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Emerging role of crystallin proteins in retinal neurodegenerative diseases

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The specific goal of this project is to better characterize the specific and respective roles of αA-and αB-crystallin, the two members of the α-crystallin sub-family in the retina and how diabetes but also retinal traumas affect them. Alpha-crystallins are important, conserved, intrinsic stress response proteins in the retina. We previously demonstrated that they are upregulated in diabetic conditions and that their biochemical properties are affected by diabetes, however their exact implication and role in retinal function and how it is affected by retinal diseases or pathological conditions remained to be clarified. Specific biochemical and cellular properties of α-crystallins in the retina were analyzed in animal models as well as human patient samples. In addition α-crystallins knockout mice were used to dissect the respective functions of α-crystallins in the retina in control and pathological conditions. Retinal function was analyzed using non-invasive methods such as optical coherence tomography and electroretinographic recordings whereas specific retinal cell survival was analyzed using cell-death assay and immunohistochemistry. This work clearly demonstrates the independent and specific respective roles of αA-and αB-crystallin in the retina in the context of chronic disease conditions such as diabetes or more acute conditions including retinal trauma. αA-and αB-crystallin proteins clearly display a different localization in the retina as well as different impact on retinal function in neurodegenerative conditions. This study demonstrates how understanding retinal intrinsic protective mechanisms could be critical in order to prevent loss of vision in patients with both acute and chronic retinal conditions but might require different strategies.

Biography

After finishing his master in Neurosciences in 2001, Fort received his Ph.D. in Living Sciences, Cellular and Molecular Aspects of Biology from the University of Strasbourg in France in 2005 for his work on the dystrophin protein Dp71 and its function in two ocular tissues, lens and retina. He then joined the Penn State Retina Research Group for a postdoctoral fellowship studying the underlying mechanisms taking place during diabetes that lead to the loss of vision. In 2008, he was promoted to a faculty position while uncovering and characterizing the upregulation of a family of proteins called crystallins that take place in the retina of several animal models of type 1 diabetes. He was then recruited in 2011 as an Assistant Professor in the Department of Ophthalmology and Visual Sciences of the University of Michigan where he continues to study the regulation of intrinsic protective mechanisms in the retina under normal and pathological conditions.

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