INVESTIGATION OF THE INVOLVEMENT OF CALCIUM REGULATION IN THE UNILATERAL ROTENONE-LESIONED MOUSE MODEL OF PARKINSON’S DISEASE

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Introduction: Neurodegeneration in Parkinson’s disease is associated with protein aggregation and the formation of neuronal Lewy bodies mainly composed of the protein alpha-synuclein. Alpha-synuclein has been shown to aggregate when intracellular calcium levels are elevated. Moreover, the calcium buffering protein, Calbindin, the expression of which results in relative sparing of Calbindin-positive neurons in PD, may provide a degree of protection against the pathological process in Parkinson’s disease, pointing to the influence of calcium dysregulation in neurodegeneration. Methods: Unilateral lesioning of the medial forebrain bundle of C57 black mice with the mitochondrial inhibitor, rotenone, was performed to induce oxidative stress and stimulate neurodegeneration. Fluorescence immunohistochemistry of brain tissue was used to determine the frequency of Calbindin-28K positive cells and alpha-synuclein aggregates. Results: Confocal microscopy of mouse brain tissue sections showed more frequent Calbindin-28K positive cells within the lesioned hemisphere (p, 0.05) than within the control hemisphere. The data indicates more frequent alpha-synuclein aggregates within the lesioned hemisphere, and that aggregates are less numerous in Calbindin-positive cells. Conclusion: These findings suggest an association between the calcium buffering protein, Calbindin-28K, and neuronal survival and alpha-synuclein aggregation in a mouse model of Parkinson’s disease and implicate the involvement of calcium dysregulation in Parkinson’s disease. (202 words).