PW01-81 - **DEFICITS OF SPATIAL MEMORY AND OXIDATIVE STRESS DAMAGE FOLLOWING EXPOSURE TO LIPOPOLYSACCHARIDE IN A RAT MODEL OF PARKINSON'S DISEASE**

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Objectives: The present work was undertaken in order to investigate the effects of systemic lipopolysaccharide endotoxin administration (250 µg/kg) on spatial memory formation and oxidative stress in rats subjected to right-unilateral lesion of the dopaminergic neurons of the substantia nigra by means of 6- hydroxydopamine (6-OHDA) compared to normal and lipopolysaccharide alone treated rats.

Methods: Thirty male Wistar rats weighing 200 ± 50 at the start of the experiment were used. The substantia nigra was right-unilateral lesioned by stereotaxic microinjections of 8 micrograms (free base) 6-OHDA. The rats were pretreated 30 min before the 6-OHDA infusion with 25 mg/kg desipramine to protect noradrenergic projections. Sham-operated rats received only an injection of desipramine, followed by vehicle in the substantia nigra. Lipopolysaccharide (LPS from Escherichia coli serotype 0111:B4, Sigma) was dissolved in pyrogen-free 0.9% NaCl at the concentration of 250 µg/kg and intraperitonealy injected in normal and 6-OHDA-lesioned rats for a period of 7 continuous days. 7 days after continuous LPS administration we assessed memory formation by means of Y-maze and radial arm-maze task, and the endogenous antioxidants activity in rat temporal cortical area.

Results: Systemic lipopolysaccharide administration significantly decreased spontaneous alternation in Y-maze task, working memory and reference memory in radial arm-maze task, suggesting impairment of both short-term memory and long term-memory, respectively. Furthermore, lipopolysaccharide administration significantly decreased activity of the biochemical markers of endogenous antioxidants in rat temporal cortical area.

Conclusions: Our results further validate that lipopolysaccharide may exacerbate the development of neurological dysfunctions associated with Parkinson's disease.

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