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Acute Stress Improves Concentration Performance

Opposite Effects of Anxiety and Cortisol

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Abstract. Acute stress can have both detrimental and beneficial effects on cognitive processing, but effects on concentration performance remain unclear. Here, we investigate the effects of acute psychosocial stress on concentration performance and possible underlying physiological and psychological mechanisms. The study sample comprised 47 healthy male participants who were randomly assigned either to a psychosocial stress situation (Trier Social Stress Test) or a neutral control task. Concentration performance was assessed using the d2 Test of Attention before and 30 min after the stress or control task. Salivary cortisol and alpha amylase were repeatedly measured before and up to 1 hr after stress. We repeatedly assessed state anxiety using the State Trait Anxiety Inventory and anticipatory cognitive stress appraisal using the Primary Appraisal Secondary Appraisal questionnaire. The stress group showed a significantly stronger improvement of concentration performance compared to the control group (p = .042). Concentration performance improvement was predicted by increased state anxiety (p = .020) and lower cortisol (stress) changes (p = .043). Neither changes in alpha amylase nor cognitive stress appraisal did relate to concentration performance. Our results show improved concentration performance after acute psychosocial stress induction that was predicted by higher state anxiety increases and lower cortisol increases. This points to a potential modulating role of specific psycho emotional and physiological factors with opposite effects.

Keywords: concentration performance, cortisol, alpha amylase, state anxiety, cognitive stress appraisal, d2 Test of Attention, Trier Social Stress Test

The ability to concentrate or to control attention after stressful experiences is especially crucial in professions such as surgeons or airport security personnel where small mistakes can have fatal consequences. Attention comprises multiple active cognitive processes employed to search, shift, focus, and maintain attention, and different types of attention include selective, focused, or sustained attention (Bates & Lemay, 2004). A core feature of attention is sustained attention (SA, often referred to as concentration), proposed to relate to both perception and processing of information (Krumm, Schmidt-Atzert, & Eschert, 2008). SA, or concentration, respectively, is defined as an attentional process involving the ability to focus on a specific task while ignoring distractions (Murphy & Moran, 2012). SA enables the maintenance of vigilance, selective and focused attention, response persistence, and continuous effort, regardless of changing conditions (Cohen, 2011). In this regard, SA constitutes a prerequisite for higher cognitive and task performances in everyday and professional life (Blotenberg & Schmidt-Atzert, 2019; Krumm et al., 2008). SA assessment typically requires to constantly perform a simple mental operation in a limited time interval (Krumm et al., 2008), as in the Revision Test (Marschner, 1980) or go/no go-tasks (Scholz et al., 2009). A prominent measure of successful SA assessment in terms of concentration performance is the *d2 Test of Attention* where participants are required to cancel targets in the context of multiple distractors (Bates & Lemay, 2004; Brickenkamp, 1994).

Whereas many studies document both positive and negative effects of acute stress on cognitive processing in general (Sandi, 2013; Shields, Sazma, & Yonelinas, 2016) with different stressors influencing cognitive processes differently (Shields, Rivers, Ramey, Trainor, & Yonelinas, 2019), comparably little is known regarding the effects of acute stress on attention. So far, first studies point to differential effects of acute stress on attention with most studies in support of negative effects. Following *acute nonpsychosocial stress induction* by means of aversive movie clips or examination stress selective attention as measured by a Stroop-like task and a telephone search task (as a part of the Test of Everyday Attention) was found to be impaired as compared to the respective control condition (i.e., neutral movie clips or nonexam period; Kohn, Hermans, & Fernández, 2017; Vedhara, Hyde, Gilchrist, Tytherleigh, & Plummer, 2000). Similar findings were observed following *acute psychosocial stress induction*. Participants stressed by a socially evaluated cold pressor task as compared to controls showed higher error rates and concentrated more on irrelevant stimuli in a detection task (Sänger, Bechtold, Schoofs, Blaszkewicz, & Wascher, 2014) but did not differ in SA as measured in a go/no-go task (Banks, Tartar, & Welhaf, 2014). After acute psychosocial stress induction by the highly potent *Trier Social Stress Test* (TSST; Dickerson & Kemeny, 2004; Kirschbaum, Pirke, & Hellhammer, 1993), SA assessed in terms of reaction times in go/no go-tasks (Scholz et al., 2009) and a card choice task (Olver, Pinney, Maruff, & Norman, 2015) was found to be impaired after stress as compared to a control group or baseline, respectively. The hitherto-only study assessing SA by means of concentration performance using the d2 Test of Attention could not observe differences after a group-TSST as compared to a nonstress control condition (von Dawans, Fischbacher, Kirschbaum, Fehr, & Heinrichs, 2012). So far, one study found positive effects of acute stress on attention. In that study, participants were exposed to a cold pressor test and compared with a control group. The stress group responded faster in a selective attention task (flanker task) without influencing accuracy or interference control, notably without prestress baseline assessment (Shields et al., 2019). Notably, all studies assessing attention after acute psychosocial stress induction compared the stressed group either with a nonstress control group or with the baseline of the stressed group. To the best of our knowledge, published peer-reviewed studies are lacking that include both, repeated assessment of SA before and after stress, in addition to a nonstress control group.

With respect to *mechanisms* underlying the effects of acute (psychosocial) stress on attention, it has been proposed that stress may influence cognitive abilities in an inverted-U shaped function depending on perceived intensity of the stressor, with relatively moderate physiological stress parameters relating to highest performance at or near the peak of the inverted-U (Arnsten, 2009; Domes, Rothfischer, Reichwald, & Hautzinger, 2005). The following physiological and psychological parameters may play a role. First, stress-induced activation of the *hypothalamic–pituitary–adrenal (HPA) axis* comprises the release of circulating glucocorticoids (GCs) from the adrenal cortex (de Kloet, Joëls, & Holsboer, 2005) that relate to either impairment of cognitive performance or attention in a dose-dependent (Hsu, Garside, Massey, & McAllister-Williams, 2003) and potentially inverted-U shaped manner (Domes et al., 2005) or are without effect on attention including SA (Olver et al., 2015; Shields et al., 2019). Second, stress activates the *sympathetic nervous system (SNS)* with catecholamine release (de Kloet et al., 2005). Noradrenergic projections have been proposed to influence PFC function in response to stress and consequently higher-order PFC abilities such as working memory and attention regulation (Arnsten, 2009). Also, (nor) adrenergic activity is required for modulation of working memory by GCs (Elzinga & Roelofs, 2005). However, salivary alpha-amylase, a surrogate marker of stress-induced SNS activation (Kuebler et al., 2014; Nater & Rohleder, 2009), was not found to relate to immediate poststress performances in attention including SA (Banks et al., 2014; Sänger et al., 2014).

With respect to psychological stress-responsive parameters, *anxiety* may play a role in mediating stress effects on SA as it can impair cognitive performance, especially when the task is complex and attentionally demanding (Derakshan & Eysenck, 2009). According to the attentional control theory, anxiety increases attention to threat-related stimuli at the expense of attentional control (Eysenck, Derakshan, Santos, & Calvo, 2007). Nevertheless, anxiety does not necessarily impair performance effectiveness in terms of quality as it can induce compensatory strategies such as, e.g., enhanced effort (Eysenck et al., 2007). However, to the best of our knowledge, effects of stress-induced state anxiety changes on attention or concentration performance, respectively, have not yet been investigated. *Anticipatory cognitive stress appraisal* might be a further potential modulating factor in the relationship between stress and cognitive performance, or SA, respectively. According to the transactional model of stress, cognitive stress appraisal results from the difference between primary (comprising perceived threat and/or challenge of a stressful stimulus) and secondary appraisal (perception of own coping abilities; Folkman, Lazarus, Dunkel-Schetter, DeLongis, & Gruen, 1986). Indeed, one pioneer study, notably in women only, found first evidence that cognitive stress appraisal negatively relates to SA in a working memory task (digit span task; Zandara et al., 2016).

Given the above-described reasoning, we set out to investigate whether acute psychosocial stress induced by TSST as compared to a nonstress control task would relate

to changes in concentration performance as measured by the d2 Test of Attention assessed repeatedly before and after the respective task. To shed light into potential underlying mechanisms, we assessed before and repeatedly after each task salivary cortisol and alpha-amylase to exploratively test for possible physiological modulation of stress effects on attention. In addition, we assessed anticipatory cognitive stress appraisal and state anxiety increases from baseline to post-task. We hypothesized that higher cortisol responses as well as higher cognitive stress appraisal and anxiety increases would predict changes in concentration performance from baseline to post-task.

Materials and Methods

Participants

The current investigation is part of a larger research project investigating the effects of acute stress on cognitive function (Thomas et al., 2014). We recruited medication-free physically and mentally healthy male volunteers aged between 18 and 40 years by online advertisement and mailing lists at the University of Zurich. Subjects were required to have normal or corrected-to-normal vision. Interested individuals were screened by a telephone interview using an extensive health questionnaire (Wirtz et al., 2006). Specific exclusion criteria as obtained by participants' self-report were the following: clinical psychosomatic and psychiatric disorders, current infectious diseases, allergies and atopic diathesis, rheumatic diseases, chronic obstructive pulmonary disease, liver and renal diseases, HIV, cancer, elevated blood sugar and diabetes, elevated cholesterol, heart disease, hypertension, varicosis, thrombotic diseases, alcohol and illicit drug abuse, smoking more than four cigarettes per day, and previous participation in studies using stress induction by TSST (Thomas et al., 2014). The initially recruited total sample size was 48 participants (Thomas et al., 2014). For this part of the study, one participant (of the control condition) had to be excluded due to incompletion of the d2 Test of Attention (please see section "Concentration Performance") after stress as main variable of interest rendering a final sample size of 47.

All study participants signed written informed consent and received research participation credits or a monetary reward of 40 Swiss Francs. The research project was approved by the institutional review board of the University of Zurich.

Design and Procedure

In anticipation of the experimental session, subjects were informed to refrain from meals and beverages other than water 1 hr prior to their appointment. Furthermore, they had to abstain from drinks containing caffeine or alcohol since the previous evening and from excessive exercise within 48 hrs before study participation. In order to control for diurnal variations in cortisol secretion (Pruessner et al., 1997), experimental sessions started between 1 p.m. and 2:30 p.m. Participants were randomly assigned to either a stress condition (n = 24) or a control condition (n = 23). Included participants were provided with complete written and oral descriptions of the study, and all subjects expected a challenging task in the study procedure. Upon arrival, participants were welcomed and seated in a quiet room. After 15 min of resting, subjects rated their state anxiety (*State-Trait Anxiety Inventory*, STAI; see below), followed by the baseline assessment of their concentration performance using the d2 Test of Attention (see below). One hour later, half of the participants were exposed to acute psychosocial stress (TSST; see below) in a separate room, while the others accomplished a nonstressful control task of identical duration (calm reading of newspapers and magazines). Five minutes after beginning of each task (i.e., after the introduction in the TSST group), all participants rated again their state anxiety and completed the *Primary Appraisal Secondary Appraisal* questionnaire (PASA; see below). Thirty minutes after completion of the stress or the control task, respectively, concentration performance was measured again. We decided for the 30-min interval between stress cessation and repeated concentration performance assessment as this interval is short enough to capture rapid-acting nongenomic effects of cortisol on cognitive performance and attention (Henckens, van Wingen, Joëls, & Fernández, 2012; Shields, Bonner, & Moons, 2015). At the same time, it is long enough to allow catecholamine levels to fully recover (Nater et al., 2006) and thus eliminate or at least reduce potential catecholamine effects on concentration performance assessment. Two saliva samples for measuring salivary cortisol and alpha-amylase were taken at baseline, i.e., prior to the d2 Test of Attention, and 1 min prior to beginning of the stress or nonstress condition. Saliva samples for poststress/nonstress cortisol and alpha-amylase assessment were taken immediately (+1 min), and 10, 20, 30, 40, 50, and 60 min after stress or nonstress cessation.

Induction of Acute Psychosocial Stress

The TSST is a well-standardized procedure to reliably induce psychosocial stress and resulting neuroendocrine responses (Dickerson & Kemeny, 2004; Kirschbaum et al., 1993). The procedure comprises three 5-min parts in a separate room in front of an unknown panel of two

evaluators and a conspicuous video camera and microphone: a preparation period after a short introduction by the experimenter, a simulated job interview, and an arithmetic task. The panel members were dressed in white laboratory coats and were presented as experts in evaluation of nonverbal behavior. Subjects assigned to the control condition were instructed to read newspapers and magazines in a quiet room corresponding to the duration of the TSST.

Concentration Performance

To assess concentration performance before and after stress/nonstress, participants completed the paper-pencil version of the d2 Test of Attention (Brickenkamp, 1994). The aim of this test is to discriminate target stimuli from visually similar distractors by canceling letters. The test comprises 14 lines, each containing 47 randomly mixed letters. The letters are either "d" or "p" with one, two, three, or four small dashes. Participants were instructed to cross out all "d" with two dashes (below and/or above) as fast and accurately as possible. They had 20 s to finish each line before the experimenter requested them to start with the next line. The test lasts for 4.40 min. Concentration performance was computed by the number of correctly canceled targets minus the number of incorrectly canceled distractors (Bates & Lemay, 2004). Reflecting speed as well as accuracy the value can be seen as an overall performance measure of the d2 Test of Attention. The d2 test has shown adequate reliability and good test retest reliability (Brickenkamp, 2002).

Physiological Measurements

Stimulated saliva samples (by chewing on cotton roles for 1 min) for cortisol and alpha-amylase determination were collected using salivette collection devices (Sarstedt, Rommelsdorf, Germany) and stored at −20 °C until biochemical analysis. To prepare for biochemical analysis, saliva samples were thawed and centrifuged at 3,000 rpm for 10 min to yield low viscosity saliva. Salivary free cortisol concentrations were measured using a commercially available chemiluminescence immunoassay with a high sensitivity of 0.16 ng/ml (LIA; IBL Hamburg, Germany). Inter- and intra-assay variability was below 11.5% and 7.7%, respectively. Alpha-amylase activity was determined using a commercially available enzymatic colorimetric assay according to IFCC with a lower detection limit of 3 U/l (Roche diagnostics GmbH, Mannheim, Germany). Inter- and intra-assay coefficients of variance were <10%.

Psychological Measurements

State Anxiety

To assess state anxiety, subjects completed the state scale of the German version of the STAI (Laux, Glanzmann, Schaffner, & Spielberger, 1981). The STAI state scale measures current anxiety as a transitory emotional state consisting of feelings of strain, worries, and nervousness in face of threatening demands. The scale comprises 20 items with responses on a 4-point Likert scale (ranging from 1 = *not at all* to 4 = *very much so*) rendering possible scores ranging from 20 to 80. The instrument asks participants to rate how they feel "right now, that is, at this moment." Sample items include "I feel calm" and "I feel nervous" with positive items being reverse coded so that higher overall scores indicate greater state anxiety. The scale has good psychometric properties with an internal consistency of α = .90 (Laux et al., 1981).

Cognitive Stress Appraisal

Based on the transactional stress model proposed by Lazarus and Folkman (1984), anticipatory cognitive stress appraisal processes were measured by self-report using the PASA questionnaire (Gaab, Rohleder, Nater, & Ehlert, 2005; Lazarus & Folkman, 1984). This questionnaire comprises the scales primary appraisal and secondary appraisal, each comprising eight items rated on a 6-point Likert scale (ranging from 1 = *strongly disagree* to 6 = *strongly agree*), as well as the global PASA scale, termed stress index. The global stress index scale provides an integrated measure of transactional stress perception by subtracting secondary appraisal from primary appraisal with a possible score range of −20 to 20. Higher scores in the stress index indicate higher stress appraisal. The questionnaire has good psychometric properties, showing good internal consistencies (α = .74 .80; Gaab, 2009).

Statistical Analysis

To detect an expected effect size of .35, the sample size of n = 48 was calculated a priori for the previous study (Thomas et al., 2014). Statistical analysis was performed using SPSS (version 25) statistical software package for Macintosh (IBM SPSS Statistics, Somers, NY). All tests were two-tailed with the significance level set at $p < .05$. All data were tested for normal distribution and homogeneity of variance using Kolmogorov Smirnov and Levene's tests prior to statistical analyses. No outliers were excluded.

Changes in both concentration performance and state anxiety after the respective treatment were calculated as percentage changes from baseline (defined as 100%) to post-treatment. Cortisol and alpha-amylase baseline levels

Table 1. Group characteristics and psychological parameters

	Stress group (n = 24)	Nonstress group (n = 23)	
	M ± *SEM* (range)	*M* ± *SEM* (range)	*p*
Age (years)	24.33 ± 0.89 (19 36)	24.78 ± 0.98 (19 33)	.77
Concentration performance at baseline	278.50 ± 3.33 (235 295)	263.17 ± 5.19 (206 293)	*.017*
Post task concentration performance	287.67 ± 2.14 (261 298)	270.09 ± 5.65 (209 296)	*.006*
State anxiety at baseline	35.13 ± 1.78 (23 59)	34.83 ± 1.97 (22 57)	.82
Post task state anxiety	46.67 ± 2.08 (27 69)	32.48 ± 1.99 (21 54)	*<.001*
Cognitive stress appraisal	−1.65 ± 1.17 (−9 to 11)	−7.52 ± 0.79 (−12.50 to 2.50)	*<.001*
Cortisol baseline levels (nmol/l)	7.76 ± 1.47 (1.90 31.69)	9.97 ± 1.12 (2.86 25.21)	*.037*
Cortisol maximum change (nmol/l)	5.21 ± 1.47 (−9.78 to 22.62)	−4.72 ± 0.69 (−12.02 to 0.32)	*<.001*
Alpha amylase baseline levels (U/ml)	94.17 ± 7.60 (46.05 205.05)	68.78 ± 11.20 (11.55 207.35)	.065
Alpha amylase maximum change (U/ml)	114.95 ± 25.40 (−12.05 to 386.30)	15.98 ± 6.89 (−34.75 to 114.10)	*<.001*

Note. Values are given as means ± *SEM* (range); *n* subsample size. Significant *p* values are highlighted in italics.

were calculated as the mean of the two baseline measurements. Cortisol change scores were computed by cortisol (stress) peak 10 min after the respective treatment minus baseline, and alpha-amylase change scores by peak at 1 min after the respective treatment minus baseline. All measures showing a skewed distribution (age, cortisol baseline, cortisol change, alpha-amylase change, state anxiety baseline, and concentration performance baseline and post-task levels) were log-transformed. While log-transformed data were used for modeling and testing, we depict untransformed data in Table 1 and in Figure 1 for reasons of clarity.

To calculate differences in group characteristics, we used univariate analyses of variance (ANOVAs). In order to test whether the TSST as compared to the control condition induced significant physiological stress responses, we calculated repeated measures ANOVA with repeated assessment of cortisol and alpha-amylase, respectively (each eight repetitions from baseline to +60 min), as the dependent variable and group as the independent variable.

To test our main hypothesis, whether acute stress induces changes in concentration performance that relate to physiological and/or psychological stress reactivity, we used a stepwise procedure. First, to test whether stress as compared to nonstress induces higher increases in concentration performance, we calculated repeated measures analysis of covariance (ANCOVA) with concentration performance percentage scores as the repeated dependent variable and group as the independent variable. We controlled for baseline concentration performance and baseline cortisol levels as covariates to account for the observed significant group differences in both parameters. Notably, baseline-adjusted change-score models have been proposed to provide unbiased effect estimates (Glymour, Weuve, Berkman, Kawachi, & Robins, 2005). Post hoc tests comprise separate reanalysis in each group. Second, to test whether concentration performance changes relate to physiological and/or psychological stress reactivity, we calculated multiple linear regression analyses with concentration performance percentage change as the dependent variable. As independent predictor variables, we entered cortisol and alpha-amylase change scores as well as state anxiety percentage change and cognitive stress appraisal (stress index) simultaneously. Huynh Feldt correction for repeated measures was applied where appropriate.

Results

Group Characteristics

Table 1 provides participants' characteristics of the stress group (n = 24) and the nonstress group (n = 23). Univariate ANOVAs revealed that at baseline the two groups did not significantly differ in age and state anxiety (p's > .77). However, the stress group displayed at baseline higher concentration performance ($F(1, 45) = 6.18$, $p = .017$, partial $\eta^2 = .12$, $f = .37$), lower cortisol concentrations ($F(1, 45) = 4.65$, $p = .037$, partial $\eta^2 = .09$, $f = .31$), and a trend toward higher alpha-amylase levels ($F(1, 45) = 3.57$, $p = .065$, partial $\eta^2 = .07$, $f = .27$). Most stress-related measures are interrelated: Higher cortisol increases correlate with higher salivary alpha-amylase ($r(45) = .49$, $p = .001$) and state anxiety increases ($r(45) = .62$, $p < .001$) as well as higher stress appraisal ($r(45) = .46$, $p = .001$). Higher state anxiety increases correlate with higher alpha-amylase increases ($r(45) = .33$, $p = .022$) and higher stress appraisal ($r(45) = .62$, $p < .001$). There were no associations between stress appraisal and alpha-amylase increases ($r(45) = .08$, $p = .58$).

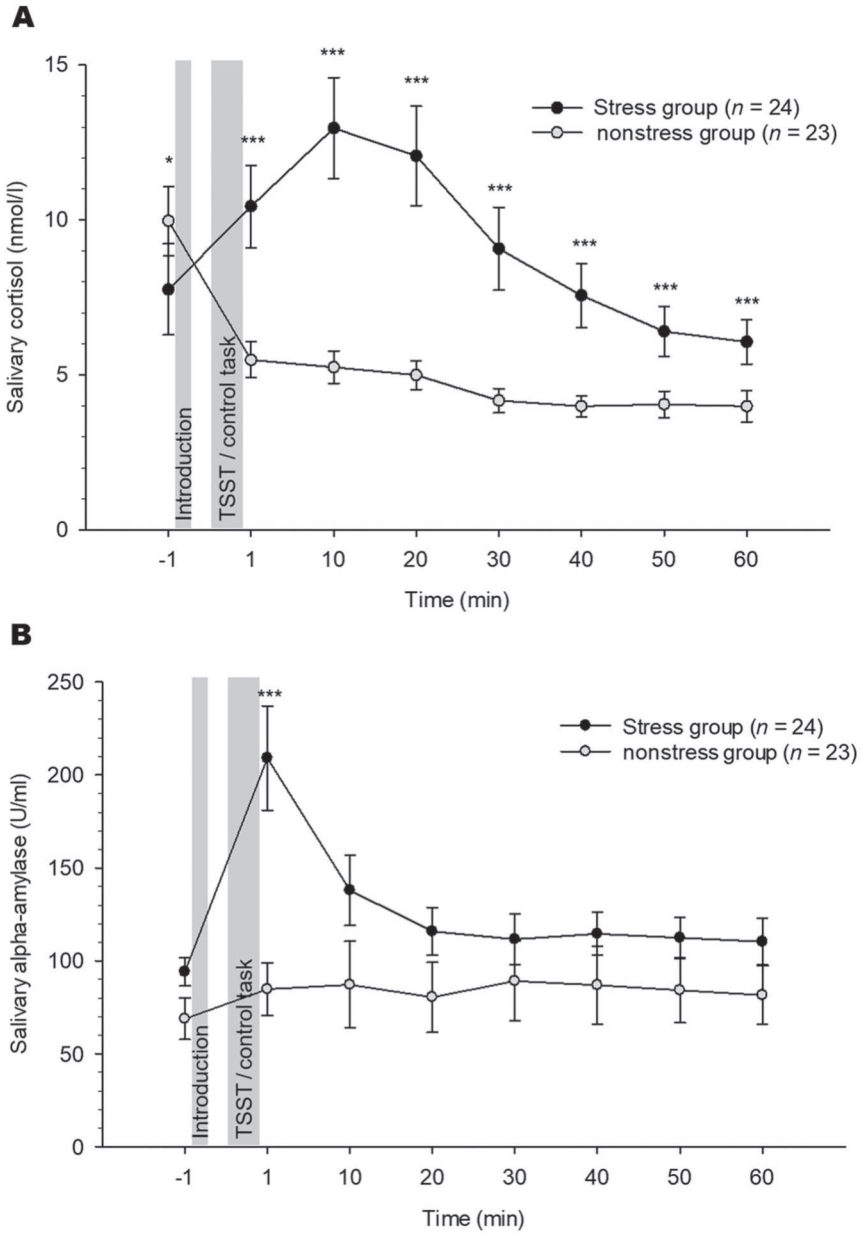

Figure 1. Physiological responses to TSST and control task (mean ± *SEM*): (A) salivary cortisol and (B) salivary alpha amylase. Asterisks indicate significant group differences between stress/nonstress with post task group differences controlled for the respective baseline levels (*$p < .05$; **$p < .01$; ***$p < .001$). TSST = Trier Social Stress Test.

Reactivity of Stress-Reactive Physiological and Psychological Measures Over Time in the Stress and Nonstress Groups

Physiological Reactivity

As depicted in Figure 1, the stress group displayed in reaction to the TSST significant increases in salivary free cortisol ($F(3.3, 149.10) = 23.94$, $p < .001$, partial $\eta^2 = .35$, $f = .73$; Figure 1A) and alpha-amylase ($F(4.6, 206.8) = 3.17$, $p = .011$, partial $\eta^2 = .07$, $f = .27$; Figure 1B) as compared to the control group indicating the successful stress induction by TSST. Accordingly, cortisol ($F(1, 45) = 37.57$, $p < .001$, partial $\eta^2 = .46$, $f = .92$) and alpha-amylase change scores ($F(1, 45) = 16.07$, $p < .001$, partial $\eta^2 = .26$, $f = .59$) were higher in the stress group as compared to the control group (see Table 1).

Psychological Reactivity

Physiological stress reactivity results were complemented by psychological stress indicators: State anxiety significantly increased from baseline to post-treatment in the stress group as compared to the control group: $F(1, 45) = 36.84$, $p < .001$, partial $\eta^2 = .45$, $f = .90$. Moreover, cognitive stress appraisal in anticipation of the stress condition was significantly higher as compared to anticipation of the resting condition ($F(1, 45) = 17.00$, $p < .001$, partial $\eta^2 = .27$, $f = .61$; see Table 1).

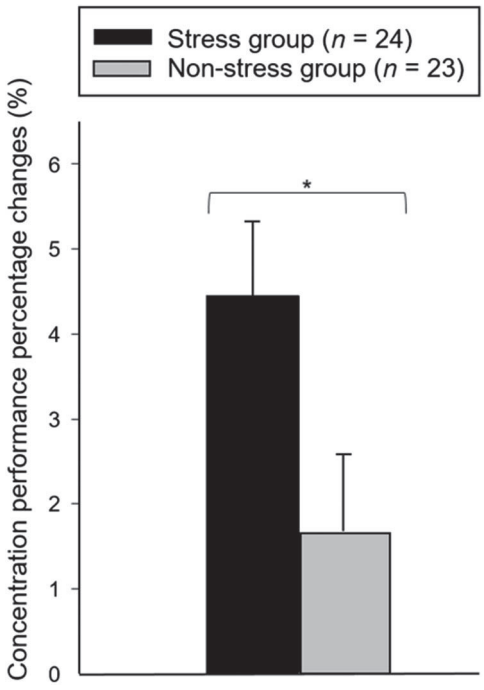

Figure 2. Percentage changes in concentration performance in the stress and nonstress groups (mean ± *SEM*). Independent of baseline differences in cortisol and concentration performance, percentage increases in concentration performance from baseline to post task were higher in the stress group as compared to the nonstress group (p = .042). Asterisks indicate significant group differences (*p < .05).

Concentration Performance Changes in the Stress and Nonstress Groups

Figure 2 depicts percentage changes in concentration performance (from baseline to post-task) in the stress and nonstress groups. Repeated measures ANCOVA revealed higher increases in concentration performance from baseline to post-task in the stress group as compared to the nonstress group independent of baseline cortisol and concentration performance (F(1, 43) = 4.38, p = .042, partial η^2 = .09, f = .31). Post hoc tests reveal that increases in concentration performance over time were significant in the stress group (F(1, 21) = 40.35, p < .001, partial η^2 = .66, f = 1.39), but not in the control group (F(1, 20) = .16, p = .70).

Stress-Related Predictors of Changes in Concentration Performance

To test whether stress/nonstress-induced changes in concentration performance relate to physiological and/or psychological mechanisms, we calculated multiple linear regression analysis (see Table 2) with percentage changes in concentration performance as the dependent variable.

Table 2. Prediction of post task percentage changes in concentration performance

Variables entered	Standardized β	t	p	ΔR^2	Tolerance	VIF
Cortisol maximum change	−.42	−2.09	*.043*	.09	.50	1.99
Alpha amylase maximum change	.08	0.50	.62	.01	.72	1.39
State anxiety change	.51	2.42	*.020*	.12	.46	2.18
Cognitive stress appraisal	−.16	−0.86	.40	.02	.57	1.76

Note. Significant p values are highlighted in italics.

As independent variables, we entered maximum cortisol and alpha-amylase changes as potential physiological predictors in addition to cognitive stress appraisal and percentage changes in post-task state anxiety as potential psychological predictors.

Post-task concentration performance improvement was predicted by lower cortisol increases (β = −.42, p = .043, ΔR^2 = .09) and higher state anxiety increases (β = .51, p = .020, ΔR^2 = .12). Neither task-related changes in alpha-amylase nor cognitive stress appraisal significantly related to post-task concentration performance (p's > .40). Tolerance and variance inflation factor (VIF) values indicate that there are no problems with multicollinearity. To graphically illustrate our findings, Figure 3 depicts groups with higher and lower levels of the significant stress-related predictors of concentration performance changes based on median splits.

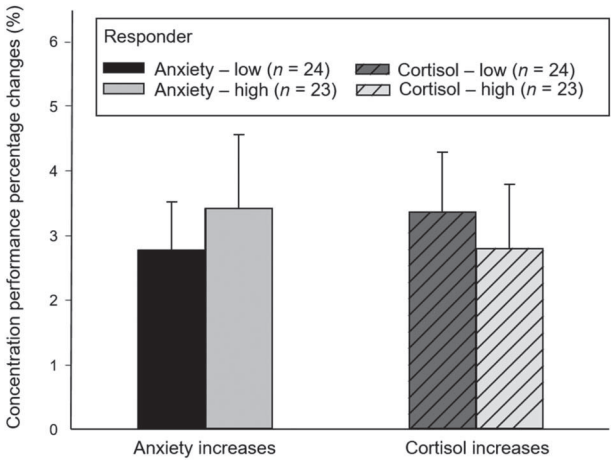

Figure 3. Higher anxiety and lower cortisol increases predict higher subsequent concentration performance increases (state anxiety increases: β = .51, p = .020, ΔR^2 = .12; cortisol increases: β = −.42, p = .043, ΔR^2 = .09). The figure depicts groups with high and low anxiety increases and high and low cortisol increases based on median splits.

Discussion

The main objective of our study was to investigate whether acute psychosocial stress would relate to changes in concentration performance using the d2 Test of Attention assessed repeatedly before and after TSST or a nonstressful control task, respectively. To elucidate potential underlying physiological and psychological mechanisms, we assessed before and repeatedly after each task salivary cortisol and alpha-amylase in addition to anticipatory cognitive stress appraisal and state anxiety increases from baseline to post-task.

While most of the above-cited studies found either impairing (Olver et al., 2015; Sänger et al., 2014; Scholz et al., 2009) or no effects (Banks et al., 2014; von Dawans et al., 2012) after acute psychosocial stress in SA, our main findings indicate that acute psychosocial stress induction by means of the TSST relates to a stronger improvement in concentration performance as compared to a control task. Notably, unlike our study, these studies did either compare post-task SA assessment(s) between a stress group and a nonstress group (Banks et al., 2014; Sänger et al., 2014; Scholz et al., 2009; von Dawans et al., 2012) or reduced SA assessment to pre poststress assessment in a stress group only without control group (Olver et al., 2015). Our finding of stress-induced SA improvement is in line with a previous study, where beneficial effects of cold pressor test-induced acute stress on attention performance were observed (Shields et al., 2019). The main difference between prior studies and our investigation was the use of different SA assessment methods (go/no-go task: Banks et al., 2014; Scholz et al., 2009; card choice task: Olver et al., 2015; detection task: Sänger et al., 2014; flanker task: Shields et al., 2019). Contradictory findings regarding stress effects on SA might thus result from differences in SA tasks and task difficulties. Similarly, inconsistent results of studies investigating stress effects on working memory might be explained by differences in working memory load with stress effects most apparent when working memory load is high (Oei, Everaerd, Elzinga, van Well, & Bermond, 2006). The contradictory finding of the hitherto-only study using the d2 Test of Attention who could not observe group differences between a stress group and a nonstress group in SA (von Dawans et al., 2012) may relate to the timing of the d2 assessment in that study at 5 min after the end of stress induction. At this timepoint, cortisol usually starts increasing but has not yet reached peak reactivity while catecholamines are in the process of decreasing to baseline levels. Our measurement timepoint of 30 min poststress allows us to capture cortisol stress reactivity without catecholaminergic influences, which may explain the differential results.

With respect to potential underlying mechanisms, we found that concentration performance improvement was predicted by lower cortisol (stress) changes and increases in state anxiety while neither changes in alpha-amylase nor cognitive stress appraisal did relate to concentration performance. Impairing cortisol effects on concentration performance improvement are in line with previous findings (Hsu et al., 2003). However, few studies could not find any relation between cortisol and attention (Olver et al., 2015; Shields et al., 2019) with potential explanations including the timing of saliva sampling omitting the cortisol stress response (Olver et al., 2015) or the potency of the stressor to induce a strong HPA axis activation (Shields et al., 2019). Discrepancies may relate to time dependency of GC effects with rapid, nongenomic effects followed by slower, genomic effects that may affect cognitive functions in opposite and complementary ways (Henckens et al., 2012; Shields et al., 2015). We could not find associations between concentration performance and stress-induced salivary alpha-amylase changes. On the one hand, this might be explained by our assessment time of 30 min poststress when alpha-amylase stress reactivity has already been terminated (Nater et al., 2006). On the other hand, previous studies could not find alpha-amylase to relate to immediate poststress performances in attention including SA (Banks et al., 2014; Sänger et al., 2014), suggesting that unlike other SNS activation markers (Arnsten, 2009), alpha-amylase may not relate to attention. Notably, as a mere surrogate marker of SNS activation, alpha-amylase, or alpha-amylase secretion, respectively, has been shown to result from catecholaminergic activation but is not causally involved in SNS activation (Kuebler et al., 2014; Nater & Rohleder, 2009).

With respect to anxiety, we found that stress-induced state anxiety increases related to concentration performance improvements. This finding corresponds, on the one hand, with the capacity theory that posits persons to have a greater amount of available attentional capacity when they are fully alert (Kahneman, 1973; Murphy & Moran, 2012). Notably, this reasoning is not in line with the attentional control theory where anxiety and stress impair attentional processes by forcibly narrowing attention to threat-related stimuli (Eysenck et al., 2007; Shields et al., 2019). On the other hand, persons might use compensatory strategies such as, e.g., enhanced effort in response to anxiety (Eysenck et al., 2007). Whether anxiety increases relate to stress-induced acceleration of executive motor activity (Shields et al., 2019) of relevance for letter canceling the d2 Test of Attention remains to be elucidated. Anticipatory cognitive stress appraisal has been shown to determine cortisol stress reactivity (Gaab et al., 2005), and in our study, stress reactivity predicted concentration performance. Indeed, previous research found cognitive

stress appraisal to negatively relate to SA in a working memory task 15 min after stress cessation (Zandara et al., 2016). As we could not find any associations between cognitive stress appraisal and concentration performance 30 min after stress, we speculate that the delay between stress cessation and the second SA assessment was too long to allow anticipatory appraisal effects to persist.

Strengths of our study include that unlike previous studies we repeatedly assessed SA before and after stress in a stress group as compared to a nonstress control group. This study design allowed us to control for individual baseline differences and learning effects in concentration performance in the d2 Test of Attention. Moreover, we induced acute psychosocial stress using a well-validated and highly potent standardized laboratory stress test (Dickerson & Kemeny, 2004). A limitation of our study is that our study sample comprised healthy young Caucasian men only. Also, our study design with one single stressor of moderate intensity and two concentration performance measurement timepoints does not allow to test for quadratic and thus inverted-U shaped associations between stress-related predictors and concentration performance. Whether our findings are generalizable to populations including women, older or younger individuals, remains unclear. Despite the random assignment of our participants to either the stress or control condition, we found baseline differences in concentration performance and salivary cortisol between the stress and nonstress groups. Notably, we accounted for baseline differences in our analyses. Nevertheless, the observed baseline differences in cortisol and concentration performance between groups limit robustness of our findings. A further limitation is that as we measured SA 30 min after stress cessation when SNS stress reactivity has returned to baseline levels, our design is not well-suited to detect potential SNS effects on concentration performance. This could be one reason why we were unable to find associations between concentration performance and cognitive stress appraisal, or salivary alpha-amylase, respectively. Also, investigation of noninvasive SNS markers other than salivary alpha-amylase would have been desirable. Moreover, the use of a placebo-TSST (Het, Rohleder, Schoofs, Kirschbaum, & Wolf, 2009) would have been a more appropriate control condition.

Our findings suggest that short-time stress or stress-induced anxiety, respectively, may have beneficial effects on concentration performance and thus SA. As we induced acute psychosocial stress using a highly potent but, compared to, e.g., life-threatening events, moderate standardized laboratory stress test, future studies are needed to elucidate the effects of acute stress of different intensities compared to our TSST. Also, effects of chronic stress or critical life events on concentration performance remain unclear. Taken together, our results suggest improved concentration performance after acute psychosocial stress induction by means of the TSST that was predicted by higher state anxiety increases and lower cortisol increases. This points to a potential modulating role of specific psycho-emotional and physiological factors with opposite effects.

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Open Data

The data of all experiments reported here are accessible via PsychArchives under http://dx.doi.org/10.23668/psycharchives.2886

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