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Gliptins have been recently shown to conquer neuronal degeneration in cell cultures via modulating glucagon-like peptide (GLP)-1. This peptide produced in the gut not only crosses the blood brain barrier, but is also synthesized in the brain and acts on GLP-1R exerting central anti-inflammatory and anti-apoptotic effects thus impeding neuronal damage. This study investigated the anti-parkinsonian effect of vildagliptin, a dipeptidyl peptidase (DPP)-4 inhibitor in a rat rotenone model targeting mainly the RAGE-NFkB/Nrf2-signaling pathways to judge the potential anti-inflammatory/antioxidant effects of the drug. Vildagliptin markedly improved the motor performance in the open field and rotarod tests, effects that were emphasized by the accompanied reduction in striatal dopamine content. It modified the striatal energy level (ADP/ATP) associated with partial antagonism of body weight reduction. This incretin enhancer suppressed nuclear factor (NF)kB, and consequently the downstream inflammatory mediator tumor necrosis factor- α . Normalization of receptor for advanced glycated end product (RAGE) is a main finding which justifies the anti-inflammatory effects of vildagliptin, together with hampering striatal inducible nitric oxide synthase (iNOS), intracellular adhesion molecule (ICAM)-1, as well as myeloperoxidase (MPO). Antioxidant potential of vildagliptin guarded against neuronal demise through an anti-apoptotic effect as reflected by the reduction of the mitochondrial matrix component cytochrome c and the key downstream executioner caspase-3. In conclusion, vildagliptin is endowed with various neuroprotective effects hence can be a promising candidate for the management of Parkinson's disease.

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