ISSN: 2641-1911
DOI: 10.33552/ANN.2023.15.000853
Archives in Neurology & Neuroscience
Iris Publishers

Review Article

The Role of Dopamine on Color Vision Impairment in Alzheimer's Disease and in MCI

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Received Date: April 06, 2023

Published Date: April 13, 2023

Abstract

Several alterations in the dopaminergic system have been reported in Alzheimer's Disease (AD), as well as in the precondition Mild Cognitive Impairment (MCI). This present mini-review reports the color vision study as a prognostic sign for dopamine pathway impairment on bot AD, and MCI patients.

Keywords: Color vision impairment; Alzheimer's Disease; Mild Cognitive Impairment; Dopamine pathway; Clinical test

Retina Vs Cortex Pathway

Two main features of the organization of the retina are central to color processing, the functional linkage between cells (coupling or convergence) laterally through the receptive field, and the feedback mechanism that horizontal cells mediate either electronically or chemically to influence the final output of the cone receptors. The former is the anatomical basis for spatial sensation and the latter for lateral inhibition, both of which are found frequently in sensory pathways. These features have a far greater consequence than mere economic walling of responses, resulting in the cells losing their independence as single units, they greatly influence the sensitivity of the eye and the response to edges or demarcations or stimuli. The retinal signals travel to the brain via axons of the ganglion cells forming one million optic nerve fibers organized in a systematic manner. Macular fibers from the fovea, signaling color, occupy approximately one third of the optic nerve area on the temporal side when they enter. They later move to a central location in the nerve, while towards the chiasma they shift medially. The fibers, carrying impulses coding color, terminate at the lateral geniculate nucleus. The color patterns red-green and blue-yellow remain unchanged from the retinal ganglion cells. The lateral geniculate nucleus receives the axons of the retinal ganglion cells and connects them by synapses with the higher centers of the brain. Finally, nerve impulses signaling color information are relayed from the lateral geniculate nucleus via the visual radiations to the main visual areas of the brain, the striate cortex [1]. The principal zones were designated Area 17 by Brodman [2] in his classical division, the occipital or para-striate cortex designated to Area 18, V4 to a non-Area and the pre-occipital cortex to Area 19 [3-4]. The aim of the present study is to evaluate color vision impairment in AD, and MCI patients.

Alzheimer's Disease and Mild Cognitive Impair on Color Vision

Several alterations in the dopaminergic system have been reported in Alzheimer's Disease (AD) patients, as well as in the precondition Mild Cognitive Impairment (MCI), including reduced

levels of dopamine and alterations in the dopamine receptors. One of the sources of dopamine in the hippocampus derives from dopaminergic neurons in the ventral tegmental area. Dopamine is a well-recognized modulator of hippocampal synaptic plasticity, and dopamine binding to dopaminergic receptors in the dorsal hippocampus is a major determinant of memory encoding. Ventral tegmental area neurons also target the accumbens nucleus and cerebral cortex, mediating the control of incentive motivation and reward processing [4-5].

AD, MCI, and Color Vision

We examined 21 male Calabrian patients: 9/21 were AD patients (age range 54–88, mean age, 72 years); 12/21 were MCI patients (age range 64-83, mean age, 66.5 years). 21 controls were matched for age, and sex. Fixed sampling of males allowed us to avoid the genetic Lyon phenomenon [6] present only in heterozygous females for X-linked diseases, such as colorblindness (the inherited red green color vision deficiency). The exclusion of females also allowed us to avoid the "false positives" for the acquired red green color vision deficiency caused both AD and MCI, altering the results' analysis. Previous results [7] showed an 8% occurrence of colorblindness in Calabria, therefore including females would have resulted in a relatively significant high number of "false positives" subjects showing a red green color vision trend. For example, a Gaussian curve can carry a minimum value (normal color vision) to a maximum value miming inherited red green colorblindness, passing for different anomalous color vision levels. Heterozygous status in females mimes normal color vision. All patients and controls underwent the three tests: Ishihara test [8]; Farnsworth Dichotomous D-15 test [9]; City University test [10].

Therefore, at the stage when no plaque deposition, hyper-phosphorylated tau tangles or any sign of neuronal loss in cortical and hippocampal regions involved in memory deficits has yet occurred, we provide evidence that a specific apoptotic process is taking place in the ventral tegmental area, causing progressive degeneration of the dopaminergic neuronal population.

Conclusion

Dopaminergic neurons act in the outer and inner retina at multiple levels, producing alterations to the flow of visual information. Dopamine is a chemical messenger for light adaptation, promoting the flow of information through cone circuits while diminishing that through rod circuits. Color vision relies on the cone photoreceptors and is therefore largely confined to the central retina. Because there are retinal blue-yellow and red-green pathways, it is possible to use color discrimination tasks to assess cone and retinal ganglion cells [11]. Functional studies performed on AD showed how visual acuity, contrast sensitivity, color vision and visual integration vary with the progression of neurodegeneration. At the stage of AD where plaque deposition occurs, neurons that employ glutamate or acetylcholine are particularly damaged, as are neurons that produce serotonin and norepinephrine. At a stage where no plaque deposition, hyper-phosphorylated tau tangles or sign of neuronal loss in cortical and hippocampal regions involved in memory deficits has occurred, a specific apoptotic process takes place in the ventral tegmental area, causing progressive degeneration of the dopaminergic neuronal population, so alterations in the dopaminergic system include reduced levels of dopamine and alterations in the dopamine receptors. Dopamine is a modulator of hippocampal synaptic plasticity and its binding to dopaminergic receptors in the dorsal hippocampus is a major determinant of memory encoding [12]. Ventral tegmental area dopaminergic neurons also target nucleus accumbens and the cerebral cortex, mediating the control of incentive motivation and reward processing. Degeneration is selective for the ventral tegmental area as dopaminergic neurons in the adjacent substantia nigra pars compacta were intact. Moreover, basal outflow of dopamine in the hippocampus and nucleus accumbens shell is reduced, likely contributing to deficits in mesolimbic cognitive and non-cognitive symptoms [13].

Acknowledgments

The Authors thank Fondazione Cassa di Risparmio di Calabria e Lucania for its contribute

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CASSA DI RISPARMIO DI CALABRIA E DI LUCANIA

Conflict of Interest

No conflict of interest.

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