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Endocannabinoid signaling in the etiology and treatment of major depressive illness.

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Abstract

The purpose of this review is to examine human and preclinical data that are relevant to the following hypotheses. The first hypothesis is that deficient CB1R-mediated signaling results in symptoms that mimic those seen in depression. The second hypothesis is that activation of CB1R-mediated signaling results in behavioral, endocrine and other effects that are similar to those produced by currently used antidepressants. The third hypothesis is that conventional antidepressant therapies act through enhanced CB1R mediated signaling. Together the available data indicate that activators of CB1R signaling, particularly inhibitors of fatty acid amide hydrolase, should be considered for clinical trials for the treatment of depression.

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