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**"Retinal Degeneration and Macroglial Function: Contributions of Müller Cells in Usher Syndrome"**

Usher Syndrome (USH1-3) is an inherited disorder that causes visual, vestibular, and hearing loss. Of the three clinical forms, USH1, the most severe form, is characterized by congenital sensorineural hearing loss, vestibular areflexia and early-onset retinitis pigmentosa, a degenerative eye disorder that severely impairs vision. Patients experience gradual deterioration of their photoreceptor cells, eventually losing their full vision. The continuous deterioration highlights the need for investigation and possible treatment measures to slow the advancement of this disease.

Microglial and macroglial cells are involved in the maintenance of the retina's health and function. Microglial cells extend throughout every retina layer and are activated in response to injury or disease. Ionized calcium binding adapter molecule-1 (IBA-1) is a marker for activated microglial cells. Macroglia cells, which include astrocytes and Müller cells, are critical for retinal neuron and photoreceptor survival. Astrocytes, found in the nerve fiber layer of the retina, maintain homeostasis by surrounding blood vessels and neuronal axons. Glial fibrillary acidic protein (GFAP) is a marker for astrocytes. Müller cells degrade neurotransmitters and span from the outer to the inner limiting membrane. These cells also express GFAP in response to retinal injury and can be identified by the presence of glutamine synthetase (GS) and vimentin. Harmonin, a scaffolding protein encoded by the USH1C gene, is expressed in photoreceptors and Müller glial cells; however, its function in vision is undefined. USH1 is caused by mutations in the *USH1C* gene. The objective of this study is to characterize macroglial cells, specifically Müller glia, in USH1C mice.

Eyes were harvested from 3-month-old USH1C and wild type littermates. Frozen retinal sections were examined using immunohistochemistry techniques to examine the expression and localization of IBA1, GFAP, GS, vimentin, and harmonin. A Zeiss confocal microscope was used to capture the images. Fluorescence intensity was analyzed using Image J Software.

The long-term objectives of this research are to advance our understanding of the role of harmonin in vision and the mechanism of USH1C retinal disease. These findings will guide future research in the development of novel therapies to prevent, slow, or cure the disease.