability to dynamically regulate the appropriate amount of goal shielding under acute psychosocial stress. This research was motivated by the observation that dynamic adjustments of goal shielding are especially important in situations of conflict (signaling potential erroneous behavior) or novelty (reflecting situations without prior experience), which are exactly those situations found to induce stress in mammals (cf. Mason, 1968). Early animal and human research demonstrated that the exposure to a situation where an individual lacks previous experience leads to physiological stress responses (e.g., Davis et al., 1962). It is, thus, likely that the same situational characteristics that demand high levels of cognitive flexibility are perceived as particularly stressful.

Despite this obvious link, remarkably little research has directly addressed the question of whether and how stress responses affect cognitive flexibility and vice versa. On a theoretical level, the assumption of a close link between the experience of stress and the flexibility of cognitive control appears reasonable. Cognitive control processes that underlie efficient action control were repeatedly found to rely on pFC functioning (Mansouri, Tanaka, & Buckley, 2009; Miller & Cohen, 2001). Importantly, evidence primarily derived from animal research indicates that pFC is particularly vulnerable to influences of both acute and chronic stress (Barsegyan, Mackenzie, Kurose, McGaugh, & Roozendaal, 2010; for a review, see Arnsten, 2009). Of

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central importance in this modulation is the increased activity of the hypothalamus–pituitary–adrenal (HPA) axis under stress, which triggers the synthesis and secretion of glucocorticoids (mainly cortisol; de Kloet, Joels, & Holsboer, 2005). When bound to mineralocorticoid and glucocorticoid receptors, which are abundant in pFC (e.g., Perlman, Webster, Herman, Kleinman, & Weickert, 2007), glucocorticoids alter local brain activity (e.g., Liston et al., 2006). This in turn might provide a physiological basis for a close interaction between stress and cognitive control processes.

Although effects of stress on hippocampus-dependent declarative memory functions are well documented (for a review, see Wolf, 2009), the few available studies addressing the link between stress and flexible cognitive control provide a relatively inconsistent picture both empirically and theoretically. An intuitive assumption is that stress depletes attentional resources and, thus, impairs cognitive control functions (e.g., Liston, McEwen, & Casey, 2009; Alexander, Hillier, Smith, Tivarus, & Beversdorf, 2007; Steinhauser, Maier, & Hübner, 2007). Stress is thought to interfere with controlled and resource-dependent processes with only little impact on automatic or highly accessible information. As a consequence, goal shielding becomes insufficient reflected in an increased susceptibility to interference by automatically activated task-irrelevant stimuli (Wegner & Erber, 1992).

An alternative view postulates an increased selectivity under stress. As compensation for stress-induced overload of the cognitive system, the scarce attentional resources may be fully engaged in task-relevant processing demanding prioritized processing. This narrowing of attention leads to reduced interference by irrelevant information and, therefore, to heightened goal shielding (e.g., Kofman, Meiran, Greenberg, Balas, & Cohen, 2006; Chajut & Algom, 2003; Wells & Matthews, 1994).

In summary, empirical studies have yielded opposing views how stress modulates cognitive control functions. Apart from differences in tasks, procedures, and examined components of cognitive control, one reason for the empirical differences might be found in the inconsistent and often relatively vague definition of stress. In the context of the present study, stress was conceptualized as a two-component response of the CNS to acute uncontrollable psychosocial stress, that is, an early activity increase of the sympathetic nervous system (SNS) and a late activity increase of the HPA axis. To stimulate this physiological stress response in our healthy volunteers, we employed the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), a standardized protocol for laboratory settings that reliably induces moderate psychosocial stress (cf. Dickerson & Kemeny, 2004) on half of our participants. The other half, however, were exposed to a standardized control situation (see Het, Rohleder, Schoofs, Kirschbaum, & Wolf, 2009). In addition, we aimed at validating the stress response on a physiological level by repeatedly collecting saliva samples and analyzing well-established biological stress markers, that is, salivary α-amylase (sAA) activity reflecting sympathetic activity (Nater & Rohleder, 2009) and free cortisol levels reflecting HPA axis activity (e.g., Kirschbaum & Hellhammer, 1994), respectively. Moreover, subjective stress levels were monitored by analyzing self-reported mental state assessed with the mental state questionnaire MDBF (Steyer, Schwenkmezger, Notz, & Eid, 1997).

Cognitive flexibility was investigated by means of a selective attention task following the treatment (i.e., TSST vs. standardized control situation) designed to study the dynamic adjustment of goal shielding to current task demands such as the occurrence of response conflicts. We used a version of the Simon task (Simon, 1990) that was administered over three blocks to capture potential time-dependent stress effects. Participants responded with left and right keypresses to the identity of stimuli that were presented to the left or right of fixation. A response conflict occurs when the irrelevant stimulus location (e.g., on the right side) activates a response (e.g., right keypress) that differs from the response mapped to the task-relevant stimulus feature (i.e., left keypress). Such incompatible trials produce slower and/or more erroneous responses than compatible trials in which relevant and irrelevant stimulus features congruently activate the same response. The performance difference between incompatible and compatible trials indicates the degree of interference and is termed the Simon effect (for a review, see Lu & Proctor, 1995). Numerous studies have shown that interference from irrelevant stimuli (i.e., the Simon effect) is reduced following conflict trials compared with trials without conflict (e.g., Fischer, Dreisbach, & Goschke, 2008; Kerns et al., 2004; Stuermer, Leuthold, Soetens, Schroeter, & Sommer, 2002; see Figure 1, left). These trial-to-trial sequential modulations of interference effects have often been taken as index of adjustments of cognitive control (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Gratton, Coles, & Donchin, 1992).[1] Importantly, recent studies have used these trial-

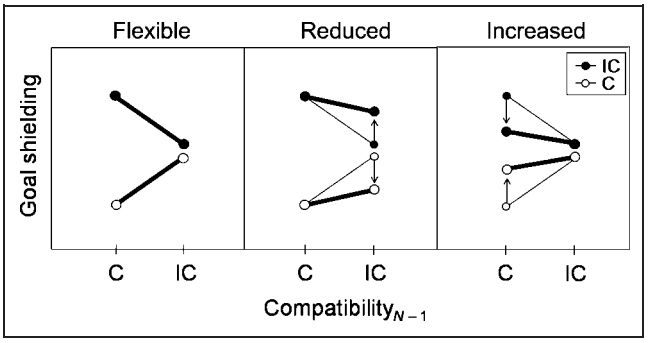

Figure 1. Sequential modulation of the Simon effect representing the administered amount of goal shielding. The *flexible* adjustment of goal shielding (left) is reflected in increased goal shielding after conflict and relaxed goal shielding following nonconflict trials. If stress leads to overall *reduced* goal shielding, interference effects should also be large after conflict trials (center). If stress leads to generally *increased* goal shielding, interference should be small also in conditions without previous conflict (right).

to-trial sequential modulations to study the flexibility of cognitive control adjustments under the influence of varying moods or reward contingencies (e.g., van Steenbergen, Band, & Hommel, 2009, 2010), thus validating this approach for the present study. We therefore used trial-to-trial sequential dependencies of interference effects as a measure of cognitive flexibility, which represents the dynamic adjustment of goal shielding to varying task demands such as the presence or absence of a response conflict. In particular, reduced Simon effects on trials following conflict trials were taken to indicate increased goal shielding, whereas larger Simon effects after trials without conflict indicate relaxed goal shielding.

Given the aforementioned link between the experience of stress and flexibility in action control, a first plausible prediction holds that acute psychosocial stress might lead to tonically *insufficient* (or overall relaxed) goal shielding. On the basis of the idea of impaired cognitive control functions under stress, one should expect generally increased interference effects by conflicting task-irrelevant information irrespective of whether the previous trial involved a response conflict. In other words, tonically insufficient goal shielding should turn the sequential modulation of interference effects (trial-to-trial adaptation) into an additive pattern of large interference irrespective of previous conflict history (Figure 1, center).

Second, and according to the idea of increased selectivity under stress, acute psychosocial stress might lead to tonically *increased* goal shielding, reflecting a compensatory increase of mental effort. The stress-induced narrowing of attention to task-relevant processing reduces interference by task-irrelevant information. Therefore, we expect that increased goal shielding, reflected in significant interference reduction, is not only evident in trials following conflict trials but also when following nonconflict trials (Figure 1, right).

Finally, acute psychosocial stress may also selectively lead to phasic increases of goal shielding as a response to conflict and, thus, should promote interference reduction exclusively in postconflict trials. In this conception, however, stress facilitates cognitive flexibility, such as the dynamic allocation of attentional control to specific task demands.

METHODS

Participants

Forty-eight healthy, medication-free volunteers (24 men, 19–29 years; mean age = 22.46 years, SD = 2.41 years) of normal weight (body mass index between 18 and 27; mean body mass index = 22.18, SD = 2.37) with normal or corrected-to-normal vision took part in the experiment. Because previous studies showed attenuated physiological stress responses in terms of lower stress-related saliva-free cortisol levels due to both habitual smoking (see review by Rohleder & Kirschbaum, 2006) and oral contraceptive intake (e.g., Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999), all participants were nonsmokers and female volunteers did not use hormone-based birth control. Participants gave their written informed consent before their inclusion in the study in accordance to the 1964 Declaration of Helsinki and received financial compensation for expenses.

Stress Induction and Stress Validation

Participants were randomly assigned to a stress group and a control group, respectively (12 men and 12 women per group). The stress group was exposed to the TSST protocol (Kirschbaum et al., 1993) which consists of a public speech, a mental arithmetic task (both in front of a committee) following an anticipatory period (total time: 15 min). Participants of the control group underwent a standardized control situation designed to resemble the TSST but without stress-inducing features (for details, see Het et al., 2009).

Saliva samples were collected using Salivette sampling devices (Sarstedt, Nümbrecht, Germany) at eight measurement time points (i.e., 15, 5, and 1 min before treatment started and 1, 10, 20, 30, and 40 min after end of treatment) for later analysis of sAA activity and free cortisol levels. They were assessed as markers of the individual physiological stress response, that is, of SNS and HPA axis activity, respectively. sAA activity was obtained by applying a quantitative enzyme kinetic method (cf. Rohleder & Nater, 2009). Free cortisol levels were analyzed using a chemiluminescence immunoassay (IBL International, Hamburg, Germany). Subjective individual stress levels were assessed with the mental state questionnaire MDBF (Steyer et al., 1997) on three dimensions (i.e., good mood vs. bad mood, calmness vs. restlessness, and alertness vs. fatigue). Participants self-reported their current mental state by answering the MDBF at five measurement time points (15 and 1 min before the start of treatment and 1, 20, and 40 min after the treatment ended).

Simon Task

We used a number versions of the Simon task (Fischer et al., 2008). Target stimuli (digits 1–9, except 5) were displayed white against black on a 17-in. monitor of an IBM-compatible personal computer. Viewing distance was approximately 60 cm. Targets (0.48°–0.67°) were presented 2.8 cm either to the left or to the right of a fixation (plus sign) resulting in a visual angle of 2.7° to the left and right, respectively. On a standard QWERTZ keyboard, participants responded with the left (Alt key) or right (Alt-Gr key) index finger to digits smaller or larger than five, respectively. Stimulus presentation and data recording were based on Presentation software (version 0.71; Neurobehavioral Systems, Inc., Albany, CA).

Each trial began with the presentation of the fixation sign. After 1000 msec, the target was added for 200 msec. The fixation sign remained until a response was given (1600 msec

max). A blank screen served as feedback (300 msec) for correct responses. Feedback for misses "zu langsam" (too slow) and for erroneous responses "falsch" (false) was accompanied by an auditory signal through headphones (sinus tone). Subsequently, the screen went blank for a random interval between 100 and 1000 msec.

Action–State Orientation

The ACS-90 questionnaire (Kuhl, 1994) assesses action versus state orientation and allows to differentiate between individuals that behave more action-oriented and individuals that primarily act state-oriented. We were particularly interested in the preoccupation dimension aimed at measuring the ability to detach from thoughts about an unpleasant past experience. The ACS provides a criterion based on the dimension total score (0–12) that allows to distinguish the two groups of action-oriented (<5) and state-oriented individuals (≥5) with regard to preoccupation. Action-oriented individuals are assumed to successfully refrain from rethinking unpleasant past events, while state-oriented individuals fail to do so. Because psychosocial stress represents an unpleasant situation, we aimed at addressing whether more action- or more state-oriented individuals (21 and 27 participants, respectively) react differently to the stressor and moreover if potential stress effects on cognitive performance within the present study are affected by this personality dimension.

Procedure

A cognitive training introduced participants to the Simon task and aimed to minimize practice effects during cognitive testing after treatment (160 trials). At 20 min after arrival, participants were exposed to either the TSST or the standardized control situation. After an interval of 5 min, the cognitive testing period consisting of three blocks with 256 trials each (total number of trials = 768) followed. Testing took place between noon and 6.30 p.m. to minimize variance due to circadian variations in cortisol levels. Because both instantaneous food and caffeine intake were found to significantly affect salivary cortisol response to stress (Lovallo, Farag, Vincent, Thomas, & Wilson, 2006; Gonzalez-Bono, Rohleder, Hellhammer, Salvador, & Kirschbaum, 2002), participants were asked to refrain from eating and consumption of sugar- or caffeine-containing drinks 2 hr before testing. Because acute glucose availability is however a condition precedent for a stress-related HPA activity increase (Kirschbaum et al., 1997), we aimed at standardizing blood glucose levels by providing all participants with 200 mL grape juice at the beginning of the session.

Data Analysis

To analyze changes in physiological and subjective stress level over the experimental session, ANOVAs with the within-subjects factor measurement time point (eight or five levels, respectively) and the between-subject factor stress (stress vs. no stress) were conducted on sAA, salivary cortisol, and total scores of the three MDBF dimensions, respectively. Regarding cognitive performance, the within-subject factors compatibility$_{N-1}$ (incompatible vs. compatible), compatibility$_N$ (incompatible vs. compatible), and block (1–3), and the between-subject factors stress (stress vs. no stress) and action–state orientation (action-oriented vs. state-oriented) were entered into ANOVAs on error rates and mean RTs, respectively. Both the first trial of each block and post-error trials were excluded (4.86%). In addition, trials with identical target repetitions were omitted (11.02%). For RT analysis only, error trials (3.37%) and RT differing more than 2.5 standard deviations from the mean RT of each individual cell mean (2.73%) were also excluded. Greenhouse–Geisser correction was applied where necessary.

RESULTS

Stress Response

Neuroendocrine Measures

ANOVAs on sAA and cortisol revealed differences in the time course between the stress group and the control group, $F(7, 315) = 3.78, p < .01, \eta^2 = .08$ and $F(7, 315) = 10.78, p < .001, \eta^2 = .19$, respectively.[2] The stress group displayed higher sAA levels than the control group 15 min prior and, more importantly, only up to 10 min following treatment, ps < .05, one-tailed. Regarding cortisol, levels were higher in stressed participants compared with volunteers of the control group at 10, 20, 30, and 40 min after treatment, all ps < .05, one-tailed (see Figure 2).

Mental State

The MDBF revealed significant differences between the stress group and the control group on the two dimensions good versus bad mood and calmness versus restlessness, $F(4, 184) = 10.58, p < .001, \eta^2 = .19$ and $F(4, 184) = 6.02, p < .001, \eta^2 = .12$. Compared with the control group, volunteers exposed to the TSST reported both worse mood and more restlessness after being instructed for treatment during the anticipatory period, $t(46) = 2.16, p < .05$, one-tailed, and $t(46) = 2.67, p < .01$, one-tailed, as well as immediately following treatment, $t(46) = 4.51, p < .001$, one-tailed, and $t(46) = 2.51, p < .01$, one-tailed. The overall increase in fatigue over time, $F(4, 184) = 11.39, p < .001, \eta^2 = .20$, was independent of stress, as was the mean fatigue level, both Fs < 1 (see Figure 3).

Cognitive Performance

Error Rates

Error rates showed a significant main effect of Simon compatibility$_N$ (incompatible: 3.89%, compatible: 2.82%), $F(1,$

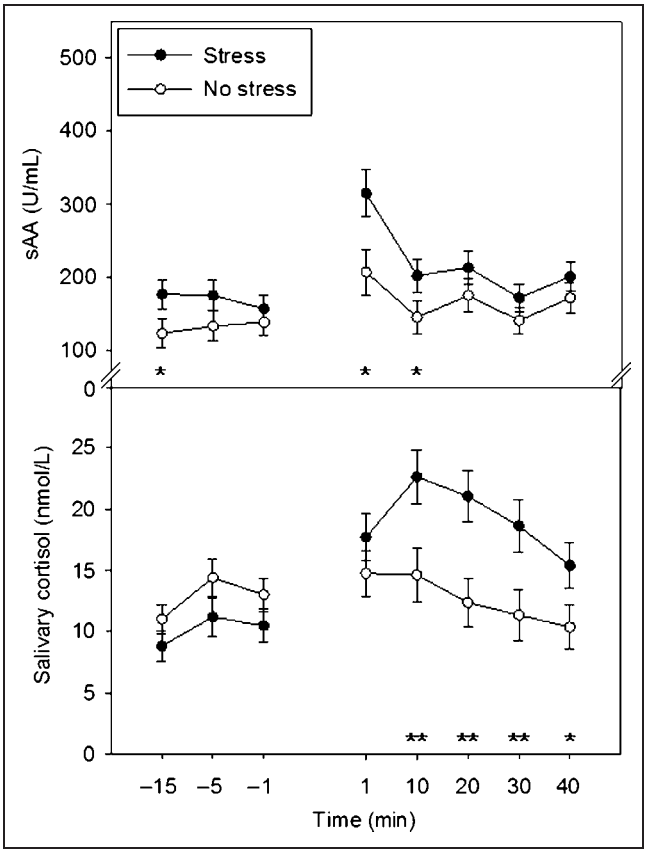

Figure 2. Mean levels of sAA and salivary cortisol as a function of time (minutes before or after treatment) for the stress group and the control group, respectively. Error bars represent *SEM*s (n = 47). *p < .05, one-tailed. **p < .01, one-tailed.

44) = 5.20, p < .05, η^2 = .11. Furthermore, the Simon effect was significantly reduced following conflict trials compared with nonconflict trials, $F(1, 44) = 48.22$, p < .001, η^2 = .52. Although stress did not influence overall error rates or compatibility$_N$, both Fs < 1, acute psychosocial stress reduced the trial-to-trial sequential modulation of compatibility,[3] $F(1, 44) = 4.45$, p < .05, η^2 = .09. In particular, the stress group compared to the control group showed a smaller Simon effect following nonconflict trials, $t(46)$ = 1.90, p < .05 (one-tailed) with no differences between both groups following conflict trials, $t(46) = 0.73$, p = .94.

The observed effect of stress on the sequential modulation was found to vary with the duration of cognitive testing, $F(2, 88) = 3.92$, p < .05, η^2 = .08. Post hoc analyses revealed that, within the stress group, the sequential modulation of the Simon effect decreased over time, $F(1, 22)$ = 5.53, p < .05, η^2 = .20 (linear). In contrast, an increase of the sequential modulation over time was found for the control group, $F(1, 22) = 4.47$, p < .05, η^2 = .17 (linear). Separate post hoc ANOVAs for the three blocks showed that with regard to the interaction Compatibility$_{N-1}$ × Compatibility$_N$ both groups did not differ in the first and second block of the cognitive testing, F < 1 and $F(1, 46)$ = 1.62, p = .21, η^2 = .03, respectively. However, within the third block, the stress group showed a significantly reduced sequential modulation compared with the control group, $F(1, 46) = 8.28$, p < .01, η^2 = .15 (see Figure 4). Again, especially after nonconflict trials, the Simon effect was much smaller for the stress group than for the control group, $t(46) = 2.29$, p < .05. Yet, the reduction in the Simon effect did not differ between groups after conflict trials, t < 1. No further effects of the factor block reached significance, all ps > .25. The finding of a time-dependent

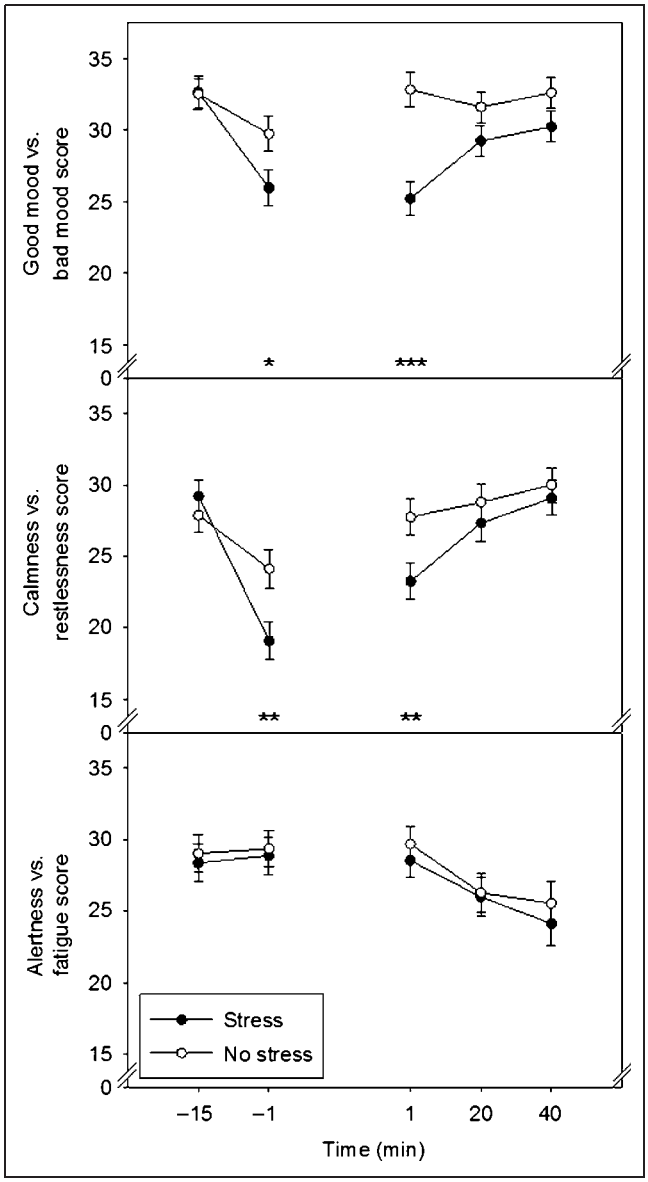

Figure 3. Mental state assessed on the three dimensions good mood versus bad mood, calmness versus restlessness, and alertness versus fatigue assessed with the mental state questionnaire MDBF (Steyer et al., 1997) as a function of time (minutes before or after treatment) for the stress group and the control group, respectively. The measurement −1 for mental state was conducted directly after instruction for the subsequent treatment (i.e., TSST or standardized control situation) as part of the anticipatory period. Error bars represent *SEM*s. *p < .05, one-tailed. **p < .01, one-tailed. ***p < .001, one-tailed.

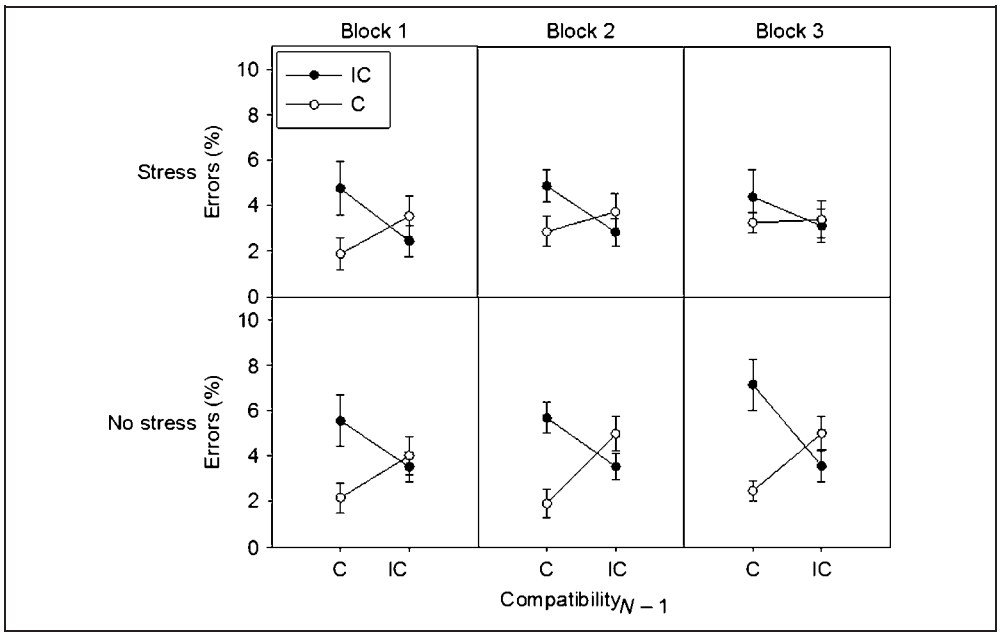

Figure 4. Compatibility in trial$_N$ as a function of compatibility in trial$_{N-1}$ (sequential modulation) across blocks for the stress group and the control group, respectively (IC = incompatible, C = compatible). Error bars represent *SEM*s.

stress effect on the cognitive flexibility measure was further substantiated by a significant negative correlation between the total increase[4] of salivary cortisol but not sAA following treatment and the amount of cognitive flexibility exclusively in the final block of testing (see Table 1).

Regarding action–state orientation, state-oriented individuals showed a numerically smaller Simon effect (0.21%) compared with action-oriented individuals (1.94%). This effect, however, failed the conventional level of statistical significance, $F(1, 44) = 3.40, p = .07, \eta^2 = .07$. No main effect of action–state orientation on error rates as well as no further interactions with one or more of the other factors reached significance, all ps $> .23$. An additional analysis further revealed neither a main effect of gender nor any interaction with one or more of the other factors, all ps $> .05$. Most importantly, the observed stress effect on the sequential modulation of the Simon effect was similar in male and female participants, $F < 1$.

Table 1. Correlations between the Total Increase of Neuroendocrine Stress Measures following Treatment and the Amount of Cognitive Flexibility for the Three Experimental Blocks

	Cognitive Flexibility		
Neuroendocrine Measures	*Block 1*	*Block 2*	*Block 3*
Salivary cortisol			
r	−0.02	−0.20	−0.41**
P	0.87	0.18	<0.01
sAA			
r	0.07	−0.27	−0.22
P	0.63	0.07	0.14

$n = 47$. Cognitive flexibility was calculated as index of the sequential modulation of interference effects (for a detailed description, see van Steenbergen et al., 2009). Neuroendocrine measures were calculated as area under the curve with respect to increase on logarithmized data (Pruessner et al., 2003).

**$p < .01$.

RT

RT analysis showed a main effect of compatibility$_N$ (incompatible: 507 msec, compatible: 491 msec), $F(1, 44) = 54.24, p < .001, \eta^2 = .55$. This Simon effect was eliminated following conflict trials (−6 msec) compared with nonconflict trials (38 msec), $F(1, 44) = 149.80, p < .001, \eta^2 = .77$. There was no main effect of stress, $F < 1$. The interaction between action–state orientation and compatibility$_N$ was not significant, $F(1, 44) = 2.95, p = .09, \eta^2 = .06$, mirroring the error data. Moreover, stress and/or action orientation did not interact with any other factor or factorial combination, all ps $> .09$.

DISCUSSION

This study provides evidence that acute psychosocial stress does not inevitably impair cognitive functioning. When exposed to an uncontrollable and novel stressful situation, our volunteers increased goal shielding to reduce interference in an experimental task. This increased focusing came, however, at the cost of reduced flexibility in situational control adjustments over time. In addition, our data are the first demonstration that the effects of acute psychosocial stress on cognitive performance parallel the time course of the HPA stress-response kinetic.

This experiment aimed at investigating the influence of acute psychosocial stress on cognitive flexibility as reflected

in the dynamic regulation of goal shielding to situational task demands. The results are rather straight forward: Whereas the nonstressed controls displayed the expected flexible adjustment of goal shielding (indicated by trial-to-trial adaptation with context-sensitive control adjustment), stressed individuals displayed a different cognitive response pattern. Although stress did not affect overall performance when solving response conflicts (Wolf et al., 2001), the trial-to-trial sequential analysis revealed that acute psychosocial stress affected the flexibility in context-sensitive control adjustments. Although the experience of a response conflict in the Simon task also resulted in reduced interference effects in the subsequent trial (like the control group); however, in contrast to the control group, such increased levels of goal shielding were not only found in trials following a response conflict but also in trials that did not call for control adjustments (i.e., trials without a conflict history in the previous trial). Put differently, whereas the control group was able to relax the allocation of attentional control (reduced goal shielding) when no adaptation was required because of the absence of conflict in $N - 1$, stressed participants showed *tonically* increased goal shielding which reduced interference irrespective of previous conflict experience.

This observation neither supports the view of increased interference under stress, nor do the data corroborate the prediction of an increased selective (phasic) conflict-related goal shielding. The present data rather converge with the assumption that diminished attentional resources under stress are specifically allocated to task-relevant processing. Although this narrowing of attentional focus ensures reliable task performance in challenging situations, it comes at the cost of reduced cognitive flexibility: Tonically increased goal shielding does not allow for the flexible adjustment of attentional control to specific varying task demands. In this respect, the observed behavioral adjustment that arises under stress does not constitute an impairment condition per se. In fact, increased goal shielding as a response to extreme stress experience might also reflect performance benefits given stable environments that call for a consistent focus on one particular action or task goal only.

To the best of our knowledge, our results also provide the first demonstration that the increase of administered goal shielding develops gradually over time (i.e., with delay to stress exposure) closely following the cortisol-response pattern (see below). Stress and control group did not differ in their performance immediately after treatment (i.e., TSST vs. standardized control situation) but displayed substantial differences in overall goal shielding at the end of the experimental session (Block 3). In fact, the higher the total increase of cortisol levels, the stronger was the reduction in the sequential modulation indexing increased goal shielding in Block 3. This finding is particularly important as it converges with recent observations in animal research describing the time course of the endocrinological response of stress, for example, delayed glucocorticoid-dependent responses in the hippocampus (Droste et al., 2008).

A major advantage of the present study is the administration of a standardized stress induction protocol (TSST) that allowed for biological validation of the individual stress response. As typical for the HPA axis, the full-blown salivary cortisol response developed with increasing delay to the stress exposure. The SNS also showed the expected stress response with increased sAA levels immediately after the end of the TSST, returning to baseline levels shortly after stress exposure with no significant difference between the two experimental groups during the cognitive testing period. In line with this, the MDBF questionnaire revealed an increased arousal level and worsened mood for the stress group immediately before and after the stress exposure. The observed stress effects on the behavioral measures (i.e., trial-to-trial adaptation) were most likely not caused by changes in mood, arousal, or fatigue because the decreased trial-to-trial adaptation was most pronounced in the final block when these subjective participants no longer differed between stressed and control subjects. Furthermore, goal shielding was tonically increased under the experience of acute psychosocial stress irrespective of action versus state orientation.

It should be noted though that acute psychosocial stress affected primarily the likelihood of error commitment but not the speed of responses. At present, we do not have an explanation for this specific modulation. Our RT data, however, do not compromise the interpretation of the error data. As we find this pattern of stress effects repeatedly in our laboratory (e.g., Plessow, Kiesel, & Kirschbaum, 2009), we suggest that further studies investigating effects of stress on cognitive performance should implement tests that provide sufficiently high error rates to avoid potential floor effects.

Our findings of stress-induced and time-dependent effects on goal shielding and cognitive flexibility support and extend findings from related fields of research. In the study of memory, for example, stress has been shown to selectively benefit rigid "habit" memory over more flexible "cognitive" memory (for a review, see Schwabe, Wolf, & Oitzl, 2010). Beversdorf and colleagues (Ishizuka, Hillier, & Beversdorf, 2007; Beversdorf, Hughes, Steinberg, Lewis, & Heilman, 1999) primarily addressed the adrenergic system of the stress response and demonstrated that its modulations (pharmacologically or by means of the cold pressure test), during creative thinking and problem-solving tasks reduced cognitive flexibility. Similarly, a recent study by Colzato, Kool, and Hommel (2008) also showed that a repeated application of the cold pressure test in between cognitive testing reduced the efficiency in low-level visuo-motor binding processes. Although all these studies seem to provide converging evidence of reduced cognitive flexibility under stress or as a response to stress, it is also obvious that a clear definition of the targeted constructs of cognitive flexibility, stress and the investigated stress components is essential to appreciate

each individual finding and to allow comparisons between them. At the same time, the diversity in research strategies provides fertile grounds to assemble a holistic picture of the interaction between various forms of stress on cognitive functions.

Finally, our finding of increased goal shielding under the experience of stress does not contradict recent observations of stress impairing working memory functions (e.g., Luethi, Meier, & Sandi, 2009; Schoofs, Preuss, & Wolf, 2008; but see Smeets, Jelicic, & Merckelbach, 2006; Kuhlmann, Piel, & Wolf, 2005). Again, these divergent results call for a clear definition of the investigated components of prefrontal executive control as such control functions are manifold (Miyake et al., 2000). Furthermore, it highlights the investigated time line of stress effects on cognitive functions, as Schoofs and colleagues (2008) found stress effects on working memory only immediately after stress exposure (suggesting an immediate sympathetic effect), whereas in the present study effects of stress increased with increasing delay to the stress experience (rather suggestive of a cortisol effect). Additional studies are now needed to disentangle the specific contributions of different biological (response) systems to variation of cognitive control in times of acute stress. These experiments may ultimately help to understand how cognitive adaptation is reorganized in response to changing environmental demands.

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Notes

1. It should be noted that the exact underlying mechanism(s) responsible for the sequential modulation of interference effects is still under debate, as previous research has shown that various aspects of low-level repetition and feature binding processes (e.g., Hommel, Proctor, & Vu, 2004) and cognitive control processes (Botvinick et al., 2001) are likely to contribute in different extents to the sequential pattern (for a review, see Egner, 2007). Although feature binding processes are hard (or rather impossible) to fully exclude, a number of precautions serve to minimize their contribution. To this end, we particularly used a Simon task with four stimuli for each response alternative, used large response–stimulus intervals, excluded identical stimulus repetitions and considered control analyses with the factor response repetition, to feel confident that control processes contribute to the trial-to-trial sequential modulation (but see Dutzi & Hommel, 2009). Moreover, from a theoretical perspective, accounts of sequential modulations of congruence effects in terms of control adaptation or episodic retrieval may not be mutually exclusive, but closely linked. In particular, it has been suggested that episodic bindings may include bindings between a task and control settings (e.g., the degree of goal shielding), which are retrieved on subsequent trials and thereby determine control settings depending on previous task episodes (Spape & Hommel, 2008). Thus, episodic retrieval of control settings may be a possible mechanism underlying flexible control adjustments.
2. Salivary samples of one participant could not be analyzed due to technical problems.
3. The trial-to-trial sequential modulation and its interaction with stress were not modulated by the additional factor response repetition/switch (all $ps > .24$).
4. Calculated as area under the curve with respect to increase on logarithmized data (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) based on the measurement time point level immediately before treatment (i.e., −1 min) as well as all measurement time points after treatment (i.e., +1, +10, +20, +30, and +40 min after treatment ended) for each individual participant, respectively.

REFERENCES

Alexander, J. K., Hillier, A., Smith, R. M., Tivarus, M. E., & Beversdorf, D. Q. (2007). Beta-adrenergic modulation of cognitive flexibility during stress. *Journal of Cognitive Neuroscience, 19,* 468–478.

Arnsten, A. F. T. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience, 10,* 410–422.

Barsegyan, A., Mackenzie, S. M., Kurose, B. D., McGaugh, J. L., & Roozendaal, B. (2010). Glucocorticoids in the prefrontal cortex enhance memory consolidation and impair working memory by a common neural mechanism. *Proceedings of the National Academy of Sciences, U.S.A., 107,* 16655–16660.

Beversdorf, D. Q., Hughes, J. D., Steinberg, B. A., Lewis, L. D., & Heilman, K. M. (1999). Noradrenergic modulation of cognitive flexibility in problem solving. *NeuroReport, 10,* 2763–2767.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108,* 624–652.

Braver, T. S., & Cohen, J. D. (1999). Dopamine, cognitive control, and schizophrenia: The gating model. *Progress in Brain Research, 121,* 327–349.

Chajut, E., & Algom, D. (2003). Selective attention improves under stress: Implications for theories of social cognition. *Journal of Personality and Social Psychology, 85,* 231–248.

Colzato, L. S., Kool, W., & Hommel, B. (2008). Stress modulation of visuomotor binding. *Neuropsychologia, 46,* 1542–1548.

Davis, J., Morrill, R., Fawcett, J., Upton, V., Bondy, P. K., & Spiro, H. M. (1962). Apprehension and elevated serum cortisol levels. *Journal of Psychosomatic Research, 6,* 83–86.

de Kloet, E. R., Joels, M., & Holsboer, F. (2005). Stress and the brain: From adaptation to disease. *Nature Reviews Neuroscience, 6,* 463–475.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130,* 355–391.

Droste, S. K., de Groote, L., Atkinson, H. C., Lightman, S. L., Reul, J. M., & Linthorst, A. C. (2008). Corticosterone levels in the brain show a distinct ultradian rhythm but a delayed response to forced swim stress. *Endocrinology, 149,* 3244–3253.

Dutzi, I. B., & Hommel, B. (2009). The microgenesis of action–effect binding. *Psychological Research, 73,* 425–435.

Egner, T. (2007). Congruency sequence effects and cognitive control. *Cognitive, Affective, & Behavioral Neuroscience, 7,* 380–390.

Fischer, R., Dreisbach, G., & Goschke, T. (2008). Context-sensitive adjustments of cognitive control: Conflict-adaptation effects are modulated by processing demands of the ongoing task. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 34,* 712–718.

Gonzalez-Bono, E., Rohleder, N., Hellhammer, D. H., Salvador, A., & Kirschbaum, C. (2002). Glucose but not protein or fat load amplifies the cortisol response to psychosocial stress. *Hormones and Behavior, 41,* 328–333.

Goschke, T. (2000). Intentional reconfiguration and involuntary persistence in task set switching. In S. Monsell & J. Driver (Eds.), *Attention and performance: XVIII. Control of cognitive processes* (pp. 331–355). Cambridge, MA: MIT Press.

Goschke, T., & Dreisbach, G. (2008). Conflict-triggered goal shielding: Response conflicts attenuate background monitoring for prospective memory cues. *Psychological Science, 19,* 25–32.

Gratton, G., Coles, M. G., & Donchin, E. (1992). Optimizing the use of information: Strategic control of activation of responses. *Journal of Experimental Psychology: General, 121,* 480–506.

Het, S., Rohleder, N., Schoofs, D., Kirschbaum, C., & Wolf, O. T. (2009). Neuroendocrine and psychometric evaluation of a placebo version of the "Trier Social Stress Test." *Psychoneuroendocrinology, 34,* 1075–1086.

Hommel, B., Proctor, R. W., & Vu, K. P. (2004). A feature-integration account of sequential effects in the Simon task. *Psychological Research, 68,* 1–17.

Ishizuka, K., Hillier, A., & Beversdorf, D. Q. (2007). Effect of the cold pressor test on memory and cognitive flexibility. *Neurocase, 13,* 154–157.

Kerns, J. G., Cohen, J. D., MacDonald, A. W., III, Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science, 303,* 1023–1026.

Kirschbaum, C., Gonzalez Bono, E., Rohleder, N., Gessner, C., Pirke, K. M., Salvador, A., et al. (1997). Effects of fasting and glucose load on free cortisol responses to stress and nicotine. *Journal of Clinical Endocrinology and Metabolism, 82,* 1101–1105.

Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology, 19,* 313–333.

Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus–pituitary–adrenal axis. *Psychosomatic Medicine, 61,* 154–162.

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The "Trier Social Stress Test"—A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28,* 76–81.

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. *Cognition and Emotion, 20,* 577–595.

Kuhl, J. (1994). Action versus state orientation: Psychometric properties of the action control scale (ACS-90). In J. Kuhl & J. Beckmann (Eds.), *Volition and personality: Action versus state orientation* (pp. 47–59). Seattle, WA: Hogrefe.

Kuhlmann, S., Piel, M., & Wolf, O. T. (2005). Impaired memory retrieval after psychosocial stress in healthy young men. *Journal of Neuroscience, 25,* 2977–2982.

Liston, C., McEwen, B. S., & Casey, B. J. (2009). Psychosocial stress reversibly disrupts prefrontal processing and attentional control. *Proceedings of the National Academy of Sciences, U.S.A., 106,* 912–917.

Liston, C., Miller, M. M., Goldwater, D. S., Radley, J. J., Rocher, A. B., Hof, P. R., et al. (2006). Stress-induced alterations in prefrontal cortical dendritic morphology predict selective impairments in perceptual attentional set-shifting. *The Journal of Neuroscience, 26,* 7870–7874.

Lovallo, W. R., Farag, N. H., Vincent, A. S., Thomas, T. L., & Wilson, M. F. (2006). Cortisol responses to mental stress, exercise, and meals following caffeine intake in men and women. *Pharmacology, Biochemistry, and Behavior, 83,* 441–447.

Lu, C.-H., & Proctor, R. W. (1995). The influence of irrelevant location information on performance: A review of the Simon and spatial Stroop effects. *Psychonomic Bulletin & Review, 2,* 174–207.

Luethi, M., Meier, B., & Sandi, C. (2009). Stress effects on working memory, explicit memory, and implicit memory for neutral and emotional stimuli in healthy men. *Frontiers in Behavioral Neuroscience, 2,* 1–9.

Mansouri, F. A., Tanaka, K., & Buckley, M. J. (2009). Conflict-induced behavioural adjustment: A clue to the executive functions of the prefrontal cortex. *Nature Reviews Neuroscience, 10,* 141–152.

Mason, J. W. (1968). A review of psychoendocrine research on the pituitary-adrenal cortical system. *Psychosomatic Medicine, 30(Suppl.),* 576–607.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24,* 167–202.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41,* 49–100.

Muller, J., Dreisbach, G., Brocke, B., Lesch, K. P., Strobel, A., & Goschke, T. (2007). Dopamine and cognitive control: The influence of spontaneous eyeblink rate, DRD4 exon III polymorphism and gender on flexibility in set-shifting. *Brain Research, 1131,* 155–162.

Nater, U. M., & Rohleder, N. (2009). Salivary alpha-amylase as a non-invasive biomarker for the sympathetic nervous system: Current state of research. *Psychoneuroendocrinology, 34,* 486–496.

Perlman, W. R., Webster, M. J., Herman, M. M., Kleinman, J. E., & Weickert, C. S. (2007). Age-related differences in glucocorticoid receptor mRNA levels in the human brain. *Neurobiology of Aging, 28,* 447–458.

Plessow, F., Kiesel, A., & Kirschbaum, C. (2009). The stressed prefrontal cortex: Acute psychosocial stress disturbs shifting and shielding in a task-switching setting. Paper presented at the 16th European Society for Cognitive Psychology (ESCoP) conference, Krakow, Poland.

Pruessner, J. C., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology, 28,* 916–931.

Rohleder, N., & Kirschbaum, C. (2006). The hypothalamic–pituitary–adrenal (HPA) axis in habitual smokers. *International Journal of Psychophysiology, 59,* 236–243.

Rohleder, N., & Nater, U. M. (2009). Determinants of salivary alpha-amylase in humans and methodological considerations. *Psychoneuroendocrinology, 34,* 469–485.

Schoofs, D., Preuss, D., & Wolf, O. T. (2008). Psychosocial stress induces working memory impairments in an *n*-back paradigm. *Psychoneuroendocrinology, 33,* 643–653.
Schwabe, L., Wolf, O. T., & Oitzl, M. S. (2010). Memory formation under stress: Quantity and quality. *Neuroscience and Biobehavioral Reviews, 34,* 584–591.
Simon, J. R. (1990). The effects of an irrelevant directional cue on human information processing. In R. W. Proctor & T. G. Reeve (Eds.), *Stimulus–response compatibility: An integrated perspective* (pp. 31–86). Amsterdam: North Holland.
Smeets, T., Jelicic, M., & Merckelbach, H. (2006). The effect of acute stress on memory depends on word valence. *International Journal of Psychophysiology, 62,* 30–37.
Spape, M. M., & Hommel, B. (2008). He said, she said: Episodic retrieval induces conflict adaptation in an auditory Stroop task. *Psychonomic Bulletin & Review, 15,* 1117–1121.
Steinhauser, M., Maier, M., & Hübner, R. (2007). Cognitive control under stress: How stress affects strategies of task-set reconfiguration. *Psycholological Science, 18,* 540–545.
Steyer, R., Schwenkmezger, P., Notz, P., & Eid, M. (1997). *Der mehrdimensionale Befindlichkeitsfragebogen (MDBF) [The multidimensional mental-state questionnaire (MDBF)]*. Göttingen: Hogrefe.
Stuermer, B., Leuthold, H., Soetens, E., Schroeter, H., & Sommer, W. (2002). Control over location-based response activation in the Simon task: Behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception and Performance, 28,* 1345–1363.
van Steenbergen, H., Band, G. P., & Hommel, B. (2009). Reward counteracts conflict adaptation. Evidence for a role of affect in executive control. *Psychological Science, 20,* 1473–1477.
van Steenbergen, H., Band, G. P., & Hommel, B. (2010). In the mood for adaptation: How affect regulates conflict-driven control. *Psychological Science, 21,* 1629–1634.
Wegner, D. M., & Erber, R. (1992). The hyperaccessibility of suppressed thoughts. *Journal of Personality and Social Psychology, 63,* 903–912.
Wells, A., & Matthews, G. (1994). *Attention and emotion: A clinical perspective*. Hillsdale, NJ: Erlbaum.
Wolf, O. T. (2009). Stress and memory in humans: Twelve years of progress? *Brain Research, 1293,* 142–154.
Wolf, O. T., Convit, A., McHugh, P. F., Kandil, E., Thorn, E. L., De Santi, S., et al. (2001). Cortisol differentially affects memory in young and elderly men. *Behavioral Neuroscience, 115,* 1002–1011.