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ABSTRACT

This study presents a protocol for induction of moderate psychosocial stress and investigates its impact on psychological and physiological responses. The proposed procedure was designed to enable researchers to assess cognitive performance under effect of various classes of stressors. The protocol's structure contains three main periods: baseline, assessment, and recovery. The assessment stage starts with task anticipation, during which audience (three-member commission) is introduced and apparatus (cameras, microphones, lights, and physiological measuring devices) stationed. Subsequently, cognitive performance was tested. The protocol was evaluated on 56 university students that were randomly assigned to control or stress (protocol) treatment and administered three cognitive tests (working memory operation span, remote associates test, and semantic fluency). Compared to control sessions, protocol induced state anxiety, interfering worry thoughts, and disturbance during recovery period. In addition, the stress group also showed elevated levels of skin conductance, higher average heart rates, and larger drops in peripheral temperature. Even though more research is needed, these results suggest that the protocol effectively induces both psychological and physiological stress responses and therefore encourages utilization in cognitive-affective and cognitive-biological fields of research.

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Anxiety; assessment;
cognition; stress; coping

Simplistic mind-computer analogy, or metaphor, that characterizes the approach of “cold” cognitive psychology in explaining the information processing, may underestimate the documented importance of the interplay between cognitive and affective systems (Pessoa, 2008). Following the cognitive paradigm, authors interested in cognitive functioning typically eliminate affective “confounds” by equalizing participants’ emotional state, making it constant in order to study desired phenomena under neutral conditions. Even though this modus operandi is often reasonable, cognitive functioning during events that are personally significant may be particularly altered by the emotional states they are accompanied with—such as stress and anxiety. Because unsuccessful resolving of a significant problematic event yields serious consequences, it is important to study how these emotional states modulate relevant cognitive processes. For this reason, we aimed to develop a tool that enables researchers to assess cognitive functioning in the

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presence of psychosocial stressors. The present study describes the protocol of this tool and empirically investigates its ability to induce stress.

Findings of a previous meta-analysis (Dickerson & Kemeny, 2004) suggest that the most effective stress-induction combines both social and cognitive evaluation in settings that are novel, unpredictable, and uncontrollable. Given the inherent tendency to protect and preserve one's social self (Baumeister & Leary, 1995; Gilbert, 1997), social evaluation can be understood as a natural stressor that poses a threat for social esteem and social status by requiring a person to manifest socially valued attributes in the presence of others (Dickerson & Kemeny, 2004).

Such threatening situational conditions have been documented to disturb homeostatic equilibrium and consequently trigger a complex repertoire of physiological and psychological responses (Allen, Kennedy, Cryan, Dinan, & Clarke, 2014; Campbell & Ehlert, 2012; Foley & Kirschbaum, 2010). These responses include the activation of the autonomic nervous system and hypothalamo-pituitary-adrenal axis, which manifests in observable physiological changes (e.g., elevated heart rate, sweating, cortisol release). On the subjective level of analysis, stress responses include affective (e.g., anxiety, fear) and perceived physical changes (e.g., perceived trembling, tension).

However, functional changes induced by acute stress also impact broad brain areas responsible for higher cognition (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; Schwabe & Wolf, 2013). Specifically, the effect has been demonstrated on various types of cognitive performance such as attention (Aston-Jones, Rajkowski, & Cohen, 1999), working memory (Elzinga & Roelofs, 2005; Oei, Everaerd, Elzinga, van Well, & Bermond, 2006; Schoofs, Preuß, & Wolf, 2008), memory consolidation (Barsegyan, Mackenzie, Kurose, McGaugh, & Roozendaal, 2010; Schwabe, Wolf, & Oitzl, 2010), cognitive flexibility (Alexander, Hillier, Smith, Tivarus, & Beversdorf, 2007; Hillier, Alexander, & Beversdorf, 2006), goal-switching (Plessow, Fischer, Kirschbaum, & Goschke, 2011; Plessow, Kiesel, & Kirschbaum, 2012), or creativity (Heilman, Nadeau, & Beversdorf, 2003; Martindale & Greenough, 1973). Several physiological mechanisms (e.g., glucocorticoid system, Barsegyan et al., 2010; locus coeruleus-noradrenergic system, Alexander et al., 2007) and psychological theories (e.g. cue utilization theory, Easterbrook, 1959; attention control theory, Eysenck, Derakshan, Santos, & Calvo, 2007) have been proposed to account for these complex stress-impairing effects.

Stress-investigating psychobiological research commonly uses well-established stress protocols such as Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), Manheim Multicomponent Stress Test (MMST; Reinhardt, Schmahl, Wüst, & Bohus, 2012), cold-pressor test (CPT; Hines & Brown, 1932), or socially evaluated cold-pressor test (SECPT; Schwabe, Haddad, & Schachinger, 2008). The former has been probably the most frequently used and recognized tool since its publication. TSST protocol consists of baseline period, anticipatory period, test period, in which participants have to deliver a speech and perform mental arithmetic in front of a committee, and final recovery. Despite the amount of research that support the well-established protocols (e.g., Foley & Kirschbaum, 2010; Hellhammer & Schubert, 2012; Het, Rohleder, Schoofs, Kirschbaum, & Wolf, 2009; Kolotylova, Pirke, & Hellhammer, 2010; Otto, Raio, Chiang, Phelps, & Daw, 2013; Porcelli et al., 2008; Schwabe & Wolf, 2010; Von Dawans, Kirschbaum, & Heinrichs, 2011), their standard structure and content may not fit the requirements necessary to study the effects of stress on other

psychological (e.g., cognitive) functions. This leads researchers to either modifying a given protocol (Alexander et al., 2007; Dedovic et al., 2005) or adding crucial tests after stressors have been removed, assuming their sustained or lagged effect (Luethi, Meier, & Sandi, 2008; Plessow et al., 2011; Schoofs et al., 2008).

However, a large body of evidence implies that the effect of stress on certain types of cognitive performance substantially differs depending on stress-to-testing latency (e.g., Elzinga & Roelofs, 2005; Schoofs et al., 2008; Schwabe & Wolf, 2013). Additionally, after the stressors are removed, their effect on subsequent performance might not only be attenuated but may even lack critical features since the stimuli are no longer physically present to distract or bias cognitive processing (Eysenck et al., 2007; LeBlanc, 2009). Hence, both protocol modification and post-protocol assessment bring doubts concerning internal validity.

In the present study, we addressed the abovementioned issue by proposing a psychosocial stress protocol which enables researchers to test cognitive performance in the presence of valid and reliable stressors. The aim of our research was to provide necessary evidence to support the protocol's validity (i.e., to support its ability to induce a stress response) at both physiological and psychological level. In contrast to control settings, we expected that stress treatment would elevate sympathetic activity as indexed by higher skin conductance level (Boucsein, 2012; Lim et al., 1997; Van Dooren, de Vries, & Janssen, 2012), increased heart rate (Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004; Turner, 1994), and a drop in peripheral temperature (Vinkers et al., 2013). Additionally, we also expected that treatment would induce higher state anxiety, more intrusive worries (cognitive interference; Sarason, Sarason, Keefe, Hayes, & Shearin, 1986) and more profound disturbance during post-stress recovery than control treatment.

Method

Participants

Fifty-six healthy first and second year university students (16 males, 40 females) in age between 18–23 years ($M_{\text{age}} = 19.9$, $SD = 1.2$) and body mass index between 17–28 ($M_{\text{BMI}} = 21.5$, $SD = 3.7$) were recruited from a larger pool of volunteers that attended various universities in western Slovakia (45% of participants studied social sciences, 29% medial communications, and 26% technology). The group was screened for trait anxiety with the Spielbergers Trait Anxiety Inventory (STAI-T, Spielberger, 1989) and divided into three blocks (low, medium, and high trait-anxiety) prior to participating. Only native Slovak speakers with no learning disability were selected. Additionally, participants had to refrain from excessive exercise, alcohol (24 hours for both), and caffeine (12 hours) prior to participation. Their abstinence was checked by a brief interview before all sessions.

Experimental and control group were equal in age, they did not differ in the percentage of females, $\chi^2(1) = .350$, $p = .554$, trait anxiety level, $\chi^2(2) = .102$, $p = .950$, body mass index, $t(54) = -.192$, $p = .849$, or session time, $t(54) = -.242$, $p = .810$. Between the groups, there were also no differences in initial state anxiety, $t(54) = .732$, $p = .465$, positive affect $t(54) = -.194$, $p = .847$, or negative affect $t(54) = 1.37$, $p = .176$. Due to measurement or administration errors, less than 2% of data were missing. The project was approved by the ethics committee of Trnava Self-Governing Region. A written informed consent was obtained from all subjects to participate.

Design and Procedure

A blind, controlled, and blocked study design was used. Experimental sessions were run between 9:00 and 17:00. Participants from each trait-anxiety block (Trait-anxiety factor) were randomly assigned either to stress or control group (Treatment factor) of equal size ($N = 30$), and individually completed three different cognitive tests (Time factor) in random order (Task-sequence factor). Each session started with a brief interview followed by assessment of initial affect (about 10 min). Subsequent experimental procedure consisted of three periods: baseline, cognitive testing, and recovery. Baseline and recovery were fixed to 10 minutes. Within this time, participants were left alone sitting quietly in a room, instructed to relax and attenuate body movements as much as possible. The cognitive tasks lasted about 30 minutes in total. Before cognitive testing, pretest State Anxiety Inventory was administered. After the testing, post-test State Anxiety Inventory and Cognitive Interference Questionnaire were completed. Recovery disturbance scale (Marko & Brezina, 2015) was administered after recovery period. At the end of the procedure, each participant was moved to second room, where the session was discussed. The exact sequence of the protocol is shown in Figure 1.

Stress and Control Treatment

Trier Social Stress Test (Kirschbaum et al., 1993) was modified for cognitive assessment purposes to induce a state of stress and anxiety (Stress treatment). Despite the modifications, the protocol contained all effective features known to reliably elicit stress (Dickerson & Kemeny, 2004). After the 10-minute period of adaptation, experimenter and three-member committee dressed in white coats entered the laboratory. Within the first three minutes, the apparatus was stationed and the committee members were introduced as experts in behavioral and psychological analysis (including the analysis of facial and speech expressions, voice, body language, logic and rationale of answers, cognitive and personality parameters). The apparatus consisted of proximal camera projecting face on feedback monitor, distal camera, spotlight and a microphone on a stand placed in front of the seated participant. Instructions informed participants that the recordings would be subjected for additional analyses for evaluation of performance, voice, and nonverbal expressions. The committee was instructed to observe participant's behavior, take notes, and provide no positive reinforcement while participants were cognitive assessed. Transitions between each cognitive task were filled by succinct questions about general knowledge and commonsense (e.g. what

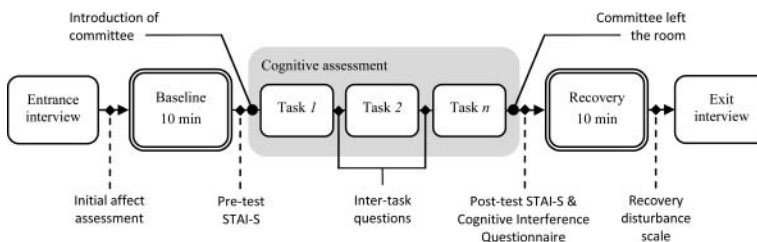


Figure 1. Time representation of the stress protocol. Each participant entered the room separately and went through all stages of the protocol: baseline, cognitive assessment (three cognitive tests in random order), and recovery. Solid lines represent elements present only in stress treatment.

time is it now in Tokyo?) in a time sensitive manner to sustain testing dynamics. The control group underwent cognitive testing without abovementioned stressors. The settings of the respective treatments are shown in Figure 2.

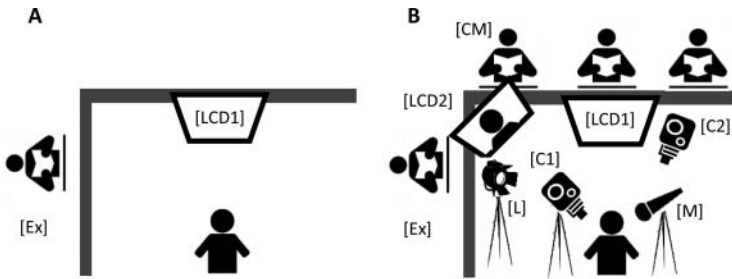


Figure 2. Spatial representation of control (A) and stress (B) treatment's settings and equipment stationing. CM = committee members; EX = experimenter; LCD1 = exposition monitor; LCD2 = feedback monitor; C1 = proximal (face) camera; C2 = distal camera; L = spotlight; M = microphone.

Cognitive Tests

Working memory capacity was tested by means of the operation span task. In each trial within nine blocks, the participant was instructed to solve an arithmetic problem (e.g., “ $7 + 7 \cdot 8 = 63$ ”) exposed on a monitor and to remember a word provided by a committee member orally (e.g., “coffee”). As recommended (Unsworth, Heitz, Schrock, & Engle, 2005; Clair-Thompson, 2012), the length was not predictable and at least 70% of arithmetic problems were explicitly required. Cognitive flexibility was assessed by Remote Associates Test (RAT; Mednick, 1962). This test required participants to generate a valid word meaningfully associated with all three cue words presented for 25 seconds. The test consisted of 22 items aligned by increasing difficulty. In semantic fluency, the participant was asked to generate as many instances of category determinant as possible in a defined time span. We assessed three categories: “Animals,” “Supermarket products,” and “Occupations.” Participants were given one minute for each.

Affective Assessment

As a possible confounding source, initial affect was assessed. Prior to testing, participants were asked to rate their actual affective state by fifteen adjectives (e.g., “Happy”) on 7-point Likert scale from 0 (*not at all*) to 6 (*very much*). Two orthogonal composites with acceptable consistency were subsequently created: positive affect (6 adjectives; $\alpha = .89$) and negative affect (9 adjectives; $\alpha = .92$). Additionally, State Anxiety Inventory was completed before and after the cognitive block in order to assess state anxiety (Spielberger, 1989). The inventory is based on a 4-point Likert scale and consists of 20 questions. Internal consistency of the pretest and the post-test for this sample was of $\alpha = .82$ and $\alpha = .95$, respectively.

Cognitive Interference and Recovery Disturbance Assessment

In order to assess self-reported cognitive interference, the “Task-oriented Worries” of Cognitive Interference Questionnaire (Sarason et al., 1986) was utilized. Participants were asked to

indicate the frequency of occurrence of task-related thoughts that intruded their mind while completing the cognitive tasks on a 5-point scale from 1 (*never*) to 5 (*very often*). Cronbach's α was .87 for this sample. Disturbance of recovery phase, which followed the cognitive block, was assessed by eight questions on a 5-point scale from 1 (*never*) to 5 (*very often*). Content of the questions mapped to what extent thoughts and feelings of previous testing disturbed instructed emotional recovery and to what extent the recovery was successful (e.g., "*Tension and anxiety of previous testing disturbed my effort to relax*," "*During the last period, I was able to unwind*," "*While relaxing, I felt worried about my previous performance*"). Cronbach's α for the ad-hoc Recovery disturbance scale was of .92 for this sample.

Physiological Measures and Signal Processing

Physiological data were acquired by a 16-bit, multimodal, portable device Neurobit Optima 4 with sampling rate of 1000 Hz. Its wireless real-time data transmission provided full galvanic isolation of the subject's body. The laboratory's average temperature of $24.5 \pm 1^\circ\text{C}$ (at 34% humidity) was equal in both conditions. Electrodermal activity was measured by two reusable 100 mm AgAgCl electrodes placed on medial phalanxes (index and ring finger) of the non-dominant hand. Skin conductance level was derived from the raw signal by applying 0.1 Hz low-pass filter. The resulting signal was subsequently corrected for drifts by down-sampling a minima in 10-second moving window and smoothed afterward. This correction removed a portion of the signal inflated by phasic dermal activity. For single-lead configuration (lead-II placement) of continuous heart rate measurement (in beats per minute), two disposable 80 mm AgAgCl spot electrodes were used. Heart rate was derived from normal-to-normal beat intervals of an electrocardiogram that was filtered by 5–35 Hz band-pass filter. Peripheral temperature was measured by a surface sensor attached to participants' dorsal side of middle finger.

Results

In order to normalize between-subject variability, the physiological variables and state anxiety in each time points were transformed into relative scores in order to get the change at a given time point relative to basal level (i.e., %SCL, %HR, %TEMP, %SA), and then compared between conditions. Variables without baseline level (cognitive interference, recovery disturbance) were computed as raw scores.

All repeated measures analyses of variance were adjusted by Greenhouse-Geisser sphericity correction. If statistically significant, they were additionally corrected for trait anxiety level and sequence of cognitive tasks. Partial eta-squared statistic (η_p^2) was computed as a standard estimate effect size. Plots showing estimated marginal means and 95% confidence intervals of all six analyses are shown in [Figure 3](#). The results of related statistical analyses are summarized in [Table 1](#) and [Table 2](#).

Physiological Indicators

Skin Conductance Level

A repeated measures ANOVA, corrected for Trait-anxiety and Task-sequence, showed a significant main effect of Time, $F(2, 53) = 147.14, p < .001, \eta_p^2 = .85$, Treatment, $F(1, 27) =$

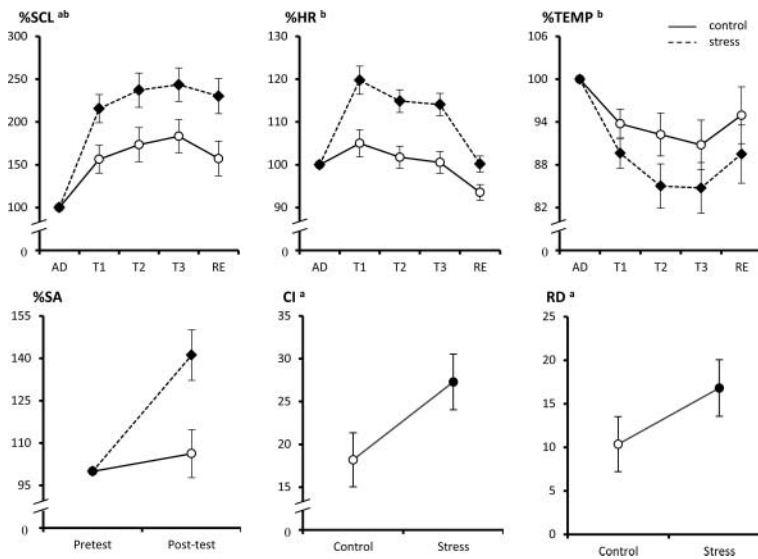


Figure 3. Estimated marginal means of physiological and psychological stress responses for stress and control treatment. All plots display 95% confidence intervals. %SCL = change in skin conductance level; %HR = change in heart rate; %TEMP = change in peripheral temperature; %SA = change in state anxiety; CI = cognitive interference; RD = recovery disturbance. AD = adaptation period; T1 to T3 = respective cognitive test; RE = recovery period. Superscript "a" and "b" indicate that ANOVA model was corrected for significant effect of Trait-anxiety and/or Task-sequence factor, respectively.

Table 1. Summary of results for physiological measures (%SCL, %HR, %TEMP).

Measure	Effect	SS	df	MS	F	p	η_p^2
%SCL	Time	320384	2.0	163330	147.14	<.001	.85
	Treatment	170142	1.0	170142	30.20	<.001	.53
	Time \times Treatment	44456	2.0	22664	20.42	<.001	.43
%HR	Time	8412	2.3	2103	97.36	<.001	.69
	Treatment	5981	1.0	5981	67.97	<.001	.61
	Time \times Treatment	2012	2.3	873	23.29	<.001	.35
%TEMP	Time	4846	2.4	2051	43.24	<.001	.51
	Treatment	1352	1.0	1352	8.59	.005	.17
	Time \times Treatment	403	2.4	171	3.59	.024	.08

30.20, $p < .001$, $\eta_p^2 = .53$, and their interaction, $F(2, 53) = 20.42$, $p < .001$, $\eta_p^2 = .41$, on skin conductance level change (%SCL). Bonferroni corrected post-hoc analyses of the interaction indicated that %SCL between baseline period and first cognitive test changed significantly more in stress group than in control group, $F(1, 27) = 28.72$, $p < .001$, $\eta_p^2 = .52$, and then it sustained elevated comparatively for the rest of the session.

Table 2. Summary of results for psychological measures (%SA, CI, RD).

Measure	Effect	SS	df	MS	F	p	η_p^2
%SA	Treatment	15736	1	15736	30.81	<.001	.36
CI	Treatment	1062	1	1062	16.29	<.001	.26
RD	Treatment	477	1	477	13.64	<.001	.24

Heart Rate Level

A second repeated measures ANOVA, corrected for Tasks-sequence, also indicated a significant main effect of Time, $F(2.3, 99) = 97.36, p < .001, \eta_p^2 = .69$, Treatment, $F(1, 43) = 67.97, p < .001, \eta_p^2 = 0.61$, and their interaction, $F(2.3, 99) = 23.29, p < .001, \eta_p^2 = 0.35$, on heart rate change (%HR). Bonferroni corrected post-hoc analyses of the interaction indicated that %HR level changed significantly more for stress group between baseline period and first cognitive test, $F(1, 43) = 42.34, p < .001, \eta_p^2 = .50$, while stress group recovered to lower heart rate level after the last cognitive test, $F(1, 43) = 11.89, p = .006, \eta_p^2 = .22$.

Peripheral Temperature

A repeated measures ANOVA, corrected for Tasks-sequence, revealed a significant main effect of Time, $F(2.3, 102) = 43.24, p < .001, \eta_p^2 = .51$, Treatment, $F(1, 43) = 8.58, p = .005, \eta_p^2 = 0.17$, and their interaction, $F(2.3, 102) = 3.60, p = .024, \eta_p^2 = 0.08$, on peripheral temperature change (%TEMP). After Bonferroni correction, the temperature drop between basal level and first task was significantly bigger in stress group than in control group, $F(1, 43) = 7.98, p = .028, \eta_p^2 = 0.16$. The drop between first and second cognitive task yielded similar trend, however, it was only marginally significant, $F(1, 43) = 6.74, p = .052, \eta_p^2 = 0.14$.

Psychological Indicators

As for state anxiety, an analysis of variance showed that stress group indicated higher post-test state anxiety change (%SA) when compared to the control group, $F(1, 44) = 30.81, p < .001, \eta_p^2 = .36$. Additionally, the stress group reported more cognitive interference, $F(1, 47) = 16.29, p < .001, \eta_p^2 = .26$, and higher recovery disturbance during recovery period, $F(1, 43) = 13.64, p < .001, \eta_p^2 = .24$ (both analyses were corrected for Trait-anxiety).

Discussion

Elevated levels of various physiological and psychological stress indicators has been reported in many previous studies (Bassett, Marshall, & Spillane, 1987; Hellhammer & Schubert, 2012; Kirschbaum et al., 1993; Reinhardt et al., 2012; Schwabe et al., 2008; Trestman et al., 1991). However, with respect to the protocols' applicability, they were designed to enable the investigation of individual differences in the pattern of stress response as such. Building on their effective features, the contribution of our study lies in the development of a standard protocol that allows assessment and investigation of cognitive performance under psychosocial stress. Although a number of studies apply procedures for similar purposes, their effectiveness is sometimes questionable. For example, Renner and Beversdorf (2010) used a war drama movie as a naturalistic stressor to study its effect on subsequent problem-solving and memory performance; Hillier et al. (2006) exposed participants to 90 dB white noise while working on verbal and visuo-spatial tasks; Coy, O'Brien, Tabaczynski, Northern, and Carels (2011) manipulated the instruction (supporting vs. evaluative) before working memory was tested; finally, Lewis, Nikolova, Chang, and Weekes (2008) tested students' working memory within periods they were naturally stressed by taking major examinations.

Progress in theory and methods can, indeed, capitalize on the systematic use of diverse stress-induction procedures. However, if these techniques lack sufficient accuracy and power

to induce the desired state of mind, they may yield inconsistent results and thus hinder theoretical synthesis. This problem is plausible to occur mainly if ad hoc protocols are used or their outcomes are not described comprehensively. For this reason, we also performed empirical verification of the protocol.

As expected, the proposed protocol had a significant effect on skin conductance level, heart rate, and peripheral body temperature, but it also affected all three psychological stress dimensions assessed after stressors were physically eliminated: state anxiety, occurrence of worrying thought, and degree of disturbance transferred to recovery period. This indicates that the protocol has a reproducible and consistent effect on those measures. More importantly, substantial effects of the manipulation on the measures were rather large. Although the temperature measured on finger's surface was the least affected measure, its effect size of approximately 6.5% ($\sim 2^\circ\text{C}$ drop) is comparable to the results of TSST reported by Vinkers et al. (2013), suggesting this indicator may be less sensitive in general. In spite of that, the proposed protocol had more profound overall impact on autonomic than psychological responses. Because autonomic responses have not been included in the meta-analyses of Dickerson and Kemeny (2004), more detailed comparisons of our results to other stress paradigms are precluded.

Following this robust evidence, we conclude that the present protocol can be validly used to induce critical changes in both physiological and psychological stress markers and therefore can be utilized in research investigating their impact on desired cognitive parameters.

However, several considerations should be mentioned. Firstly, previous research indicated that personality traits related to emotional sensitivity and appraisal plausibly influence the pattern of stress response (Allen et al., 2014). Consistently with this evidence, we found that trait anxiety had statistically and substantially significant effect on skin conductance, cognitive interference, and recovery disturbance. Blocking and controlling of such intervening personality variables is therefore recommended. Secondly, our results also indicated that characteristics of specific cognitive tests and their order are plausible to influence the observed stress response. Post-hoc qualitative analysis of task sequences revealed that Remote Associates Test was possibly less stressful than the other two tests, since it induced relatively smaller initial change in skin conductance and heart rate when administered as first. The limited stressfulness of RAT might be due to smaller effort requirements and less restricted testing dynamics (e.g., timing, stimuli change). These findings thus emphasize that cognitive tasks incorporated into the protocol should be selected thoughtfully; tasks bearing no stress or no time constraints might partially diminish the desired stress response. Consideration of each candidate task should be therefore done in advance. Furthermore, the complete cognitive assessment lasted approximately 30 minutes in total. The course of the effect therefore cannot be predicted with a certainty. However, we assume that 30-minute testing span might be satisfactory for most research needs, and any prolonged testing could become exhausting. Although it was not of the primary interest, the contribution of the protocol's individual features might also be worth studying. A step-wise addition and combination of stress-inducing agents (e.g., committee, instruction, and equipment) may provide systematic means for estimating their separate effects on the stress response pattern. Such analysis can lead to further refinement in the research design.

Finally, the presence of an experimenter in control settings might have been thought to affect observed stress responses. However, studies by Dickerson, Mycek, and Zaldivar (2008) and Wiemers, Schoofs, and Wolf (2013) have showed that the mere social presence of others lack the effects of direct social evaluation. It's therefore implausible that this aspect biased observed treatment differences.

Our research has several limits. Participation in the study was restricted to university students with rather narrow age boundaries. Broader generalization therefore requires further research on participants of different ages and education. Likewise, the assessment of participants' emotional profile was also restricted. Various emotions (e.g., anger, fear, or sadness) may be associated with the observed stress response; therefore, it would be important to support internal validity of the protocol (i.e., verify that only stress-related changes occurred) by mapping these emotional states via established psychological measures. More importantly, the present study did not address more prolonged stress-induced response of the hypothalamus-pituitary-adrenal axis, which leads to the synthesis and release of glucocorticoids (mainly cortisol) into the bloodstream (e.g., de Kloet, Joels, & Holsboer 2005). Glucocorticoids pass the blood-brain barrier and bind to glucocorticoid receptors in the prefrontal cortex which modulates higher cognition (Barsegyan et al., 2010; Roozendaal, McReynolds, McGaugh, 2004; Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012). It has been documented that some protocols are less capable of provoking activity of hypothalamus-pituitary-adrenal axis (e.g., Denson, Creswell, & Granville-Smith, 2012; Schwabe et al., 2008; Wiemers et al., 2013). However, this deficiency is plausibly related to the absence of social-evaluative elements (Dickerson et al., 2008) which have been documented to have robust effects on the hypothalamus-pituitary-adrenal axis (Dickerson & Kemeny, 2004). Because the proposed protocol includes social evaluation, it could be assumed that it also activates the release of cortisol. This assumption needs further investigation.

In conclusion, our study described a modified psychosocial protocol and provided supporting evidence of its validity to induce the pattern of responses that typically occur under moderate stress. These results imply that the union of simultaneous psychosocial stressors and cognitive testing forms an effective and reliable tool suitable to investigate stress-induced modulation of cognitive functioning. The findings may partially aid the methodological issues related to the questionable efficiency and validity of ad hoc stress protocols (and controversial adaptations of standard stress protocols) that often appear in psychophysiological research, and also deal with the aforementioned stress-to-testing latency problem. Therefore, the presented structure of experimental stress settings may not only enhance the related research methodology, but may also expand the possibilities of a researcher during research designing stage. Further projects are however needed in order to broaden the scope of its applicability and resolve the aforementioned limits.

Author Note

Martin Marko is a researcher at the Comenius University in Bratislava. His current research interests concern modulation of cognition under stress and anxiety, structure and dynamics of idea generation, and cognitive flexibility.

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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*(3), 468–478. doi.org/10.1162/jocn.2007.19.3.468
- Allen, A. P., Kennedy, P. J., Cryan, J. F., Dinan, T. G., & Clarke, G. (2014). Biological and psychological markers of stress in humans: focus on the Trier Social Stress Test. *Neuroscience and Biobehavioral Reviews*, *38*, 94–124. doi.org/10.1016/j.neubiorev.2013.11.005
- Aston-Jones, G., Rajkowski, J., & Cohen, J. (1999). Role of locus coeruleus in attention and behavioral flexibility. *Biological Psychiatry*, *46*(9), 1309–1320.
- 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 of the United States of America*, *107*(38), 16655–16660. doi.org/10.1073/pnas.1011975107
- Bassett, J. R., Marshall, P. M., & Spillane, R. (1987). The physiological measurement of acute stress (public speaking) in bank employees. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *5*(4), 265–273.
- Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin*, *117*, 497–529.
- Boucsein, W. (2012). *Electrodermal activity*. Boston, MA: Springer.
- Campbell, J., & Ehlert, U. (2012). Acute psychosocial stress: does the emotional stress response correspond with physiological responses? *Psychoneuroendocrinology*, *37*(8), 1111–1134. doi.org/10.1016/j.psychneuen.2011.12.010
- Clair-Thompson, H. S. (2012). Ascending versus randomised list lengths in working memory span tasks. *Journal of Cognitive Psychology*, *24*(3), 335–341. doi.org/10.1080/20445911.2011.639760
- Coy, B., O'Brien, W. H., Tabaczynski, T., Northern, J., & Carels, R. (2011). Associations between evaluation anxiety, cognitive interference and performance on working memory tasks. *Applied Cognitive Psychology*, *25*(5), 823–832. doi.org/10.1002/acp.1765
- de Kloet, E. R., Joels, M., & Holsboer, F. (2005). Stress and the brain: from adaptation to disease. *Nature Reviews Neuroscience*, *6*(6), 463–475.
- Dedovic, K., Renwick, R., Mahani, N. K., Engert, V., Lupien, S. J., & Pruessner, J. C. (2005). The Montreal Imaging Stress Task: using functional imaging to investigate the effects of perceiving and processing psychosocial stress in the human brain. *Journal of Psychiatry and Neuroscience*, *30*(5), 319–325.
- Denson, T. F., Creswell, J. D., & Granville-Smith, I. (2012). Self-focus and social evaluative threat increase salivary cortisol responses to acute stress in men. *Journal of Behavioral Medicine*, *35*(6), 624–633. doi.org/10.1007/s10865-011-9393-x
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*(3), 355–391. doi.org/10.1037/0033-2909.130.3.355
- Dickerson, S. S., Mycek, P. J., & Zaldivar, F. (2008). Negative social evaluation, but not mere social presence, elicits cortisol responses to a laboratory stressor task. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association*, *27*(1), 116–121. doi.org/10.1037/0278-6133.27.1.116
- Easterbrook, J. A. (1959). The effect of emotion on cue utilization and the organization of behavior. *Psychological Review*, *66*(3), 183–201.

- Elzinga, B. M., & Roelofs, K. (2005). Cortisol-induced impairments of working memory require acute sympathetic activation. *Behavioral Neuroscience*, *119*(1), 98–103. doi.org/10.1037/0735-7044.119.1.98
- Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: attentional control theory. *Emotion (Washington, D.C.)*, *7*(2), 336–353. doi.org/10.1037/1528-3542.7.2.336
- Foley, P., & Kirschbaum, C. (2010). Human hypothalamus-pituitary-adrenal axis responses to acute psychosocial stress in laboratory settings. *Neuroscience and Biobehavioral Reviews*, *35*(1), 91–96. doi.org/10.1016/j.neubiorev.2010.01.010
- Gilbert, P. (1997). The evolution of social attractiveness and its role in shame, humiliation, guilt and therapy. *British Journal of Medical Psychology*, *70*, 113–147.
- Heilman, K. M., Nadeau, S. E., & Beversdorf, D. O. (2003). Creative innovation: possible brain mechanisms. *Neurocase*, *9*(5), 369–379. doi.org/10.1076/neur.9.5.369.16553
- Hellhammer, J., & Schubert, M. (2012). The physiological response to Trier Social Stress Test relates to subjective measures of stress during but not before or after the test. *Psychoneuroendocrinology*, *37*(1), 119–124. doi.org/10.1016/j.psyneuen.2011.05.012
- 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*(7), 1075–1086. doi.org/10.1016/j.psyneuen.2009.02.008
- Hillier, A., Alexander, J. K., & Beversdorf, D. Q. (2006). The effect of auditory stressors on cognitive flexibility. *Neurocase*, *12*(4), 228–231. doi.org/10.1080/13554790600878887
- Hines, E. A., & Brown, G. E. (1932). A standard stimulus for measuring vasomotor reactions: its application in the study of hypertension. *Proceedings of the Staff Meetings of the Mayo Clinic*, *7*, 209–217.
- 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*(1–2), 76–81. doi.org/119004
- Kolotylova, T., Koschke, M., Bär, K.-J., Ebner-Priemer, U., Kleindienst, N., Bohus, M., & Schmahl, C. (2010). Development of the “Mannheim Multicomponent Stress Test” (MMST). *Psychotherapie, Psychosomatik, Medizinische Psychologie*, *60*(2), 64–72. doi.org/10.1055/s-0028-1103297
- Kudielka, B. M., Schommer, N. C., Hellhammer, D. H., & Kirschbaum, C. (2004). Acute HPA axis responses, heart rate, and mood changes to psychosocial stress (TSST) in humans at different times of day. *Psychoneuroendocrinology*, *29*(8), 983–992. doi.org/10.1016/j.psyneuen.2003.08.009
- LeBlanc, V. R. (2009). The effects of acute stress on performance: implications for health professions education. *Academic Medicine: Journal of the Association of American Medical Colleges*, *84*(10 Suppl), S25–33. doi.org/10.1097/ACM.0b013e3181b37b8f
- Lewis, R. S., Nikolova, A., Chang, D. J., & Weekes, N. Y. (2008). Examination stress and components of working memory. *Stress (Amsterdam, Netherlands)*, *11*(2), 108–114. doi.org/10.1080/10253890701535160
- Lim, C. L., Rennie, C., Barry, R. J., Bahramali, H., Lazzaro, I., Manor, B., & Gordon, E. (1997). Decomposing skin conductance into tonic and phasic components. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, *25*(2), 97–109.
- Luethi, M., Meier, B., & Sandi, C. (2008). Stress effects on working memory, explicit memory, and implicit memory for neutral and emotional stimuli in healthy men. *Frontiers in Behavioral Neuroscience*, *2*, 5. doi.org/10.3389/neuro.08.005.2008
- Lupien, S. J., Maheu, F., Tu, M., Fiocco, A., & Schramek, T. E. (2007). The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. *Brain and Cognition*, *65*(3), 209–237. doi.org/10.1016/j.bandc.2007.02.007
- Marko, M., & Brezina, I. (2015). *Posudzovanie miery rozrušenia odpočinku po indukcii psychosociálneho stresu*. Paper presented at Psychologic Assessment, Brno. Brno: Faculty of Social Studies.
- Martindale, C., & Greenough, J. (1973). The differential effect of increased arousal on creative and intellectual performance. *The Journal of Genetic Psychology*, *123*(2d Half), 329–335. doi.org/10.1080/00221325.1973.10532692

- Mednick, S. A. (1962). The associative basis of the creative process. *Psychological Review*, *69*, 220–232.
- Oei, N. Y. L., Everaerd, W. T. a. M., Elzinga, B. M., van Well, S., & Bermond, B. (2006). Psychosocial stress impairs working memory at high loads: an association with cortisol levels and memory retrieval. *Stress (Amsterdam, Netherlands)*, *9*(3), 133–141. doi.org/10.1080/10253890600965773
- Otto, A. R., Raio, C. M., Chiang, A., Phelps, E. A., & Daw, N. D. (2013). Working-memory capacity protects model-based learning from stress. *Proceedings of the National Academy of Sciences*, *110* (52), 20941–20946. doi.org/10.1073/pnas.1312011110
- Pessoa, L. (2008). On the relationship between emotion and cognition. *Nature Reviews. Neuroscience*, *9*(2), 148–158. doi.org/10.1038/nrn2317
- 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*(11), 3218–3227. doi.org/10.1162/jocn_a_00024
- 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*(3), 397–408. doi.org/10.1007/s00221-011-2943-1
- Porcelli, A. J., Cruz, D., Wenberg, K., Patterson, M. D., Biswal, B. B., & Rypma, B. (2008). The effects of acute stress on human prefrontal working memory systems. *Physiology & Behavior*, *95*(3), 282–289. doi.org/10.1016/j.physbeh.2008.04.027
- Reinhardt, T., Schmahl, C., Wüst, S., & Bohus, M. (2012). Salivary cortisol, heart rate, electrodermal activity and subjective stress responses to the Mannheim Multicomponent Stress Test (MMST). *Psychiatry Research*, *198*(1), 106–111. doi.org/10.1016/j.psychres.2011.12.009
- Renner, K. H., & Beversdorf, D. Q. (2010). Effects of naturalistic stressors on cognitive flexibility and working memory task performance. *Neurocase*, *16*(4), 293–300. doi.org/10.1080/13554790903463601
- Roozendaal, B., McReynolds, J. R., & McGaugh, J. L. (2004). The basolateral amygdala interacts with the medial prefrontal cortex in regulating glucocorticoid effects on working memory impairment. *The Journal of Neuroscience*, *24*(6), 1385–1392. doi.org/10.1523/JNEUROSCI.4664-03.2004
- Sarason, I. G., Sarason, B. R., Keefe, D. E., Hayes, B. E., & Shearin, E. N. (1986). Cognitive interference: Situational determinants and traitlike characteristics. *Journal of Personality and Social Psychology*, *51*(1), 215–226. doi.org/10.1037/0022-3514.51.1.215
- Schoofs, D., Preuß, D., & Wolf, O. T. (2008). Psychosocial stress induces working memory impairments in an n-back paradigm. *Psychoneuroendocrinology*, *33*(5), 643–653. doi.org/10.1016/j.psyneuen.2008.02.004
- Schwabe, L., Haddad, L., & Schachinger, H. (2008). HPA axis activation by a socially evaluated cold-pressor test. *Psychoneuroendocrinology*, *33*(6), 890–895. doi.org/10.1016/j.psyneuen.2008.03.001
- Schwabe, L., Joëls, M., Roozendaal, B., Wolf, O. T., & Oitzl, M. S. (2012). Stress effects on memory: an update and integration. *Neuroscience and Biobehavioral Reviews*, *36*(7), 1740–1749. doi.org/10.1016/j.neubiorev.2011.07.002
- Schwabe, L., & Wolf, O. T. (2010). Learning under stress impairs memory formation. *Neurobiology of Learning and Memory*, *93*(2), 183–188. doi.org/10.1016/j.nlm.2009.09.009
- Schwabe, L., & Wolf, O. T. (2013). Stress and multiple memory systems: from “thinking” to “doing.” *Trends in Cognitive Sciences*, *17*(2), 60–68. doi.org/10.1016/j.tics.2012.12.001
- Schwabe, L., Wolf, O. T., & Oitzl, M. S. (2010). Memory formation under stress: quantity and quality. *Neuroscience and Biobehavioral Reviews*, *34*(4), 584–591. doi.org/10.1016/j.neubiorev.2009.11.015
- Spielberger, C. D. (1989). *State-trait anxiety inventory: Bibliography* (2nd ed.). Palo Alto, CA: Consulting Psychologists Press.
- Trestman, R. L., Coccaro, E. F., Bernstein, D., Lawrence, T., Gabriel, S. M., Horvath, T. B., & Siever, L. J. (1991). Cortisol responses to mental arithmetic in acute and remitted depression. *Biological Psychiatry*, *29*(10), 1051–1054.
- Turner, J. R. (1994). *Cardiovascular reactivity and stress*. Boston, MA: Springer. Retrieved from <http://link.springer.com/10.1007/978-1-4757-9579-0>

- Unsworth, N., Heitz, R. P., Schrock, J. C., & Engle, R. W. (2005). An automated version of the operation span task. *Behavior Research Methods*, *37*(3), 498–505.
- Van Dooren, M., de Vries, J. J. G. G.-J., & Janssen, J. H. (2012). Emotional sweating across the body: comparing 16 different skin conductance measurement locations. *Physiology & Behavior*, *106*(2), 298–304. doi.org/10.1016/j.physbeh.2012.01.020
- Vinkers, C. H., Penning, R., Hellhammer, J., Verster, J. C., Klaessens, J. H. G. M., Olivier, B., & Kalkman, C. J. (2013). The effect of stress on core and peripheral body temperature in humans. *Stress (Amsterdam, Netherlands)*, *16*(5), 520–530. doi.org/10.3109/10253890.2013.807243
- Von Dawans, B., Kirschbaum, C., & Heinrichs, M. (2011). The Trier Social Stress Test for Groups (TSST-G): A new research tool for controlled simultaneous social stress exposure in a group format. *Psychoneuroendocrinology*, *36*(4), 514–522. doi.org/10.1016/j.psyneuen.2010.08.004
- Wiemers, U. S., Schoofs, D., & Wolf, O. T. (2013). A friendly version of the trier social stress test does not activate the HPA axis in healthy men and women. *Stress (Amsterdam, Netherlands)*, *16*(2), 254–260. doi.org/10.3109/10253890.2012.714427