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Influence of buspirone on cytokines in Parkinson's disease: A role for 5-HT1A receptors

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Background and Aims: The available literatures show that Parkinson's disease (PD) is largely resulted from a selective degeneration of nigrostriatal dopaminergic neurons, probably as a result of neuroinflammation and the presence of inflammatory mediators (TNF-α, IL-1β,and IL-6,) in the cerebrospinal fluid (CSF).Because of neuroprotective role of 5-HT1A receptor agonists,this study aimed to investigate the effect of buspirone, a partial agonist of 5-HT1A receptor, on the level of TNF-α, IL-1β,and IL-6, in the CSF of parkinsonian rats.
Methods: We attempted to evaluate the effect of buspirone at doses of 0.5,1 and 2 mg/kg(ip) on the level of inflammatory mediators in the CSF of 6-hydroxydopamine (6-OHDA)-lesioned male Wistar rats.Catalepsy was induced by unilateral infusion of 6-OHDA (8 µg/2 µl/rat) into the central region of the substantia nigra pars compacta (SNc) and assayed by the bar test. Ten days after ip injection of buspirone we collected CSF and measured the level of TNF-α, IL-1β, and IL-6, in parkinsonian rats pre-treated with effective anti-cataleptic dose of buspirone (1mg/kg) by ELISA method.
Results: Our data showed that TNF-α raised in parkinsonian rats and buspirone decreased it to the level of control group.The levels of IL-6 and IL-1β diminished following 6-OHDA injection when compared with control, while buspirone increased their level in the CSF.According to our results 6-OHDA could increaseTNF-α but the levels of IL-6 and IL-1β were decreased.It seems that degeneration of microglias may lead to reduction in IL-6 and IL-1β levels.
Conclusions: We suggest possible involvement of inflammatory cytokines in anti-cataleptic effect of buspirone in parkinsonian rats. Further studies are needed to establish a neuroanti-inflammatory effect for buspirone in PD.

Keywords: Buspirone; Parkinson; Disease; Neuroinflammation; 5-HT1A receptore