Visual impairments are affecting millions of people worldwide, and the number of visually disabled people is increasing due to the aging of the population and chronic diseases. By 2050, 61 million people will be blind, and 474 million will have moderate and severe vision. Decrease or loss of vision impacts the lives of those affected, making daily activities difficult, leading to a loss of independence, and increasing the risk of mental diseases. That is why it is crucial to look for effective therapies that will slow disease progression and restore vision. Recently, viral gene therapies providing defective genes or optogenetic tools appeared to be the most effective ways to restore vision. It is mainly related to privileged immune status and easy accessibility for treatment delivery of the eye. The First AAV-based gene therapy that improves the vision of patients with retinal dystrophy was approved by the US FDA in 2017. There are also multiple AAV-based gene therapies focused on delivering different variants of microbial Channelrhodopsin 2 (Chr2) subjected to clinical trials. As recent clinical trials focus on opsins that perform their function in high light intensities, there is still a need to design an optogenetic tool that would be successfully activated by relatively low light stimulation. In a healthy retina, signals received from light-activated photoreceptors are processed from bipolar cells to amacrine and ganglion cells. However, in the degenerated retina, irreversible changes and mutations are causing profound loss of light-detecting rods and cones. Despite the loss of photoreceptors and structural changes, surviving cells remain functional. **This project aims to develop novel proof-of-concept chimeric opsins with exceeded functionality to mimic natural circuit mechanisms and information processing within a degenerated retina.** We propose that chimeric RecRho variants may constitute a new gene therapy strategy in retinal diseases. We hypothesize that moderate light levels can activate chimeric RecRho variants delivered to surviving cell populations within the degenerated retina, will transform transduced cells into direct light detectors, and restore high-level vision. Besides converting transduced cells into direct light detectors, we believe that our chimeric proteins will also positively impact structure and synaptic plasticity on a large scale through the whole retina. We will utilize single-neuron recordings and behavioral visual discrimination tasks to estimate visual network selectivity in response to complex and moving stimuli. Such an approach of synthetic protein with multiple functions, based on the mGluR1 transduction pathway, wasn’t proposed and used before.