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Differential effects of intraperitoneal and oral rotenone administration on Parkinson's-disease-linked genes, brain urea levels and gut pathology in a rat model of Parkinson's disease

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Abstract

Parkinson's disease (PD) is a progressive disease characterized by basal ganglia dysfunction caused by degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc) and development of fibrillar cytoplasmic inclusions that contain alpha synuclein (α-syn) and ubiquitin. Many pivotal works have plausibly linked autophagy and mitochondrial energy dysfunction with PD. In other neurodegenerative conditions, including Alzheimer's disease (AD) and Huntington's disease (HD), brain urea levels are elevated, although the relative cause for these changes is unknown. Several studies demonstrated that gut microbiota may contribute to PD, and these effects could increase α-syn expression in gut tissue. While there is ample evidence that emphasizes the importance of both brain and gut pathological changes in PD, there is no definitive proof as to which organ is affected first.

Rodent rotenone models of PD have been widely employed to explore molecular mechanisms involved in the disease pathogenesis and to test the efficacy of therapeutic drugs. However, it is unknown whether or not the impact of oral (po) and intraperitoneal (ip) rotenone promotes the same PD pathogenesis. To this end, we explored whether po and ip rotenone treatment induced a similar PD-like progression. We examined various protein and gene expression changes as well as urea levels in brain samples and the results indicate that sub chronic systemic rotenone administration altered α-syn and tyrosine hydroxylase levels in the corpus striatum, data that are indicative of rotenone-induced neuronal damage. Gene expression changes in autophagy- and energy-homeostasis-related genes as well as brain urea levels suggest the rotenone impairs normal cellular processes. Intriguingly, there were apparent differences between ip and po rotenone administration, where the former method more robustly affected these measures. Finally, the notable gut histopathology alteration induced by systemic rotenone administration implicates the gut microbiota and/or ENS in the PD-like pathology.

Biography

SuchitraKavuri has completed her PG in Medical Biochemistry from Kasturba Medical College, Manipal University, India and completed her PhD from Saveetha University, India. She is working as Assistant Professor in the Department of Biochemistry at ASRAM Medical College, India since March, 2002 and actively involved in teaching Medical Biochemistry for medical graduates, paramedical and nursing students. She plays a role as Quality Manager in the Central Clinical Laboratory and maintains Quality Assurance Department as per ISO: 15189, 2012 at ASRAM Medical College & Hospital.

Publications

1. Oxidative stress and antioxidant status in rotenone induced rat models of Parkinson's disease
2. Evaluation of Haematological Alterations in Intraperitoneal and Oral Rotenone Induced Parkinson's Disease Wistar Rats.
3. A study of variations in the iron profile and vitamin – b12 levels as predictive bio-chemical markers for gestational diabetes mellitus (gdm) in pregnant women (Funded work).
4. Comparison of Prothrombin Time and Activated Partial Thromboplastin Time between patients with diabetes mellitus and diabetics with hypertension.
5. Oxidative stress in pediatric nephrotic syndrome

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