Dr. Larry N. Thibos, MS, PhD, has received funding for a new project, concerning optical aberrations and myopia development. Collaborators on the project include Professor Rowan Candy, Dr. Jesson Martin (Postdoctoral Fellow), Dr. Vidhyapriya Sreenivasan (Postdoctoral Fellow), and Tao Liu (graduate student).

Myopia (nearsightedness) has a high prevalence in many regions of the world. In the USA, myopia prevalence has increased sharply over the past 30 years and now affects 33% of adults. Of greatest concern with this condition is its possible progression to high myopia (>6.00 diopters), which dramatically affects one's ability to function without optical aids. High myopia is also associated with an increased risk of retinal disease, cataract, and glaucoma. Developing contact lenses to slow or stop the progression of myopia would be of great interest and benefit to public health. However, achieving this objective requires a rigorous understanding of the optical stimuli that cause myopia progression. The source of the prevalence data is Susan Vitale, PhD, of the National Eye Institute in Bethesda, Md., December 2009 issue of Archives of Ophthalmology.

Abundant evidence suggests that accommodation to near work (focusing on close items) causes myopia to worsen, i.e. myopia progression. A small, but significant, literature further suggests that peripheral areas of the retina are involved in the visual control of myopia progression. What is unclear, however, is the causal mechanism by which these two factors might exert their influence. One major hypothesis, called "grow to clarity" suggests that eye elongation occurs when the eye has insufficient refractive power to clearly focus the retinal image on the cone photoreceptors. Under this condition of hyperopic blur, the retinal image plane lies posterior to cone apertures. If the eye has not yet achieved adult size, then it will continue to grow until axial elongation is sufficient for the retina to "catch up" with the under-powered retinal image. When that happens, growth stops because the signal to keep growing (hyperopic blur) stops. In this model, growth is an active process caused by optical defocus; stopping growth is a passive process that occurs when the active signal to grow is removed.

An alternative hypothesis suggests the opposite: stopping growth is an active process that occurs when the retinal image is habitually well focused. If the eye is underpowered, the resulting hyperopic defocus prevents the stop signal from arresting eye growth. The puzzling aspect of this model is that the stop-growing signal remains when the eye is over-powered (i.e. myopic defocus). Obviously for this hypothesis to be tenable, the retina must be able to detect the sign of defocus in order to initiate the stop-command appropriately. There are numerous clues in the retinal image that could be used to detect the sign of defocus, but none have yet been proven to be the elusive signal that controls eye growth. Our study will evaluate the possibility that the interaction of defocus with the eye's higher-order aberrations resolves the sign-of-defocus ambiguity.

The funding for this project, titled “Accommodation and Myopia,” runs from 12/01/2011 - 12/31/2012 and is provided by Vistakon, Inc., Johnson and Johnson.