

Dr. McKay Research Update:
Retinas of Treated Mice Demonstrate Improvement in Cell Number!
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Overview: The goal of this research is to determine whether OA1 signaling activation can rescue normal retinal development in albino animals. Our hypothesis is that all forms of albinism exhibit the same developmental changes in the retina because all forms of albinism cause a reduction in OA1 signaling.

Current work: In the study funded by The Vision for Tomorrow Foundation, we are testing whether this hypothesis is correct, and whether that information can be used to rescue retinal development. We are using two mouse strains, one is tyrosinase deficient, so it is a complete albino animal. The other is OA1 deficient, so this strain has ocular albinism, but a normal brown coat. We supplement pregnant females of the two mouse strains then measure specific properties of retinal development known to be altered in albino animals. In the case of mice, these changes include decreased ganglion cells, decreased photoreceptors, and loss of the uncrossed retinal projection at the optic chiasm. Our hypothesis is that the supplementation will rescue mice deficient in tyrosinase, but not rescue OA1 deficient mice.

To date, our efforts have indicated that supplementation of tyrosinase deficient mice leads to an increase in ganglion cells and photoreceptors (14% and 18%, respectively). These results were achieved with 18 animals. Our OA1 deficient animals have been slower to breed (small litter size), so we have had 12 animals to study. Supplementation has had no effect in this population, suggesting that OA1 is required. However, we believe that this result is not solid yet. The animal to animal variability encountered in this group has indicated that we will need to plan at least 12 more animals to firm up the results.

We have dissected and investigated the chiasm routing in 12 animals so far. We have developed procedures that will allow us to actually count the uncrossed axons at the chiasm, making this a rigorous experimental design. We have not generated enough samples using the same procedures to provide any comparative data.