EFFECT OF CHRONIC TREATMENT WITH DEXAMETHASONE ON BRAIN DOPAMINE RECEPTORS IN MICE

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Glucocorticoids are expressed in the central nervous system. Radioligand binding studies have shown their presence in the neurons of the limbic system, a structure involved in mood control and subtle regulation of hypothalamic-pituitary-adrenal (HPA) axis. Structures of the limbic system are also rich in dopaminergic innervation. It has been hypothesized that glucocorticoids may be important in causing and perpetuating depression.

Our previous study has demonstrated that dexamethasone decreases the locomotor activity of mice and counteracts the hyperactivity induced by agonists of dopamine receptors.

The aim of the present study was to find the possible mechanism responsible for these behavioral effects of dexamethasone. So we sought to examine the influence of chronic dexamethasone treatment on selective radioligand binding to dopamine D1 ([3H]SCH 23390) and D2 ([3H]spiperone) receptors in the brain of mice.

The male Albino Swiss mice received dexamethasone (4, 8 or 16 mg/kg/day) for 14 days. The striatum and limbic system structures were isolated and the binding procedure was performed 3.5 or 48 h after the last injection.

It was shown that 3.5 h after the last dose of dexamethasone (4 mg/kg/day), specific D2 receptor binding was statistically significantly increased (by 64%) in the limbic system. On the contrary, the tendency to the reduction of specific D2 receptor binding was observed in the striatum. Dexamethasone treatment did not influence the specific binding to D1 receptors in any structure of the brain.

Key words: dexamethasone, brain dopamine receptors, mice