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Oxidative stress and neuroinflammation in schizophrenia detected by novel neuroimaging approaches

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Schizophrenia (SZ) is a common and severe psychiatric disorder characterized by abnormal cognition and perception. Despite its public health impact and a century of biological research, the pathophysiology of SZ remains poorly understood. Recently accumulated evidence suggests that an immuno-oxidative pathway including oxidative stress, NMDAR hypofunction and neuroinflammation may contribute to disruptions in brain circuits in SZ. The redox pair of Nicotinamide adenine dinucleotide (NAD⁺) and its reduced form NADH have long been implicated in biological activities such as cellular energy metabolism, calcium homeostasis, gene expression and immunological functions. Despite the crucial roles of NAD⁺ and NADH in cellular metabolism and physiology, its non-invasive in vivo detection is extremely challenging. Recently we demonstrate the feasibility of 31P MRS-based NAD quantification at 4 T MRI-scanner and apply this novel method in patients with SZ. We found a substantial and significant reduction in the redox ratio (i.e., NAD⁺/NADH) in the chronic and first-episode SZ patients. Intracellular redox ratio is influenced by multiple cellular signaling events and may constitute a metabolic integrator for local metabolic status within cells. Therefore, our work provides new insights into the pathophysiology of SZ, as well as a biomarker for tracking the impact of treatment interventions. In addition, the identification of pharmacological compounds acting on brain redox status as an innovative therapeutic approach for first-episode psychosis treatment and neuroimaging biomarker measurements such as glutathione (antioxidant) and glutamate/glutamine (index of NMDAR hypofunction) will be discussed.

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