Hydrocortisone Counteracts Adverse Stress Effects on Dual-Task Performance by Improving Visual Sensory Processes

Lisa J. Weckesser[1], Nina C. Alexander[1,2], Clemens Kirschbaum[1], Eva Mennigen[3,4], and Robert Miller[1]

Abstract

■ The impact of acute stress on executive processes is commonly attributed to glucocorticoid-induced disruptions of the pFC. However, the occipital cortex seems to express a higher density of glucocorticoid receptors. Consequently, acute stress effects on executive processes could as well be mediated by glucocorticoid (e.g., cortisol)-induced alterations of visual sensory processes. To investigate this alternative route of stress action by demarcating the effects of acute stress and cortisol on executive from those on visual sensory processes, 40 healthy young men completed a standardized stress induction (i.e., the Trier Social Stress Test) and control protocol in two consecutive sessions. In addition, they received either a placebo or hydrocortisone (0.12-mg/kg bodyweight) pill and processed a dual and a partial report task to assess their executive and visual sensory processing abilities, respectively. Hydrocortisone administration improved both partial report and dual-task performance as indicated by increased response accuracies and/or decreased RTs. Intriguingly, the hydrocortisone-induced increase in dual-task performance was completely mediated by its impact on partial report performance (i.e., visual sensory processes). Moreover, RT measures in both tasks shared approximately 26% of variance, which was only in part attributable to hydrocortisone administration ($\Delta R^2 = 8\%$). By contrast, acute stress selectively impaired dual-task performance (i.e., executive processes), presumably through an alternative route of action. In summary, the present results suggest that cortisol secretion (as mimicked by hydrocortisone administration) may counteract adverse residual stress effects on executive processes by improving visual sensory processes (e.g., the maintenance and amplification of task-relevant sensory information). ■

INTRODUCTION

A fundamental challenge in cognitive neuroscience is to understand how acute stress impacts on executive processes. Initially, acute stress was believed to globally compromise the efficiency of pFC functioning, thereby impairing executive processes (Keinan, 1987; see Arnsten, 2009, for a review). These stress-induced impairments were attributed to catecholamines and glucocorticoids that are secreted whenever an individual is confronted with novel, uncontrollable, and/or ego-threatening situations (Dickerson & Kemeny, 2004). Specifically, the unbound fraction of circulating glucocorticoids like cortisol is able to diffuse through the blood–brain barrier and to affect pFC functioning by the activation of glucocorticoid receptors (Arnsten, 2009; Perlman, Webster, Herman, Kleinman, & Weickert, 2007; Carrasco & Van de Kar, 2003).

Adopting this line of reasoning, the even higher density of glucocorticoid receptors in the occipital cortex suggests that the impact of cortisol on this brain region might also contribute to stress-induced changes of executive task performances (Öğmen, Ekiz, Huynh, Bedell, & Tripathy, 2013; Perlman et al., 2007; Webster, Knable, O'Grady, Orthmann, & Weickert, 2002). Indeed, many executive tasks rely on the presentation of visual stimulus material. Accordingly, the early detection and selection of task-relevant visual information, which is mediated by the occipital cortex, constitutes an indispensable prerequisite for all subsequent processing levels (Miller, Weckesser, Smolka, Kirschbaum, & Plessow, 2015; Öğmen et al., 2013). Taking this reliance of executive task performance on a variety of cognitive processes into account (see also Miyake et al., 2000), stress-induced changes of executive processes are probably also driven by cortisol actions on more basic, visual sensory processes (Miller et al., 2015; Öğmen et al., 2013).

Indeed, such an alternative route of stress action on executive processes receives support by increasing evidence that suggests a more complex effect pattern of acute stress and cortisol exposure including improved (Stauble, Thompson, & Morgan, 2013; Oei, Tollenaar, Spinhoven, & Elzinga, 2009; Kofman, Meiran, Greenberg, Balas, & Cohen, 2006), impaired (Plessow, Kiesel, & Kirschbaum, 2012; Schoofs, Preuss, & Wolf, 2008; Steinhauser, Maier, & Hübner, 2007), and unaffected (Kuhlmann, Kirschbaum, & Wolf,

[1]Technische Universität Dresden, [2]Medical School Hamburg, [3]The University of New Mexico, [4]University Hospital Carl Gustav Carus

2005; Hoffman & Al'Absi, 2004; Monk & Nelson, 2002) indicators of executive processes. Although some of these mixed results have been attributed to time-dependent interactions of catecholamines and cortisol on distinct brain circuits (Shields, Bonner, & Moons, 2015; Hermans, Henckens, Joëls, & Fernández, 2014), differential cortisol effects on basic visual sensory versus executive processes might also contribute to some of these ambiguous findings (Miller et al., 2015). To investigate how cortisol and residual (i.e., all remaining physiological and psychological) stress components impact on visual sensory as compared with executive processes, this study draws on a partial report and a dual task, respectively (Pashler, 1994; Coltheart, 1980).

Cognitive Concepts of Partial Report and Dual-Task Performance

Usually, the partial report task serves to assess the so-called iconic memory (IM). IM is believed to enable the transient maintenance of all information from a visual scene (at a specific moment in time) that are subjected to subsequent change (Gugerty, 1998; Coltheart, 1980). While driving a car, for example, IM provides a schematic, spatial representation of nearby traffic (Gugerty, 1998). This representation facilitates higher-order responses such as the decision to dodge or to brake when a slowly moving vehicle suddenly changes to your freeway lane and there is not enough time left to check for the presence of other vehicles behind you or on adjacent lanes. However, IM also rapidly decays to prevent interference with subsequently encoded and thus putatively more relevant information such as other fast approaching vehicles (Gugerty, 1998; Gegenfurtner & Sperling, 1993). In partial report tasks, this rapid IM trace decay is experimentally challenged by introducing a delay (i.e., SOA) between the onset of a briefly presented but complex target stimulus and the onset of a spatial cue marking the subset of information that has to be reported from that target stimulus (Coltheart, 1980). Increasing this SOA reduces the probability for a correct retrieval of the cued stimulus features (Gegenfurtner & Sperling, 1993). Whereas the high performance under conditions of short SOAs constitutes the so-called partial report superiority effect (PRSE; Coltheart, 1980) that is attributed to the large capacity of IM, the performance decrease with increasing cue delays is traditionally explained by the limited retention period of IM (Gegenfurtner & Sperling, 1993). Henceforth, we will refer to these fundamental IM-governing processes of visual cognition as "visual sensory processes."

In contrast to partial report tasks, dual tasks serve to characterize our limited ability to process two tasks at once. This limitation is reflected by a response delay to either one or both of the two tasks (Miller, Ulrich, & Rolke, 2009; Pashler, 1994). Referring to the abovementioned driving example, the preoccupation with other devices such as cell phones or the car radio will postpone the response to any other stimulus, such as the decision to swerve or brake because of the sudden lane change of another vehicle. In analogy to such situations, traditional dual tasks continuously present two simple stimuli (S1 and S2) in rapid temporal succession. Whereas RTs to S1 are little influenced by a succeeding S2 presentation, mean RTs to S2 are markedly delayed as compared with the RT when S2 is presented in isolation. This RT slowing to S2 was termed psychological refractory period (PRP) effect and decreases when the temporal interval (SOA) between S1 onset and S2 onset increases (Pashler, 1994). Two of the most common model classes to explain the PRP effect are the standard bottleneck and the shared capacity models. According to standard bottleneck models, the PRP effect is caused by a central processing level that cannot simultaneously operate on multiple targets and/or responses at once, thereby forming a cognitive bottleneck (Kahneman, 1973). Thus, while this bottleneck engages in S1 processing, S2 processing has to wait until the former is completed. Because this waiting period delays response selection and/or execution to S2, RT to S2 is increased (Miller et al., 2009; Pashler, 1994). Alternatively, shared capacity models explain the PRP effect by a limited amount of mental resources that can be differentially allocated to S1 and/or S2 processing (Miller et al., 2009; Pashler, 1994). Whenever an equal amount of mental resources is allocated to the processing of S1 and S2, respectively, the total processing time of both tasks theoretically doubles. However, dual-task instructions prioritize the processing of S1. In consequence, a higher amount of resources is allocated to S1, which delays the subsequent S2 processing (Pashler, 1994). Although both model classes attribute the PRP effect to different sources, they agree that these PRP sources are located at hierarchically higher levels of information processing than IM. Most frequently, these sources are located at a level of response selection and/or decision-making in working memory (WM; Dux et al., 2009; Miller et al., 2009; Miyake et al., 2000; Pashler, 1994). Thus, dual tasks commonly serve to assess executive processes that coordinate the concurrent processing of multiple tasks (e.g., the inhibition of prepotent responses and the updating of WM contents; Miyake et al., 2000).

A Biophysical Network Model Integrating Partial Report and Dual-Task Performance

The outlined traditional cognitive concepts attribute limitations in partial report and dual-task processing to largely distinct levels of information processing (Pashler, 1994; Coltheart, 1980). However, the biophysical network model of Zylberberg, Dehaene, Mindlin, and Sigman (2009) provides an integrative conceptualization of the processes underlying information maintenance during the SOA period, which is a crucial design feature of both tasks (Miller et al., 2009; Coltheart, 1980). Basically, the SOA serves to constrain the optimal time of attention allocation that is required to (a) selectively report a cued

subset of information in partial report tasks or (b) respond to S2 in dual tasks after the prioritized S1 processing has been completed (Zylberberg et al., 2009; Finke et al., 2005; Pashler, 1994). Thus, the SOA period is not exclusively considered a passive waiting period (see also Miller et al., 2009, p. 280) but conceptualized as a period during which active information processing can be either beneficial or disadvantageous for successful task completion, depending on the demands of the processed task.

More precisely, the model assumes that task-relevant information must be actively maintained across the SOA period to become available to further processing levels like response selection or verbal report (Zylberberg et al., 2009; Dehaene, Sergent, & Changeux, 2003). In both tasks, stimulus presentation induces the formation of sensory representations of task-relevant and irrelevant information in IM. These initial representations are fragile because they are subjected to information decay because of neural competition and/or incoming noise. To prevent this decay, top–down amplification processes can be recruited to allocate all attentional resources to a limited subset of sensory information and initiate their subsequent transformation into more persistent WM representations (Zylberberg et al., 2009; Wong & Wang, 2006; Gegenfurtner & Sperling, 1993). However, this comes at the cost of disregarding any other sensory information that cannot be amplified anymore if the visual scene changes rapidly.

In the specific context of the partial report task, the task-relevant target information is unknown directly after the brief stimulus presentation. Thus, the immediate allocation of attentional resources should be actively withheld across the SOA period, thereby accepting the progressive decay of the transiently available sensory information until the cue is finally presented and attention can be directed to the target (see Figure 1A).[1] Thereafter, their transformation into more persistent representations, that is, the transfer of sensory information from IM to WM (Zylberberg et al., 2009; Wong & Wang, 2006; Gegenfurtner & Sperling, 1993), requires a certain amount of time during which all not-attended information further decays to a noninformative noise level. By contrast, dual tasks enable the immediate allocation of attentional resources to the sensory S1 representations (see Figure 1B). The resulting information transfer from IM to WM (which refers to response selection in this context) also requires a certain amount of time during which neural competition is biased toward S1 processing (Pashler, 1994) and all further incoming sensory information is subjected to decay. Consequently, the task-relevant sensory information that is provided by S2 can hardly be amplified whenever the SOA period is shorter than the period required for S1 response selection (see also Egner & Hirsch, 2005). To compensate for the resulting weaker sensory S2 representations that finally accumulate after the attention focus has switched (see Figure 1D), the period required for response selection needs to be extended. This will then give rise to the PRP effect.

In accordance with the predictions of this model, the massive change in partial report and dual-task performance

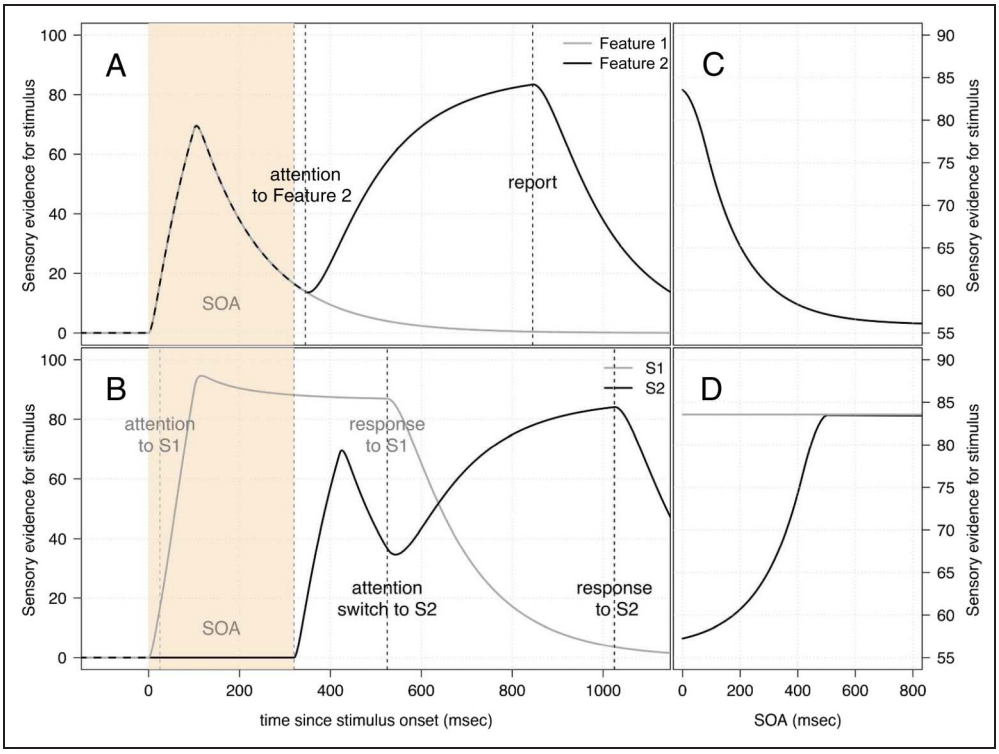

Figure 1. A model showing how sensory evidence is accumulated and used for response selection during one trial of (A) a partial report task and (B) a dual task: In both tasks, the presentation of the respective stimuli is supposed to provide information that is subjected to either subsequent attention-driven amplification or decay processes. In contrast to the partial report task (A), the top–down amplification of S2 in the dual-task setting (B) does not occur in synchrony with the SOA but is delayed because attentional resources are still allocated to S1 (in the illustrated scenarios, response selection is assumed to require 500 msec of directed attention). The resulting SOA dependencies of the sensory evidence that is accumulated after attention allocation for response selection are shown in C for the partial report task and in D for the dual task.

across SOAs occurs in the same time interval between 0 msec and approximately 1000 msec (e.g., Pashler, 1994, p. 222; Coltheart, 1980). Thus, the temporally delayed top–down amplification of (by then) noisy sensory information is the hypothesized proximate cause of both the PRSE and the PRP effect. Nonetheless, the PRP can only occur because of a preceding response selection process that binds the available attentional resources, and that cannot be distinguished from visual-sensory amplification processes by only investigating behavioral dual-task performance (cf. Kahneman, 1973, pp. 4–5).

The Impact of Acute Stress and Cortisol on Partial Report and Dual-Task Performance

The biophysical network model outlined in the previous section predicts that partial report and dual-task performances are susceptible to any modulation of amplification and decay processes during the SOA period. Crucially, these processes are accomplished by processing units that mimic slow acting, glutamatergic *N*-methyl-D-aspartate receptors (NMDARs; Zylberberg et al., 2009; Wong & Wang, 2006). In animal models, acute stress and glucocorticoids have been shown to modulate NMDAR-related neurotransmission and associated behavior (e.g., Barsegyan, Mackenzie, Kurose, McGaugh, & Roozendaal, 2010; Yuen et al., 2009). Therefore, stress-related cortisol secretion probably also impacts on NMDAR-related information processing during the SOA period in humans, which in turn should alter partial report and dual-task performances.

Indeed, there is first evidence suggesting that pharmacologically increased cortisol availability exerts a SOA-specific impact on partial report performance (Miller et al., 2015). More precisely, high cortisol availability reduced partial report performance under conditions of short SOAs but also seemed to improve partial report performance at long SOAs. Proceeding from the biophysical network model (Zylberberg et al., 2009), such an effect pattern could be explained by a cortisol-induced acceleration of information decay, which reduces the interference of task-irrelevant stimuli on the level of IM and in turn improves the cue-induced top–down amplifications under conditions of long SOAs. Similar cortisol-induced reductions of stimulus interference have also been reported for other cognitive tasks (Plessow, Fischer, Kirschbaum, & Goschke, 2011; Oei et al., 2009).

Regarding dual-task performance, Beste, Yildiz, Meissner, and Wolf (2013) found a significant improvement of processing efficiency (particularly faster S2 RTs) that was highly correlated with stress-induced cortisol secretion. By contrast, Plessow, Schade, Kirschbaum, and Fischer (2012) observed a stress-related impairment of dual-task performance (slower S2 RTs) that was virtually unrelated to stress-induced cortisol secretion. Considering this inconsistency and the inability to differentiate between visual sensory processes (e.g., amplification/decay) and executive processes (e.g., response selection) solely by means of dual tasks, it is relatively unclear how cortisol and residual stress components impact on each of the constituents of dual-task performance.

Aims of This Study

This study aims to investigate how acute stress and cortisol impact on partial report and dual-task performances. According to traditional cognitive notions, performance on both tasks is supposed to rely on comparably distinct levels of information processing (Beste, Stock, Ness, Epplen, & Arning, 2012; Miyake et al., 2000; Pashler, 1994). Therefore, any specific stress and/or cortisol impact on high-level dual-task performance should occur independently of their effects on low-level partial report performance (but not vice versa). Conversely, the biophysical network model emphasizes that both partial report and dual-task performance are susceptible to active information maintenance during the SOA period and their subsequent selective transfer from IM to WM (Öğmen et al., 2013; Wang et al., 2013; Zylberberg et al., 2009). Thus, partial report and dual-task performances should share a substantial amount of variance. Any stress and/or cortisol effect on these shared processes would not be task specific but be comparably reflected by both dual-task and partial report performances.

As we expected that cortisol could be the crucial mediator of stress effects on performances in both tasks (Miller et al., 2015; Beste et al., 2013), we decided to experimentally manipulate both the cause (i.e., stress using standardized protocols) and the mediator (i.e., cortisol by administering hydrocortisone [HC], which is biochemically identical to endogenous cortisol; Shields et al., 2015). Proceeding from this rationale, 40 participants processed a partial report and a dual task after completing the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) and a psychosocial control protocol (C-TSST; Het, Rohleder, Schoofs, Kirschbaum, & Wolf, 2009) and after having either ingested a HC or a placebo (PL) pill. Importantly, each participant received the same substance under both protocols that were completed in counterbalanced order with a time lag of 1 week. Adopting a sequential information processing perspective, we hypothesized that stress and cortisol effects on dual-task performance might be mediated by visual sensory processes as indicated by partial report performance (Beste, Stock, et al., 2012; Pashler, 1994).

METHODS

Participants

Forty healthy, nonsmoking men, aged 19–30 years (M = 23.70, SD = 2.85) with normal or corrected-to-normal vision and no self-reported history of psychiatric or other (chronic) disorders or chronic medication or drug

consumption (excluding smoking up to five cigarettes per day), were recruited from the participant database of the Department of Psychology, Technische Universität Dresden. Male participants were exclusively recruited to reduce gender-specific variations in cortisol availability and thus to increase the internal validity of this study (Perogamvros, Aarons, Miller, Trainer, & Ray, 2011; Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999). All participants had previously taken part in psychological experiments (including partial report task processing) but were naive toward the TSST procedure (Kirschbaum, Pruessner, & Stone, 1995). Their body mass index ranged from 20 to 26 kg/m^2 (M = 22.72, SD = 1.79). To minimize any interfering effects of glucose and caffeine ingestion on cortisol measurement, all participants were instructed to consume nothing but water for at least 2 hr before testing (Kirschbaum et al., 1997). This study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committee of the Dresden University Hospital (dossier: EK 230062014). All participants provided their written informed consent before participation and received monetary compensation (€90).

Study Design and Procedure

Separated by 1 week, all participants completed two testing sessions of approximately 3 hr. Proceeding from the low endogenous cortisol secretion in the afternoon (Veldhuis, Iranmanesh, Lizarralde, & Johnson, 1989), all sessions were completed between 2:30 pm and 6:00 pm to maximize the effectiveness of the experimental manipulations. In a double-blinded, factorial split-plot design (Kirk, 2013), each participant completed the TSST and C-TSST in counterbalanced order (within-participant factor) and concurrently received either an HC (0.12-mg/kg bodyweight; Miller et al., 2015) or a PL pill (between-participant factor). Thus, half of the participants received PL under both psychosocial protocols, whereas the other half received HC. Accordingly, each participant completed two of four experimental conditions of this study.

Except for the standardized protocol (i.e., [C-]TSST) and the administered substance (i.e., HC or PL), both sessions were identical with respect to the procedure: After completion of two practice blocks including the partial report and dual task with performance feedback, participants ingested the PL or HC pills. After substance intake, all participants were immediately exposed to the (C-) TSST to prevent a potential suppression of subsequent, stress-related cortisol responses. Such suppression effects may probably occur when bioavailable (i.e., absorbed) HC causes the pituitary corticotrophs to cease any secretory activity that is required to initiate endogenous cortisol synthesis and thus cortisol secretion (Reuter, 2002; cf. Spiga, Walker, Terry, & Lightman, 2014). Thereafter, all participants immediately started to complete five testing blocks (18 min each) that included the partial report and dual task without performance feedback. Throughout the sessions, subjective mood (Steyer, Schwenkmezger, Notz, & Eid, 1997) and saliva were sampled every 20 min. After the practice and testing blocks, respectively, participants completed the Mental Workload Test (MWT, German translation; Di Stasi et al., 2009). In the MWT, individual ratings of 13 task dimensions ([1] central processing; [2–5] spatial, verbal, visual, and auditory processing; [6] response processing; [7–8] manual and speech responses; [9] time pressure; [10–11] overall and physical effort; [12] self-evaluation; [13] frustration) were aggregated to an overall index of the mental effort invested into each task, ranging between 0% and 100% (Tsang & Velazquez, 1996). At the end of the second session, participants received their compensation.

Task Design and Technical Setup

Both cognitive tasks were presented using E-prime 2.0 experimental software (PST Inc., Pittsburgh, PA) on a 19-in. CRT monitor in a sound-attenuated room. The screen refresh rate of the monitor was set to 100 Hz. All visual stimuli were presented in white Serif font on a black background. Participants were seated in front of the monitor with a viewing distance of approximately 102 cm. Except for the performance feedback, all task blocks were identical and involved the alternating presentation of 168 trials of the partial report task followed by 96 trials of the dual task. A typical trial of each task is visualized in Figure 2.

Partial Report Task

The partial report task was adopted from Lu, Neuse, Madigan, and Dosher (2005; see also Miller et al., 2015). Within each trial, a fixation cross (viewing angle = 0.56°) was presented for 800 msec, followed by a letter array (2.81°) presentation for 100 msec. The array consisted of eight capital letters (0.39°) that were circularly arranged around the fixation cross. At each of the eight positions, the letters "C", "D", "H", and "U" were presented twice and in counterbalanced order. The letters were chosen because of their morphological dissimilarity and their comparable frequency in German texts (Graziano & Sigman, 2008; Best, 2005). After a varying SOA of 120, 140, 180, 260, 420, 740, or 1380 msec (chosen in counterbalanced order), a small red dot (0.11°) was presented to mark the position of the letter that had to be reported in the given trial. Within the following 2 sec, participants could enter their response by pressing the corresponding letter key on a standard computer keyboard. After an intertrial interval of 700 msec, a new trial began with the presentation of the fixation cross. Each block consisted of 168 trials (i.e., 24 trials per SOA). In total, participants completed 336 practice and 840 testing trials per session.

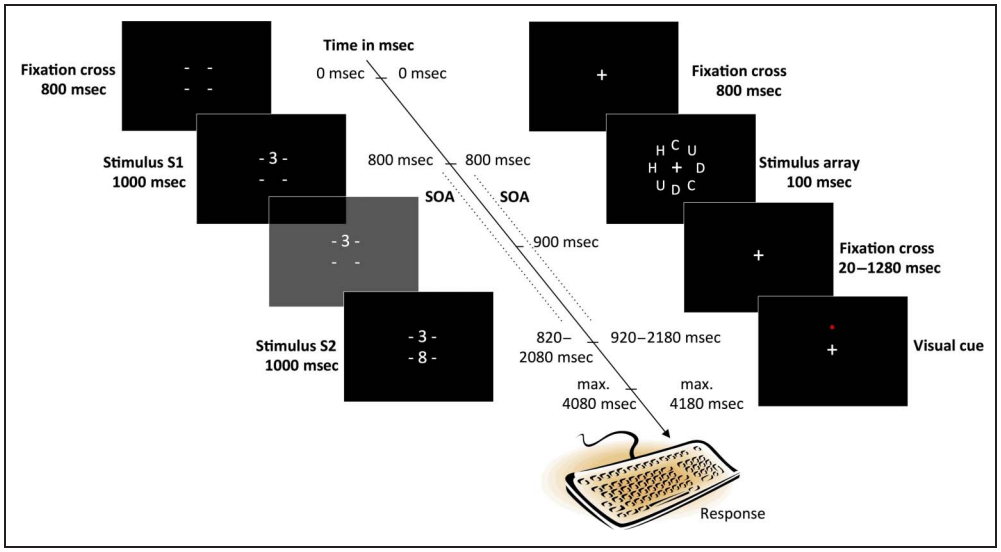

Figure 2. Illustration of a typical trial of the employed dual task (left side) and partial report task (right side): Any task trial starts with the presentation of a fixation cross for 800 msec, followed by the presentation of S1 for 1000 msec or the letter array for 100 msec, respectively. After a variable SOA, S2 or the visual cue is presented. The dual task requires classifying S1 and S2 as being larger or smaller than "5," whereas the partial report task requires reporting the letter that has been previously presented at the position of the visual cue. Max. = maximum.

Dual Task

The dual task was adopted from Fischer and Hommel (2012). Both tasks require a categorization of digits between 1 and 9 (except for 5) as being larger or smaller than 5. The digits 3 and 7 served as Task 1 stimuli (S1), whereas the digits 2, 4, 6, and 8 functioned as Task 2 stimuli (S2). The digits (0.56°) were presented in counterbalanced order. Participants were instructed to prioritize the S1 response and to subsequently respond to S2 as quickly and accurately as possible. Within each trial, a fixation display (1.35°) was presented for 800 msec that consisted of four horizontal dashes of 4 mm (0.11°) indicating the position of S1 and S2 10.5 mm above and 10.5 mm below the screen center. Thereafter, S1 was presented in between the upper dashes for 1000 msec. With varying SOA of 20, 80, 320, or 1280 msec, S2 was added in between the lower dashes for 1000 msec. A black screen was presented until participants entered their responses on a standard computer keyboard. The maximal response latency was set to 2 sec. Participants responded to S1 by pressing the keys "A" and "S" with their left index and middle fingers and to S2 by pressing the keys "K" and "L" with their right index and middle fingers (keys were labelled with "<" and ">" on a green or blue ground for each task). After an intertrial interval of 700 msec, a new trial began with the presentation of the fixation display. Each block consisted of 96 trials (i.e., 24 trials per SOA). In total, participants completed 192 practice and 480 testing trials per session.

Saliva Sampling and Glucocorticoid Analysis

During the testing sessions, saliva samples were obtained using Salivette collection devices (Sarstedt, Nümbrecht, Germany). After session completion, all samples were frozen (−20°C) until analysis. As HC is biochemically identical to cortisol, its ingestion contaminates the oral cavity, thereby yielding saliva cortisol concentrations that overestimate the amount of bioavailable cortisol in blood (Perogamvros et al., 2011; Perogamvros, Keevil, Ray, & Trainer, 2010). By contrast, salivary cortisone—the enzymatically inactivated form of cortisol—is less susceptible to this contamination and therefore the preferred marker after oral HC challenge (Perogamvros et al., 2010). Accordingly, we determined salivary cortisol and cortisone using liquid chromatography-tandem mass spectrometry, as described in detail elsewhere (Gao, Stalder, & Kirschbaum, 2015). The lower detection limit and intrarun and interrun coefficients of variation of the protocol were 0.08 nmol/l and below 10%, respectively. To enable general linear model-based analyses, salivary cortisol and cortisone were fourth-root transformed before they were submitted to further analyses (Miller & Plessow, 2013).

Statistical Analysis

All analyses were performed using the *lme4* and *mediation* packages (Bates, Mächler, Bolker, & Walker, 2015; Tingley, Yamamoto, Hirose, Keele, & Imai, 2014) with R 3.2.0 statistical software (R Core Team, 2015) as well as G*Power 3.1 (Faul, Erdfelder, Buchner, & Lang, 2009).

RTs and Accuracies

The mean RTs to correctly report the cued letter in the partial report task (RT_{PR}) or to correctly classify S1 (RT_{S1}) and S2 (RT_{S2}) in the dual task were estimated using the Kaplan–Meier method (Kaplan & Meier, 1958). Essentially, this method combines the information provided by RTs from correct and incorrect responses (assuming that incorrect responses are censored correct responses) by increasing the weight of ordered correct RTs whenever a faster incorrect response has occurred (see also

Blurton, Greenlee, & Gondan, 2015). RT_{S1} and RT_{S2} were further used to calculate several secondary measures of dual-task performance that were only of interest for enabling an integrative discussion of our data with regard to previous investigations of stress effects on dual-task processing: The total RT (TRT) was calculated as the sum of RT_{S1} and RT_{S2} and served as an indicator of processing efficiency (Miller et al., 2009). The category-match effect (CME) was calculated as RT_{S1} difference between trials requiring incompatible versus compatible responses to S1 and S2 (Plessow, Schade, et al., 2012). The proportions of correctly reported letters (AC_{PR}) or correctly classified S2 digits (AC_{S2}) in the partial report and dual task, respectively, were used as accuracy measures.

Hierarchical Regression

To evaluate whether acute stress (TSST exposure) and cortisol (HC administration) comparably impact on partial report (RT_{PR}, AC_{PR}) and/or dual-task (RT_{S2}, AC_{S2}) performance, hierarchical regression analyses were conducted on response ACs or Kaplan–Meier adjusted RTs, respectively. Apart from the randomly varying intercept to account for the repeated measurements, all predictors were sequentially entered. The employed procedure was sufficiently sensitive to detect relative increments in the portion of explained variance of $f^2 = \Delta R^2/(1 - R^2) \geq 0.21$ with a statistical power of at least 80% (Faul et al., 2009).

After inclusion of the baseline covariates (i.e., a general order effect and the sequence of [C]-TSST exposure), we investigated the amount of variance that was explained by the experimental manipulations (i.e., SOA, HC administration, TSST exposure, and their interaction). The corresponding baseline and fully parameterized regression models are expressed by the Equations 1A and 1B, respectively.

$$\mathrm{RT}|\mathrm{AC} = \beta_0 + \alpha_p + \beta_1\,\mathrm{order} + \beta_2\,\mathrm{sequence} \quad (1A)$$

$$\begin{aligned}\mathrm{RT}|\mathrm{AC} = {} & \beta_0 + \alpha_p + \beta_1\,\mathrm{order} + \beta_2\,\mathrm{sequence} \\ & + \beta_3\,\mathrm{SOA} + \beta_4\,\mathrm{HC} + \beta_5\,\mathrm{TSST} + \beta_6\,\mathrm{HC} \times \mathrm{TSST}\end{aligned} \quad (1B)$$

Equation 1A formalizes models in which the variance of RTs or ACs is solely explained by systematic variance because of the stability of the RT or AC within each participant p (i.e., random intercept, α_p) and the following baseline covariates: a general order effect representing the change of RTs/ACs from the first to second sessions (β_1 order; 0 = first session, 1 = second session) and a differential order effect because of the sequence in which the TSST and C-TSST protocol were completed (β_2 sequence; 0 = if the TSST in the first session was followed by C-TSST in the second session, 1 = if the C-TSST in the first session was followed by TSST in the second session). Please further note that β_0 indicates the mean RT or AC across all participants. Equation 1B formalizes the fully parameterized models that explain additional variance by the experimental manipulations: SOA in seconds (β_3 SOA), HC administration (β_4 HC; 0 = PL, 1 = HC), and TSST exposure (β_5 TSST; 0 = C-TSST, 1 = TSST) and their interaction (β_6 HC × TSST).

Mediation Analyses

For mediation analysis, the trapezoidal areas under the RT-log(SOA) curves[2] and the AC-log(SOA) curves (AUCs; cf. Fekedulegn et al., 2007) were calculated to obtain a single dependent performance variable for each participant and session. Thus, AUC(RT_{PR}) and AUC(RT_{S2}) indicate the individual, SOA-integrated partial report and dual-task RTs, whereas AUC(AC_{PR}) and AUC(AC_{S2}) indicate the individual, SOA-integrated partial report and dual-task accuracies. Correlations between the AUCs were determined using nonparametric bootstrapping. To facilitate the interpretability of the results, all AUCs were fully standardized before they were submitted to mediation analysis.

To investigate if the impact of TSST exposure and/or HC administration on dual-task performance is actually mediated by partial report performance, we simultaneously regressed the partial report AUCs on the contrasts between (a) the PL condition and each of the three intervention conditions (i.e., the mediator model) and (b) the dual-task AUCs on partial report AUCs under each condition (i.e., the outcome model). Within-participant variance was also appropriately estimated via random effects. Within these analyses, we used 10,000 iterations of a quasi-Bayesian Monte Carlo simulation to estimate the direct and indirect mediation effects (Tingley et al., 2014). The significance of these effects was determined from the 2.5th and 97.5th percentiles of all simulated effects.

RESULTS

Manipulation Check

Salivary Cortisol and Cortisone

Overall, the experimental manipulation of salivary cortisol and cortisone by TSST exposure and/or HC administration (dose: $M = 8.95$ mg, $SD = 0.8$ mg, range = 7.07–10.05 mg) was successful. As intended, participants were unable to identify HC beyond guessing probability (P[hit] = .41, $p = .95$].

As illustrated in Figure 3A and B, mean cortisol (CortL) and cortisone (CortN) concentrations increased across sampling time (CortL: $\chi^2(9) = 382.36$, $p < .001$; CortN: $\chi^2(9) = 251.26$, $p < .001$) because of TSST exposure (CortL: $\chi^2(1) = 7.98$, $p < .01$; CortN: $\chi^2(1) = 40.49$, $p < .001$) and HC administration (CortL: $\chi^2(1) = 38.67$, $p < .001$; CortN: $\chi^2(1) = 21.02$, $p < .001$) and their interaction (CortL: $\chi^2(1) = 0.93$, $p = .33$; CortN: $\chi^2(1) = 16.32$, $p < .001$). These findings were complemented by the

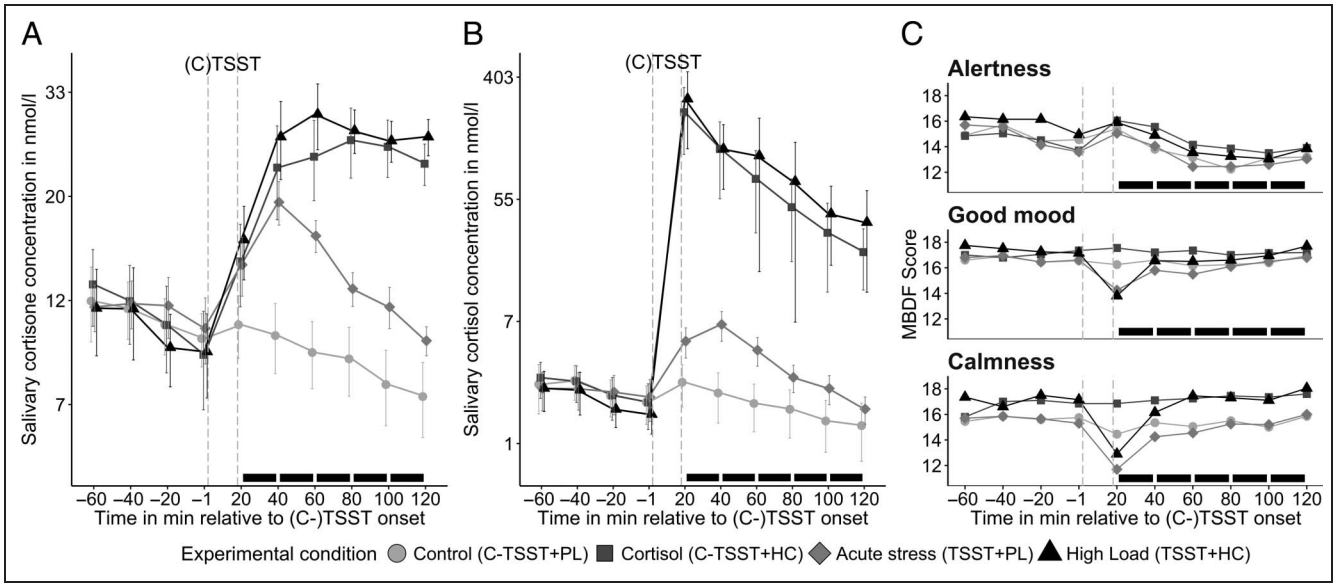

Figure 3. Mean salivary cortisone (A) and cortisol (B) concentrations and mood ratings (C) relative to (C-)TSST onset at t_0 = 0 min in the four experimental conditions that resulted from crossing TSST and C-TSST exposure with an HC or PL administration. Black rectangles indicate the onset/offset of the five cognitive testing blocks. Error bars in A and B constitute the 95% confidence interval (Please note that log-scaled concentrations distort the symmetry of the shown confidence intervals).

significant interaction of sampling time and HC administration or TSST exposure (all ps < .05).

In accordance with Perogamvros and colleagues (2010, 2011), salivary cortisol was strongly contaminated by oral HC intake (see Figure 3B). This contamination was also reflected by a strong correlation of salivary cortisol and cortisone before HC administration (including four saliva samples assessed during the first 60 min of each session; r = .74, p < .001) but an attenuated correlation across the whole session (r = .53, p < .001; cf. Perogamvros et al., 2010, 2011).

Mood Ratings

Overall, TSST exposure successfully induced a subjective stress response as indicated by a change in mood and calmness. As illustrated in Figure 3C, TSST exposure induced a significant drop in good mood ($\chi^2(1)$ = 10.61, p < .01) and calmness ratings ($\chi^2(1)$ = 8.66, p < .01). More precisely, good mood was significantly affected by sampling time ($\chi^2(9)$ = 67.13, p < .001) and TSST exposure and their interaction ($\chi^2(1)$ = 72.71, p < .001) but unaffected by HC administration ($\chi^2(1)$ = 1.32, p = .25). Calmness ratings were significantly affected by sampling time ($\chi^2(9)$ = 133.77, p < .001) and TSST exposure and their interaction ($\chi^2(1)$ = 85.48, p < .001) but also by HC administration ($\chi^2(1)$ = 9.21, p < .01; cf. Reuter, 2002). In accordance with previous studies, alertness ratings only decreased over time (i.e., fatigue increased; $\chi^2(9)$ = 149.85, p < .001), irrespective of TSST exposure ($\chi^2(1)$ = 0.14, p = .71) and its interaction with time ($\chi^2(1)$ = 11.63, p = .24) or HC administration ($\chi^2(1)$ = 1.00, p = .32; Plessow, Kiesel, et al., 2012; Plessow, Schade, et al., 2012).

RTs

To evaluate the impact of TSST exposure and/or HC administration on RTs that were required to report the cued letter in the partial report task (RT_{PR}) or to classify the second digit in the dual task (RT_{S2}), hierarchical multiple regressions were performed on Kaplan–Meier adjusted RTs. Changing the order of predictor inclusion did not change any of the below reported results. Table 1 provides detailed information about the RTs in both tasks for each experimental condition.

RTs in the Partial Report Task

Overall, RT_{PR} significantly decreased from the first to second testing sessions (β_1 = −52.39 ± 8.70 msec, $\chi^2(1)$ = 35.12, p < .001), irrespective of the psychosocial intervention ([C-]TSST) that was implemented during the respective session (β_2 = 50.73 ± 35.51 msec, $\chi^2(1)$ = 2.09, p = .15). This general order effect explained approximately 3% of the RT_{PR} variance and was most likely because of general learning effects.

As visualized in Figure 4A, RT_{PR} significantly decelerated when SOA was increased (β_3 = 151.83 ± 7.79 msec, $\chi^2(1)$ = 286.33, p < .001), with SOA accounting for approximately 18% of the total RT_{PR} variance. HC administration significantly reduced RT_{PR} (β_4 = −91.34 ± 32.70 msec, $\chi^2(1)$ = 7.65, p < .01) and incrementally explained approximately 9% of its variance. Notably, neither

Table 1. Mean (*M*) Partial Report and Dual-Task S2 RTs (in Milliseconds) with Standard Deviations (*SD*s) for All SOAs and Experimental Conditions

	Control (N = 20), C-TSST + PL		*Cortisol (N = 20), C-TSST + HC*		*Stress (N = 20), TSST + PL*		*High Load (N = 20), TSST + HC*	
SOA	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Partial report RTs (RT_{PR})								
120	846.14	145.20	751.13	95.49	847.92	162.82	766.01	101.57
140	880.91	149.81	763.91	84.02	865.85	174.82	785.36	105.03
180	885.08	116.09	795.37	99.98	890.75	145.19	788.95	103.55
260	900.59	134.81	820.38	109.41	887.68	147.09	828.75	122.44
420	972.48	143.32	873.31	110.80	960.74	149.32	875.82	116.16
740	998.79	133.87	893.07	122.31	1009.60	142.03	897.01	142.80
1380	1040.58	150.93	967.30	117.80	1053.98	147.16	955.91	139.45
Overall	932.08	152.04	837.78	126.63	930.93	166.45	842.54	133.83
$AUC(RT_{PR})$	5.56	0.71	4.98	0.51	5.54	0.80	5.03	0.62
Dual-task S2 RTs (RT_{S2})								
20	922.14	110.68	833.55	129.70	978.73	161.51	838.39	120.97
80	870.42	109.56	793.43	143.10	924.45	153.68	792.61	130.40
320	784.76	147.25	682.98	162.87	830.37	198.40	671.34	138.68
1280	668.79	163.63	563.51	136.50	706.68	215.03	564.11	119.49
Overall	811.53	132.78	718.37	143.04	860.06	182.15	716.61	127.38
$AUC(RT_{S2})$	2.45	0.38	2.17	0.42	2.60	0.52	2.17	0.37

The conditions resulted from crossing the standardized stress-induction and control protocols (TSST and C-TSST) with an HC or a PL administration. AUCs indicate the standardized areas under the RT-log(SOA) curves (in sec × log[sec]).

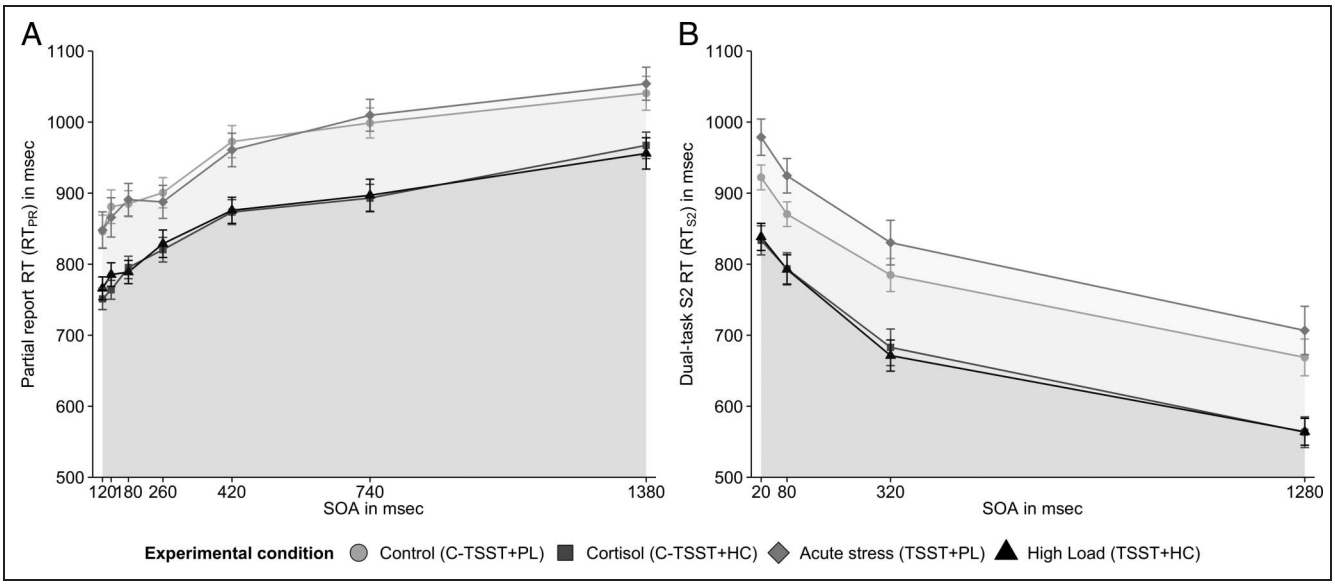

Figure 4. Mean partial report (A) and dual-task S2 (B) RTs for all four experimental conditions that resulted from crossing a TSST or C-TSST exposure with an HC or PL administration. Shaded areas indicate the task-specific areas under the RT-SOA curves ($AUC[RT_{PR}]$ and $AUC[RT_{S2}]$). Error bars represent the respective standard errors.

TSST exposure ($\beta_5 = 1.81 \pm 6.62$ msec, $\chi^2(1) = 0.07, p = .78$) nor its interaction with HC administration ($\beta_6 = 5.92 \pm 13.25$ msec, $\chi^2(1) = 0.20, p = .65$) significantly affected RT_{PR}. The fully parameterized model (including the random effect of participants on the intercept at $\beta_0 = 877.82 \pm 29.03$ msec) accounted for 32% (74%) of the total RT_{PR} variance.

RTs in the Dual Task

Overall, RT_{S2} significantly decreased from the first to second testing sessions ($\beta_1 = -48.89 \pm 14.40$ msec, $\chi^2(1) = 11.33, p < .001$), irrespective of the psychosocial intervention ([C-]TSST) that was implemented during the respective session ($\beta_2 = 18.83 \pm 46.28$ msec, $\chi^2(1) = 0.17, p = .68$). This general order effect explained about 2% of the RT_{S2} variance.

As illustrated in Figure 4B, RT_{S2} significantly decreased when SOA was increased ($\beta_3 = -191.46 \pm 8.44$ msec, $\chi^2(1) = 293.40, p < .001$). This SOA effect accounted for approximately 26% of the total RT_{S2} variance. HC administration induced an additional and significant reduction in RT_{S2} ($\beta_4 = -118.30 \pm 42.68$ msec, $\chi^2(1) = 7.55, p < .01$) and explained another 9% of the RT_{S2} variance. Most importantly, TSST exposure significantly increased RT_{S2} ($\beta_5 = 23.39 \pm 8.44$ msec, $\chi^2(1) = 7.65, p < .01$), explaining approximately 0.5% of the variance in RT_{S2}. Concurrent HC administration, however, completely reversed this adverse effect of TSST exposure ($\beta_6 = -50.29 \pm 16.64$ msec, $\chi^2(1) = 9.11, p < .01$). As a consequence, RT_{S2} even dropped below the RTs after PL administration or C-TSST exposure. Nevertheless, this interaction effect only accounted for another 0.5% of RT_{S2} variance. The fully parameterized model (including the random effect of participants on the intercept at $\beta_0 = 907.93 \pm 37.82$ msec) accounted for 38% (85%) of the total RT_{S2} variance.

Accuracies

To evaluate the impact of TSST exposure and/or HC administration on response accuracies in reporting the cued letter in the partial report task (AC_{PR}) or in classifying the second digit in the dual task (AC_{S2}), hierarchical multiple regressions were performed on the portion of correct responses (ACs). Changing the order of predictor inclusion did not change any of the below reported results. Table 2 provides detailed information about the ACs in both tasks for each experimental condition.

Accuracies in the Partial Report Task

Overall, AC_{PR} increased from the first to second testing sessions ($\beta_1 = 2.16 \pm 0.99\%$, $\chi^2(1) = 4.71, p < .05$), irrespective of the psychosocial intervention ([C-]TSST) that was implemented during the respective session ($\beta_2 = -2.61 \pm 3.30\%$, $\chi^2(1) = 0.65, p = .42$). This general order effect explained approximately 0.5% of the AC_{PR} variance.

As illustrated in Figure 5A, AC_{PR} significantly decreased when SOA was increased ($\beta_3 = -21.3 \pm 0.70\%$, $\chi^2(1) = 531.24, p < .001$), with SOA accounting for approximately 35% of the total AC_{PR} variance. HC administration additionally increased AC_{PR} ($\beta_4 = 8.29 \pm 3.05\%$, $\chi^2(1) = 7.28, p < .01$) and explained another approximately 7% of the AC_{PR} variance. Finally, neither TSST exposure ($\beta_5 = -0.25 \pm 0.60\%$, $\chi^2(1) = 0.18, p = .67$) nor its interaction with HC administration ($\beta_6 = 0.93 \pm 1.20\%$, $\chi^2(1) = 0.61, p = .44$) significantly affected AC_{PR}. The fully parameterized model (including the random effect of participants on the intercept at $\beta_0 = 70.72 \pm 2.71\%$) accounted for 42% (79%) of the total AC_{PR} variance.

Accuracies in the Dual Task

As illustrated in Figure 5B, the consistently high AC_{S2} indicated a pronounced ceiling effect around $\beta_0 = 96.61 \pm 0.73\%$. Accordingly, only the SOA exerted a slight but statistically significant impact on AC_{S2} ($\beta_4 = 0.56 \pm 0.27\%$, $\chi^2(1) = 4.49, p < .05$), explaining approximately 1% of the total AC_{S2} variance (all other ps $> .35$). When AC_{S2} was predicted by TSST exposure and HC administration (which were not predictive when considered in isolation) and their interaction, participants made significantly more mistakes after TSST exposure and concurrently HC administration ($\beta_6 = -1.12 \pm 0.55\%$, $\chi^2(1) = 4.22, p < .05$) as compared with either C-TSST exposure and/or PL administration. The fully parameterized model (including the random effect of participants on the intercept) accounted for 2% (49%) of the total AC_{S2} variance.

Mediation Analyses

As explained in the Introduction, this study aimed at evaluating two mediation hypotheses about (1) a mediation effect of acute stress on partial report and/or dual-task performance by cortisol and (2) a mediation effect of acute stress and/or cortisol on dual-task performance by partial report performance.

Mediation hypothesis (1) was investigated by the experimental manipulation of stress (by TSST exposure) and its mediator cortisol (by HC administration): As summarized in Tables 1 and 2, AC_{PR}/RT_{PR} and RT_{S2} were both significantly affected by cortisol, but only RT_{S2} was also significantly affected by acute stress. In contrast to the hypothesized comparability of acute stress and cortisol effects, the effect direction of stress and cortisol on RT_{S2} diverged. Specifically, the common impact of acute stress and HC administration was not only dominated by HC (as indicated by the same effect direction), but their joint administration further boosted the beneficial impact of HC on RT_{S2}. An additional cortisol secretion in response to TSST exposure presumably caused this boost. Therefore, the present results suggest that high cortisol availability (as, for instance, induced by HC administration) suppresses adverse stress effects on RT_{S2}.

Table 2. Mean (*M*) Partial Report and Dual-Task S2 Accuracies (Scaled in Percentage) with Standard Deviations (*SD*s) for All SOAs and Experimental Conditions

	Control (N = 20), C-TSST + PL		*Cortisol (N = 20), C-TSST + HC*		*Stress (N = 20), TSST + PL*		*High Load (N = 20), TSST + HC*	
SOA	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Partial report accuracies (AC_{PR})								
120	77.67	13.11	85.42	7.29	76.79	12.81	84.49	7.52
140	72.83	13.09	82.38	7.19	71.88	15.15	80.89	8.62
180	68.42	14.44	75.89	8.04	66.29	12.53	76.43	8.38
260	62.13	12.66	70.54	6.84	63.83	13.50	71.79	9.84
420	57.04	13.63	66.71	8.48	57.75	12.91	66.06	8.96
740	53.54	12.05	59.08	9.33	51.08	11.10	60.63	9.20
1380	47.71	13.16	54.13	7.49	46.71	10.85	55.33	9.66
Overall	62.76	16.34	70.59	13.22	62.05	16.06	70.80	13.22
$AUC(AC_{PR})$	3.77	0.73	4.24	0.38	3.73	0.71	4.26	0.44
Dual-task S2 accuracies (AC_{S2})								
20	96.17	3.33	97.13	2.89	96.21	3.02	95.74	5.22
80	96.92	3.10	97.71	2.28	97.00	2.65	96.97	4.99
320	96.92	2.62	96.75	2.07	97.38	2.35	96.66	4.79
1280	97.58	2.77	97.46	2.09	98.21	1.46	96.41	4.92
Overall	96.90	2.95	97.26	2.34	97.20	2.50	96.44	4.91
$AUC(AC_{S2})$	2.91	0.08	2.92	0.06	2.92	0.06	2.90	0.14

The conditions resulted from crossing the standardized stress-induction and control protocols (i.e., TSST and C-TSST) with an HC or a PL administration. AUCs indicate the standardized areas under the AC-log(SOA) curves (in AC × log[sec]).

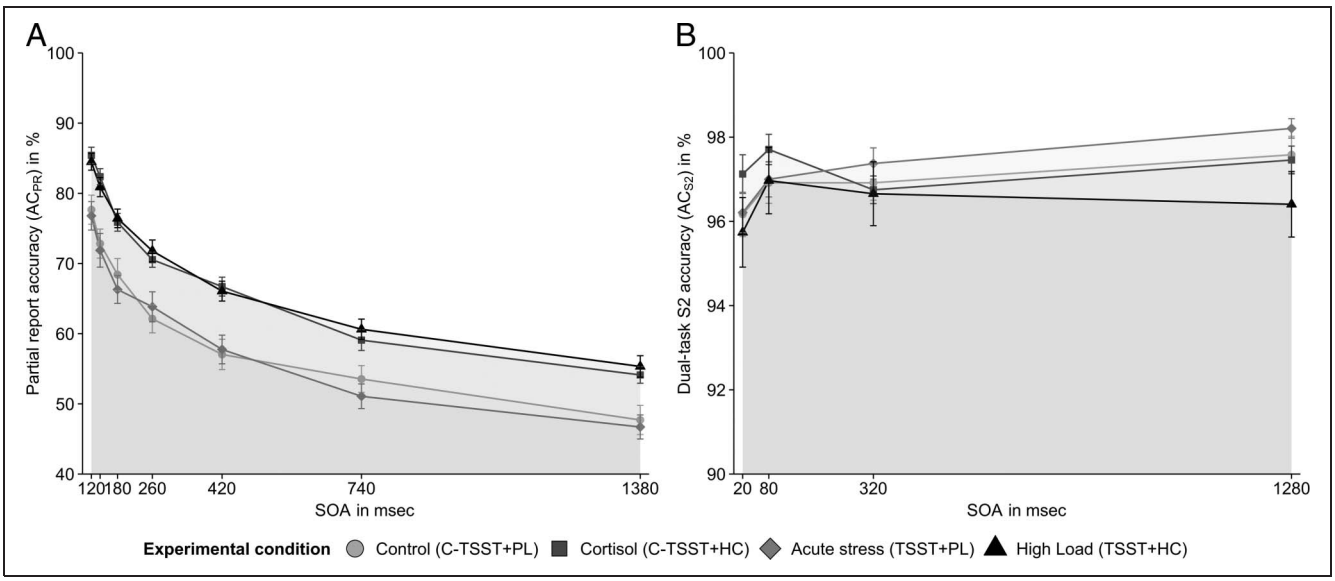

Figure 5. Mean partial report (A) and dual-task S2 response (B) accuracies scaled in percentage for all four experimental conditions that resulted from crossing a TSST or C-TSST exposure with an HC or PL administration. Shaded areas indicate the task-specific areas under the AC-SOA curves ($AUC[AC_{PR}]$ and $AUC[AC_{S2}]$). Error bars represent the respective standard errors.

The regressions that were used to evaluate if (2) the impact of TSST exposure and/or HC administration on AUC(RT_{S2}) or AUC(AC_{S2}) was mediated by AUC(RT_{PR}) or AUC(AC_{PR}) are visualized in Figure 6. More detailed information on the AUCs is provided in the lower part of Tables 1 and 2.

Mediation: Partial Report RT and Dual-Task RT

AUC(RT_{S2}) and AUC(RT_{PR}) correlated significantly (r = .51, 95% CI [0.32, 0.67]), which indicates that they shared at least approximately 26% of variance. On the basis of their reduced correlation after controlling for HC administration ($r_{partial}$ = .42, 95% CI [0.22, 0.50]), approximately 8% of their common variance was attributable to HC administration.

The impact of HC administration on AUC(RT_{S2}) was completely mediated by AUC(RT_{PR}). As illustrated in Figure 6A, the regression coefficients that relate the two HC conditions to AUC(RT_{PR}) were significant, as were the coefficients relating these conditions to the AUC(RT_{S2}). The significant mediation effect given HC administration after C-TSST exposure amounted to −0.32 (95% CI [−0.64, −0.07]). Similarly, the significant mediation effect given HC administration after TSST exposure amounted to −0.29 (95% CI [−0.61, −0.05]). By contrast, the impact of TSST exposure after PL administration on AUC(RT_{S2}) was not mediated by AUC(RT_{PR}) (M = −0.01, 95% CI [−0.13, 0.11]).

Mediation: Partial Report AC and Dual-Task AC

AUC(AC_{PR}) and AUC(AC_{S2}) were most likely unrelated (r = .02, 95% CI [−0.27, 0.26]). Thus, neither the impact of HC administration on AUC(AC_{S2}; M = 0.03, 95% CI [−0.19, 0.26]) nor the effects of TSST exposure

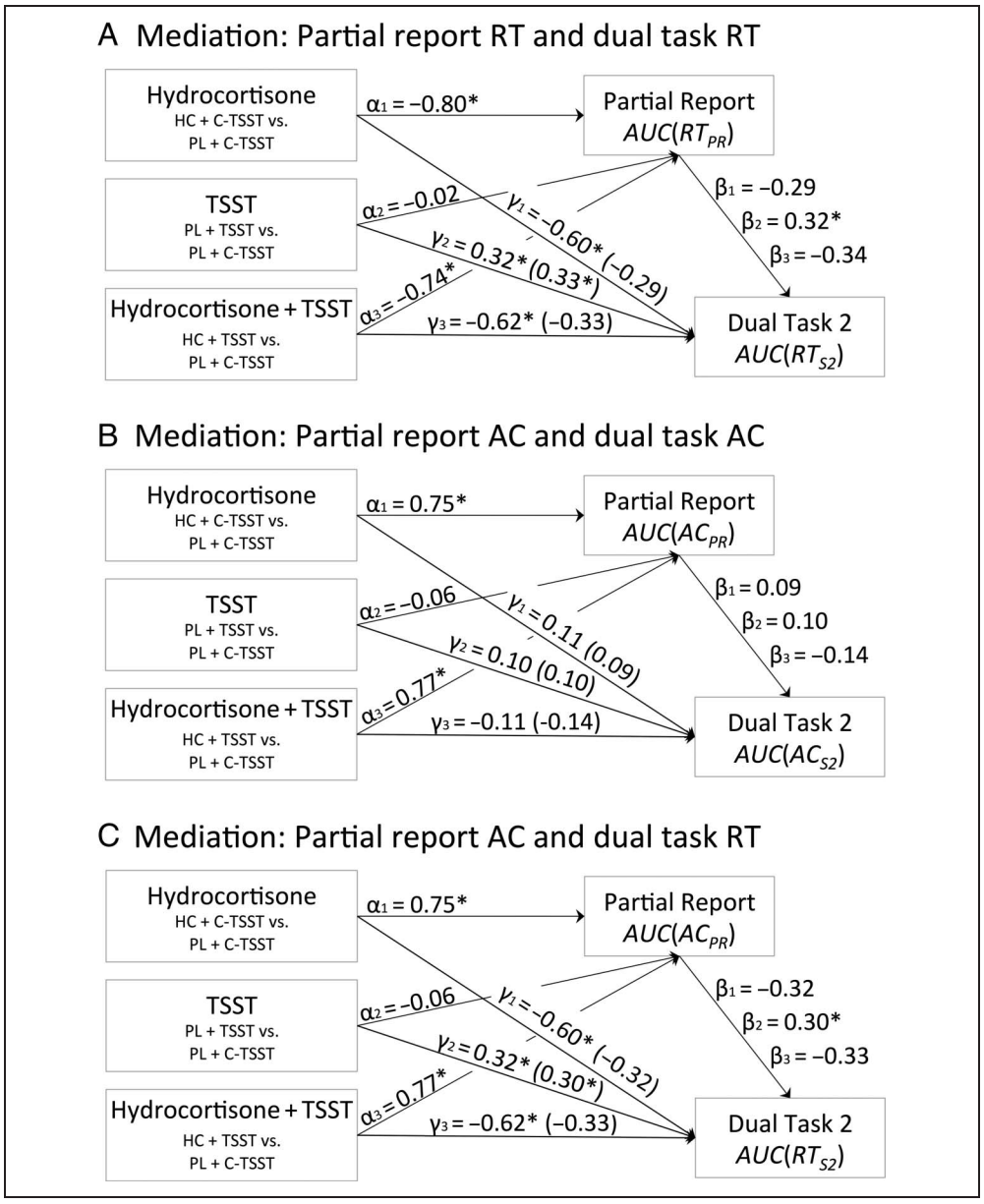

Figure 6. Regression coefficients (α, β, γ) that relate HC administration and/or TSST exposure to the standardized dual-task performance AUCs (AUC[RT_{S2}] and AUC[AC_{S2}]) and partial report task performance AUCs (AUC[RT_{PR}] and AUC[AC_{PR}]). In A, RT AUCs are investigated (AUC[RT_{PR}] and AUC[RT_{S2}]); in B, response accuracy AUCs are investigated (AUC[AC_{PR}] and AUC[AC_{S2}]); and in C, the AUC(RT_{S2}) and AUC(AC_{PR}) are investigated as proxies for the PRSE and PRP effect, respectively. Coefficients in parentheses indicate the relation between the experimental condition and the dual-task performance AUCs after controlling for partial report AUCs. (Mediation effects are provided in the text.) *p < .05.

($M = -0.00$, 95% CI [−0.04, 0.03]) or their co-occurrence on AUC(AC_{S2}; $M = 0.03$, 95% CI [−0.20, 0.27]) were significantly mediated by AUC(AC_{PR}; see Figure 6B).

Mediation: Partial Report AC and Dual-Task RT

AUC(RT_{S2}) and AUC(AC_{PR}) correlated significantly ($r = -.38$, 95% CI [−0.60, −0.16]), indicating that they shared approximately 15% of variance. This correlation decreased after controlling for HC administration ($r_{partial} = -.27$, 95% CI [−0.51, −0.05]), indicating that approximately 7% of their common variance was attributable to HC administration.

The impact of HC administration on AUC(RT_{S2}) was completely mediated by AUC(AC_{PR}). As illustrated in Figure 6C, regression coefficients that relate the two HC conditions to AUC(AC_{PR}) were significant, as were the coefficients relating these conditions to the AUC(RT_{S2}). The significant mediation effect given HC administration after C-TSST exposure amounted to −0.28 (95% CI [−0.63, −0.04]). The significant mediation effect of HC administration after TSST exposure amounted to −0.29 (95% CI [−0.65, −0.05]). By contrast, the impact of TSST exposure after PL administration on AUC(RT_{S2}) was not mediated by AUC(AC_{PR}; $M = 0.02$, 95% CI [−0.04, 0.10]).

DISCUSSION

This study examined how acute stress and cortisol impact on executive and visual sensory processes. For this purpose, we assessed dual-task and partial report performances after combining an HC or a pharmacological PL intervention with a psychosocial stress-induction protocol (TSST) or a psychological PL (C-TSST). Overall, HC significantly improved both partial report and dual-task performances as indicated by reduced RTs (RT_{PR} and RT_{S2}) and increased response accuracy (AC_{PR}). Notably, the beneficial HC effect on dual-task performance (AUC [RT_{S2}]) was completely mediated by improved partial report performance (i.e., AUC[RT_{PR}] and AUC[AC_{PR}]). By contrast, TSST exposure selectively impaired dual-task performance as indicated by increased RT_{S2}. Proceeding from our mediation analysis, this adverse stress effect was mediated through an alternative route of action that is not sensitive to cortisol but to the residual components of acute stress. In summary, cortisol (i.e., HC) did not only alleviate the adverse impact of acute stress on dual-task performance but also improved the performance in both SOA-dependent cognitive tasks beyond the levels under the respective control condition. Consequently, the presented results support the assumption that glucocorticoids could play a key role in counteracting the initially adverse stress effects on executive processes by improving visual sensory processes (Hermans et al., 2014; Putman & Roelofs, 2011; Het & Wolf, 2007).

Interestingly, partial report and dual-task performances (AUCs) shared approximately 15–26% of their variances. This common variance was only in part attributable to HC-related cortisol availability (approximately 7–8%). Therefore, the present data also support the notion that partial report and dual-task performances rely to a considerable degree on shared cognitive processes. Proceeding from the biophysical network model outlined in the Introduction, these processes presumably drive the active maintenance of task-relevant information across the SOA period and/or its selective transfer from IM to WM (Öğmen et al., 2013; Zylberberg et al., 2009).

A Common, Cortisol-sensitive Source of Partial Report and Dual-Task Performance

The precise nature of the processes that give rise to the HC susceptibility of partial report and dual-task performance cannot be ultimately inferred from the present data. Nonetheless, the alleged involvement of information maintenance and selective information transfer is also supported by earlier research, particularly the so-called attention selectivity approach proposed by Chajut and Algom (2003). Similar propositions have been advocated to explain the positive HC effect on the processing of emotionally arousing material (e.g., Oei et al., 2009; Het & Wolf, 2007; see Putman & Roelofs, 2011, for a review).

Through the use of different selective attention tasks, Chajut and Algom (2003) reasoned that acute stress may improve the processing of task-relevant information (i.e., enhances attention selectivity) and at the same time reduce the distractibility by task-irrelevant information. More precisely, manipulating the geometrical form and/or size of visual stimuli, their readability, and/or the semantic match that characterized the salience of target and distracting stimulus features only affected overall task performances but not the stress-induced improvement of selectivity (Chajut & Algom, 2003). Importantly, these beneficial stress effects occurred over a period of approximately 50 min (see Experiment 1 in Chajut & Algom, 2003). Traditionally, this period is believed to exceed the duration of noradrenergic and/or subjective stress responses on cognitive processes but perfectly matches the period of slow-occurring glucocorticoid actions (Hermans et al., 2014; Putman & Roelofs, 2011; Dickerson & Kemeny, 2004). Because partial report and dual-task performances also rely on the selective allocation of attentional resources to the respective target stimulus features (i.e., the cued stimulus feature and S2, respectively; Finke et al., 2005; Gegenfurtner & Sperling, 1993; Kahneman, 1973), an enhanced attention selectivity and decreased distractibility are well compatible with the presented HC effects. Moreover, the attention selectivity explanation also conforms to previous findings showing a cortisol-related reduction in stimulus interference because of emotional distractors (Oei et al., 2009) or overlearned response tendencies (Plessow et al., 2011).

Note that this cognitive attention framework also perfectly fits the biophysical network model introduced

in the Introduction. According to this model, partial report and dual-task performances should share variance because they both depend on the maintenance and selective amplification of task-relevant information under varying SOA periods. As SOAs temporally bracket the presentation of stimuli, they impose constraints on the utility of top–down amplifications for an adaptive maintenance of task-relevant information or a maladaptive maintenance of task-irrelevant (i.e., distracting) information (Miller et al., 2015; Zylberberg et al., 2009). Conceptually, such processes of top–down amplification are equivalent to a selective allocation of attention resources (Finke et al., 2005; Gegenfurtner & Sperling, 1993). Thus, any cortisol-related acceleration of top–down amplification ergo attention selectivity may bias neuronal competition toward the respective task-relevant stimulus features at hierarchically lower levels that are shared by SOA-dependent cognitive tasks like the partial report and the dual task (Miller et al., 2015; Zylberberg et al., 2009; Chajut & Algom, 2003). In consequence, the processing efficiency of the respective task-relevant stimulus features would increase, and partial report and dual-task performances should be consistently enhanced under conditions of cortisol exposure (e.g., Beste et al., 2013).

Importantly, this explanation of the shared variance between both tasks is also compatible with modern bottleneck models (e.g., Tombu et al., 2011) and cognitive models including a preceding perceptual bottleneck (e.g., Öğmen et al., 2013). In the former, partial report and dual-task performances should share variance because of a common, unitary bottleneck. In the latter, they should share some variance because of a common bottleneck of information encoding that might be cortisol sensitive and relevant for both partial report and dual-task performances. However, partial report performance and the PRSE are widely disregarded in the standard dual-task literature and/or considered as a preattentive perceptual phenomenon that does not involve any cognitive bottleneck attributed to dual-task performance and the PRP effect (Beste, Stock, et al., 2012; Pashler, 1994; but see Saneyoshi, Niimi, Suetsugu, Kaminaga, & Yokosawa, 2011; Finke et al., 2005). Instead, the PRP effect is often believed to exclusively arise from some capacity-limited level of response selection and/or decision-making that is not sensitive to visual sensory processes as indicated by partial report performance (Pashler, 1994; but see Saneyoshi et al., 2011; Pashler, 1991, p. 1032) or other executive processes (cf. Miyake et al., 2000). Proceeding from this reasoning, it is understandable that previously published findings on mixed stress and cortisol effects in dual-task settings have been primarily attributed to the response-selection bottleneck and/or hierarchically higher levels of information processing (Yildiz, Wolf, & Beste, 2014; Yildiz, Chmielewski, & Beste, 2013; Plessow, Schade, et al., 2012). From a sequential perspective on information processing (i.e., the encoding and maintenance of sensory information precedes response selection), however, the presented joint HC effects on partial report and dual-task performances can only arise at a shared, hierarchically lower level of information processing.

To further distinguish between this impact of cortisol on visual sensory versus executive processes in future studies, we recommend to simultaneously assess performance in multiple SOA-constrained tasks like the attentional blink (Zylberberg et al., 2009), *n*-back (Schoofs et al., 2008), task switching (Plessow, Kiesel, et al., 2012), or continuous performance task (Braver & Barch, 2002; Umbricht et al., 2000). Regarding the investigation of the neurophysiological mechanism underlying these effects, NMDAR-related glutamatergic signal transduction (e.g., Beste, Wascher, Dinse, & Saft, 2012; Umbricht et al., 2000) that is supposed to accomplish active information processing in SOA-constrained tasks may also be a worthwhile target of experimental manipulations (Zylberberg et al., 2009).

The Selective Stress Effect on Dual-Task Performance

Besides the shared variance in both tasks that was partially attributable to HC, we also observed a selective impairment of dual-task performance (RT_{S2}) after acute stress (Plessow, Schade, et al., 2012). Mediation analyses indicated that acute stress did not affect RT_{S2} by the same processes driving the cortisol-related improvements in dual-task and partial report performances. Thus, our results instead suggest that the residual components of acute stress may alter dual-task performance at hierarchically higher levels of (executive) information processing (Yildiz et al., 2013, 2014). To evaluate whether previously proposed concepts of processing efficiency (Beste et al., 2013), resource depletion (Plessow, Kiesel, et al., 2012), and/or cognitive adaptivity (Plessow, Schade, et al., 2012) may contribute to explain these residual stress effects, we also discuss several secondary cognitive measures that are reported in Table A1 (see Appendix).

In accordance with Miller and colleagues (2009), dual-task processing efficiency is reflected in the TRT that is needed to complete both tasks. Relying on this measure, Beste and colleagues (2013) reported evidence for stress-induced reductions in the TRT and therefore stress-induced enhancements of dual-task processing efficiency (Miller et al., 2009). At the same time, stress-induced cortisol increases were accompanied by reduced TRT (i.e., enhanced processing efficiency). In this study, the HC-induced reduction of the TRT (which we would rather attribute to visual sensory processes) is completely consistent with these findings. However, we did not observe a significant impact of acute stress on TRT. Thus, the most likely explanation for these diverging stress effects relates to the inability to distinguish the impact of cortisol and residual stress components on the processing efficiency solely by means of stress-induction protocols. Nonetheless, the

design parameters of cognitive tasks and/or the specific type of stress-induction protocol were previously also identified as potential effect moderators (Shields et al., 2015; Hermans et al., 2014; Dickerson & Kemeny, 2004). Indeed, Beste and colleagues (2013) presented their dual task in different modalities and employed a stress protocol that also features nonpsychosocial stress-induction components (Dickerson & Kemeny, 2004). As numeric deteriorations of dual-task processing efficiency (i.e., longer RT_{S2}) have been found in dual-task settings that are similar to ours (Plessow, Schade, et al., 2012), the abovementioned moderators have probably contributed to the diverging direction of acute stress effects on dual-task processing efficiency as indicated by the TRT.

Resource depletion approaches provide an alternative explanation for acute stress effects on cognitive processes by assuming that acute stress occupies mental resources, which are no longer available for cognitive task processing (Chajut & Algom, 2003). Accordingly, the resulting cognitive performance impairment should be stronger, the more demanding and/or complex a task is (Plessow, Kiesel, et al., 2012). However, we could not confirm that stress affects the mental resources invested to process the dual task, because the partial report task was perceived as more demanding than the dual task although the latter is considered to indicate more demanding/complex executive processes (Miyake et al., 2000; Pashler, 1991, 1994). As the mental workload ratings detected general learning and practice effects, a general lack of MWT sensitivity unlikely explains the present findings (Scerra & Brill, 2012; Tsang & Velazquez, 1996). Therefore, "pure" resource depletion seems to be of minor importance for explaining the effect of residual stress components on dual-task performance (cf. Chajut & Algom, 2003).

Cognitive adaptivity approaches assume that our cognitive system can dynamically switch between different processing modes and will choose the one that is most promising for achieving a specific task goal (Plessow, Schade, et al., 2012). Thus, under conditions of stress-induced resource depletion, the most parsimonious dual-task processing mode should be implemented to spare mental resources (Plessow, Schade, et al., 2012). As two extreme manifestations of such processing modes, S1 and S2 can be concurrently processed (i.e., in parallel) or strictly one after another (Miller et al., 2009). Although the parallel processing mode is perceived as less demanding on a subjective level (Lehle, Steinhauser, & Hübner, 2009), it is also associated with higher dual-task interference and thus higher RT_{S2} under almost all experimental SOA conditions (Lehle et al., 2009; Miller et al., 2009). Within this study, acute stress indeed entailed significantly higher RT_{S2}, which might support the engagement of a rather parallel processing mode under conditions of acute stress. Besides this RT_{S2} operationalization, the CME (i.e., the S1 RT difference between trials in which S1 and S2 require the same vs. different categorizations) has been proposed to specifically reflect dual-task interference and associated processing modes. Here, large dual-task interference should be reflected by a high CME (i.e., parallel processing), whereas low dual-task interference should be reflected by a low CME (i.e., serial processing). Relying on this interpretation, Plessow, Schade, and colleagues (2012) argued for a rather parallel dual-task processing mode under conditions of acute stress. Within this study, however, the CME was only significantly increased after HC administration but was virtually unaffected by acute stress. Thus, our data do not unambiguously confirm that residual stress components lead to an engagement of a more parsimonious (i.e., parallel) processing modes to trade off between sparing mental resources and maintaining high performance levels (cf. Plessow, Schade, et al., 2012). Nonetheless, this conclusion is restricted to the interpretability of the CME as a measure of cognitive adaptivity in dual-task settings.

The outlined ability to dynamically switch between different task processing modes as a function of varying situational task demands has previously been attributed to "cognitive flexibility." This term has also been used to interpret stress effects on the ability to switch between different task sets (which were not manipulated within this study; Beste et al., 2013; Liston, McEwen, & Casey, 2009) and lexical network contents (Alexander, Hillier, Smith, Tivarus, & Beversdorf, 2007). Thus, the term "cognitive flexibility" subsumes a large variety of cognitive processes that can be operationalized with considerable variability (Alexander et al., 2007). The resulting interpretational heterogeneity of study results highlights the need for a more precise nomenclature in terms of a clear distinction between the utilized measures of cognitive constructs (e.g., CME or TRT) and their subjective interpretation (e.g., cognitive adaptivity or processing efficiency). In this regard, we want to emphasize that all result explanations being discussed in this section should be considered with caution until more precise information about how acute stress components jointly and differentially impact on different tasks and performance measures become available.

Methodological Considerations

The combination of (C-)TSST exposure with an HC or PL enabled us to disentangle the effects of endogenous cortisol availability and residual components of the complex, psychosocial stress response. Among the latter, catecholamine secretion has been discussed as one very important mediator of endocrine stress effects on cognitive processes (Arnsten, 2009). Notably, HC intake immediately before the (C-)TSST exposure did not completely suppress the stress-related cortisol secretion through the hypothalamic–pituitary–adrenal feedback (cf. Spiga et al., 2014; Het & Wolf, 2007; Reuter, 2002). Thus, catecholamines and other stress components were probably also present after the TSST exposure and may have contributed

to the reported, specific stress effect on dual-task performance (which was not attributable to cortisol).

To explain the comparably weaker effect of such residual stress components, the timing and length of the cognitive testing periods (in this study, approximately 100 min) seem to be of crucial importance (Shields et al., 2015; Hermans et al., 2014). This is because hormone effects rely on the concentration-dependent saturations of their respective receptors that vary substantially across time whenever hormones exhibit substantially differing kinetic profiles. Specifically, catecholamine secretion occurs almost immediately in response to stress, whereas cortisol is secreted with a delay of approximately 15 min (Engert et al., 2011). Moreover, the elimination half-life of catecholamines ranges from 2 to 5 min (Vendsalu, 1960), whereas the half-life of cortisol ranges from 60 to 100 min (Veldhuis et al., 1989). Thus, the saturation of catecholamine receptors has likely returned to baseline levels after approximately 20 min of testing and impacted less on performance, whereas glucocorticoids (HC) probably dominated the reported effects during the remaining 80 min. This sensitivity to cortisol effects could probably not be compensated by the employed factorial split-plot design, although it had higher statistical power to detect acute stress effects (i.e., the within-participant factor) and their interaction with HC administration (cf. Kirk, 2013). Accordingly, future studies that strive to specifically investigate the impact of residual stress components on cognitive processes should account for such a dominance of glucocorticoid effects by adjustments of the cognitive testing period.

Apart from these considerations, this study includes some limitations that are worth to mention. First, we only tested male participants, to eliminate gender-specific variance in cortisol availability under acute stress and resting conditions (Perogamvros et al., 2011; Kirschbaum et al., 1999) and therefore increase the statistical power of the design. As neither partial report (Coltheart, 1980) nor dual-task performance (Plessow, Schade, et al., 2012) seems to depend on participant gender, we do not expect our conclusions to be restricted to men. Second, most of our participants were university students, which restricted the sample variance in psychometric intelligence and age as compared with the general population. Age was previously shown to strongly modulate both partial report (Lu et al., 2005) and dual-task performances (Verhaeghen, Steitz, Sliwinski, & Cerella, 2003). Similarly, intelligence positively impacts on partial report (Miller, Rammsayer, Schweizer, & Troche, 2010) and dual-task performances (Ben-Shakhar & Sheffer, 2001). Therefore, the present results primarily apply to healthy and comparably young people, and future work should probably strive to examine population-based samples to validate their further generalizability.

APPENDIX

Table A1. Additional Information: Mean (*M*) Dual-Task S1 RTs (RT_{S1}), TRTs ($TRT = RT_{S1} + RT_{S2}$), CMEs ($CME = RT_{S1,Incompatible} - RT_{S1,Compatible}$) in Milliseconds with Standard Deviations (*SD*s) for All SOAs and Experimental Conditions

	Control (N = 20), C-TSST + PL		*Cortisol (N = 20), C-TSST + HC*		*Stress (N = 20), TSST + PL*		*High Load (N = 20), TSST + HC*	
SOA	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Dual-task S1 RTs (RT_{S1})								
20	788.94	116.93	693.72	123.74	829.41	144.79	702.70	108.34
80	796.69	113.84	714.31	142.09	832.89	127.41	709.62	110.17
320	904.41	187.84	769.21	222.25	937.49	190.28	761.04	192.27
1280	1332.34	558.00	951.92	548.72	1389.65	555.16	952.04	529.27
Overall	955.60	373.89	782.29	321.43	997.36	381.61	781.35	303.90
TRT								
20	1711.08	213.18	1527.27	243.88	1808.15	297.76	1541.10	220.34
80	1667.11	209.95	1507.74	275.76	1757.34	270.81	1502.23	229.70
320	1689.16	323.52	1452.19	377.36	1767.86	378.10	1432.39	321.06
1280	2001.14	693.71	1515.42	679.61	2096.32	738.98	1516.15	639.67
Overall	1767.12	425.65	1500.66	422.81	1857.42	473.62	1497.96	386.28
CME								
20	97.72	460.65	66.03	375.61	98.54	502.12	54.49	348.37
80	96.53	442.37	68.24	365.71	111.57	528.33	61.08	352.34
320	102.43	469.53	54.81	355.04	101.13	519.18	41.59	340.66
1280	111.85	478.62	65.83	365.85	116.80	512.01	63.55	350.11
Overall	102.13	462.90	63.73	365.61	107.00	515.42	55.21	347.93
Mental workload ratings (MWT), dual task								
Practice T1	31.18	10.23	27.48	16.18	38.02	11.66	29.13	12.41
Testing T1	28.18	9.45	27.63	16.15	28.97	14.08	29.08	14.52
Practise T2	25.68	10.96	26.64	13.81	26.95	10.04	25.68	17.65
Testing T2	25.15	11.94	25.61	13.92	26.65	10.32	25.88	17.61
Mental workload ratings (MWT), partial report task								
Practice T1	41.72	11.43	34.89	10.70	47.78	11.21	38.56	9.88
Testing T1	37.86	13.47	33.76	11.49	37.61	13.35	37.15	13.43
Practise T2	35.13	14.65	30.53	17.54	35.46	14.40	34.38	16.14
Testing T2	32.76	13.52	32.82	16.66	34.28	12.87	31.54	18.45

Mental workload ratings (MWT in percentage) refer to practice and testing blocks in the first (T1) and second (T2) testing sessions, respectively.

Hierarchical regressions indicated a significant impact of SOA on RT_{S1} and TRT (all $ps < .001$); of HC administration on RT_{S1}, TRT, and CME (all $ps < .05$); and of their interaction on RT_{S1} and TRT (all $ps < .001$). The impact of TSST exposure and/or any other interaction of experimental manipulations on these variables were nonsignificant (all $ps > .15$). MWT ratings significantly decreased from the first to second testing sessions ($\chi^2(1) = 21.67$, $p < .001$) and from practice to testing blocks ($\chi^2(1) = 6.49$, $p < .05$). Partial report processing was rated as more effortful than dual-task processing ($\chi^2(1) = 77.95$, $p < .001$). The significant overall effect of TSST exposure on MWT ratings ($\chi^2(1) = 6.49$, $p < .01$) vanished when the practice block ratings (that were assessed before [C-]TSST exposure) were omitted ($t(79) = 1.01$, $p = .16$). All other effect contributions were nonsignificant (all $ps > .38$).

Acknowledgments

We thank Antoniya Dancheva, Kornelius Schmidt, and Rebekka Röndigs for their assistance with data collection. The German Research Foundation supported this work (DFG, SFB 940/1) but was not involved in the design of the study, the collection and analysis of data, or the decision to publish the presented data.

Reprint requests should be sent to Lisa J. Weckesser, Department of Psychology, Technische Universität Dresden, D-01062 Dresden, Germany, or via e-mail: lisa.weckesser@tu-dresden.de.

Notes

1. To prevent a complete loss of information at long SOAs, Zylberberg and colleagues (2009) postulate that top–down amplification will eventually be randomly allocated if the time of cue presentation exceeds a self-paced deadline. Given that such random amplifications are less accurate than cue-induced directed amplifications, partial report performance decreases with increasing SOAs (Figure 1C) but remains above noise level.
2. The SOA effect on partial report and dual-task performance is most pronounced when there is no delay in between the presentation of the two adjacent stimuli and (exponentially) decreases when the SOA increases (see also Figures 3 and 4 of the present article; Miller et al., 2009; Coltheart, 1980). Therefore, we chose to pool RTs across log-scaled SOAs using nonparametric approximations to the task-specific AUCs.

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.

Bates, D., Mächler, M., Bolker, B. M., & Walker, S. C. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software, 67*. doi:10.18637/jss.v067.i01.

Ben-Shakhar, G., & Sheffer, L. (2001). The relationship between the ability to divide attention and standard measures of general cognitive abilities. *Intelligence, 29,* 293–306.

Best, K.-H. (2005). Zur Häufigkeit von Buchstaben, Leerzeichen und anderen Schriftzeichen in deutschen Texten. *Glottometrics, 11,* 9–31.

Beste, C., Stock, A.-K., Ness, V., Epplen, J. T., & Arning, L. (2012). Differential effects of ADORA2A gene variations in pre-attentive visual sensory memory subprocesses. *European Neuropsychopharmacology, 22,* 555–561.

Beste, C., Wascher, E., Dinse, H. R., & Saft, C. (2012). Faster perceptual learning through excitotoxic neurodegeneration. *Current Biology, 22,* 1914–1917.

Beste, C., Yildiz, A., Meissner, T. W., & Wolf, O. T. (2013). Stress improves task processing efficiency in dual-tasks. *Behavioural Brain Research, 252,* 260–265.

Blurton, S. P., Greenlee, M. W., & Gondan, M. (2015). Cross-modal cueing in audiovisual spatial attention. *Attention, Perception, & Psychophysics, 77,* 2356–2376.

Braver, T. S., & Barch, D. M. (2002). A theory of cognitive control, aging cognition, and neuromodulation. *Neuroscience and Biobehavioral Reviews, 26,* 809–817.

Carrasco, G. A., & Van de Kar, L. D. (2003). Neuroendocrine pharmacology of stress. *European Journal of Pharmacology, 463,* 235–272.

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.

Coltheart, M. (1980). Iconic memory and visible persistence. *Perception & Psychophysics, 27,* 183–228.

Dehaene, S., Sergent, C., & Changeux, J.-P. (2003). A neuronal network model linking subjective reports and objective physiological data during conscious perception. *Proceedings of the National Academy of Sciences, U.S.A., 100,* 8520–8525.

Di Stasi, L. L., Álvarez-Valbuena, V., Cañas, J. J., Maldonado, A., Catena, A., Antolí, A., et al. (2009). Risk behaviour and mental workload: Multimodal assessment techniques applied to motorbike riding simulation. *Transportation Research Part F: Traffic Psychology and Behaviour, 12,* 361–370.

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.

Dux, P. E., Tombu, M. N., Harrison, S., Rogers, B. P., Tong, F., & Marois, R. (2009). Training improves multitasking performance by increasing the speed of information processing in human prefrontal cortex. *Neuron, 63,* 127–138.

Egner, T., & Hirsch, J. (2005). Cognitive control mechanisms resolve conflict through cortical amplification of task-relevant information. *Nature Neuroscience, 8,* 1784–1790.

Engert, V., Vogel, S., Efanov, S. I., Duchesne, A., Corbo, V., Ali, N., et al. (2011). Investigation into the cross-correlation of salivary cortisol and alpha-amylase responses to psychological stress. *Psychoneuroendocrinology, 36,* 1294–1302.

Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods, 41,* 1149–1160.

Fekedulegn, D. B., Andrew, M. E., Burchfiel, C. M., Violanti, J. M., Hartley, T. A., Charles, L. E., et al. (2007). Area under the curve and other summary indicators of repeated waking cortisol measurements. *Psychosomatic Medicine, 69,* 651–659.

Finke, K., Bublak, P., Krummenacher, J., Kyllingsbaek, S., Müller, H. J., & Schneider, W. X. (2005). Usability of a theory of visual attention (TVA) for parameter-based measurement of attention I: Evidence from normal subjects. *Journal of the International Neuropsychological Society, 11,* 843–854.

Fischer, R., & Hommel, B. (2012). Deep thinking increases task-set shielding and reduces shifting flexibility in dual-task performance. *Cognition, 123,* 303–307.

Gao, W., Stalder, T., & Kirschbaum, C. (2015). Quantitative analysis of estradiol and six other steroid hormones in human saliva using a high throughput liquid chromatography–tandem mass spectrometry assay. *Talanta, 143,* 353–358.

Gegenfurtner, K. R., & Sperling, G. (1993). Information transfer in iconic memory experiments. *Journal of Experimental Psychology: Human Perception and Performance, 19,* 845–866.

Graziano, M., & Sigman, M. (2008). The dynamics of sensory buffers: Geometric, spatial, and experience-dependent shaping of iconic memory. *Journal of Vision, 8,* 1–13.

Gugerty, L. (1998). Evidence from a partial report task for forgetting in dynamic spatial memory. *Human Factors, 40,* 498–508.

Hermans, E. J., Henckens, M. J., Joëls, M., & Fernández, G. (2014). Dynamic adaptation of large-scale brain networks in response to acute stressors. *Trends in Neurosciences, 37,* 304–314.

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.

Het, S., & Wolf, O. T. (2007). Mood changes in repsonse to psychosocial stress in healthy young women: Effects of pretreatment with cortisol. *Behavioral Neuroscience, 121,* 11–20.

Hoffman, R., & Al'Absi, M. (2004). The effect of acute stress on subsequent neuropsychological test performance. *Archives of Clinical Neuropsychology, 19,* 497–506.

Kahneman, D. (1973). *Attention and effort*. Englewood Cliffs, NJ: Prentice Hall.

Kaplan, E. L., & Meier, P. (1958). Nonparametric estimation from incomplete observations. *Journal of the American Statistical Association, 53,* 457–481.

Keinan, G. (1987). Decision making under stress: Scanning of alternatives under controllable and uncontrollable threats. *Journal of Personality and Social Psychology, 52,* 639–644.

Kirk, R. E. (2013). Split-plot factorial design: Design with group-treatment confoundig. In *Experimental design: Procedures for the behavioral sciences* (4th ed., pp. 541–620). Thousand Oaks, CA: Sage.

Kirschbaum, C., Bono, E. G., 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., 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.

Kirschbaum, C., Pruessner, J. C., & Stone, A. A. (1995). Persistent high cortisol responses to repeated psychological stress in a subpopulation of healthy men. *Psychosomatic Medicine, 57,* 468–474.

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 & Emotion, 20,* 577–595.

Kuhlmann, S., Kirschbaum, C., & Wolf, O. T. (2005). Effects of oral cortisol treatment in healthy young women on memory retrieval of negative and neutral words. *Neurobiology of Learning and Memory, 83,* 158–162.

Lehle, C., Steinhauser, M., & Hübner, R. (2009). Serial or parallel processing in dual tasks: What is more effortful? *Psychophysiology, 46,* 502–509.

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.

Lu, Z.-L., Neuse, J., Madigan, S., & Dosher, B. A. (2005). Fast decay of iconic memory in observers with mild cognitive impairments. *Proceedings of the National Academy of Sciences, U.S.A., 102,* 1797–1802.

Miller, J., Ulrich, R., & Rolke, B. (2009). On the optimality of serial and parallel processing in the psychological refractory period paradigm: Effects of the distribution of stimulus onset asynchronies. *Cognitive Psychology, 58,* 273–310.

Miller, R., & Plessow, F. (2013). Transformation techniques for cross-sectional and longitudinal endocrine data: Application to salivary cortisol concentrations. *Psychoneuroendocrinology, 38,* 941–946.

Miller, R., Rammsayer, T. H., Schweizer, K., & Troche, S. J. (2010). Decay of iconic memory traces is related to psychometric intelligence: A fixed-links modeling approach. *Learning and Individual Differences, 20,* 699–704.

Miller, R., Weckesser, L. J., Smolka, M. N., Kirschbaum, C., & Plessow, F. (2015). Hydrocortisone accelerates the decay of iconic memory traces: On the modulation of executive and stimulus-driven constituents of sensory information maintenance. *Psychoneuroendocrinology, 53,* 148–158.

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.

Monk, C. S., & Nelson, C. A. (2002). The effects of hydrocortisone on cognitive and neural function: A behavioral and event-related potential investigation. *Neuropsychopharmacology, 26,* 505–519.

Oei, N. Y., Tollenaar, M. S., Spinhoven, P., & Elzinga, B. M. (2009). Hydrocortisone reduces emotional distracter interference in working memory. *Psychoneuroendocrinology, 34,* 1284–1293.

Öğmen, H., Ekiz, O., Huynh, D., Bedell, H. E., & Tripathy, S. P. (2013). Bottlenecks of motion processing during a visual glance: The leaky flask model. *PLoS One, 8,* e83671.

Pashler, H. (1991). Shifting visual attention and selecting motor responses: Distinct attentional mechanisms. *Journal of Experimental Psychology: Human Perception and Performance, 17,* 1023–1040.

Pashler, H. (1994). Dual-task interference in simple tasks: Data and theory. *Psychological Bulletin, 116,* 220–244.

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.

Perogamvros, I., Aarons, L., Miller, A., Trainer, P. J., & Ray, D. W. (2011). Corticosteroid-binding globulin regulates cortisol pharmacokinetics. *Clinical Endocrinology, 74,* 30–36.

Perogamvros, I., Keevil, B. G., Ray, D. W., & Trainer, P. J. (2010). Salivary cortisone is a potential biomarker for serum free cortisol. *Journal of Clinical Endocrinology and Metabolism, 95,* 4951–4958.

Plessow, F., Fischer, R., Kirschbaum, C., & Goschke, T. (2011). 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. *Journal of Cognitive Neuroscience, 23,* 3218–3227.

Plessow, F., Kiesel, A., & Kirschbaum, C. (2012). The stressed prefrontal cortex and goal-directed behaviour: Acute psychosocial stress impairs the flexible implementation of task goals. *Experimental Brain Research, 216,* 397–408.

Plessow, F., Schade, S., Kirschbaum, C., & Fischer, R. (2012). Better not to deal with two tasks at the same time when stressed? Acute psychosocial stress reduces task shielding in dual-task performance. *Cognitive, Affective & Behavioral Neuroscience, 12,* 557–570.

Putman, P., & Roelofs, K. (2011). Effects of single cortisol administrations on human affect reviewed: Coping with stress through adaptive regulation of automatic cognitive processing. *Psychoneuroendocrinology, 36,* 439–448.

R Core Team. (2015). R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing.

Reuter, M. (2002). Impact of cortisol on emotions under stress and nonstress conditions: A pharmacopsychological approach. *Neuropsychobiology, 46,* 41–48.

Saneyoshi, A., Niimi, R., Suetsugu, T., Kaminaga, T., & Yokosawa, K. (2011). Iconic memory and parietofrontal network: fMRI study using temporal integration. *NeuroReport, 22,* 515–519.

Scerra, V. E., & Brill, J. C. (2012). Effect of task modality on dual-task performance, response time, and ratings of operator workload. *Proceedings of the Human Factors and Ergonomics Society Annual Meeting, 56,* 1456–1460.

Schoofs, D., Preuss, D., & Wolf, O. T. (2008). Psychosocial stress induces working memory impairments in an *n*-back paradigm. *Psychoneuroendocrinology, 33,* 643–653.

Shields, G. S., Bonner, J. C., & Moons, W. G. (2015). Does cortisol influence core executive functions? A meta-analysis of acute cortisol administration effects on working memory, inhibition, and set-shifting. *Psychoneuroendocrinology, 58,* 91–103.

Spiga, F., Walker, J. J., Terry, J. R., & Lightman, S. L. (2014). HPA axis-rhythms. *Comprehensive Physiology, 4,* 1273–1298.

Stauble, M. R., Thompson, L. A., & Morgan, G. (2013). Increases in cortisol are positively associated with gains in encoding and maintenance working memory performance in young men. *Stress, 16,* 402–410.

Steinhauser, M., Maier, M., & Hübner, R. (2007). Cognitive control under stress: How stress affects strategies of task-set reconfiguration. *Psychological Science, 18,* 540–545.

Steyer, R., Schwenkmezger, P., Notz, P., & Eid, M. (1997). *Der Mehrdimensionale Befindlichkeitsfragebogen (MDBF)*. Göttingen, Germany: Hogrefe.

Tingley, D., Yamamoto, T., Hirose, K., Keele, L., & Imai, K. (2014). Mediation: R Package for causal mediation analysis. *Journal of Statistical Software, 59,* 1–38.

Tombu, M. N., Asplund, C. L., Dux, P. E., Godwin, D., Martin, J. W., & Marois, R. (2011). A unified attentional bottleneck in the human brain. *Proceedings of the National Academy of Sciences, U.S.A., 108,* 13426–13431.

Tsang, P. S., & Velazquez, V. L. (1996). Diagnosticity and multidimensional subjective workload ratings. *Ergonomics, 39,* 358–381.

Umbricht, D., Schmid, L., Koller, R., Vollenweider, F. X., Hell, D., & Javitt, D. C. (2000). Ketamine-induced deficits in auditory and visual context-dependent processing in healthy volunteers. *Archives of General Psychiatry, 57,* 1139–1147.

Veldhuis, J. D., Iranmanesh, A., Lizarralde, G., & Johnson, M. L. (1989). Amplitude modulation of a burstlike mode of cortisol secretion subserves the circadian glucocorticoid rhythm. *The American Journal of Physiology, 257,* E6–E14.

Vendsalu, A. (1960). Plasma concentrations of adrenaline and noradrenaline in human plasma. *Acta Physiologica Standinavica. Supplementum 173, 49,* 36–48.

Verhaeghen, P., Steitz, D. W., Sliwinski, M. J., & Cerella, J. (2003). Aging and dual-task performance: A meta-analysis. *Psychology and Aging, 18,* 443–460.

Wang, M., Yang, Y., Wang, C.-J., Gamo, N. J., Jin, L. E., Mazer, J. A., et al. (2013). NMDA receptors subserve persistent neuronal firing during working memory in dorsolateral prefrontal cortex. *Neuron, 77,* 736–749.

Webster, M. J., Knable, M. B., O'Grady, J., Orthmann, J., & Weickert, C. S. (2002). Regional specificity of brain glucocorticoid receptor mRNA alterations in subjects with schizophrenia and mood disorders. *Molecular Psychiatry, 7,* 985–994.

Wong, K.-F., & Wang, X.-J. (2006). A recurrent network mechanism of time integration in perceptual decisions. *Journal of Neuroscience, 26,* 1314–1328.

Yildiz, A., Chmielewski, W., & Beste, C. (2013). Dual-task performance is differentially modulated by rewards and punishments. *Behavioural Brain Research, 250,* 304–307.

Yildiz, A., Wolf, O. T., & Beste, C. (2014). Stress intensifies demands on response selection during action cascading processes. *Psychoneuroendocrinology, 42,* 178–187.

Yuen, E. Y., Liu, W., Karatsoreos, I. N., Feng, J., McEwen, B. S., & Yan, Z. (2009). Acute stress enhances glutamatergic transmission in prefrontal cortex and facilitates working memory. *Proceedings of the National Academy of Sciences, U.S.A., 106,* 14075–14079.

Zylberberg, A., Dehaene, S., Mindlin, G. B., & Sigman, M. (2009). Neurophysiological bases of exponential sensory decay and top–down memory retrieval: A model. *Frontiers in Computational Neuroscience, 3,* 1–16.