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Does Pedophile Obsessive-Compulsive Disorder (POCD) Promote Neuroinflammatory Pathways while Alleviating Neurogenesis?

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About the Study

According to a growing body of scientific research reveal that surgical trauma can stimulate the innate immune system, which in turn activates peripheral immune cells which resulting in Blood Brain Barrier (BBB) disruption turn to trigger neuronal and glial cells activation, causing the release of inflammatory mediators leading to POCD pathogenesis and impair neurogenesis. The pathophysiology behind the processes of POCD is increasingly focusing on the roles of oxidative stress and neuroinflammation, which are both prominently associated with anaesthesia and surgery. Focusing on discovering novel treatment targets based on their capacity to biological signalling network pathways in POCD, surgery-induced inflammation creates long-term repercussions that cause cognitive deficits. However, we are far away from a common hypothesis how and why peripheral innate immune mechanisms affect brain immunity, leading to neuroinflammatory and cognitive impairment in POCD.

Bhuiyan and his colleagues employed bioinformatics and systems biology approaches to investigate the potential hub genes that are differentially expressed and associated with mechanisms involving inflammatory, neurogenesis, and modulation of synaptic transmission and also repurposing drug inhibitors in POCD [1]. According to our network biology analysis, we postulate that POCD may directly affect neuronal biological responses by altering intraneuronal pathways such as neurogenesis, neuron differentiation, postsynaptic transmission, catabolic processes of neuroimmune activity, and cellular protein modification of neuronal cells. The uncovering of the hub genes (AKT1, CHUK, PRKCZ, TYMS, RBBP4, BPTF and AKT2) implicated in important neuroimmune and neuroinflammatory signaling pathways in the pathogenesis of POCD was made possible by the use of different omics approaches integrating protein-protein interaction. This is the very first instance in which bioinformatics network-based integrative approaches have been implemented to elucidate numerous complex signaling pathways, such as the CD40L signaling pathway, AKT phosphorylation of cytosolic targets, TNFR2 signaling pathway, EGF receptor signaling pathway, PDGF signaling pathway, nicotinic acetylcholine receptor signaling pathway, Alzheimer disease-amyloid secretase pathway, FGF signaling pathway, chaperones that modulate interferon signaling pathway, IL17 signaling pathway, and AGE/RAGE pathway that is suggested to involve in the pathogenesis of POCD. To support our idea, it is imperative to conduct wet lab research and explore novel therapeutic interventions.

By drastically reducing the production of Brain-Derived Neurotrophic Factor (BDNF), a crucial protein that encourages neuronal survival and development, neuroinflammation, a frequent response to surgical trauma, may impede neurogenesis [2]. By affecting the function of neurotransmitters and receptors that regulate neuronal proliferation and maturation, where anesthesia, which is frequently used during surgery, can also have an impact on neurogenesis. The hippocampus, which is important in memory formation and consolidation, as well as synaptic plasticity and learning in different regions of the brain, may be impaired by POCD [3,4]. Genes involved in synaptic plasticity, such as BDNF and glutamate receptor subunits, may have altered epigenetic states as a result of POCD. The term "epigenetics" describes alterations to DNA or histone proteins that modify gene expression without altering DNA sequence. Environmental triggers like stress or anesthesia can cause epigenetic alterations, which can last for a very long time. By controlling the transcription of genes that encode synaptic proteins or by altering the accessibility of chromatin to transcription factors, epigenetic alterations can have an impact on synaptic plasticity. As a result, the balance between neuroinflammation and neurogenesis in the brain could potentially have an impact on POCD. POCD may be prevented or treated using therapies that can modify these processes, such as anti-inflammatory medications, preconditioning, or cognitive training [5]. Therefore, we speculate that it will open up a new window for research into the pathophysiology of POCD, with a particular emphasis on how neuroinflammation is related to impair neurogenesis.

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