



Original Articles

Sympathetic arousal, but not disturbed executive functioning, mediates the impairment of cognitive flexibility under stress

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ARTICLE INFO

Keywords:

Semantic memory
Working memory
Stress
Noradrenaline
Norepinephrine
Dopamine

ABSTRACT

Cognitive flexibility emerges from an interplay of multiple cognitive systems, of which lexical-semantic and executive are thought to be the most important. Yet this has not been addressed by previous studies demonstrating that such forms of flexible thought deteriorate under stress. Motivated by these shortcomings, the present study evaluated several candidate mechanisms implied to mediate the impairing effects of stress on flexible thinking. Fifty-seven healthy adults were randomly assigned to psychosocial stress or control condition while assessed for performance on cognitive flexibility, working memory capacity, semantic fluency, and self-reported cognitive interference. Stress response was indicated by changes in skin conductance, heart rate, and state anxiety. Our analyses showed that acute stress impaired cognitive flexibility via a concomitant increase in sympathetic arousal, while this mediator was positively associated with semantic fluency. Stress also decreased working memory capacity, which was partially mediated by elevated cognitive interference, but neither of these two measures were associated with cognitive flexibility or sympathetic arousal. Following these findings, we conclude that acute stress impairs cognitive flexibility via sympathetic arousal that modulates lexical-semantic and associative processes. In particular, the results indicate that stress-level of sympathetic activation may restrict the accessibility and integration of remote associates and bias the response competition towards prepotent and dominant ideas. Importantly, our results indicate that stress-induced impairments of cognitive flexibility and executive functions are mediated by distinct neurocognitive mechanisms.

1. Introduction

Stress is a complex adaptive response that affects multiple brain areas responsible for cognitive functioning and modulates distinct cognitive systems, such as attention, memory, and problem solving (Arnsten, 2015; Byron, Khazanchi, & Nazarian, 2010; Hermans, Henckens, Joëls, & Fernández, 2014; Schwabe, 2017; Shields, Sazma, & Yonelinas, 2016). A handful of studies showed that one of the functions that seems to be overly sensitive to stress is cognitive flexibility (Alexander, Hillier, Smith, Tivarus, & Beversdorf, 2007; Hillier, Alexander, & Beversdorf, 2006; Martindale & Greenough, 1973). Drawing upon these reports here we refer to cognitive flexibility as the flexibility to access and combine remote elements in lexical-semantic and associative networks in insightful problem solving, which has been most widely assessed by the Remote Associates Test (RAT; Mednick, 1962). In contrast to more constrained attentional set-shifting and multitasking paradigms so defined cognitive flexibility involves

changing how we think about a problem (i.e., conceptual shifts and idea exploration) to overcome mental inertia formed by habitual structure of thought (see Ionescu, 2012 for a detailed discussion). In our study, the term “cognitive flexibility” thus refers to this form of lexical-semantic and associative flexibility, whereas “executive switching” is used to denote an executive function related to the attentional control component of the working memory system (see Engle & Kane, 2004).

Numerous findings have implied that cognitive flexibility depends on processes of activation, retrieval, and integration of distant memory representations (Abraham, 2014; Davelaar, 2015; Smith, Huber, & Vul, 2013) as well as the connectivity of lexical-semantic and associative networks in which such representations are stored (Kenett, Anaki, & Faust, 2014; Marupaka, Iyer, & Minai, 2012; Schilling, 2005). Research suggests that these processes are sensitive to acute stress. For instance, it has been shown that acute stress impairs memory retrieval (Schwabe, Joëls, Roozendaal, Wolf, & Oitzl, 2012; Schwabe & Wolf, 2013). In particular, acute stress decreases access to remote memory

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representations while biasing retrieval towards close associations (Harkins, 2006; Storbeck & Clore, 2008). This indicates that the impairment of cognitive flexibility under stress may result from modulations of lexical-semantic processes and networks.

On the other hand, flexible thought heavily relies on executive attentional functions of the working memory system (Beatty, Benedek, Barry Kaufman, & Silvia, 2015; Benedek, Jauk, Sommer, Arendasy, & Neubauer, 2014; Metuki, Sela, & Lavidor, 2012). Specifically, cognitive flexibility requires inhibition of prepotent associations (Gupta, Jang, Mednick, & Huber, 2012), attentional shifts to alternative retrieval candidates (Katz & Pestell, 1989; Radel, Davranche, Fournier, & Dietrich, 2015; White & Shah, 2006), and maintenance of relevant information in working memory (Chein & Weisberg, 2014; Lee, Huggins, & Theriault, 2014). Ample evidence also indicates that acute stress modulates executive functions. For instance, it has been shown that psychosocial stress decreases performance on working memory tasks (e.g., digit span task, Elzinga & Roelofs, 2005; reading span task, Luethi, Meier, & Sandi, 2009; operation span task, Schoofs, Wolf, & Smeets, 2009; and n-back tasks, Schoofs, Preuss, & Wolf, 2008), impairs switching of attention (Elling et al., 2012), and overall “top-down” attentional control (Shackman, Maxwell, McMenamin, Greischar, & Davidson, 2011; Starcke, Wiesen, Trotzke, & Brand, 2016; Sängler, Bechtold, Schoofs, Blaszkewicz, & Wascher, 2014). This suggests that such stress-induced deterioration of executive functioning may play a major role in the impairment of cognitive flexibility under stress.

The motivation for our study was to better understand the mechanisms which underlie the impairing effects of acute stress on cognitive flexibility. To this aim we assessed the effects of acute stress on cognitive flexibility, working memory capacity, semantic fluency, and self-reported cognitive interference in the same individuals. While working memory capacity and interference control inherently employ the functionality of executive attention (Engle & Kane, 2004; McCabe, Roediger, Mcdaniel, Balota, & Hambrick, 2010), semantic fluency is mainly related to lexical-semantic access, search and retrieval, and the involvement of executive functioning in semantic fluency is relatively minor (Henry & Crawford, 2004; Shao, Janse, Visser, & Meyer, 2014; Whiteside et al., 2015). With this selection of tasks, two alternatives could be tested. First, cognitive flexibility under stress may be impaired due to downregulation of executive functioning: i.e., stress dysregulates optimal cognitive control over information maintenance and retrieval that is required for flexible thinking. In such a case we would expect that working memory capacity and cognitive interference mediates the effect of stress on cognitive flexibility. Second, stress-induced potentiation of closely related associates could hinder access to remote concepts required for flexible thinking: i.e., stress restricts the “range” of accessible information within lexical-semantic and associative networks. In this case, we would expect that acute stress would decrease cognitive flexibility but would have no or even a facilitating effect on semantic fluency, for which typical and strongly associated category instances may be utilized. This prediction is also of interest given that in absence of stress semantic fluency and cognitive flexibility seem to be positively associated (Benedek, Könen, & Neubauer, 2012).

Importantly, the effect of stress on cognitive flexibility was observed early after the onset of stressors in previous studies (Alexander et al., 2007; Hillier et al., 2006), indicating that a rapid stress mechanism is involved in the impairment. We therefore used a modified psychosocial stress paradigm, in which the cognitive assessment was superimposed on the stressors early after their introduction and was carried out concurrently during their presence (Marko, 2016). This setup was advantageous because of two reasons. First, the presence of stressors imposed an ongoing threat and continuous distraction during the cognitive performance (this important feature is absent in paradigms in which cognitive tests are administered after the stressors have terminated). Second, since the cognitive tasks were completed before peak corticoadrenal activation (Droste et al., 2008; Hermans et al., 2014), this method enabled us to focus preferentially on the early neural rather

than late hormonal stress effects. The magnitude of these rapid effects was estimated using the physiological measures of the sympathetic autonomic nervous system activation (skin conductance level and heart rate). We expected that the increase in sympathetic arousal would be associated with decreased flexibility and included sympathetic arousal in serial mediation models alongside the behavioral measures in order to disentangle the processes contributing to the impairment of cognitive flexibility under stress.

2. Method

2.1. Participants

Sixty healthy adults were sampled from a larger pool of volunteers to participate in the study following an a priori power analysis (5% Type I error rate, 20% Type II error rate, and effect size from Byron et al., 2010, were used for the calculation). Due to technical problems, data of three participants were excluded. The final group thus consisted of 57 participants (39 females and 18 males, mean age = 19.9 ± 1.3 years). All participants met the following inclusion criteria: age between 18 and 25 years, Slovak primary language, absence of a mental disorder, cardiovascular disease or chronic health problems, and no current pharmacological treatment (except contraceptives). The participants were asked to abstain from alcohol and intense physical exercise 24 h before testing and from caffeine 12 h before testing. A written informed consent was obtained from all participants. The protocol was approved by the appropriate ethics committee. Research has been conducted in accordance with the Declaration of Helsinki.

The participants were randomly divided into a group undergoing stress procedure (Stress, $N = 28$) and a control group ($N = 29$). The stress and the control group did not differ in the proportion of gender, $\chi^2(1, N = 57) = 0.230, p = .631$, mean age, $t(55) = -0.190, p = .850$, mean BMI, $t(55) = 0.209, p = .836$, or session time, $t(55) = -0.058, p = .954$. The groups were also equivalent in terms of trait anxiety level, $\chi^2(2, N = 57) = 0.026, p = .987$, initial state anxiety, $t(55) = 0.933, p = .355$, positive affect, $t(55) = -0.130, p = .897$, distress $t(48.5) = 1.630, p = .107$, self-confidence $t(55) = -0.522, p = .604$, and general self-efficacy, $t(55) = 0.324, p = .747$, prior to stress induction and cognitive assessment (see Section 2.6 for details of the used self-report methods). Finally, the control and the stress group did not differ in the frequency of the individual task sequences, $\chi^2(5, N = 57) = 0.230, p = .631$, nor in the administration order of individual cognitive tasks, $\chi^2(2, N = 57) < 0.846, p > .655$ (see Tables S1 and S2 in supplementary online material for the exact frequencies and further details).

2.2. Design and procedure

The experiment included two randomly assigned between-subjects factors, Stress (as a main factor of interest defining the testing conditions) and Task sequence (a control factor defining the assessment order of cognitive tests; 6 levels), and one fixed within-subjects factor, Time block (this factor was used to define specific time windows for repeated physiological measurements). Administration order of individual cognitive tests (administered as first, second, or third; 3 levels) was derived from Task sequence as a separate factor.

Experimental sessions were run between 9:00 and 17:00 and followed the procedure depicted in Fig. 1. Each session started with a brief interview followed by assessment of affective state and general self-efficacy (from -30 to -12 min with respect to stressor onset; see Section 2.5). Subsequent experimental procedures included 5 time blocks: baseline, 3 cognitive tests (administered in a random order), and recovery. Baseline (from -12 to -2 min) and recovery (from $+35$ to $+45$ min) were fixed to 10 min, during which participants were sitting alone in a quiet room and were instructed to relax. The cognitive testing lasted up to 30 min (from $+3$ to $+30$ min). Before cognitive

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