Dexamethasone reduces locomotor stimulation induced by dopamine agonists in mice

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Abstract:
The interaction between glucocorticoids and the dopaminergic system has attracted considerable attention in recent years since this link could be involved in certain psychopathological conditions including depression. Radioligand binding studies have shown the presence of glucocorticoid receptors in 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.
The aim of the present study was to investigate the effect of dexamethasone (DEX) on hyperactivity induced by dopamine agonists (amphetamine, amantadine, quinpirole and bromocriptine) in mice. Male Albino Swiss mice received DEX at a single dose (2, 4 or 8 mg/kg) or for 14 days at the doses of 0.5, 2 or 4 mg/kg/day. After a single or the last injection (in the chronic experiment) of DEX, dopamine agonists were given in the following regimen: D-amphetamine (0.4 mg/kg) and quinpirole (3 mg/kg) – 30 min, amantadine (50 mg/kg) – 60 min and bromocriptine (10 mg/kg) 180 min before the measurement of locomotor activity. The obtained results show that DEX may decrease the locomotor activity and reduce the hyperactivity induced by dopamine agonists in mice. These observations may suggest that DEX weakens the activity of dopamine agonists in the mesolimbic system.

Key words:
dexamethasone, dopamine agonists, mice