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Favorable effects of vildagliptin on metabolic and cognitive dysfunctions on drug induced Alzheimer's disease in rats with metabolic syndrome

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Many growing evidences suggest the presence of several similarities in the molecular mechanisms underlying the neurodegenerative diseases and metabolic abnormalities. Adults who develop Metabolic Syndrome (MS) at later stages are at a higher risk of developing Alzheimer's disease (AD). Pharmacological agents, like dipeptidyl peptidase-4 (DPP-4) inhibitors that increase the levels of glucagon like peptide 1 (GLP-1) and ameliorate symptoms of MS, have become an auspicious candidate as disease modifying agents in the treatment of AD. The present study investigates the beneficial effects of Vildagliptin, a DPP-4 inhibitor in counteracting cognitive decline in different models of AD targeting the AKT-signaling pathway and hippocampal Klotho expression, to judge the potential neuroprotective, anti- apoptotic and anti-inflammatory effects of the drug. Cognitive decline was induced by either administration of high fat high sugar (HFHS) diet for 45days, or oral administration of AlCl3 (100 mg/k.g/day) for 60 days. Rats were orally administered Vildagliptin (10mg/k.g) for 60 days along with AlCl3 administration. Vildagliptin treatment markedly improved spatial memory compared to control groups in morris water maze (MWM) test. Results revealed an increase of both hippocampal klotho and AKT expression in treatment groups compared to control groups. On the other hand, Vildagliptin treatment decreased hippocampal levels of both inflammatory and apoptotic biomarkers as TNF-α and caspase-3 along with serum glucose and lipids compared to control groups. These findings demonstrate that treatment with vildagliptin has a protective role in improving both metabolic abnormalities and memory deficits, possibly via its anti-inflammatory and anti-apoptotic effects, together with elevation of hippocampal Klotho expression.