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ROLE OF FRMD7 IN SYNAPTIC CONNECTIVITY IN THE RETINA

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Nystagmus is a condition of the eye characterized by an involuntary and uncontrolled movement. It has a significant impact on vision in general and can affect patient's ability to lead an independent life. FERM domain-containing 7 plasma membrane protein (*FRMD7*) is a member of the band 4.1superfamily of plasma proteins, found to be mutated in families with nystagmus. The lack of *Frmd7* leads to deficit of direction selectivity in mice retina by loss of asymmetric inhibition of direction-selective ganglion cells (DSGC's) by the starburst amacrine cells; the cells that express *Frmd7*. Experiments show no morphological alterations in these cells upon loss of *Frmd7*. However, no evidence if the synapses between the starburst amacrine cells and DSGC's are affected. The aim of this poster is to examine the integrity of the synapses of the starburst amacrine cells.

Methods: Synapses in freshly perfused C57Bl6 controls vs *Frmd7* transgenic knockout (*Frmd7*.^{tm1b}) retina (N=5) were studied by immunohistochemistry of frozen retinal sections.

Results: Initial results show no significant deficit in the integrity of synapses in the starburst amacrine (Acetylcholine transferase (ChAT) expressing) cells in the mouse retina. Density and intensity of ChAT expressing cells in C57Bl6 retina is similar to that of the *Frmd7*.^{tm1b}. Also, density of the presynaptic and postsynaptic markers, synaptophysin and PDS95 respectively, is normal. In addition, density of gamma-Aminobutyric acid (GABA), which is also expressed by the starburst amacrine cells, was also normal.

Conclusion: *Frmd7* is somehow involved in modulating inhibitory signals from the starburst amacrine cell to the DSGC's in the retina. Level of general pre-synaptic and post-synaptic markers in the *Frmd7*.^{tm1b} transgenic retina seem to be indistinguishable from wild type controls, so as expression levels of acetylcholine and GABA, which indicates the synaptic markers in the *Frmd7*.^{tm1b} mice are similar to the wild type control mice. However, the inhibitory feedback from the starburst amacrine cells to the DSGC's is compromised.

Biography

Ahmed Salman obtained his BSc Honours degree in Genetics form the University of Glasgow in 2009. He then studied for his MSc by research in Brasenose college, University of Oxford, under the supervision of Professor Elizabeth Robertson, investigating genes involved in early mouse embryonic development. After finishing his Masters, he stayed in the Robertson lab as a research assistant before joining the Welcome Trust Centre for Human Genetics in Oxford, where he worked on the mechanisms of double-stranded break repair for a year, before starting his PhD in the University of Southampton in 2014, working on the role of Frmd7 gene in nystagmus, a significant eye disease characterised by involuntary eye movements. He is currently in the final year of studies aiming to submit his thesis at the end of 2018.

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