Mitochondria, Metabolism, and Redox Mechanisms in Psychiatric Disorders

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Article

Abstract

Significance: Our current knowledge of the pathophysiology and molecular mechanisms causing psychiatric disorders is modest, but genetic susceptibility and environmental factors are central to the etiology of these conditions. Autism, schizophrenia, bipolar disorder and major depressive disorder show genetic gene risk overlap and share symptoms and metabolic comorbidities. The identification of such common features may provide insights into the development of these disorders.

Recent Advances: Multiple pieces of evidence suggest that brain energy metabolism, mitochondrial functions and redox balance are impaired to various degrees in psychiatric disorders. Since mitochondrial metabolism and redox signaling can integrate genetic and environmental environmental factors affecting the brain, it is possible that they are implicated in the etiology and progression of psychiatric disorders.

Critical Issue: Evidence for direct links between cellular mitochondrial dysfunction and disease features are missing.

Future Directions: A better understanding of the mitochondrial biology and its intracellular connections to the nuclear genome, the endoplasmic reticulum and signaling pathways, as well as its role in intercellular communication in the organism, is still needed. This review focuses on the findings that implicate mitochondrial dysfunction, the resultant metabolic changes and oxidative stress as important etiological factors in the context of psychiatric disorders. We also propose a model where specific pathophysiologies of psychiatric disorders depend on circuit-specific impairments of mitochondrial dysfunction and redox signaling at specific developmental stages.