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Neuroprotective effects of saxagliptin against scopolamine-induced Alzheimer's-like pathology in rats

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Alzheimer's Disease (AD), the most common form of dementia, is characterized by the loss of normal functions of brain cells and neuronal death, ultimately leading to memory loss. Recent accumulating evidences have demonstrated that disruption of neuronal insulin signaling is involved in the progression of AD. Moreover, it was proposed that saxagliptin, a dipeptidyl peptidase (DPP)-4 inhibitor and an oral hypoglycemic might have neuroprotective properties. However, the signaling mechanisms underlying this action have not been fully delineated yet. Thus, the current study was directed to investigate the possible role of neuronal insulin signaling cascade and its interaction with cholinergic and GABAergic systems as potential mechanisms by which saxagliptin protects against scopolamine-induced Alzheimer's like pathology in adult male rats. AD-related pathology was induced by a daily intraperitoneal injection of scopolamine at a dose of 3mg/kg for six weeks. Animals were orally administered saxagliptin, one hour before scopolamine injection, at a dose of 3mg/kg for six weeks. Saxagliptin mitigated scopolamine-induced cognitive and spatial memory deficits in rats. Such effects were accentuated by the associated increase in hippocampal acetylcholine. Furthermore a reduction in β -amyloid plaques, tau phosphorylation and its upstream glycogen synthase kinase 3- β (GSK-3 β) was observed. Moreover, saxagliptin restored scopolamine-induced impairment of neuronal insulin. Collectively, our results suggest that saxagliptin can have a promising therapeutic effect in mitigating scopolamine-induced disruption of insulin signaling and other pathological aberrations in Alzheimer's disease.

Biography

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