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Comment

The quartet theory: Implications for autism spectrum disorder

Comment on “The quartet theory of human emotions: An integrative and neurofunctional model” by S. Koelsch et al.

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Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder characterized by social and communication deficits as well as restricted and repetitive behaviors [1]. Specific deficits include failure to initiate reciprocal social interactions, verbal and non-verbal communication difficulties, decreased sensitivity to social and emotional cues, and limited perspective-taking abilities. Social withdrawal, avoidance or indifference to affection or physical contact, lack of eye contact, and decreased joint attention and facial responsiveness are also common [2]. In addition to these core features, there is a growing body of literature that describes problematic patterns of emotional reactivity (increased negative and decreased positive emotions) and emotion regulation (increased use of maladaptive and decreased use of adaptive emotion regulation strategies) [3–5]. The present comment seeks to link difficulties in socio-emotional domains to the *Quartet Theory of Human Emotions* by mapping characteristic ASD social deficits and emotion dysregulation onto two of the affect systems described in this theory: the hippocampal and orbitofrontal-centered systems.

According to the *Quartet Theory*, the hippocampal system (consisting of hippocampus, parahippocampus, entorhinal cortex and temporal pole) is involved in the generation of tender positive and joyful “attachment-related affects” [6, p. 16]. By contrast, the orbitofrontal-centered affect system is thought to perform “a fast and automatic (non-conscious) cognitive appraisal of both external and internal information” [6, p. 21] and is “involved in the control of emotional behaviour” [6, p. 24]. The role of the orbitofrontal-centered system is to evaluate sensory cues “with reference to internalized knowledge” [6, p. 21]. It thus interacts with the hippocampal system by integrating current sensory information with memory content provided by the hippocampal system. In healthy individuals, a successful integration of hippocampal memory is essential for top-down regulatory influences by the orbitofrontal cortex (OFC) on lower level sensory and other processing streams, such as the amygdala, which is a target area for self-regulation in socio-emotional contexts [7]. Taking the *Quartet Theory* as a model to explain socio-emotional difficulties in ASD, we hypothesize a dysfunctional interaction between these two affect systems. In coordination with the hippocampal system, the OFC “performs a (...) non-conscious imbue ment of information with emotional valance” [6, p. 22] and

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may in the case of failure be linked to problematic patterns of emotional reactivity and regulation, such as those observed in ASD. From this perspective, increased maladaptive emotion regulation – such as remaining focused on the stressor, meltdowns, or social withdrawal – may be seen as an indicator of poor performance of the orbitofrontal system in evaluating relevant stimuli, resulting in diminished regulation of socio-emotional behavior.

Pathophysiological evidence corroborates dysfunctional interactions of the hippocampal and orbitofrontal affect systems. Studies using rodent models of ASD have shown that impaired social behavior is associated with alterations of glutamate receptor concentrations in hippocampal and frontal cortices induced by disruptions of candidate ASD genes [e.g. 8]. Interestingly, morphometric studies in ASD draw an inconclusive picture on hippocampal volume in this disorder [9]. However, the frontal cortex, including the orbitofrontal region, has been shown to be a main target area of early brain overgrowth in ASD [10]. In addition, malformations in the frontal cortex through neuroinflammatory responses or migration defects seem to persist regardless of developmental influences [11]. Based on frontal lobe abnormality, Courchesne and Pierce [11] put forward the underconnectivity hypothesis in ASD, which posits impaired large-scale connections of the frontal lobe, and implicates reduced integration of information and reduced control over lower-level systems. This hypothesis is consistent with the approach taken by Shalom [12], who suggested a deficient integration of information through lack of cognitive control as a prominent feature of ASD. This proposal was based upon studies showing deficits in recognizing blurred faces, revealing insufficient cognitive regulation from frontal regions during integration of perceptual information [e.g. 13]. Structurally, the OFC and hippocampal systems are connected via the uncinate fasciculus, a pathway involved in memory and higher-order socio-emotional processing, which connects the OFC with the anterior temporal lobe and the rhinal cortex, which in turn is connected to the hippocampus [14]. Studies using diffusion tensor imaging (DTI) to characterize microstructure in white matter tracts have repeatedly reported disruptions of the uncinate fasciculus in ASD [e.g. 15] supporting the long-distance underconnectivity hypothesis [16] and in particular the potential dysfunctional interaction of the hippocampal and orbitofrontal systems.

A recent study investigating the neural underpinnings of emotion regulation deficits in ASD has reported reduced prefrontal-amygdalar connectivity during down-regulation of emotions using cognitive reappraisal [17], a regulation strategy that aims to modify one's emotional response by reinterpreting the emotional significance of the stimulus. The authors concluded that impaired fronto-limbic top-down regulation might be an underlying mechanism of emotion dysregulation in ASD. As described above, the OFC integrates information, most likely provided by the hippocampal system. Therefore, a dysfunctional interaction of the hippocampal and orbitofrontal-centered affect systems might indirectly influence downstream regulation of the OFC to the amygdala and thereby hamper effective emotion regulation in ASD.

We believe that the *Quartet Theory* may be useful in generating hypotheses regarding the neural bases of problematic patterns of emotion reactivity and regulation in ASD. From the perspective of the *Quartet Theory*, it might be interesting to test how other affect systems (e.g. brainstem, diencephalon) or coordinate systems (e.g. the amygdala) exert protective influence on the hippocampal system during stressful situations in ASD. The extent to which accessibility and vividness of memory retrieval and integration helps individuals with ASD to regulate their emotions using cognitive reappraisal might also bear examination in future research.

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