SHORT COMMUNICATION

Influence of psychosocial stress on cognitive flexibility

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Stress-induced changes substantially impact multiple brain areas responsible for cognitive functioning. The present paper addresses recent findings indicating that this effect may be specifically deteriorative for cognitive flexibility and investigates its underlying mechanism.

Cognitive flexibility represents the ability to inhibit strong preferences or dominant tendencies in thinking, which enables the exploration of alternative solution paths (Alexander *et al* 2007). It is utilized mainly in complex problems whose ill-constrained nature requires broad conceptual search. Due to breaking mental inertia formed by habitual structure of thought, cognitive flexibility is often associated with creative innovation and insight. Recent research has indicated that productive attributes of thought may result from a flexible combination of conceptual information stored in semantic networks. Implementing network theory approach, neurodynamical simulations (Marupaka *et al* 2012) and human studies (Kenett *et al* 2014) indicate that important properties and features of thinking (e.g. cognitive flexibility, insight, psychopathology) plausibly emerge from the structure in which the mind organizes knowledge and conceptual information. Although flexible thought may be grounded in the connectivity of semantic networks, extensive evidence indicates that it is also profoundly modulated by acute stress. Behavioral research demonstrated that induction of stress impairs flexible thought (Martindale & Greenough 1973). Apart from the effect on perceptual and processes (Budáč *et al* 2014), elevated arousal and increase informational selectivity (Easterbrook 1959) which may impair the access to distant conceptual information. Because arousal is elevated under stress, stressors are expected to deteriorate cognitive flexibility. More recent studies investigating the mechanisms of stress-induced impairments in cognitive flexibility attributed the critical role to locus coeruleus – noradrenergic system (Alexander *et al* 2007). The locus coeruleus is a neuromodulatory nucleus involved in physiological responses to stress, which supplies noradrenaline throughout the neuraxis and also plays a crucial role in initiation and regulation of physiological arousal (Samuels & Szabadi 2008). Noradrenergic activity was documented to alter broad scope of behavioral and cognitive performance (Aston-Jones & Cohen 2005). The involvement of central noradrenergic system in cognitive flexibility performance was supported by administration of propranolol (Alexander *et al* 2007), which reversed the effect of acute stress on cognitive flexibility. Similarly, stimulation of vagus nerve, which is thought to activate the noradrenergic system, decreased cognitive flexibility (Ghacibeh *et al* 2006). Several studies have addressed the modulatory effect of noradreline directly. At the information processing level, catecholamines appear to affect the ability to detect a signal when it is embedded in noise. Detection-enhancing effects of catecholamines were argued to be a consequence of improved rejection of internal noise within the brain (Hasselmo *et al* 2007). These studies suggested that high gain may eliminate the representation of weaker signals in favor of the dominant, fixating stereotyped responses which can lead to a decreased variability in responding of the system. In line with similar previous research documenting a decline of flexible thought under experimentally induced states of high arousal, anxiety, and stress, our research also indicated that the psychosocial stressors substantially impair cognitive flexibility (Marko 2016). We hypothesized that the underlying mechanism responsible for this impairment rests in the elevated sympathetic activation as a common feature intersecting the abovementioned psychological conditions (i.e. arousal, stress, and anxiety). The proposed mediating effect of sympathetic activation was subsequently supported by more specific analyses (Marko 2016). This evidence is consistent with the proposed explanation related to locus coeruleus – noradrenergic system (Alexander *et al* 2007) which both regulates autonomic functions through projections to the spinal cord and autonomic nuclei (Samuels & Szabadiz 2008), and also inhibit cortical noise (Hasselmo *et al* 2007), an effect that presumably narrows the space of available alternative ideas, biasing and limiting problem solving towards proponent and dominant response. An alternative explanation is however also possible: cognitive flexibility may be also impaired due to decreased ability to detect weaker (alternative) signals under stress. Several brain imaging studies revealed that preparation for flexible thinking is associated with activation of anterior cingulate cortex (Kounios *et al* 2006). It was hypothesized that higher activity of anterior cingulate cortex enables the detection of weaker neuronal activation that represent subdominant ideas and switching the attention to them (Kounios *et al* 2006). The activity of anterior cingulate cortex was documented to increase during relaxed states and positive mood (Subramaniam *et al* 2009) and decrease under acute stress (Feng *et al* 2011). Following these findings, it could be expected that distress may decrease the ability to detect weaker (background) neural activity patterns and so impair the accessibility of weakly connected or remote concepts. This effect may plausibly manifest in an impairment cognitive flexibility. Thirdly, it has been documented that corticosteroid system affects frontal-lobe functions, which are responsible for high-order cognition, and interacts with sympathetic system in a complex way. However, the role of the hypothalamic–pituitary–adrenal axis in modulation of cognitive flexibility is not well-understood. To our knowledge, there is no systematic study that can unequivocally disentangle between the abovementioned neurophysiological mechanisms. Future research is thus required to take account for the corticosteroid system and other physiologically related markers engaged in stress response and the activity of locus coeruleus – noradrenergic system specifically (e.g. pupil diameter). Furthermore, simultaneous pharmacological manipulation and brain activity recording might provide the necessary apparatus for deeper understanding of the neurocognitive and neuroendocrine mechanisms involved in cognitive flexibility modulation.

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