ANNEXON biosciences

STOPPING NEUROINFLAMMATION AT ITS SOURCE

CORPORATE PRESENTATION | MAY 2026

Nasdaq: ANNX

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Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended, which are subject to the "safe harbor" created by those sections. All statements other than statements of historical facts contained in this presentation are forward-looking statements. These forward looking statements include, but are not limited to statements regarding the potential therapeutic benefits of our product candidates; our clinical and preclinical programs, timing and commencement of future nonclinical studies and clinical trials and research and development programs, timing of clinical results, anticipated timing and results of regulatory interactions related to our product candidates, including the timing of our planned biologics license application (BLA) submission to the U.S. Food and Drug Administration (FDA); our ability to achieve regulatory approval for our product candidates; the potential for vonaprument to be the first drug approved for dry AMD with GA; the potential for vonaprument and tanruprubart to reset the standard of care; strategic plans for our business and product candidates, including additional indications which we may pursue, our ability to commercialize our product candidates, if approved; the potential for us to deliver significant value for patients and our stakeholders; our financial position, runway and anticipated milestones. 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 the negative of these terms or other similar expressions that are predictions of or indicate future events and future trends.

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: our history of net operating losses; our ability to obtain necessary capital to fund our clinical programs; the potential for delays in our clinical trials; the potential for our product candidates to not receive regulatory approval, including if the FDA and comparable foreign regulatory authorities determine that our submission package is not sufficient or require us to provide additional data in patients that are not feasible to obtain; the early stages of certain of clinical development of our product candidates; the effects of 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" and in the other cautionary statements contained in our Annual Report on Form 10-K for year ended December 31, 2025, our subsequent Quarterly Reports on Form 10-Q and our other filings with the Securities Exchange Commission. Any forward-looking statements that we make in this presentation are made pursuant to the Private Securities Litigation Reform Act of 1995, as amended, and represent our management's beliefs and assumptions 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.

This presentation concerns drug candidates that are under clinical investigation, and which have not yet been approved for marketing by the FDA. These are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates or statistical data. Neither we nor any other person makes any representation as to the accuracy or completeness of such data or undertakes any obligation to update such data after the date of this presentation.

Two Registrational Programs with Blockbuster Potential in Large, Underserved Markets

Tanruprubart

Establishing first potential targeted rapid-acting treatment for GBS

- Comprehensive EU MAA filed
- Initial FORWARD study data anticipated 2026
- US BLA planned 2026

Vonaprument

Establishing first potential vision-preserving treatment for GA

- 1st significant vision-sparing data in P2 evaluating vision
- 1st P3 trial evaluating vision - data anticipated Q4 2026

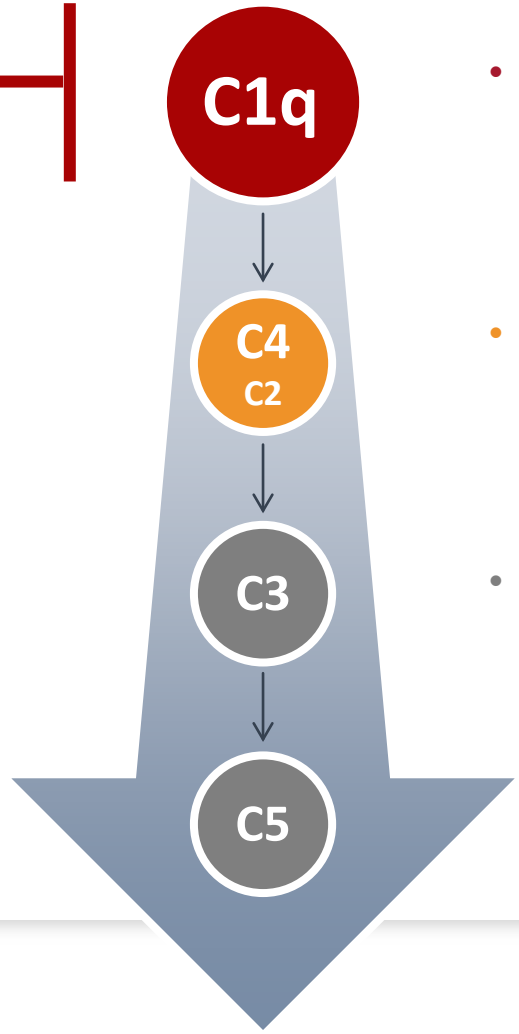
Pipeline expansion opportunities including 1st oral inhibitor of classical complement in the clinic (ANX1502), and 1st demonstration of inhibition of C1q inflammation in CNS for an array of diseases (e.g., HD, TBI)

C1q Inhibition Platform Provides Competitive Advantage

Annexon's C1q Approach

Stops neuroinflammation at its source
before amplification and damage
vs. downstream C3 & C5 inhibitors

Classical Complement Cascade



- Binds neurons and activates pathway
- Amplifies inflammation and tissue damage
- Inflammation and damage and removal of cells

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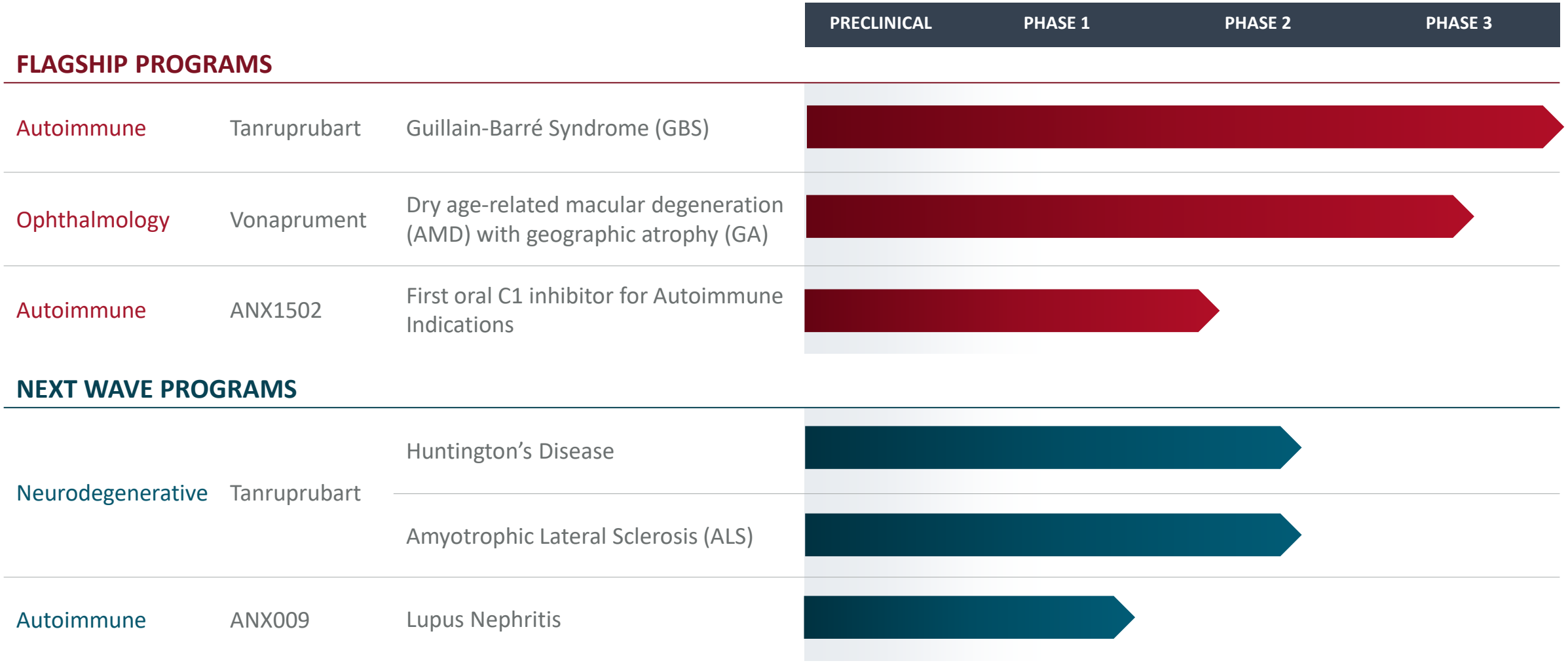
Late-Stage Pipeline with Multiple Blockbuster Opportunities

Diverse and informed drug candidates addressing neuroinflammatory diseases for ~10 million patients



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Annexon Poised to Capture Asymmetric Value Opportunity

Positioned to unlock significant value with multiple late-stage assets

First Generation
C5 Inhibition

ALEXION

Pioneered C5 inhibition
Acquired by AstraZeneca

$39B[1]

IVERIC BIO

Pioneered C5 inhibition in GA
Acquired by Astellas

$5.9B[2]

Second Generation
C3 Inhibition

Apellis

Pioneered C3 inhibition in GA
Acquired by Biogen

~$5.6B[3]

Next Generation
C1q Inhibition

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Pioneering C1q Inhibition
in several diseases

~$1.1B[4]

[1]https://www.astrazeneca.com/media-centre/press-releases/2020/astrazeneca-to-acquire-alexion.html#
[2]https://investors.ivericbio.com/news-releases/news-release-details/astellas-enters-definitive-agreement-acquire-iveric-bio
[3]https://investors.biogen.com/news-releases/news-release-details/biogen-completes-acquisition-apellis-pharmaceuticals
[4]Based on 200M fully diluted shares outstanding, including 163.8M common shares and 36M prefunded warrants at $5.80 per share, the last 30-day average closing price of the company's stock on 5/4/2026.
Comparisons to complement companies are purely for illustrative purposes only. Actual results for the Company will vary and nothing in this presentation should be regarded as a representation by any person that similar results will be achieved.

Established Leadership with Drug Development through Commercial Depth

Doug Love
President & CEO

élan AMGEN

Genentech
A Member of the Roche Group

Ted Yednock, PhD
Chief Innovation Officer

élan TYSABRI (natalizumab)

Jamie Dananberg, MD
Chief Medical Officer

UNITY BIOTECHNOLOGY

Lilly Takeda

Rick Artis, PhD
Chief Scientific Officer

élan

Plexxikon

Jennifer Lew
Chief Financial Officer

ADURO BIOTECH EY

DYNAVAX

Michael Overdorf, MBA
Chief Business Officer

Lilly

Shikhar Agarwal
Head of Commercial

argenx AMGEN

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Anticipated Catalysts Supported by Cash Runway into 2H 2027

Tanruprubart in GBS

- ✓ **EU MAA filed in Jan 2026** for first targeted therapy for GBS

FORWARD U.S. & EU initial data anticipated 2026

FDA BLA filing anticipated 2026

Vonaprument in GA

Topline Phase 3 data anticipated Q4 2026 for first vision-sparing program in dry AMD with GA

ANX1502 in Autoimmune

POC study completion anticipated 2026 for first oral C1 inhibitor

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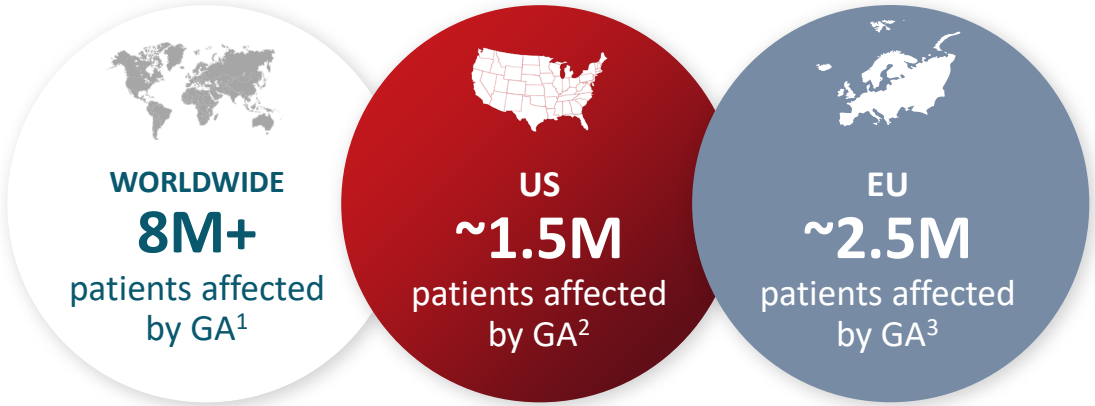
Vonaprument: First Potential Treatment to Preserve Vision for Dry AMD with Geographic Atrophy

Global Pivotal Program with Potential Blockbuster Market Opportunity

Geographic Atrophy Remains a Significant Unmet Need

No approved treatments demonstrating vision preservation



GA SEVERELY LIMITS INDEPENDENCE AND IS A LEADING CAUSE OF BLINDNESS IN THE ELDERLY

- Avg. Age of GA Patient: 79 years
- GA greatly impacts quality of life, interfering with reading, driving, recognizing faces
- **Incidence projected to increase due to aging population**

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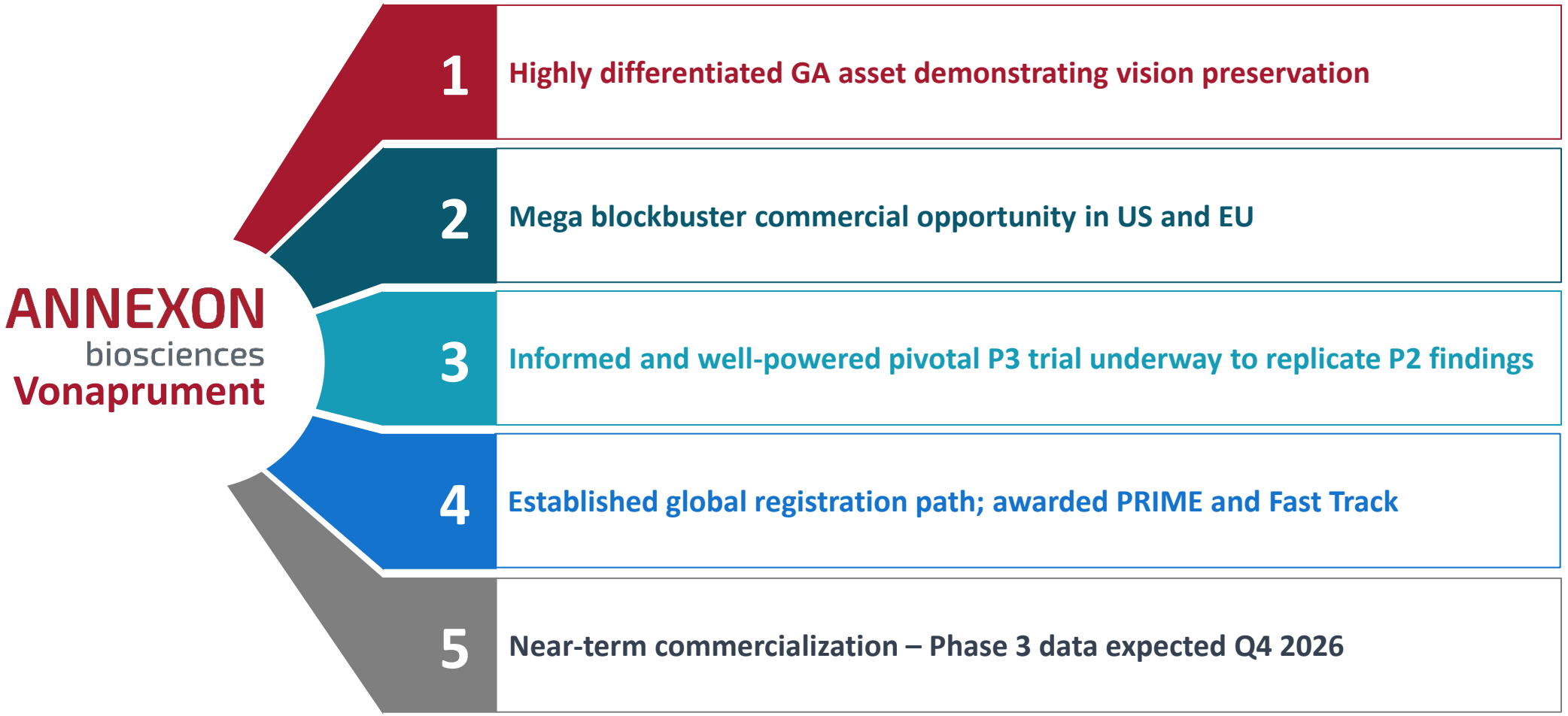
Vonaprument (ANX007) is a Disruptive Blockbuster Commercial Opportunity in GA



Vonaprument Poised to Capture and Drive Immense GA Market

Pursuing vision preservation to drive a fundamental shift in standard of care

Lesion-sparing medicines

~$1.5B

Combined current sales[1]

SYFOVRE® (pegcetacoplan injection)

izervay™ (avacincaptad pegol intravitreal solution)

1st generation IVT drugs have established patient demand, but lagged expectations due to benefit-risk profile

Vision-preservation medicines

>$7B

Global peak sales[2]

Vonaprument

Vision preservation offers enhanced benefit-risk to tap full market

Differentiated profile: Small, non-pegylated, low viscosity, limited conversion to CNV

[1]Analyst estimates

[2]ClearView Healthcare Partners analysis of 2037 worldwide sales

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GA is a Chronic Progressive Neurodegenerative Disease

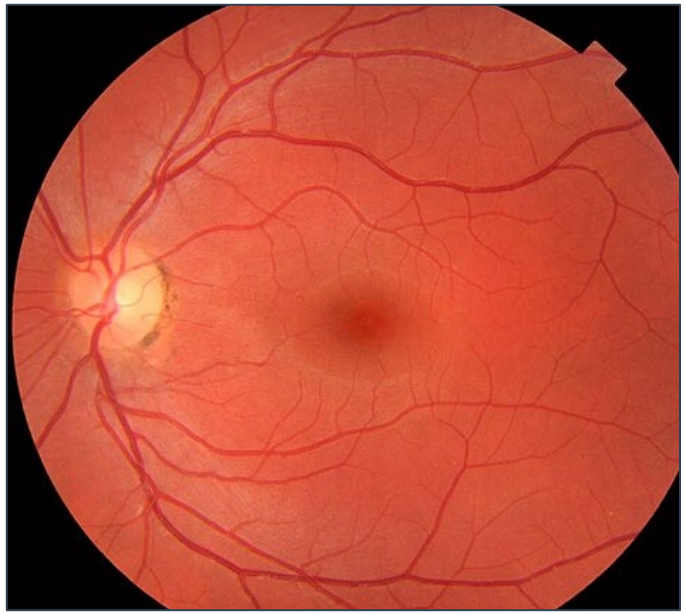
GA is Advanced Form of Dry AMD:

Loss of Photoreceptors –
neurons that sense light in the retina
leads to loss of vision

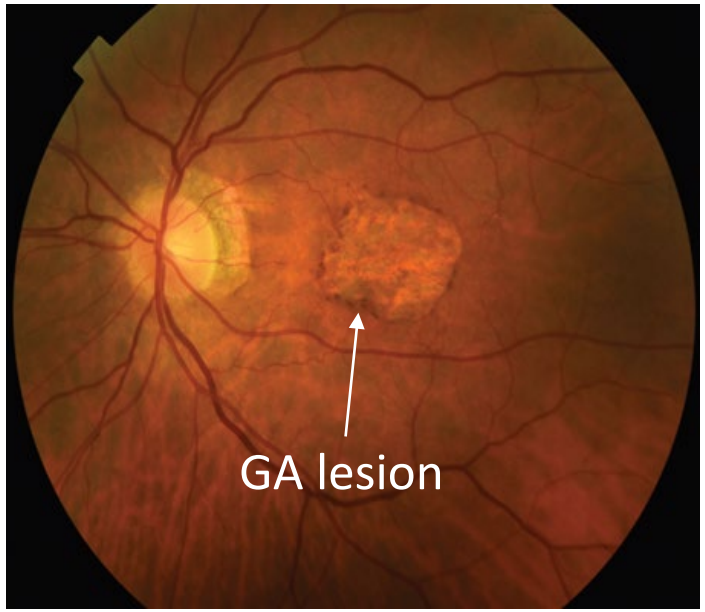
▼

Subsequent atrophy of Retinal Pigment Epithelium (RPE) –
cells that support the photoreceptors
shown in GA lesion growth

Normal retina



Retina with GA



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Geographic Atrophy Treatment Landscape Rapidly Evolving

C3/C5 therapies designed to stop RPE atrophy/lesions

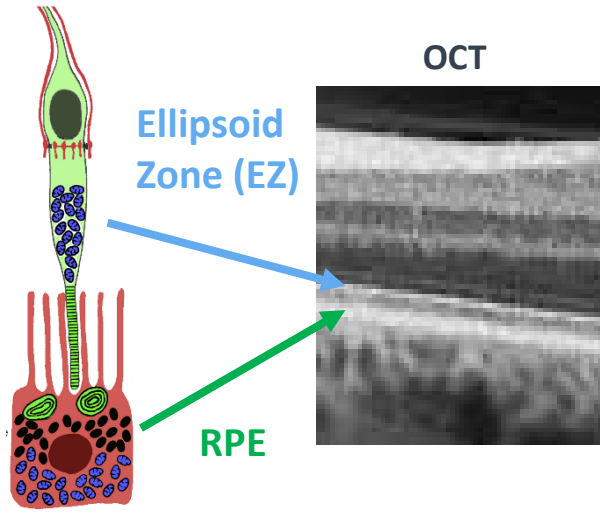
- Human genetics established complement inhibition as a therapeutic strategy for GA
- C3/C5 designed to target the alternative pathway to preserve RPE cells/reduce lesion growth
- Protection of RPE has not resulted in preservation of vision

SYFOVRE (pegcetacoplan injection)

izervay (avacincaptad pegol intravitreal solution)

Recent advances: PR neurons are increasingly understood as locus of disease based on OCT/EZ

- **GA is a neurodegenerative disease** resulting in photoreceptor and vision loss
- Loss of PRs via the classical pathway/C1q precedes RPE loss



Today's Opportunity

- Vision-preserving therapy will meaningfully improve patient lives

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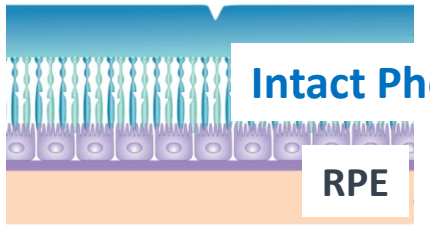
GA is a Neurodegenerative Disease that Starts at Photoreceptor Synapses and Cells Necessary for Vision

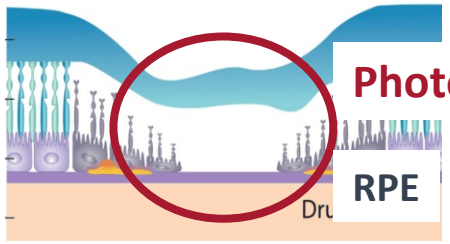
NORMAL RETINA

Photoreceptors convert to electrical signals



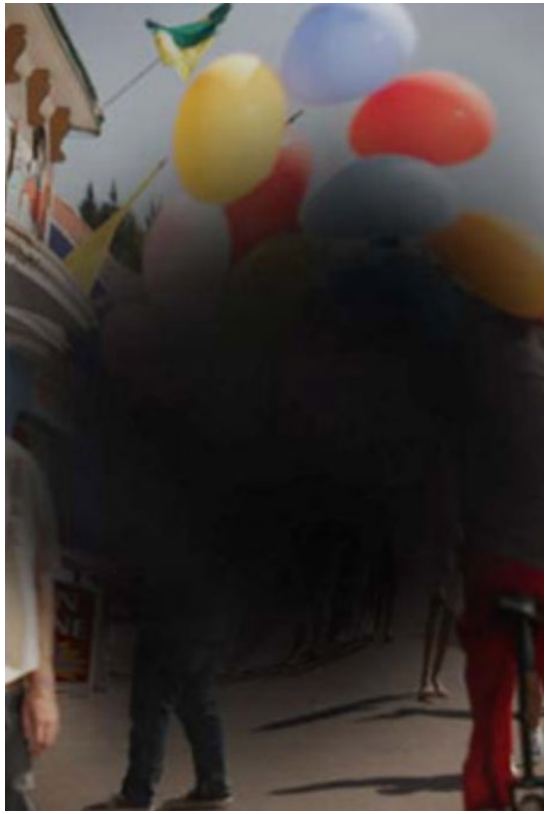
RETINA WITH GA

Loss of signals due to damaged or missing photoreceptors



Normal Vision

Reduced Vision



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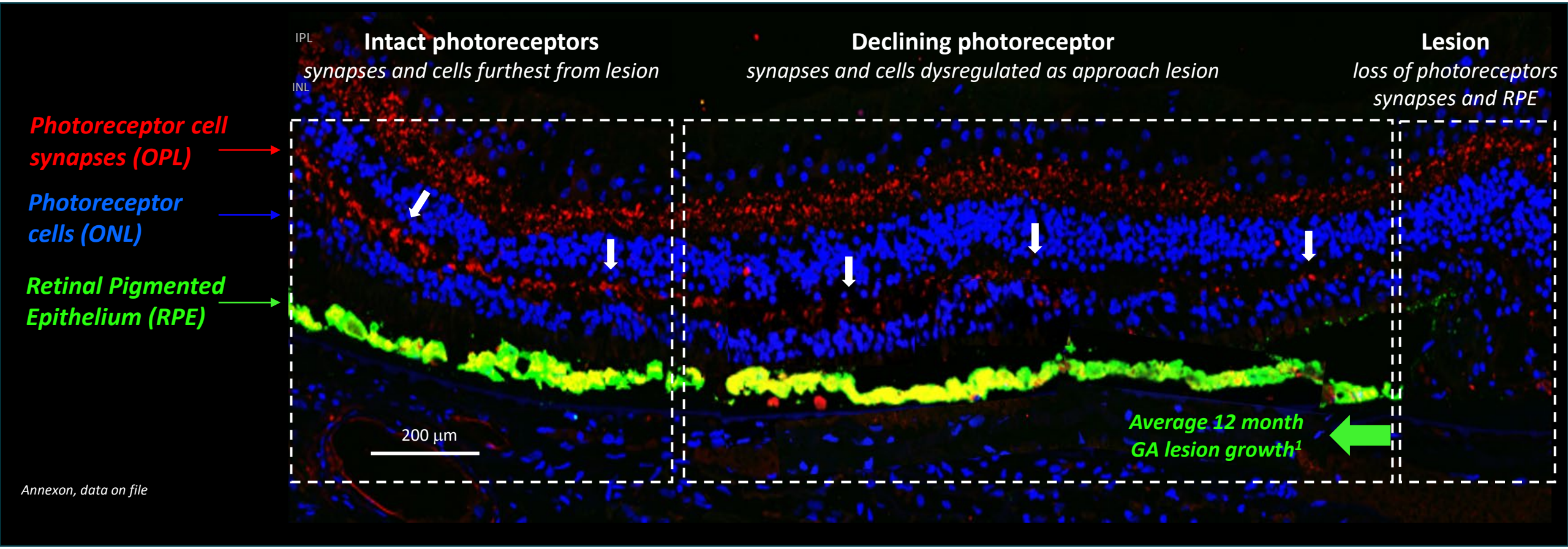
In GA Photoreceptors (PR) and Synapses are Lost Before RPE Loss

PR loss occurs prior to RPE loss; PR loss = vision loss



Photoreceptor Health	Functional retina	Reduced function	Lost function

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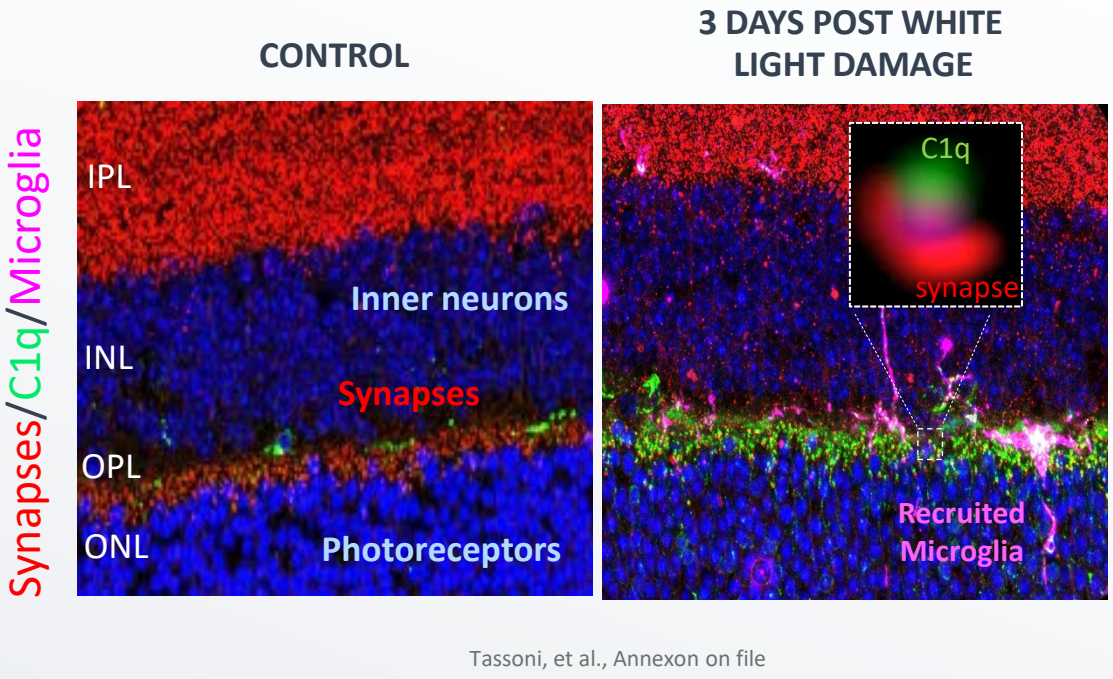
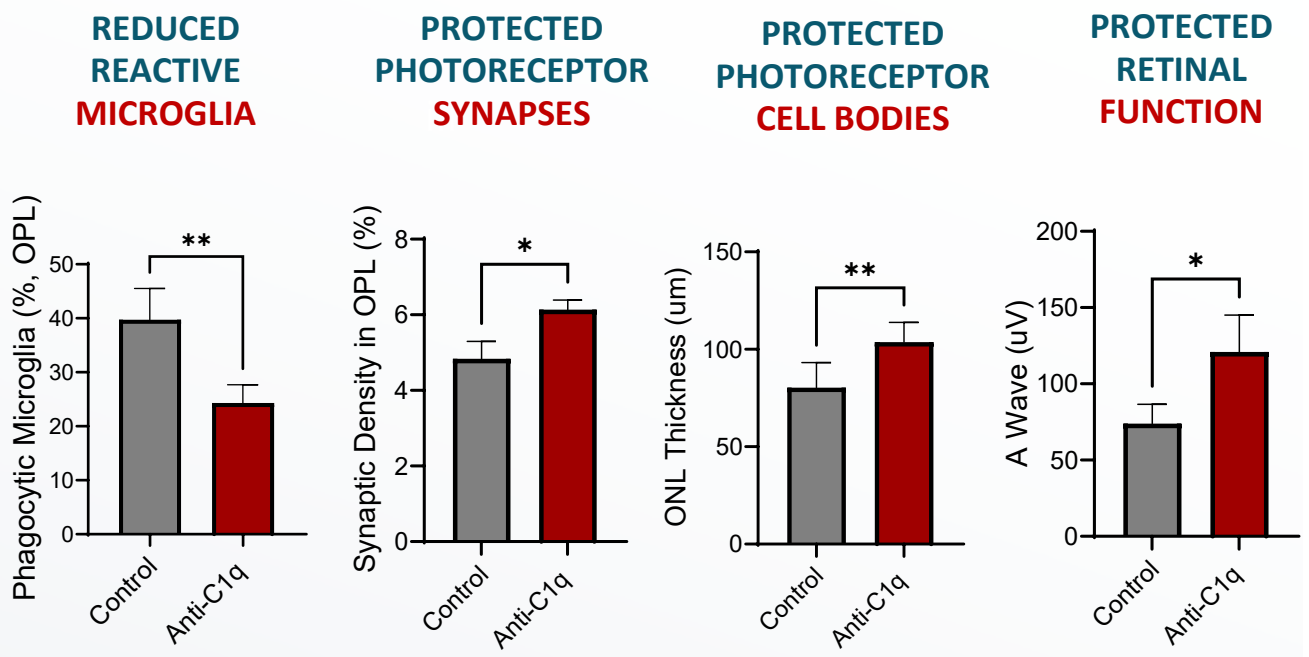
Anti-C1q Protected Photoreceptor Cells and Their Function in Models of Photoreceptor Damage

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



Tassoni, et al., Annexon on file

Anti-C1q Protected Photoreceptors and Function



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ARCHER: Phase 2 Trial of C1q Inhibitor Vonaprument 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)

Vonaprument 5mg every other month (EOM)
(n=92)

Vonaprument 5mg monthly (EM)
(n=89)

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

See full ARCHER phase 2 trial results in Vonaprument presentation at ir.annexonbio.com

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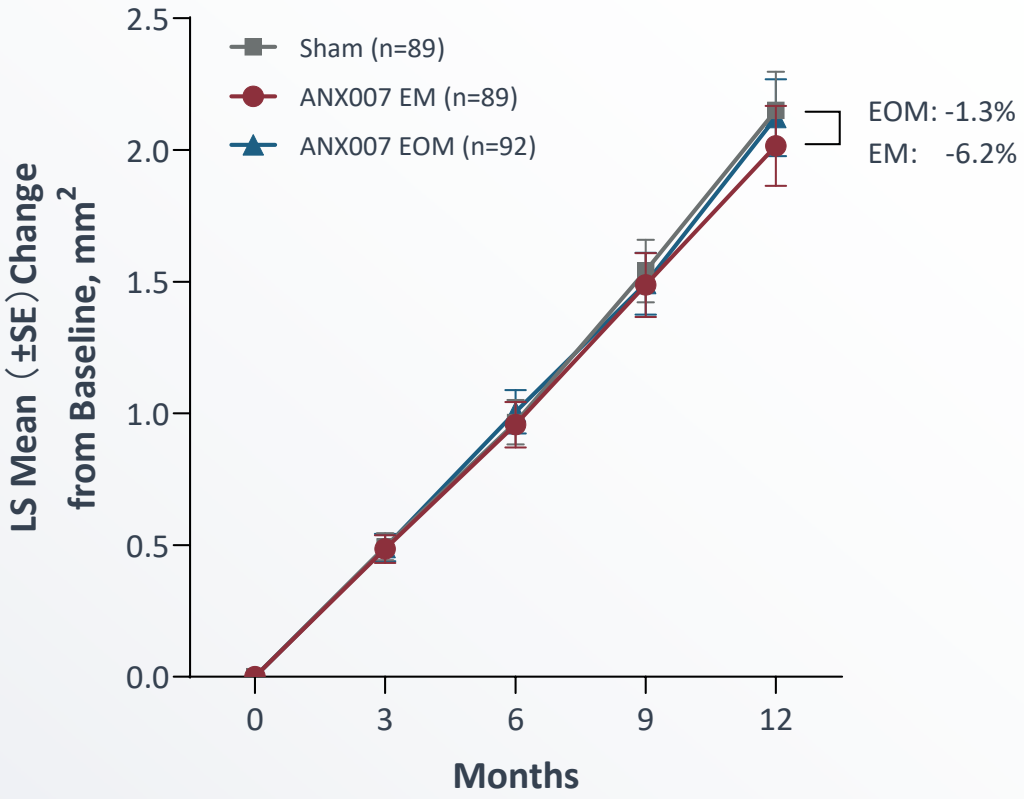
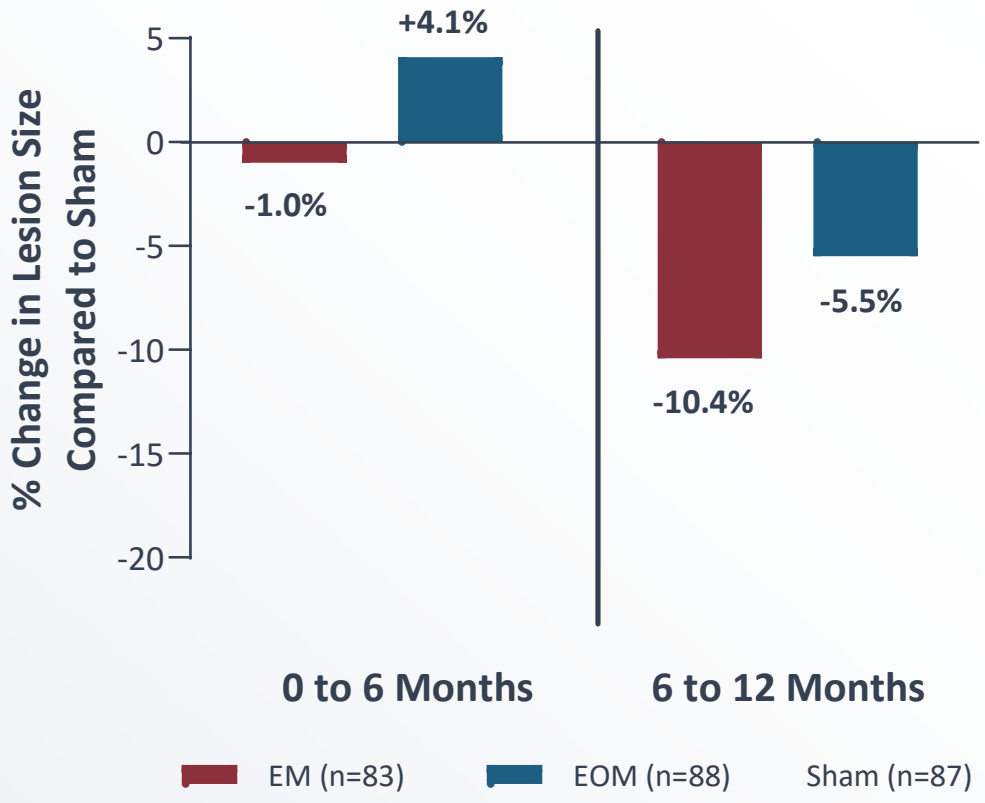
Phase 2: Vonaprument Treatment Effect on RPE Loss through 12 Months

No significant reduction at month 12, but effects increased over 2nd half of study

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.

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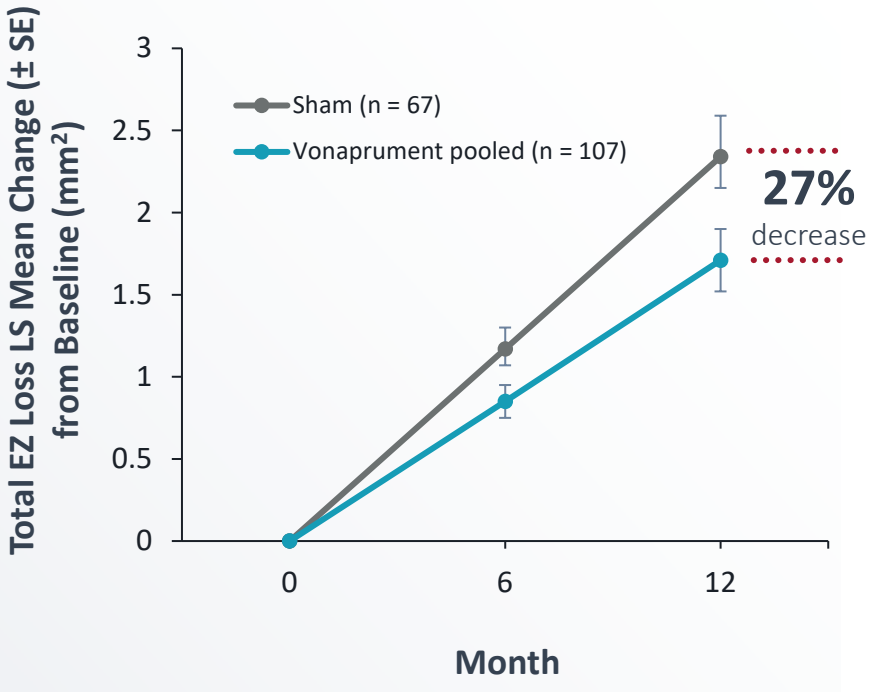
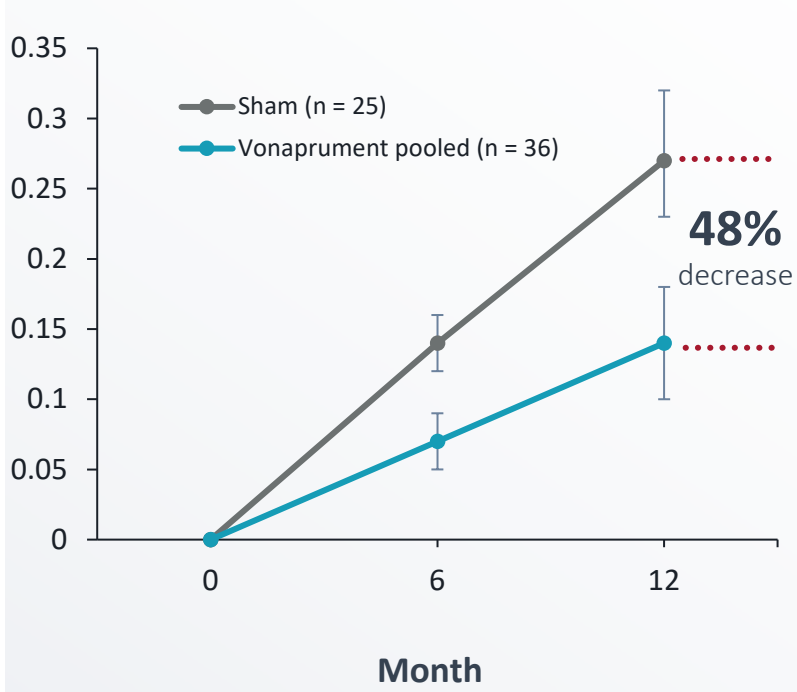
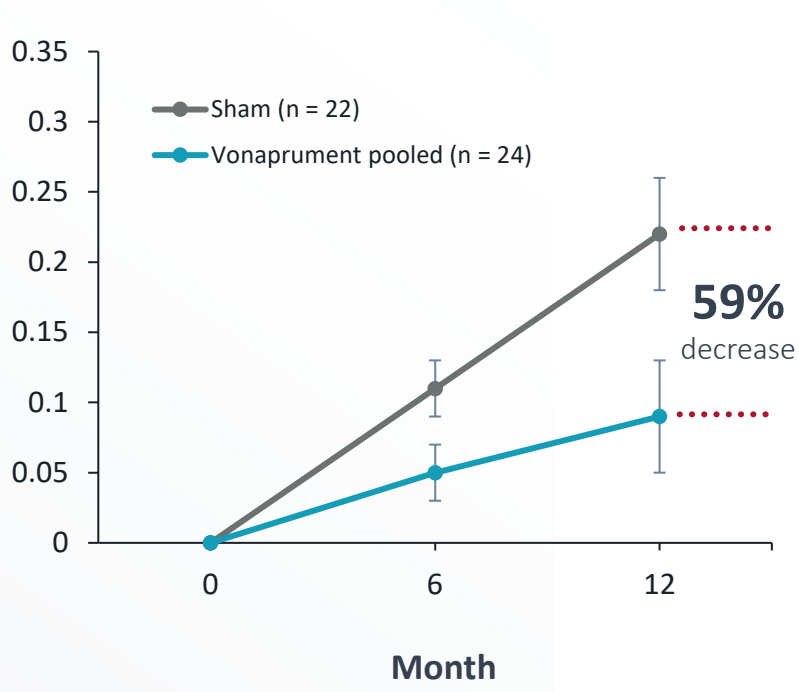
Phase 2: Greater Protection Against EZ Loss in Central Retina Necessary for Visual Acuity







Nominal p-value^		
	Vonaprument Pooled vs Sham	0.0457
	Vonaprument Pooled vs Sham	0.0218
	Vonaprument 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 and at least one post-baseline OCT image , excludes patients with >98% atrophy/attenuation at baseline

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A Vision-Sparing Therapy in GA: Greatest Unmet Need in Retina

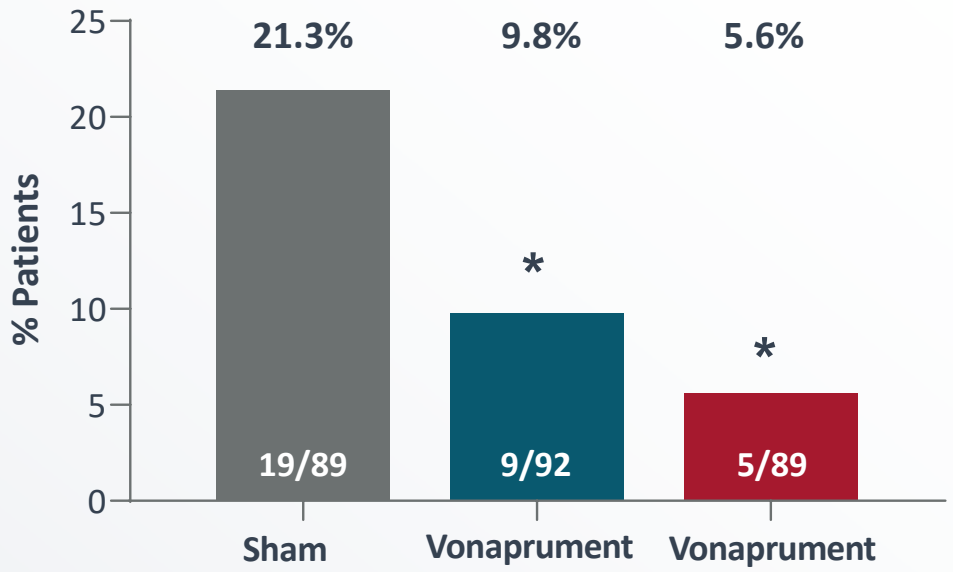
PRODUCT	FUNCTIONAL PRIMARY ENDPOINT
Wet AMD	
Lucentis	Trial 1 & 2: BCVA ≥15 letter Trial 3 & 4: mean BCVA change
Eylea	BCVA ≥15 letter
Eylea HD	Mean BCVA change
Vabysmo	Mean BCVA change
DME	
Lucentis	BCVA ≥15 letter
Eylea	Mean BCVA change
Eylea HD	Mean BCVA change
Vabysmo	Mean BCVA change
Iluvien	BCVA ≥15 letter
Ozurdex	BCVA ≥15 letter
GA	
Syfovre	N/A
Izervay	N/A

No Approved Vision-Preserving Treatments in GA

Phase 2: Vonaprument Demonstrated Dose-Dependent Protection From Vision Loss as Measured by BCVA ≥15-Letter Loss

Consistent treatment effect when month 12 loss is confirmed at month 15

PATIENTS WITH CONFIRMED BCVA ≥15-LETTER LOSS THROUGH MONTH 12 OR LAST VISIT#

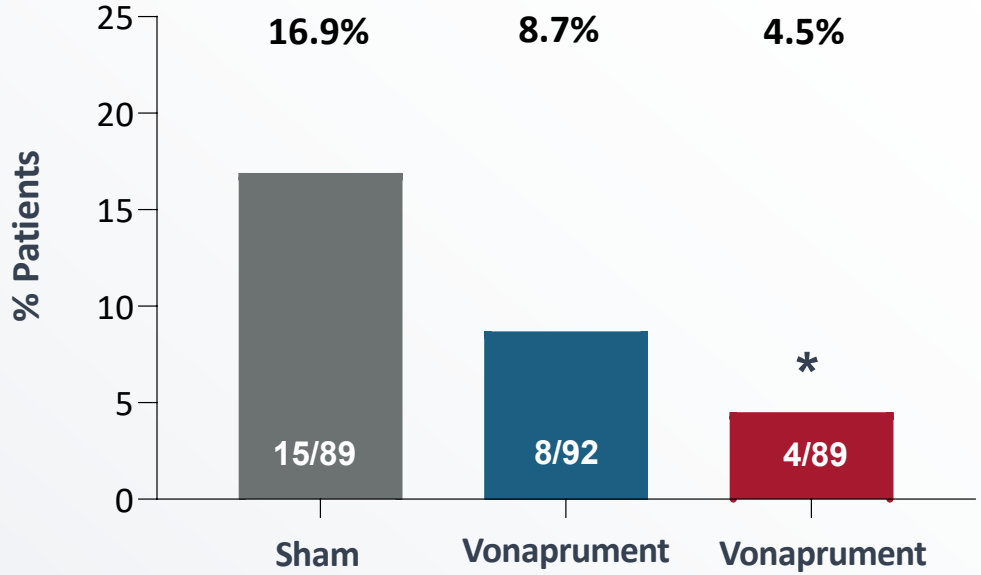


Nominal p-value vs Pooled Sham^	---	0.032	0.0021

#Confirmed 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

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

Month 12 event confirmed at month 15



Nominal p-value vs Pooled Sham^	---	0.0994	0.0076

##Confirmed at two consecutive visits through month 12; month 12 event confirmed at month 15
^Nominal p-value from a Chi-square test in ITT population: *Nominal p < 0.05

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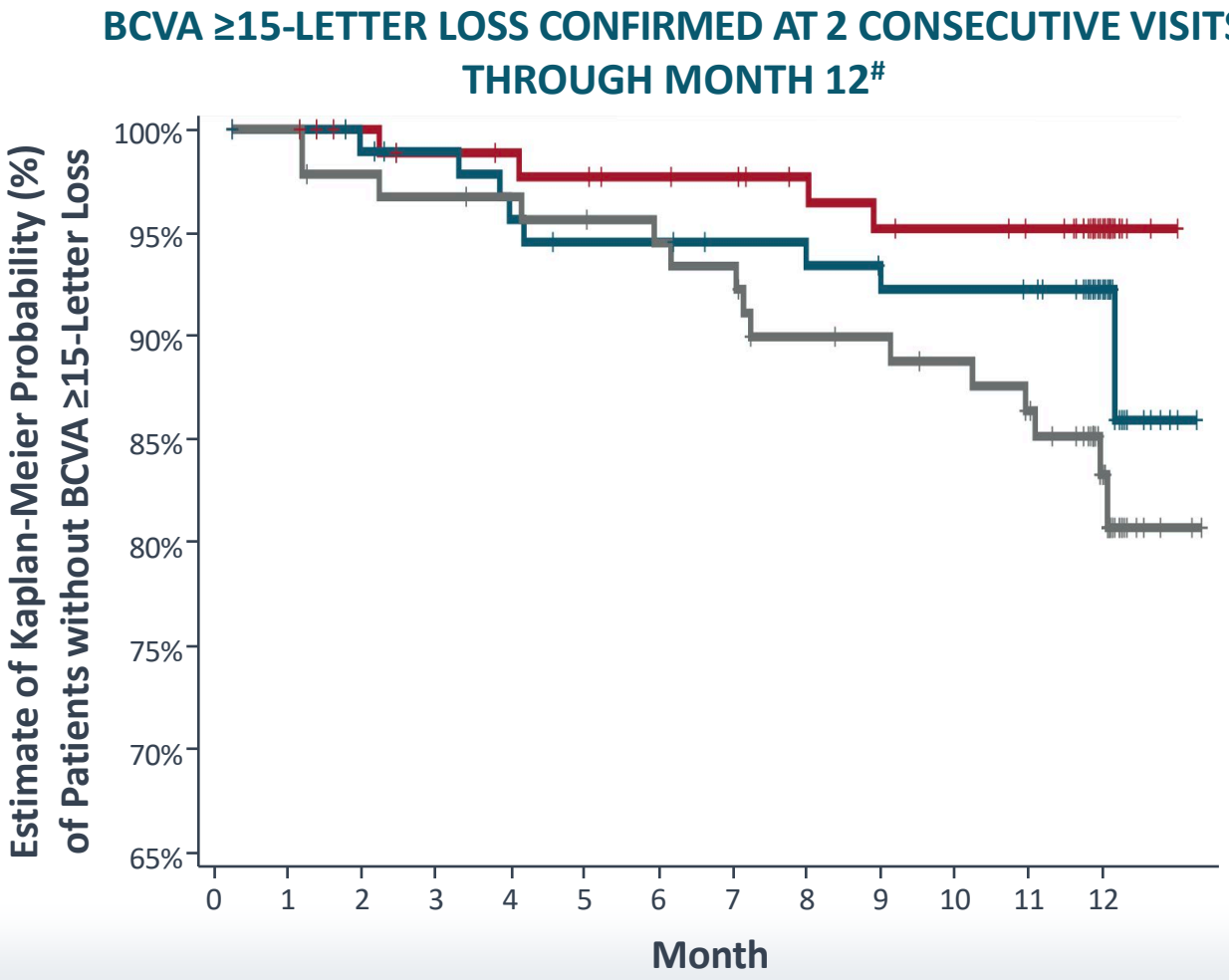
Phase 2: Vonaprument Monthly Treatment Provided 73% Reduced Risk of ≥15-Letter Vision Loss at Month 12



EM (n=89)

EOM (n=92)

Sham (n=89)

73% Risk Reduction Vonaprument EM

HR (CI) = 0.272 (0.090 to 0.819); Nominal p = 0.0119

50% Risk Reduction Vonaprument EOM

HR (CI) = 0.504 (0.214 to 1.189); Nominal p = 0.1098

INCREASING VONAPRUMENT IMPACT OVER TIME

HR, hazard ratio; Nominal log-rank test (versus sham) p-values are presented;

Confirmed BCVA 15-LL at two consecutive visits including month 12 supported by ensuing (off-treatment) visit

Note: vertical tick marks represent patients that have exited the study without 15-letter loss event

Final data

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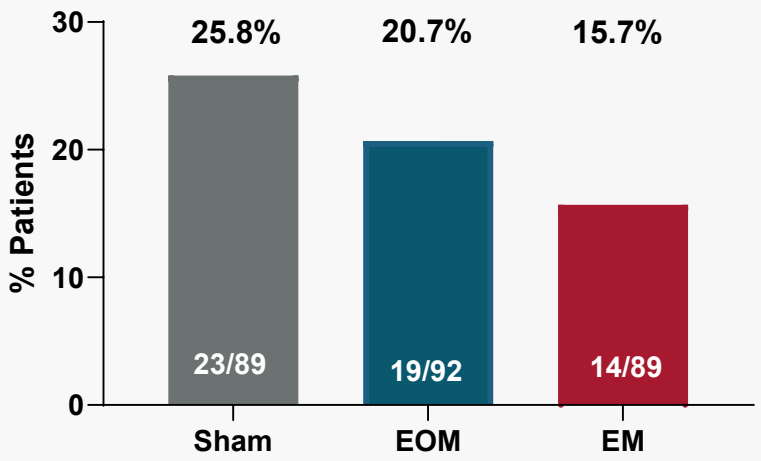
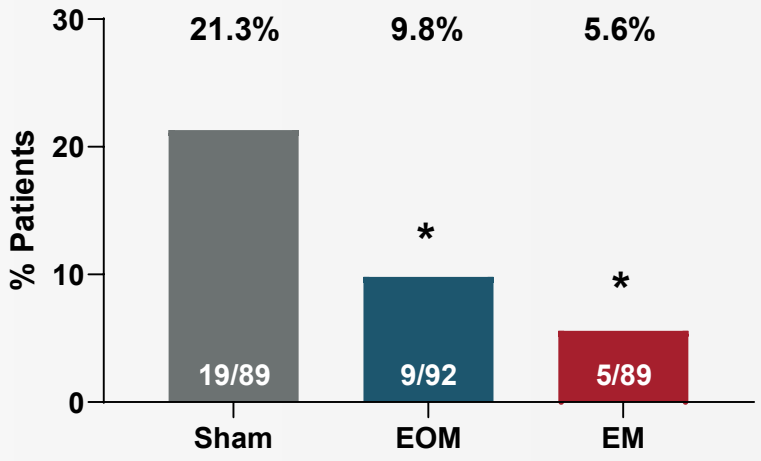
Phase 2 Sensitivity Analysis: Consistent Dose Dependent Protection from Vision Loss with BCVA ≥10, ≥15 and ≥20- Letter Assessments

CONFIRMED BCVA VISION LOSS THROUGH MONTH 12#

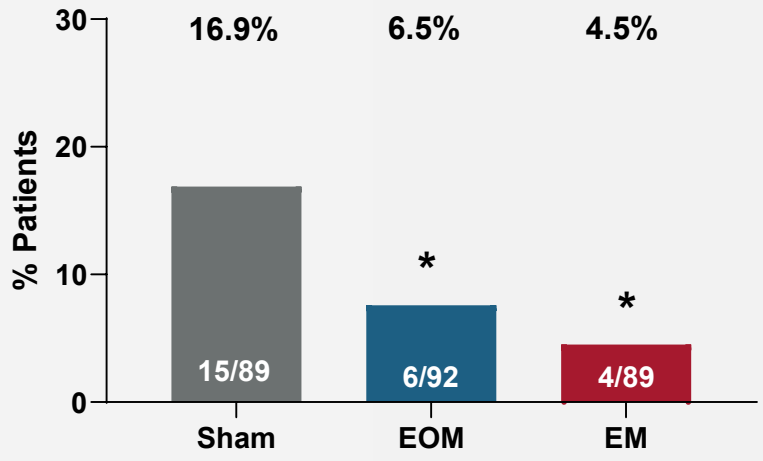
≥10-LETTER LOSS



≥15-LETTER LOSS



≥20-LETTER LOSS



	Sham	EOM	EM
Nominal p-value vs sham^	---	0.408	0.096
	---	0.032	0.002
	---	0.030	0.008

#Confirmed 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
Final data

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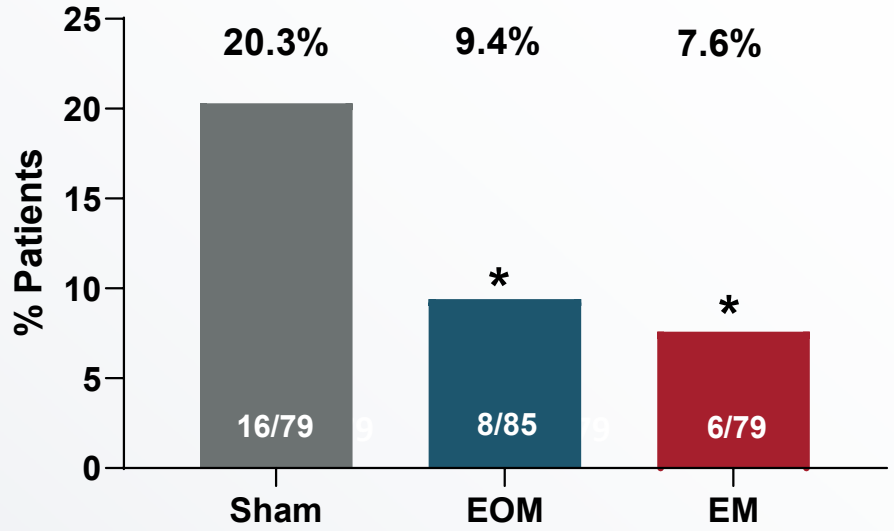
Phase 2: Vonaprument Consistent Dose Dependent Vision Protection Measured by LLVA and LLVD

LLVA ≥15-LETTER LOSS THROUGH MONTH 12#

Low Luminance Visual Acuity (LLVA): visual acuity assessed in low light conditions



Nominal p-value vs sham^	--	0.0497	0.0216

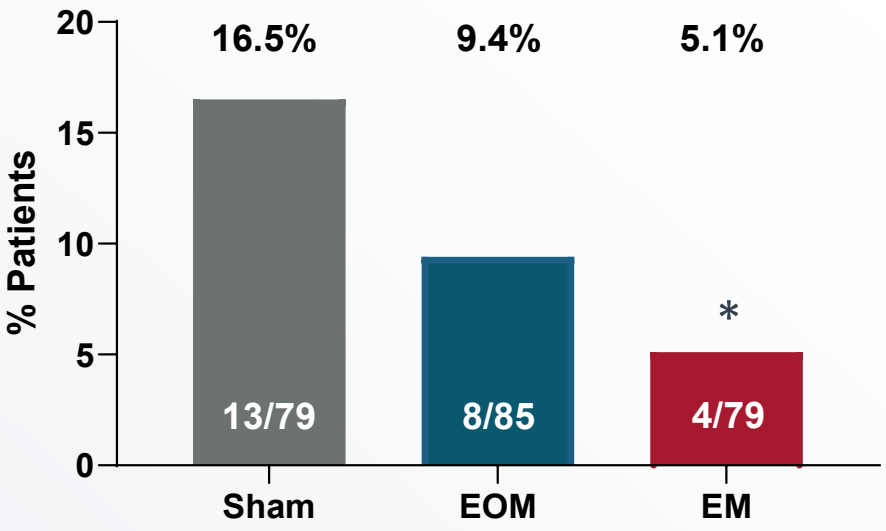
+Patients with at least one post-baseline LLVA measurement and two consecutive or last visit 15-letter loss events

^Nominal p-value from a Chi Square test; *p<0.05

Final data

LLVD ≥15-LETTER WORSENING THROUGH MONTH 12#

Low Luminance Visual Deficit (LLVD): difference between BCVA and LLVA to assess disease progression



Nominal p-value vs sham^	--	0.1774	0.0209

#≥15-letter worsening in LLVD was defined as a change from Baseline of ≥15-letters across 2 consecutive visits, or at the last visit, through month 12

^Nominal p-value from a chi-square test in the ITT set

*p<0.05

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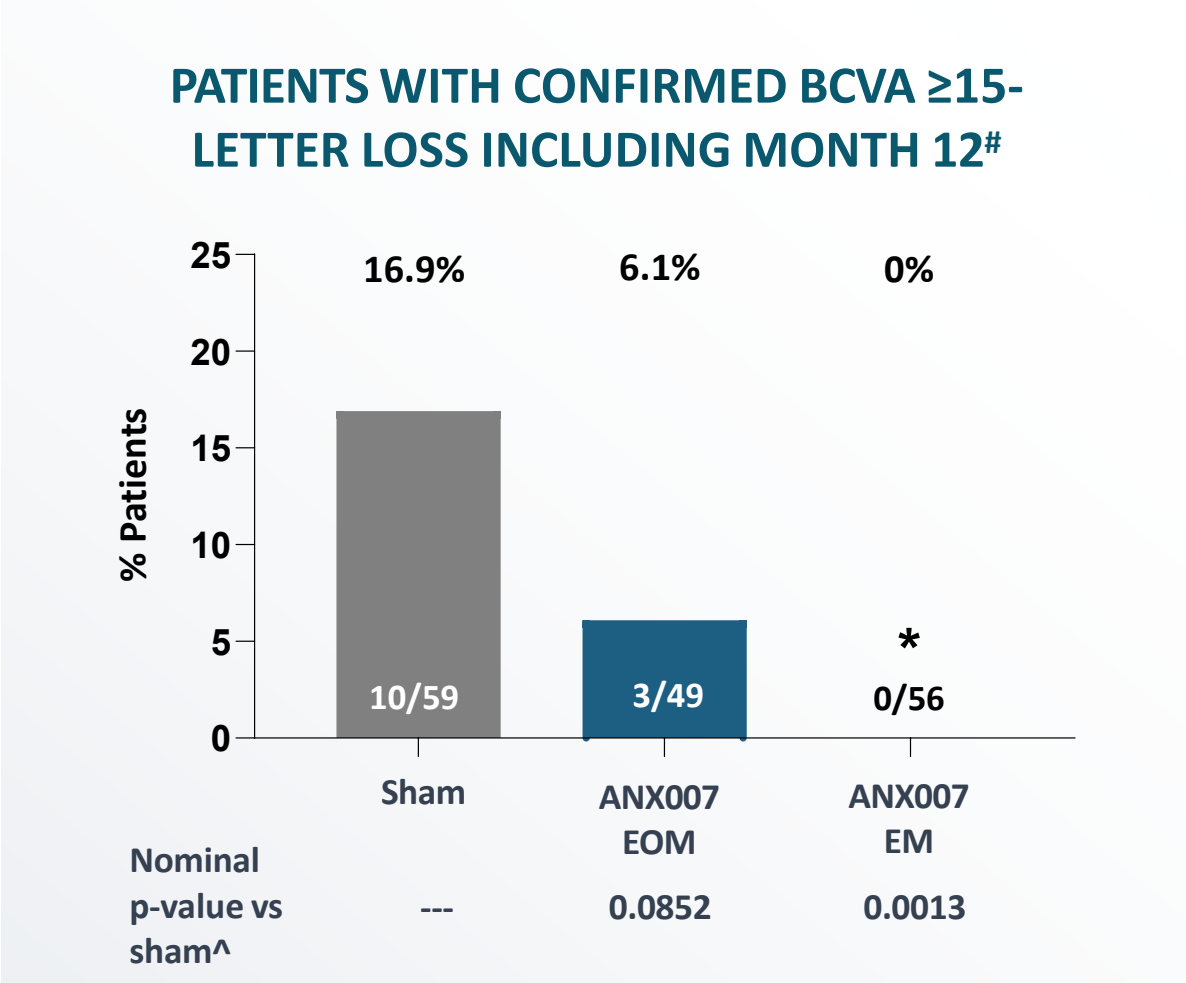
Phase 2: Profound Effect of Vonaprument on BCVA in Eyes with Less Advanced Disease (LLVD < 30[1])



No Monthly treated patients experienced confirmed BCVA ≥15-letter loss

#Confirmed for two consecutive visits including month 12

^Nominal p-value from a Cochran Mantel-Haenszel test (General Association) in ITT population; * Nominal p < 0.05

Final data

[1]LLVD <30 at baseline were designated as eyes with less advanced disease, based on mean baseline values in lampalizumab Phase 3 studies.Holz FG, et al. Efficacy and Safety of Lampalizumab for Geographic Atrophy Due to Age-Related Macular Degeneration: Chroma and Spectri Phase 3 Randomized Clinical Trials. JAMA Ophthalmol. 2018;136(6):666–677.

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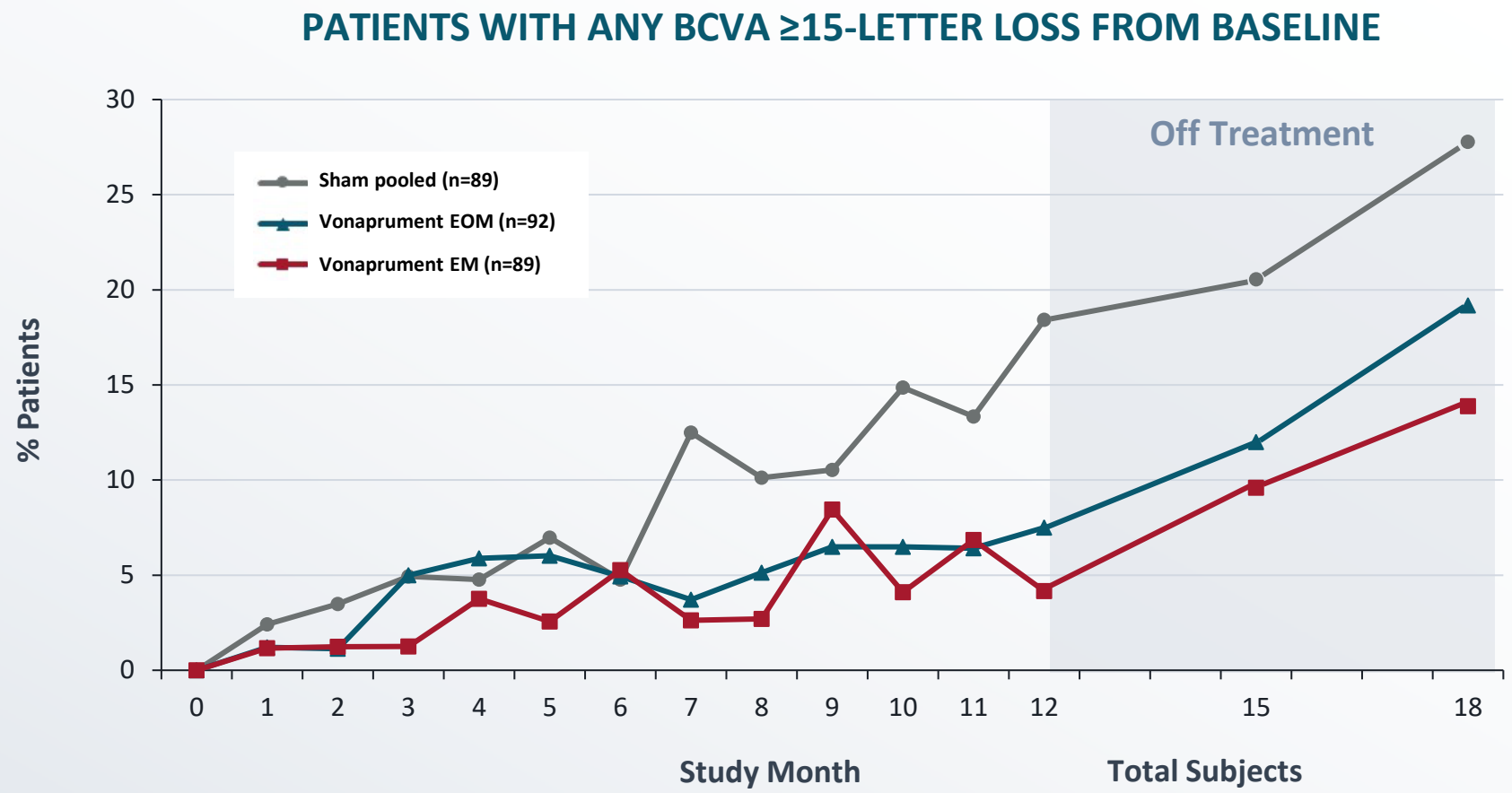
Phase 2: Vonaprument Demonstrated Clear Protection of Vision During On-Treatment vs Off-Treatment Period



Total Subjects

	Month 12	Month 15	Month 18
Sham	76	73	72
EOM	80	75	73
EM	72	73	72

Reinforces on-treatment drug effect and disease-modifying mechanism of action

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Safety Overview: Vonaprument was Well-Tolerated in Phase 2

ADVERSE EVENTS OF SPECIAL INTEREST n (%)	SHAM (N=89)	VONAPRUMENT EM (N=89)	VONAPRUMENT 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	0	0	0
Intraocular Inflammation+	0	2 (2.2%)	1 (1.1%)
Ischemic Optic Neuropathy+	0	0	0

CNV rate generally similar across sham and treatment arms

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

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

Vonaprument, formerly ANX007

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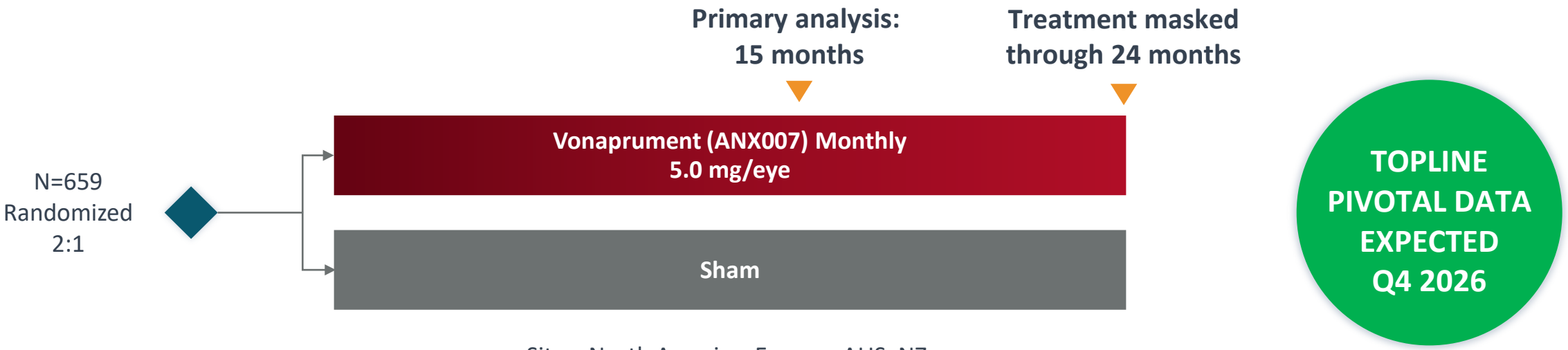
Phase 3 Pivotal Study: Well-Informed Design and Powering

Leverages Phase 2 learnings and enriched for patients with higher risk of vision loss



Sites: North America, Europe, AUS, NZ

GLOBAL REGISTRATION PATH[1]

Prime designation in EU
Selected by EMA for PDC[2] program
FDA Fast Track designation

PRIMARY ENDPOINT

Proportion of patients who experience a BCVA ≥15-Letter Loss confirmed at two consecutive visits

SECONDARY ENDPOINTS

Safety, LLVA, EZ integrity

[1] Single protocol analyzed as two sub-studies addresses FDA two-trial recommendation ; [2]Product Development Coordinator.

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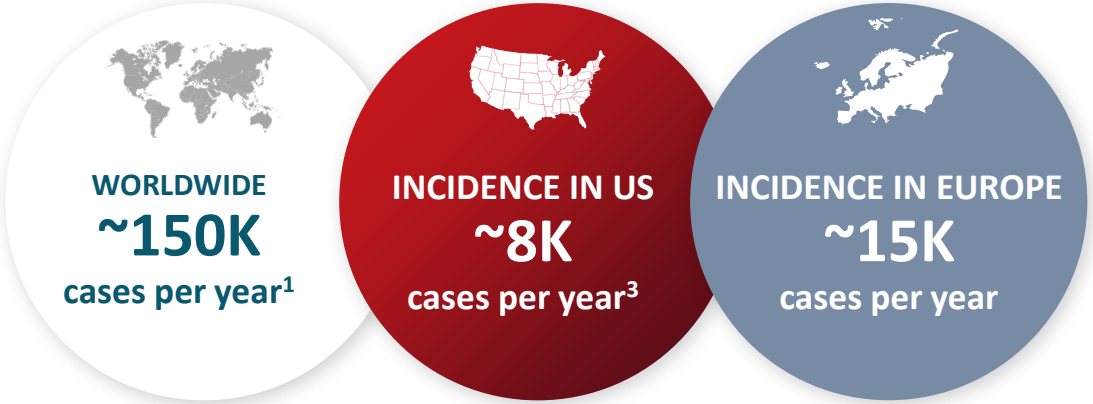
Tanruprubart: First-in-Kind Targeted Therapy for Guillain-Barré Syndrome

Potential Blockbuster Opportunity Poised to Replace Standard of Care

GBS: Sudden Neurological Emergency

No FDA-approved therapies

IVIg used off-label



SIGNIFICANT DISEASE BURDEN DESPITE IVIG TREATMENT [1,2,3,4,5,6,7]

~30%
admitted to ICU

~75%
in ICU require ventilation

~20%
can't walk a year after treatment

- Most common cause of acute neuromuscular paralysis
- Typically caused by infection, vaccine side effects, other therapies
- **>$7B annual burden associated with the disease[8]**

[1]ClearView Health Market Research (2024); [2]Hughes et al. (2003). *Neurology, 61*, 736-40; [3]Hund et al. (1993). *Crit Care Med, 21,* 433-46; [4]Doets, et al. (2018). *Brain, 141*, 2866-77; [5]Van den Berg et al. (2014). *Nat Rev Neurol, 10*, 469-82; [6]Leonhard et al. (2019). *Nat Rev Neurol, 15*, 671-83; [7]Inflation- and population-adjusted cost estimates from Frenzen (2008). *Neurology, 71*(1), 21-7; [8]Ongoing Annexon study submitted for AAN presentation

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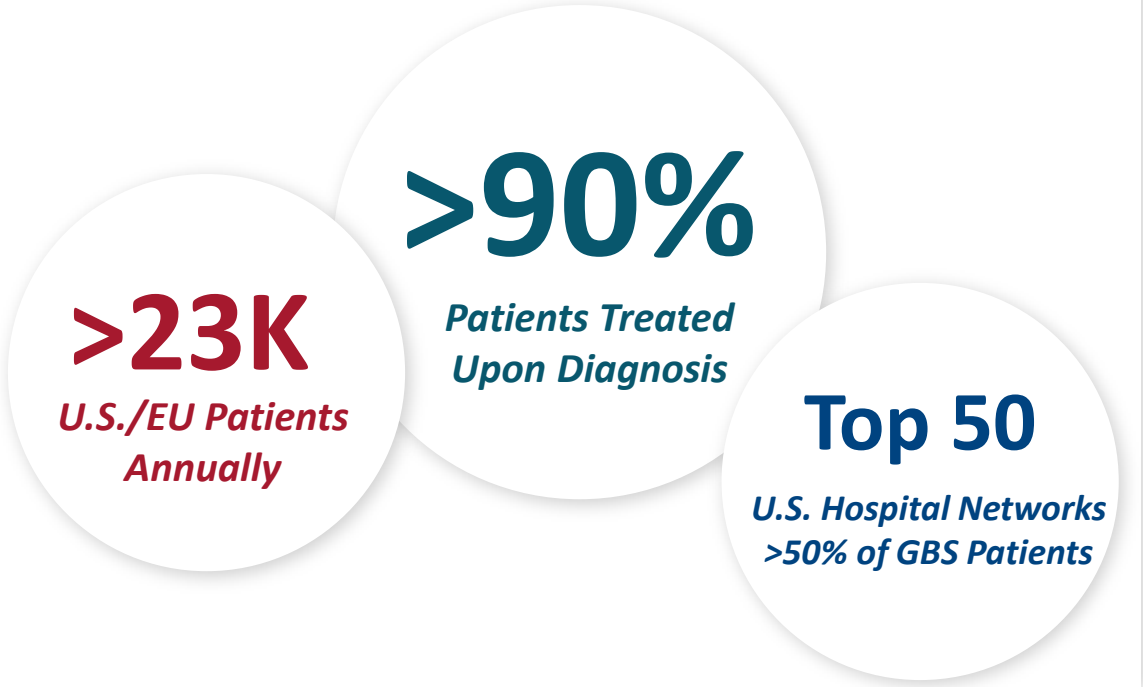
GBS Provides a Compelling Market Opportunity

Tanruprubart is first targeted therapy designed to create a new standard of care in GBS

BLOCKBUSTER MARKET OPPORTUNITY FOR GBS



Tanruprubart

- **Single infusion** halts neuroinflammation
- **~90% of treated patients improved** by week 1
- **Safety** data comparable to placebo
- **Significant potential savings to U.S. healthcare** system over IVIg/PE

Current treatments are slow and suboptimal
Most patients suffer from incomplete benefit

Targeted treatment offers faster, more complete recovery for patients to regain their independence

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Pivotal Phase 3 Trial Design

Randomized, Double-Blind, Placebo-Controlled Study

PATIENT SELECTION

- GBS Disability Score 3, 4 or 5
- <10 days from onset of weakness till treatment
- IVIg or PE not available to patients
- Stratified by baseline prognostic factors: muscle strength and time from onset of weakness

KEY ENDPOINTS

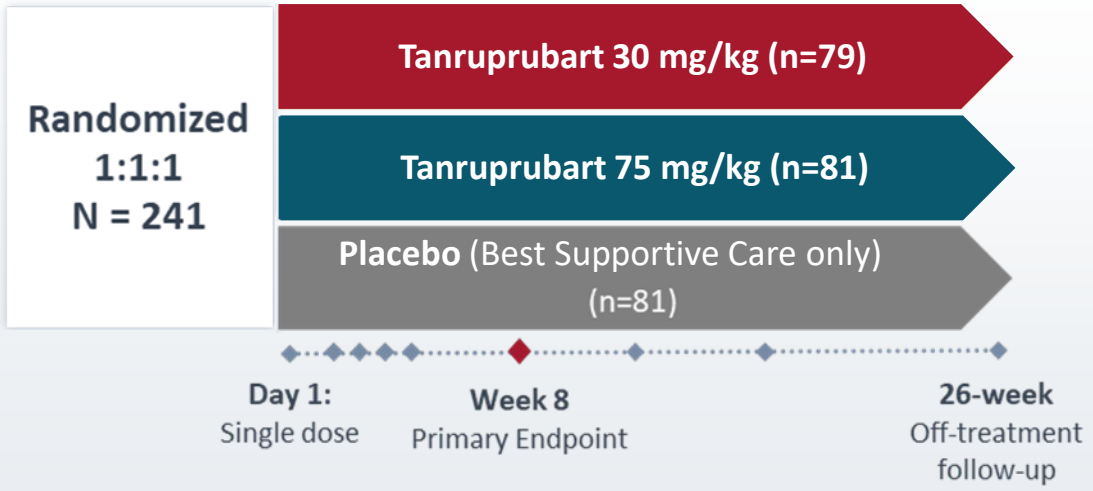
- **Primary Outcome Measure:** GBS Disability Score (GBS-DS) at week 8
- **Secondary Endpoints:** Muscle strength (MRC sumscore), Motor Disability (ONLS), Duration of Ventilation

DOSE SELECTION

- Phase 1b suggested tanruprubart 30 mg/kg may be most efficacious (approximately 1 week of complement inhibition)
- Determine optimal dose assuming both or either dose could be efficacious

STATISTICAL ANALYSIS PLAN

- Both doses tested independently and simultaneously vs placebo
- Analysis of primary and key secondary endpoints alpha-protected with Hochberg procedure



Conducted at Sites in Bangladesh and the Philippines

Tanruprubart (ANX005) administered as a single intravenous dose

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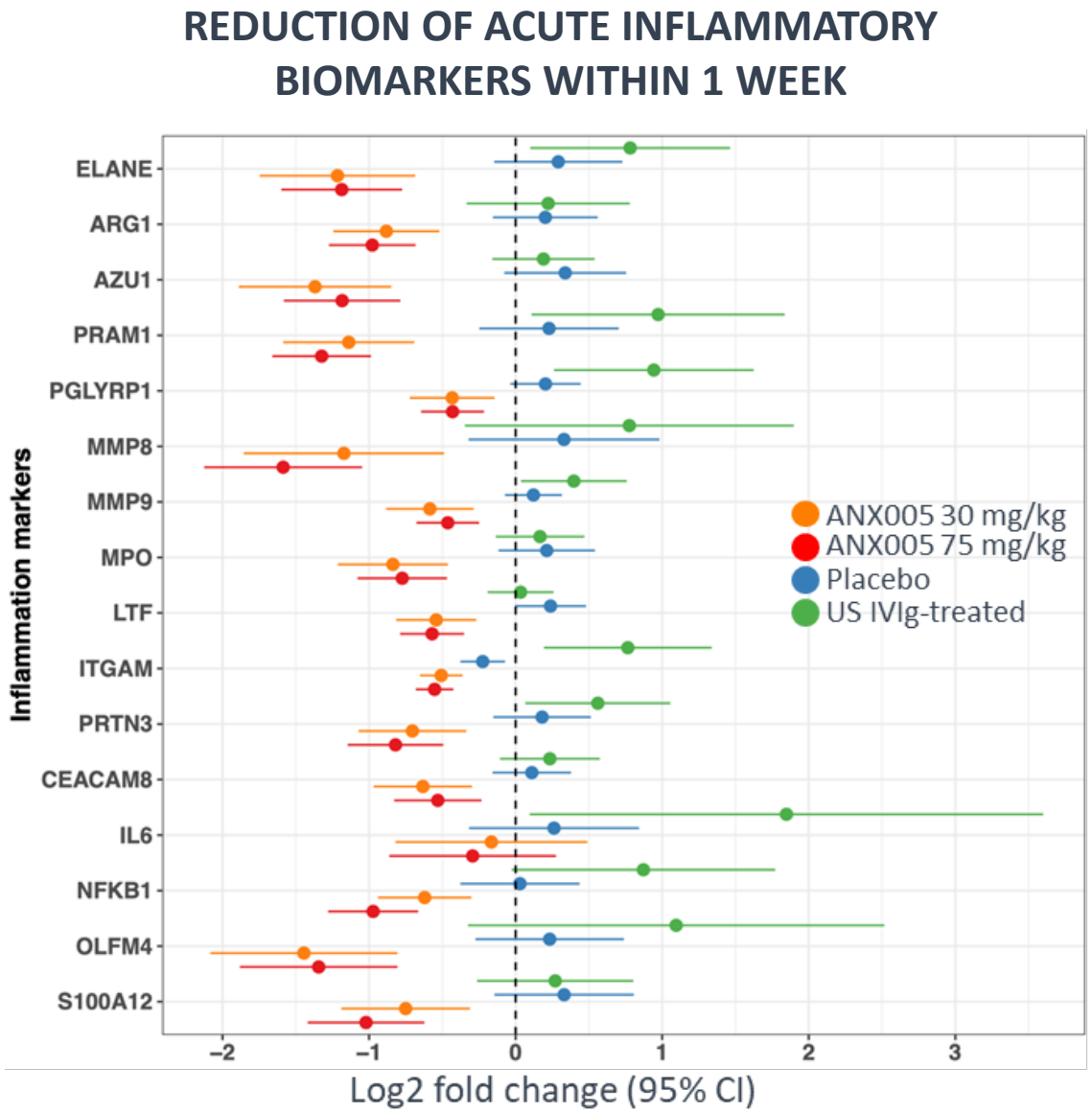
Phase 3: Unlike IVIg or Placebo, Tanruprubart Significantly Reduced Key Markers of Neuroinflammation Within 1 Week



Resulted in Rapid Muscle Strength and Motor Function Recovery

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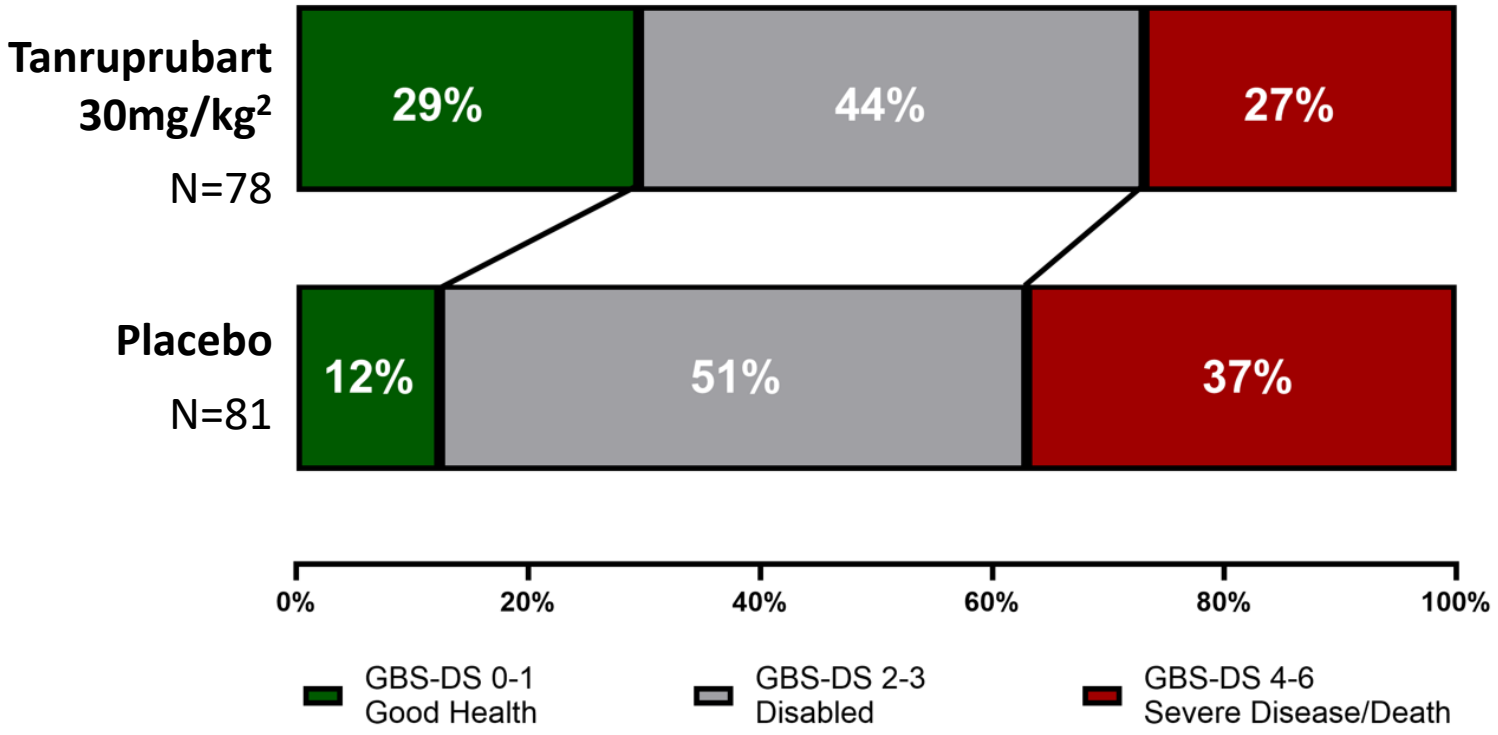
Phase 3: Tanruprubart Demonstrated Highly Significant and Clinically Meaningful Treatment Effect on Primary Endpoint, GBS-DS, at Week 8

2.41-fold higher likelihood of being in a better state of health relative to placebo

Grotta Bar of GBS-DS[1] at Week 8



OR 2.41, p=0.0058

[1]GBS-DS is the GBS disability scale
[2]Phase 3 data reported on the 30 mg/kg dose

Topline Results Subject to Change

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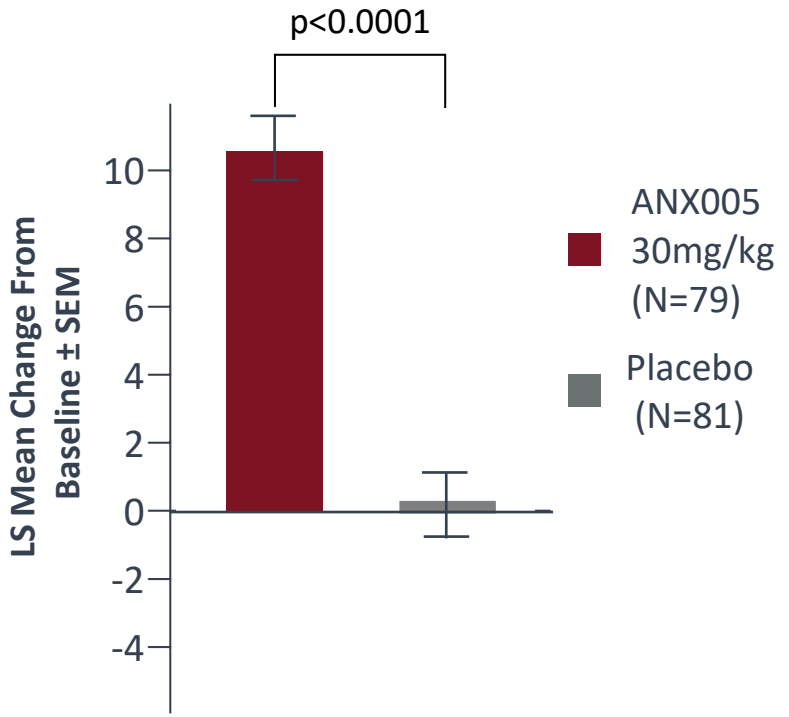
Phase 3: Tanruprubart Treatment Outcomes Were Rapid and Durable, with >2x More Treated Patients Fully Recovered at Month 6

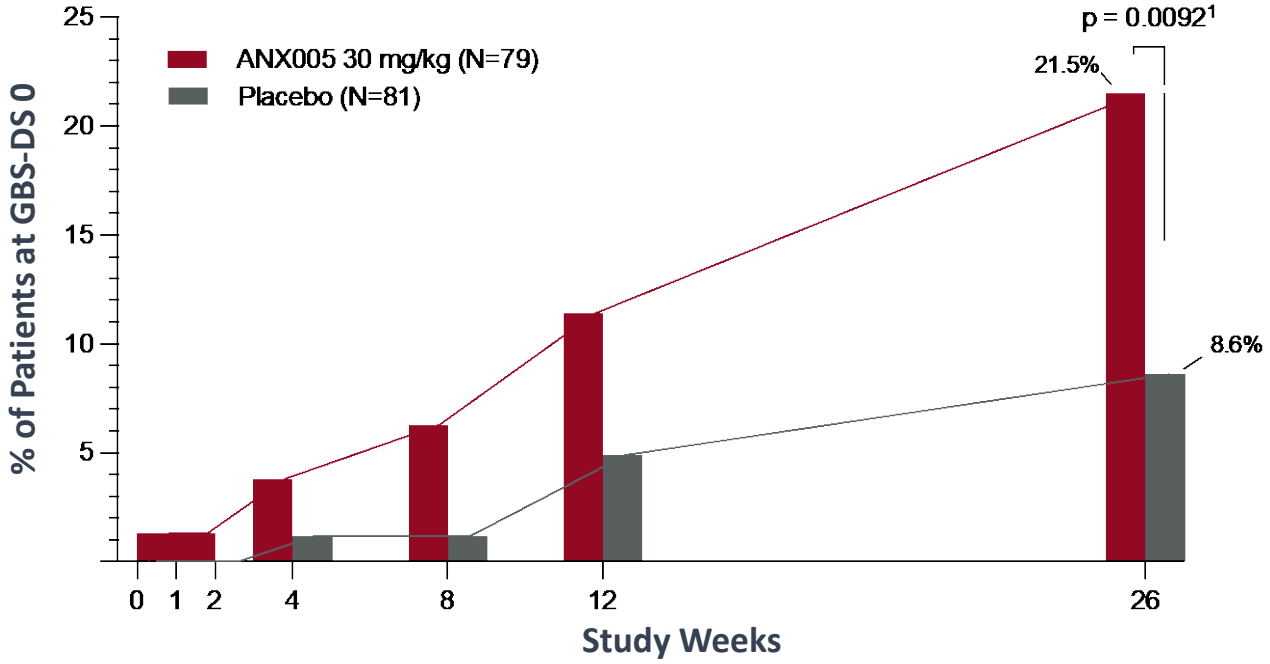
86% OF PATIENTS TREATED WITH ANX005 IMPROVED IN WEEK 1 (10 PTS ON AVG)

MRC Sumscore at Week 1



>2 TIMES MORE TREATED PATIENTS FULLY RECOVER AT WEEK 26 (GBS-DS = 0)

GBS-DS=0 Through Week 26



[1]MRC Sumscore
[2]nominal

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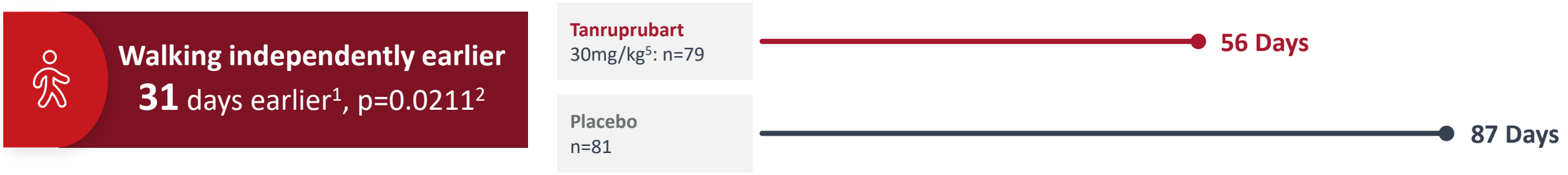
Phase 3: Tanruprubart Demonstrated Profound Impact on Measures Most Important To Patients, HCPs and Payers

Helped Patients Achieve Their Independence Sooner versus Placebo

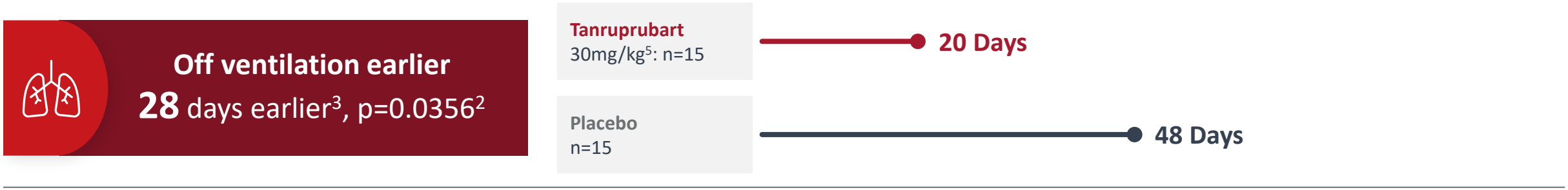
Walking independently earlier

31 days earlier[1], p=0.0211[2]



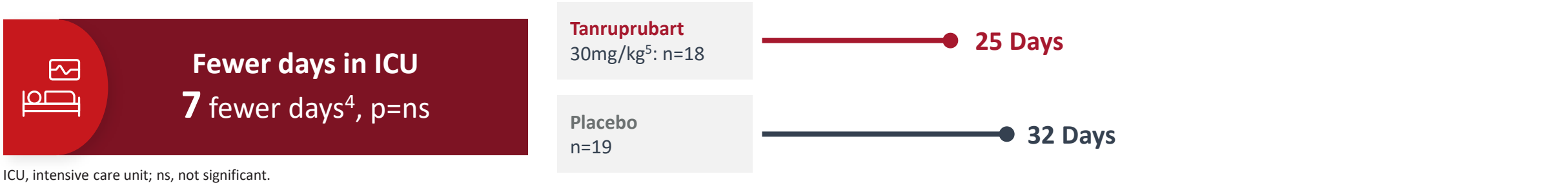
Off ventilation earlier

28 days earlier[3], p=0.0356[2]



Fewer days in ICU

7 fewer days[4], p=ns



ICU, intensive care unit; ns, not significant.

[1]Based on first scheduled visit of recording [2]Nominal [3]Among patients ventilated [4]Among patients requiring ICU

*Phase 3 data reported on the 30 mg/kg dose

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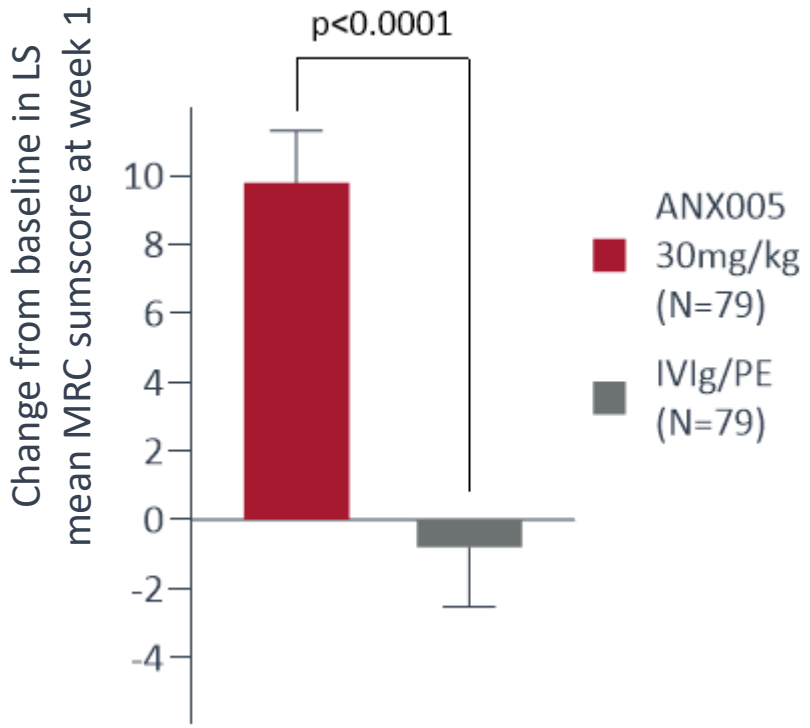
Real World Evidence Study: Tanruprubart Treatment Showed Early & Durable Benefit over Matched IGOS IVIg/PE Treated Patients

MORE THAN A 10-POINT IMPROVEMENT IN MRC SUMSCORE[1] OVER IVIG/PE

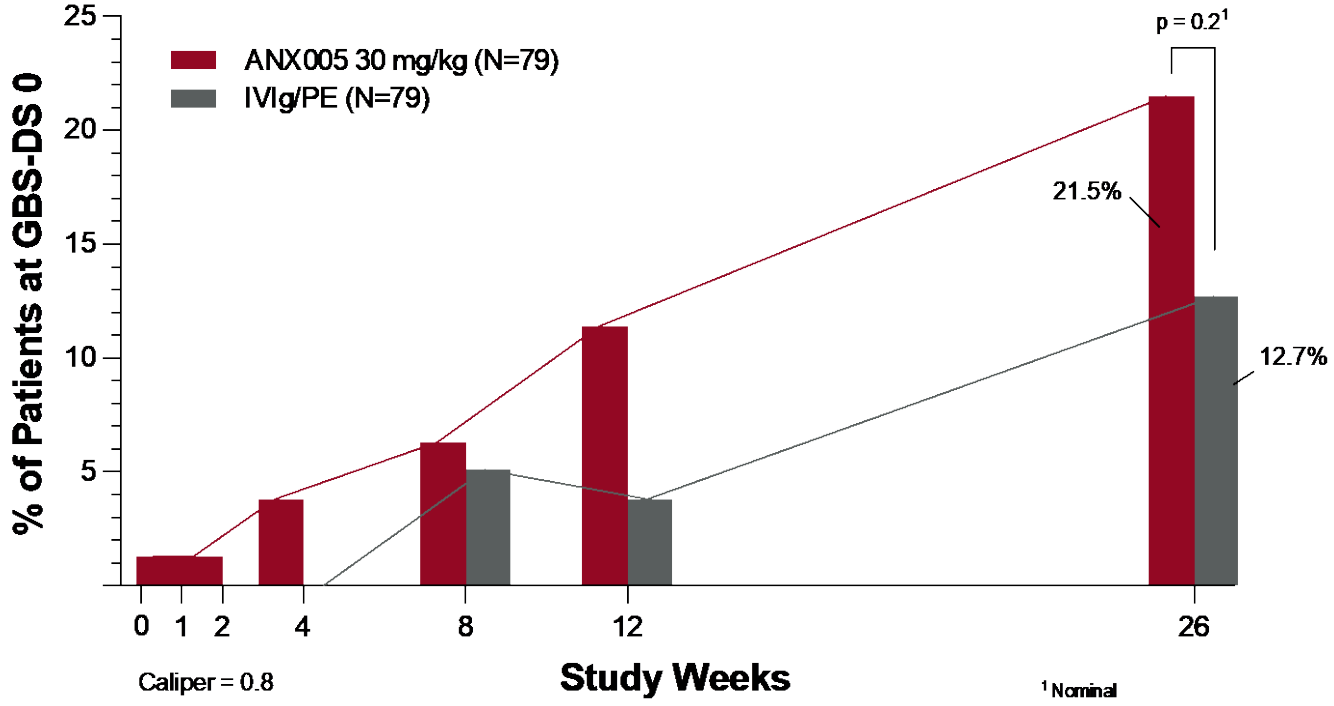
MRC Sumscore at Week 1



[1]MRC Sumscore
[2]nominal

MORE TANRUPRUBART PATIENTS FULLY RECOVERED AT WEEK 26 (GBS-DS = 0) COMPARED TO IVIG/PE

GBS-DS=0 Through Week 26



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Early Experience with Tanruprubart Treatment in EU & US Suggests Rapid and Consistent Effect Across Geographies

Example Patient: Moderate to severe

- **Baseline: bed-bound, hospitalized**
- Treated with tanruprubart within 4 days from onset
- **Day 8[1]: discharged from hospital, walking with assistance**
- **Day 29[1]: walking independently**

Initial

FORWARD STUDY Data

anticipated in 2026

BLA planned in 2026

[1]Post tanruprubart treatment

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On the Path to Bringing Tanruprubart to GBS Patients Worldwide

PHASE 3 PLACEBO-CONTROLLED TRIAL POSITIVE OUTCOMES AND FAVORABLE SAFETY PROFILE

Faster and more complete recovery with ANX005 30mg/kg vs. placebo

REAL WORLD EVIDENCE DEMONSTRATED BENEFIT OVER IVIg

Ph 3 patients matched with majority Western World population

Earlier and greater benefits of ANX005 30 mg/kg single dose over IVIg/PE

NEXT STEPS

- ✓ **MAA Filed in Europe Jan '26**
- ➢ **BLA Submission Planned 2026**
- ➢ **Ramping disease education and engaging payers on disease burden to optimize coverage and reimbursement**

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ANX1502: First Oral C1 Inhibitor for Complement-Mediated Diseases

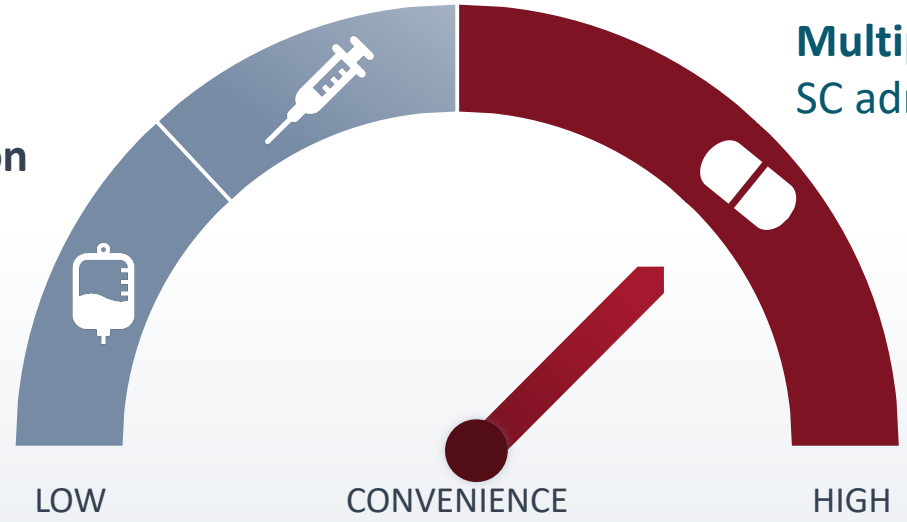
First-in-kind potential to disrupt biologics-treated indications

ANX1502 Next Generation Oral Therapy

Potential to Transform the Treatment of Neuroinflammatory Autoimmune Diseases

Daily self- or HCP-administration
Self-administered needle-phobia
Less flexibility
Less convenience

In clinic administration
Time consuming, expensive, and inconvenient



ANX1502 ORAL DOSING

Clinically Validated Target

Multiple Blockbuster Therapies with IV & SC administration

Best-in-class Potential with enhanced convenience & equivalent or better efficacy and safety profile

100K+ PATIENTS WITH AUTOIMMUNE DISEASES
currently treated with biologics in US*

*Data on file.

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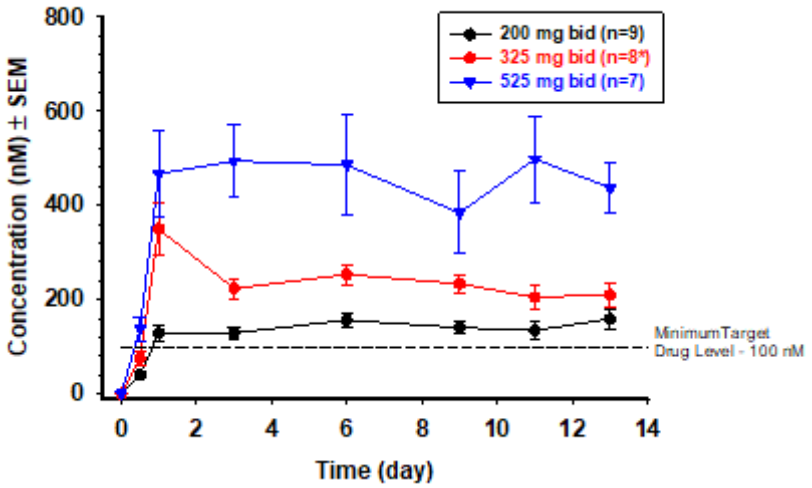
ANX1502 Well Tolerated & Achieved Target Dosing Objectives in Phase 1

Target drug levels reached in fasted healthy volunteers with oral twice-daily dosing; supportive impact on PD biomarker

SAFETY AND TOLERABILITY WITH LIQUID SUSPENSION

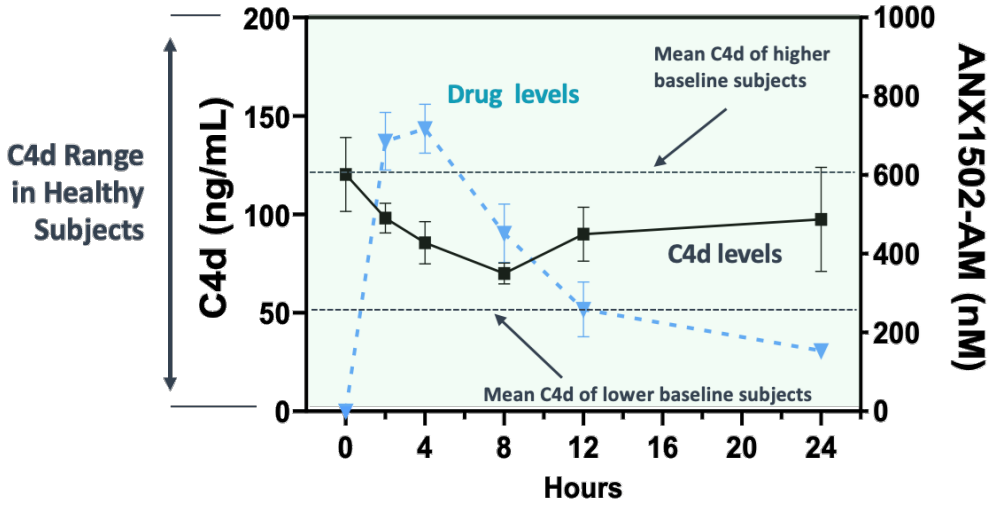
- All treatment-emergent adverse events (TEAEs) mild or moderate
- Most frequent TEAEs were GI related[1]
- **No serious adverse events (SAEs)**
- **No significant clinical/lab findings[2]**

TARGET LEVELS OF ACTIVE DRUG CONSISTENT WITH BID DOSING (MAD STUDY)



- Dose-proportional PK (AUC) was observed in the MAD cohorts

INITIAL *IN VIVO* PD SIGNAL WITH COMPLEMENT ACTIVATION BIOMARKERS (SAD STUDY)



- C4d used as a biomarker reflects drug's *in vivo* impact on C1s activation
- ANX1502 suppressed C4d serum levels in healthy volunteers w/ higher than median baseline C4d

[1]Gastro-intestinal (nausea, emesis, and diarrhea)
[2] Liver enzymes, serum chemistry, or hematology

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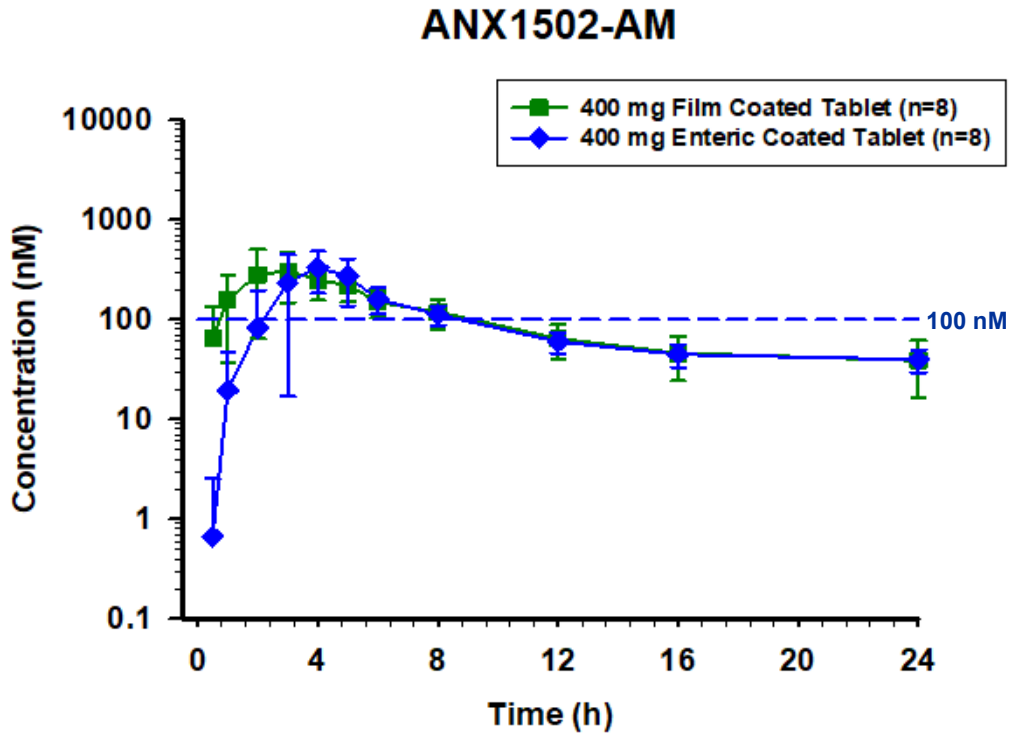
Transitioned to ANX1502 Enteric-coated Tablet For Enhanced Tolerability; Similar Exposure in Healthy Volunteers to Earlier Tablet Formulation

ANX1502 IS THE FIRST ORAL INHIBITOR OF THE CLASSICAL PATHWAY

- Single dose data with enteric-coated tablet show similar exposure to earlier tablet formulation
- Target concentration levels maintained with BID dosing
- Enteric-coated tablet generally well-tolerated in healthy volunteers

SINGLE 400 MG DOSE OF ANX1502 ENTERIC COATED TABLET FORMULATION VS FILM COATED TABLET



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Currently Assessing ANX1502 in POC Cold Agglutin Disease (CAD); Aim to Pursue in Multiple Neuroinflammatory Autoimmune Diseases

Pioneering Clinical Development of First Oral C1 Inhibitor

WELL-TOLERATED TABLET formulation

POTENCY observed to be comparable to approved therapies with twice daily dosing

CLINICALLY EVALUATING EFFICACY in CAD patients using objective markers

Potential to serve **HIGH UNMET NEED** in neuromuscular and large autoimmune indications

NEXT STEPS

Dosing ongoing to enhance understanding of ANX1502's PK/PD profile and effect on complement and clinical markers of hemolysis

CAD PoC trial completion anticipated in 2026

MISSION DRIVEN

helping millions of people
impacted by devastating
neuroinflammatory diseases to

LIVE THEIR BEST LIVES

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