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Elevated immune-inflammatory signaling in mood disorders: a new therapeutic target?

McNamara RK[1], Lotrich FE.

Author information

[1]Department of Psychiatry, Division of Bipolar Disorders Research, University of Cincinnati College of Medicine, Cincinnati, OH, USA. robert.mcnamara@uc.edu

Abstract

Converging translational evidence has implicated elevated immune-inflammatory signaling activity in the pathoetiology of mood disorders, including major depressive disorder and bipolar disorder. This is supported in part by cross-sectional evidence for increased levels of proinflammatory eicosanoids, cytokines and acute-phase proteins during mood episodes, and prospective longitudinal evidence for the emergence of mood symptoms in response to chronic immune-inflammatory activation. In addition, mood-stabilizer and atypical antipsychotic medications downregulate initial components of the immune-inflammatory signaling pathway, and adjunctive treatment with anti-inflammatory agents augment the therapeutic efficacy of antidepressant, mood stabilizer and atypical antipsychotic medications. Potential pathogenic mechanisms linked with elevated immune-inflammatory signaling include perturbations in central serotonin neurotransmission and progressive white matter pathology. Both heritable genetic factors and environmental factors including dietary fatty-acid composition may act in concert to sustain elevated immune-inflammatory signaling. Collectively, these data suggest that elevated immune-inflammatory signaling is a mechanism that is relevant to the pathoetiology of mood disorders, and may therefore represent a new therapeutic target for the development of more effective treatments.

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