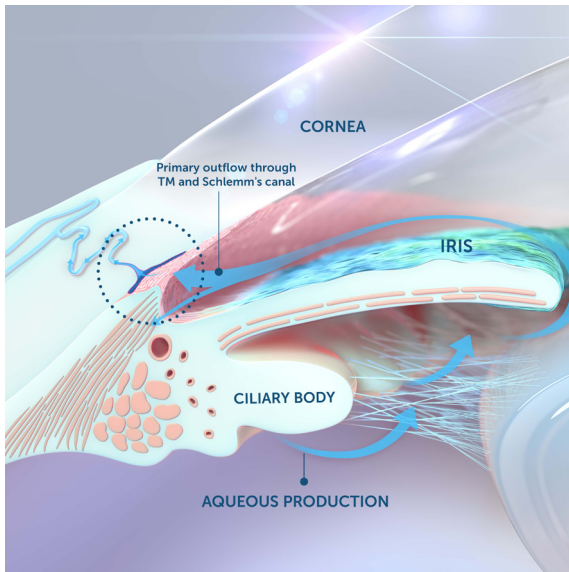


# IOP HOMEOSTATIC FEEDBACK LOOP

**CLINICAL AND SCIENTIFIC DATA POINT TO NITRIC OXIDE** as an endogenous regulator of conventional aqueous humor outflow that may also impact intraocular pressure (IOP).<sup>1-7</sup>

 NITRIC OXIDE     INACTIVE eNOS     ACTIVE eNOS     ENDOTHELIN-1

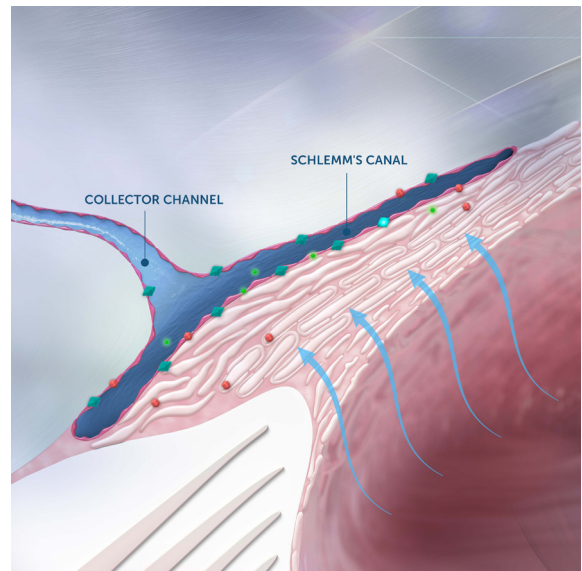


## CONVENTIONAL OUTFLOW PHYSIOLOGY

The trabecular meshwork (TM) is a dynamic, 3-dimensional network that senses and regulates conventional aqueous outflow and IOP. The majority of outflow resistance is generated within the TM and the inner wall of Schlemm's canal. Additional resistance may be generated at distal sites, including collector channels and episcleral veins.<sup>2,8-10</sup>

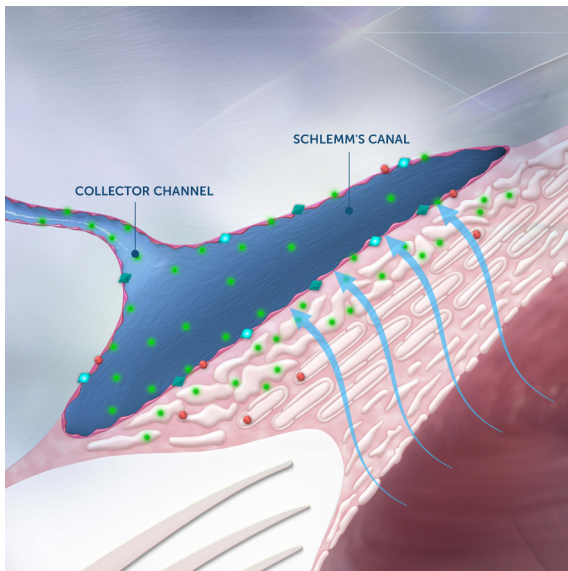
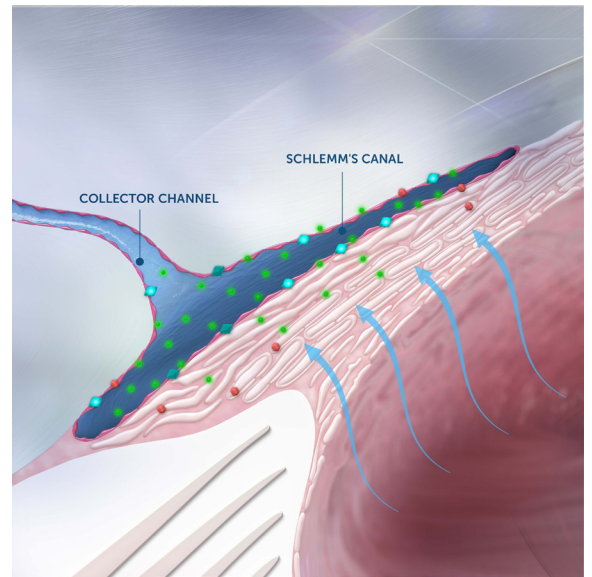
## ELEVATED IOP

When IOP is elevated, the TM pushes outward toward Schlemm's canal, producing a mechanical stretch that narrows the canal and increases shear stress to levels that are comparable to those in large arteries.<sup>11</sup>



## eNOS ACTIVATION AND NITRIC OXIDE PRODUCTION

Within seconds, increased stretch and shear stress in Schlemm's canal triggers activation of endothelial nitric oxide synthase (eNOS) and nitric oxide production.<sup>11</sup>



## RESTORED HOMEOSTASIS

Nitric oxide diffuses bidirectionally throughout the conventional outflow pathway to induce<sup>11</sup>:

1. Relaxation of the TM
2. Dilation of distal vessels
3. Increased permeability of Schlemm's canal inner wall

The result is increased aqueous outflow and reduced IOP.



Explore additional resources on how nitric oxide plays a pivotal role in ocular health and disease.

Discover more at [GlaucomaHorizons.com](https://www.GlaucomaHorizons.com)

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