Role of kinins receptor b2 in therapy of Parkinson's disease in animal modell

H. D. N de Souza[1], A. Oliveira, H. Ulrich
[1]Departmente of Biochemistry, Universidade de Sao Paulo, Brazil

Parkinson´s disease (PD) a neurodegenerative disorder, is characterized by the loss of dopaminergic neurons in the substantia nigra and its projections into the striatum causing various motor deficits. Nowadays, treatment mostly relies on L-DOPA administration; however, effects produced by this drug are limited and cause diverse side effects. Treatment of PD is initiated at progressed stages of PD, since symptoms only become evident after a loss of at least 50 % of dopaminergic neurons in the substantia nigra accompanied by a drastic reduction of dopamine content in the striatum. The slow and progressive death of dopaminergic neurons let to suggest therapeutic strategies aiming at protection of the remaining ones against apoptosis and stimulation of neurogenesis for replacement of lost neurons. In view of that, the exploration of neuroprotective, self-renewal of stem cells inducing and neuroregenerative properties of bradykinin may help to substitute lost dopaminergic neurons in addition to enhance the survival of reminiscent neurons. The bioactive peptide bradykinin obtained from cleavage of precursor kininogens activates the kinin-B2 receptor functioning in induction of inflammation and vasodilatation. Recent evidence suggests that bradykinin participates in kidney and cardiovascular development and neuronal differentiation. Here we show that kinin-B2 receptors and the participation of bradykinin in neuroregeneration in a rat model of PD induced 6-OH-dopamine injection. Bradykinin injection following establishment of PD symptoms resulted in improvements in the lesioned areas as studied by tyrosine hydroxylase immunostaining and motor functions.