

Abstract

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The role of zinc in neurodegenerative inflammatory pathways in depression.

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BACKGROUND: According to new hypothesis, depression is characterized by decreased neurogenesis and enhanced neurodegeneration which, in part, may be caused by inflammatory processes. There is much evidence indicating that depression, age-related changes often associated with impaired brain function and cognitive performances or neurodegenerative processes could be related to dysfunctions affecting the zinc ion availability.

FINDINGS: Clinical studies revealed that depression is accompanied by serum hypozincemia, which can be normalized by successful antidepressant treatment. In patients with major depression, a low zinc serum level was correlated with an increase in the activation of markers of the immune system, suggesting that this effect may result in part from a depression-related alteration in the immune-inflammatory system. Moreover, a preliminary clinical study demonstrated the benefit of zinc supplementation in antidepressant therapy in both treatment non-resistant and resistant patients. In the preclinical study, the antidepressant activity of zinc was observed in the majority of rodent tests and models of depression and revealed a causative role for zinc deficiency in the induction of depressive-like symptoms, the reduction of neurogenesis and neuronal survival or impaired learning and memory ability.

SUMMARY: This paper provides an overview of the clinical and experimental evidence that implicates the role of zinc in the pathophysiology and therapy of depression within the context of the inflammatory and neurodegenerative hypothesis of this disease.

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