As specialists in managing a chronic disease of aging, glaucoma clinicians care for their patients over many years and play an important role in many of their lives. When I recently saw a young mother of two who first came to me as a teenager with advanced glaucoma secondary to iris melanoma, I felt almost as though she were a childhood friend. Over the years, we have gone through her plaque treatment, cataract extraction, diode laser ablation, and a host of other treatments with tears and laughter. Witnessing her remarkable journey humbly reminds me how satisfying it is to be a clinician.

I also derive great personal satisfaction from my scientific research. The fast-paced learning of residency and fellowship continues, as I stay up to date on the latest basic scientific research, formulate new questions and experimental approaches, and analyze my own data in addition to caring for patients. As a clinician, I wonder why some patients’ disease worsens when their IOPs are low. As a scientist in the laboratory, I have developed a line of mice that has glaucoma without high IOP to try to address this question. Are the mechanisms of retinal ganglion cell degeneration fundamentally different in so-called low-tension glaucoma than in glaucoma with high IOP? I hope that, by answering this long-standing question, my research contributes to the development of treatments for glaucoma independent of lowering IOP. If I am able to achieve this goal, I like to think that, in the long run, my research effort will benefit a much larger number of patients than I could possibly serve in the clinic.

As a clinician, I ask about every patient’s family history of glaucoma. As a scientist, I appreciate that this genetic predisposition offers a great opportunity for discovery by letting genetics point to potentially unanticipated mechanisms. In my recent research, this approach proved powerful when my colleagues and I identified a novel glaucoma gene in a colony of dogs that carry inherited glaucoma. The gene, ADAMTS10, is involved in the function of microfibrils, which are nanometer-scale structures in the extracellular matrix that both contribute to tissue elasticity and control localization and activation of transforming growth factor beta. Based on this finding together with previous studies implicating other microfibril-associated genes, we have formed a new hypothesis that microfibril deficiencies cause glaucoma. My research is currently focused on testing this hypothesis using mice with microfibril deficiencies and investigating new approaches to glaucoma therapy that are known to be effective in treating other microfibril deficiencies in humans. My colleagues and I are excited that the microfibril-deficient mice have low-tension glaucoma manifested with retinal nerve fiber layer thinning and retinal ganglion cell loss. Although finding data consistent with our microfibril hypothesis is exhilarating, understanding the mechanism and trying new treatments will be even more exciting and clinically relevant.

It gives me great pleasure to share my motivation and experiences with my peers, especially those considering careers as clinician-scientists in glaucoma. Although I must acknowledge the difficulty of maintaining both a busy clinical practice and conducting research, the choice I made has proven to be highly rewarding.

Section Editor Tony Realini, MD, MPH, is an associate professor of ophthalmology at West Virginia University Eye Institute in Morgantown. Dr. Realini may be reached at (304) 598-6884; realinia@wvuh.com.

Rachel Kuchtey, MD, PhD, is an associate professor of ophthalmology and visual sciences at Vanderbilt University in Nashville, Tennessee. Dr. Kuchtey may be reached at (615) 936-7190; rachel.w.kuchtey@vanderbilt.edu.

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