Glaucoma is a leading cause of irreversible blindness due to progressive degeneration of retinal ganglion cells (RGCs) and their axons. Many risk factors associated with glaucoma have been identified, including exfoliation syndrome (XFS), a systemic condition with elastic fiber defects. The identification of single nucleotide polymorphisms within Lysyl Oxidase-Like 1 (LOXL1) that are associated with XFS and glaucoma secondary to XFS (XFG) were landmark discoveries in glaucoma research. LOXL1 is responsible for crosslinking elastin, which serves as the core of elastic fibers. Microfibrils surrounding the elastin core are equally important in terms of maintaining the stability and elastic strength of elastic fibers. Microfibrils are principally composed of fibrillin-1 encoded by the FBN1 gene. Functional relationships between fibrillin-1 and LOXL1 have been increasingly recognized. Studies have shown concomitantly reduced levels of LOXL1 and fibrillin-1 in the lamina cribrosa of human patients with XFS, although the precise mechanisms causing these reductions remain unknown. In this talk, I will present ocular structural and biomechanical changes in our mouse models carrying different mutations, including our newly created model with dual defects of fibrillin-1 and LOXL1. The objective of this talk is to stimulate scientific discussions for further investigation and potentially new development of treatment strategies for glaucoma.