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STOP THE START

of classical complement-driven diseases

Update: ANX007 Phase 3 Program in GA

August 2024

Forward-Looking Statements and Disclaimers

This presentation contains “forward-looking” statements about Annexon, Inc. and our industry that involve substantial risks and uncertainties. All statements other than statements of historical facts, including statements regarding topline data from the ARCHER Phase 2 trial and post-hoc analyses, our clinical and preclinical programs, timing and commencement of future nonclinical studies and clinical trials and research and development programs, timing of clinical results, strategic plans for our business and product candidates, including additional indications which we may pursue, our financial position, runway and anticipated milestones, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as “aim,” “anticipate,” “assume,” “believe,” “contemplate,” “continue,” “could,” “design,” “due,” “estimate,” “expect,” “focus,” “goal,” “intend,” “may,” “objective,” “plan,” “positioned,” “potential,” “predict,” “seek,” “should,” “target,” “will,” “would” and other similar expressions that are predictions of or indicate future events and future trends, or the negative of these terms or other comparable terminology.

Forward-looking statements are not guarantees of future performance and are subject to risks and uncertainties that could cause actual results and events to differ materially from those anticipated, including, but not limited to, risks and uncertainties related to: the ongoing off-treatment follow-up portion of the ARCHER Phase 2 trial and final results from the ARCHER Phase 2 trial; our history of net operating losses; our ability to obtain necessary capital to fund our clinical programs; the early stages of clinical development of our product candidates; the effects of COVID-19 or other public health crises on our clinical programs and business operations; our ability to obtain regulatory approval of and successfully commercialize our product candidates; any undesirable side effects or other properties of our product candidates; our reliance on third-party suppliers and manufacturers; the outcomes of any future collaboration agreements; and our ability to adequately maintain intellectual property rights for our product candidates.

These and other risks are described in greater detail under the section titled “Risk Factors” contained in our Annual Report on Form 10-K and Quarterly Reports on Form 10-Q and our other filings with the Securities Exchange Commission (SEC) Annual Report on Form 10-K filed with the Securities Exchange Commission (SEC) on March 26, 2024 and our other filings with the SEC from time to time. All forward-looking statements in this presentation speak only as of the date of this presentation. Except as required by law, we undertake no obligation to publicly update any forward-looking statements, whether as a result of new information, future events or otherwise.

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Comparisons to third-party studies are provided for illustrative purposes only. Differences exist between trial designs, study sites, subject populations and applicable products or candidates, and caution should be exercised when comparing outcomes across studies.

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A bold mission to free the body, brain and eye from complement-mediated disease

Overview of ANX007 Geographic Atrophy Program

Structure-confirmed vision benefit in Phase 2 ARCHER study; Phase 3 ARCHER II ongoing

- ✓ Unique neuroprotective MOA, blocking C1q-mediated synapse and neuron elimination
- ✓ Consistent, significant, dose & time-dependent vision protection across pre-specified endpoints
  - — Multiple lines of evidence, including: 12 months on-treatment, fellow-eye and off-treatment analyses
  - — Benefits demonstrated on multiple visual acuity measures
- ✓ First-in-kind visual function benefit supported by protection of structures correlated with visual function
  - — Significant protection of photoreceptors across retina
  - — Enhanced protection of photoreceptors and RPE specifically in the foveal center subdomains – structures correlated with visual acuity
- ✓ Generally well tolerated; no CNV increase in treated vs. sham; no reported cases of vasculitis
- ✓ ANX007 1st and only EMA PRIME Designation in GA – based on functional benefit
- ✓ Global Phase 3 program to confirm ARCHER findings **NOW ENROLLING**

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Anti-C1q Mechanism of Action

Classical Complement-Mediated Neurodegeneration Extensively Researched in Ophthalmic and Neurological Diseases

Functional clinical benefit previously demonstrated in Huntington's disease and ALS, and now in GA

Ben Barres, M.D., Ph.D.
Discoverer of C1q Technology
Scientific Co-Founder, Annexon

Anti-C1q protective in several models, including:

- Geographic atrophy (photoreceptor damage)
- Glaucoma
- Retinal ischemia
- Huntington's disease
- Amyotrophic lateral sclerosis
- Alzheimer's disease
- Frontotemporal dementia
- Spinal muscular atrophy
- Traumatic brain injury

ANTI-C1q PROTECTS AGAINST SYNAPSE LOSS AND NEURODEGENERATION

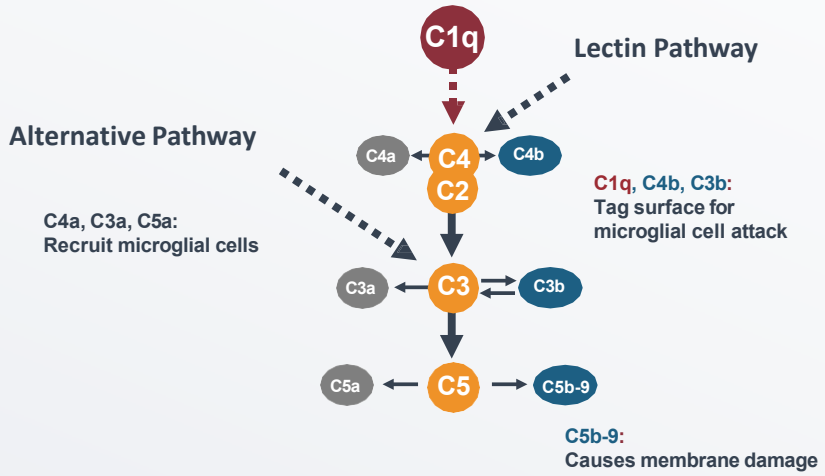
- Discovered by Annexon co-founder, Ben Barres, spawning an entire field and validated in multiple labs[1]
- Synapse loss correlates with functional decline[2]
- Synapse loss precedes neuronal loss[3]

[1]Stevens, et al., 2007 DOI 10.1016/j.cell.2007.10.036; Schafer et al., 2012 DOI 10.1016/j.neuron.2012.03.026; Hong, et al., 2016 doi: 10.1126/science.aad8373; Lui et al., 2016 doi.org/10.1016/j.cell.2016.04.001; [2]Davies et al., 1987 *J Neurological Sci* **78**:151; Terry, et al., 1991 *Ann Neurol* **30**:572; [3]Yoshiyama et al., 2007 DOI 10.1016/j.neuron.2007.01.010

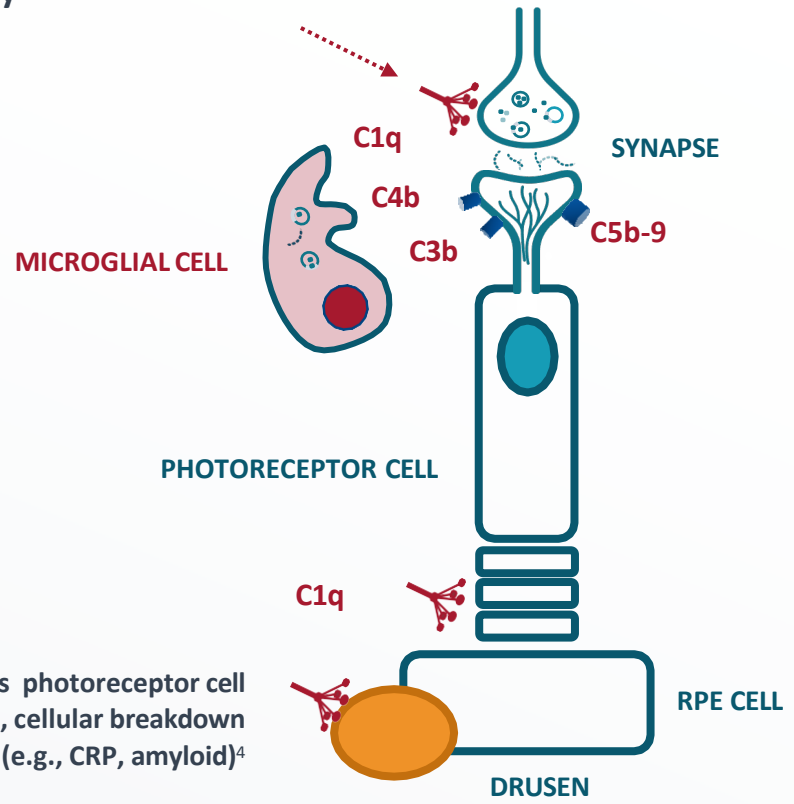
Anti-C1q: A Distinct Neuroprotective Mechanism

C1q initiates classical complement cascade to drive photoreceptor synapse & cell loss and neuroinflammation

- C1q is a **key driver of neurodegeneration**[1]
- C1q anchors classical pathway activation on **photoreceptor cells to cause inflammation and loss**[2]
- **ANX007 inhibits C1q** and all damaging components of the classical pathway[3]



C1q binds stressed photoreceptor synapses and activates the classical pathway



In GA, **C1q** also binds photoreceptor cell outer segments, cellular breakdown products and drusen (e.g., CRP, amyloid)[4]

[1]Stevens, 2007, *Cell* **131**:1164; Howell, et al., 2011 *J Clin Invest*. **121**:1429; Schafer, et al., 2012 *Neuron* **74**: 691; Stephan et al., 2012 *Annu Rev Neurosci* **35**:369; Hong, et al., 2016 *Science*. **352**:712; Lui, et al., 2016 *Cell* **165**:921; Dejanovic, et al., 2018 *Neuron* **100**:1322; Vukojicic , et al., 2019, *Cell Rep.* **29**:3087; Williams, et al., 2016 *Mol Neurodegener* **11**:26; [2]Tassoni, et al., SFN 2022; Annexon data on file; Jiao, et al., 2018 *Mol Neurodegener* **13**:45; Katschke, 2018 Sci Rep. **8**:7348. [3]Lansita, et al., 2017 *International Journal of Toxicology*, **36**:449; [4]Yednock, et al., 2022 *Int J Retina Vitreous* **8**:79

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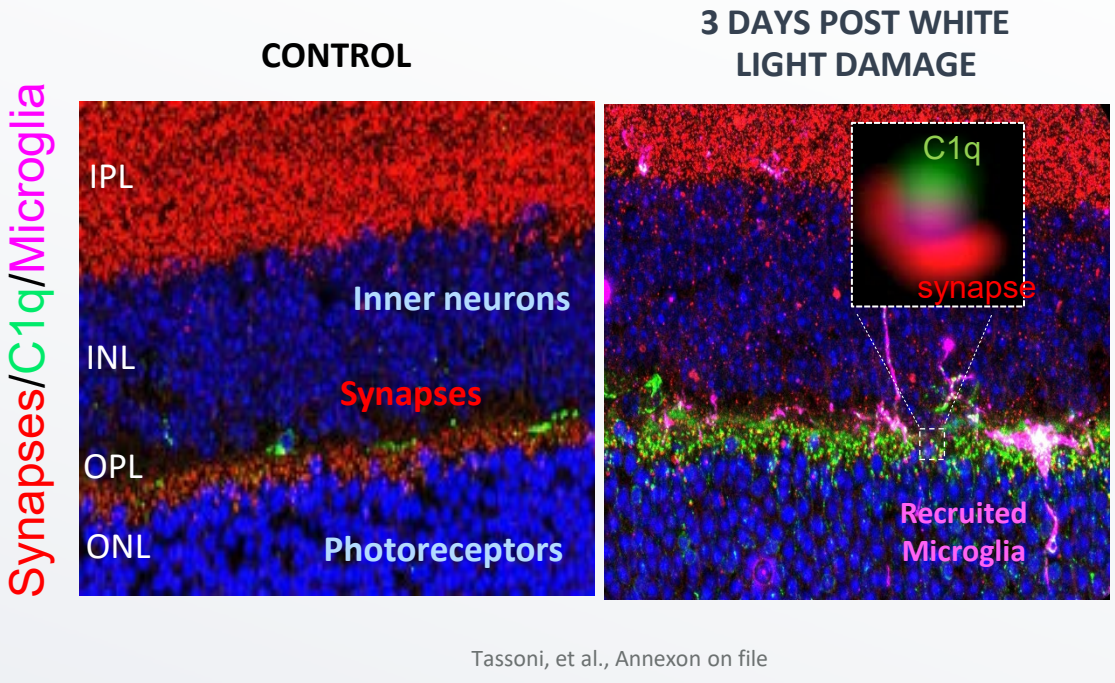
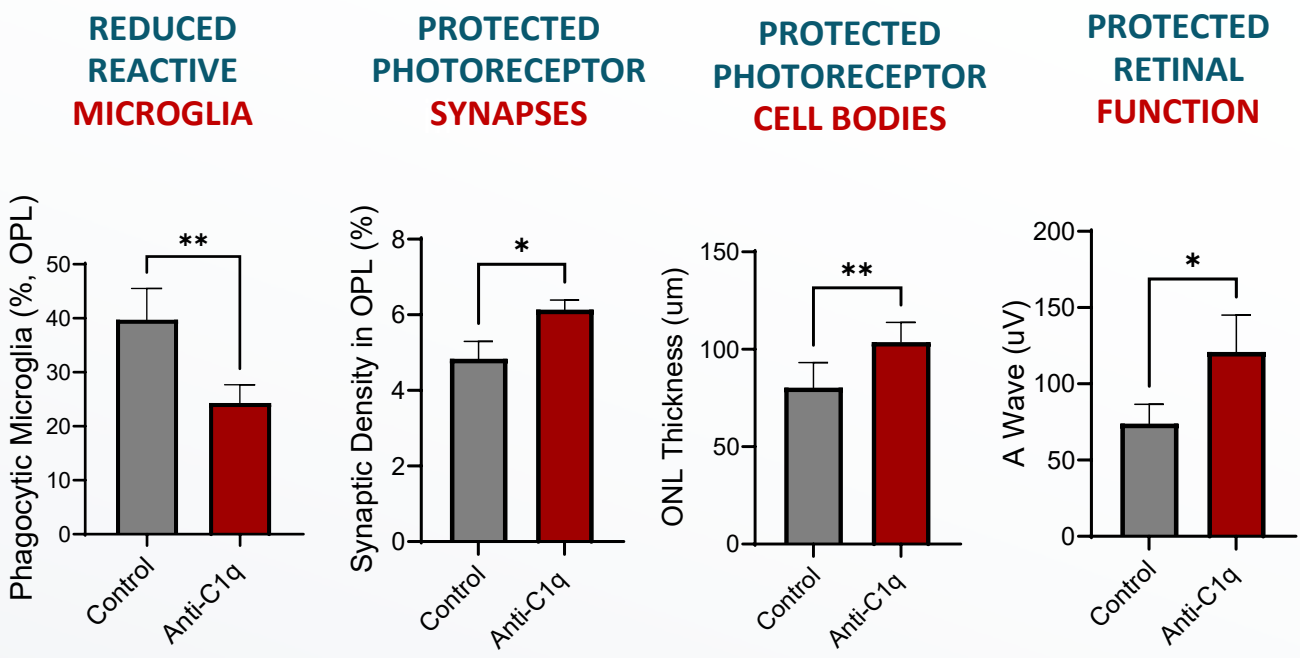
Anti-C1q Protected Photoreceptor Cells and Their Function in Models of Photoreceptor Damage

C1q Deposition on Photoreceptor Cells and Synapses with Light-Induced Damage



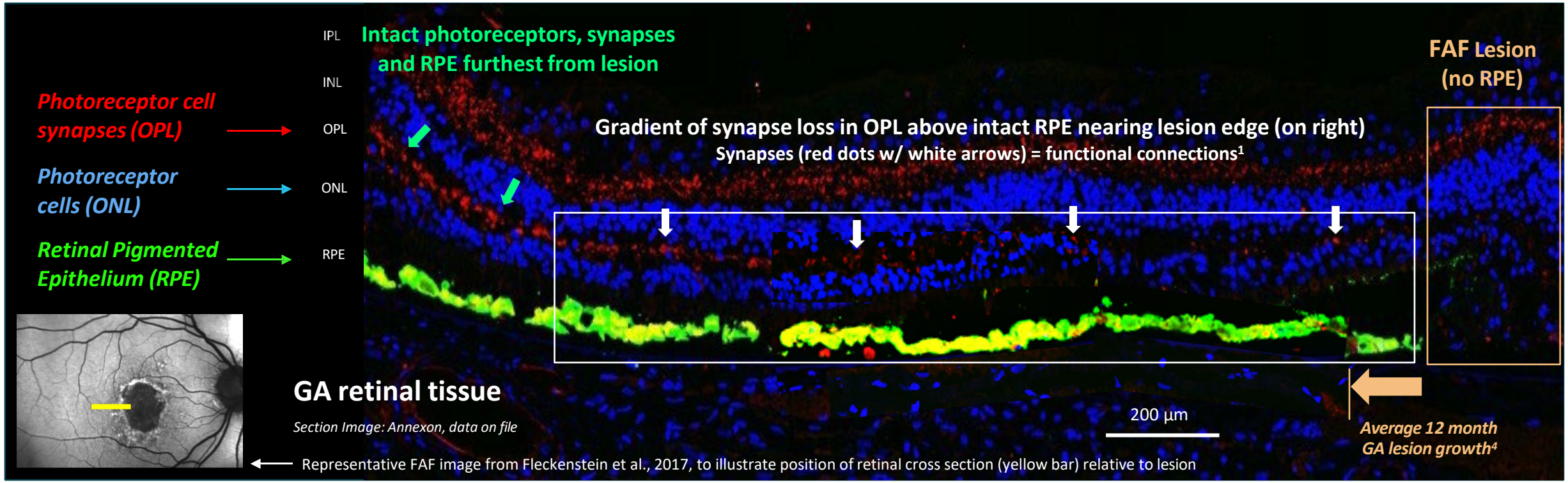
Tassoni, et al., Annexon on file

Anti-C1q Protected Photoreceptors and Function



Photoreceptor Cells, Synapses & Function Lost Prior to RPE in GA

- Photoreceptor cells and their synapses are lost over intact RPE (white box)
  - Decreasing gradient of red-labeled synapses (w/ white arrows) moving toward the lesion on right - loss of synapses is loss of function[1]
  - Also, decreasing gradient of blue-labeled photoreceptor cells toward lesion – photoreceptors are lost prior to RPE[2]
- FAF measures RPE loss/lesion growth, but not photoreceptor or synapse loss and correlates poorly w/ visual function[3]



[1]Selkoe, 2002 doi: 10.1126/science.1074069; Burger, et al., doi.org/10.1016/j.ydbio.2021.04.001; [2]Bird et al., 2014 *JAMA Ophthalmol* doi:10.1001/jamaophthalmol.2013.5799; Li, et al., 2018 *Retina* 38:1937; Pfau, et al., 2020 10.1001/jamaophthalmol.2020.2914; Sarks, et al., 1988 *Eye* 2:552; [3]Heier, et al., 2020 Ophthalmology Retina **4**:673; [4]Shen, et al., 2020 *Ophthalmol Retina* **4**:899

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ARCHER Trial Overview

Geographic Atrophy (GA): Progressive and Life-altering Disease that Remains a Leading Cause of Blindness in Elderly People

- Advanced form of age-related macular degeneration (AMD)
- Chronic, progressive neurodegenerative disease of the eye with irreversible vision loss
- 1M people diagnosed in US; 2.5M in EU
- Diagnosis can be traumatic and impact the social and financial aspects of patients lives, including reading, daily activities and recognizing faces
- **No currently approved therapies have demonstrated preservation of visual function**
- **Urgent unmet need to protect against vision loss**

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ANX007: Differentiated Inhibitor of C1q and Classical Complement to Treat Geographic Atrophy

ANX007

IVT administered antigen-binding fragment (Fab)

KEY ATTRIBUTES

- ✓ **Design: Modeled after established IVT-administered Fab antibodies**; **same antigen recognition structure as ANX005** – full length anti-C1q antibody well tolerated as IV treatment in GBS
- ✓ **Profile:** 50kD Fab antibody; **low viscosity / non-pegylated**; <10 pM potency formulated for intravitreal administration
- ✓ **Dosing:** 5 mg / 100 microliters in Ph2; PK in patient aqueous humor supports monthly/every other month dosing; **25 microliter dose in Ph3**
- ✓ **Specificity: Full target engagement** / inhibition of classical complement pathway observed; **lectin and alternative pathway in place** for immune and homeostatic functions[1]

[1]Sun, et al., 2023 Ophthal Sci 3(2):100290

ARCHER: Phase 2 Trial of C1q Inhibitor ANX007 in GA Patients

Randomized, double-masked
Included **foveal and non-foveal** lesions
Stratified for lesion location and lesion size
12 months (n=270)

- **Sham monthly or every other month** (n=89)
- **ANX007 5mg monthly (EM)** (n=89)
- **ANX007 5mg every other month (EOM)** (n=92)

PRIMARY BIOMARKER ENDPOINT

Change in GA lesion area as assessed by fundus autofluorescence at Month 12

PRESPECIFIED SECONDARY FUNCTIONAL ENDPOINTS

Best Corrected Visual Acuity (BCVA)
Low Luminance Visual Acuity (LLVA) & Deficit (LLVD)

Off-treatment
(6 months)

END OF STUDY
Month 18

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Patient Demographics and Study Eye Characteristics Generally Well-Balanced Across Groups

CHARACTERISTIC	SHAM POOLED (N=89)	ANX007 EM (N=89)	ANX007 EOM (N=92)
Age, mean (SD)	79.8 (7.49)	79.7 (8.64)	80.5 (8.53)
Female, n (%)	59 (66.3%)	47 (52.8%)	60 (65.2%)
Caucasian, n (%)	87 (97.8%)	87 (97.8%)	89 (96.7%)
Mean BCVA, mean (SD)	**58.5 (16.2)**	**58.8 (17.2)**	**58.3 (15.0)**
Foveal Lesion	49.4%	57.3%	53.3%
GA Lesion Size (mm^2), mean (SD)	7.28 (3.99)	7.28 (3.96)	7.53 (4.10)
GA Lesion < 7.5 mm^2	61.8%	58.4%	57.6%
Fellow Eye CNV	22.5%	24.7%	17.4%
Multifocality, n (%)	65 (73.0%)	61 (68.5%)	67 (72.8%)

Discontinuations Consistent with Previous GA Studies

	SHAM POOLED (N=89)	ANX007 EM (N=89)	ANX007 EOM (N=92)
Discontinued treatment	**10 (11.2%)**	**13 (14.6%)**	**11 (12.0%)**
Withdrawal by subject			
Unrelated health issues	2	1	2
Moved / travel / time	1	2	2
Personal reasons / no reason given	2	3	2
Other	1	2	---
Death	2	2	3
Lost to follow-up	1	2	2
Physician decision	1	1	---

BCVA: Widely Accepted Functional Endpoint of Visual Acuity

BCVA 15-letter change or Mean BCVA change used in multiple sham-controlled pivotal trials

BEST CORRECTED VISUAL ACUITY (BCVA)

15-Letter Loss

20/60 to 20/120 vision

PRODUCT	PRIMARY ENDPOINT MEASURE
Wet AMD	
Lucentis	Trial 1 & 2: BCVA ≥15 letter Trial 3 & 4: mean BCVA change
Eylea	BCVA ≥15 letter
Vabysma	Mean BCVA change
DME	
Lucentis	BCVA ≥15 letter
Eylea	Mean BCVA change
Vabysma	Mean BCVA change
Iluvien	BCVA ≥15 letter
Retinal Vascular Occlusion (BRVO/CRVO)	
Lucentis	BCVA ≥15 letter
Eylea	BCVA ≥15 letter
Ozurdex	BCVA ≥15 letter

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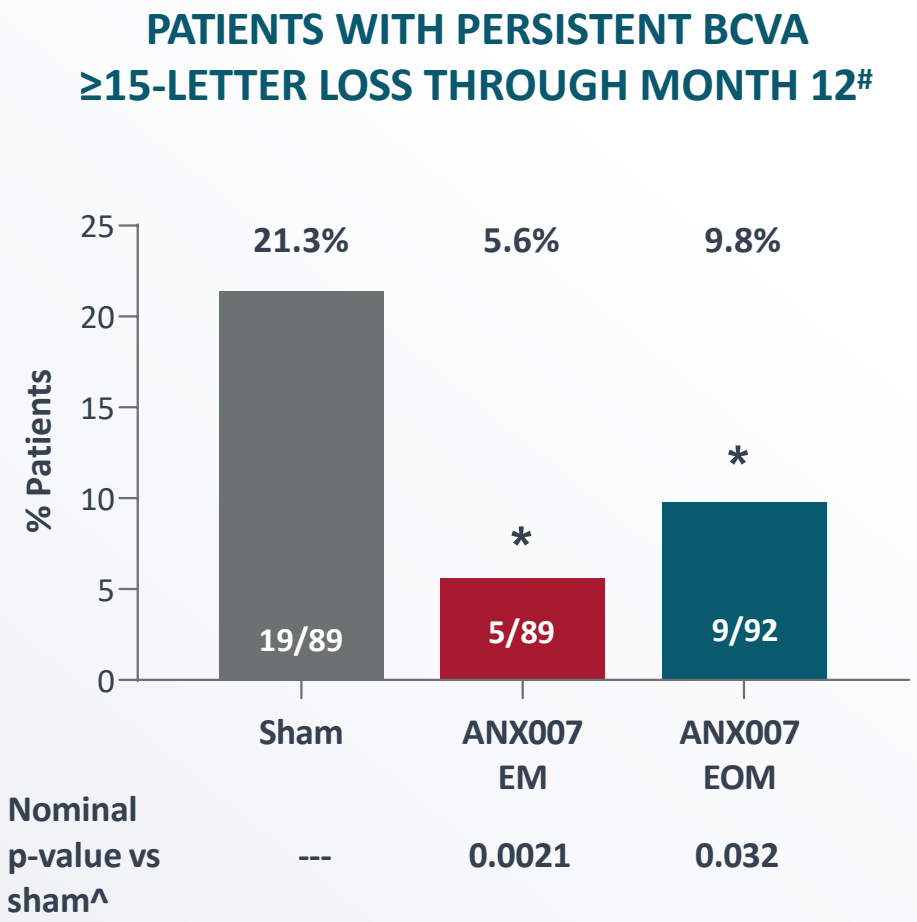
ARCHER Trial
Visual Acuity Results

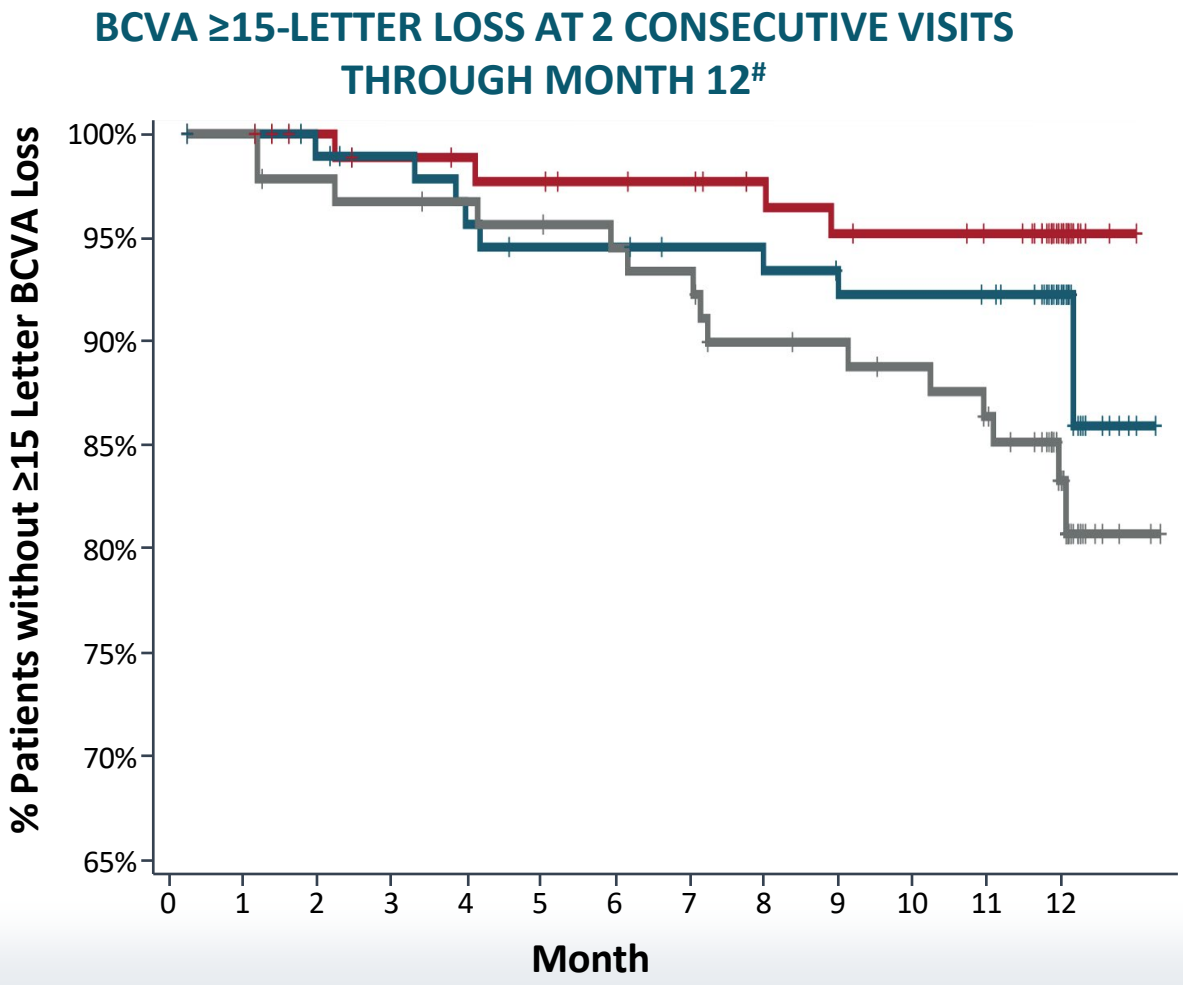
ANX007 Demonstrated Statistically Significant Protection From Vision Loss as Measured by BCVA ≥15-Letter Loss



#Persistent for two consecutive visits through month 12 or at last study visit
^Nominal p-value from a Chi-square test in ITT population: * Nominal $p < 0.05$
Final data

- First known significant preservation of vision in GA
- Dose-dependent response
- BCVA ≥15-letter loss universally deemed clinically meaningful

Significant, Time-Dependent Protection From ≥15-Letter Vision Loss with ANX007 Monthly Treatment



EM (n=89)

73% Risk Reduction ANX007 EM

HR (CI) = 0.272 (0.090 to 0.819); p = 0.0098

EOM (n=92)

50% Risk Reduction ANX007 EOM

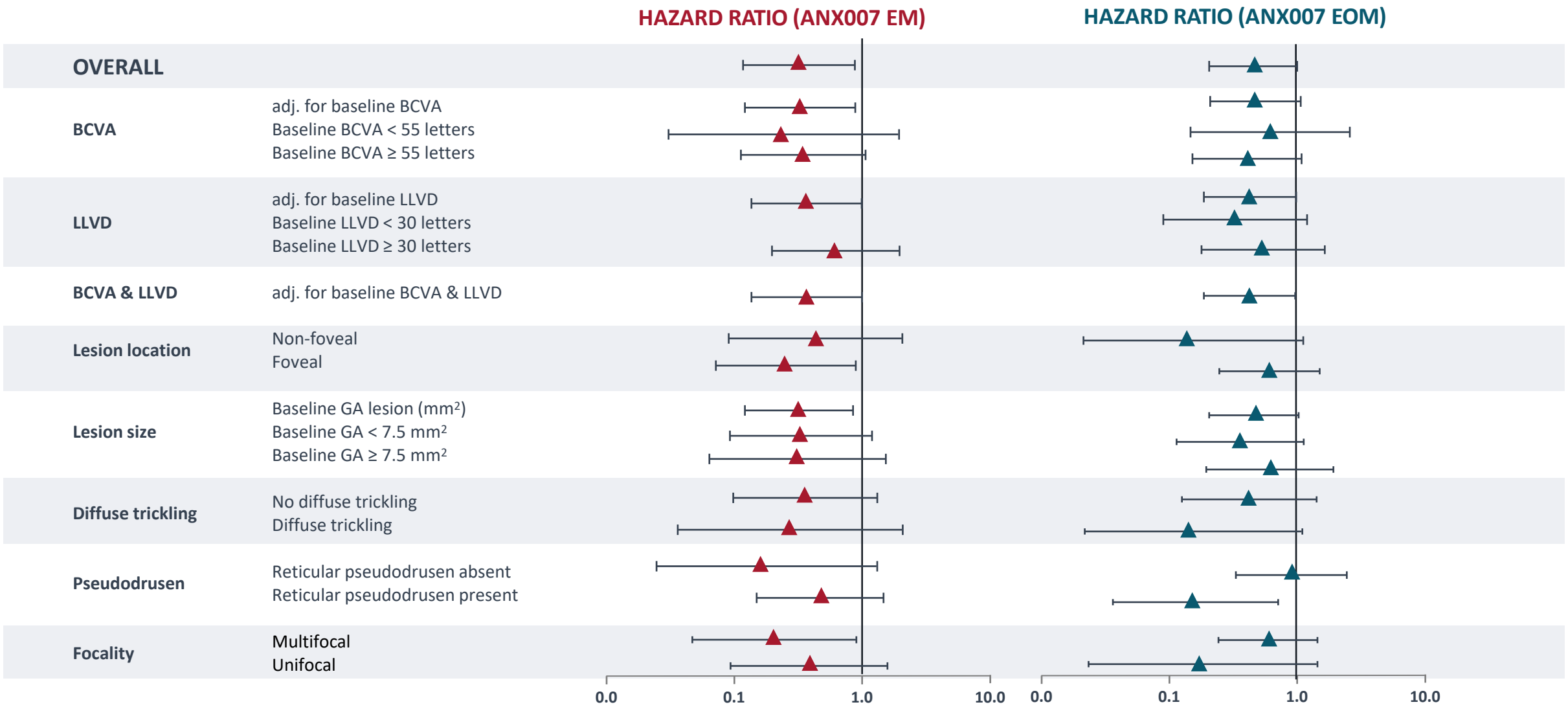
HR (CI) = 0.504 (0.214 to 1.190); p = 0.0788

Sham (n=89)

Increasing ANX007 Impact Over Time

HR, hazard ratio; Nominal log-rank test (versus sham) p-values are presented;
#Persistent BCVA 15-LL at two consecutive visits including month 12 supported by ensuing (off-treatment) visit
Final data

ANX007 Protection from Vision Loss Consistent Across Baseline Characteristics



*persistent for two consecutive visits through month 12 or at last visit; Hazard ratios are from Cox regressions accounting for event time and censorship
NOTE: Hazard ratio not estimated for ANX007 EM vs Sham with baseline LLVD < 30 due to zero (0) event in ANX007 EM group for the subgroup.

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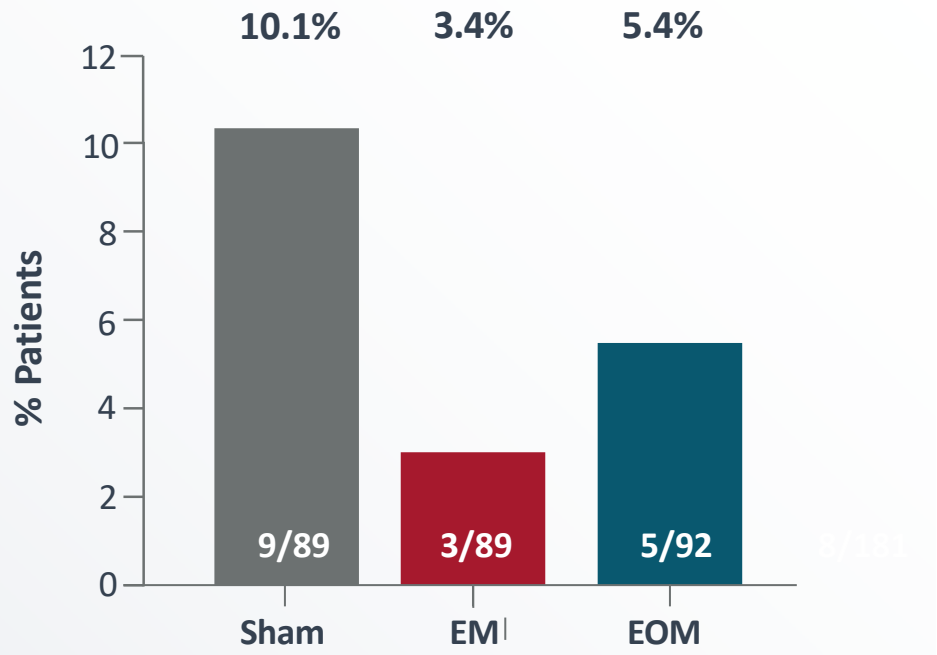
ANX007 Effect on BCVA ≥15-Letter Loss Improves with Longer Treatment

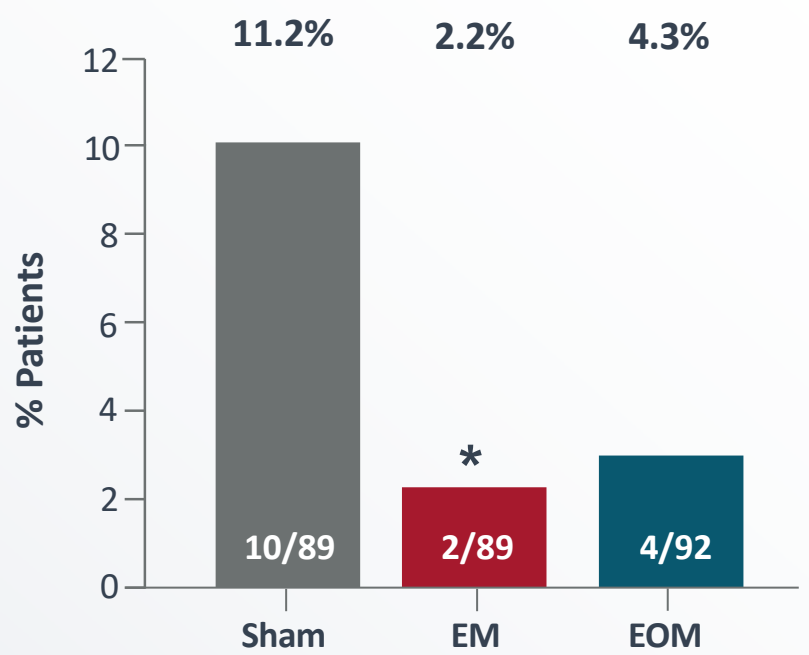
PATIENTS WITH PERSISTENT BCVA ≥15-LETTER LOSS

STUDY MONTHS 0-6



Nominal p-value vs sham^	---	0.0729	0.2389

STUDY MONTHS 6-12



Nominal p-value vs sham^	---	0.0168	0.0829

⁺Persistent for two consecutive visits through month 12 or at last visit; ^Nominal p-value from a Chi-square test in ITT population; Nominal P < 0.05*

Increasing ANX007 Impact Over Time

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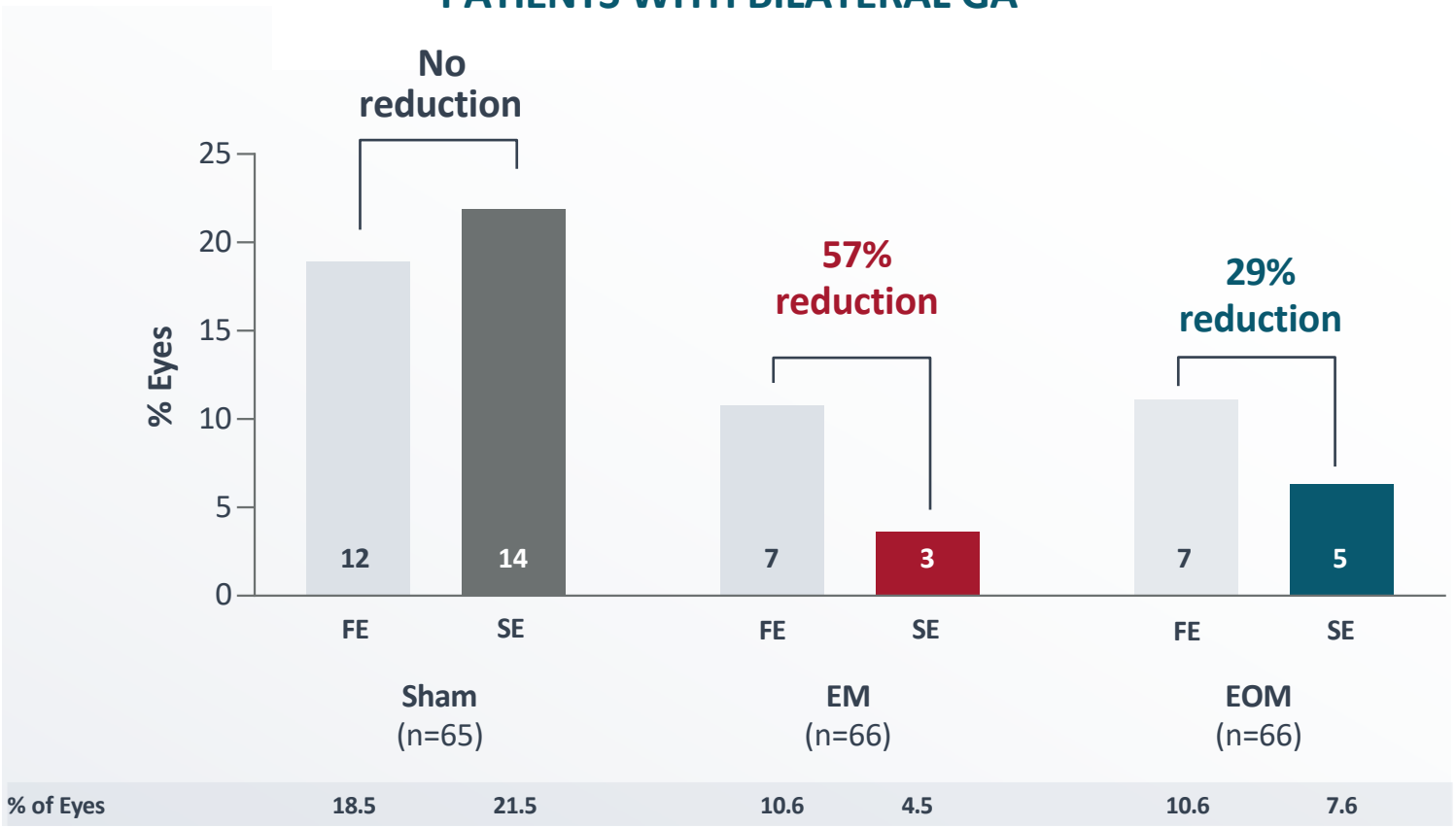
Protection From Vision Loss Supported by Fellow Eye Analysis

EYES WITH ≥15-LETTER BCVA LOSS AT MONTH 12 IN ALL PATIENTS WITH BILATERAL GA



EM, every month; EOM, every other month; Pooled: EM+EOM; FE, fellow eye; SE, study eye
All patients with bilateral GA were included due to small sample size

- Sham: No reduction in BCVA vision loss study vs. fellow eye
- Dose dependent protection from vision loss in ANX007 treated study eyes relative to fellow eyes
  - EM: 57% reduction in 15-letter loss
  - EOM: 29% reduction in 15-letter loss

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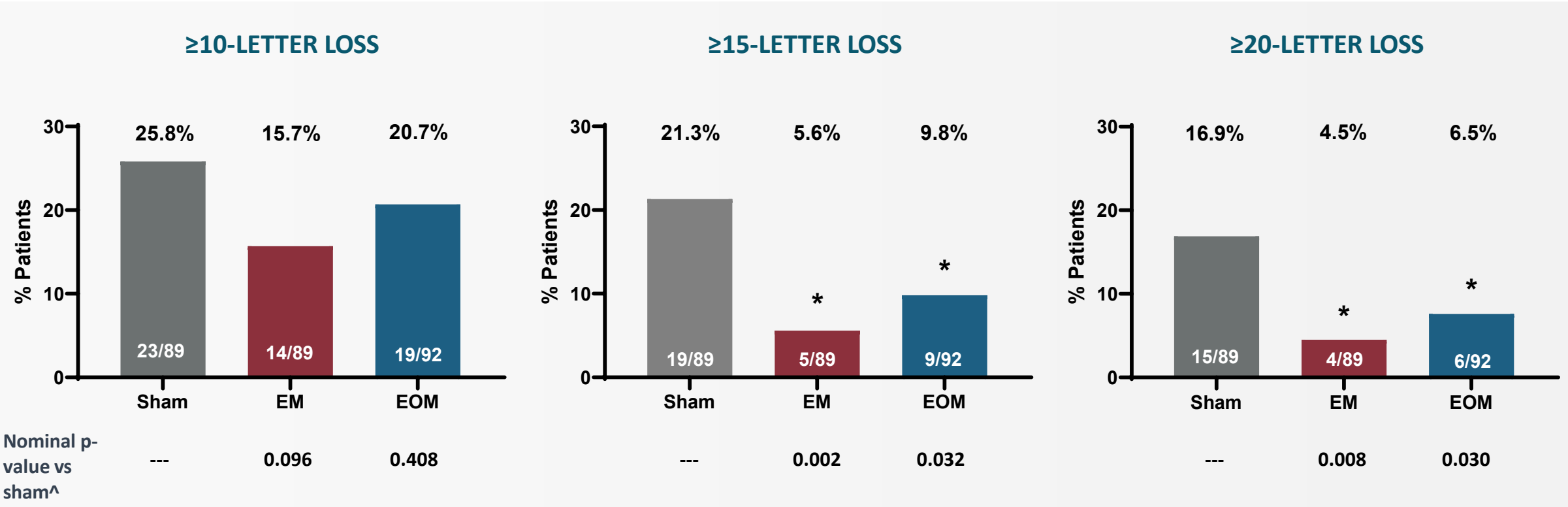
Consistent Protection from Vision Loss with BCVA ≥10, ≥15 and ≥20-Letter Assessments

Persistent BCVA Vision Loss Through Month 12#



#Persistent for two consecutive visits through month 12 or at last visit
^Nominal p-value from a Chi-square test in ITT population
* P < 0.05

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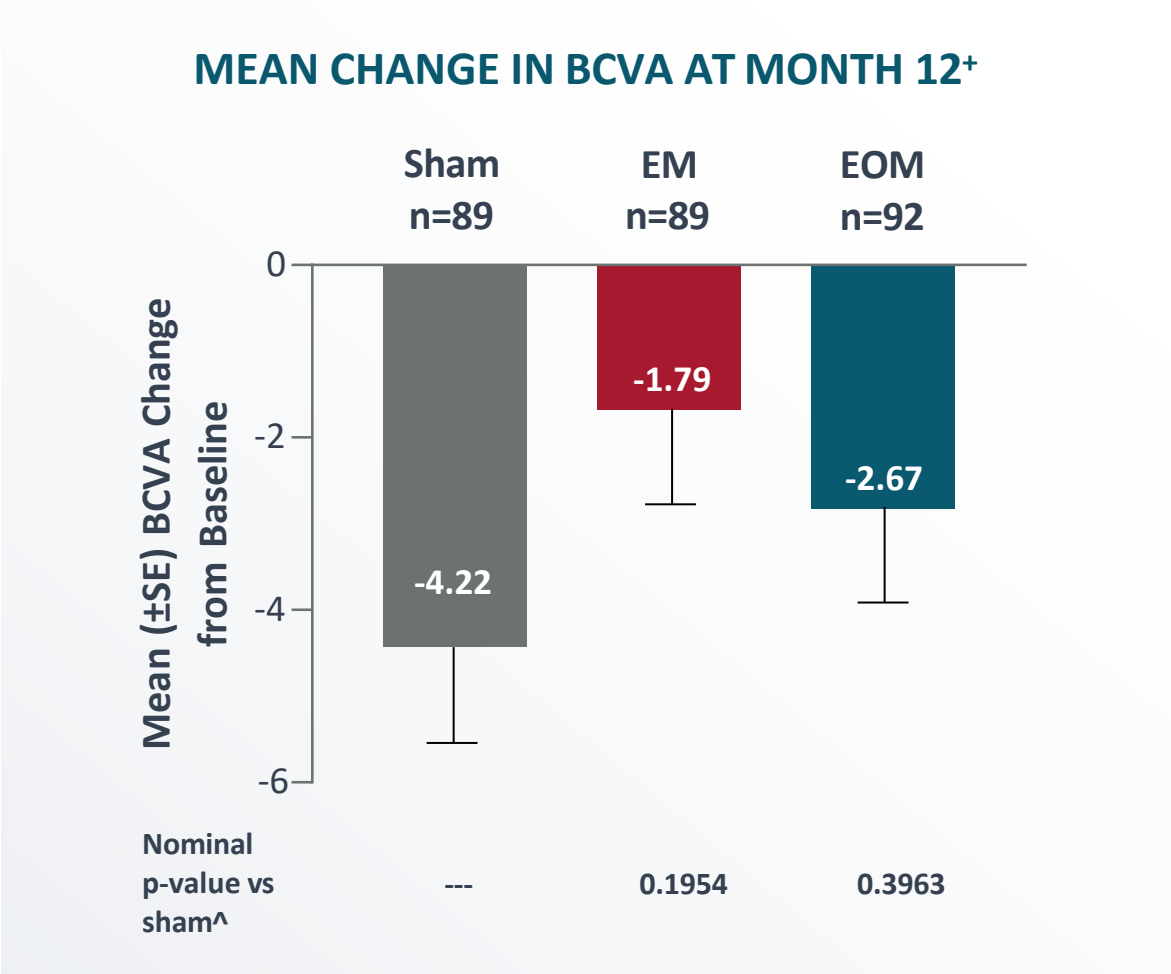
Mean Change in BCVA at Month 12 Further Supports Consistent Protection From Vision Loss with ANX007 Treatment



- Trend for dose-dependent response in ANX007 treated groups
- BCVA loss in sham through 12 months consistent with previous GA trials[1,2,3]

+Mean, standard error (SE), and p-value based on MMRM adjusting for baseline lesion location, lesion focality, baseline GA lesion, and the baseline GA lesion by visit interaction.
^Nominal p-value from MMRM adjusting for baseline lesion location, lesion focality, baseline GA lesion, and the baseline GA lesion by visit interaction in ITT population

[1]Liao et al (2020) *Ophthalmology* 127: 186-195; [2]Holtz et al (2018) *JAMA Ophthalmology* 136:666-677;
[3]Heier et al, *Retina Society* 2022

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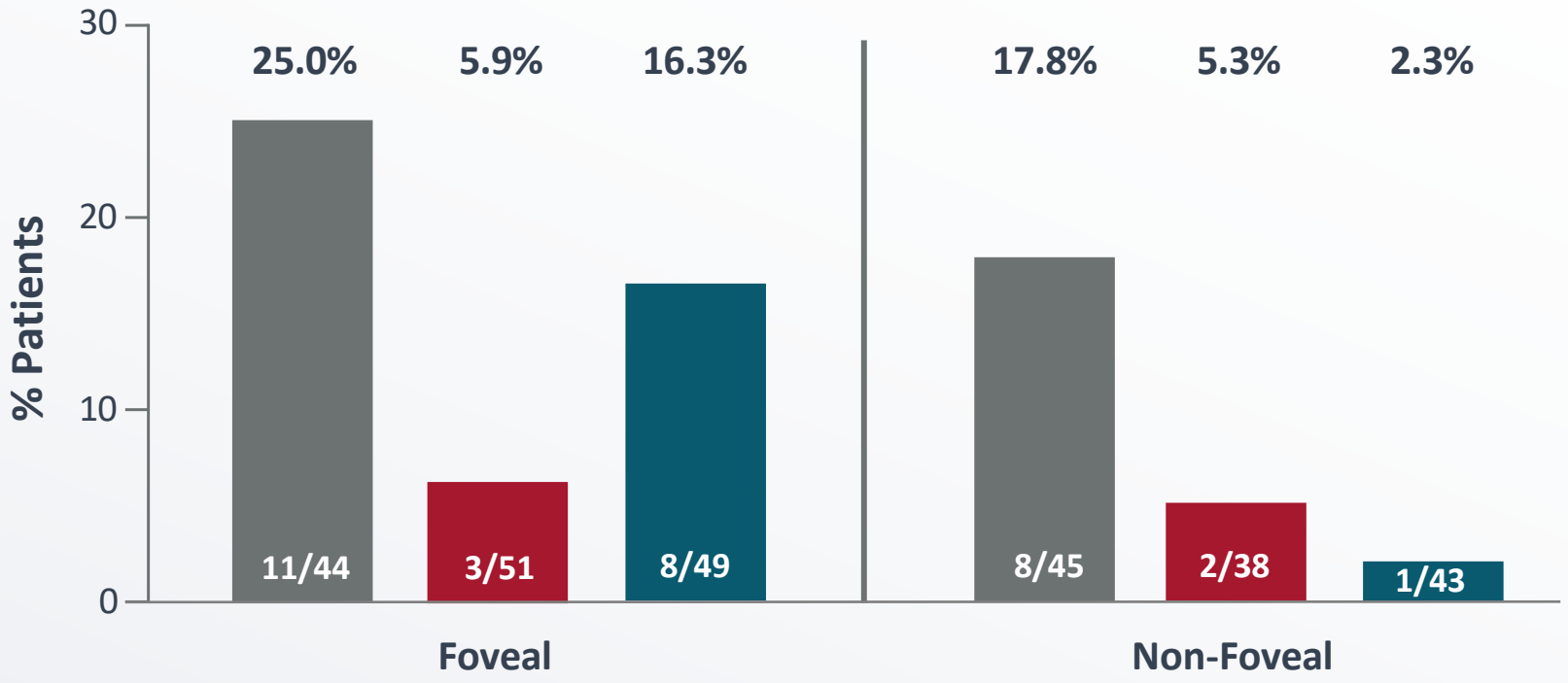
ANX007 BCVA Subgroup Analysis: Protection from Vision Loss Observed in Both Foveal and Non-Foveal Patients

PATIENTS WITH PERSISTENT BCVA ≥15-LETTER LOSS THROUGH MONTH 12#

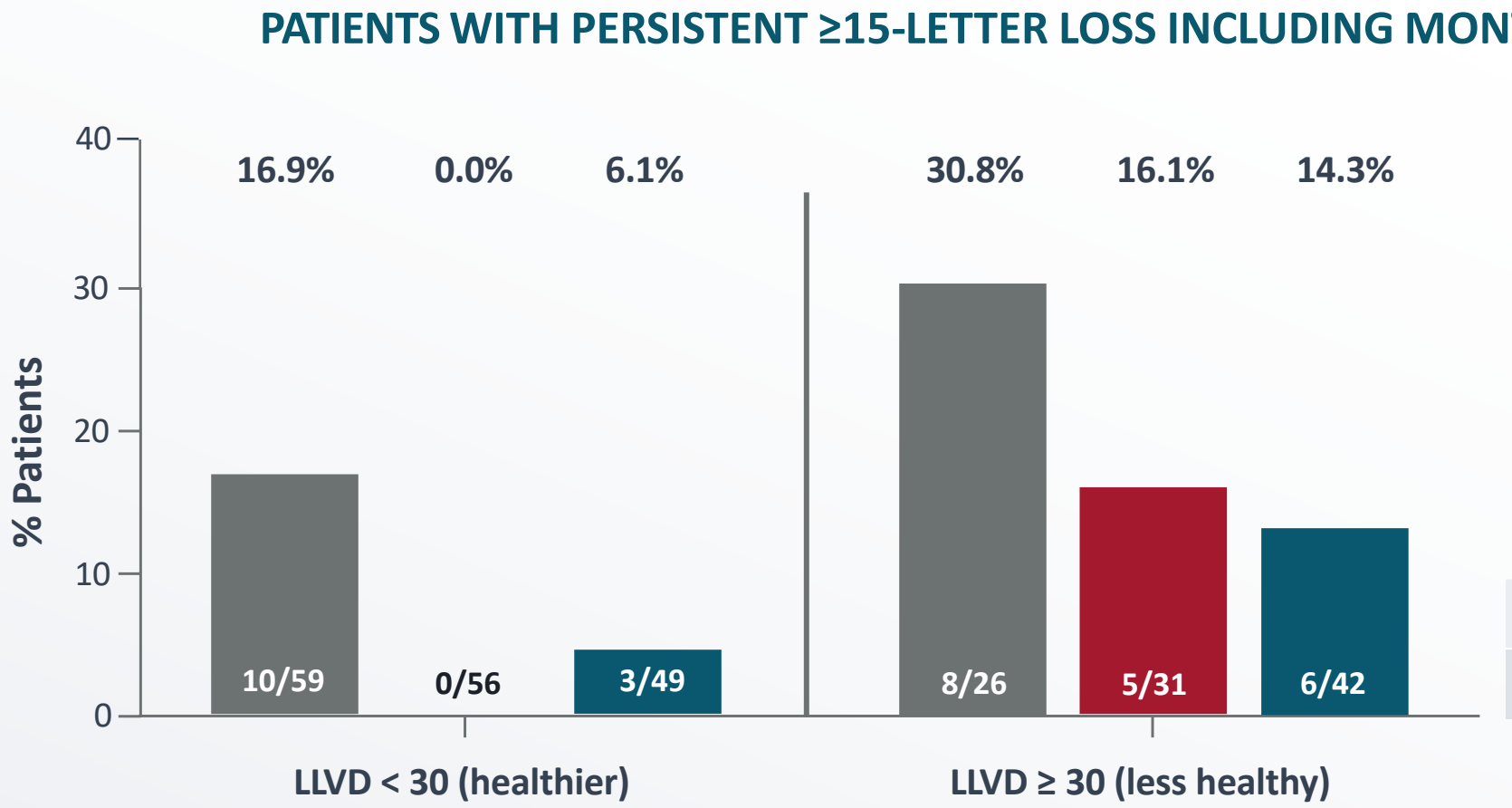


Nominal summary p-value vs. sham^	
ANX007 EM	0.0019
ANX007 EOM	0.0254

#Persistent for two consecutive visits at any time through month 12 or at last study visit
^Nominal p-value from a Cochran Mantel-Haenszel test (General Association) in ITT population
Final data

Greatest Effect of ANX007 in Earlier / Healthier Patients

Protection from vision loss (BCVA ≥15-Letter) based on retina health at baseline



Nominal summary p-value vs. sham^	
EM	0.0016
EOM	0.0183

#Persistent for two consecutive visits including month 12
^Nominal p-value from a Cochran Mantel-Haenszel test (General Association) in ITT population

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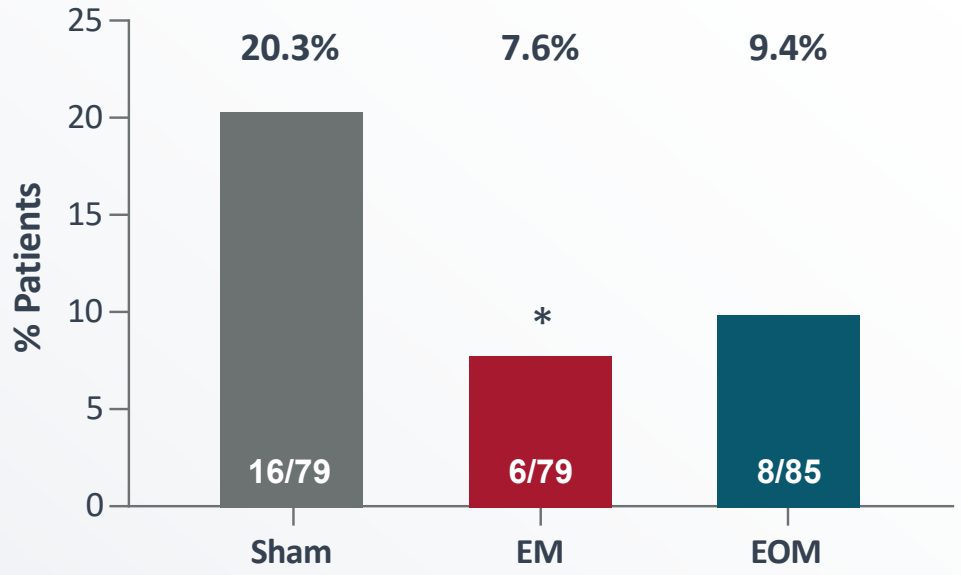
Consistent Protection From Vision Loss with ANX007 Treatment Also Demonstrated with LLVA

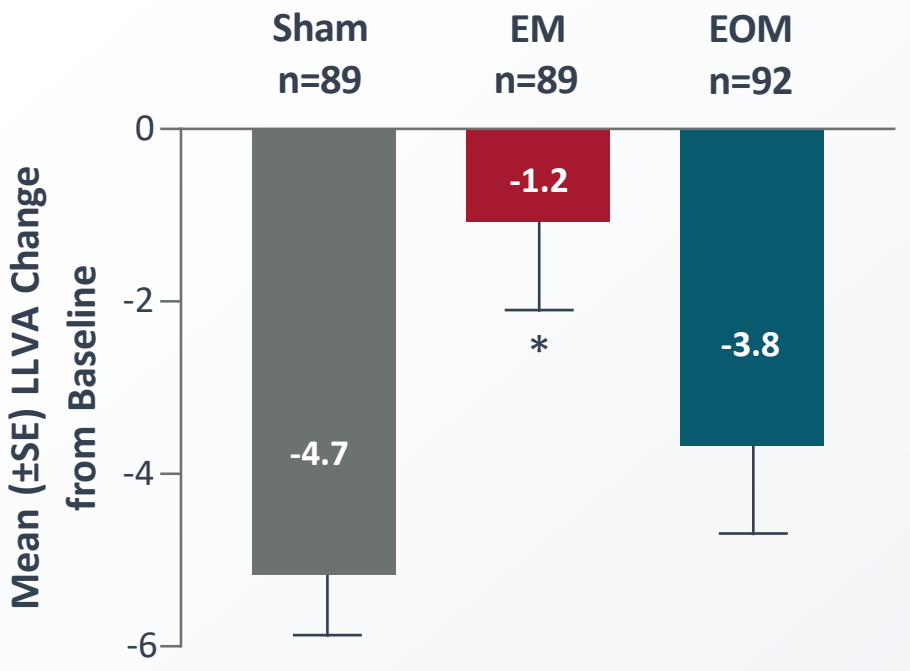
LLVA ≥15-LETTER LOSS THROUGH MONTH 12#



Nominal p-value vs Sham^	--	0.022	0.050

#Patients with single LLVA ≥15-letter loss event and at least one post-baseline LLVA measurement
^Nominal p-value from a Chi-square test
Final data

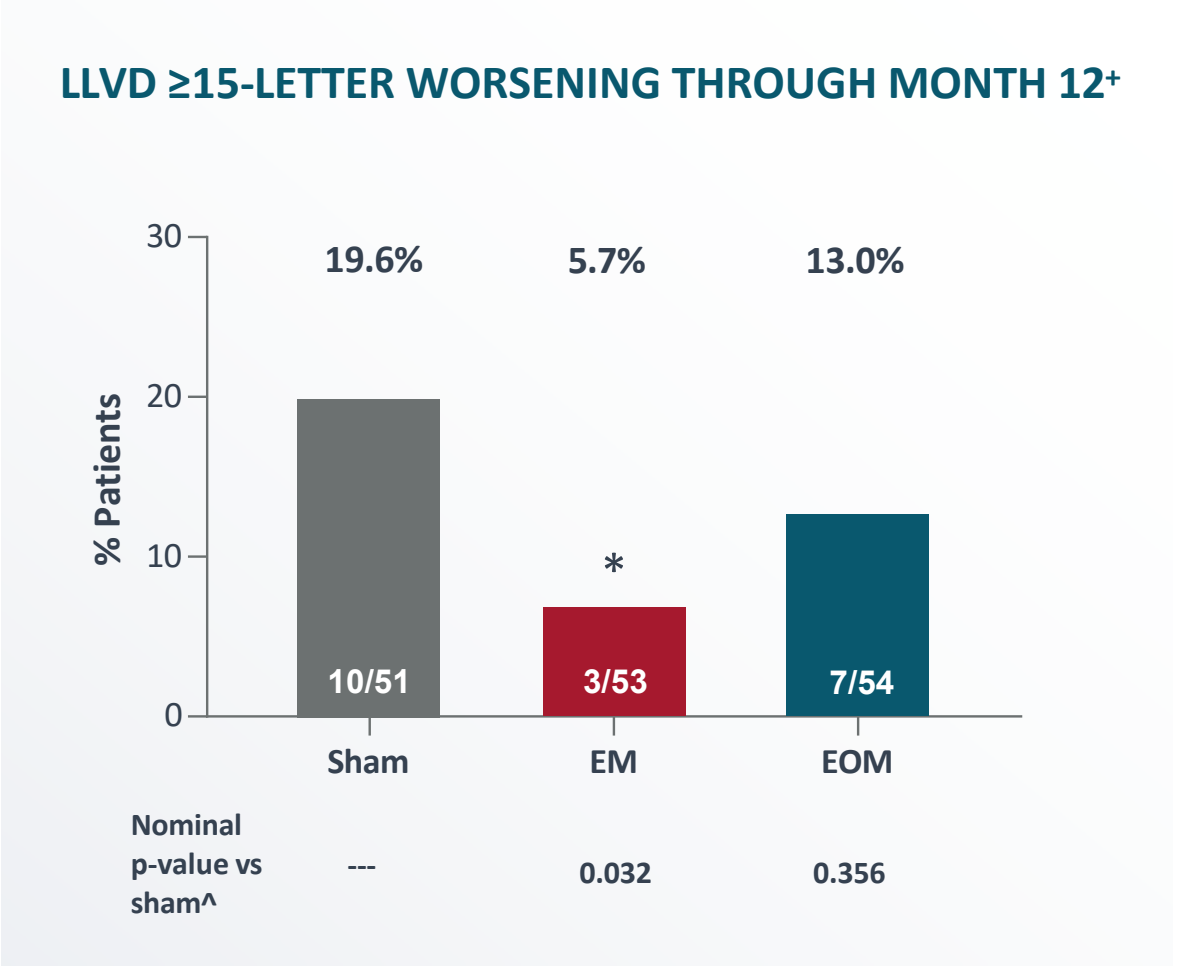
MEAN CHANGE IN LLVA AT MONTH 12+



Nominal p-value vs sham^	---	0.0354	0.5890

+Mean, standard error (SE), and p-value based on MMRM adjusting for baseline lesion location, lesion focality, baseline GA lesion, and the baseline GA lesion by visit interaction.
^Nominal p-value from a Chi-square test in ITT population
* Nominal P < 0.05
Final data

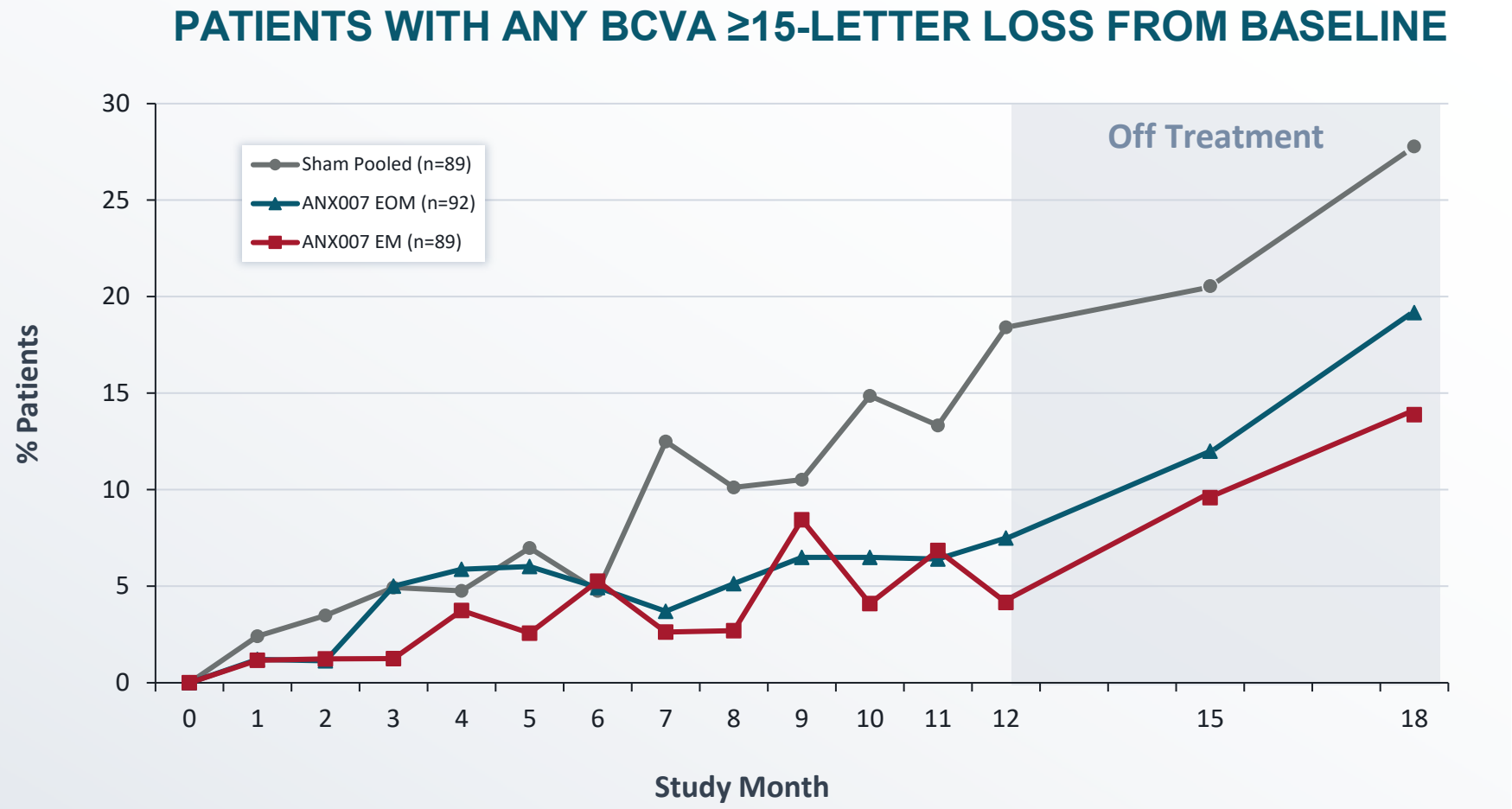
ANX007 Provided Consistent Protection from Vision Loss by LLVD



+in subjects with BCVA ≥55
^Nominal p-value from a Chi Square test
*p<0.05

BCVA ≥15-Letter Loss Accelerated After Cessation of Treatment

Consistent with true on-treatment drug effect and disease-modifying mechanism of action



- Low frequency (<0.6% per month) of single BCVA ≥15-letter losses in EM- and EOM-treated groups during 12-month treatment period
- While benefit was maintained after treatment cessation the rate of BCVA≥15 LL increased to parallel that of sham (>1.6% per month)

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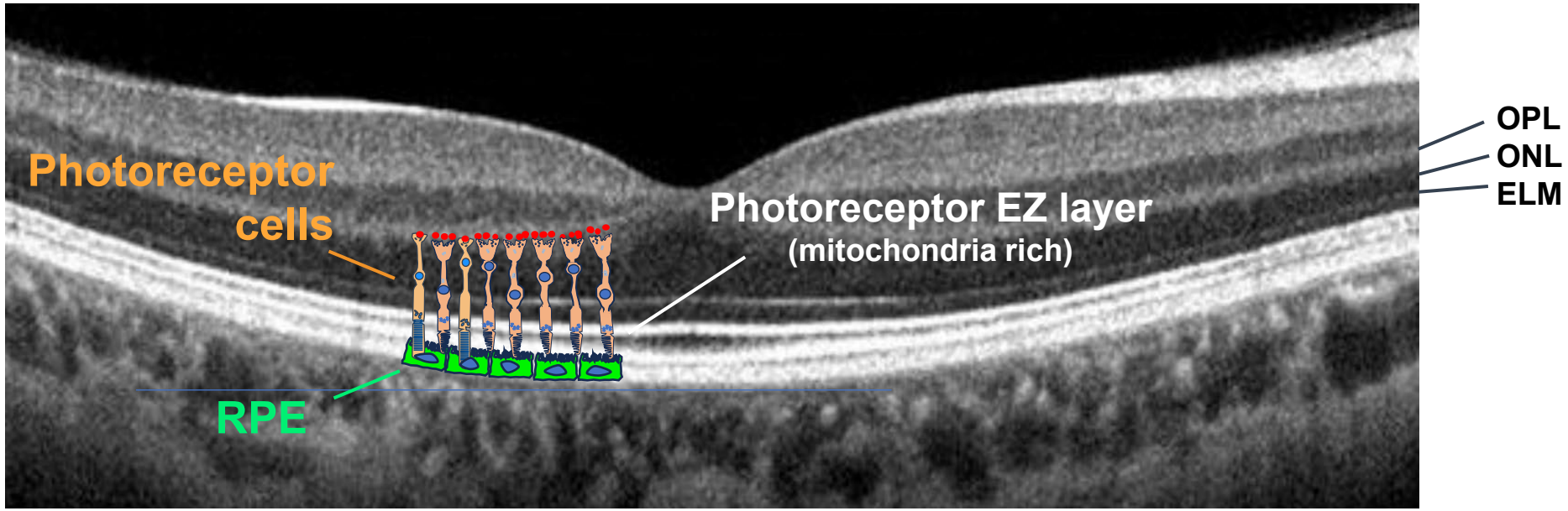
ANX007 Impact on Retinal Structure

Change in OCT Ellipsoid Zone (EZ) Directly Measures Photoreceptor Anatomy



octscans.com

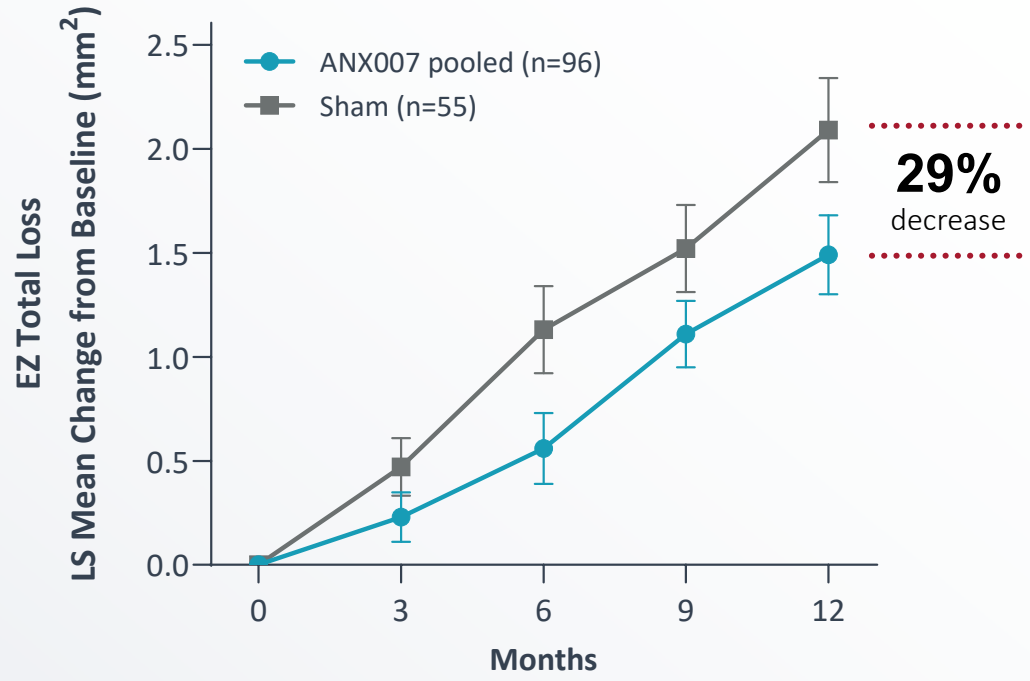
ARCHER EZ Population

Sham	ANX007 EM	ANX007 EOM	Total
71	60	62	193

- 193 patients with OCT scans from Heidelberg Spectralis
- Patient demographics and study eye characteristics were generally well balanced across groups
- Same treatment effect between sham, EM and EOM groups as in whole study population

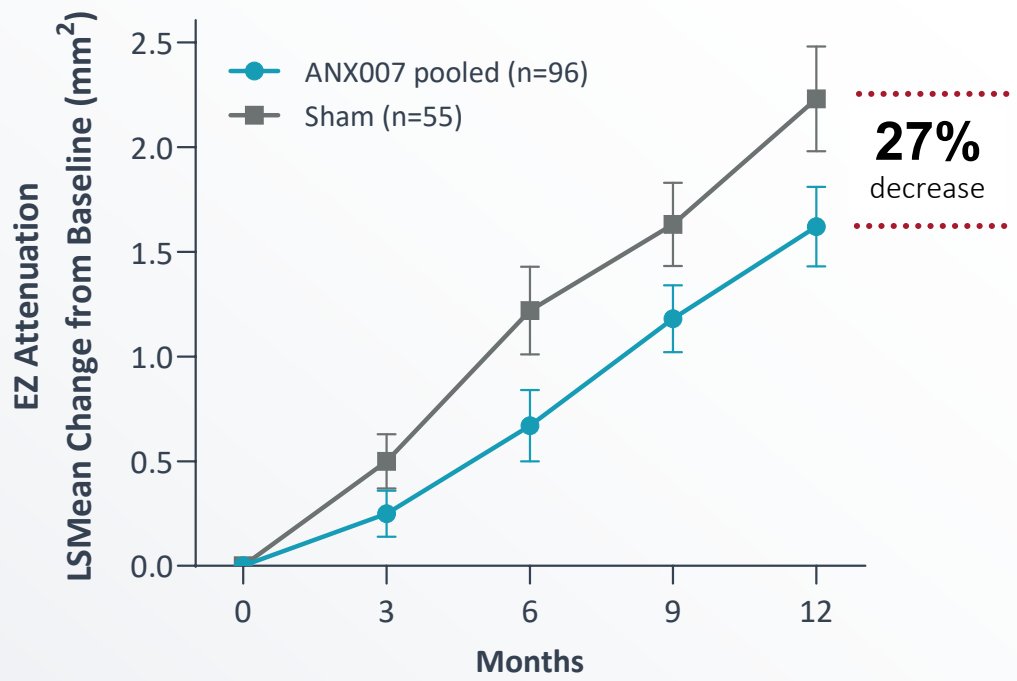
ANX007 Significantly Protected Photoreceptors Across Retina Through 12 Months

EZ TOTAL LOSS (EZ = 0 μm)*



Nominal p-value vs sham^ ANX007 0.017

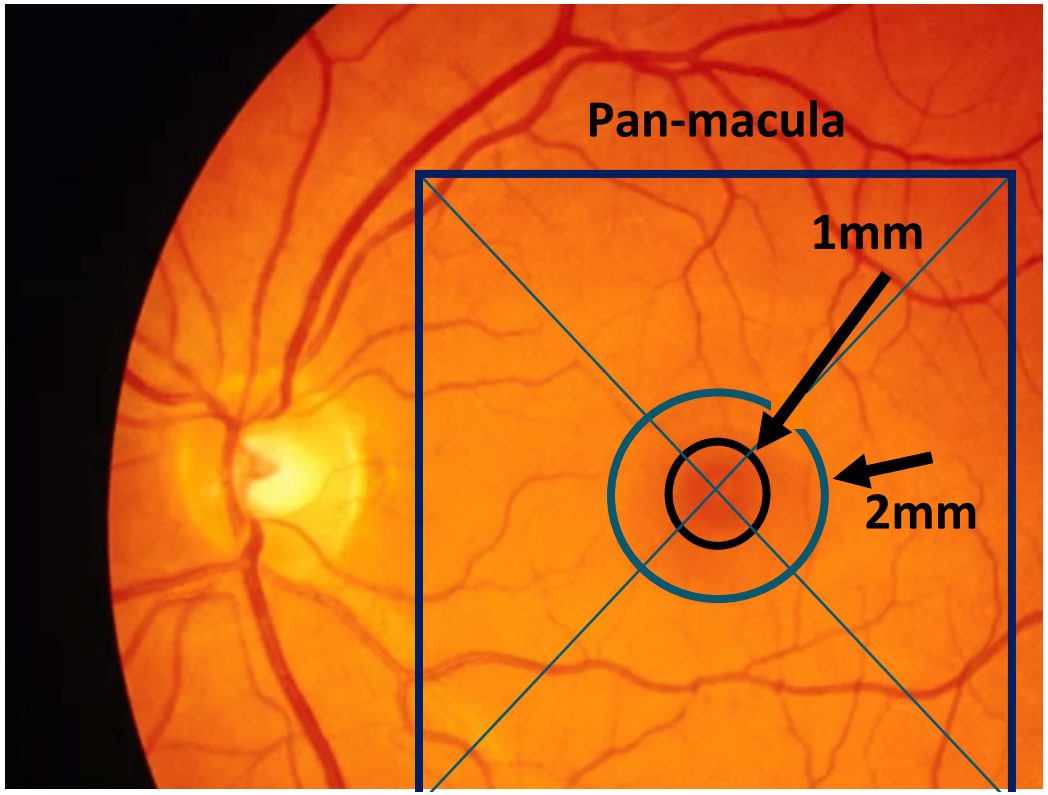
EZ ATTENUATION (EZ < 20 μm)*



Nominal p-value vs sham^ ANX007 0.021

^Nominal p-values from a mixed model for repeated measures (MMRM) analysis; Heidelberg Spectralis OCT population with baseline OCT data (n=151)
*Two treatment groups (EM and EOM) were not different statistically

EZ Disruption in Central Fovea, Not Across Full Retina, Correlates with BCVA in GA Patients^



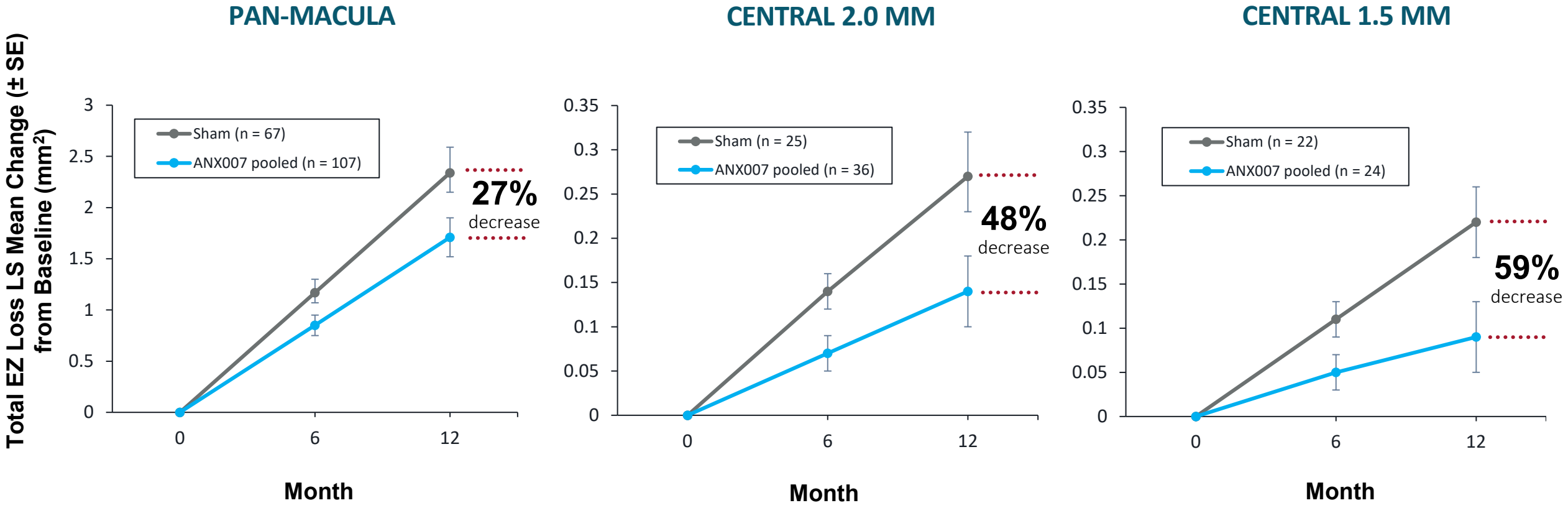
Parameter	Region	Correlation with GA Eyes (Pearson r value)
EZ Loss	1mm	**-0.49***
	2mm	**-0.54***
	Pan-macula	-0.34 (ns)

*p≤0.05

^From Yordi et al (2024) J Pres Med 14: 543

Photoreceptor Protection Through 12 Months in Central Fovea

More robust protection with ANX007 in center, area best associated with vision, compared to pan-macula

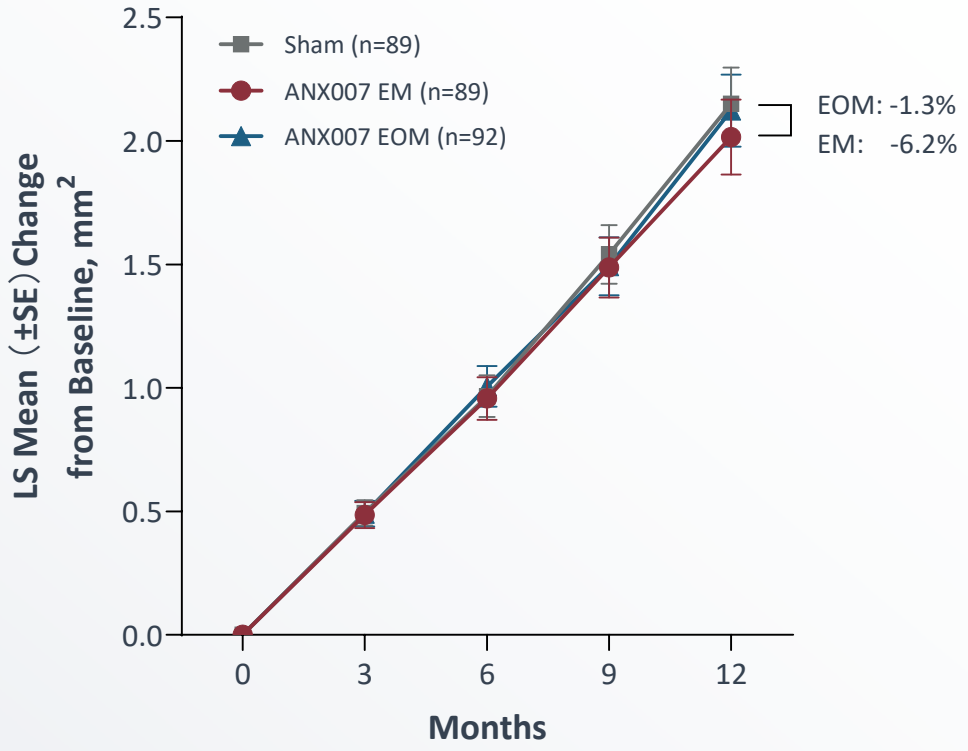


Nominal p-value^		
	ANX007 Pooled vs Sham 0.0457	ANX007 Pooled vs Sham 0.0218	ANX007 Pooled vs Sham 0.0319

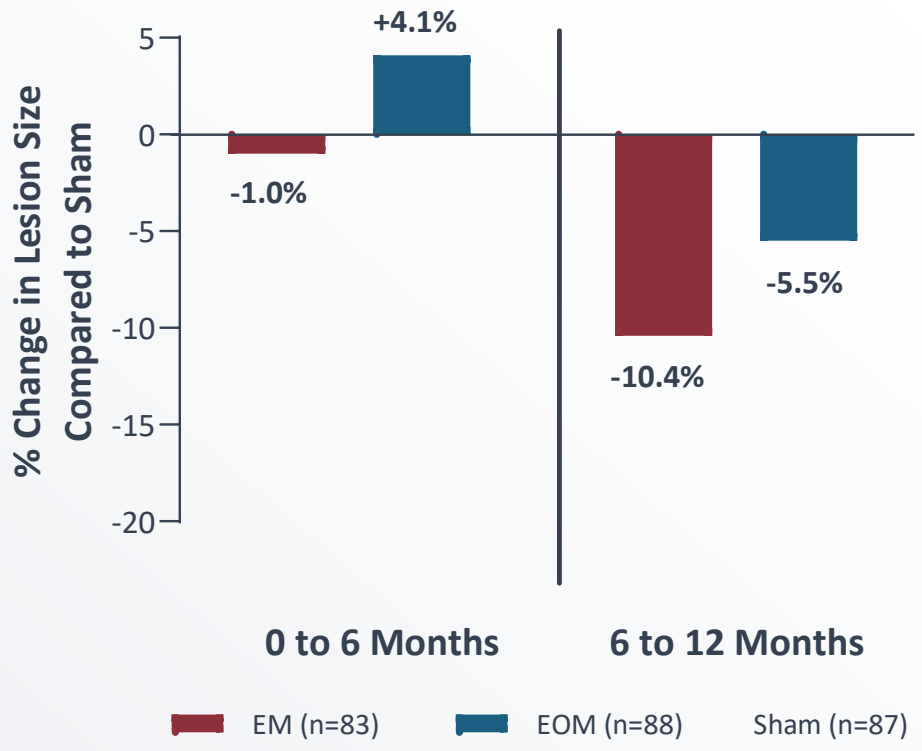
^Nominal p-values from a linear mixed model for repeated measures model (slope) analysis; Heidelberg Spectralis OCT population with baseline OCT data, excludes patients with >98% atrophy/attenuation at baseline

ANX007 Did Not Significantly Reduce RPE Loss Across Full Retina, but Effects Increased Over Time

RPE LOSS FROM BASELINE TO MONTH 12#



CHANGE IN RPE LOSS OVER TIME#

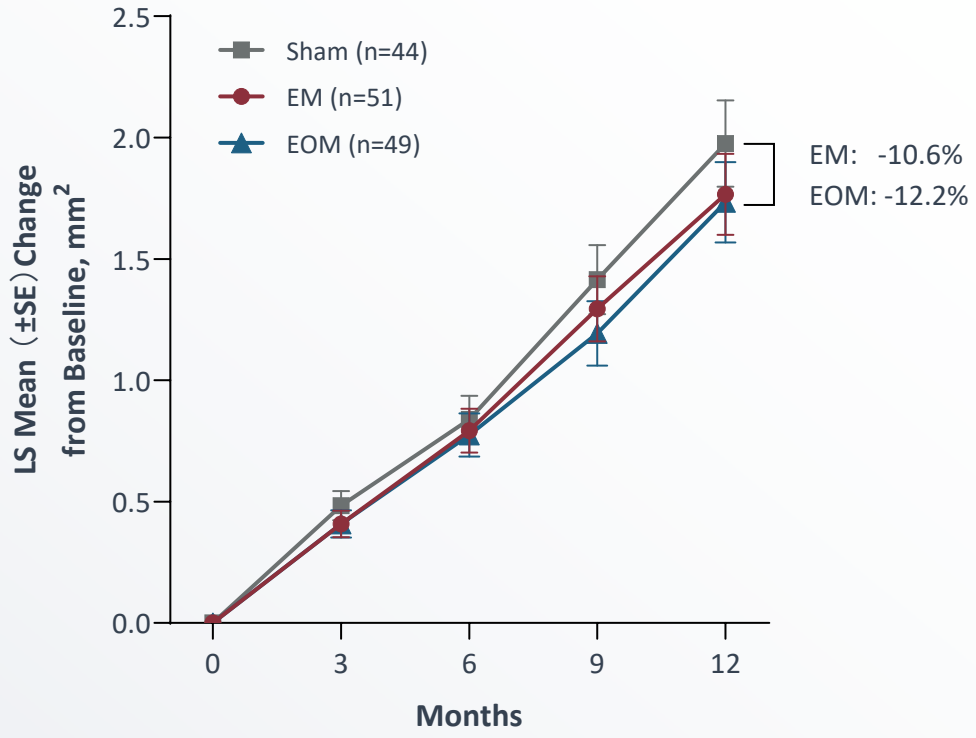


#Least-square (LS) mean and its standard error (SE) are based on a mixed-effect model for repeated measures (MMRM) adjusting for baseline lesion location, lesion focality, baseline GA lesion, and the baseline GA lesion by visit interaction.

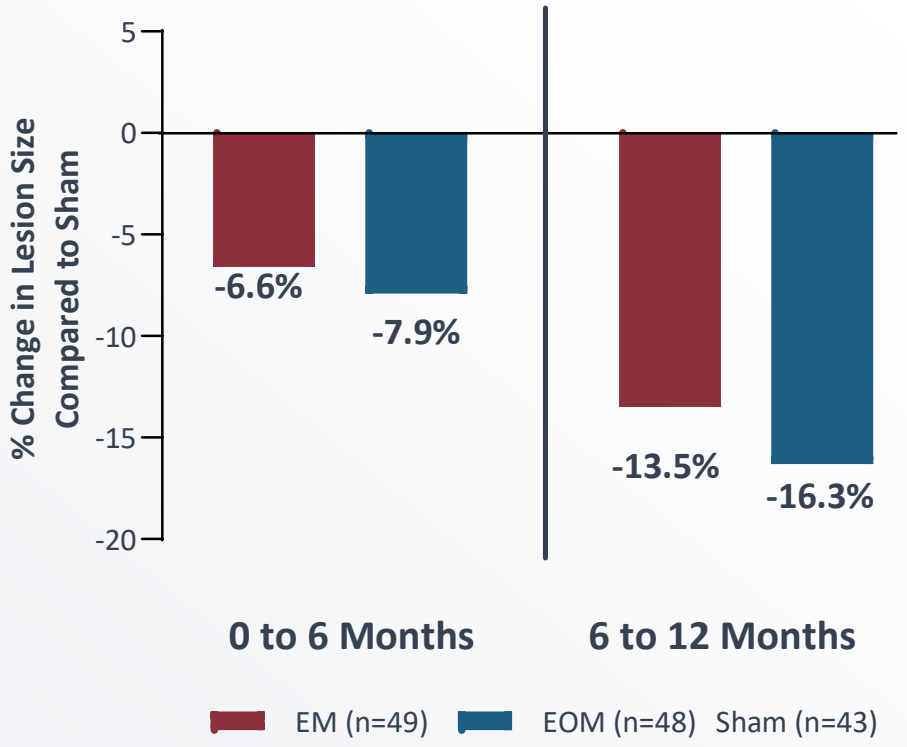
Stronger Impact on RPE Loss in Patients with Foveal Involvement at Baseline – Suggesting Differential ANX007 Effect in Foveal Center

Greater protection of RPE in region responsible for visual acuity

RPE LOSS FROM BASELINE TO MONTH 12[#] FOVEAL PATIENTS



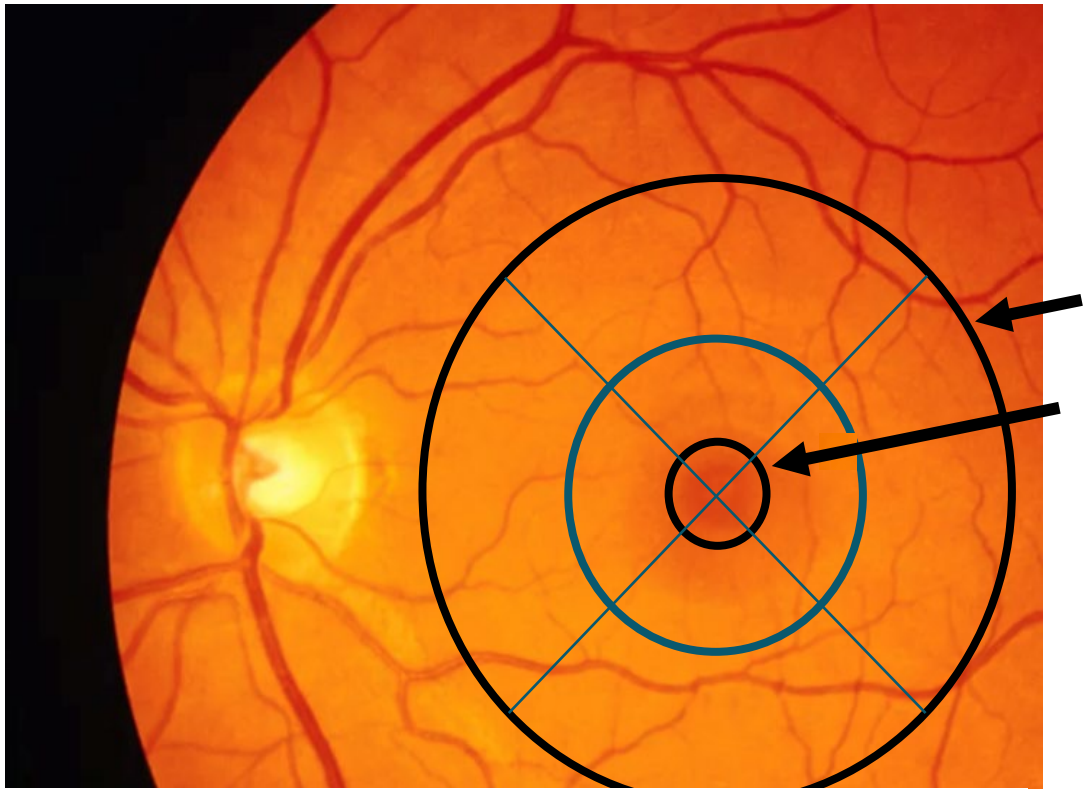
CHANGE IN RPE LOSS OVER TIME[#] FOVEAL PATIENTS



[#]Least-square (LS) mean and its standard error (SE) are based on a mixed-effect model for repeated measures (MMRM) adjusting for baseline lesion location, lesion focality, baseline GA lesion, and the baseline GA lesion by visit interaction.

RPE Loss within the Central Fovea Correlates with BCVA Loss[1]

Correlation in central 1mm seen as early as 6 months; RPE loss across full retina not well correlated with BCVA loss



Spearman Correlation Coefficients Comparing the Changes in RPE Area with BCVA Change Over Time

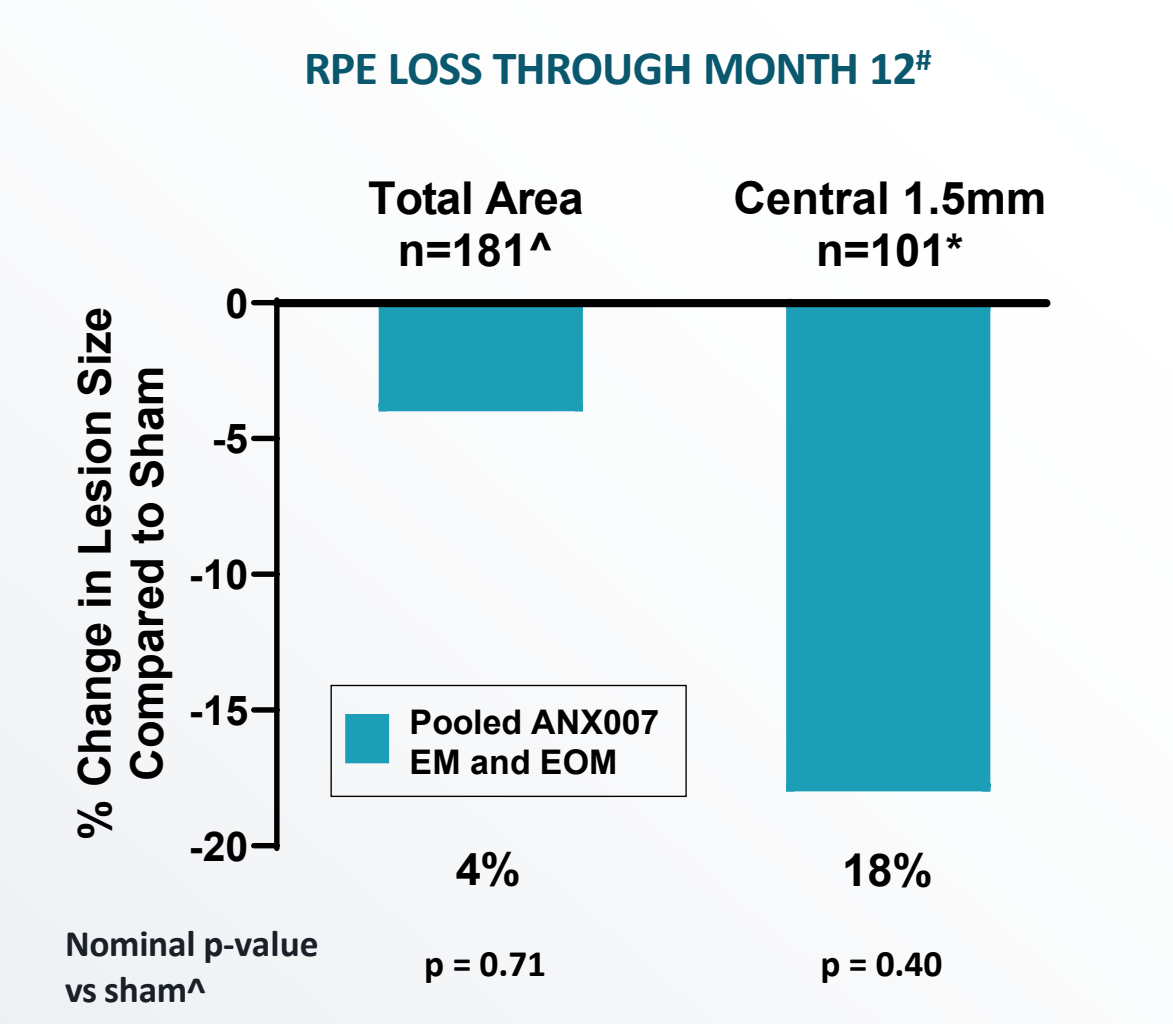
Location	Month 6	Month 12	Month 18
Full 6 mm diameter	p=0.59	p=0.15	**p=0.03**
1mm foveal center	**p=0.03**	**p=0.001**	**p<0.0001**

- Correlation in central 1mm as early as 6 months
- Overall lesion growth correlates after 18 months

1. Sayegh et al, Am J Ophthalmology 2017 179:118-128

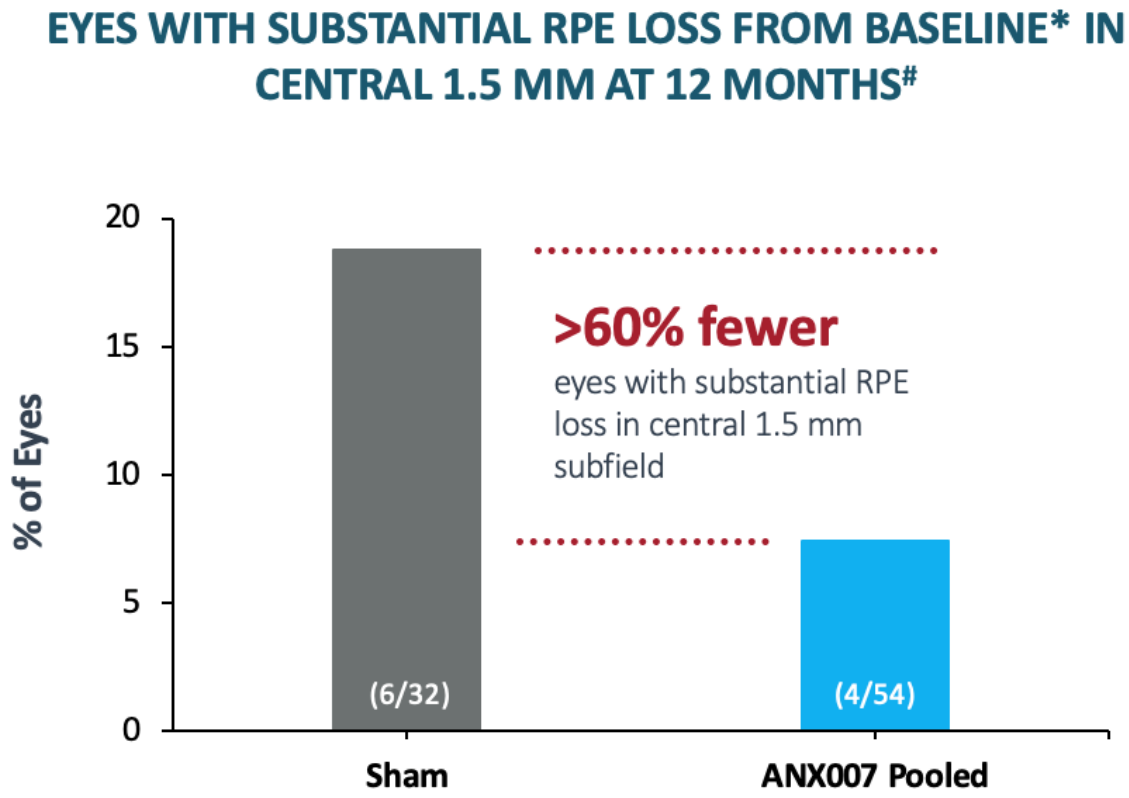
ANX007 Protection from RPE Loss More Robust in 1.5 mm Foveal Center

Consistent with treatment that protects from vision loss



#From a mixed model for repeated measures (MMRM) analysis; ^ITT population
*Heidelberg Spectralis OCT population with baseline OCT data, excludes patients with >98% atrophy at baseline

In Patients with Foveal Center RPE Remaining, ANX007 Reduced Patients Experiencing Substantial RPE Loss by 60%



#Eyes with at least 25% of RPE intact in the central 1.5mm at baseline (n = 86) in patients with Heidelberg Spectralis OCT scans (overall total n=193)
*Substantial RPE loss defined as 25% absolute loss of RPE

ANX007 Generally Well-Tolerated

ADVERSE EVENTS OF SPECIAL INTEREST n (%)	SHAM (N=89)	ANX007 EM (N=89)	ANX007 EOM (N=92)
Choroidal Neovascularization	3 (3.4%)	4 (4.5%)	4 (4.3%)
Endophthalmitis	0	1 (1.1%)	2 (2.2%)
Retinal Vascular Occlusion	0	0	1^ (1.1%)
Retinal Vasculitis – No Cases Reported			
Intraocular Inflammation+	0	2 (2.2%)	1 (1.1%)
Ischemic Optic Neuropathy+ - No Cases Reported			

^Isolated cilioretinal artery occlusion; no vasculitis confirmed by DSMC and reading center
+Not AESI, included because of current interest

INTRAOCULAR INFLAMMATION DETAILS* n

Iritis – 1
Resolved with topical steroids in 2 days
No Vasculitis

Vitritis – 1
Resolved with topical steroids in 9 days
No Vasculitis

Vitreous Debris – 1
KP on endothelium, prior treatment with topical steroids
No Vasculitis

*Event Verbatim term listed

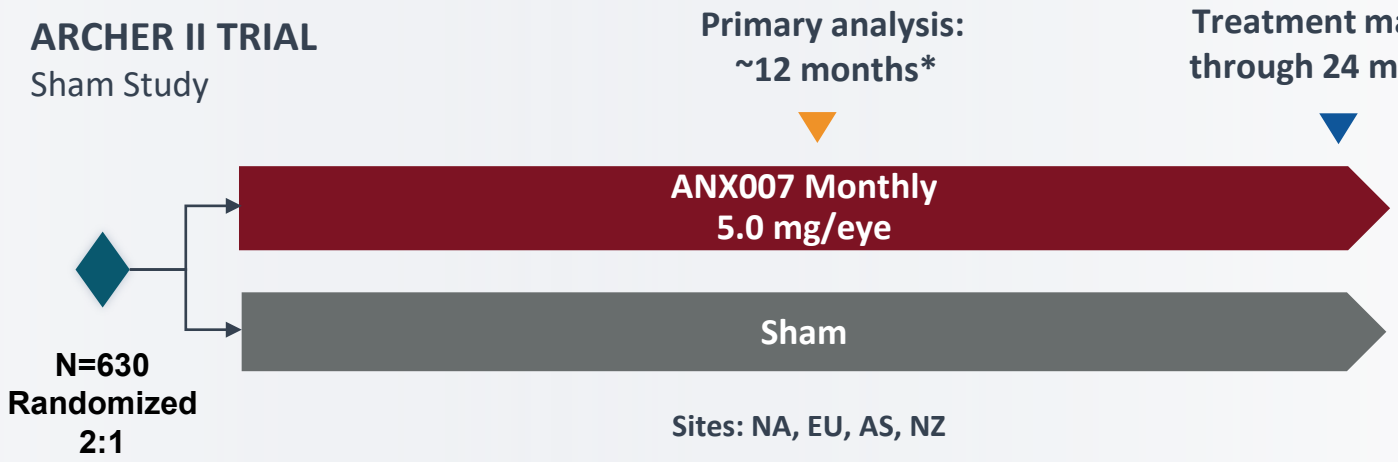
ANNEXON
biosciences

Phase 3 Program Overview

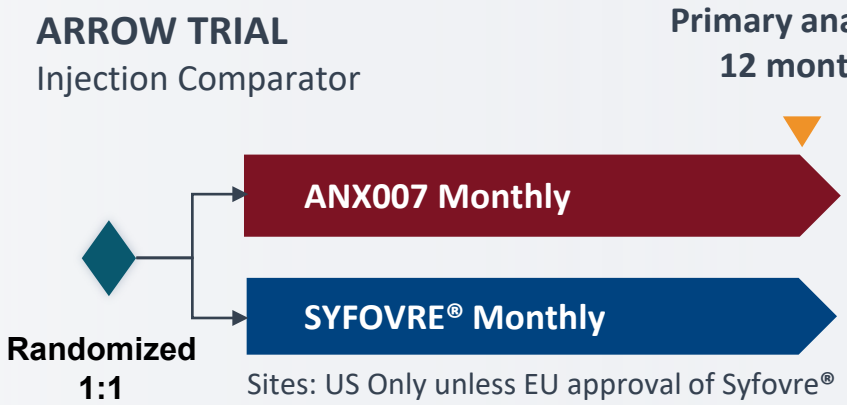
ANX007 Global GA Pivotal Program INITIATED

ARCHER II enrollment ongoing; ARROW trial initiation in late-2024

ARCHER II TRIAL
Sham Study



ARROW TRIAL
Injection Comparator



PRIME Designation from EMA

PRIMARY ENDPOINT

Persistent BCVA ≥15-Letter Loss through ~12 months*

*Primary analysis based on accumulation of BCVA ≥15-letter loss target events assessed between months 12-18 from initiation of dosing

SECONDARY ENDPOINTS

Safety, Low Luminance VA (LLVA), Anatomic assessments

ANX007: A Novel Neuroprotective Agent Demonstrating Vision Protection Supported by Structure Protection Now in Phase 3

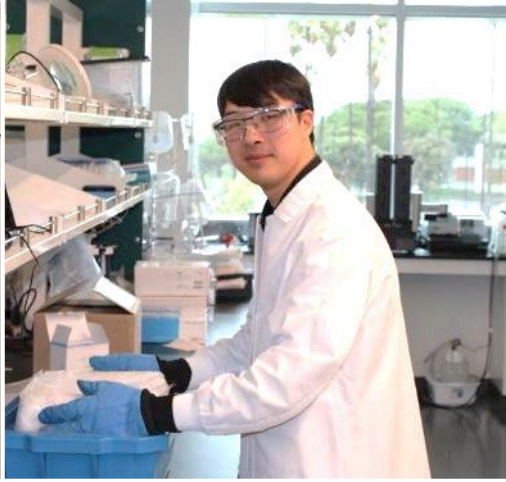
Blocking C1q for neuroprotection, prevented synapse loss and protected photoreceptors from elimination

ANX007, an anti-C1q Fab antibody administered IVT, **consistently protected against the loss of visual acuity** in the Phase 2 ARCHER study

Visual function benefit **supported by protection of retinal structures,** particularly those structures closely associated with visual function – **photoreceptors and foveal RPE**

ANX007 treatment was **generally well-tolerated;** no CNV increase;
no reported cases of vasculitis

Regulatory-aligned Phase 3 program NOW ONGOING



To the patients, families, caregivers, physicians and medical teams who participated in our trial, we are eternally grateful for your support and contributions!

To our employees, collaborators and advisors, thank you for your Warrior Spirit and All For One commitment!