

## عنوان مقاله:

Attenuation Effect of Cannabinoid Type \ Receptor Activation on Methamphetamine-Induced Neurodegeneration and Locomotion Impairments among Male Rats

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## خلاصه مقاله:

Background: A number of neuroimaging studies on human addicts have revealed that abuse of Methamphetamine (METH) can induce neurodegenerative changes in various brain regions like the cerebral cortex and cerebellum. Although the underlying mechanisms of METH-induced neurotoxicity have been studied, the cellular and molecular mechanisms of METH-induced neurotoxicity remain to be clarified. Previous studies implicated that cannabinoid type V receptors (CBVRs) exert neuroprotective effects on several models of cerebral toxicity, but their role in METH-induced neurotoxicity has been rarely investigated. Moreover, the cerebellum was considered as a potential target to evaluate the effects of cannabinoids on locomotion activity as the CBVRs are most widely distributed in the molecular layer of cerebellum. Therefore, the present study was carried out to evaluate whether neurodegeneration induced in the cerebellum tissue implicated in locomotion deficit induced by METH.Methods : In the current study, open field test was used to examine locomotor activity. Using hematoxylin and eosin (H&E) staining, morphology of the cerebellar vermis was investigated after repeated exposure to METH. Then, the effects of CBVRs antagonist [SRVVYFVA, V+ mg/kg, intraperitoneally (IP)] and CBVRs agonist [WIN&b, YVY-Y (WIN), Y mg/kg] against METH-induced neurodegeneration and locomotor deficit were assessed.Findings : The results of the present study demonstrated that repeated exposure to METH increased cerebellar degeneration level as compared to the saline and dimethyl sulfoxide (DMSO) groups. In addition, METH-treated rats showed hyperactivity as compared to the saline and DMSO groups. Pretreatment with WIN significantly attenuated neurodegeneration and hyperactivity induced by METH.Conclusion: The findings of this study provided evidence that CBVRs may serve as a therapeutic strategy for attenuation of METH-induced locomotor deficits.

کلمات کلیدی: Neurodegeneration, Methamphetamine, cerebellum, CB۱ receptor

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