DEPRESSION-LIKE SYMPTOMS AND NEURODEGENERATION UNDER ZINC-DEFICIENT CONDITIONS

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INTRODUCTION

It has been repeatedly observed that there is a link between serum zinc level and mood disorders. Patients with major depression exhibit a significantly lower serum zinc levels compared to the control subjects. This may be associated with a new theory of depression assuming hyperactivity of the glutamatergic system. Recent studies have consistently indicated that disturbances of glutamatergic transmission are involved in the processes leading to depression. Zinc is a modulator/potent inhibitor of the glutamatergic NMDA receptor complex. Its deficiency contributes to higher glutamate concentration and hyperexcitation of the hypothalamic-pituitary-adrenal (HPA) axis. Our and other recent results have shown that zinc deficiency induces the development of “pro-depressive” alterations, which suggest that zinc deficiency is a cause of depressive disorder. Preclinical and clinical studies have shown that zinc exhibits antidepressant-like activity. Zinc can enhance the antidepressant effect of drugs when given as an adjunct to ineffective doses of antidepressants. Moreover, the activity of zinc in augmentation therapy was demonstrated in major depression whilst diminished blood zinc levels are concurrent with depression.

AIM OF THE STUDY

The aim of this study was to examine the contribution of zinc to the development of depressive-like behavior in the forced swim test (FST), and to correlate this behavior with serum zinc level, corticosterone concentration and expression of neurotrophins.

RESULTS AND CONCLUSIONS

Zinc-deficient mice showed increased immobility time compared to zinc adequate, which means that zinc deficiency contributes to development of depressive-like behavior.

Administration of escitalopram to zinc deprived mice induces lower response in comparison to adequate diet in FST. This results indicate that zinc deficiency may contribute to development of treatment-resistant depression.

There were no changes in locomotor activity in all groups, control and zinc deficient.

Depressive-like behavior in zinc deficiency is correlated with lower serum zinc level, which is normalized after chronically escitalopram administration.

Zinc deficiency diet caused increase in serum corticosterone concentration, which means hyperactivity of HPA axis under zinc-deficient conditions.

Zinc-deficient animals showed a significant reduction of neurotrophins, which indicates involvement of zinc deficiency in the neurodegenerative processes.

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