Abstract

PTSD is a condition that develops after an individual has experienced a major trauma and associated with inability to extinguish these fear memories, increase anxiety behaviors & hippocampal atrophy, HPA axis dysfunction & increase the risk of neurodegenerative disease. Studies showed that apoptosis is a neurodegenerative process that has important role in that, although volume & function of hippocampus reduce by the chronic effect of glucocorticoids. Hippocampal steroid receptors control the HPA axis. Glucocorticoids involve in stress response, they also have activity in immune system, control of physiologic & basal level of metabolism and memory consolidation. The SSRIs have been considered as a first-line medication choice but the response of rates rarely exceed 60%. Exercise is advocated as a behavioral intervention to alleviate neurological deficits. Adult female Wistar rats divided to SPS & sham groups. We used to (SPS) model as an animal model for PTSD. After 14 days SPS & sham group rats divided to two subgroups: 1) Exercise group: moderate treadmill exercise for 4 weeks & 5 days in each week. 2) Control group without any intervention. At the end of the intervention, rats from each group were decapitated, trunk blood was collected & serum was used for the corticosterone assay. The hippocampi were also removed and the mRNA expression of pro-apoptotic (Caspase 3) proteins was determined by using RT-PCR method. Statistical analysis showed that corticosterone levels significantly increased in PTSD rats and exercise can alleviated this factor in these animals, expression of Caspase 3 mRNA increased in the SPS rats, and exercise reduced expression of Caspase 3 mRNA in this animals. This study have shown that moderate treadmill exercise can be reduce the rate of apoptosis in hippocampus PTSD rats by regulating corticosterone secretion.

Keywords: Stress, Exercise, Corticosterone & Hippocampal Apoptosis, PTSD Rats

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