

UNIQUE PATHOPHYSIOLOGY OF THE OPTIC NERVEHEAD: RELEVANCE TO GLAUCOMA

This presentation highlights the unique structural and functional biology that constitutes the mammalian optic nerve head. Multifactorial processes contribute to the pathogenesis of glaucoma, with a major recent emphasis on the role of astrocytes and the blood vessels of the ONH. The challenge confronting the field is to understand the changes in the functional capabilities of astrocytes and pericytes with ageing and how this relates to glaucoma pathology. Our earlier studies investigated changes in astrocyte density, morphology, proliferation and apoptosis occurring in the retina during 'physiological aging'. The density and total number of parenchymal astrocytes in the retina increased between 3 and 9 months of age but decreased markedly between 9 and 12 months in rats. Proliferation of astrocytes was detected at 3 months but virtually ceased beyond that age, whereas the proportion of astrocytes that were apoptotic increased progressively with aging. In addition, in aged retina astrocytes exhibited gliosis-like morphology and loss of Pax2 reactivity. A small population of Pax2+/GFAP+ cells was detected in both young adult and aged retina. Pericyte-endothelial ratio is also significantly reduced in aging, likely affecting blood flow regulation. Taken together, the reduction in the availability of astrocytes and pericytes in aged retina may have a significant impact on the ability of supporting cells in the region of the optic nerve head to maintain homeostasis and support neuronal function in old age. We will also detail our current studies of the changes in pericytes, astrocytes and blood vessels of the human ONH during 'physiological aging'.