

ABSTRACT

Characterization of protein expression and retinal structure upon GABA Receptor Deficiency

NITHIN RAMACHANDRAN

It has long been known that schizophrenia is a severe chronic psychiatric disorder that is approximately present in 1% of the world population and that about 62% of adults with this disorder experience damages to vision. Recent studies have furthermore found that vision damage occurs due to a change in the retinal architecture. However, not much has been done to investigate the exact root cause for this change in retinal architecture. I was interested in determining what the root cause of these vision problems is and first sought to study whether this disease affects the integrity or the apical processes of the retinal pigmented epithelium cell layer, as this layer is responsible for mediating photoreceptor function. To do this, immunohistochemistry was performed on retinal sections of the wild type (control) and GABA receptor deficient mice (diseased) to compare the expression of Kir 7.1 (a functional protein) and Ezrin (a structural protein). With this, the effects of GABAergic receptor deficiency on the apical processes of these RPE cells was determined. My results concluded that there was no qualitative difference in the colocalization of both Kir 7.1 and Ezrin between the control and GABA receptor deficient mice, indicating that GABA receptor deficiency likely does not have an effect on the apical processes of the RPE cells. On top of this analysis, I sought to study whether this disease affects retinal cells or synapse by altering the overall structure of the retina. I imaged Hematoxylin and Eosin stained retinal sections of the control and GABA receptor deficient mice. With these images, I performed manual nuclear counts within the outer and inner nuclear layer structures of the retina and measured the thickness of the outer and inner plexiform layers. Finally, I performed a statistical data analysis to compare the control and GABA receptor deficient mice to evaluate whether there is any significant difference in the structure of the respective layers between them. However, the H&E stained retinal sections of the GABA receptor deficient mice received were poor, so the statistical comparison of retinal structures between the control mice and GABA receptor deficient mice was not performed. Which is why no conclusion about the effects of GABA receptor deficiency on retinal structure was made. Further studies would pursue new H&E stained retinal sections for both control and GABA receptor deficient mice to better understand the effects of GABA receptor deficiency on retinal structure and eventually understand the root cause of the vision problems caused by this disease. This information will be key in the synthesis of various treatments to help those with vision damage caused by schizophrenia.