ANGPTL7, a Gene Highly Induced by Elevated IOP, Affects Adhesion of Trabecular Meshwork Cells to their Extracellular Matrix

William Hope, B.S. Biology
Dr. Terete Borrás, Department of Ophthalmology
**Background**

- Proteomic studies show that ANGPTL7 is present in trabecular meshwork of glaucomatous patients
- Dysfunction of the trabecular meshwork leads to a decrease in aqueous humor outflow which results in elevated intraocular pressure (IOP)

**Hypothesis**

- Upregulation of ANGPTL7 decreases adhesion of HTM cells to the ECM which may lead to an increase of aqueous humor outflow

**Goals**

- To further understand the role of ANGPTL7 in the adhesion of Human Trabecular Meshwork cells (HTM) to their extracellular matrix
- To determine whether ANGPTL7 influences the levels and localization of the focal adhesion proteins paxillin and α5β1
- To study the adhesion properties of HTM cells overexpressing ANGPTL7
A: Decrease in Adhesion of HTM cells expressing ANGPTL7
B: Decrease in Adhesion of AD293 cell stably transformed with ANGPTL7
C: Decrease and distributional changes in the focal adhesion proteins Paxillin and α5β1

Conclusion: a decrease in the adhesion of trabecular meshwork cells may provide a natural pathway for the body to increase aqueous humor outflow and reduce intraocular pressure, the major risk factor for glaucoma.