JOJ Ophthalmology
ISSN: 2473-5477

Juniper Publishers
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Letter To Editor
Volume 11 Issue 5 - November 2024
DOI: 10.19080/JOJO.2024.11.555822

JOJ Ophthalmol

Impair Color Vision by Impair Cerebellum Pathway: Comparison with Parkinson's Disease

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Submission: October 08, 2024; **Published:** November 05, 2024

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Keywords: Impair Color Vision; Parkinson's Disease; Retinal Receptors; Granule Cells

Letter To Editor:

Dear Editor,

From the retinal receptors to the visual cortex can originate any visive abnormality, anywhere in the visual pathway. Previously, we showed as Parkinson's Disease affects dopaminergic substantia nigra neurons within the brain system, and the decline in color vision may be predicted by the level of retinal dopamine deficiency. Previously, we showed the involvement of an olive-cerebellum-rubral-thalamic loop did not seem to influence color vision in Essential Tremor Disease, too. Anatomical and physiological studies have established that the color center begins in V1 visive cortex area and sends signals to extra-striate areas V2 e V4 for further processing, in particular because V4 has the strength of the color receptive fields in its neurons. Lesions in the ventral occipital lobe causing achromatopsia, suggested that ventral occipital area plays an important role in color vision. Reduced color vision is reported in patients with Parkinson's Disease because of the abnormal phosphorylation of human alpha-synuclein localized to the inner retina cells. The influence of Parkinson's Disease is most noticeable in the pathway of short-wave cones because these are widely separated. In the retina, the small bistratified ganglion cells, which are the morphological substrate of the short-wave cone pathway, have much longer receptive fields than the midget ganglion cells and may be more dependent upon long range spatial interactions mediated by dopaminergic inter-plexiform amacrine cells. Retinal parvocellular, koniocellular, and magnocellular damage in Parkinson's Disease also confirms the impairments by the long- wave pathway contrasting with the pathway typically seen in aging. A Parkinson's Disease staging system suggests that alpha-synuclein deposition commences in olfactory, autonomic, and sleep/mood centers before affecting the substantia nigra or the cortex.

The existing evidence for a transmitting role of amino acid from granule cells in cerebellar cortex is primarily neurochemical. In particular, it has been demonstrated that in the conditions in which there is a substantially reduced number of granule cells in the cerebellum, there is a pronounced reduction in the levels of glutamate released by granule cells parallel fibers in the cerebellar cortex. In Essential Tremor Disease, showing an impair color vision both on red/green and blue/yellow axes, allowed us to assess the involvement of the cerebellum structure in the color vision pathway, too. This result can be evidence of the favoring transmitter role for glutamate at the synapses from cerebellum granule cells via the parallel fibers onto Purkinje and other neurons in the cerebellar cortex but few direct studies on the effects of antagonists on this pathway have been made. There is evidence that glutamate could be the neurotransmitter released by granule cell parallel fibers in the cerebellar cortex.

Acknowledgement

Authors thank Fondazione Cassa di Risparmio di Calabria e Lucania for its contribution

FONDAZIONE
CASSA DI RISPARMIO DI CALABRIA E DI LUCANIA

DOI: 10.19080/JOJO.2024.10.555822

How to cite this article: Anna P, Federico R, Teresa I, Teresa La R, Gabriele C, et al. Impair Color Vision by Impair Cerebellum Pathway: Comparison with Parkinson's Disease. JOJ Ophthalmol. 2024; 11(5): 555822. DOI: 10.19080/JOJO.2024.10.555822