May 2nd, 12:00 AM

**Effect of Dosage on Severity of Dysphagia in a Toxicological Rat Model of Parkinson's Disease**

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Introduction

Parkinson’s Disease (PD) is a prevalent neurodegenerative disorder that causes progressively worsening motor symptoms affecting oropharyngeal and locomotor function (Kwon and Lee, 2019). There is limited research in animal models on swallowing dysfunction in PD. The goal of this study was to examine how neurodegeneration in PD produces progressive impairment in the oropharyngeal and locomotor processes.

Hypotheses

H1: Animal models injected with the rotenone will exhibit oropharyngeal dysfunction with increased deficit that correlates with prolonged treatment.

H2: Animal models injected with rotenone will exhibit locomotor deficiency with a positive correlation between treatment length and deficit.

Material and Methods

18 Lewis rats received either 2.75 mg/kg of rotenone or vehicle intraperitoneal injections (Cannon et al. 2009, Gould et al 2018). Animals received daily injections for 17 or 8 days, respectively. At the end of the injections in each group, the rats’ brains were immunostained for striatal tyrosine hydroxylase (TH). Videofluoroscopic recording was taken of the rats and total chewing time and average chew cycle length were collected from days 0, 1, 4, 7 and 16 of those recordings.

The number of rears by a rat in a glass cylinder over 4 minutes was counted on the same days to assess locomotor behavior.

Hypothesis were tested using linear mixed models to control for interindividual variation.

Results

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H1 is partially supported. Total chewing time and average chew cycle duration increased in rotenone-treated rats from day 0 to day 4, but the difference disappeared from day 7 onwards.

H2 is fully supported. Rearing behavior was consistently reduced in rotenone-treated rats for all 16 days. TH staining intensity in the striatum decreased consistently over the course of the injections.

Discussion

These results suggest that rotenone-treated rats have temporary/short term deficiency in chewing function. The restoration of function after day 4 may indicate a learned behavior to compensate for this dysfunction in chewing. It may also point to a different neurological impairment than that seen in locomotor behavior. Locomotor impairment however persisted throughout the experiment. Dysphagia is a crucial nonmotor symptom in PD and the pathophysiology of the preparation and execution of swallowing is complex and has not yet been fully understood. Further research needs to be undertaken to understand how the rotenone model of PD produces dysfunction in the swallowing sequence in order to advance its use in the study of parkinsonian dysphagia.

References


Acknowledgements

The authors would like to thank the vivarium staff at Rowan-Virtua School of Osteopathic Medicine for the outstanding animal care and support. This work was funded by Rowan-Virtua seed funding to Dr. Gould.