Are cyclooxygenase-2 and nitric oxide involved in the dyskinesia of Parkinson's disease induced by L-DOPA?

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Supplementary figures

Supplementary Figure 1. Timeline of treatments and analyses of abnormal involuntary movements. Effects of nNOS inhibitor against the development of the L-DOPA-induced abnormal involuntary movements in hemi-Parkinsonian rats. NOS inhibitor, the 7NI or vehicle (saline-DMSO 50%, i.p.) was given 30 min prior to L-DOPA administration for 21 days from the 1st day of L-DOPA treatment. The abnormal involuntary movements were scored at 60 and 120 min after L-DOPA on the indicated days and rats were killed 3h after the drug treatment. After the behavioural analysis, the animals were subjected to biochemical analyses.
**Supplementary Figure 2.** COX2-immunoreactivity in the hippocampus and dorsal striatum of control rats. Photomicrographs showing COX2-immunopositive cells on hippocampus (A-C). In the dorsal striatum (D) COX2-immunopositive cells were sparse or absent.
**Supplementary Figure 3.** Expression and co-localization of COX2 and calretinin immunopositive cells of 6-OHDA-hemiparkinsonian rats with L-DOPA-induced dyskinesia. Photomicrographs of striatal (A-C) and cortical (D-E) sections showing immunofluorescence staining for COX2 (green) in combination with calretinin-immunopositive neurons (in red). No co-localization was observed. (C-E) Merged images. Scale bars: striatum 50 µm; cortex 100 µm.
Supplementary Figure 4. Confocal laser scanning micrography of COX2 and GFAP immunoreactivity in the stritatum of a 6-OHDA lesioned rat treated with L-DOPA. Photomicrographs of a striatal section showing immunofluorescence staining for COX2 (green) and GFAP (glial fibrillary acidic protein, red). (A and B) No colocalization was observed for COX2 and GFAP immunopositive cells. (B) High magnification showing proximity of COX2 and GFAP immunopositive cells.