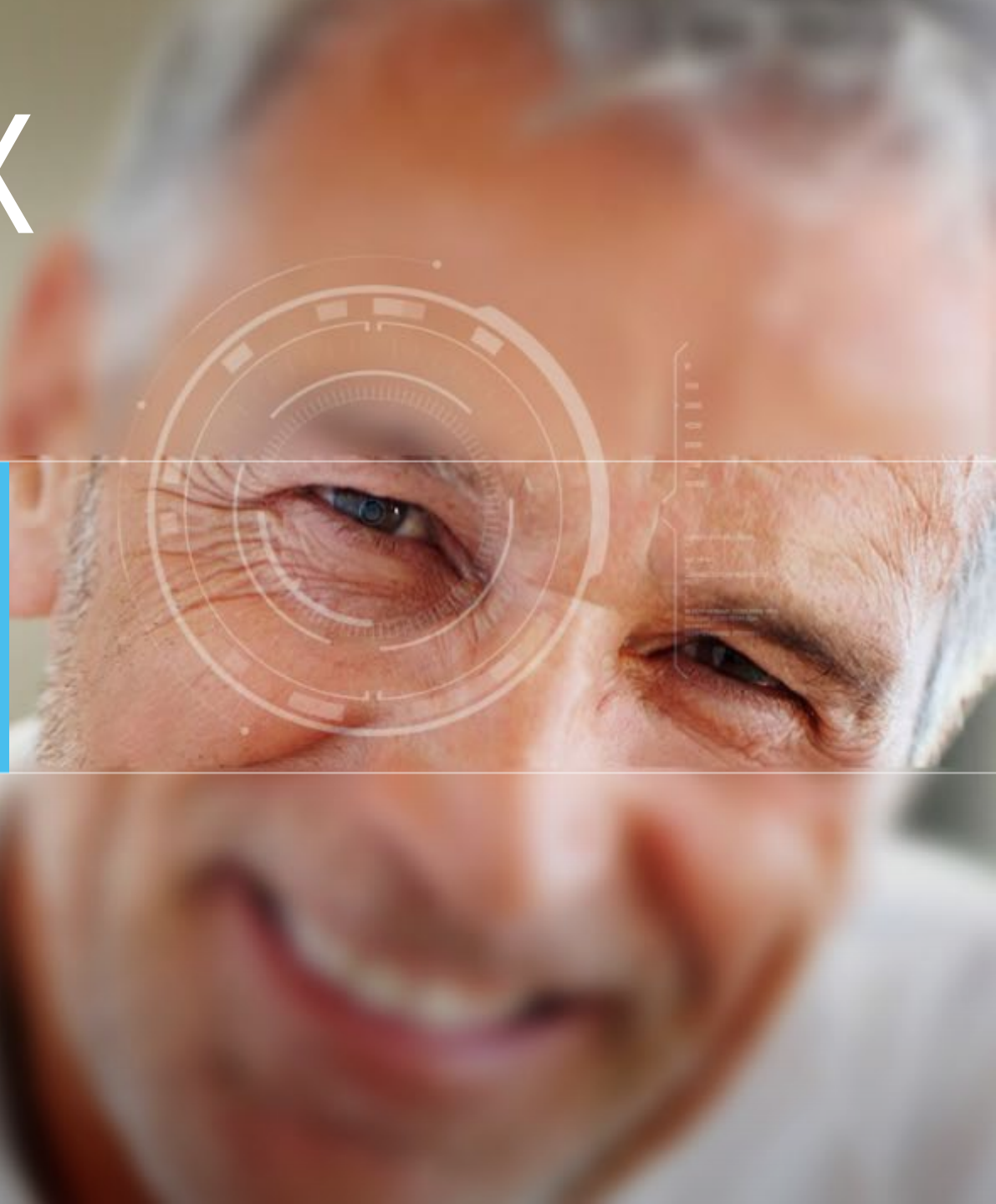
OCUGENIX

A New Approach to Angio-Fibrotic Diseases

Company Presentation

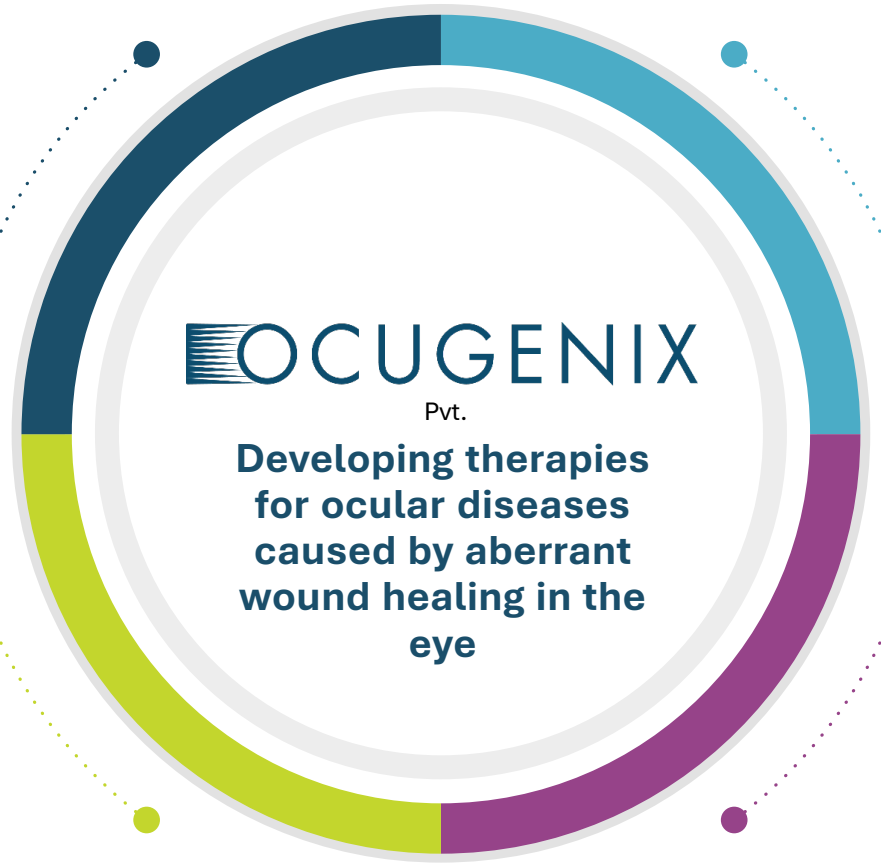
Q3 2026



Ocugenix at a Glance

Corporate History

- Clinical-stage biotech on track to complete the pivotal trials for NDA by 2028
- Supported by $22 million of NIH-funded research into molecular mechanisms of wound healing
- Additional $12 million in financing raised from local venture and family offices / mostly in the form of convertible notes
- Former Chair of Ophthalmology and Vice Chair of Pathology at UPMC among Ocugenix's co-founders



Clinical Highlights of Lead Program in wAMD (OGX110)

- 1st-in-class with disease-modifying results showing increased interval between treatments in wAMD
- >20 patients dosed with meaningful functional and anatomical changes observed in AMD
- improved BCVA over SoC while showing a 55% reduction in treatment burden
- Resolved the markers of fibrosis and cleared residual fluids

Our Platform: Scientific Approach

- Ocugenix is focused on activation of the CXCR3 pathway – the biological "switch" that turns off wound repair and turns on regenerative healing
- Activation of the pathway prevents and reverses pathological vasculature and fibrosis, and reduces inflammation
- Ocugenix is developing peptide mimics of naturally occurring activators of this CXCR3 pathway; new chemical entities
- Ocugenix pipeline includes wet AMD, Geographic atrophy, and Proliferative Diabetic Retinopathy and indications in the eye and other organs.

Strong IP and Market Position

- Issued worldwide composition of matter and disease process/MoA patents
- New chemical entity, and exclusivity through at least 2043
- Only compound in the clinic that is anti-angiogenic, anti-fibrotic and a suppressor of inflammation
- Compound can be developed both as a monotherapy and combination therapy

A new approach for the treatment of wet AMD

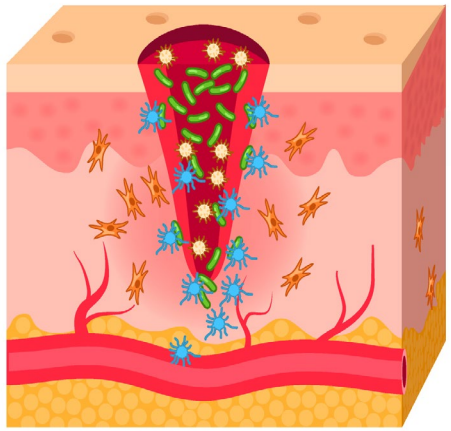
- We are a Phase II company develop a novel approach for the treatment of wet AMD based on the molecular mechanism of wound healing
- Wet AMD is caused by an aberrant wound response where a wound healing process fails to resolve
- Our early clinical data has shown good activity in all of the pathologies of wAMD including ones where anti-VEGFs have little to no. effect
- Only compound in clinical development that is:
  - Anti-fibrotic
  - Anti-angiogenic *and angio-regressive*
  - A longer persistence of effect than any other product in the clinic
  - Disease modifying
- 55% reduction in treatment burden relative to Vabysmo while demonstrating improved response on all anatomical features

Angiofibrotic Retinal Diseases Are a Result of Aberrations in the Wound Healing Process

IP-10 Activation of the CXCR3 Pathway is the Primary Driver Restoring Homeostasis

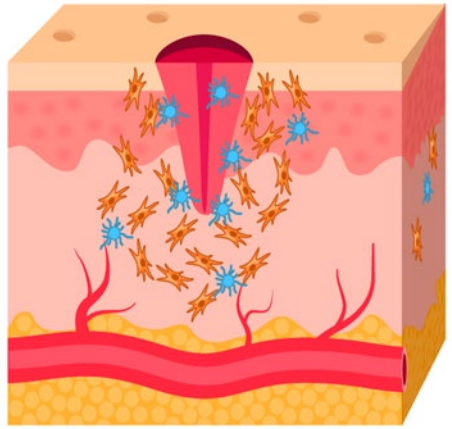
TURNING OFF the body's wound response via CXCR3 activation

HEMOSTASIS/ INFLAMMATORY

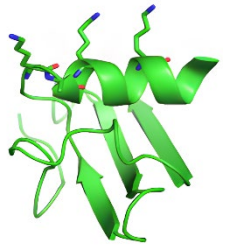


Following injury, HB-EGF, VEGF, EGF and many other factors promote tissue replacement with vessels, cells and matrix

TISSUE REPLACEMENT



IP-10 (aka CXCL10)

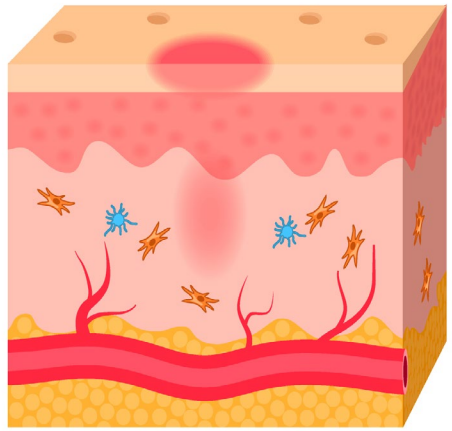


Activates CXCR3 (CXCR3 present on all cells during wound repair)

WOUND RESOLUTION

Angio-regression
Matrix regression
M2-MΦ inflammation suppression

HOMEOSTASIS



CXCR3 activation has been shown to be anti-fibrotic and anti-inflammatory in skin, heart, lung, liver, kidney and eye models by multiple groups

- With the wound response turned off:
  - over 90% of the recruited vasculature regresses, and
  - over 70% of fibrosis regresses
- This pathway is dominant over – and separate from – the VEGF and other growth factor pathways

Wells, Alan et al. “Skin Tissue Repair: Matrix Microenvironmental Influences.” Matrix Biology vol. 49,(2016): 25-36., Wells, Alan. ”Role of CXCR3 in Fibrotic Tissue Responses” Int J Biochem & Cell Biol vol 152 (2022): e106311.

Ocugenix's OGX110 Mimics IP-10/CXCL10, a Naturally Occurring Activator of CXCR3

IP-10/CXCL10

- 8.7 kDA cytokine that binds to the CXCR3 receptor, inducing the regression of angiogenesis and fibrosis
- Labile and hydrophobic, so not amenable to scale up

CXCR3 Receptor

- Activation causes a regenerative process in tissue stem cells that drives the restoration of HOMEOSTASIS
- This activation results in "turning off" the wound response and resetting the microenvironment to its previous state
- Cellular changes are seen within 6 hours of administration, and tissue changes within 24 hours

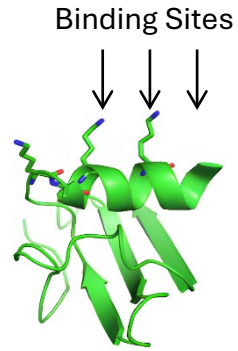
OGX110

- Experimental validations demonstrate that OGX110 has equivalently high selectivity for CXCR3 as naturally occurring chemokines
- Modifications in OGX110 allow for Composition of Matter patents through at least 2043
- Stable for at least 3 years as product and 2 years in formulation, and highly soluble in aqueous solutions

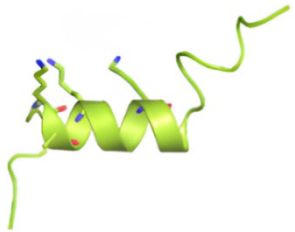
Endogenous Protein

IP-10 (aka CXCL10)

- 98 amino acid chemokines present in every cell
- Very high selectivity



OGX110



- 22 amino acids in length
- Mimics ligand to CXCR3 binding site
- Easily synthesized
- Worldwide CoM patent

Pathology of Wet AMD Follows This Wound Model

1 Natural accumulation of drusen in Bruch's membrane...

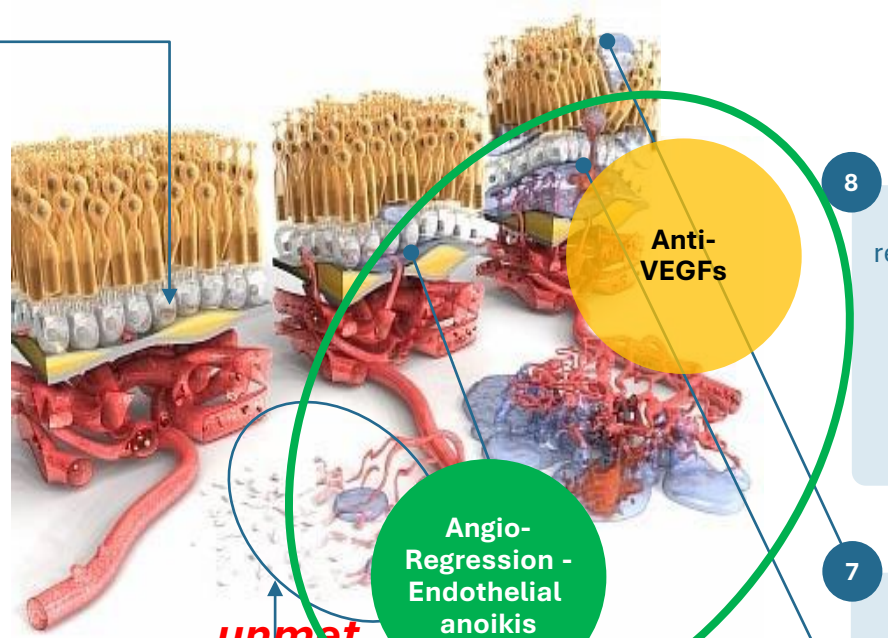
1 Step #1 of Wound Response

2 ...triggers recruitment of inflammatory cells...

Anti-Inflammatory – M2-like MΦ Sup Tcells

unmet need

3 ...which release numerous growth factors (e.g. VEGF) into the choroidal space...



unmet need

4 ...causing growth of new vessels...

5 ...which can penetrate through Bruch's membrane

6 The membranes of the new vessels are inherently permeable and leak fluid into the retinal space...

7 ...leading to fluid accumulation, and ultimately...

8 ...death of photoreceptors, which results in permanent central vision loss

unmet need

No resolution of wound response

Progressive retinal scarring

Anti-Fibrotic – Resolving Fibroblasts

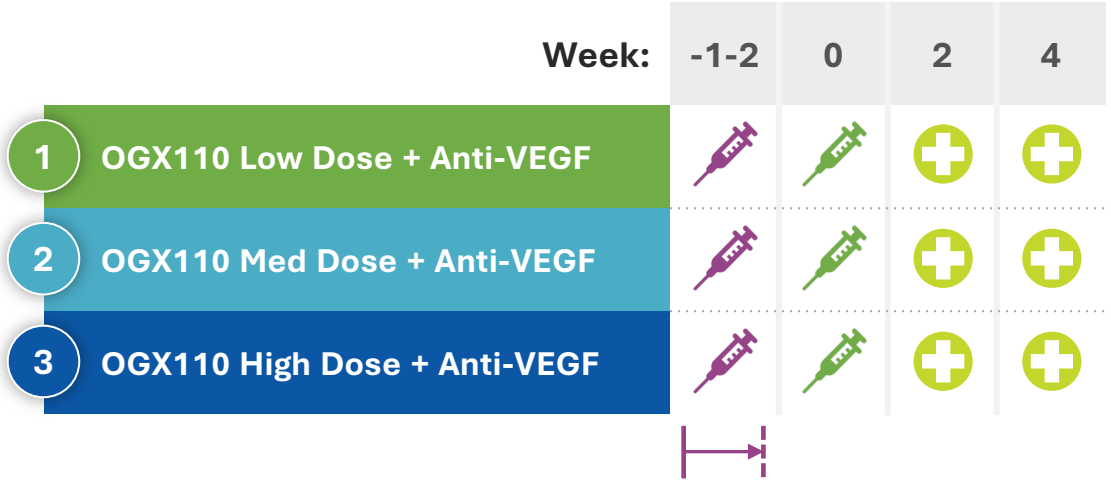
2 Step #2 of Wound Response

Homeo-Stasis – Recruit/support Tissue Stem Cells

Wound response never turned off by body

Schematic of wAMD with the key properties of CXCR3 activation by OGX110 superimposed on the pathologic processes.

Our Phase I Trial Demonstrated *Increased Visual Acuity* over Eylea Monotherapy



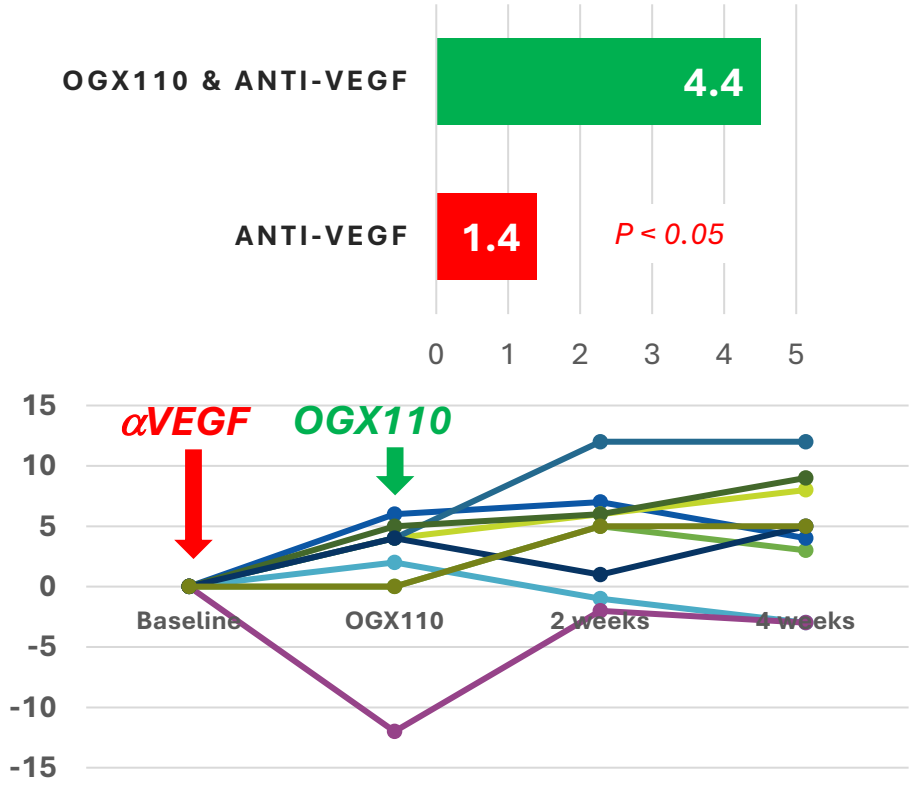
= Anti-VEGF

= OGX110

= Clinical examination w/ physician discretion for rescue

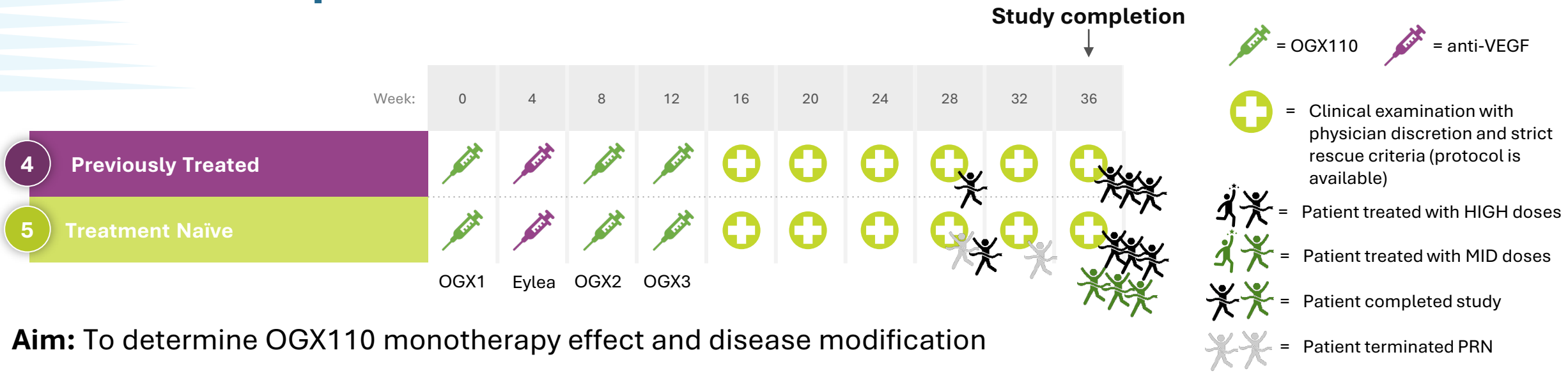
Previously-treated patients; persistent disease; 3 per dose

Impact of OGX110 on BCVA



- *Complete data set available under CDA*
- *6/9 patients gained 3 or more letters from OGX110, 2/9 were flat (within 2 letters) and 1/9 decreased 4 letters*

Phase IIa Trial ("AMD-2"), With Initial OGX110 Monotherapy, Shows Multiple Benefits of OGX110



Aim: To determine OGX110 monotherapy effect and disease modification

Population: Previously Treated & Treatment Naïve Cohorts

Principal Goal of the Study: Safety

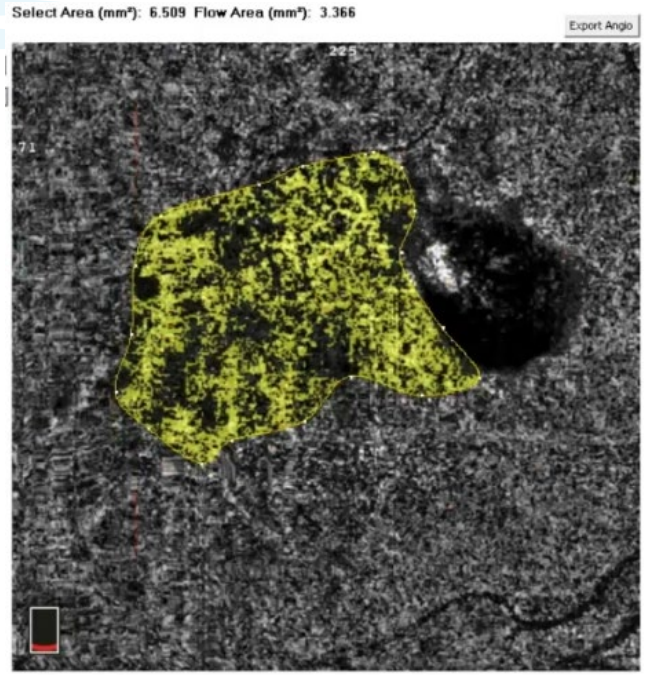
Secondary Goal: Positive impact on BCVA, improve retinal anatomical measurements, vascular regression, lesion shrinkage, establish disease stabilization

Visits: All included BCVA, SD-OCT, and fundus examination, FA and OCT-A at entry, week 16 and exit

Analyses: Change in BCVA and CRT, Investigator determination of fluids (IRF, SRF) and SHRM [binary +/-], anatomic evaluation of retinal fluids and structures [quantitative]

Regression of the Pathological Vasculature Without Retina Atrophy – Representative of 4 out of 5 patients showing OGX110-induce regression

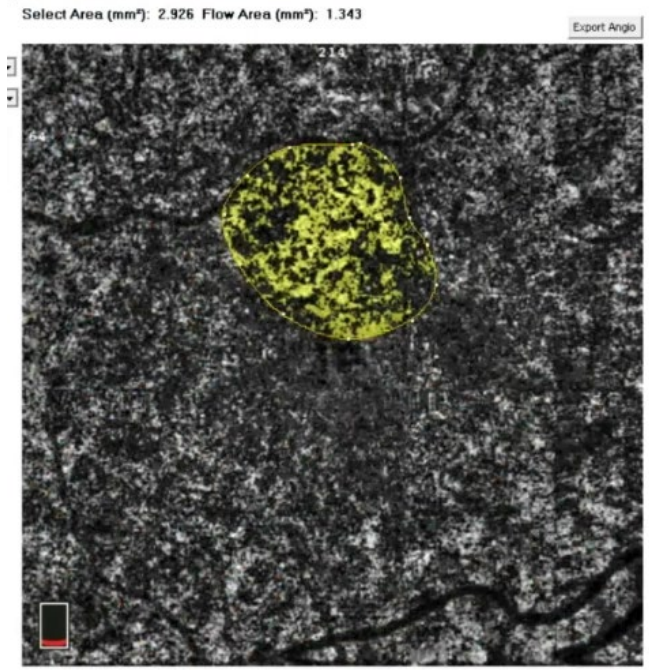
Visit: Baseline



Poor quality due to motion artifact and smoothing. There is a clearly visible PED with focal atrophic change. A visible CNV network is seen temporal to the PED, but the exact margins are poorly defined. With some careful consideration, we have traced the CNV. It involves central subfield and almost entire inner ring and some of superior and temporal outer ring. CNV area measurement 6.509.

Visit: 1 month after full course

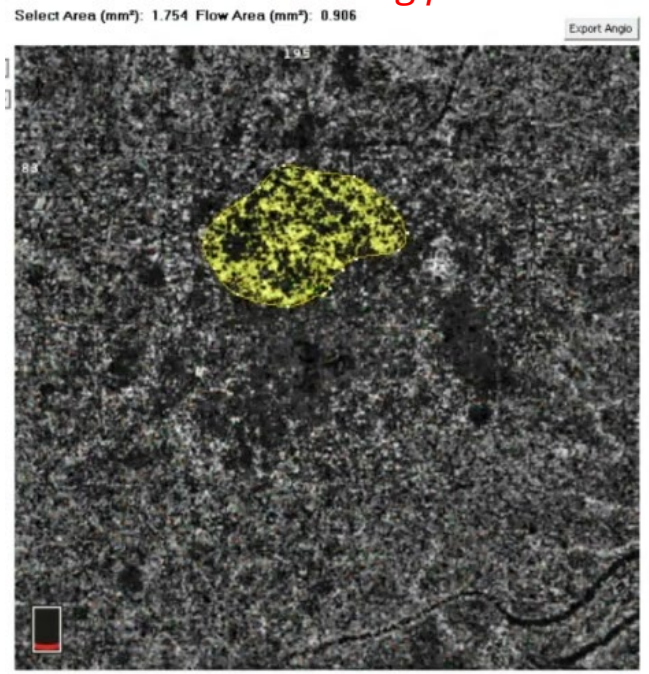
3 weeks after OGX110 is cleared



Better quality study with less motion. PED resolved. CNV in central subfield and superior and nasal inner ring but not as extensive as in past. CNV area measurement is 2.926.

Visit: 6 months after full course

6 months with no drug present



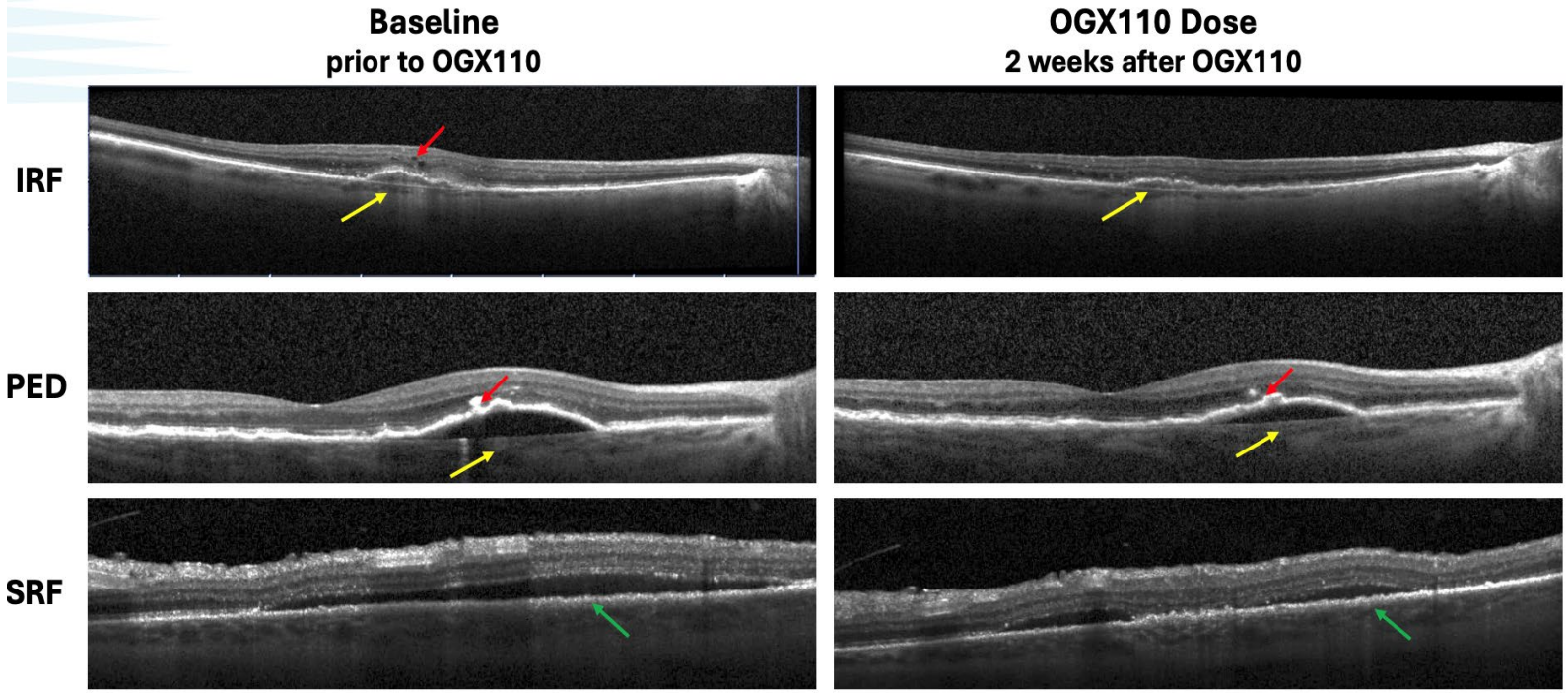
Better quality study. More well-defined region of CNV. Some gravitational SRF noted in fovea. CNV area measurement is 1.745. CNV does NOT involved central subfield but mostly in superior and slightly in temporal inner ring.

Patient 103-009; (CNV regression without atrophy) *4 of 5 patients* show this

Regression continues even after treatment, demonstrating wound resolution and remission

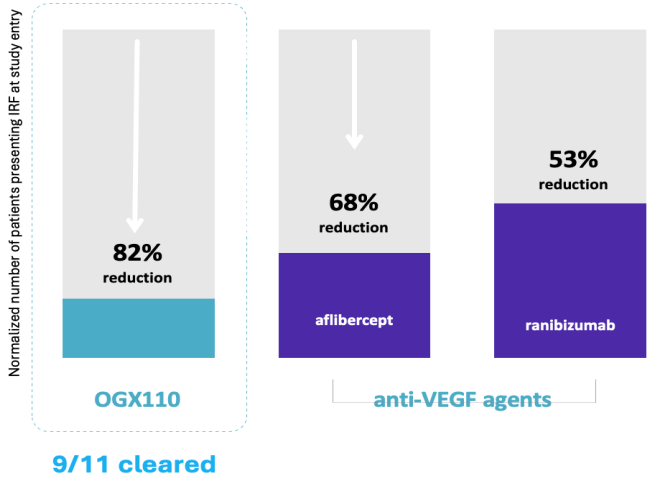
OGX110 Alone Decreases Fluid and the Treatment Clears Residual Fluids in More Patients

One IVT of OGX110 Initiates Fluid Resolution



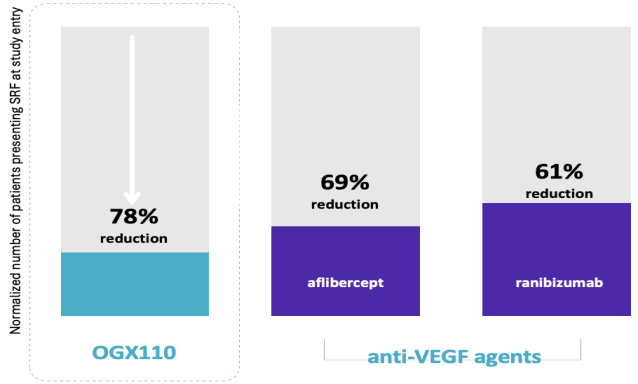
In the three treatment-naïve patients with exudative AMD show evident reductions and disappearance of intraretinal fluid (IRF; red arrow in 103-008 and 103-009), subretinal fluid (SRF; green arrow in 103-011), and reduced RPE elevation/PED (yellow arrow in 103-008 and 103-009).

After a loading course of OGX110, IRF disappears in > 80% of patients who initially present with IRF; anti-VEGF agents eliminate IRF in only 70% of presenting patients



9/11 cleared

Similarly, after a loading course of OGX110, SRF disappears in ~ 80% of patients who initially present with SRF; anti-VEGF agents eliminate SRF in about 2/3 of presenting patients



7/9 cleared

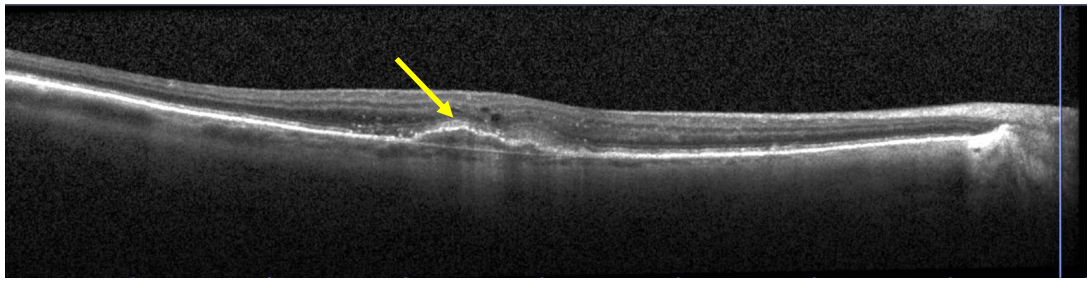
OGX110 data based on patients who completed the course of treatment; Eylea based on the TENAYA/LUCERNE trials Ophthalmology 2025; Lucentis based on EXCITE trial Ophthalmology 2014. Bars are normalized to all patients who present with the type of fluid retention, and percent absent of fluid type.

OGX110 Reduces SHRM, an Early Anti-Fibrotic Effect

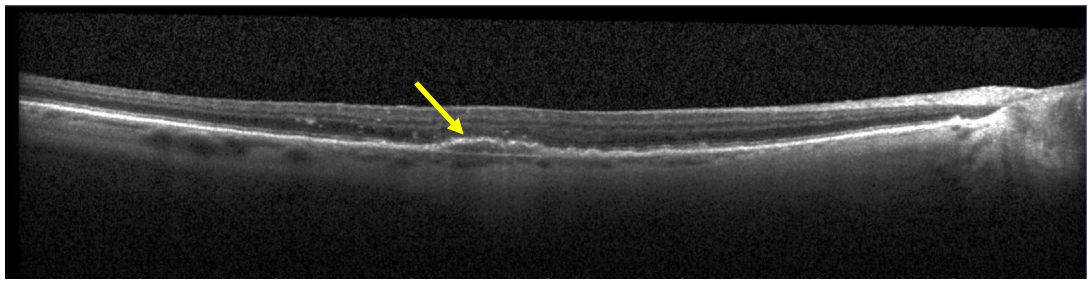
One IVT of OGX110 Reduces SHRM

Anatomic evidence of 90% reduction in SHRM (yellow arrow) 2 weeks after one dose of OGX110

Baseline

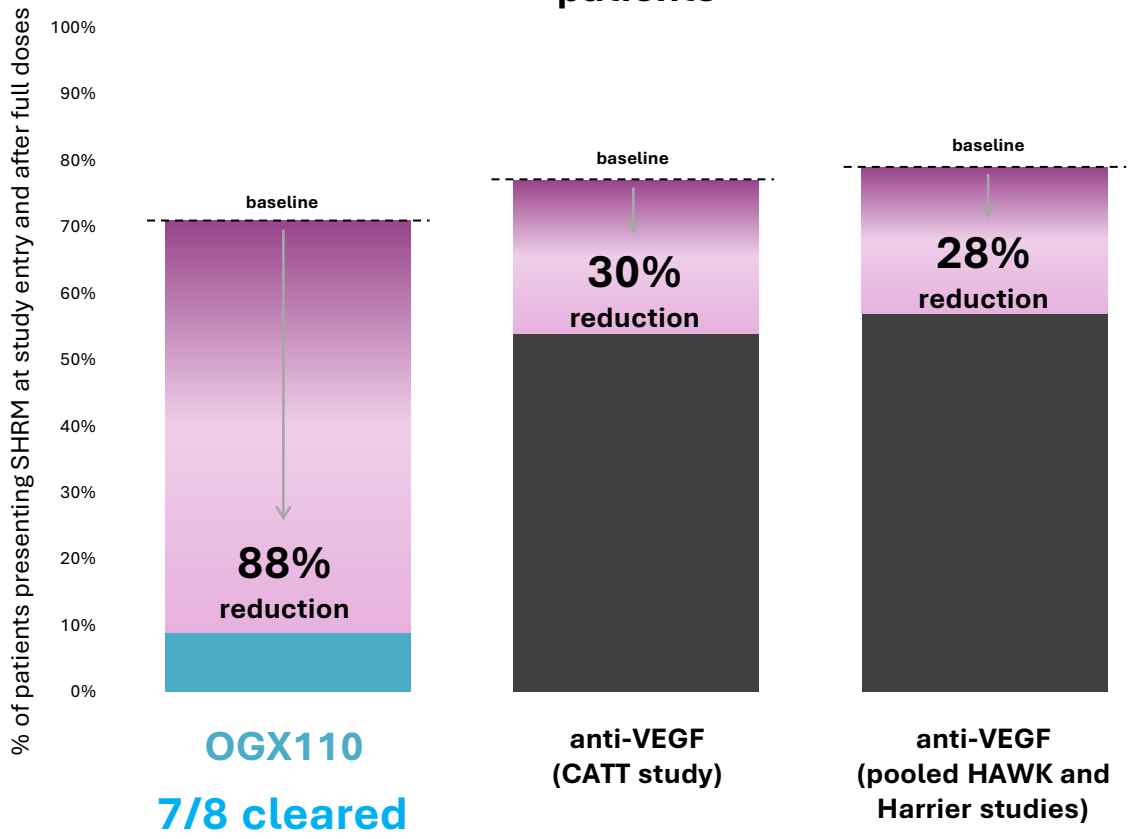


OGX110 14d



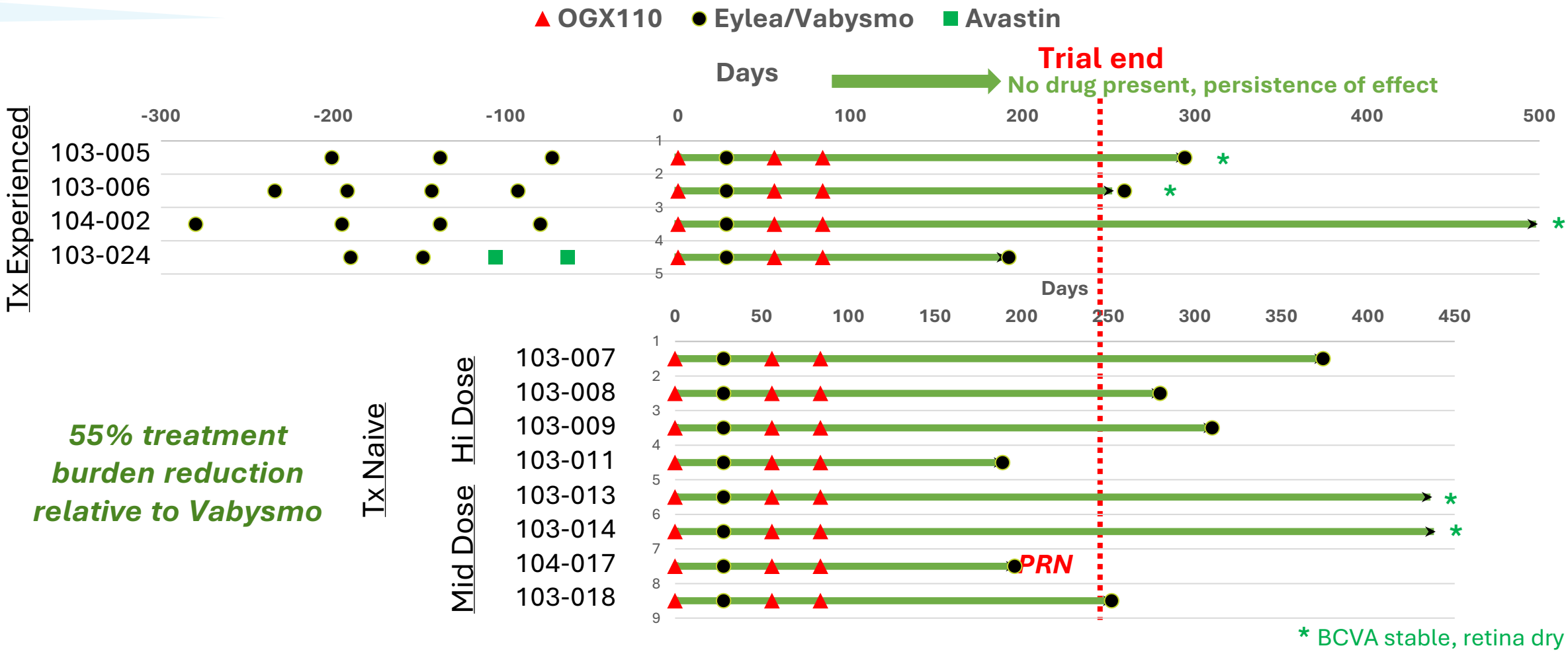
N.B.: Anti-VEGFs showed 50% reduction in SHRM thickness (HAWK and HARRIER studies)

After a loading course of OGX110, SHRM is absent in > 80% of patients who present with SHRM; anti-VEGF therapies eliminate SHRM in only 20-30% of such patients



OGX110 data based on all patients who completed the course of treatment; anti-VEGF data based on cited studies; based on presence or absence of SHRM.

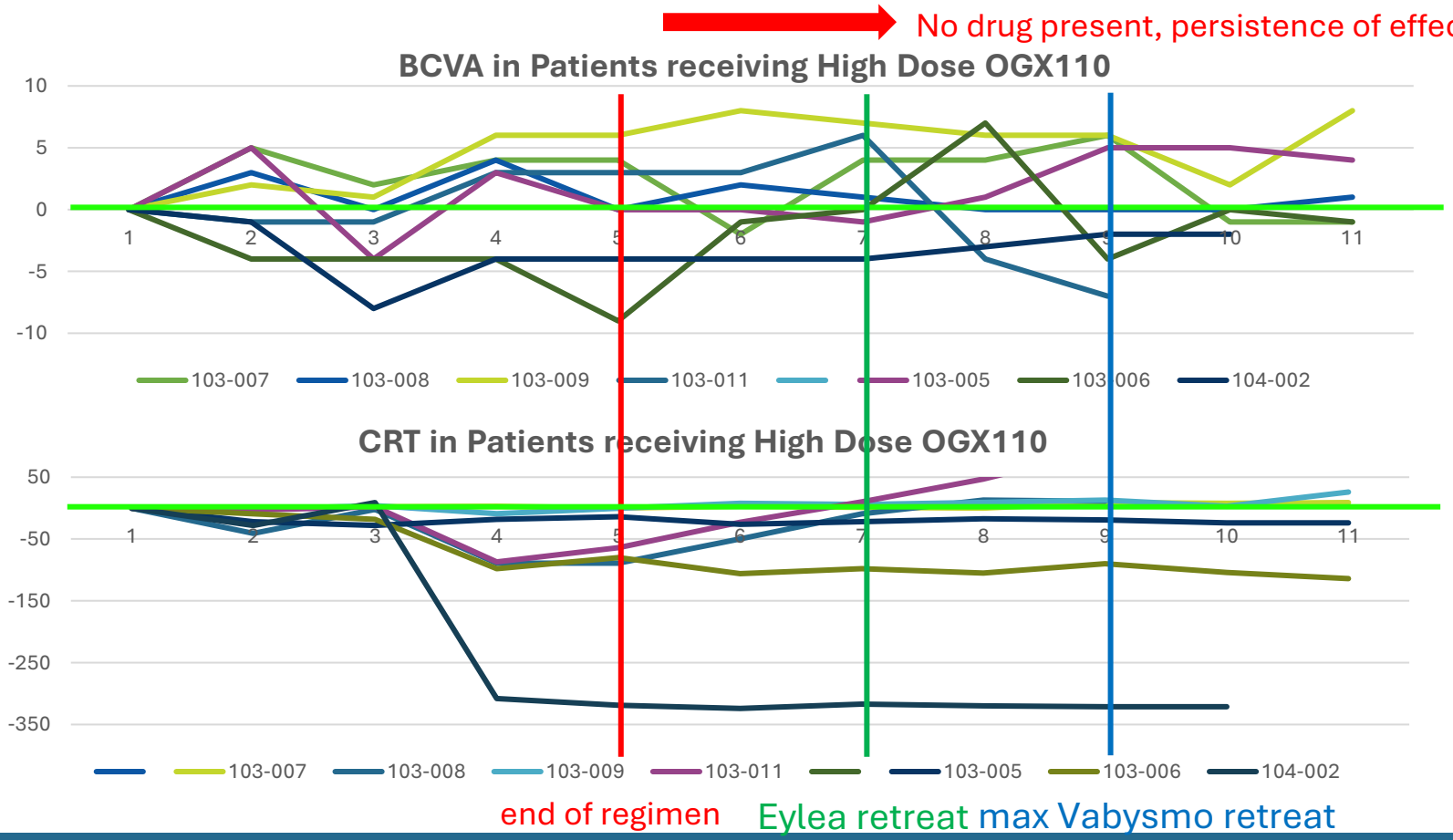
OGX110 Extends Treatment-Free Interval – Achieving Remission in a subset of Patients



55% treatment burden reduction relative to Vabysmo

OGX110 Effected a Response in Most All Patients – Stable Disease

- Stable disease: 7 of 11 patients had extended progression-free disease by BCVA gain *and* CRT reduction; 2 more were improved by one measure



All patients showed persistence of effect two to three times that of Vabysmo.

Real world experience is +0-1 letters in Experienced and +3 letters in Naïve patients at one year with 8-week treatment schedules

Persistence of effect and anatomical changes both demonstrate **OGX110 is disease modifying**

Next studies will measure persistence of effect out nine months

Ocugenix Has Performed Trials That Have Built a Compelling Story in Several Critical Areas of Wet AMD

Angioregression

We found:

Pathological vessels should regress as CXCR3 activation causes detachment of the underlying pathological vasculature. This manifested in **vascular regression and reduction of edema/decrease of fluid in active disease.**

Suppression of Fibrosis/Scarring/ Atrophy

We found:

By causing the matrix to transition to tissue regeneration, the drivers of fibrosis are absent and countered, and development of fibrosis should be suppressed. While this will take 9-12 months, **early elimination of SHRM** is supportive

Anti-Inflammatory

We found:

By reducing the inflammation in the eye, **BCVA vision improvement** well beyond that noted with Eylea in our Phase 1a patients

Restores Homeostasis

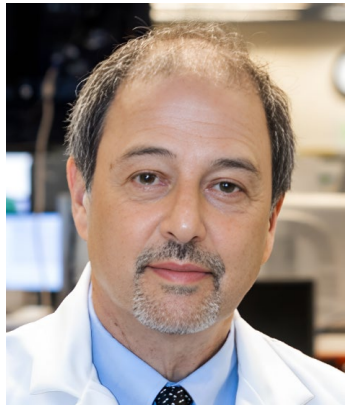
We found:

CXCR3 signaling restored homeostasis as noted by **prolonged disease-free intervals** until the patients needed to be treated again

Management Team and Advisors

Sean McDonald, CEO and Co-Founder

- Successful Serial Healthcare Founder & Entrepreneur
- Group President, McKesson (over 7 divisions)
- Founder/CEO of Automated Healthcare; grew to $200M
- Respironics Board Member to $5B exit
- CEO of Precision Therapeutics, a pioneering company in personalized medicine
- Entrepreneur in Residence - Carnegie Mellon University



Alan Wells, MD, CSO and Co-Founder

- 30 years of NIH-funded research in wound healing
- Executive Vice-Chair Pathology, University of Pittsburgh and UPMC
- Medical Director of Clinical Laboratories, UPMC Health System
- Past President, Wound Healing Society

Richard Kollender Board Member / Advisor	CEO, Reaction Biology, with extensive experience as a life sciences executive / board member and venture capital investor
David Guyer Advisor	CEO, EyeBio (acquired by Merck for up to $3bn) and former CEO, Ophthotech (now Iveric Bio, acquired by Astellas for ~$6bn)
Bruce Peacock Advisor	Extensive biotech experience across former C-suite roles, including CEO (Adolor, Alba, Orthovita), and CFO of Ophthotech
Ian Conner Co-Founder	Associate Professor of Ophthalmology, University of Pittsburgh
Joel Schuman Co-Founder	Vice Chair for Research Innovation and Co-Director, Glaucoma Service Wills Eye Center
Cecilia Yates Co-Founder	Associate Professor of Nursing, University of Pittsburgh
Barb Berglund CMC & Quality	11 ophthalmic IND programs
Bill Brock Toxicology	35 years experience in both ocular and eye drop submissions
Gary Novak Regulatory	Over 55 successful FDA ophthalmology submissions

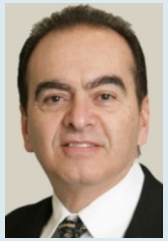
Clinical Advisors

Robert Avery, MD

- Founder and CEO, California Retina Consultants
- Co-Founder, California Retina Research Foundation

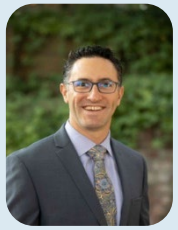
David Boyer, MD

- Senior Partner, Retina-Vitreous Associates Medical Group
- Adjunct Clinical Professor of Ophthalmology, Keck School of Medicine of USC



Don D'Amico, MD

- Chair, Ophthalmology, Weill Cornell Medical College
- Ophthalmologist in Chief, NY Presbyterian Hospital
- Fellow of ARVO, Awards from AAO and ASRS



Roger Goldberg, MD, MBA

- Vitreoretinal Surgeon, Bay Area Retina Associates
- Active Member, American Society of Retinal Specialists, the Retina Society, and the American Academy of Ophthalmology

Raj Maturi, MD, FASRS

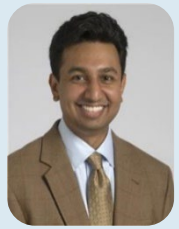
- Founder and Partner, Retina Partners Midwest
- Adjunct Clinical Associate Professor of Ophthalmology, Indiana University School of Medicine

SriniVas Sadda, MD, FARVO

- President, Association for Research in Vision and Ophthalmology (ARVO)
- Director, Artificial Intelligence and Imaging Research at the Doheny Eye Institute
- Professor of Ophthalmology, UCLA

Jason Slakter, MD

- Vitreoretinal Surgeon, Vitreous Retina Macular Consultants NY, and NYU School of Medicine
- Head of Imaging, Voiant



Sunil Srivastava, MD

- Uveitis and Vireo-Retinal Specialist, Cole Eye Institute at the Cleveland Clinic
- Senior Achievement Award Recipient, American Academy of Ophthalmology