

عنوان مقاله:

High-intensity interval training reduced oxidative stress and apoptosis in the hippocampus of male rats with type 2 diabetes: The role of the PGC1 α -Keap1-Nrf2 signaling pathway

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خلاصه مقاله:

Objective(s): This study aimed to determine the effect of 8-week high-intensity interval training (HIIT) on oxidative stress and apoptosis in the hippocampus of male rats with type 2 diabetes (T2D). The study focused on examining the role of proliferator-activated receptor gamma co-activator 1 α (PGC1 α)/Kelch-like ECH-associated protein Keap1/nuclear factor erythroid 2-related factor 2 (Nrf2) signaling pathway. **Materials and Methods:** Twenty-eight 8-week-old Wistar rats were randomly assigned to one of four groups (n=7): control (Con), type 2 diabetes (T2D), exercise (Ex), and exercise + type 2 diabetes (Ex+T2D). The Ex and Ex+T2D groups completed an 8-week exercise program consisting of 80-100% Vmax and 4-10 intervals. The homeostasis model assessment of insulin resistance (HOMA-IR) index was used to assess insulin resistance. The levels of Bcl2, BAX, musculoaponeurotic fibrosarcoma (Maf), Nrf2, Keap1, and PGC1 α in the hippocampus were assessed using the western blot method. Additionally, the levels of antioxidant enzymes in the hippocampus were measured using ELISA. **Results:** The findings indicated that the T2D group had lower levels of antioxidant enzymes, Maf, Bcl2, PGC1 α , and Nrf2, and higher levels of BAX and Keap1 in the hippocampus. Conversely, the HIIT group exhibited increased levels of antioxidant enzymes, Maf, Bcl2, Nrf2, and PGC1 α , along with decreased levels of BAX and Keap1 in the hippocampus. **Conclusion:** The study demonstrated that 8-week HIIT was effective in reducing hippocampal apoptosis and oxidative stress induced by T2D by activating the PGC1 α -Keap1-Nrf2 signaling pathway. The metabolic changes induced by exercise may lead to an increase in PGC1 expression, which is the primary stimulator of the Keap1-Nrf2 signaling pathway.

کلمات کلیدی:

Anti-oxidant enzymes, Apoptosis, Hippocampus, PGC1 α , Type 2 diabetes

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