Cai et al., demonstrated that basic fibroblast growth factor (bFGF), as a neurotrophic factor, inhibited Endoplasmic reticulum (ER) stress-induced neuronal cell apoptosis and that Oxidopamine (6-hydroxydopamine 6-OHDA)-induced ER stress was involved in the progression of Parkinson's disease (PD) in rats. bFGF administration improved motor function recovery, increased tyrosine hydroxylase (TH)-positive neuron survival, and upregulated the levels of neurotransmitters in PD rats.

The 6-OHDA-induced ER stress response proteins were inhibited by bFGF treatment. Meanwhile, bFGF also increased expression of TH. The administration of bFGF activated the downstream signals PI3K/Akt and Erk1/2 in vivo and in vitro. Inhibition of the PI3K/Akt and Erk1/2 pathways by specific inhibitors partially reduced the protective effect of bFGF. This study provides new insight towards bFGF translational drug development for PD involving the regulation of ER stress [1)].

1)
Cai P, Ye J, Zhu J, Liu D, Chen D, Wei X, Johnson NR, Wang Z, Zhang H, Cao G, Xiao J, Ye J, Lin L. Inhibition of Endoplasmic Reticulum Stress is Involved in the Neuroprotective Effect of bFGF in the 6-OHDA-Induced Parkinson's Disease Model. Aging Dis. 2016 Jan 17;7(4):336-449. doi: 10.14336/AD.2016.0117. eCollection 2016 Aug. PubMed PMID: 27493838; PubMed Central PMCID: PMC4963188.