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Carbamylated erythropoietin-FC protects hippocampus against Aβ induced memory deterioration in a rat model of Aβ toxicity: Considering Akt/GSK-3β, MAPKs and MMP-2

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Alzheimer's disease (AD) is a debilitating neurodegenerative disease, characterized by extracellular deposition of senile plaques, mostly amyloid β-protein (Aβ) and neuronal loss. It has been reported that erythropoietin (EPO) has neuroprotective effects in some models of neurodegenerative disease but because of its hematopoietic side effects, its derivatives lacking hematopoietic bioactivity is recommended. This study evaluated the neuroprotective effects of carbamylated erythropoietin-FC (CEPO-FC) against beta amyloid induced memory deficit. Adult male Wistar rats weighing 250–300 g were cannulated bilaterally into CA1. Aβ25–35 was administered intra hippocampally for four consecutive days (5 μg/2.5 μL/each side/day). CEPO-FC (500 or 5000 IU) was injected intraperitoneally during the days 4–9. Learning and memory performance of rats was assessed on days 10–13 using Morris water maze and then the hippocampi were isolated and the amount of activated forms of hippocampal MAPKs subfamily, Akt/GSK-3β and MMP-2 were analyzed by western blot. The behavioral results revealed that CEPO-FC treatment in both 500 and 5000 IU significantly reversed Aβ-induced learning and memory deterioration. Molecular analysis showed an increment of MAPKs and MMP-2 activity and an imbalance in Akt/GSK-3β signaling after Aβ25–35 administration. CEPO-FC treatment prevented the elevation hippocampal of P38, ERK, MMP-2 activity and also Akt/GSK-3β signaling impairment induced by Aβ25–35; however, it had no effect on c-Jun N-terminal kinase (JNK). It seems that CEPO-FC prevents Aβ-induced learning and memory deterioration and modulates hippocampal MAPKs, Akt/GSK-3β and MMP-2 activity. This study suggests CEPO-FC can be considered as a potential therapeutic strategy for memory deficits like AD.

Biography

Etrat Hooshmandi is a PhD student of Neuroscience at Shahid Beheshti University of Medical Sciences, Tehran, Iran. Her thesis and projects are about therapeutic targets and molecular signaling in rat models of Alzheimer's diseases.

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