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Pramipexole combined with the BDNF gene transfection to surviving dopamine neurons rescues dendritic spines and motor behavior in the rat model of Parkinson´s disease

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For a treatment to be successful in treating Parkinson´s disease, it should control the atrophy of the dendrites and loss of spines of the striatal MSNs. The dendritic abnormalities are not only due to a diminished dopamine delivery, but also to a reduced BDNF delivery because of the degeneration of the dopamine nigral neurons. Both dopamine D3 receptors and BDNF are required for the survival, protection and proliferation of dopamine nigral neurons, and apparently, they act synergistically. We have been studying the effects of long-term activation of dopamine D3 receptors combined with the BDNF gene transfection to dopamine neurons surviving the 6-OHDA-induced degeneration. Here, we studied the effect of the long-term administration of oral Pramipexole combined with the non-viral BDNF gene transfection to dopamine nigral neurons surviving the 6-OHDA-induced degeneration. The combined treatment rescued the dendritic spines of the MSNs and the dopamine nigral neurons, which was associated with the full recovery of motor behavior and normal muscle tone (muscular rigidity abolished). The recovery apparently was permanent because it persisted 3 months after the end of the treatment, which is consistent with the recovery of the dendritic spines of the striatal neurons. Thus, the treatment appears to be a promising disease modifying treatment for Parkinson´s disease.

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Estrogen affects iron metabolism in astroctyes and neurons

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Epidemiological studies have demonstrated that the postmenopausal women harbor a higher level of body iron than premenopausal women. Nigral iron accumulation is involved in the etiology of Parkinson's disease. Recent studies demonstrated that the women are on average 2.1 years older than the men at time of diagnosis. Moreover, medical conditions leading to estrogen depletion increase the risk of PD. The importance of estrogens and iron to physiology and disease has been known for decades, but we often overlook that these two factors interact. In this study, we investigated the effect of estrogen on the iron transport proteins as well as its mechanisms in midbrain. The results were as follows: Iron exporter ferroportin1 (FPN1) and iron importer divalent metal transporter 1 (DMT1) were up-regulated after estrogen was treated in primary cultured astrocytes, while hypoxia inducible factor-1alpha (HIF-1α) was up-regulated, but hypoxia inducible factor 2 alpha (HIF-2α) remained unchanged. In neurons, DMT1 was decreased but FPN1 was up-regulated after estrogen was treated. IRP1 was down-regulated while HIF-1α and HIF-2α remained unchanged after estrogen was treated in primary cultured neurons. The results suggest that the regulations for iron metabolisms of estrogen on astroctyes and neurons are different. Estrogen can increase FPN1 and DMT1 expressions by elevating HIF-1α in astrocytes. However, the decreased expression of IRP1 may account for the decreased DMT1 and increased FPN1expressions in neurons.

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