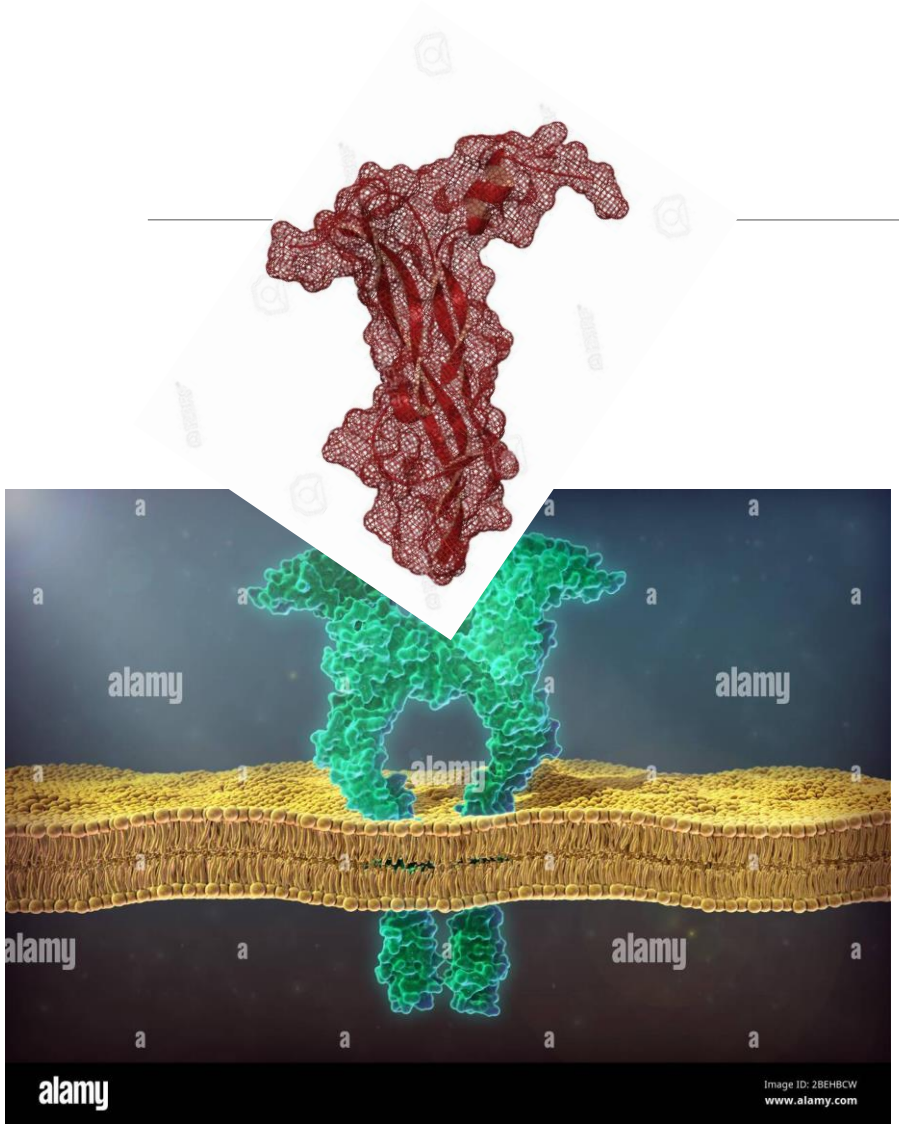
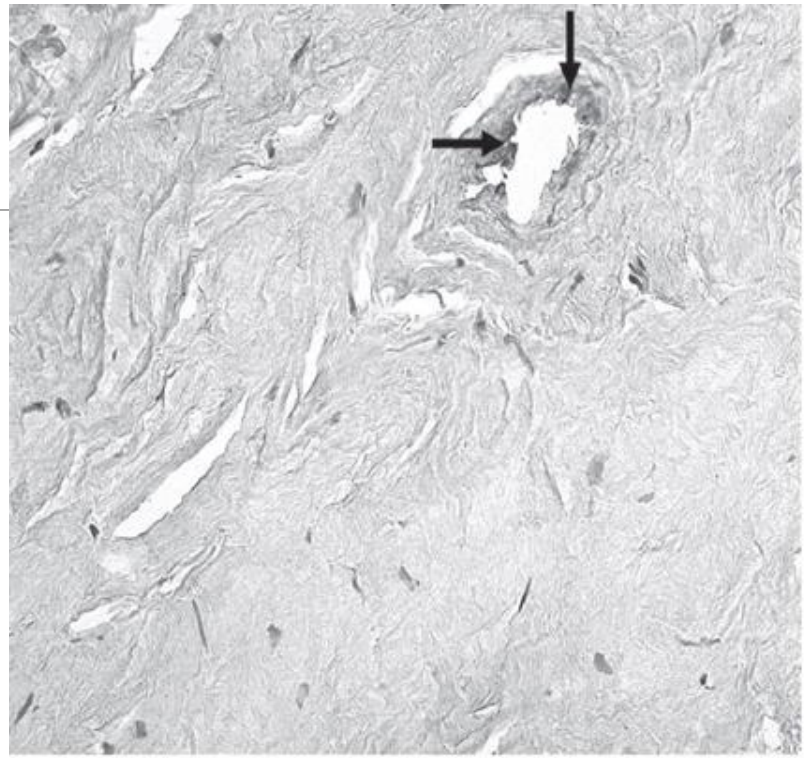
GESTIONE DELL'EDEMA MACULARE NEL PAZIENTE GLAUCOMATOSO



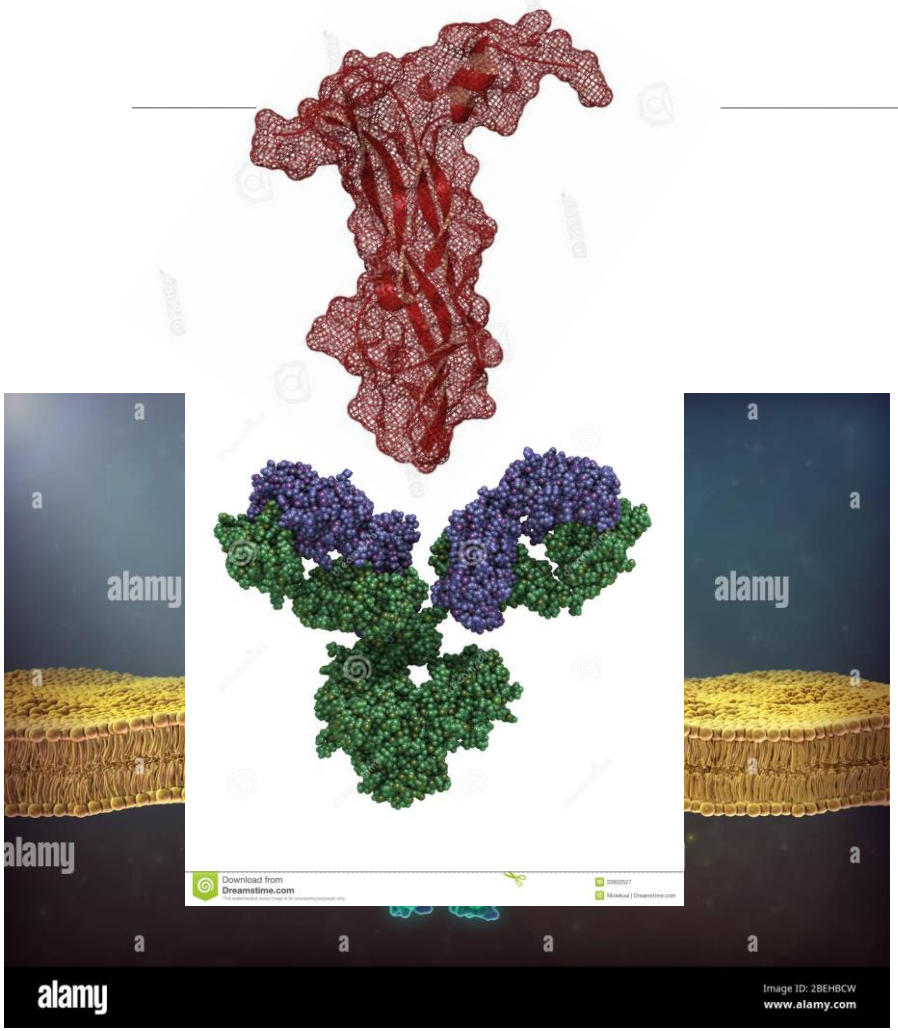


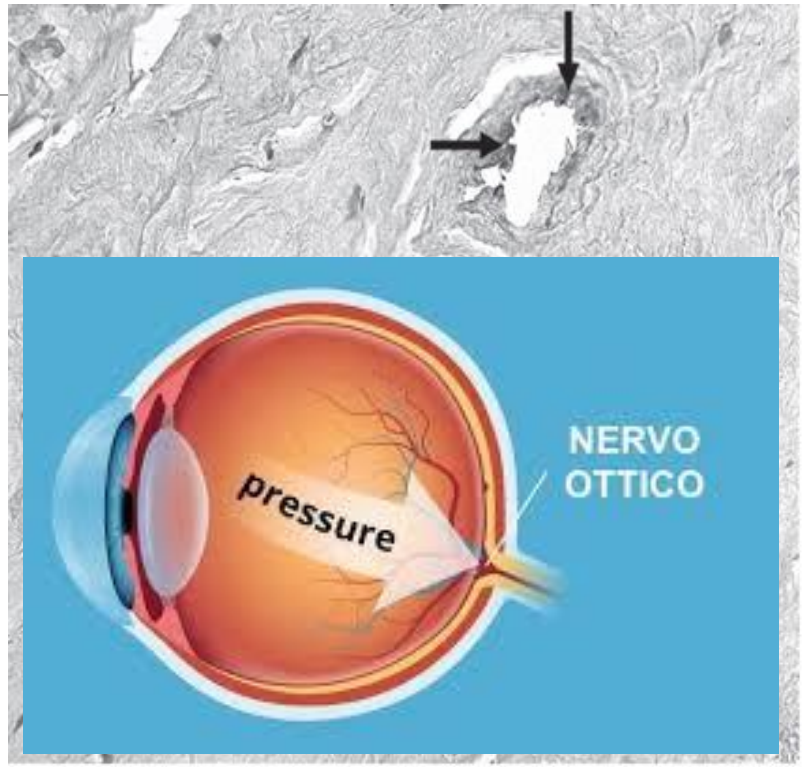
UNIVERSITÀ DEGLI STUDI DI FERRARA
- EX LABORE FRUCTUS -

Dott. Giuseppe Lamberti
U.O. Oculistica
Dir: Prof. Marco Mura

GESTIONE DEL GLAUCOMA NEL PAZIENTE CON EDEMA MACULARE

Dott. Giuseppe Lamberti





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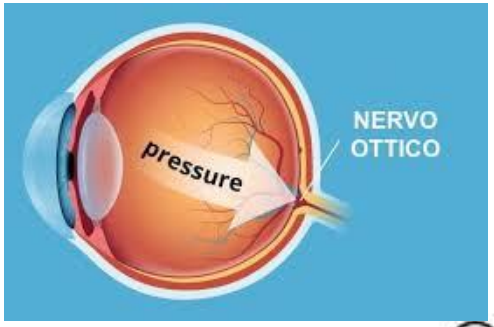
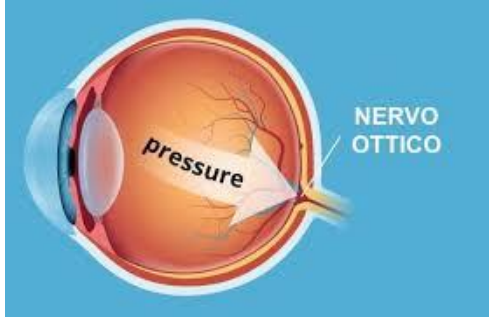




Retinal pigment epithelium
Glial cells (Müller cells and microglia)

Pro-inflammatory cytokines (eg, IL-1β, TNFα, and IL-6)

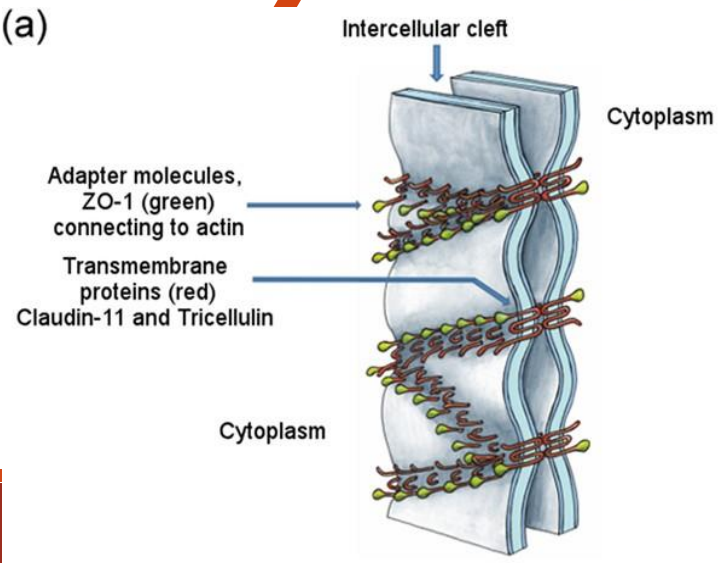
VEGF

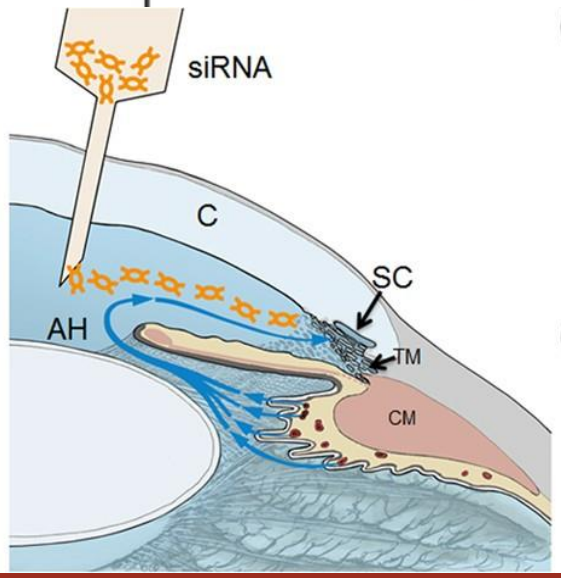
Intravitreal corticosteroids

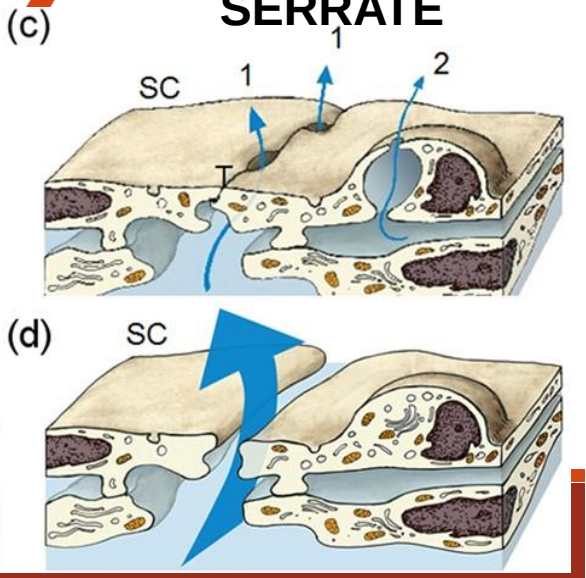
Anti-VEGF agents

Breakdown of the blood–retinal barrier

CONTROLLO APERTURA GIUNZIONI SERRATE



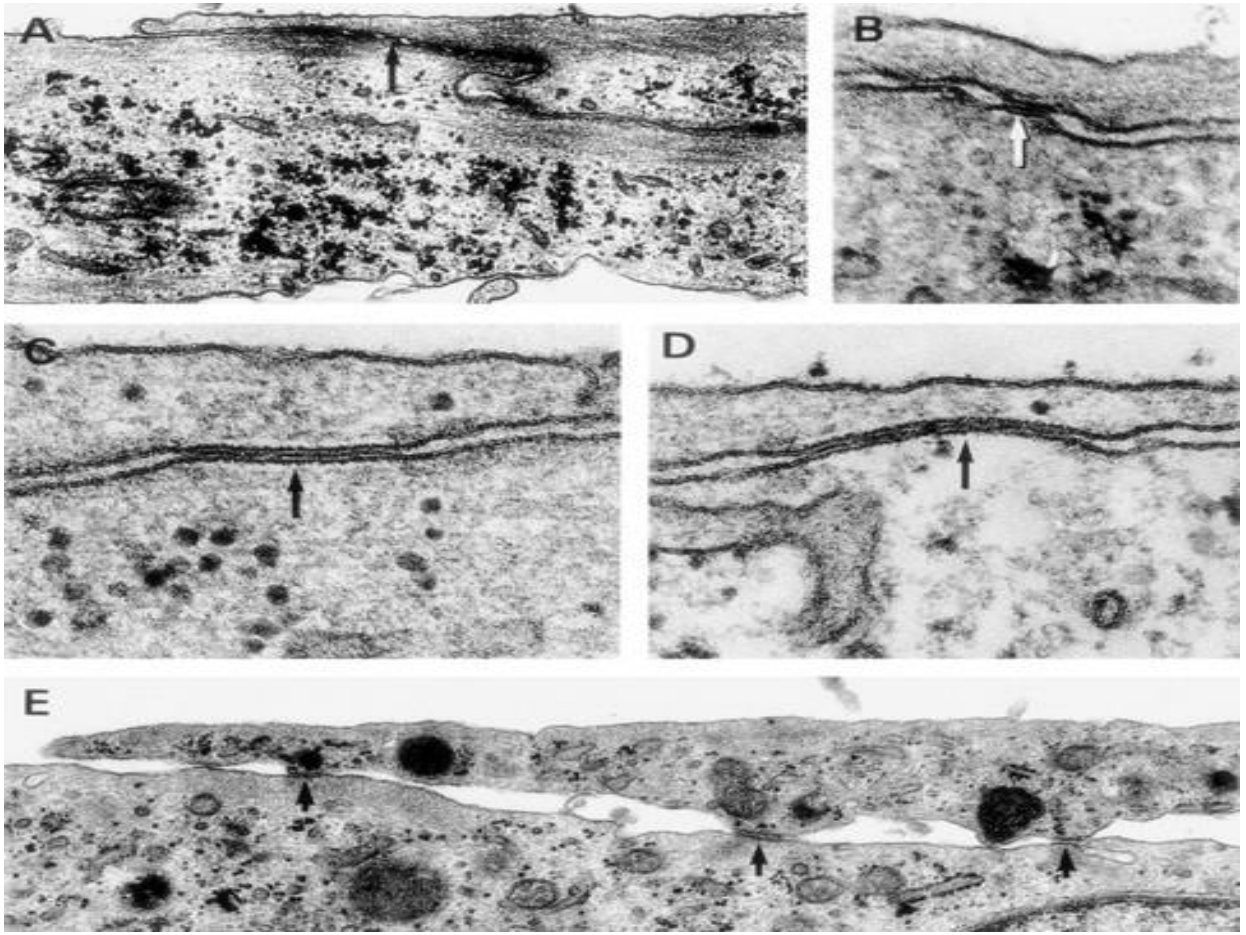




Glucocorticoids regulate transendothelial fluid flow resistance and formation of intercellular junctions

Johnnie L. Underwood

,

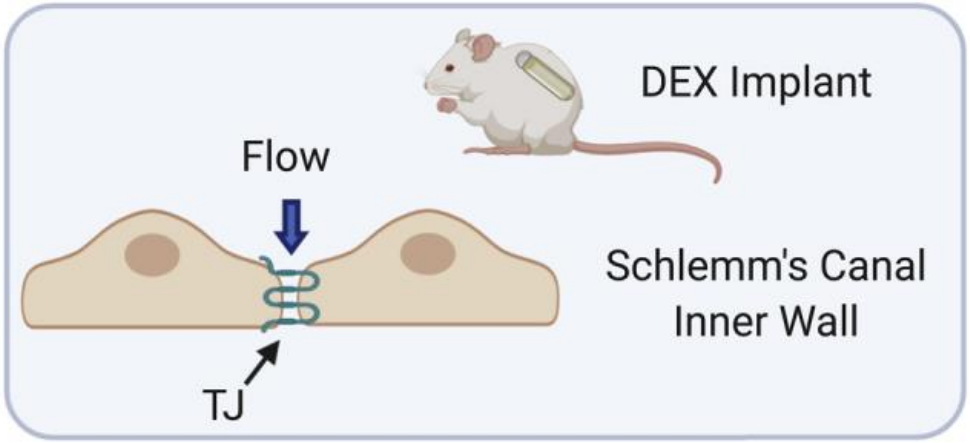


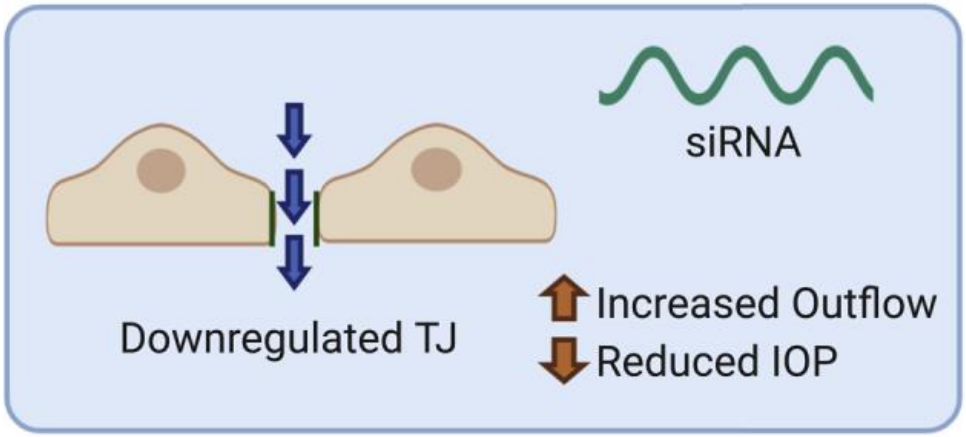
CONTROLLO
APERTURA
GIUNZIONI
SERRATE

DEX

NO DEX

GC treatment can have a range of effects on the outflow pathway, including altering TM cell functions, extracellular matrix metabolism, and gene expression, which may be responsible for the increase in outflow resistance associated with GC treatment.





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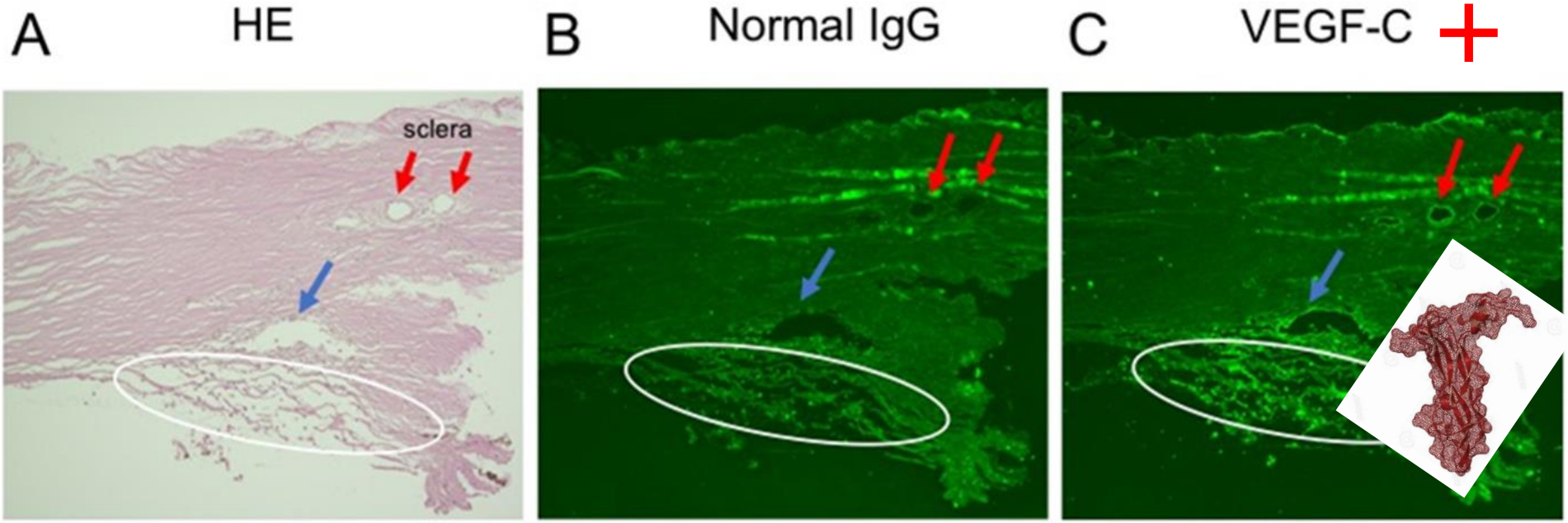
SERRATE

GESTIONE DELL'EDEMA MACULARE NEL PAZIENTE GLAUCOMATOSO

Article

Expression of Vascular Endothelial Growth Factor-C in the Trabecular Meshwork of Patients with Neovascular Glaucoma and Primary Open-Angle Glaucoma

J. Clin. Med. **2021**, 10, 2977



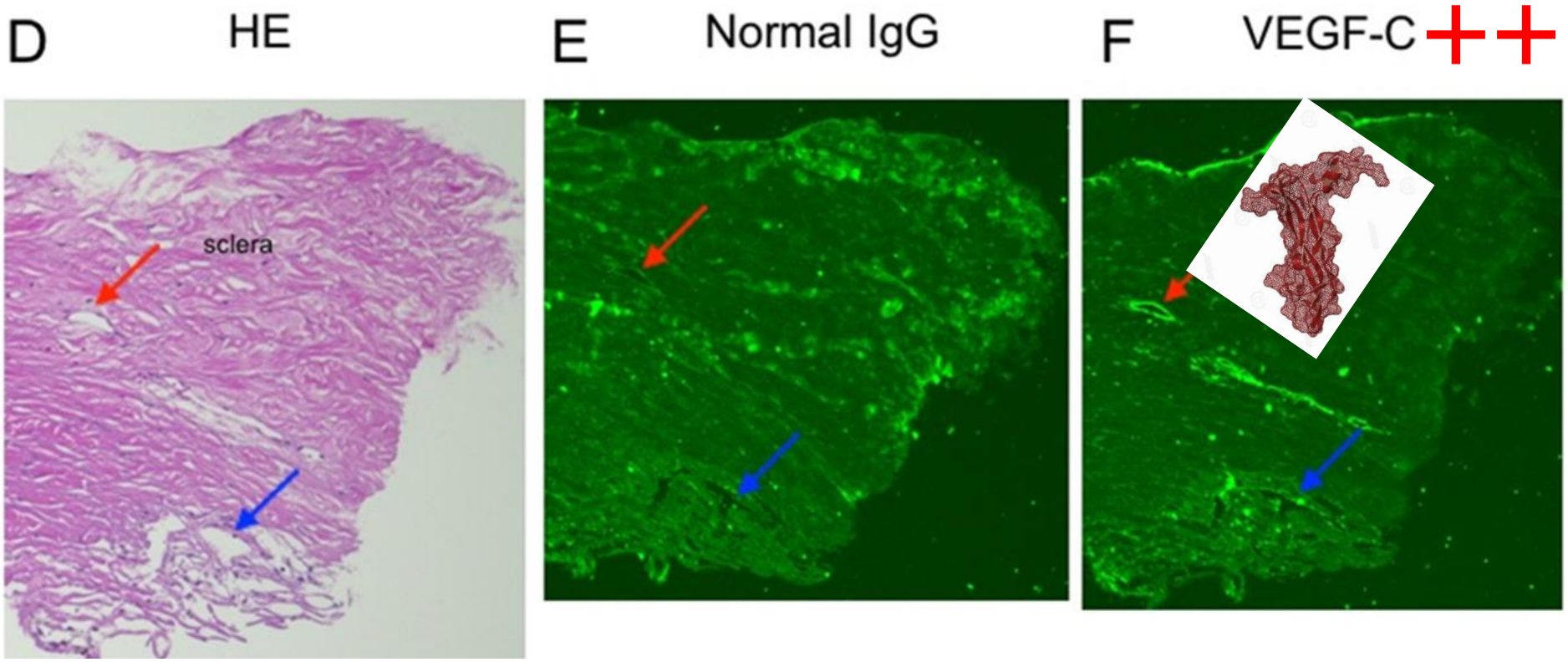
GLAUCOMA NEOVASCOLARE

GESTIONE DELL'EDEMA MACULARE NEL PAZIENTE GLAUCOMATOSO

Article

Expression of Vascular Endothelial Growth Factor-C in the Trabecular Meshwork of Patients with Neovascular Glaucoma and Primary Open-Angle Glaucoma

J. Clin. Med. **2021**, 10, 2977



POAG

GESTIONE DELL'EDEMA MACULARE NEL PAZIENTE GLAUCOMATOSO

Article

Expression of Vascular Endothelial Growth Factor-C in the Trabecular Meshwork of Patients with Neovascular Glaucoma and Primary Open-Angle Glaucoma

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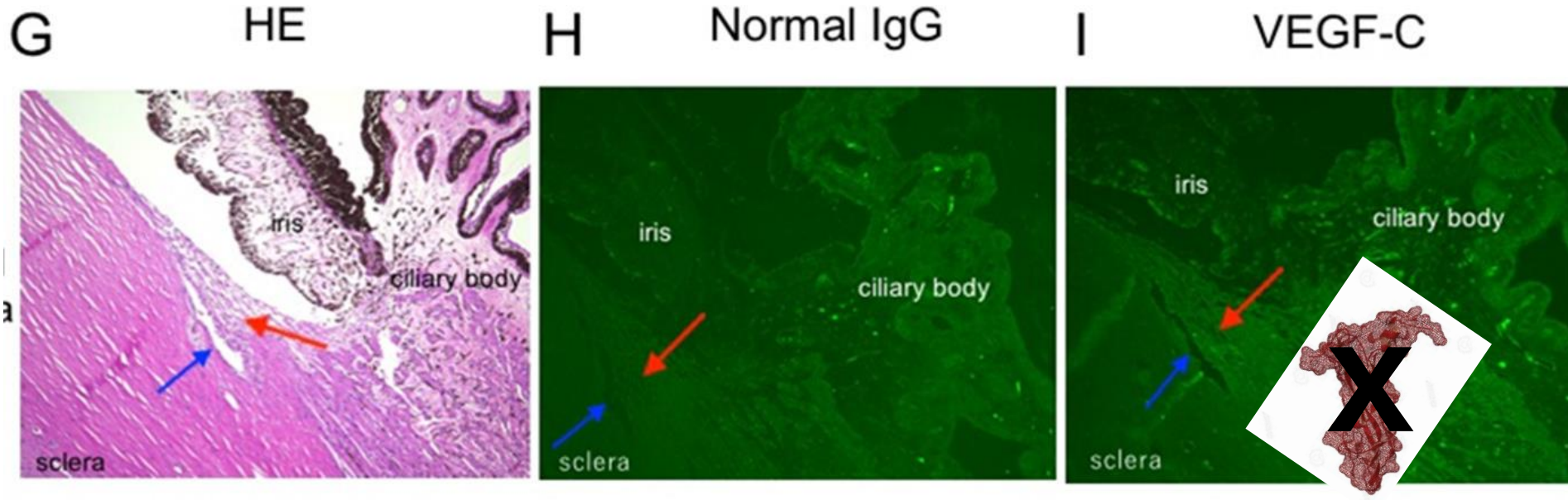
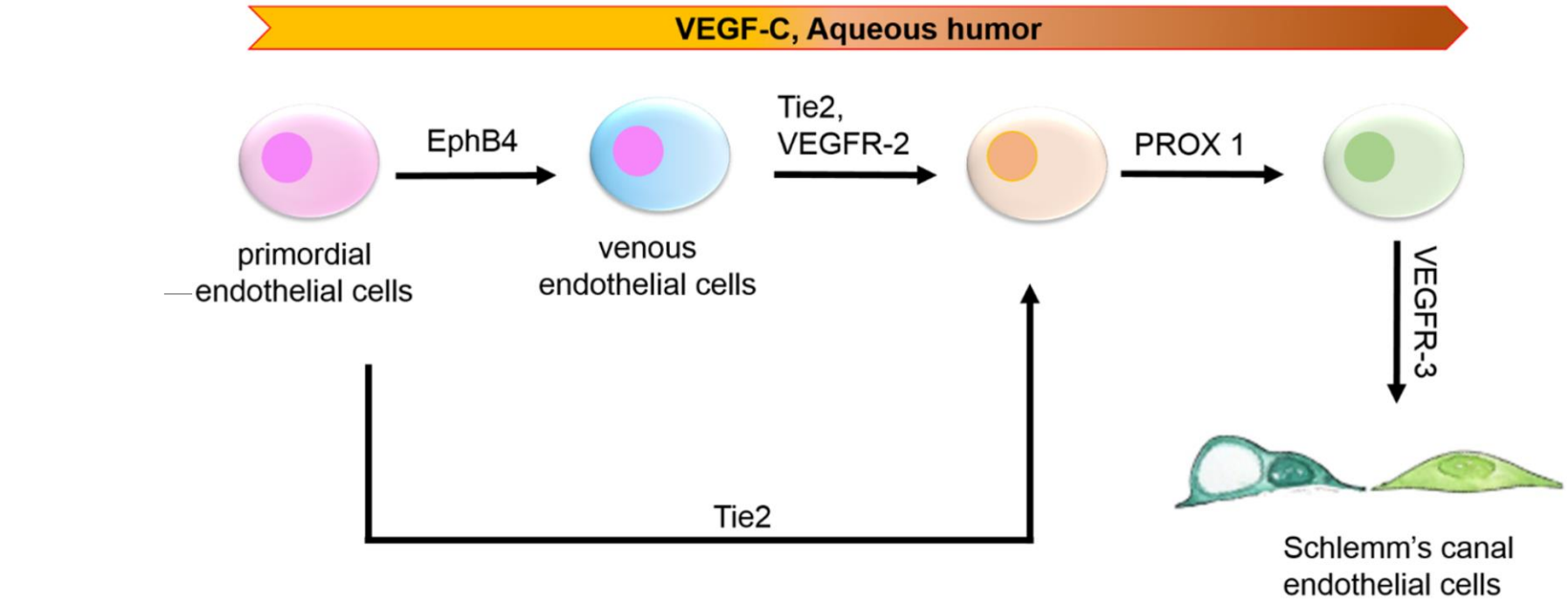
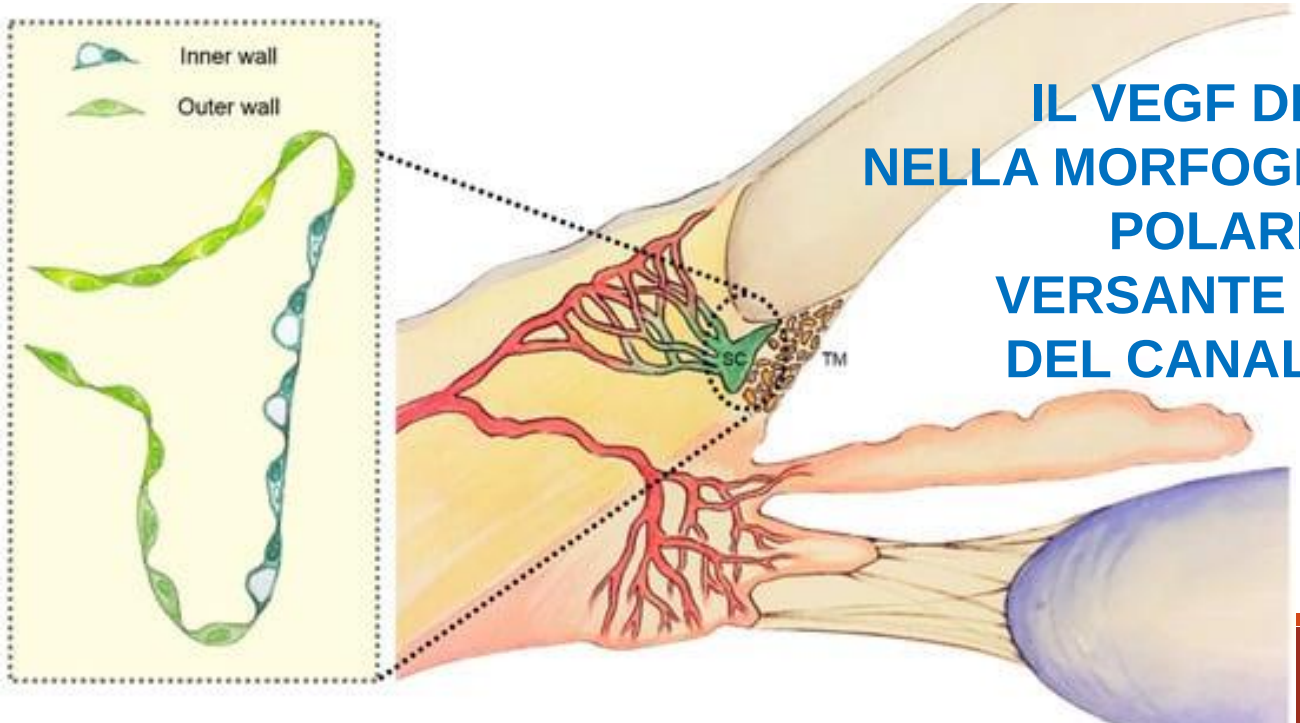
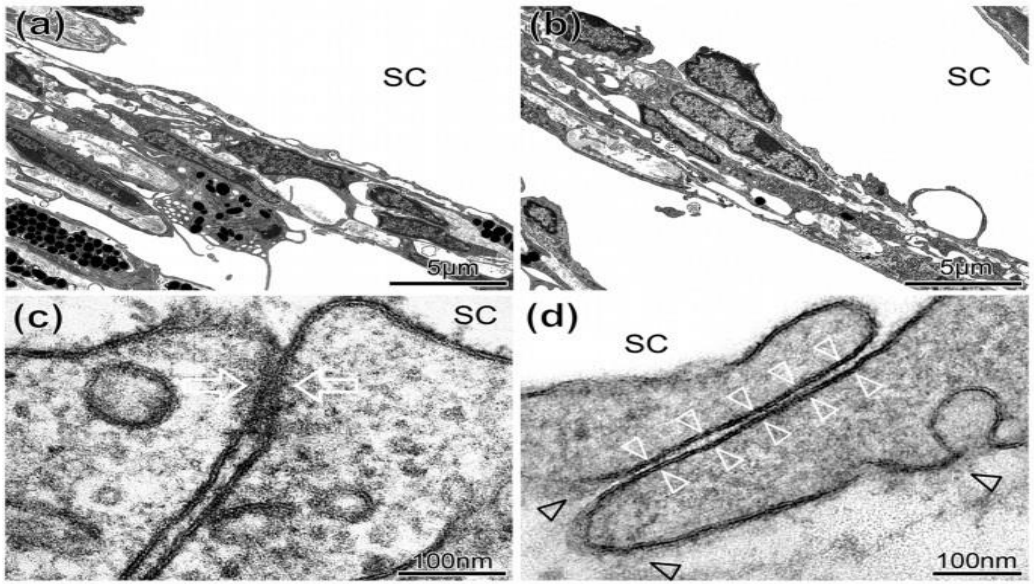


NO GLAUCOMA



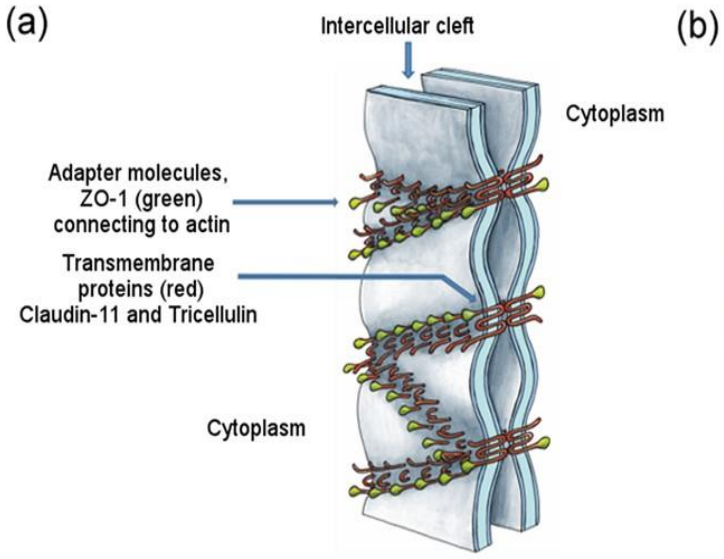


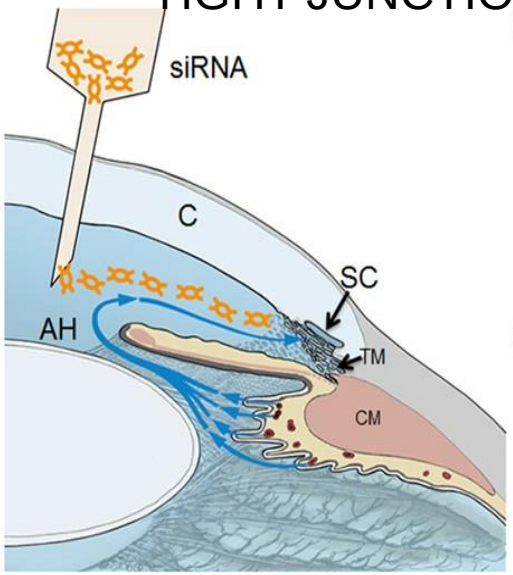
IL VEGF DETERMINANTE NELLA MORFOGENESI ENDOTELIALE POLARIZZATA DEL VERSANTE TRABECOLARE DEL CANALE DI SCHLEMM

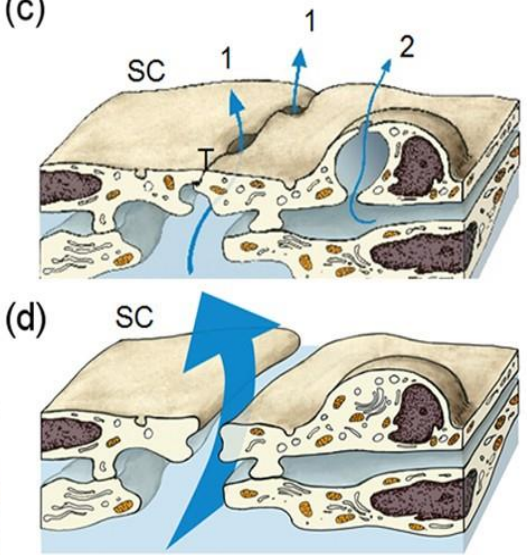


Mechanistically, chronic shear stress regulates eNOS activity through two pathways; regulation of eNOS transcription and contrl of eNOS mRNA stability. In response to the onset of shear stress, transcription of eNOS is transiently upregulated by NFkB activation and p50/p65 binding to a GAGACC sequence in the eNOS promoter at the shear stress responsive element (SSRE)[3]

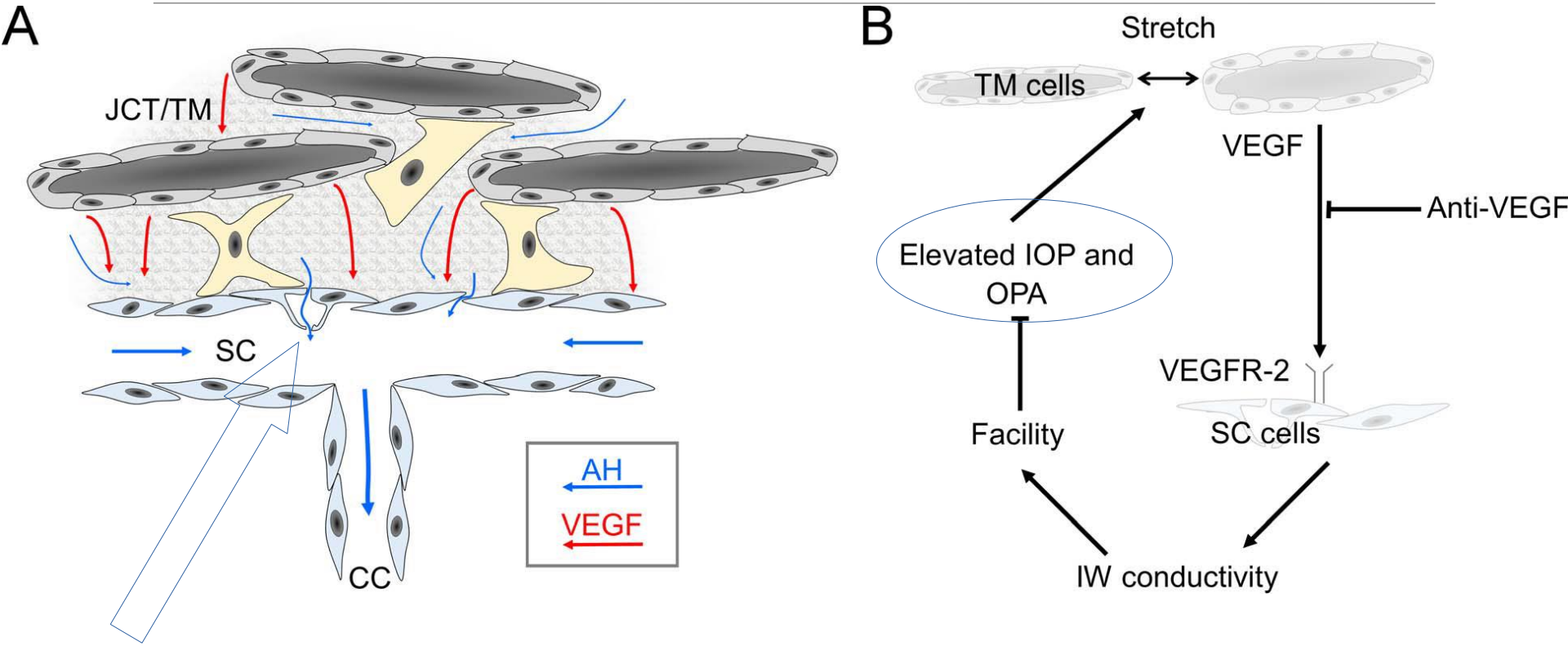
TIGHT JUNCTION DOWN REGULATION





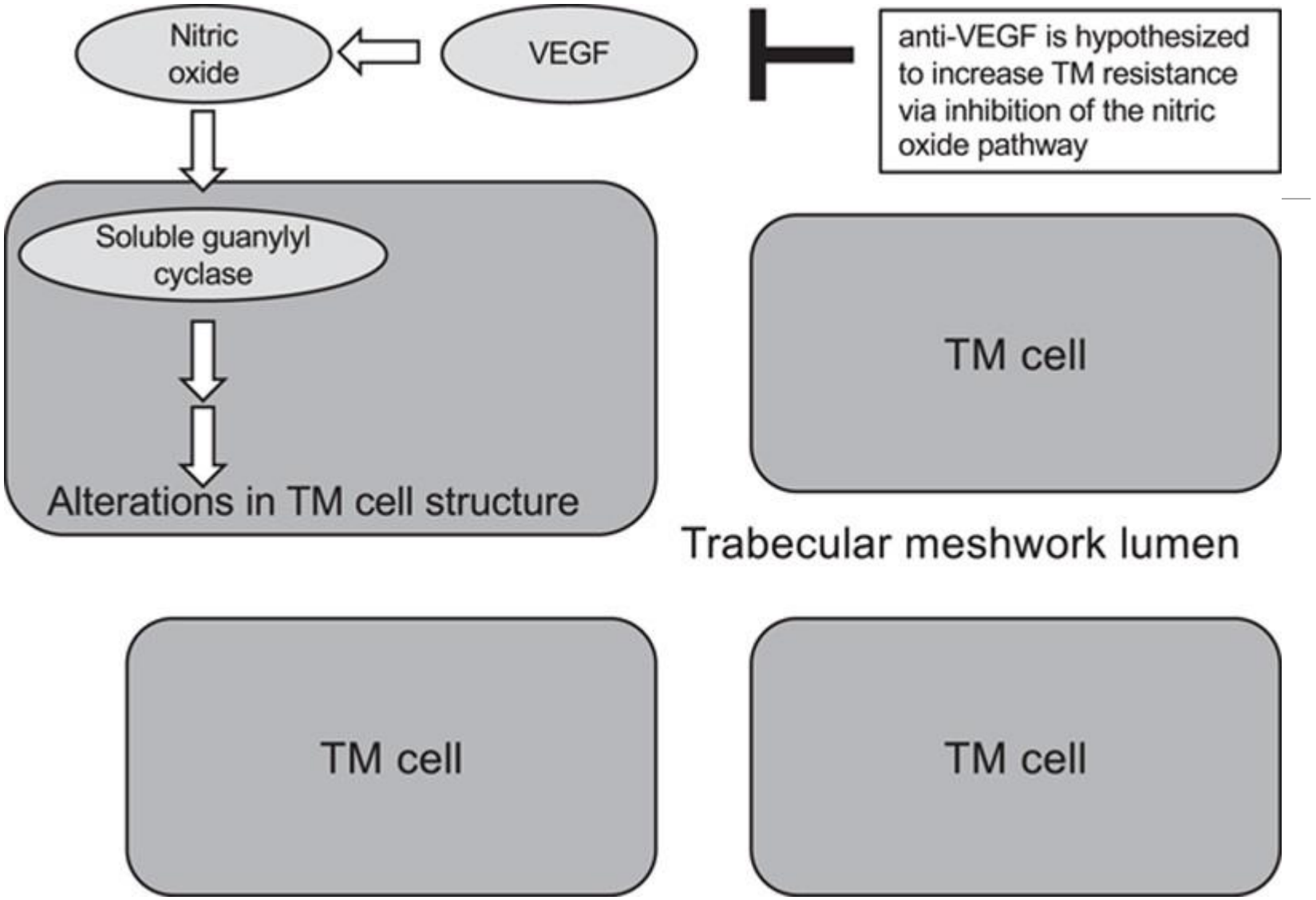


VEGF RIDUCE LA RESISTENZA AL DEFLUSSO PRIMARIO TRABECOLARE

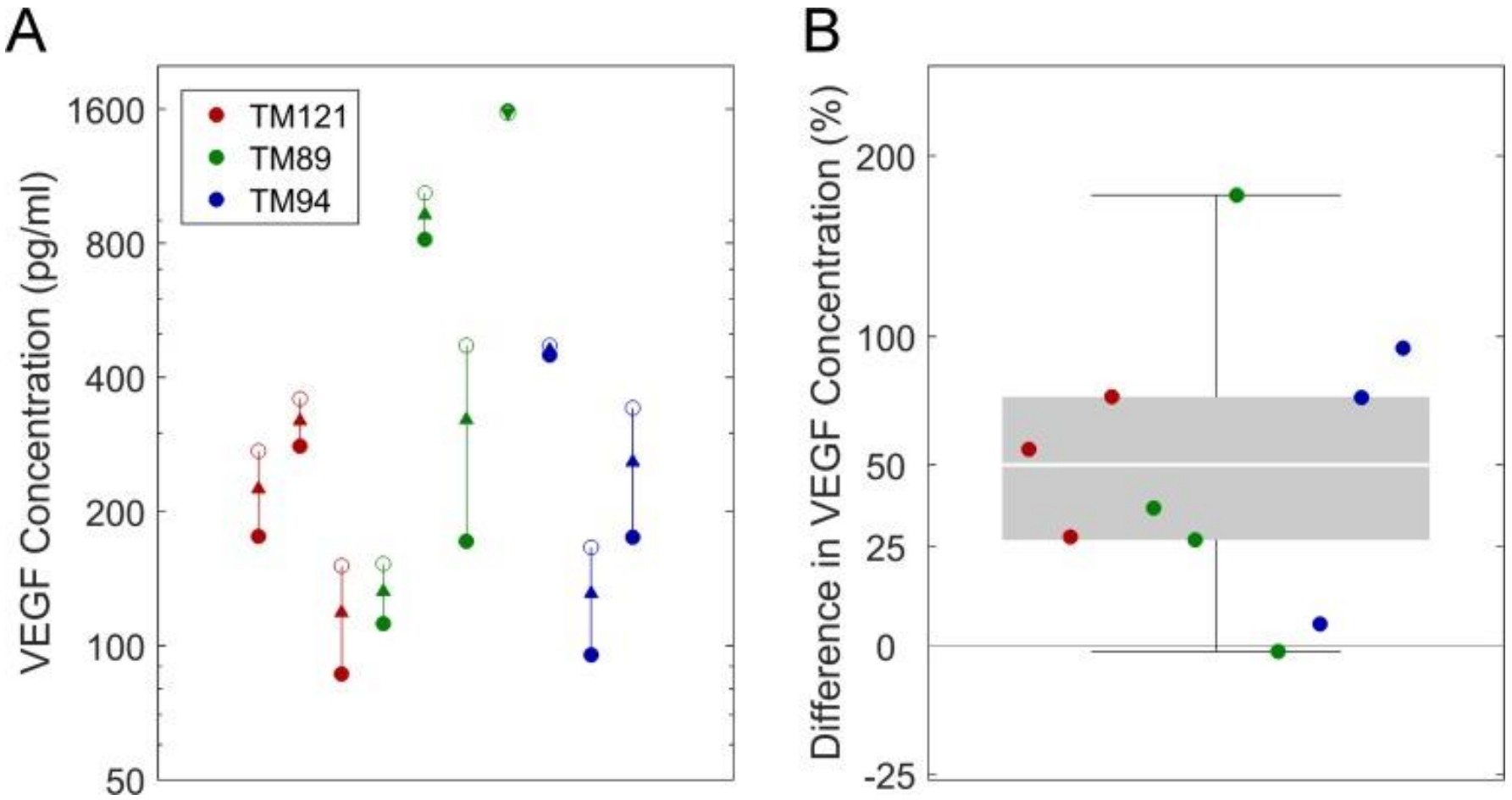


VEGF as a Paracrine Regulator of Conventional Outflow Facility

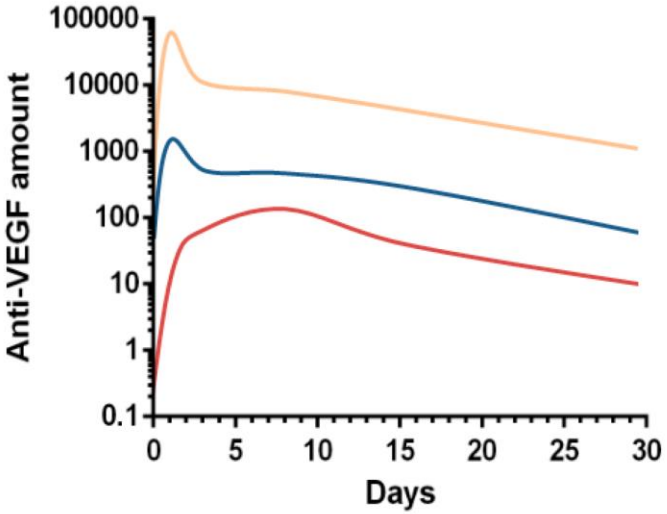
Ester Reina-Torres,

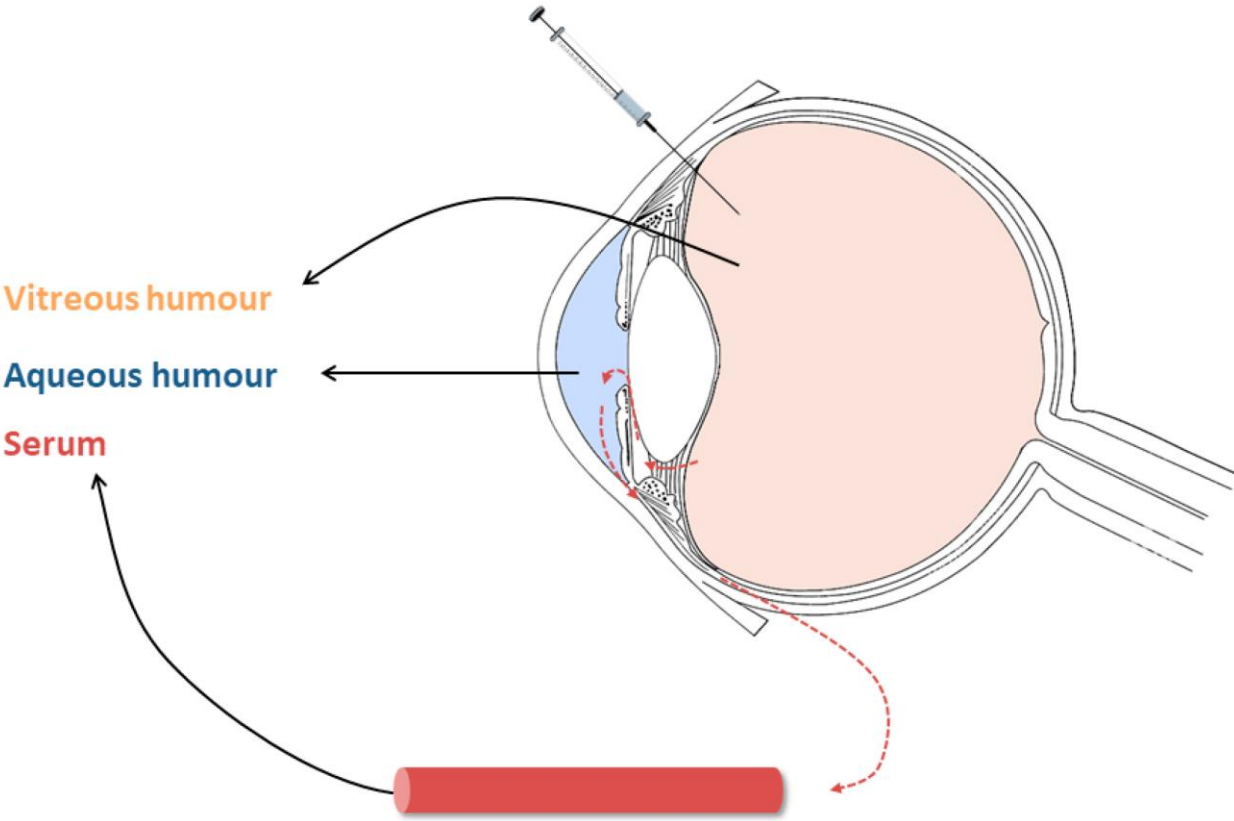

Nitric oxide
VEGF
anti-VEGF is hypothesized to increase TM resistance via inhibition of the nitric oxide pathway
Soluble guanylyl cyclase
Alterations in TM cell structure
TM cell
Trabecular meshwork lumen
TM cell
TM cell

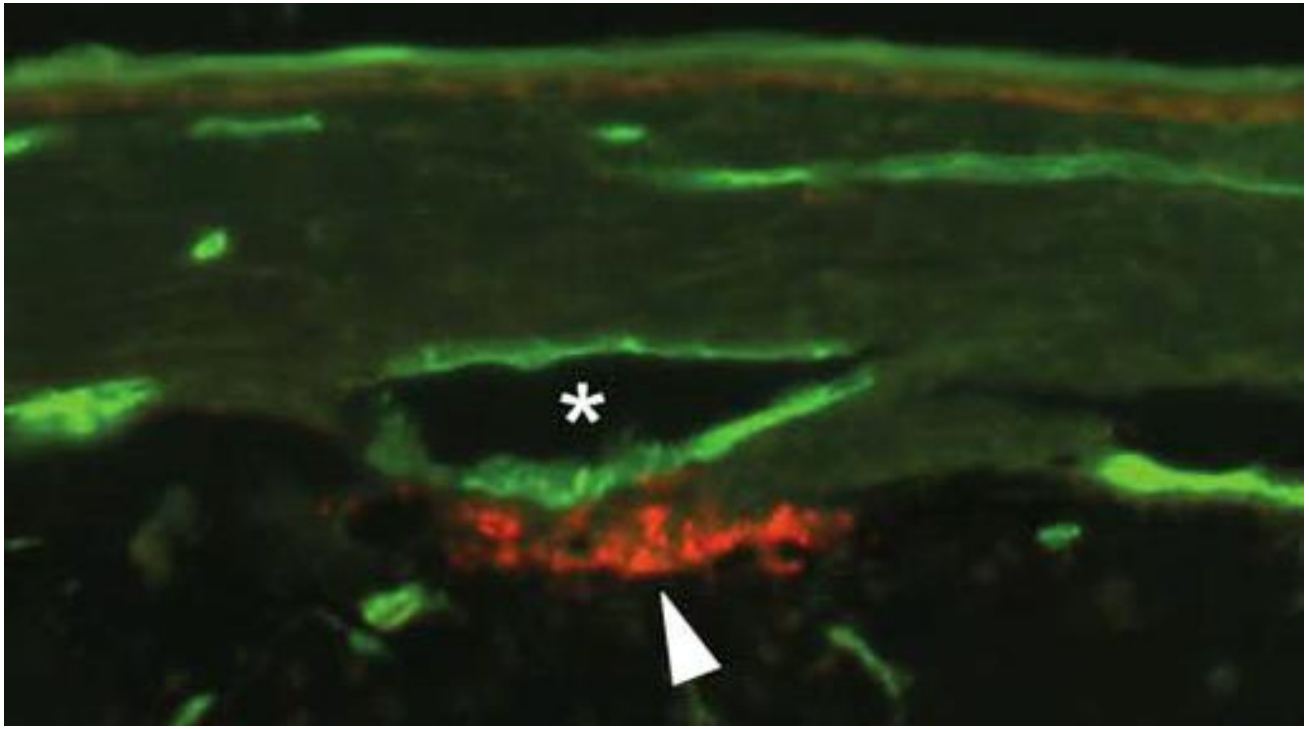
VEGF-A Is Secreted by TM Cells in Response to Mechanical Stretch



ANTI VEGF

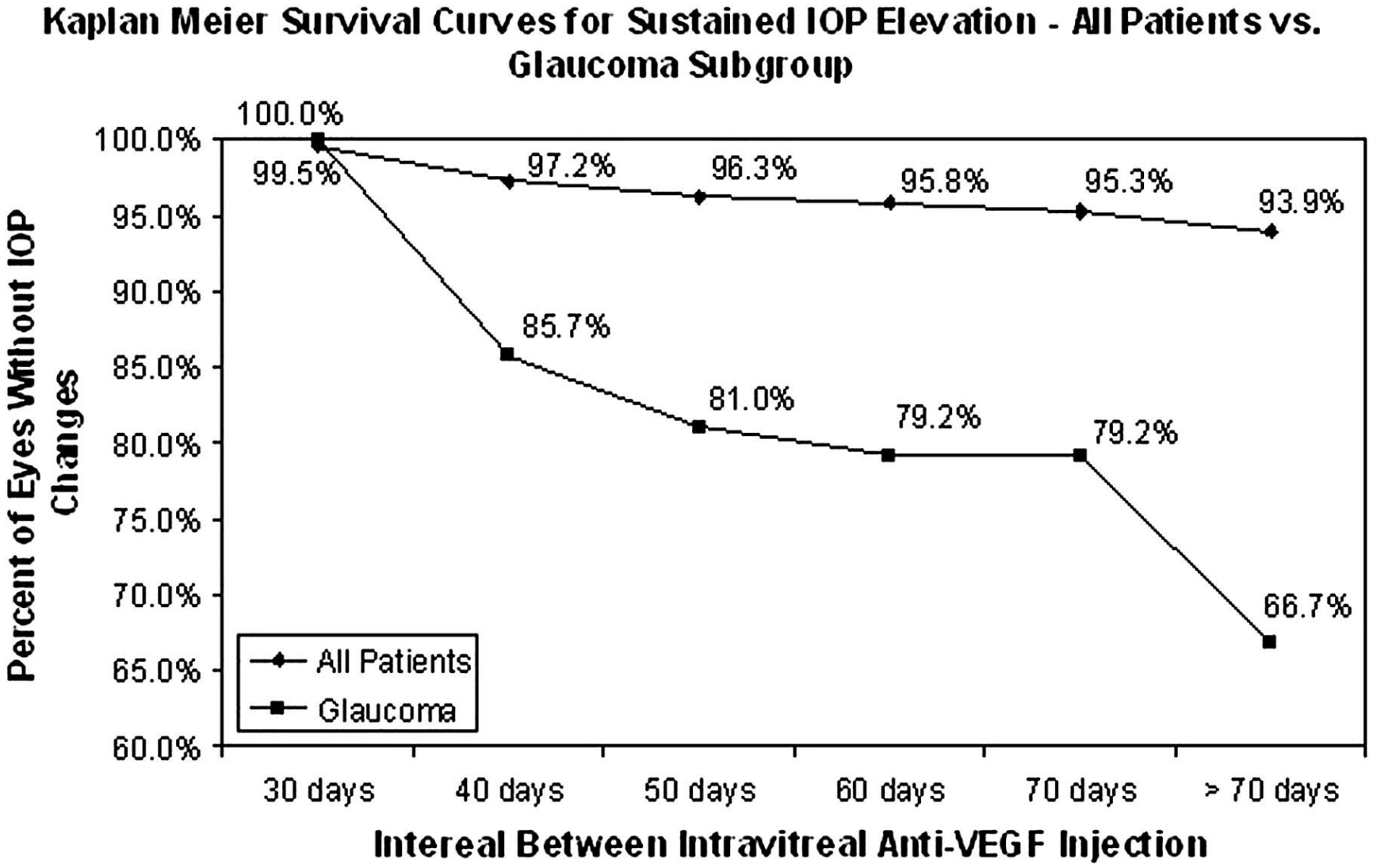

Anti-VEGF amount
100000
10000
1000
100
10
1
0.1
0
5
10
15
20
25
30
Days

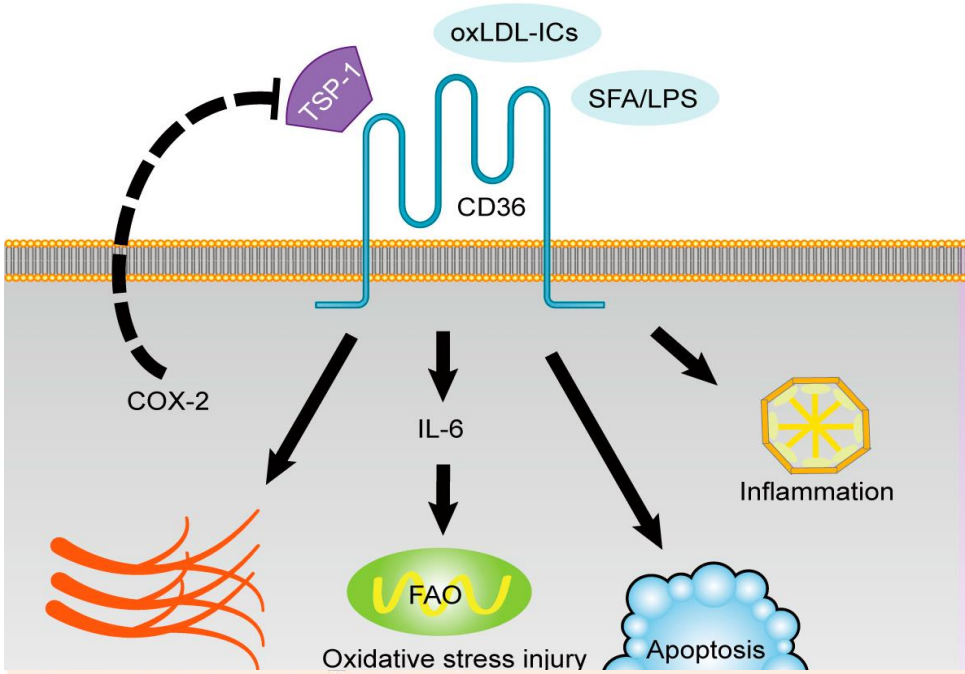

Vitreous humour
Aqueous humour
Serum

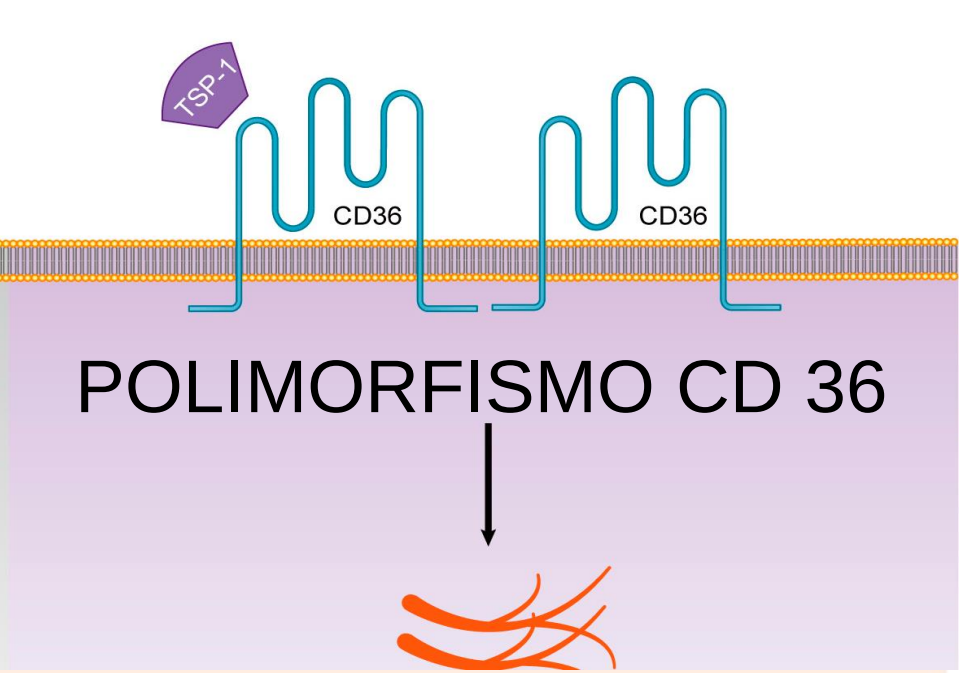


ANTI VEGF

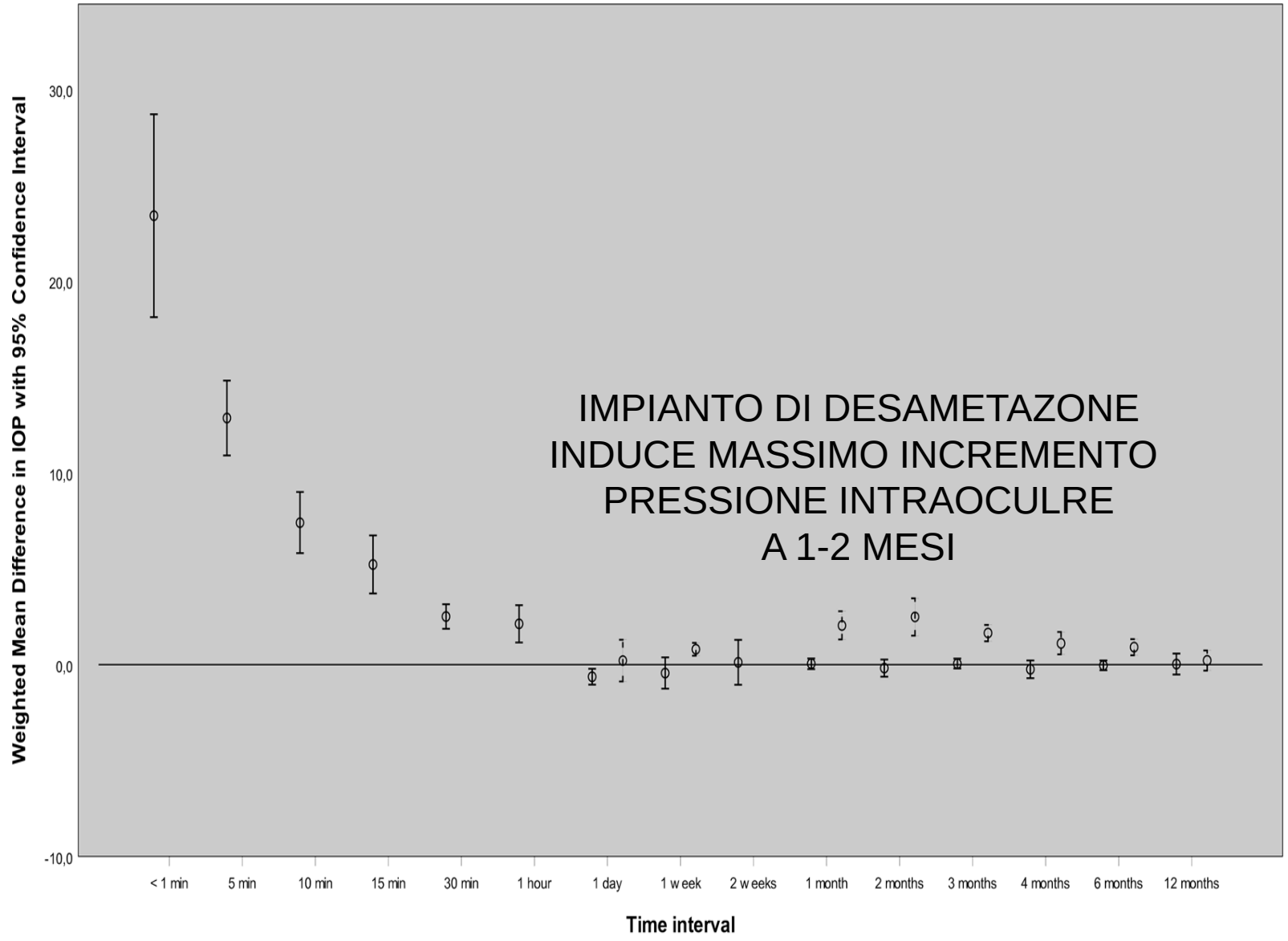
I PAZIENTI GLAUCOMATOSI PRESENTANO UNA CRITICITA' CLINICA SPECIFICA





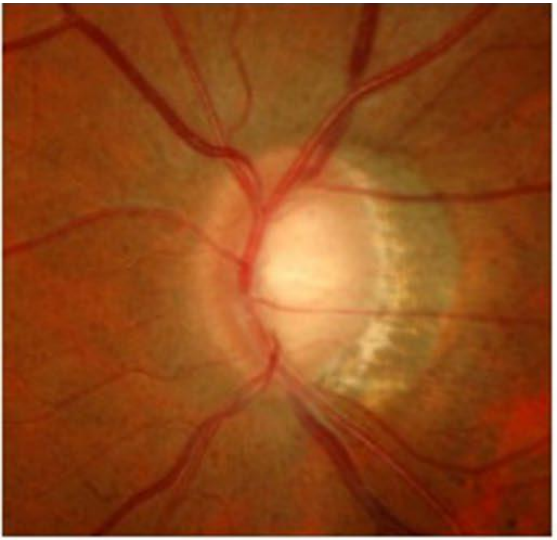


- Immediately following an injection of ranibizumab, nearly 90 percent of eyes have been reported to have an intraocular pressure of > 30 mmHg.
- A recent analysis of the IRIS registry found a small and probably not clinically significant decrease in IOP from anti-VEGF injections over time, but a small percentage of patients had an IOP increase that could be clinically significant.
- Research has suggested that the rise in IOP may be related to anti-VEGF interaction with nitric oxide physiology and/or three polymorphisms of the CD36 gene.
- Further clinical studies are needed to better understand the mechanism responsible for chronic IOP increases after anti-VEGF injections.

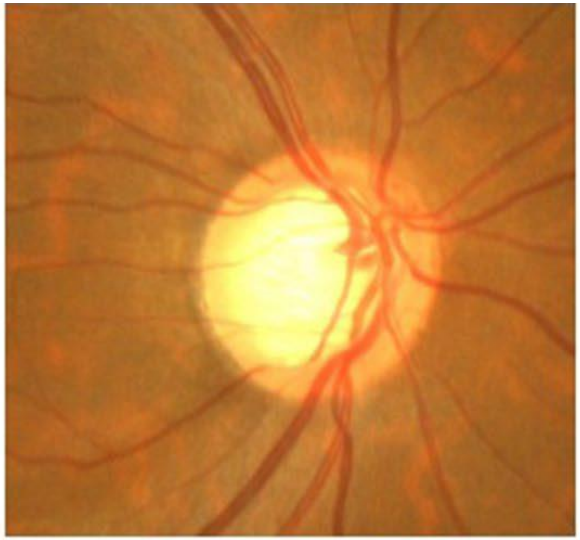


N (studies)	15	11	10	6	19	7	18	6	10	4	5	17	21	5	8	16	16	4	5	15	20	4	7
N (eyes)	943	388	562	163	1134	368	1196	263	411	613	248	562	1535	225	869	546	1291	208	166	594	1496	229	974

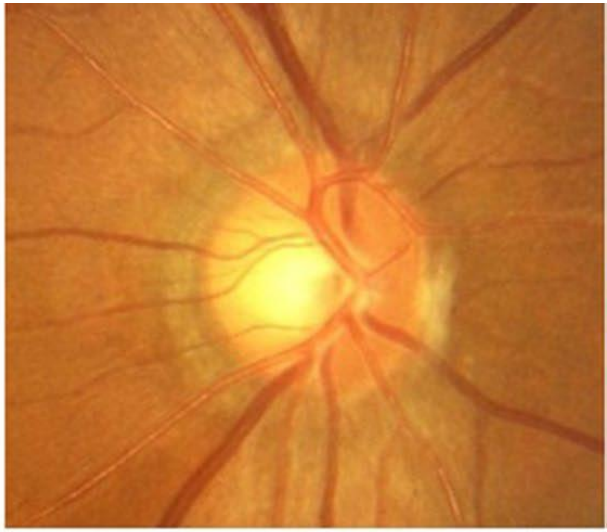
MONITORAGGIO MORFOSTRUTTURALE



Myopic glaucomatous (MG) disc type



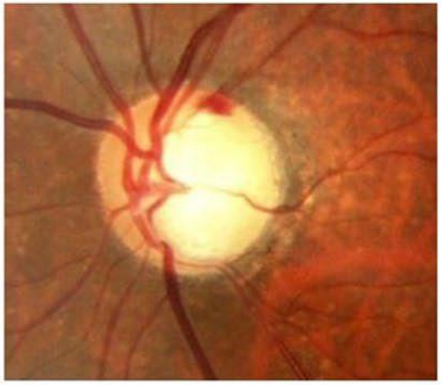
Generalized enlargement of cup (GE) disc type



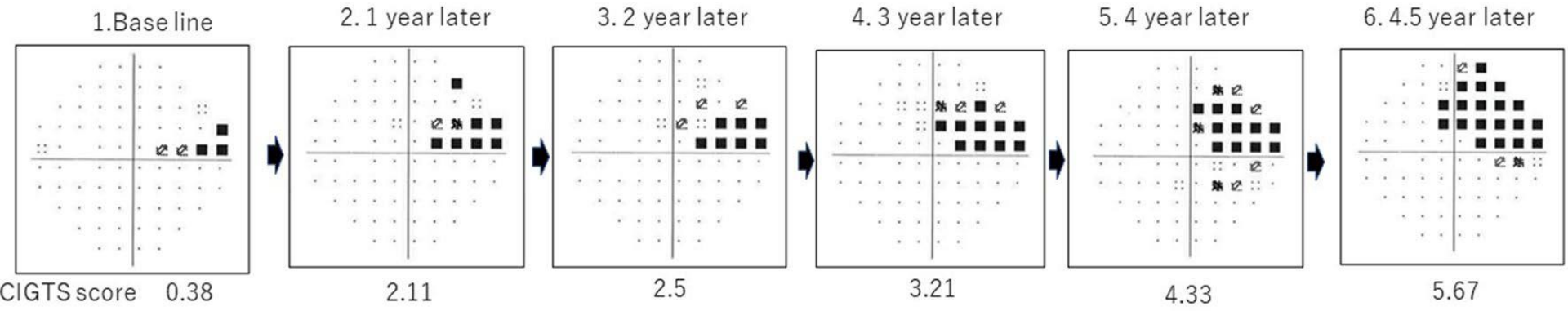
Focal glaucomatous (FG) disc type

MONITORAGGIO MORFOFUNZIONALE

Example of disc hemorrhage

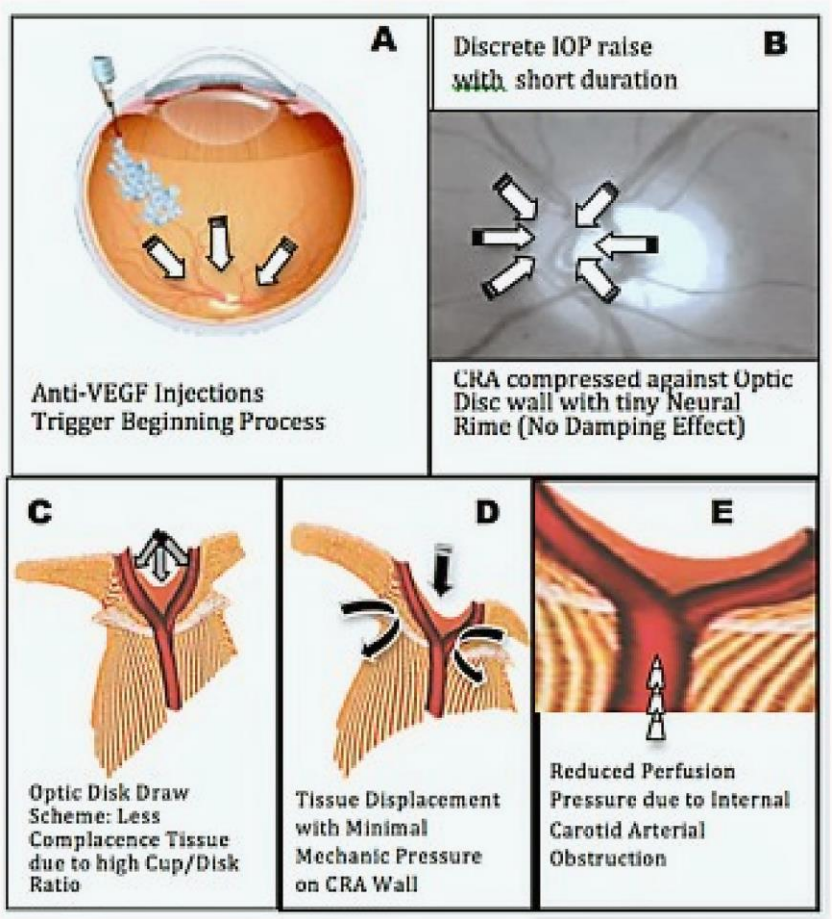


Examples of Visual field deterioration

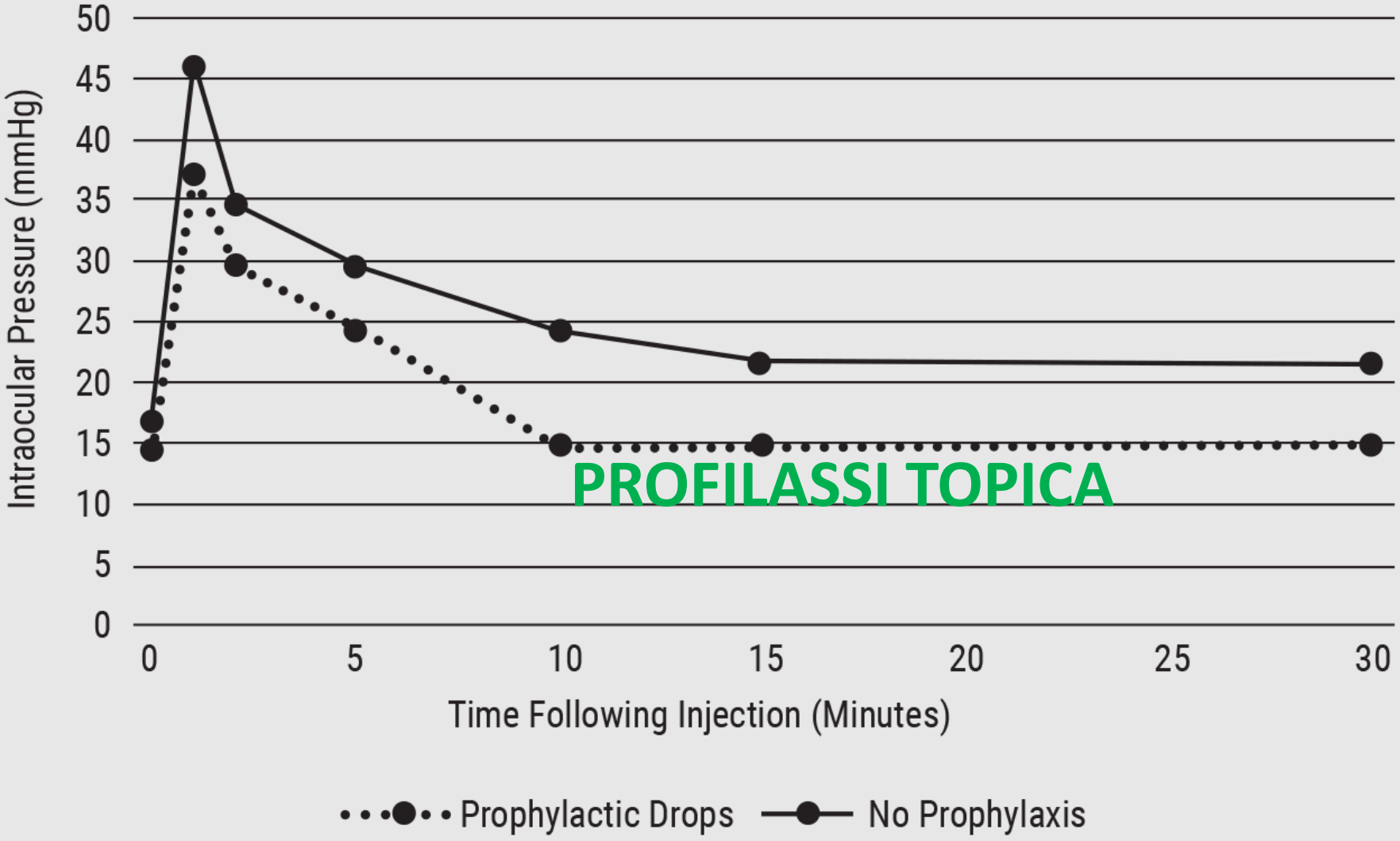


Transient central retina artery occlusion in patients undergoing intravitreal anti vegf injections
Nadyr A Damasceno

CUP/DISC RATIO



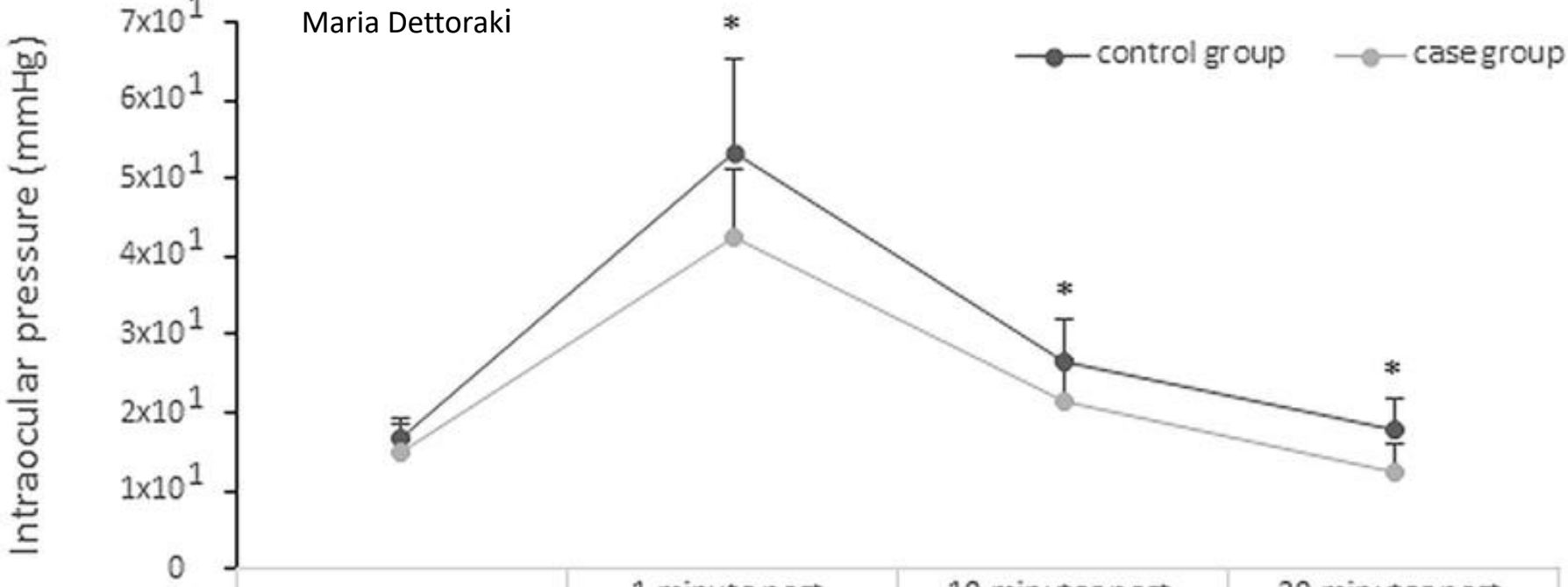
IMPACT OF TOPICAL DROPS GIVEN PRIOR TO INTRAVITREAL ANTI-VEGF INJECTIONS



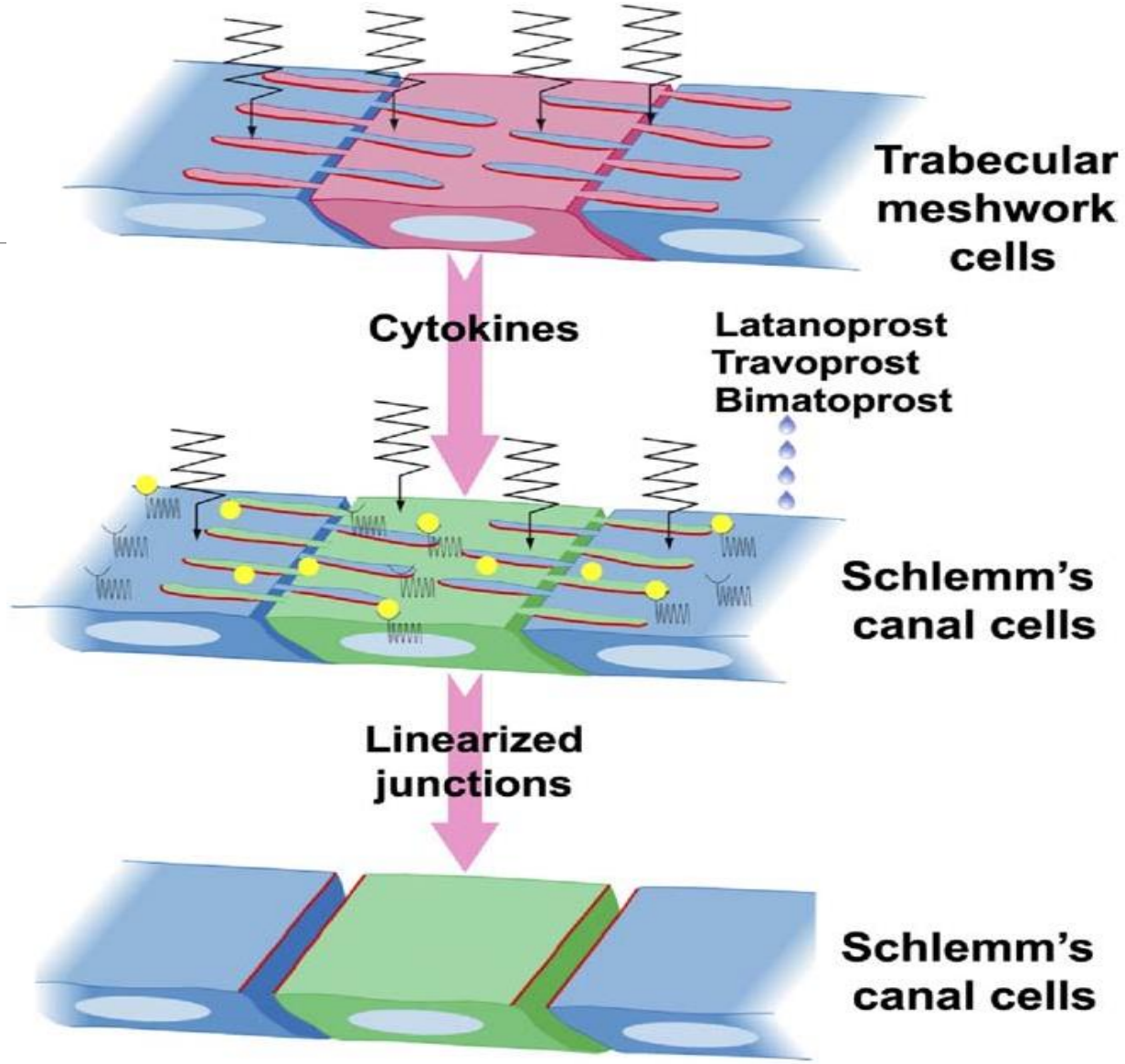
Terapia topica

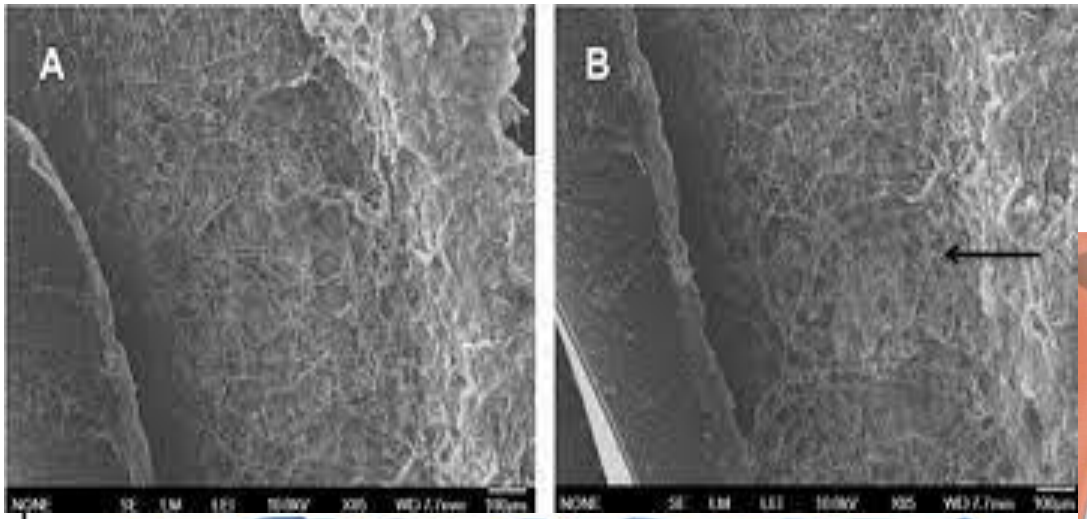
Effect of brinzolamide–brimonidine fixed combination on intraocular pressure spikes after intravitreal anti-VEGF injections

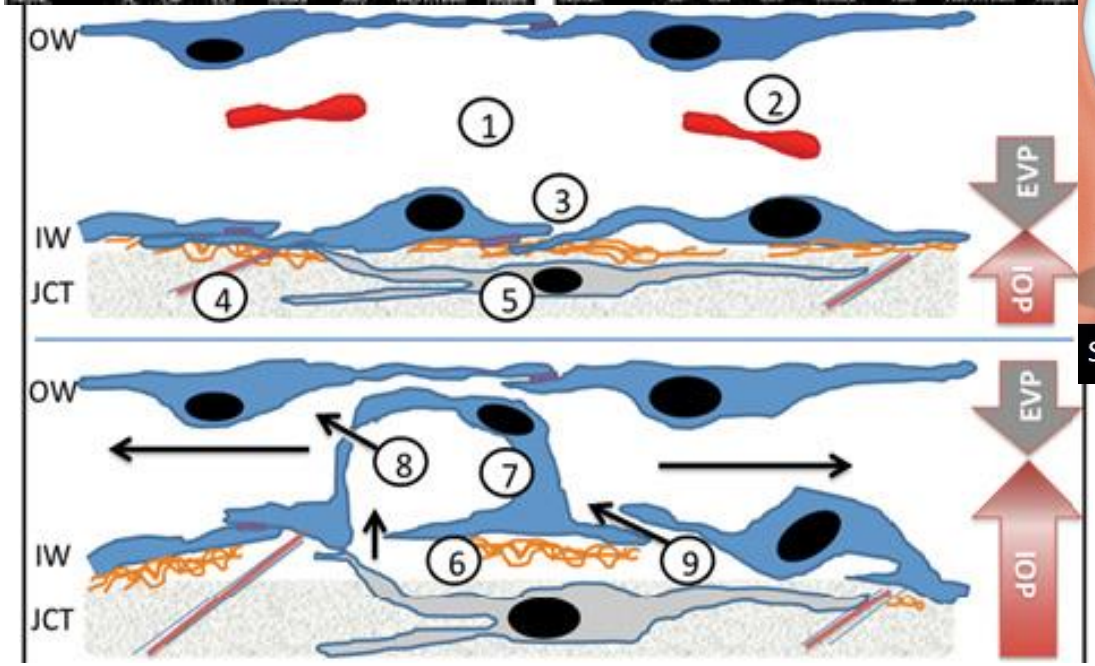
Maria Dettoraki

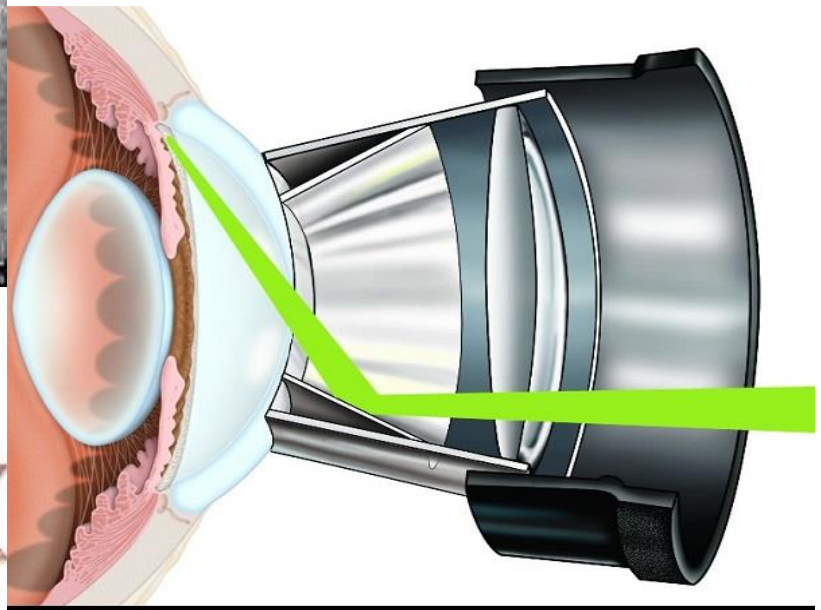


	pre-injection	1 minute post-injection	10 minutes post-injection	30 minutes post-injection
control group	16.6	53.4	26.4	17.9
case group	15.1	42.6	21.4	12.4


Trabecular meshwork cells
Cytokines
Latanoprost
Travoprost
Bimatoprost
Schlemm's canal cells
Linearized junctions
Schlemm's canal cells

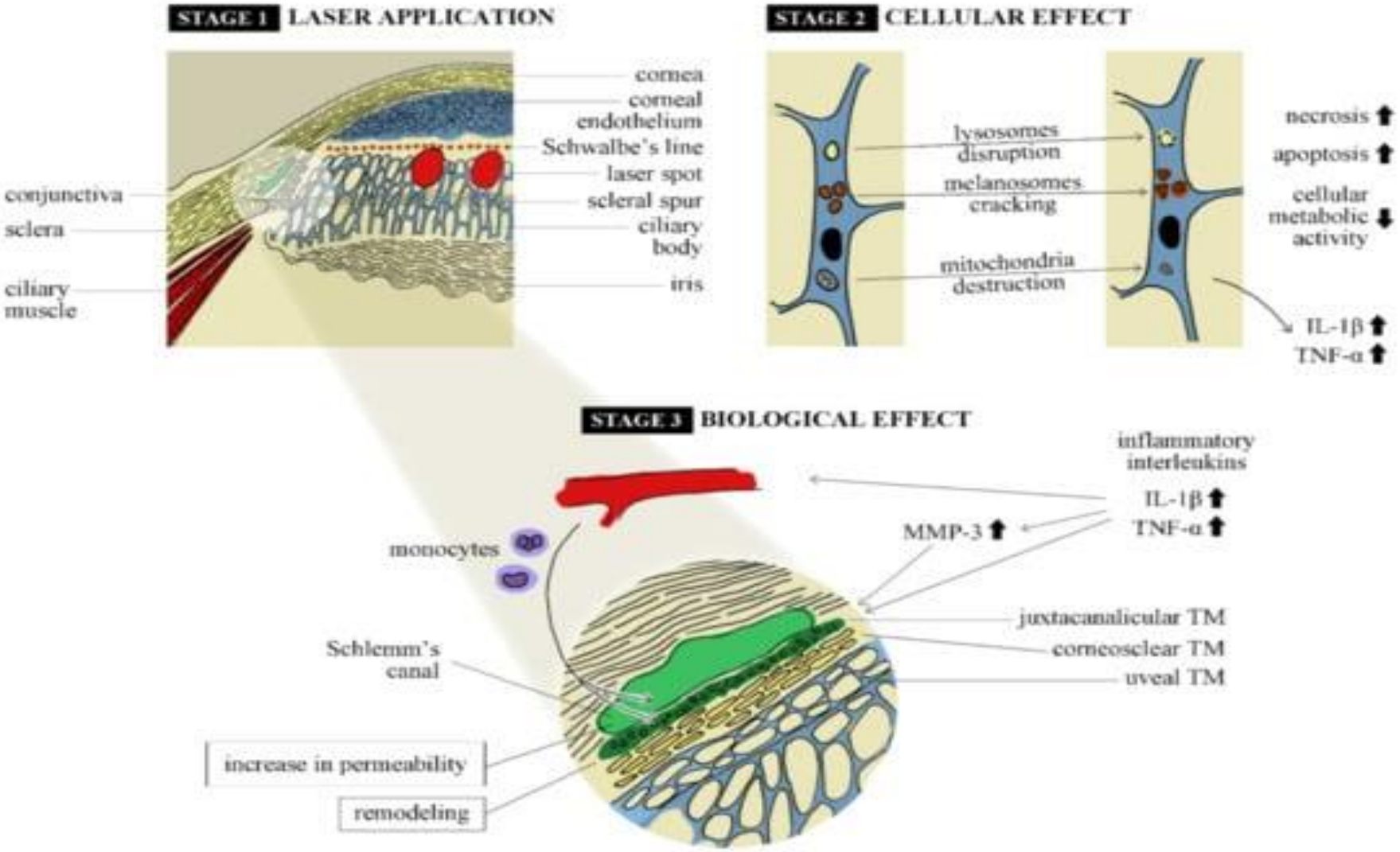






Selective laser trabeculoplasty

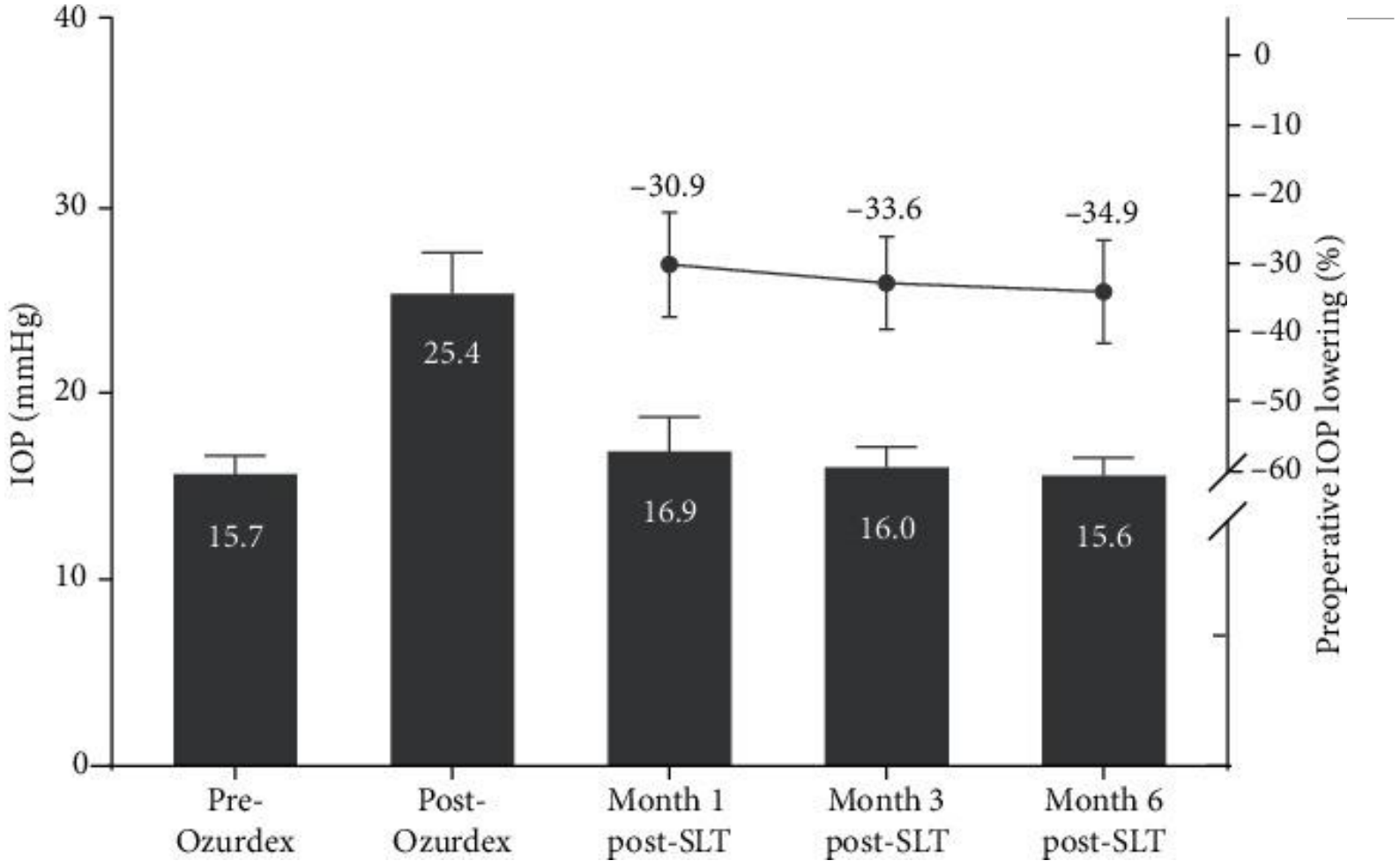
TRABECULOPLASTICA SELETTIVA

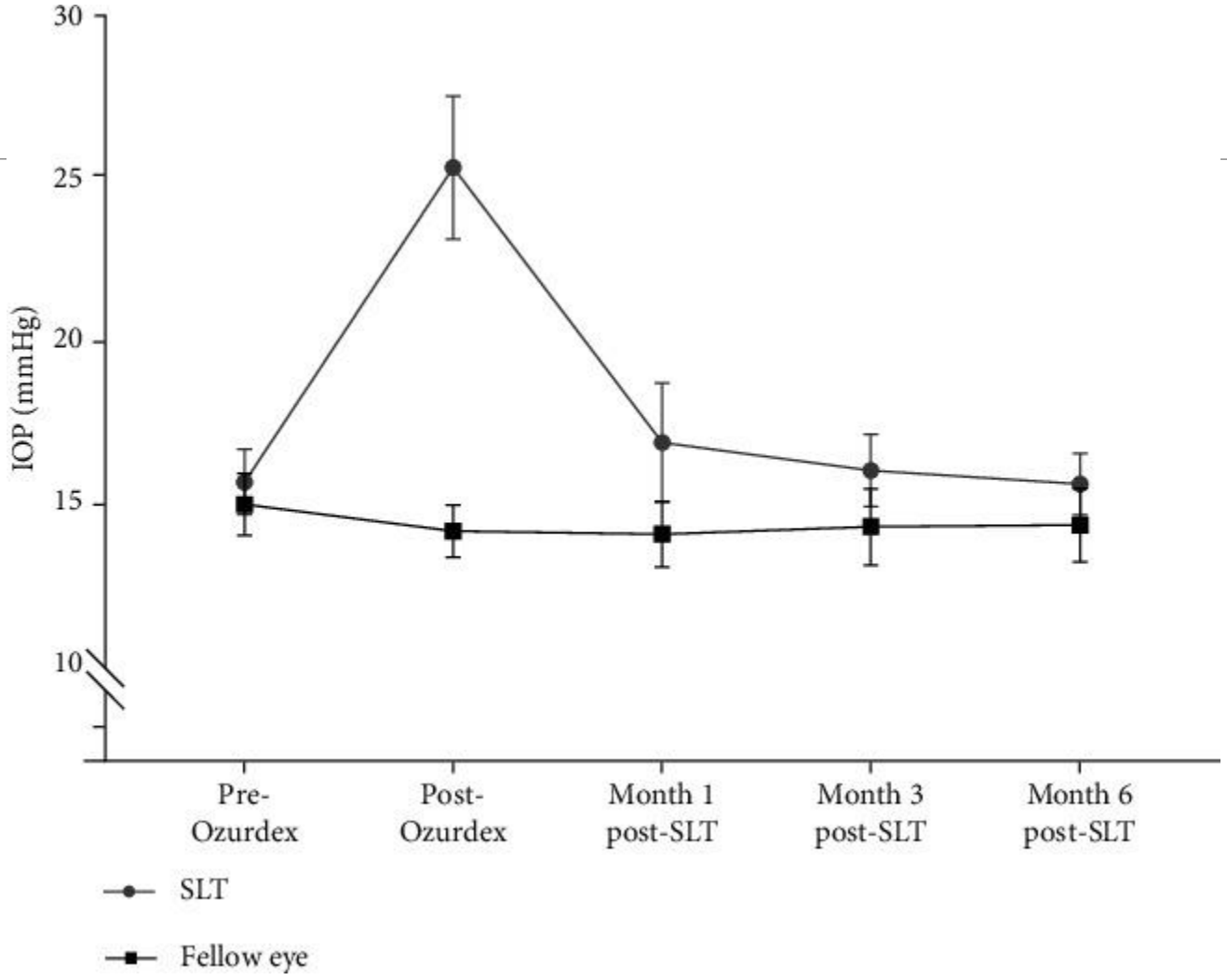


J Ophthalmol. 2020;

The Effect of Selective Laser Trabeculoplasty on Intraocular Pressure in Patients with Dexamethasone Intravitreal Implant-Induced Elevated Intraocular Pressure

Amin Bennedjai,




30
25
20
15
10
IOP (mmHg)
Pre-
Ozurdex
Post-
Ozurdex
Month 1
post-SLT
Month 3
post-SLT
Month 6
post-SLT
SLT
Fellow eye

Two-year analysis of changes in the optic nerve and retina following anti-VEGF treatments in diabetic macular edema patients

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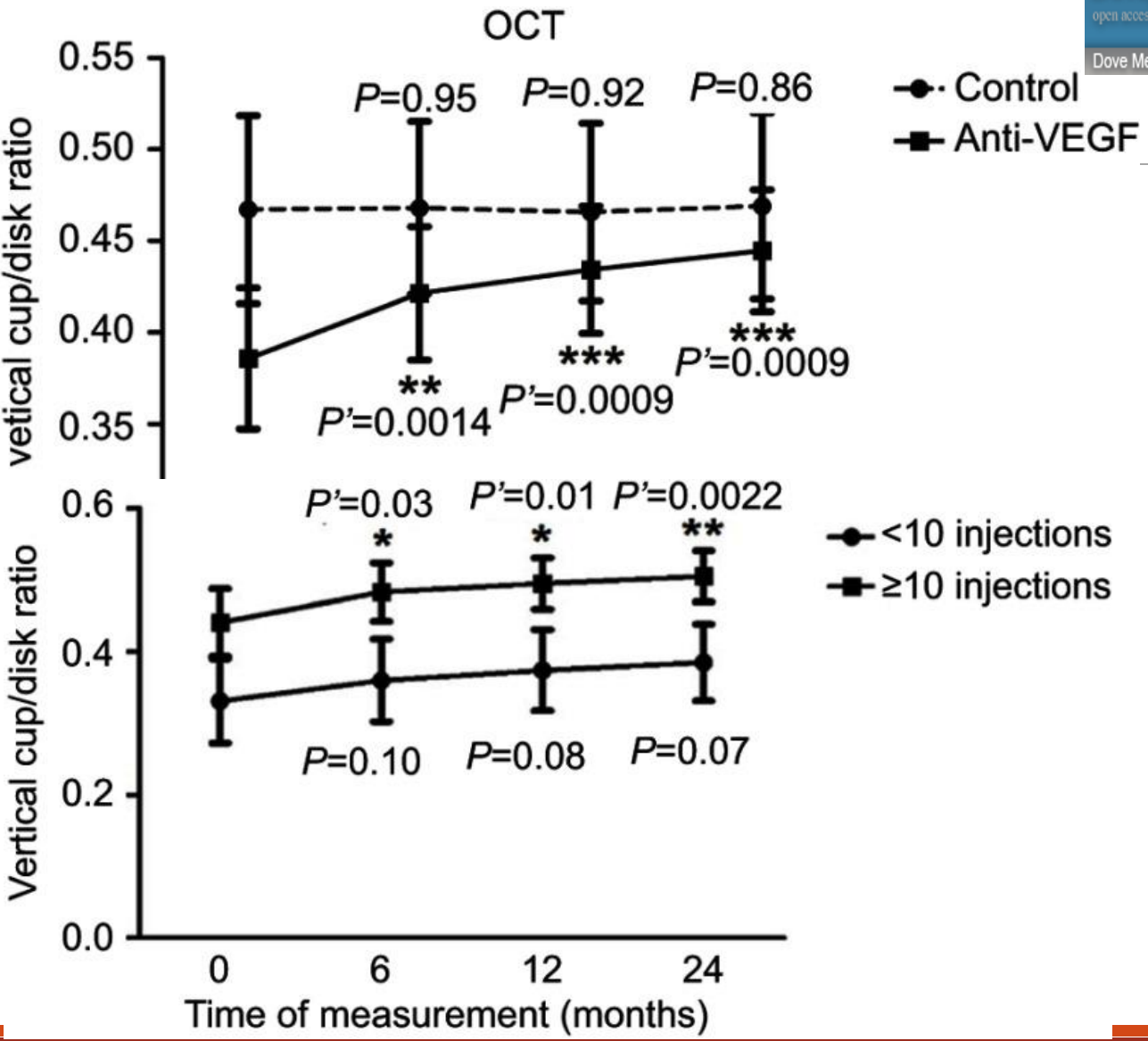
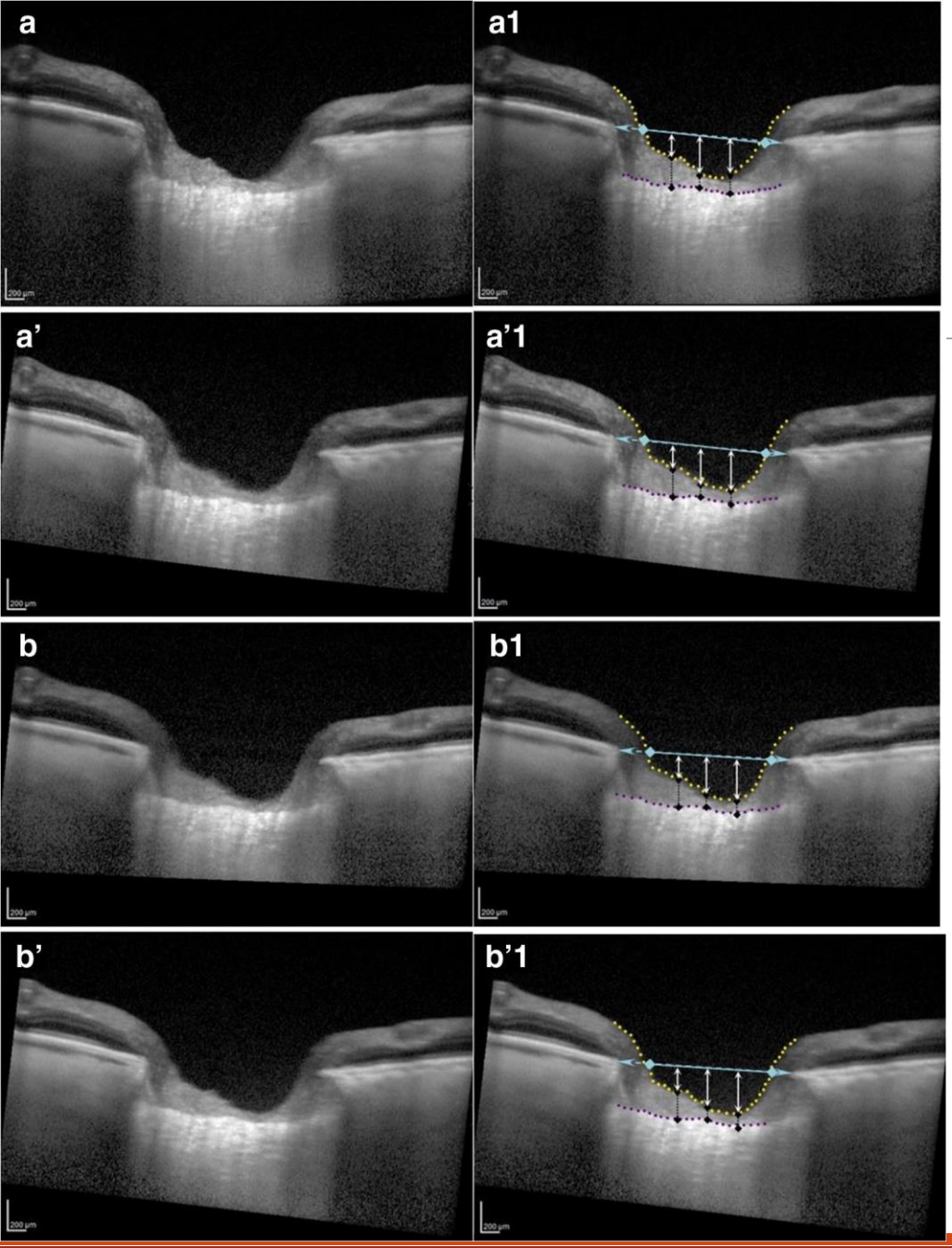




Irreversible and significant changes were observed in BMO (from 1666 to 1686 µm), inferior CD (from 143 to 156 µm) and inferior PPT (from 180 to 169 µm) after 1 year

Intraocular pressure increase after intravitreal anti-VEGF

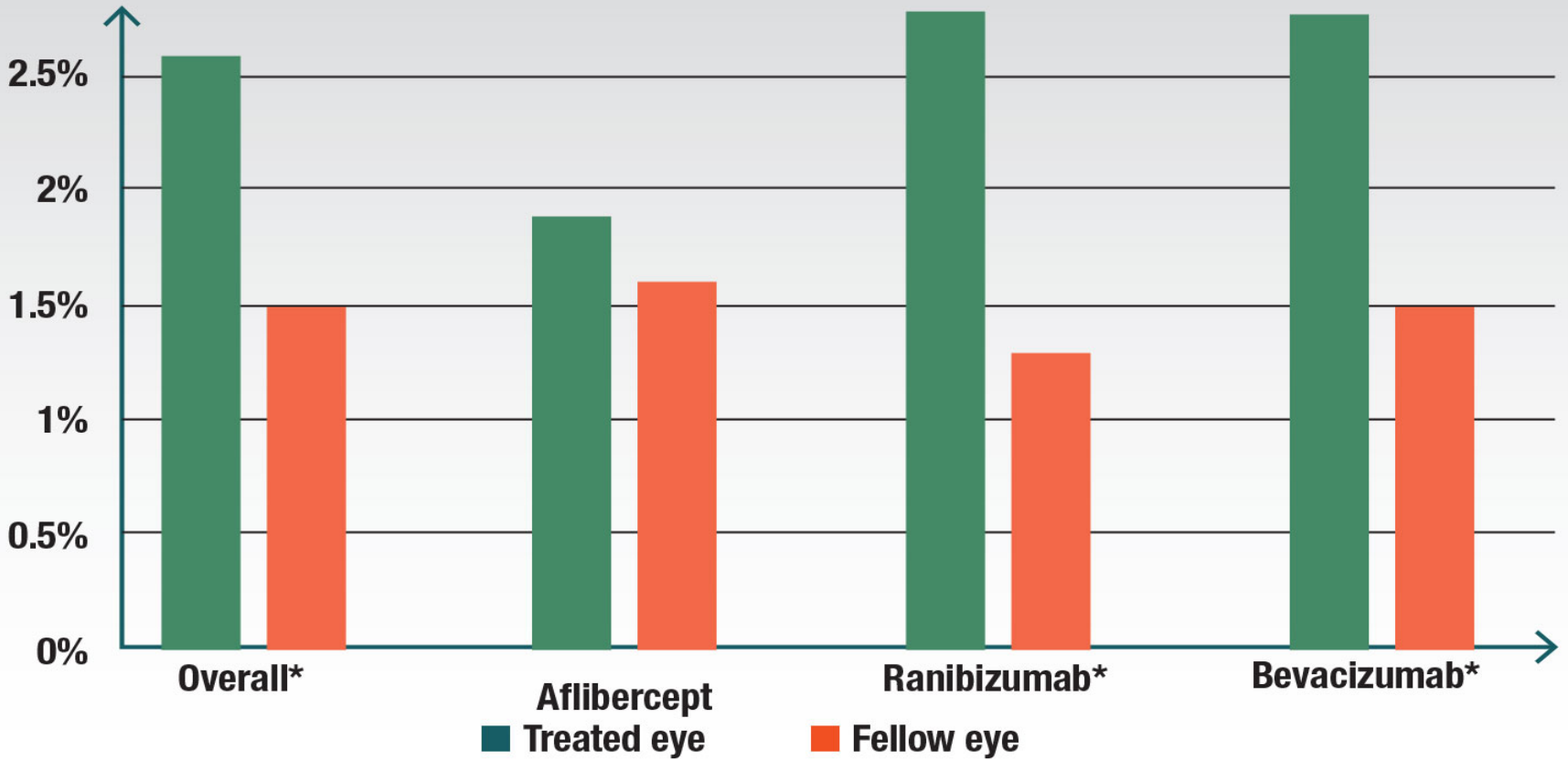


Figure. This graph shows the percentage of patients receiving ≥12 intravitreal anti-VEGF injections with a statistically significant intraocular pressure rise in the treated eye in comparison to the fellow untreated eye (*indicates statistically significant difference between treated and fellow eye). (Adapted from Atchison EA, Wood KM, Mattoz CG, et al. The real-world effect of intravitreous anti-vascular endothelial growth factor drugs on intraocular pressure. Ophthalmology. 2018;125:676-682.).

GESTIONE DELL'EDEMA MACULARE NEL PAZIENTE GLAUCOMATOSO

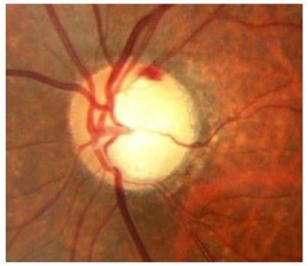
GESTIONE DEL GLAUCOMA NEL PAZIENTE CON EDEMA MACULARE

- **I PAZIENTI GLAUCOMATOSI AFFETTI DA EDEMA MACULARE COSTITUISCONO UNA VARIABILE CLINICA CON ELEMENTI DI CRITICITA' A LIVELLO DELLE OPZIONI TERAPEUTICHE**

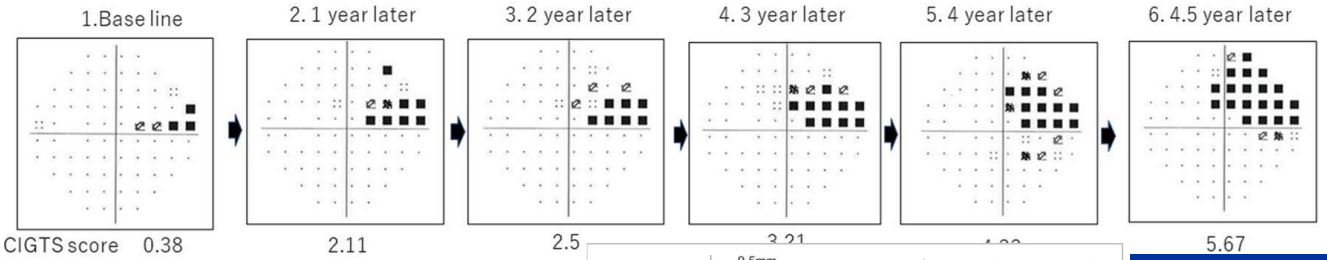
- **I PAZIENTI GLAUCOMATOSI DEVONO RICEVERE ADEGUATA PROFILASSI, MONITORAGGIO E TERAPIA TOPICA , PARACHIRURGICA ED EVENTUALMENTE CHIRURGICA**

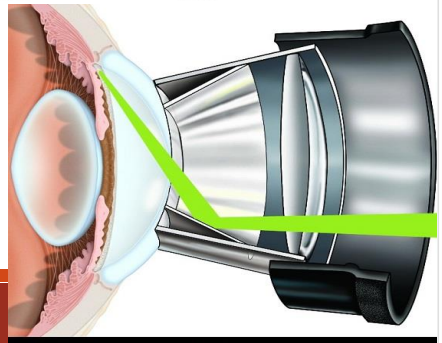
- **LA PRESENZA DEL GLAUCOMA NON DEVE RIDURRE LE CHANCE DI TRATTAMENTO DELL'EDEMA MACULARE**

Example of disc hemorrhage

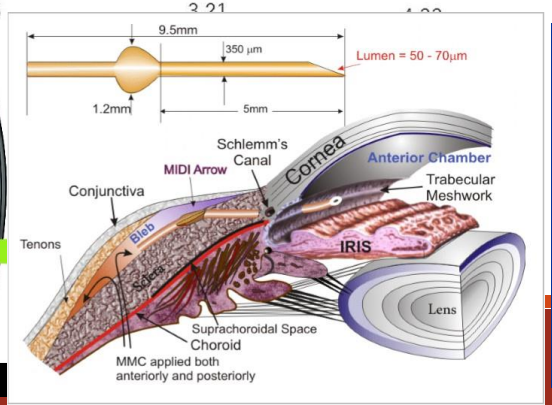


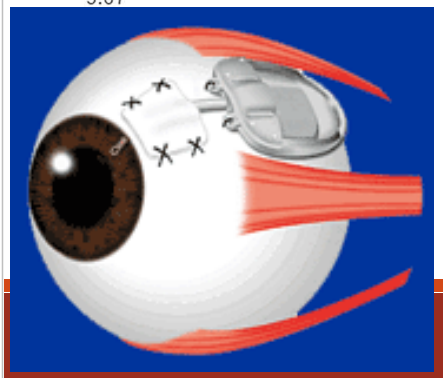
Examples of Visual field deterioration





Selective laser trabeculoplasty





GRAZIE PER L'ATTENZIONE