GLYCOGEN SYNTHASE KINASE 3B IN PARKINSON DISEASE INDUCED BY PARAQUAT

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Introduction: Parkinson’s disease (PD) is progressive neurodegenerative disease influenced by genetic and environmental factors. In recent years, the investigation on paraquat (PQ) toxicity has suggested that this herbicide might contributing to PD. Till now nothing is known about the role of glycogen synthase kinase-3β (GSK-3β) in PD and in PQ toxicity.

Aims: The aim of the study was to examine the influence of short (3,24 hours, 4 weeks) and long (37 weeks) administration of PQ in rats of the total GSK-3β and its active, tyrosine 216 (pY216) - phosphorylated form in the midbrain, as well as of the striatum.

Methods: The study was carried out using male Wistar rats. This model was described previously by Songin et al.2010. Paraquat was administered i.p (40mg/kg and 10mg/kg per b.w) . Quantitative PCR and immunochemical methods were used.

Results: The results revealed that the short time of PQ administration evoked significant increase of gene expression for iNOS and COX-2 in midbrain and striatum but GSK-3β is not altered. However the long-term PQ administration increased the levels of total and active forms of GSK-3β in the midbrain, whereas decreased them in the striatum. These differences may be connected with alteration of axonal transport between these two brain parts evoked by PQ.

Conclusions: The present data indicate that the long-term exposure of rats to PQ, a commonly used herbicide, diversely alters levels of GSK-3β in different brain structures, which may be associated with their vulnerability to its toxicity.

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