Research Description:
The primary interest of my laboratory is vision neuroscience research to study molecular mechanisms underlying neuronal diseases and to identify new treatment strategies targeting the core molecules in the disease process. Our current focus is on age-related macular degeneration (AMD), the leading cause of severe vision loss affecting millions of individuals over 60 years of age in the US, known as, is more difficult to treat with unknown etiology. We have characterized a new genetic mutant mouse model which mimics essential features of retinal angiomatous proliferation (RAP), a newly recognized subtype of neovascular AMD. It is so far the only animal model for RAP. Using this animal model, our group is the first to identify the target cells expressing very low density lipoprotein receptor (VLDLR) in the retina and discovered an early involvement of inflammatory cytokines and macrophage activation before subretinal angiogenesis. These findings have significantly advanced our understanding of the initial step of the abnormal vessel growth in RAP and provided a reproducible animal model to facilitate studies of a potential pathway mediating RAP. Taking the advantage offered by this model, we then demonstrated that VLDLR is a potent endogenous inhibitor negatively regulating a broad spectrum of retinal endothelial cell properties, and that loss of VLDLR activates these cells and promotes angiogenesis in vitro and in vivo. We are continuing to investigate the downstream signaling molecules regulated by VLDLR so that we can better understand the molecular mechanisms of angiogenic processes in RAP. In addition to these mechanistic studies, we are using this unique model to explore translational research trying new treatment strategies to control retinal angiogenesis through collaborations.

Representative publications:
