Abstract

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Project Title : Identification of circadian rhythm of clock and clock controlled genes in the brain regions involved cognition function of the type2 diabetic rats

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Abstract:

Modern societies have promoted the intake of high-calorie diets and sedentary lifestyles, causing the abnormal function of biological clock and accelerated body weight gain which increase the risk to develop metabolic syndrome. Chronic hyperglycemia is a primary factor in the pathophysiology of diabetes. The present study aimed to investigate the effect of high-fat diet (HFD)-fed and streptozotocin (STZ)-induced hyperglycemia condition and to examine the effect of melatonin on the adult hippocampal neurogenesis. In HFD-fed and STZ-treated rats, the reduction of neurogenesis in the hippocampus was observed as shown by the reduction of nestin, doublecortin (DCX) and β-III tubulin immunoreactivities. Likewise, the reduction of ionotropic glutamate receptor (NR2A) and synaptic proteins, synaptophysin, PSD-95 expression were detected whereas, the level of GFAP was increased in the hippocampus of HFD-fed and STZ-treated rats. Melatonin administration significantly increased the level of neurogenesis markers, glutamate receptor and synaptic proteins whereas significantly decreased the GFAP proteins expression in the HFD-fed and STZ-treated rats compared to that observed in the vehicle-injected HFD-fed and STZ-treated rats. Moreover, melatonin improved the decreases of melatonin receptors (MT1 and MT2) and insulin receptor-β, including p-IR-β and p-ERK, its downstream signaling, that occurred in the hippocampus of HFD-fed and STZ-treated rats. Therefore, the results suggest that melatonin ameliorates the decrease of neurogenesis and synaptic formation, and prevents the astrocytic activity which may also be involved in the protection of insulin signaling impairment via melatonin receptor and protect the damage of insulin receptors under the hyperglycemia condition.

Keywords : hippocampus, clock gene, neurogenesis, hyperglycemia