Stress and Cognition in Humans

Current Findings and Open Questions in Experimental Psychology

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Stress is a ubiquitous phenomenon. Stress or stress responses occur as a concert of adaptive psychological and physiological reactions to expected or perceived environmental challenges. In the broadest sense, stress can be conceptualized as any psychological or bodily response that is elicited by any internal or external stimulus that challenges the individual's resources for adaptation. In a more specific sense and in order to distinguish these reactions from any adaptive reaction to environmental stimuli, stress or the stress response comprises the activation of a number of neurophysiological systems that help an organism to cope with the actual or perceived threat or loss of homeostasis. Two main physiological systems have been identified as crucial for the stress response and relevant for coping: the activation of the hypothalamus-pituitary-adrenal axis (HPA axis) and the autonomic nervous system (ANS). Both systems exert their action by secretion of hormones (glucocorticoids and catecholamines) at different time scales and are regulated by complex nervous and endocrine regulatory feedback at the central and peripheral level (Charmandari, Tsigos, & Chrousos, 2005). Endocrine action involves glucocorticoid-binding receptors of different affinity (mineralocorticoid and glucocorticoid receptors) in the central nervous system (CNS; de Kloet, 2014), whereas catecholamines exert CNS effects by binding to adrenal receptors located in the brain stem and in the periphery (Chrousos, 2009).

Chronic stress-induced physiological activation has been implicated in the development of somatic illness, such as coronary heart disease, hypertension and high blood pressure, diabetes, infectious diseases, and mental disorders, such as affective and anxiety disorders (McEwen, 2000). Aside from studies focusing on the long-term sequelae of elevated stress and stress-related physiological activity, another line of experimental research focuses on the short-term psychological effects of stress and the acute impact of physiological stress reactions (e.g., Frings & Domes, 2019).

Stress-induced CNS effects of glucocorticoids and adrenergic activity have been assumed to be involved in the mediation of stress-induced modulation of cognitive processes (Sandi, 2013). Following this approach, basic cognitive functions have been extensively investigated during the past decades in animals and humans. Most studies on humans investigating the effects of acute stress on cognition using laboratory stress and pharmacological approaches have focused on basic memory functions such as memory consolidation (Roozendaal & McGaugh, 2011) and retrieval (Gagnon & Wagner, 2016).

Yet, over the last two decades, other cognitive domains have gained increasing interest regarding the potential modulation by acute stress. Recent meta-analyses quantitatively reviewing experimental research on the acute effects of stress on human cognition demonstrate an accumulating effort to understand how stress modulates human cognitive functioning beyond the effects on memory (Shields, Sazma, McCullough, & Yonelinas, 2017; Shields, Sazma, & Yonelinas, 2016). The majority of studies (in this special issue and other literature) used laboratory stress induction methods, most commonly the Trier social stress test (TSST) which was developed almost 30 years ago (Kirschbaum, Pirke, & Hellhammer, 1993). The TSST has been shown to induce robust and reliable subjective and physiological stress responses, including activation of the HPA axis, which seems to be mainly driven by the experienced social evaluative threat and uncontrollability (Dickerson & Kemeny, 2004). Although the TSST has become the gold standard for stress induction in the laboratory, other stress induction methods are conceivable, varying, for example, the social dimension of stress (e.g., the extent of social evaluation) or the modality of aversiveness (e.g., physical pain vs. social interaction). One example is the cold pressor test, for which a social variant has been introduced and used over the last decade (Schwabe & Schächinger, 2018). It is possible that the extent to which the core feature of the stressor and the cognitive function correspond (e.g., social vs. nonsocial content) explains parts of the effects that acute stress exerts. Thus, studies systematically varying the correspondence of stressor and affected cognitive function could be a promising approach to shed light on the remaining "dark variance" of stress-induced cognitive effects.

Recent Findings in This Special Issue

In this special issue, we have assembled studies from different cognitive domains that follow a common experimental approach to investigate the effects of acute stress on cognition either by inducing psychological stress with standardized protocols or by manipulating stressassociated physiological systems. While some studies focus on quite basal cognitive functions like motor responses or memory retrieval, others investigate more complex behavior like divergent thinking or prosocial behavior.

Finke and Schächinger analyzed the impact of a pharmacologically induced stress response on motor performance. They yielded evidence for a nonlinear relationship between stress-induced central sympathetic nervous system activation and motor performance - a result corroborating the long-standing notion that the way performance is modulated by arousal may crucially depend on task complexity (Finke & Schächinger, 2020). Degroote and Wirtz (2020) investigated the relationship between attention and acute stress - a relationship that is inconsistent in the literature at best - and observed one potential modulator for this complex relationship, namely, anxiety (Degroote & Wirtz, 2020). Hartogsveld, van Ruitenbeek, Quaedflieg, and Smeets (2020) analyzed the modulating effects of acute stress on instrumental learning, suggesting that the instrumental learning and outcome devaluation procedures are boundary conditions to stress-induced shifts in instrumental responding (Hartogsveld et al., 2020).

As mentioned above, the experimental approach to the effects of acute stress is heavily founded in memory research, and accordingly, this Special Issue also includes memory-related studies. Lüers and colleagues focused on the inconsistent empirical evidence for moderating effects of cortisol on working memory. They observed contrary effects of cortisol levels on working memory performance for women and men in older participants (Lüers, Kaszynska, & Pruessner, 2020). Pastötter and colleagues found that acute stress does not modulate the forward effect of testing; that is, stress does not impact the enhanced memory for information learned after testing previous material (Pastötter, von Dawans, Domes, & Frings, 2020).

Finally, some studies looked at more complex human behavior and how stress possibly modulates it. Von Dawans, Zimmer, Spenthof, and Domes (2020) showed that psychosocial stress increases detection sensitivity for positive facial expressions, a pattern that is consistent with the tendency to seek social support for coping with stress (von Dawans et al., 2020). Passarelli and Buchanan analyzed the possible effects of acute stress on the relation between social closeness and prosocial behavior; however, prosocial behavior was not increased under stress (Passarelli & Buchanan, 2020). Finally, the study by Meier et al. (2020) looked at the "opposite" of stress, namely, how a state of relaxation affects cognition. By applying a standardized vagus nerve massage, they could increase performance in divergent thinking (Meier et al., 2020).

Open Questions

Research on the cognitive effects of acute stress in humans has begun to extend the scope toward complex, higherorder cognitive functions. In addition to the large body of research on basic memory functions, this extension encompasses decision-making in different contexts, social cognitive functioning, and other complex functions, such as prosocial behavior or social perception (e.g., Passarelli & Buchanan, 2020; von Dawans et al., 2020). Additional studies on these so far under-researched cognitive domains may not only broaden our fundamental understanding of the acute effects of stress but might also help to estimate the cognitive effects of acute stress in everyday life and the impact chronic stress might have on cognitive functioning in health and mental disease (Lupien, McEwen, Gunnar, & Heim, 2009).

Aside from exploring the effects of acute stress in different cognitive domains, the role of specific psychophysiological mechanisms remains largely unexplored. Correlational approaches, often conducted as secondary analyses to exploit the within-group variance observed in the stress condition, provide a valuable starting point. However, rigorous experimental studies are needed to investigate the cause and effect of specific physiological systems or mediators. One possible attempt is the simulation of stress-induced physiological responses by using pharmacological challenges or using specific pharmacological agonists or antagonists, for example, following dose-response approaches to mimic different doses of stress exposure (e.g., Schilling et al., 2013). In addition, blocking parts of the stress-related physiological cascade while performing an acute stressor could also be a

promising avenue exploring the contribution of specific stress-related factors (such as cortisol or the sympatheticadrenal-medullary system) to stress-related cognitive effects. Combinations of the TSST with dexamethasone or propranolol have been recently introduced (Andrews, D'Aguiar, & Pruessner, 2012; Andrews & Pruessner, 2013).

Many studies suggest that the effects might be modulated by more or less stable individual factors such as sex and personality traits, as also demonstrated in some of the current papers in this special issue (Degroote & Wirtz, 2020; Lüers et al., 2020). Among these individual differences, the individual's sex is of special relevance as sex-related physiological differences, for example, endocrine profile associated with the menstrual cycle, are well known to modulate physiological stressresponsiveness (Kudielka & Kirschbaum, 2005). Thus, systematic investigation of these modulating factors or at least thorough experimental control seems rational.

Taken together, the effects of acute stress on cognition in the studied literature are heavily biased toward memory research, and while memory (and its modulations by stress) is a very important research topic, there is no reason to limit the research on stress effects to memory. In addition, some studies follow a correlational approach to investigate stress effects (and sometimes, this is the only choice). Here, we emphasize that researchers should more strongly expand the research on stress effects beyond the memory domain and, when possible, adhere to an experimental approach.

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Conflict of Interest

The authors declare no conflict of interest.

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