Fibrillin-1, versican, and hyaluronan form a unique complex in the ciliary non-pigmented epithelium. Wagner vitreoretinopathy is a vitreoretinal degeneration inherited as an autosomal dominant trait. Interestingly, the disease manifests in only eye. Affected individuals present with an ‘empty’ vitreous cavity with fibrillar condensation or avascular strands and veils but peripheral traction retinal detachments usually becomes most problem. Wagner syndrome can be caused by heterozygous mutation in the gene encoding versican. The fibrillin-versican-hyaluronan complex and its cleavage products may be indispensable for the physiological properties important to the ciliary body and vitreous body. Furthermore, disruptions of the non-pigmented ciliary epithelial cells, constituents of the blood–ocular barrier (BOB), are clinically recognized as uveitis. Uveitis is often associated with increased tumor necrosis factor-alpha (TNF-α) in the serum and aqueous humor. The expression of MMP-1, MMP-3, and MMP-9 increased in the presence of TNF-α in non-pigmented ciliary epithelial cells. These MMPs degraded claudin-1 and occludin, essential components of the tight junctions in non-pigmented ciliary epithelial cells, which increased permeability through the cellular barrier. Infliximab effectively attenuated the TNF-α-induced increases in MMP expression in cells that comprised the BOB. These findings may provide a basis for the clinical prevention of uveitis by infliximab.