ANNEXON biosciences

STOP THE START

of classical complement-driven diseases

CORPORATE PRESENTATION | AUGUST 2024

Nasdaq: ANNX

Forward-Looking Statements

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 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 of submission of a Biologics Licensing Application, 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.

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A bold mission to enable MILLIONS of PATIENTS impacted by complement-mediated diseases of the body, brain and eye LIVE THEIR BEST LIVES

Annexon Bio: Intentionally and Rigorously Tackling an Array of Classical Complement-Mediated Diseases

Stopping the Start of Classical Pathway Neuroinflammation

Broad Therapeutic Application of Late-Stage Clinical Platform

Multiple Near-Term Clinical Catalysts

ON A JOURNEY TO HELP PATIENTS REGAIN THEIR INDEPENDENCE

Well-researched MOA demonstrated differentiated functional outcomes across GBS, CAD, GA and HD

Suite of fit-for-purpose drug candidates for diseases of the body, brain and eye

- ✓ *GBS pivotal Ph3 data readout (Q2)*
- ✓ *GA pivotal Ph3 initiation (mid-yr)*
- *Oral program POC data readout (2H)*

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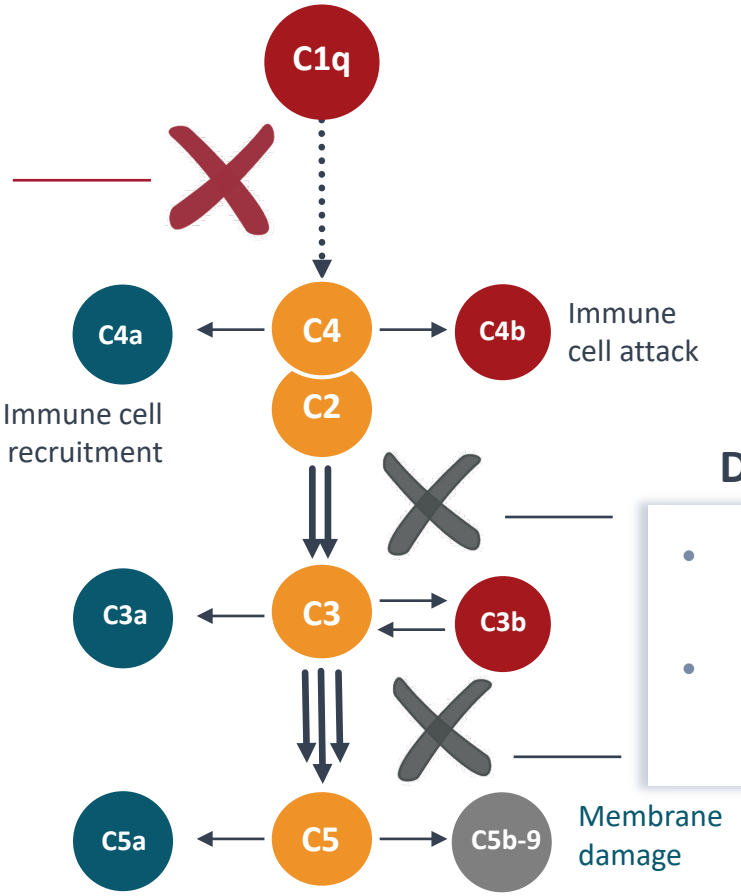
ANX005 Rapidly Shuts Down Activation of the *ENTIRE* Classical Complement Cascade on the Nerve to Prevent Acute Injury

Classical Complement Drives Harmful Inflammation and Tissue Destruction

STOPPING AT THE START

- Blocks upstream and downstream[1] inflammation & tissue damage
- Before downstream bypass mechanisms (breakthrough) and pathway amplification
- Differentiated functional outcomes shown in GBS, GA, HD and ALS



DOWNSTREAM APPROACHES (C3/C5)

- Do not block ongoing inflammatory pressure of upstream classical pathway
- More susceptible to complement bypass mechanisms / inflammatory breakthrough

[1]Lansita, et al., 2017; DOI: 10.1177/1091581817740873

GBS: Guillain-Barré Syndrome; GA: geographic atrophy; HD: Huntington's Disease; ALS: amyotrophic lateral sclerosis

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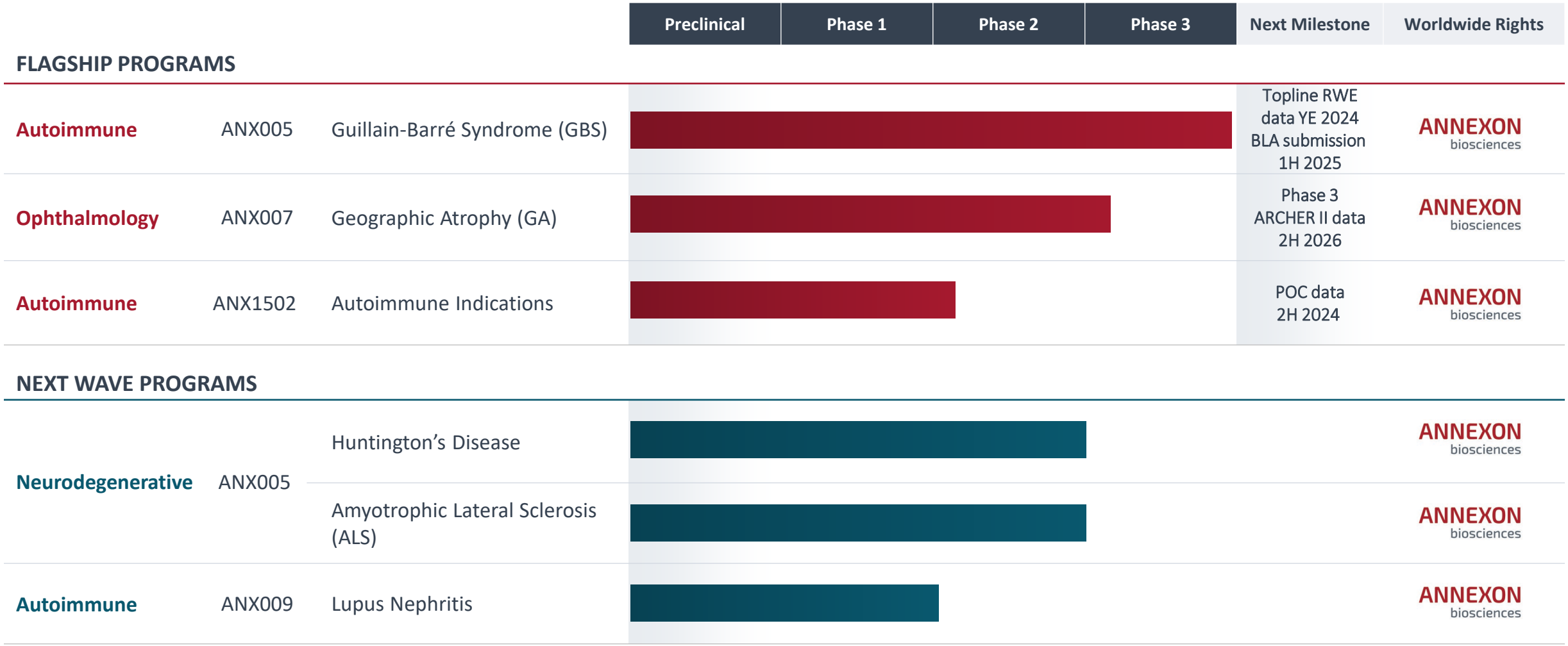
Industry's Leading Complement-Focused Pipeline

Diverse late-stage clinical platform for classical complement-mediated neuroinflammatory diseases of the body, brain and eye



POC: Proof-of-Concept

Annexon GBS Phase 1b data presented at American Academy of Neurology 2020 and Peripheral Nerve Society Annual Meetings 2021-2022.

Maximizing Pipeline Potential with THREE Clinical Priorities

1

Deliver
1st placebo-controlled pivotal dataset for GBS in 40 years

2

Initiate
1st global pivotal program for GA using vision preservation
as primary outcome measure

3

Advance
1st-in-kind oral classical complement inhibitor to **clinical proof-of-concept**

GBS: Guillain-Barré Syndrome. GA: Geographic Atrophy.

2024: Transformational Year with Several Program Catalysts

Operating runway into **2H 2026** funding **multiple clinical catalysts**

2024 ANTICIPATED MILESTONES

ANX005	✓ **GBS pivotal trial readout**
ANX007	✓ **GA P3 ARCHER II trial initiation**
ANX1502	✓ **Bridging study to twice-daily tablet formulation**
ANX007	✓ **GA P3 ARROW trial initiation**
ANX1502	CAD proof-of-concept trial readout
ANX005	GBS topline RWE comparability data

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ANX005: First-in-Kind C1q Inhibitor for Guillain-Barré Syndrome

Positive Topline Results from Pivotal Phase 3 Trial

Shane S.
53-year-old patient with GBS

Summary: ANX005 Breakthrough Phase 3 Win for GBS Patients Worldwide

A single infusion demonstrated robust, consistent benefit across multiple endpoints

Met Primary Endpoint

OR[1] 2.4, P=0.0058

2.4-fold higher likelihood of being in a better state of health on GBS-DS at Week 8

- ✓ FDA-agreed primary endpoint
- ✓ Multiple sensitivity analyses of the primary endpoint show consistent improvements
- ✓ Larger effect in sub-group with western baseline characteristics

Expedited Recovery

Patients Got Better Sooner

Early, robust & clinically meaningful benefit on multiple outcome measures @ Week 8

- ✓ Able to walk earlier vs placebo
- ✓ Able to run earlier vs placebo
- ✓ Less nerve damage vs placebo

Durable Treatment Effect

Maintained improvement over placebo at all timepoints across multiple measures

- ✓ Less time on ventilation
- ✓ Less overall disability

Generally Well Tolerated

Safety data was similar to placebo

- ✓ No new safety signals
- ✓ No increased infection rate while not requiring vaccination or prophylactic antibiotics
- ✓ No difference in all-cause mortality

[1]Odds Ratio: Likelihood that a patient on ANX005 is in a better state of health relative to placebo

Topline Results Subject to Change

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GBS is a Neurological Emergency with Long-Term Disability

Requires a targeted and effective intervention to immediately block the Classical Complement Pathway

POST-INFECTIOUS COMPLEMENT-MEDIATED DISEASE

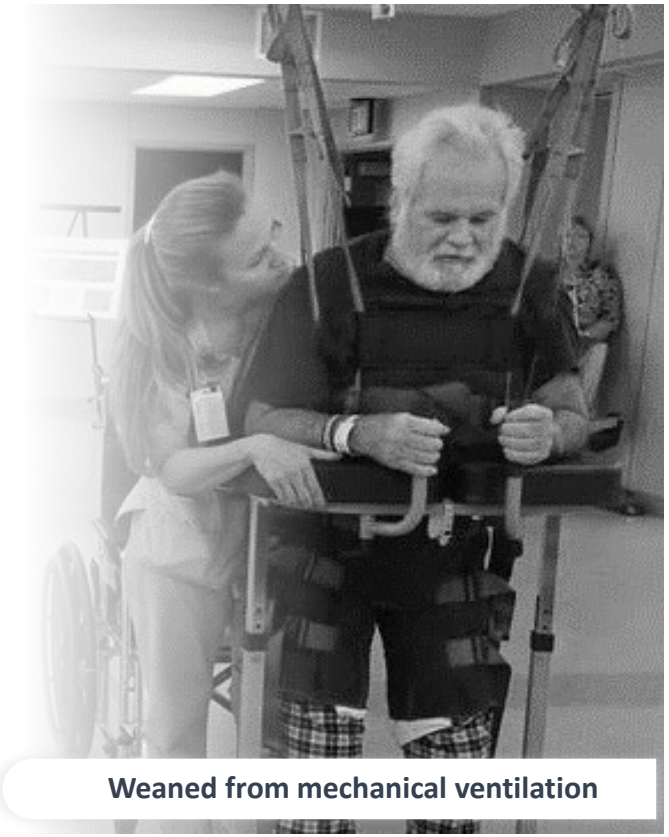
- Following infection, complement-activating autoantibodies attack nerves leading to nerve damage & acute paralysis

HIGH UNMET MEDICAL NEED

- 22,000 patients hospitalized in US & Europe annually
- Global annual incidence ~150,000
- IVIg not FDA approved, no pbo-controlled IVIg trials in GBS
- IVIg requires 5-day treatment, black-box warning for thrombosis

SIGNIFICANT MORBIDITY

- Despite IVIg, GBS results in:
  - Severe weakness and paralysis
  - Ventilation in 25% of patients
  - Uncertain and incomplete recovery



Weaned from mechanical ventilation

Classical complement drives neuroinflammation and tissue destruction in GBS

ANX005 is an anti-C1q antibody that rapidly shuts down the entire classical complement pathway

1) van Doorn, 2013; Willison et al., 2016 2(van den Berg et al., 2014) 3 Walgaard et al (2021) Lancet Neurology 20(4):275, 4AAN Guidelines "Immunotherapy for GBS", 4Hund EF et al (1993) Crit Care Med 21:433, 454Fletcher D, et al. (2000) Neurology 27;54(12), 46, 5Van den Berg B, et al (2014) *Nat Rev Neurol* Aug;10(8), 6Stephan et al (2012) Neuroscience 35:369, 7Lansita et al (2017) Int. J. Toxicol. 36:449

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Well Designed and Executed Pivotal Phase 3 Trial

Randomized, Double-Blind, Placebo-Controlled Study (Best Supportive Care, no IVIg or PE)

PATIENT SELECTION

- Baseline GBS-DS score 3-5
- <10 days from onset of weakness
- Stratified by baseline prognostic factors: muscle strength and time from onset of weakness

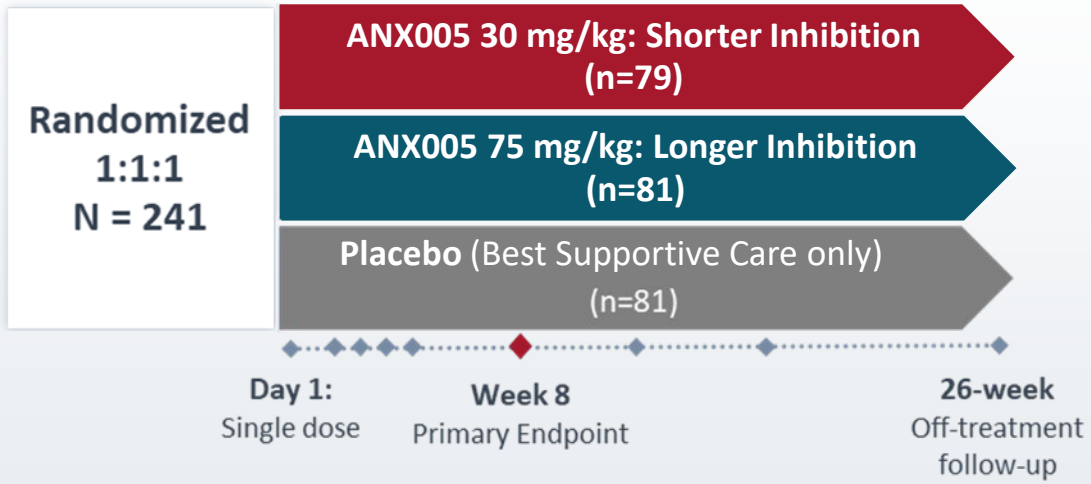
KEY ENDPOINTS

- **Primary Outcome Measure:** GBS-DS at week 8: well-accepted regulatory endpoint assessing functional status
- **Secondary Endpoints:** Muscle strength (MRC sumscore), Motor Disability (ONLS), Duration of Ventilation

KEY LEARNINGS

- **Shorter duration of complement inhibition with 30 mg/kg resulted in better outcomes**
- **Confirms initial observations from Phase 1b study**

2 DOSES SELECTED TO DETERMINE MOST EFFECTIVE DURATION OF INHIBITION



Conducted at Sites in Bangladesh and Philippines

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Baseline Characteristics Similar and Well Balanced Across Treatment Groups

Stratified by Key Prognostic Factors: MRC Sumscore and Time Since Onset of Weakness

Baseline Characteristic	Placebo (N=81)	ANX005 30mg/kg (N=79)	ANX005 75mg/kg (N=81)
Age at Screening (years); mean (SD)	34.2 (12.59)	36.9 (14.88)	37.9 (15.29)
Sex, n (%) Male	57 (70.4)	51 (64.6)	51 (63.0)
Baseline GBS-DS Score, n (%)			
3 Able to walk 10 meters across open space with help	7 (8.6)	12 (15.2)	10 (12.3)
4 Bedridden or chair bound	64 (79.0)	56 (70.9)	60 (74.1)
5 Requiring assisted ventilation for at least part of the day	10 (12.3)	11 (13.9)	11 (13.6)
Baseline MRC Sumscore (range 0-60), n (%)			
21-60 Mild/moderate loss of muscle strength	42 (51.9)	41 (51.9)	44 (54.3)
0 - 20 Severe loss of muscle strength	38 (46.9)	38 (48.1)	37 (45.7)
Time since of onset of weakness to randomization Days, mean (SD)	6.4 (1.7)	6.3 (1.9)	6.5 (2.0)
Time since of onset of weakness to treatment Days, mean (SD)	6.5 (1.7)	6.3 (1.9)	6.6 (2.0)
Electrophysiology by Hadden criteria, n (%)			
Acute Inflammatory Demyelinating Polyneuropathy (AIDP)	18 (22.2)	16 (20.3)	16 (19.8)
Acute Motor Axonal Neuropathy (AMAN)	49 (60.5)	50 (63.3)	50 (61.7)
Other	14 (17.3)	13 (16.5)	15 (18.5)

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Summary of Primary and Key Secondary Results

Statistical testing hierarchy of clinically relevant endpoints

Primary	Endpoint	Assesses	Timepoint	30 mg/kg Efficacy	P-value
1	**GBS-DS**	**GBS disability**	**Week 8**	**OR[1] = 2.41**	**0.0058**

Secondary Hierarchy	Endpoint	Assesses	Timepoint	30 mg/kg Efficacy	P-value
2	**Overall Neuropathy Limitations Scale (ONLS)**	Functional disability	Week 8	-0.8[2]	0.0965[3]
3	**MRC Sumscore**	Muscle strength	**Week 8**	**4.0[2]**	**0.0351[3]** *Nominal*
4			**Day 8**	**10.0[2]**	**<0.0001[3]** *Nominal*
5	**Ventilation**	Duration of ventilation[3]	**Week 26**	**Median 28 fewer days**	**0.0356[4]** *Nominal*

[1]Odds Ratio: Likelihood that a patient on ANX005 is in a better state of health relative to placebo

[2]LS mean point improvement relative to placebo

[3]P-values for nominal testing using 2-sided α=0.05

[4]For those requiring ventilation

Overview of Primary Endpoint: GBS-DS at Week 8

FDA accepted endpoint with alignment on statistical methodology

GBS-DS SCALE COLLAPSED INTO 3 CATEGORIES

Enhances Clinical Interpretability

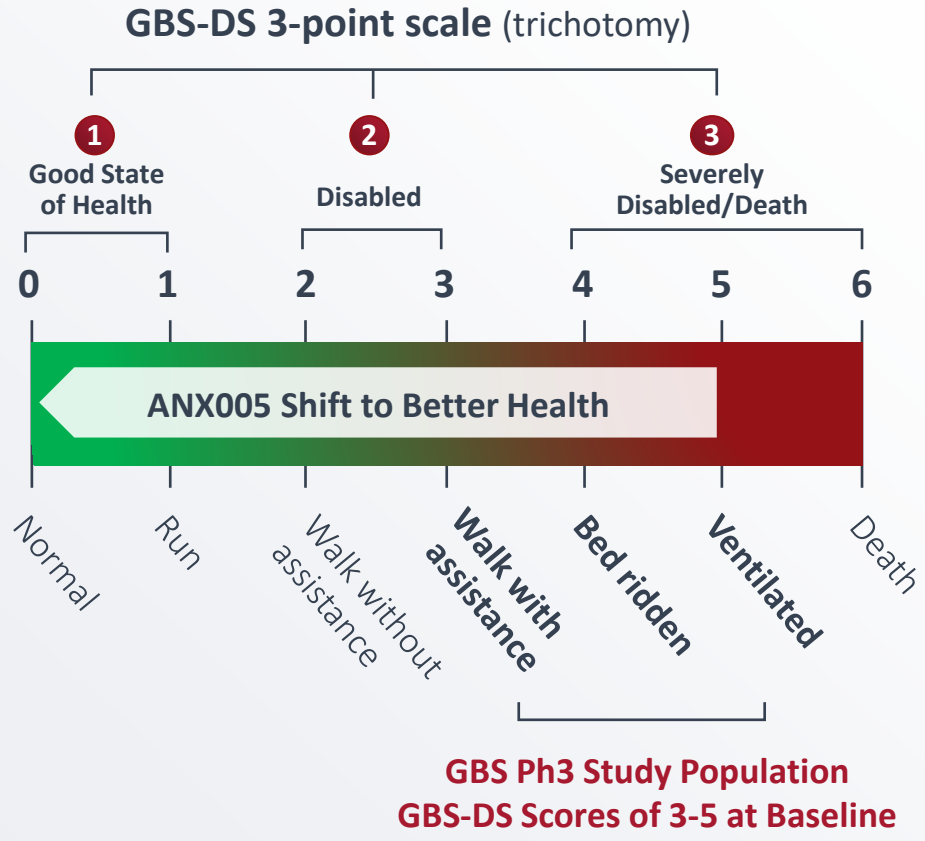
Approach: Collapse 7-grade scale to a 3-grade scale (trichotomy)

- **0-1:** Good State of Health
- **2-3:** Disabled
- **4-6:** Severely Disabled/Death

Rationale:

- ✓ Enhances clinical interpretability by focusing on a subject's actual health status at week 8 after receiving ANX005 or placebo
- ✓ Evaluates patients across all health states
- ✓ Most efficient statistical analysis approach

GBS-DS SCALE FOR PIVOTAL PHASE 3



Topline Results Subject to Change

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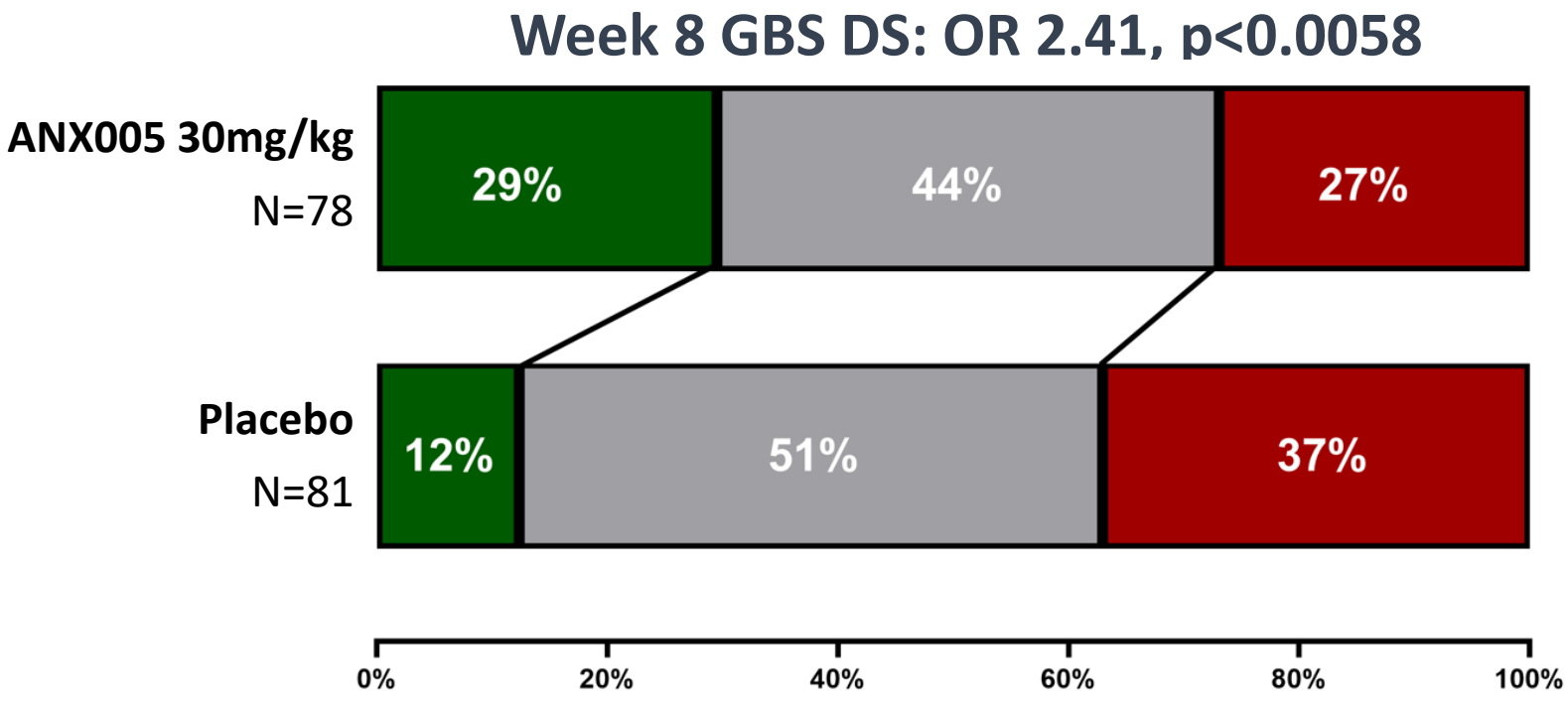
Highly Significant, Clinically Meaningful Treatment Effect at Week 8

Primary Endpoint: 2.41-fold more likely to be in better state of health vs. placebo with ANX005 30 mg/kg



Approach: Collapse 7-grade scale to a 3-grade scale (trichotomy)

- **0-1:** Good State of Health
- **2-3:** Disabled
- **4-6:** Severely Disabled/Death

Topline Results Subject to Change

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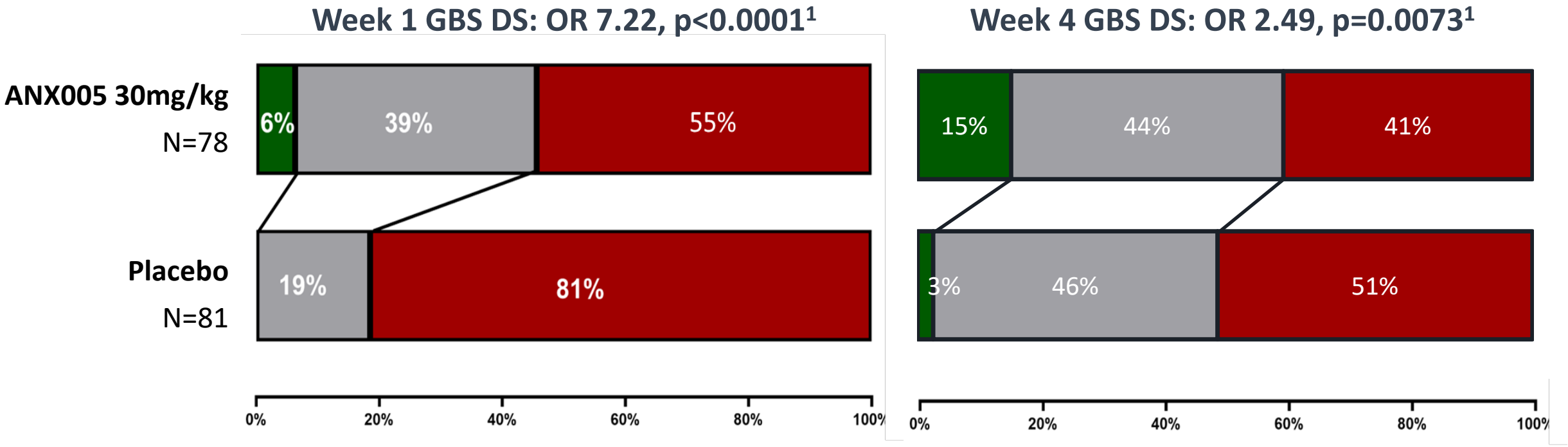
Early, Robust Treatment Effect on GBS-DS at Week 1 and Week 4



[1]Nominal

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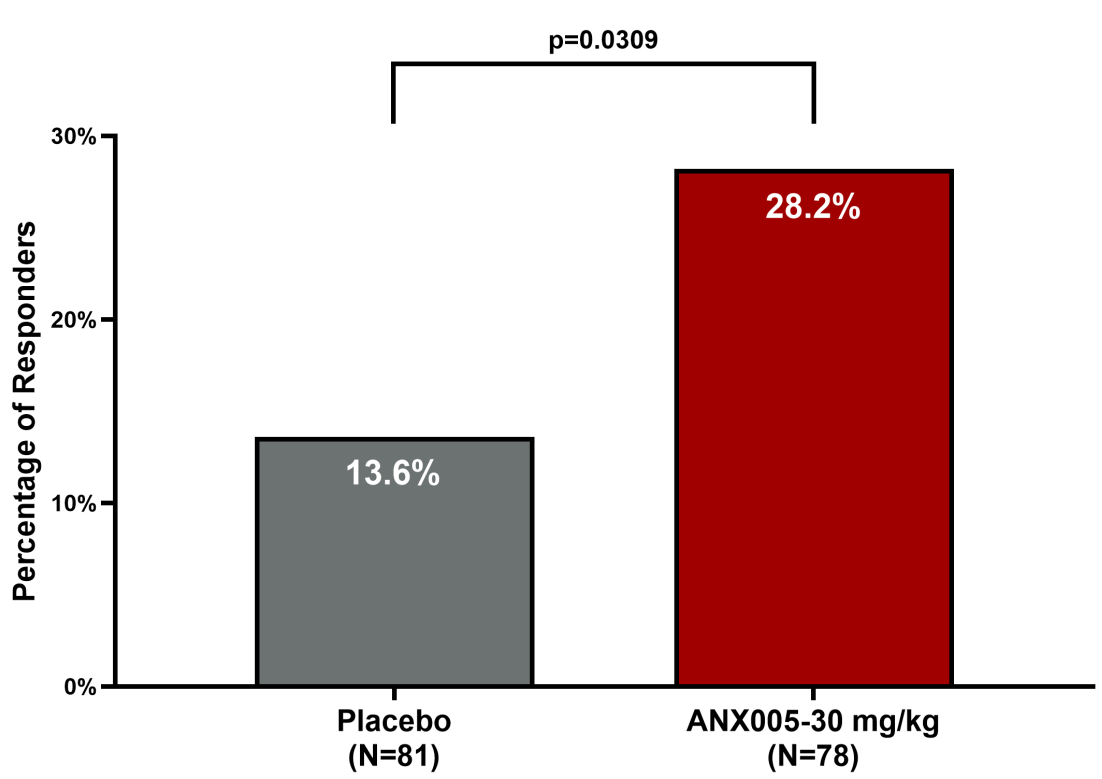
Robustness of Week 8 GBS-DS Sensitivity Analyses

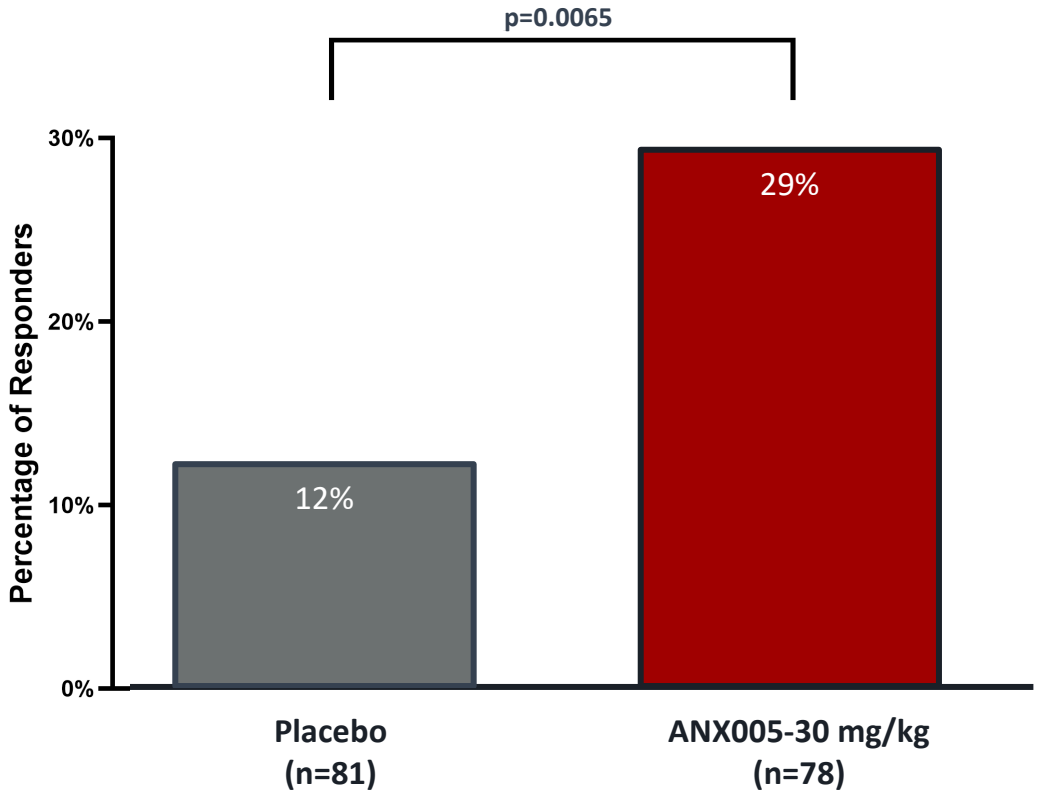
Responder Analysis: ≥3-point at week 8

2x More Patients Improved 3 Points or More



Traditional Dichotomy: (0-1, 2-6) at week 8

2.5x More Patients Were Able to Run or Better (OR 3.34)



[1]Nominal

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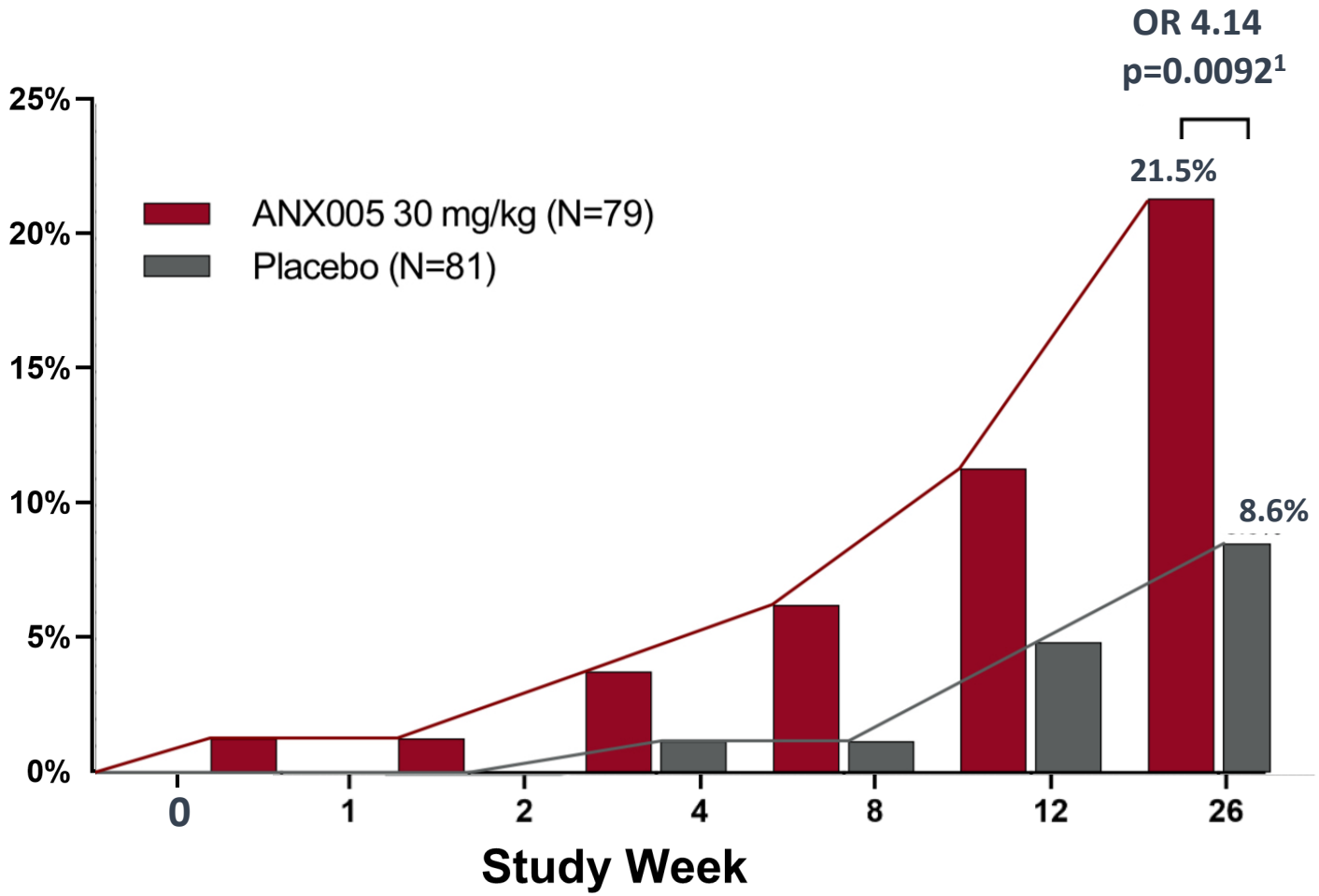
Accelerated Path to Recovery at Week 26

Significantly more patients reached 'normal' by Week 26 in prespecified analysis

2.5 times more treated patients fully recover at Week 26 (GBS-DS = 0)

Effect begins early and grows throughout study



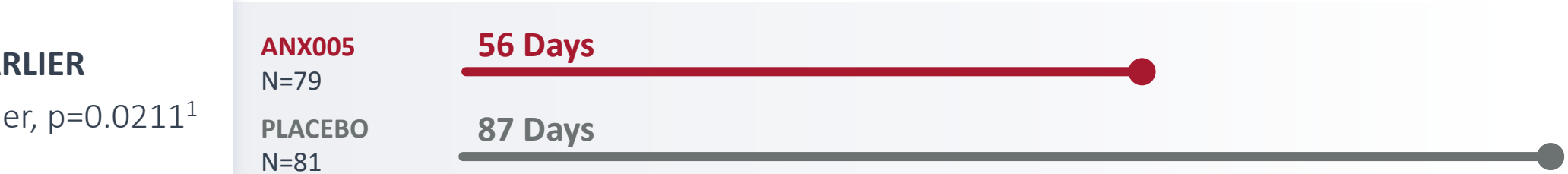
[1]Nominal

Getting Better Sooner: Helping Patients Achieve Independence

ANX005 30 mg/kg consistently showed faster recovery across clinically important measures

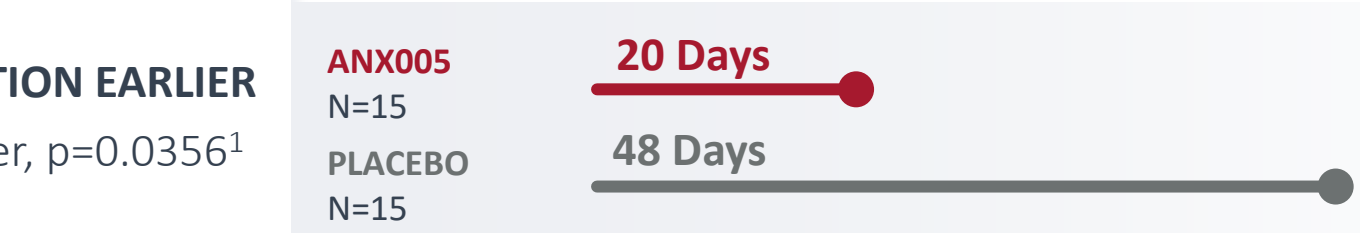
WALKING EARLIER

31 days earlier, p=0.0211[1]



OFF VENTILATION EARLIER

28 days earlier, p=0.0356[1]



[1]Nominal

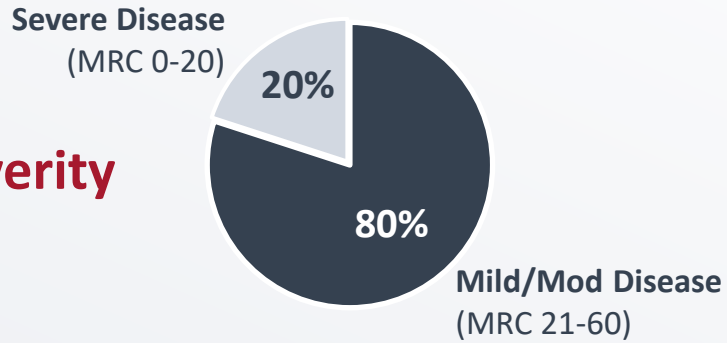
GBS Phase 3 Results are Highly Relevant to US and European Populations

Significant treatment effect in western-world type patients

US and European GBS Demographics

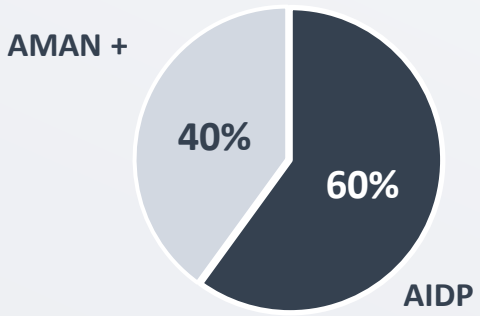
80% have baseline **MRC of >20**

Disease Severity



60% have **AIDP**

Neurotype



IGOS Database

ANX005 Phase 3 Study Results

52% with MRC >20

GBS-DS	**3.03x more likely to be better** vs. pbo at week 8, **p=0.0102**[1]
MRC	**8.8-point improvement** vs. pbo at week 1, **p<0.0001**[1]

21% with AIDP

GBS-DS	**5.31x more likely to be better** vs. pbo at week 8, **p=0.0130**[1]
MRC	**11.9-point improvement** vs. pbo at week 1, p=0.0002[1]

[1]Nominal

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ANX005 Generally Safe and Well-Tolerated

Majority of AEs were mild (Grade 1) to moderate (Grade 2)

- Most common related events were infusion related reactions
  - Majority were mild transient rashes
- No autoimmune related adverse events reported
- No increased infection rate while not requiring vaccination or prophylactic antibiotics
- One discontinuation in each dose group
- SAEs and Grade 3 AEs balanced across groups, characteristic of disease morbidity

Deaths

- No difference observed in incidence of all-cause mortality - 3 deaths in each dose group
- Mortality rate of 3.7% consistent with rates seen in US and EU
- Deaths occurred in older & more severe subjects

	Placebo N=81	ANX005 30mg/kg N=79	ANX005 75mg/kg N=81
	All Grades	All Grades	All Grades
Number of Subjects Reporting TEAEs, n (%)	79 (97.5)	79 (100.0)	80 (98.8)
Number of Subjects with Infusion Related Reaction	4 (4.9)	24 (30.4)	32 (39.5)
Rash (most common with IRR)	2 (2.5)	20 (25.3)	25 (30.9)
Most Common TEAEs (non-IRR), n (%)			
Blood CPK Increased	46 (56.8)	44 (55.7)	35 (43.2)
Musculoskeletal Pain	35 (43.2)	36 (45.6)	26 (32.1)
ALT Increased	23 (28.4)	21 (26.6)	23 (28.4)
Urinary Tract Infection	18 (22.2)	19 (24.1)	18 (22.2)
Hypokalemia	24 (29.6)	16 (20.3)	11 (13.6)
Constipation	10 (12.3)	15 (19.0)	17 (21.0)
AST Increased	16 (19.8)	11(13.9)	17 (21.0)

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Real-World Evidence to Support Planned Regulatory Submission

Interim RWE Data Support Comparability & Relevance of Phase 3 Findings to the West

- **FDA agreed that a single pivotal study would be sufficient for BLA assuming it demonstrates:**
  - Substantial evidence of ANX005's treatment effect vs. placebo
  - Comparability between Ph3 population & Western patients
- **Annexon has developed a real-world evidence (RWE) comparability protocol with IGOS (ANX005-GBS-04)**
- **IGOS data supports ongoing comparability study, including:**
  - >50% of all Western IGOS patients met the entry criteria for GBS Ph3
  - Robust ANX005 impact on 'Western World' type Phase 3 patients
  - Preparing matched cohort for comparison with IVIg

Annexon + IGOS RWE Comparability Study

IGOS

Global GBS Real-World Evidence Cohort

↕

Annexon Phase 3 Study

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GBS is an Untapped Commercial Opportunity and Annexon is Pursuing a Tailored Approach

Significant commercial opportunity for ANX005 achieved through focused commercial footprint

22,000 people in US & Europe **hospitalized** with GBS **every year**

90% of GBS patients **treated** with **off-label IVIg in US**

- **Daily infusions** over **5 days**
- **Non-specific mechanistic approach** to treating GBS

>$2B annual cost burden on patients, caregivers, hospitals, and payers[1]

Majority of patients treated in **major metro areas and large community hospitals**[2]

ANX005
First-line, monotherapy treatment for GBS

ANX005 helped **GBS patients Get Better Sooner**

- ✓ **Single infusion, targeted mechanism**
- ✓ **Faster recovery / independence**
- ✓ **Potential for significant cost reductions for health care system**

Robust HEOR plan to demonstrate **reduced cost of care**

Focused and targeted commercial launch plan

Commercial manufacturing partnership with Lonza

GBS a beachhead for **mechanistically-related neuro and autoimmune** indications

[1]Frenzen, PD (2008) Neurology 71:21-27 7, [2]ClearView Health market research

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ANX007: Phase 3-ready Complement Therapy for Geographic Atrophy

First Global Pivotal Program for GA Using Vision Preservation as Primary Outcome Measure

Nancy S.
wife and caregiver

Paul S.
85-year-old patient with GA

Global Opportunity for New GA Treatments that Preserve Vision

Chronic, progressive neurodegenerative disease of the eye resulting in vision loss

Paul S., 85-year-old patient with GA

"I look normal. My eyes look normal. **But what I see through my eyes is not what you see through your eyes.** It's cloudy, it's hazy, it's fuzzy. It's not clear, it's not crisp…I don't drive anymore. It really impacts my photography hobby. **Nothing is like it used to be.** "

Nancy S., wife and caregiver

"**He isn't able to function in the way he once did. Eye problems can take a toll not just on your sight, but emotionally too**. …When we are walking somewhere I get very tense. I try to tell him if the ground changes, but then it can start to get demeaning if I'm telling him things all the time. **I walk on eggshells**."

1 MILLION people diagnosed in US; **8 MILLION** people globally[1]

ZERO

FDA-approved treatments demonstrating **preservation of visual function**

Treatments approved in the EU or Asia

SIGNIFICANT DISEASE BURDEN

PROGRESSIVE DISEASE
leading to vision loss

2.5 YEARS
median time to developing central GA from diagnosis[2]

TRAUMATIC IMPACT ON PERSONAL LIVES AND DAILY LIVING, including limited or no ability to read, drive, or recognizing faces

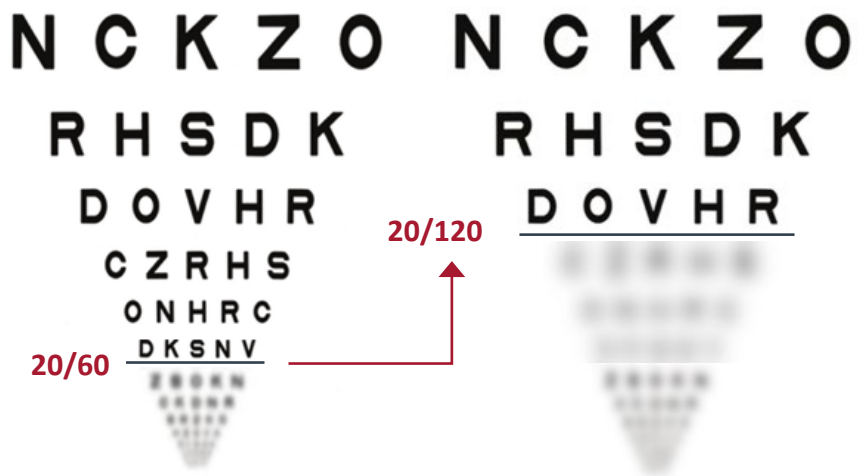
[1] https://eyewiki.aao.org/Geographic_Atrophy. [2] Arch Ophthalmol. 2009 Sep; 127(9): 1168–1174.

Vision Preservation is the Most Important Outcome for GA Patients

BCVA ≥15-letter loss is rigorous and meaningful as it represents 50% loss of a patient's central visual acuity

BEST CORRECTED VISUAL ACUITY
(BCVA)

15 Letter Loss
20/60 to 20/120 vision



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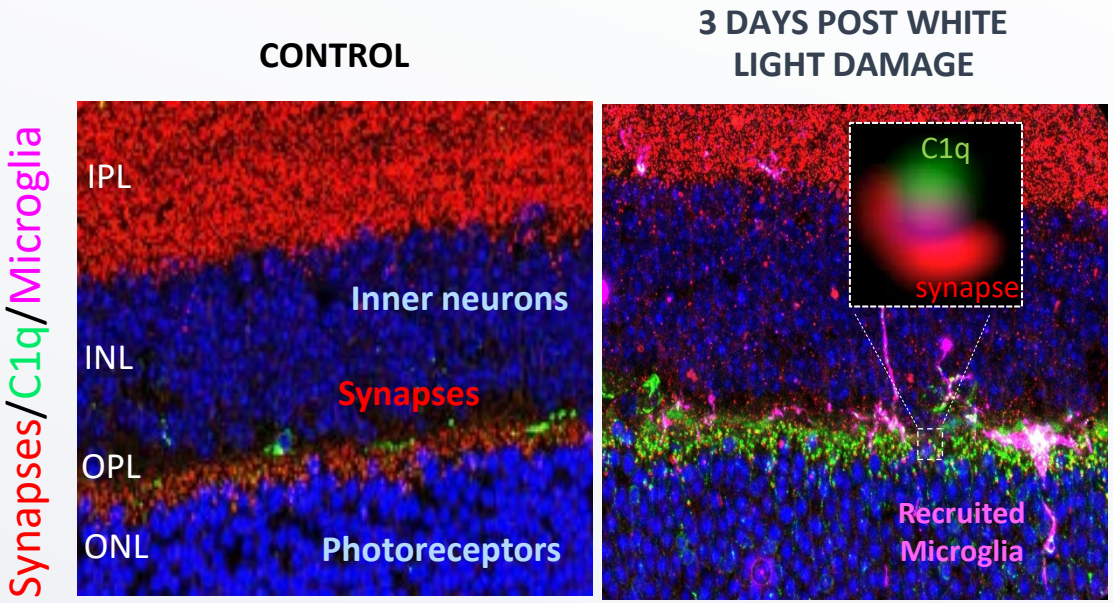
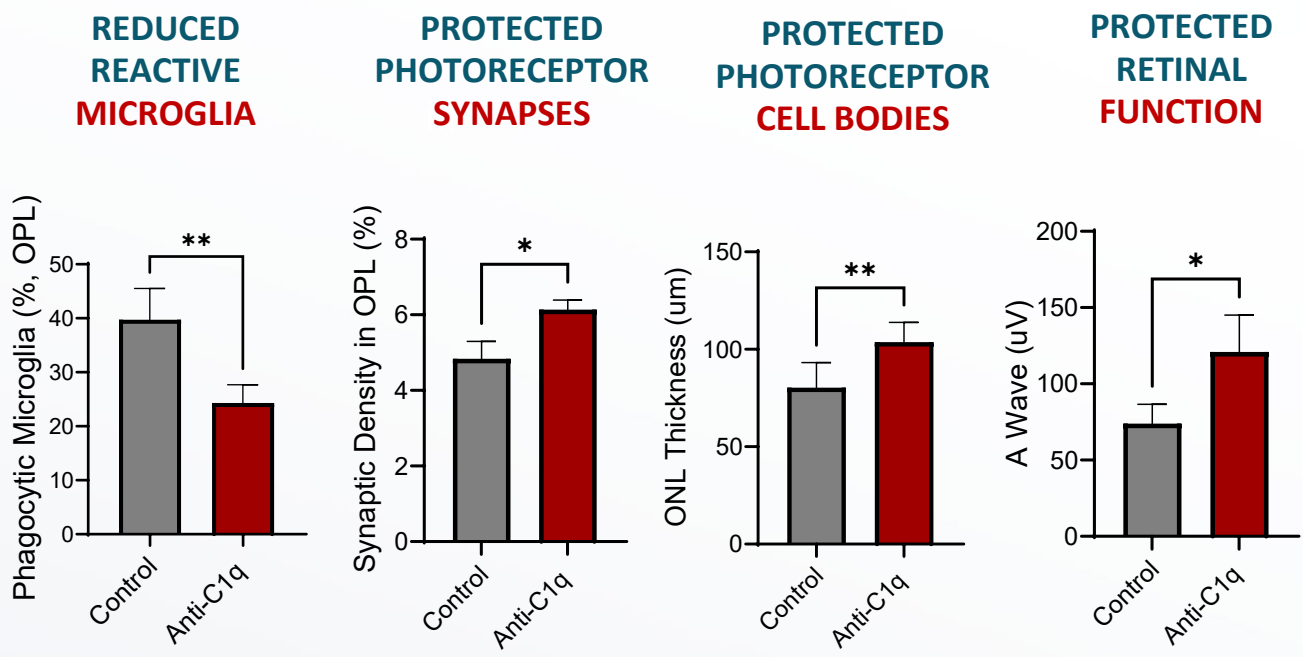
Anti-C1q Protected Photoreceptor Cells and Their Function in Models of Photoreceptor Damage

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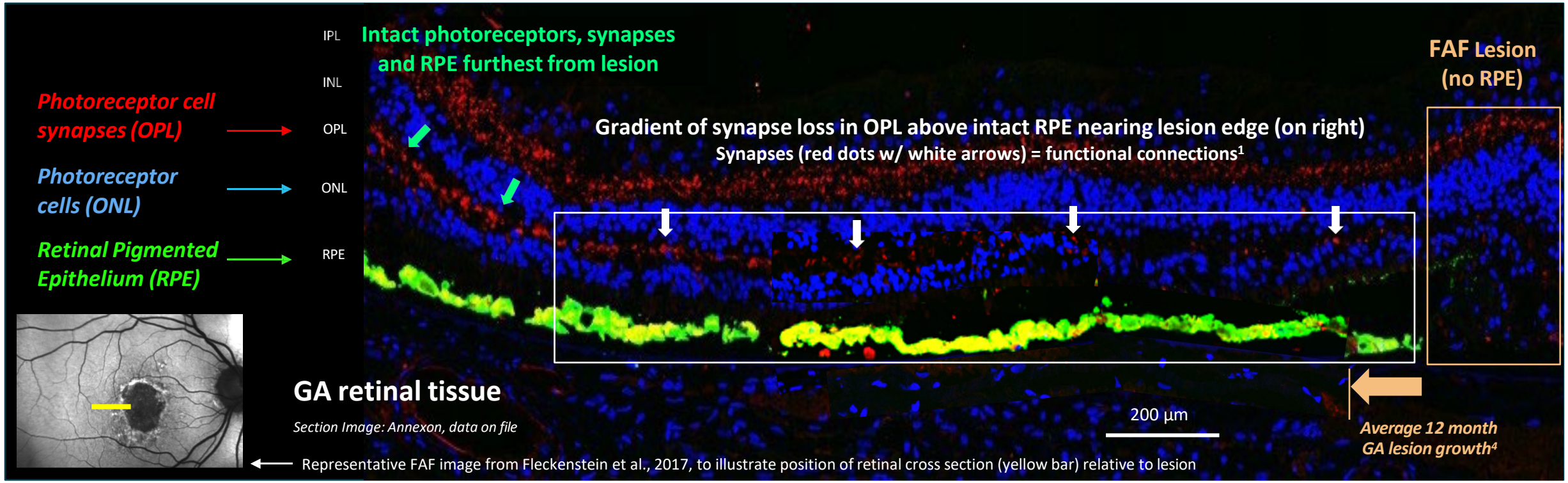
Photoreceptor Cells, Synapses & Function Lost Prior to RPE in GA

- 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

ANNEXON biosciences

Overview of ANX007 Geographic Atrophy Program

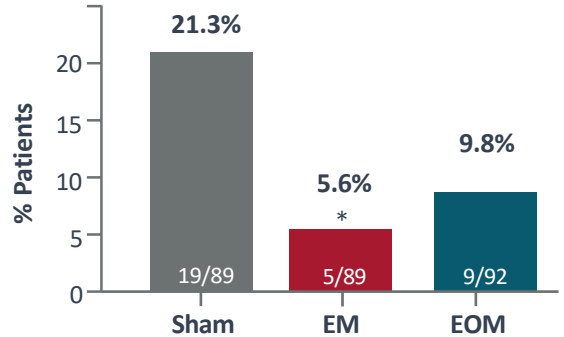
Structure-confirmed vision benefit in Phase 2 ARCHER study; Phase 3 ARCHER II ongoing

- ✓ 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**

ANX007 ARCHER Proof-of-Concept Trial – 1st Significant Demonstration of Vision Preservation in GA Patients

SIGNIFICANT VISION PROTECTION MEASURED BY BCVA ≥15-LETTER LOSS

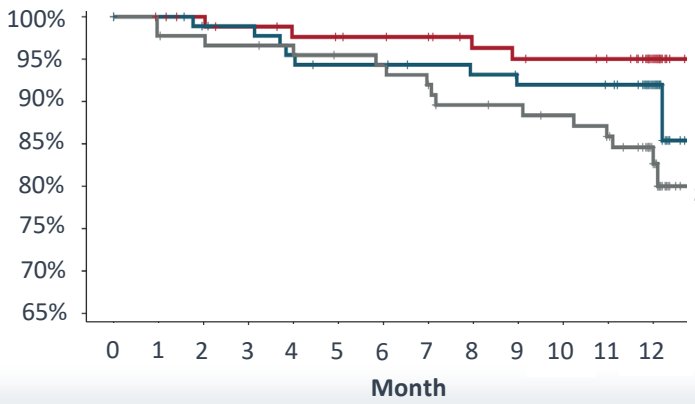
Patients with persistent BCVA ≥15-letter loss through month 12⁺



Nominal p-value vs sham^	---	0.0021	0.032

SIGNIFICANT TIME AND DOSE-DEPENDENT VISION PROTECTION

BCVA ≥15-letter loss at 2 consecutive visits

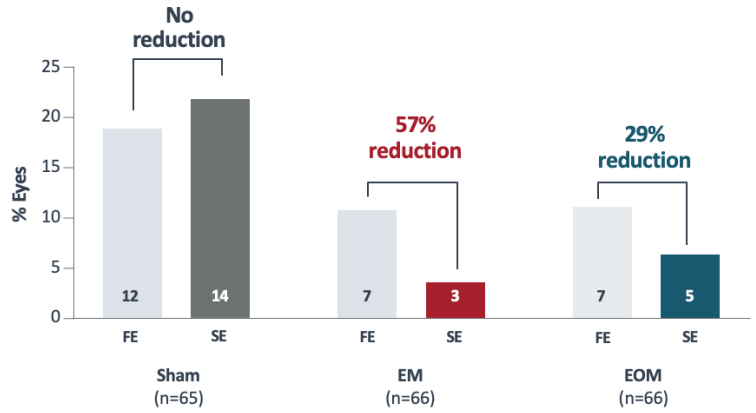


ANX007 EM 73% Risk Reduction
HR (CI) = 0.272 (0.090 to 0.819)
p = 0.0207

ANX007 EOM 50% Risk Reduction
HR (CI) = 0.504 (0.214 to 1.190)
p = 0.1181

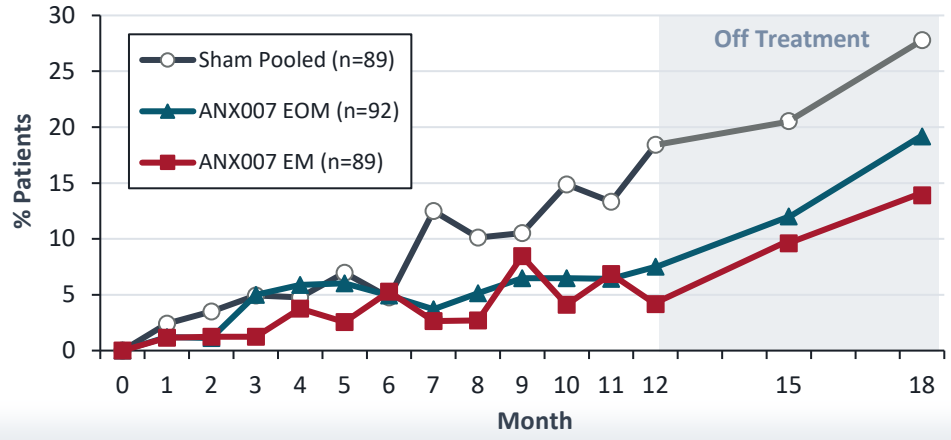
FELLOW-EYE ANALYSIS: VISION PROTECTION IN STUDY EYE (SE) BUT NOT IN NON-TREATED FELLOW EYE (FE)

Eyes with BCVA ≥15-letter loss at month 12 in all patients with bilateral GA



OFF TREATMENT ANALYSIS: ON-TREATMENT VISION PROTECTION WANES POST-TREATMENT

% of patients with any BCVA ≥15-letter loss from baseline



⁺Persistent for two consecutive visits including month 12
^Nominal p-value from a Chi-square test in ITT population

HR, hazard ratio; Nominal log-rank test (versus sham) p-values are presented
EM: monthly dosing; EOM: every other month dosing

Annexon ARCHER Phase 2 data presented American Society of Retina Specialists and American Academy of Ophthalmology 2023.

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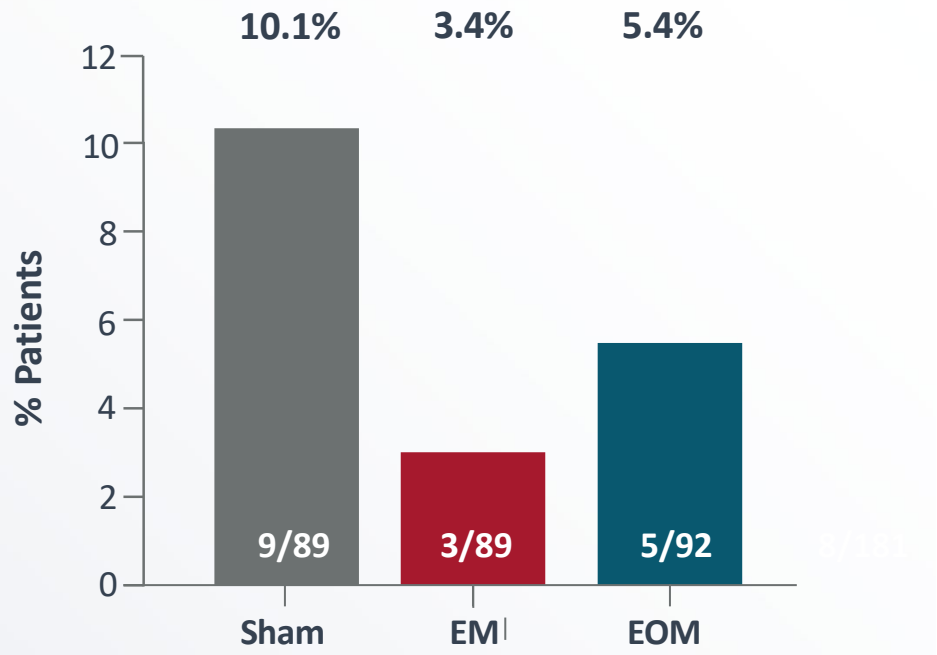
ANX007 Effect on BCVA ≥15-Letter Loss Improves with Longer Treatment

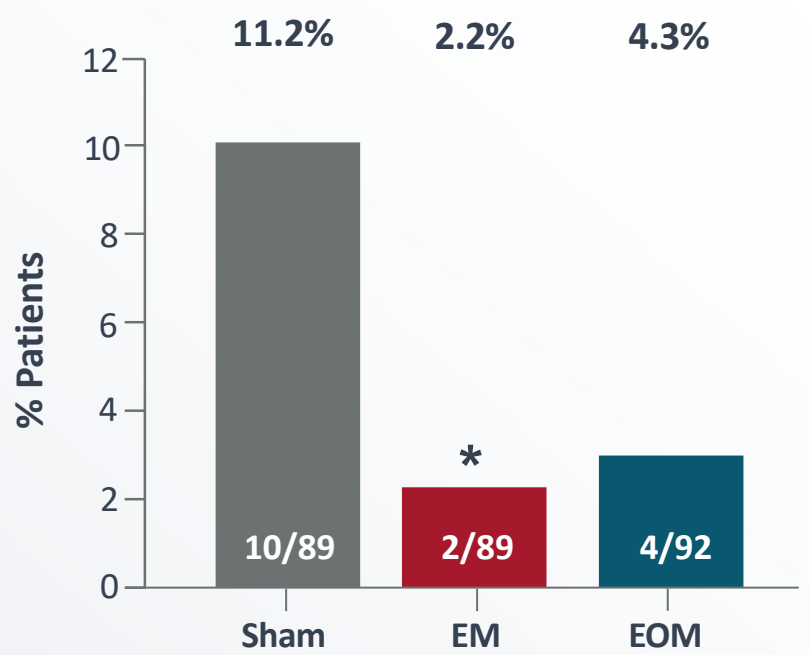
PATIENTS WITH PERSISTENT BCVA ≥15-LETTER LOSS

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

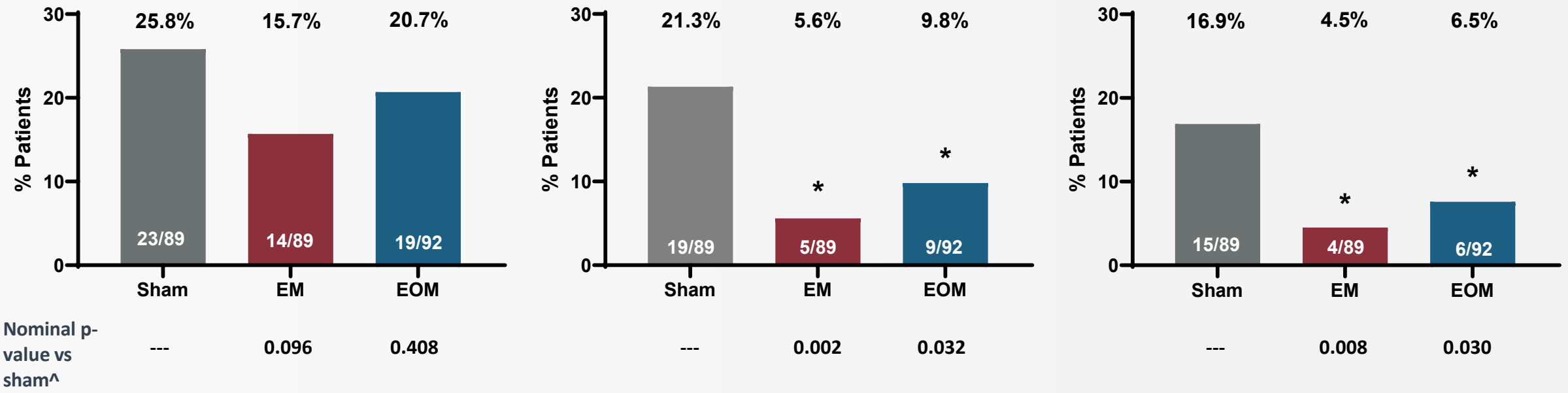
⁺Persistent for two consecutive visits through month 12 or at last visit; ^Nominal p-value from a Chi-square test in ITT population; Nominal P < 0.05*

Increasing ANX007 Impact Over Time

ANNEXON biosciences

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

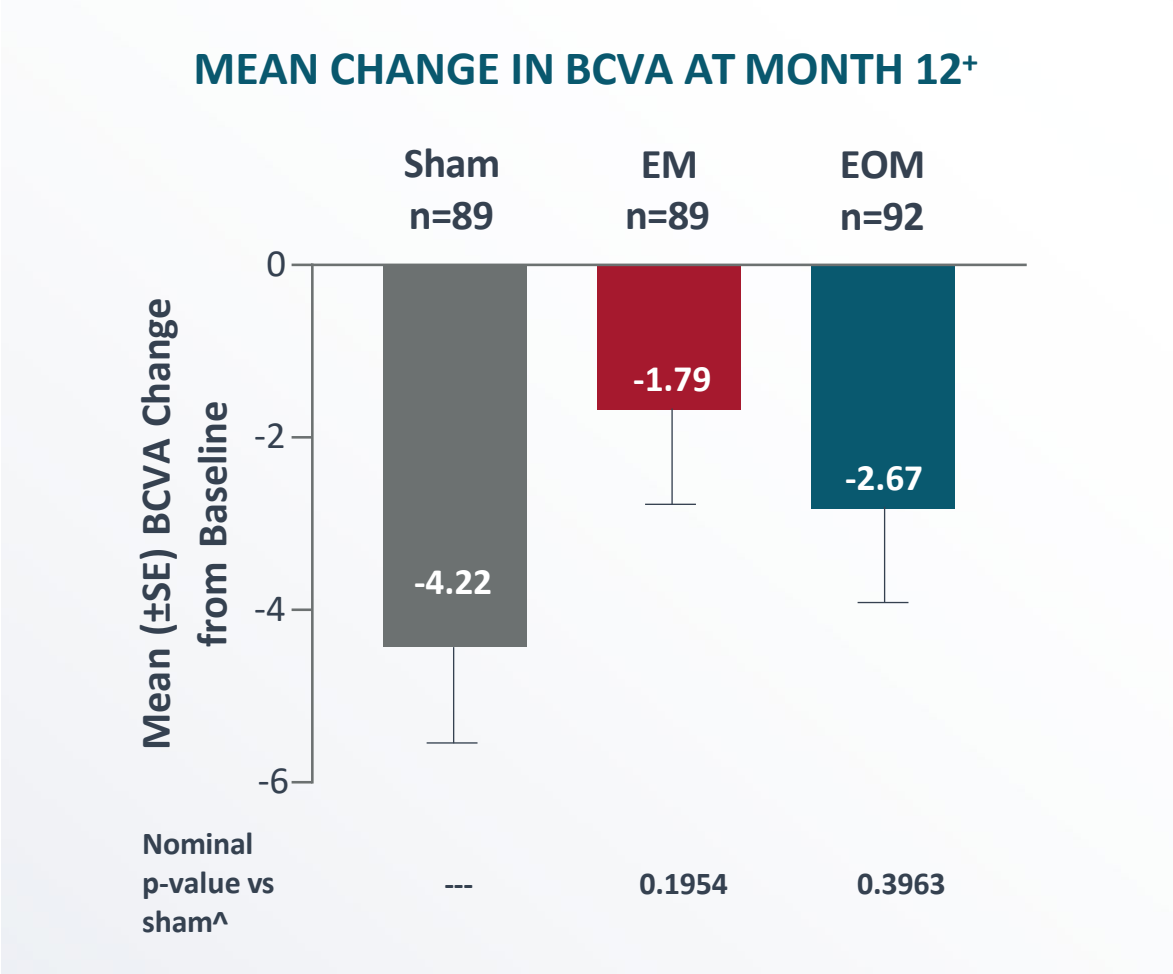
Persistent BCVA Vision Loss Through Month 12#



#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

ANNEXON biosciences

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]

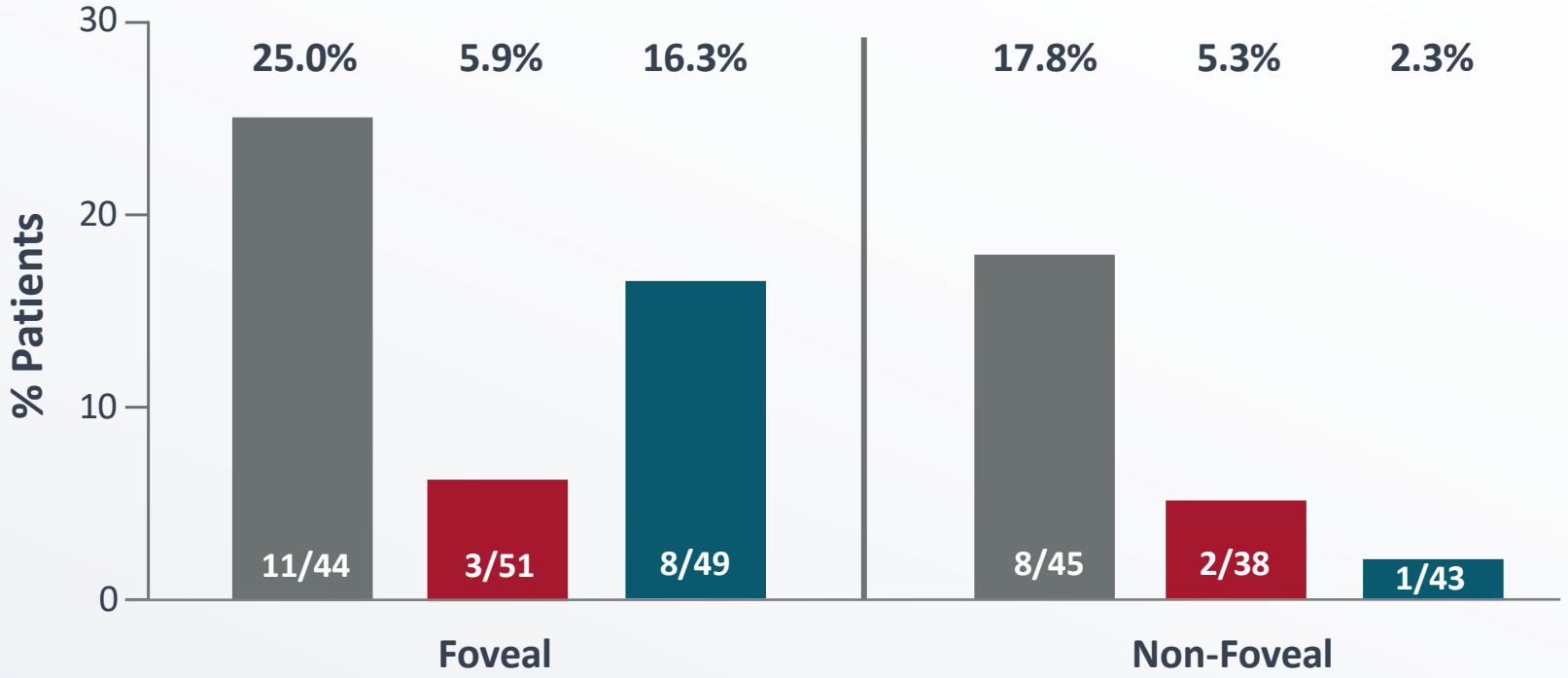
+Mean, standard error (SE), and p-value based on MMRM adjusting for baseline lesion location, lesion focality, baseline GA lesion, and the baseline GA lesion by visit interaction.
^Nominal p-value from MMRM adjusting for baseline lesion location, lesion focality, baseline GA lesion, and the baseline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

ANNEXON biosciences

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#

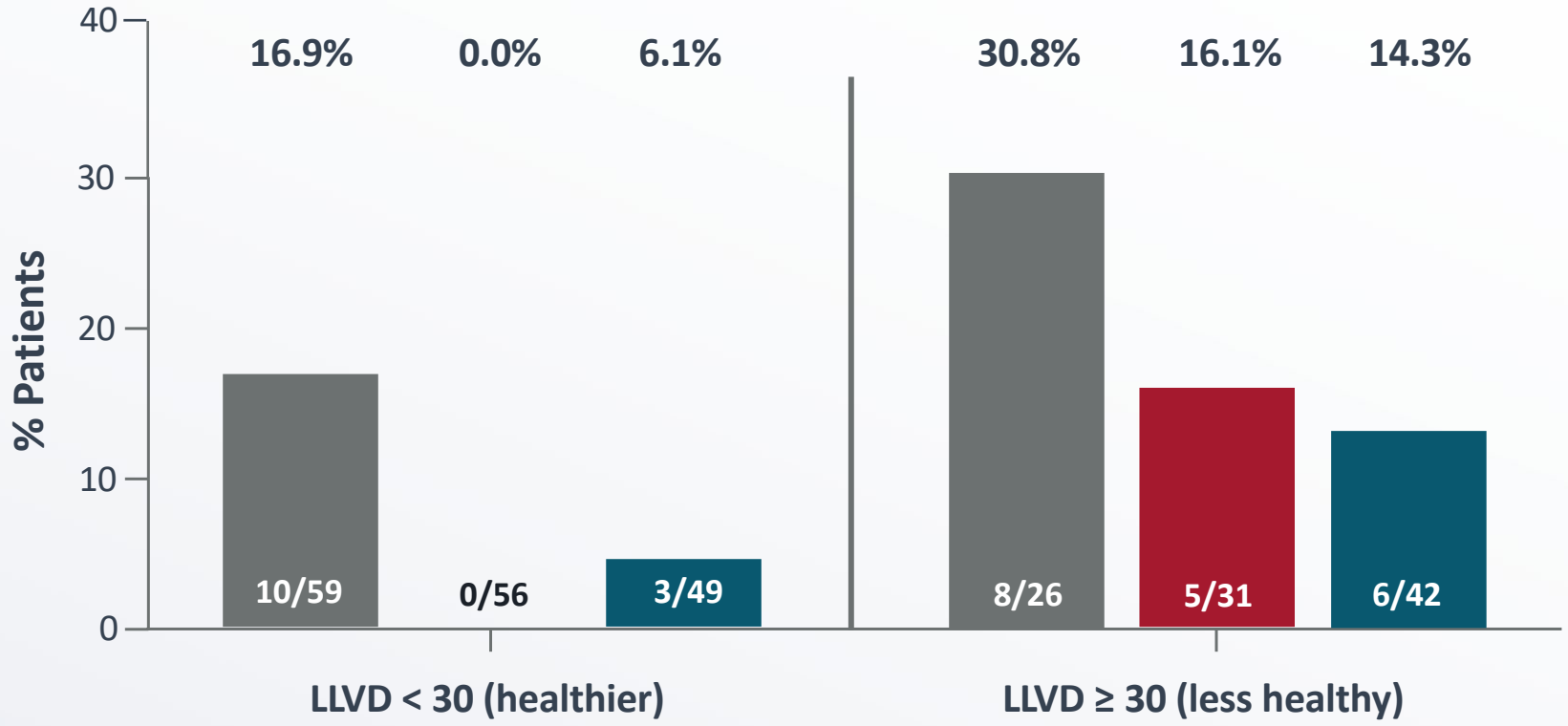


Nominal summary p-value vs. sham^	
ANX007 EM	0.0019
ANX007 EOM	0.0254

#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



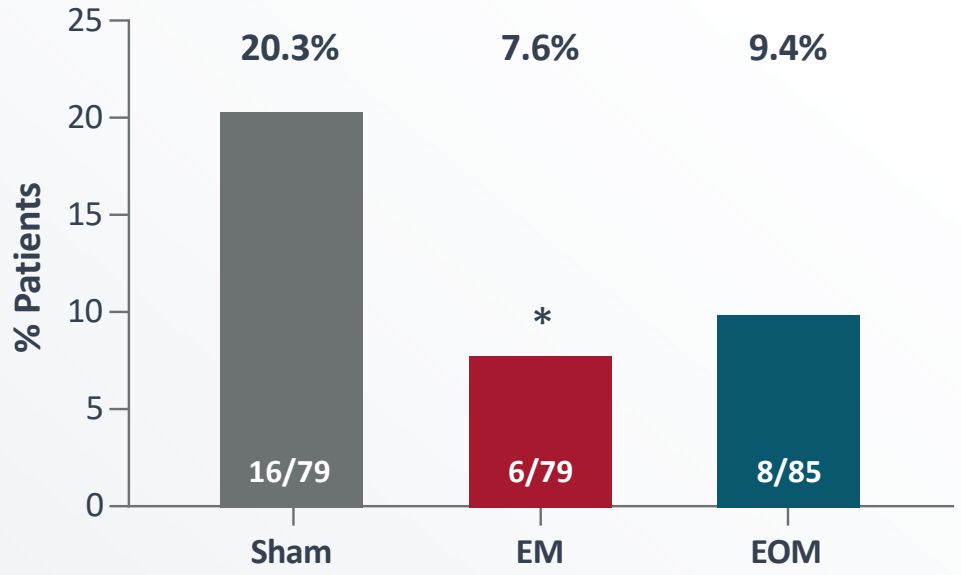
Nominal summary p-value vs. sham^	
ANX007 EM	0.0016
ANX007 EOM	0.0183

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

ANNEXON biosciences

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

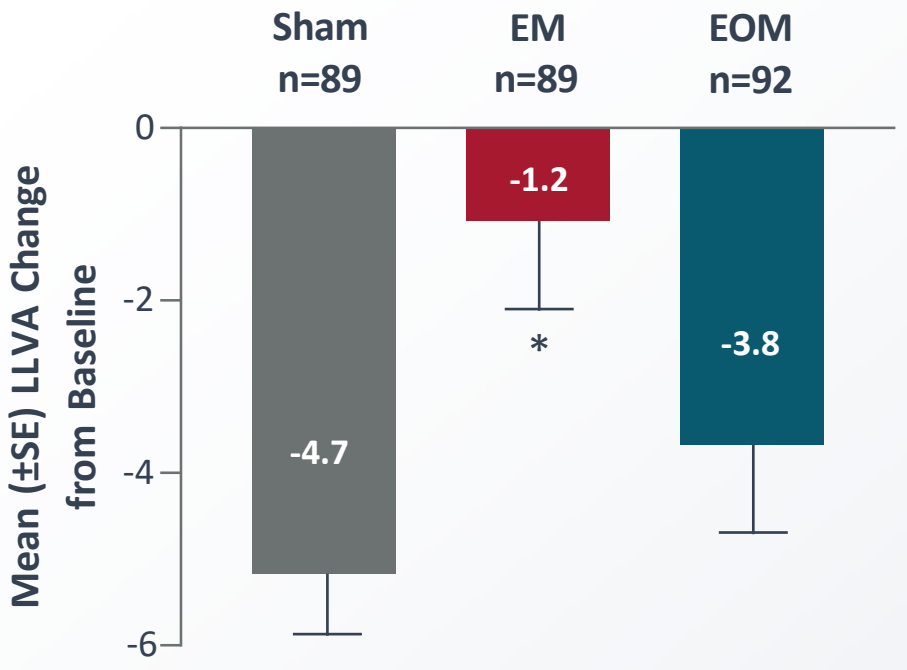
LLVA ≥15-LETTER LOSS THROUGH MONTH 12#



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

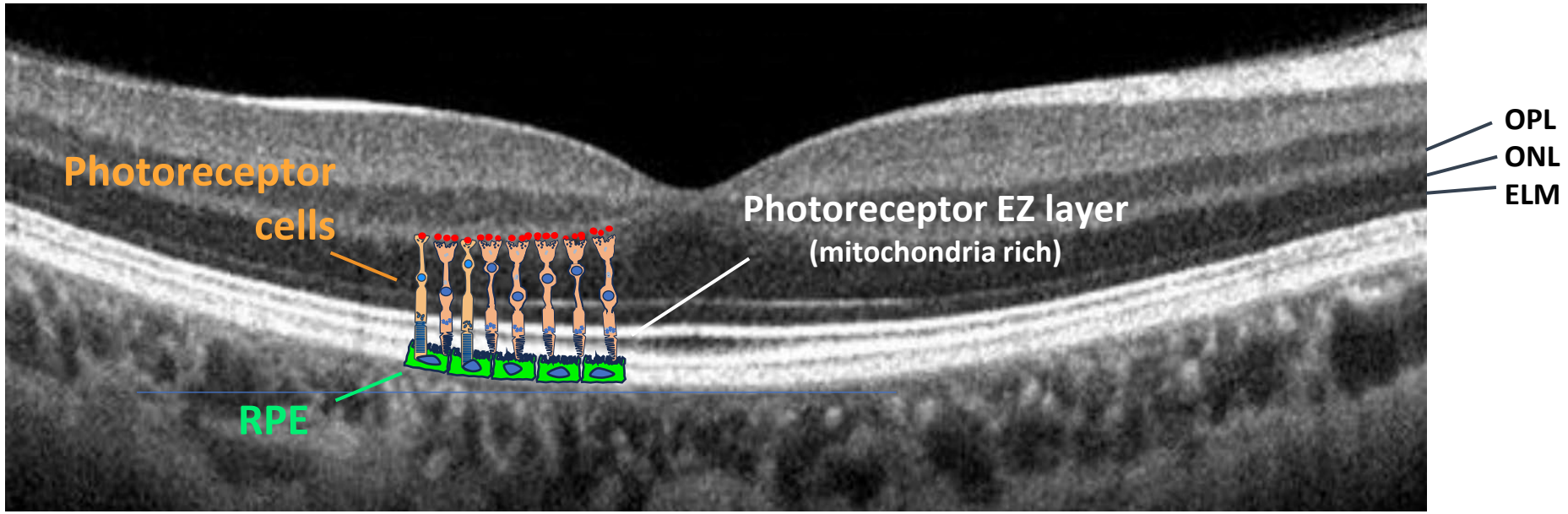
MEAN CHANGE IN LLVA AT MONTH 12+



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

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



octscans.com

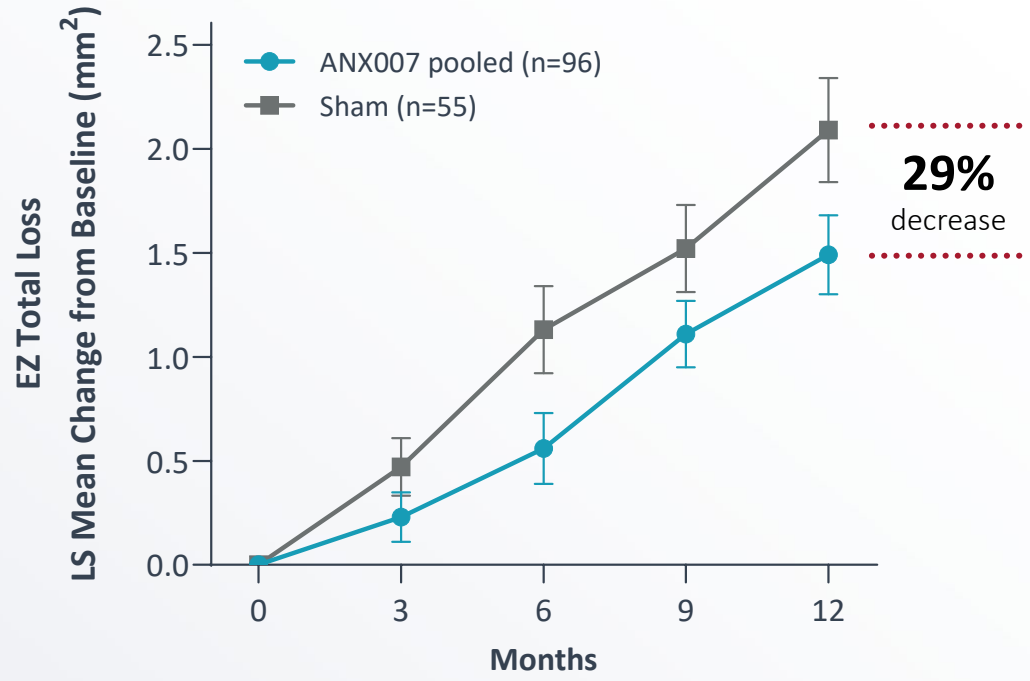
ARCHER EZ Population

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

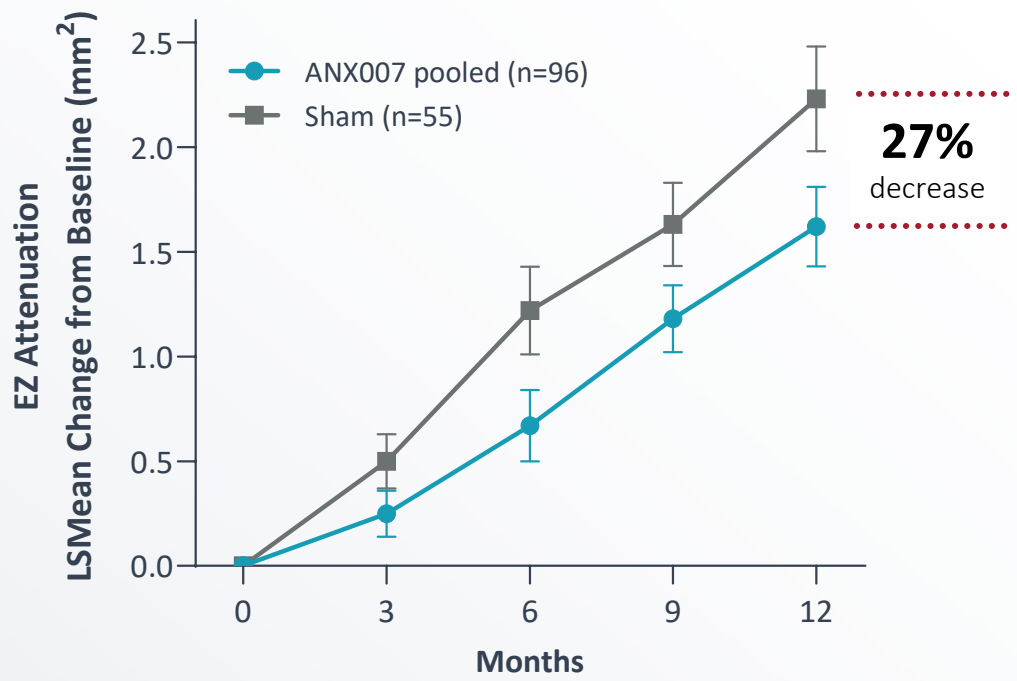
ANX007 Significantly Protected Photoreceptors Across Retina Through 12 Months

EZ TOTAL LOSS (EZ = 0 μm)*



Nominal p-value vs sham^ ANX007 0.017

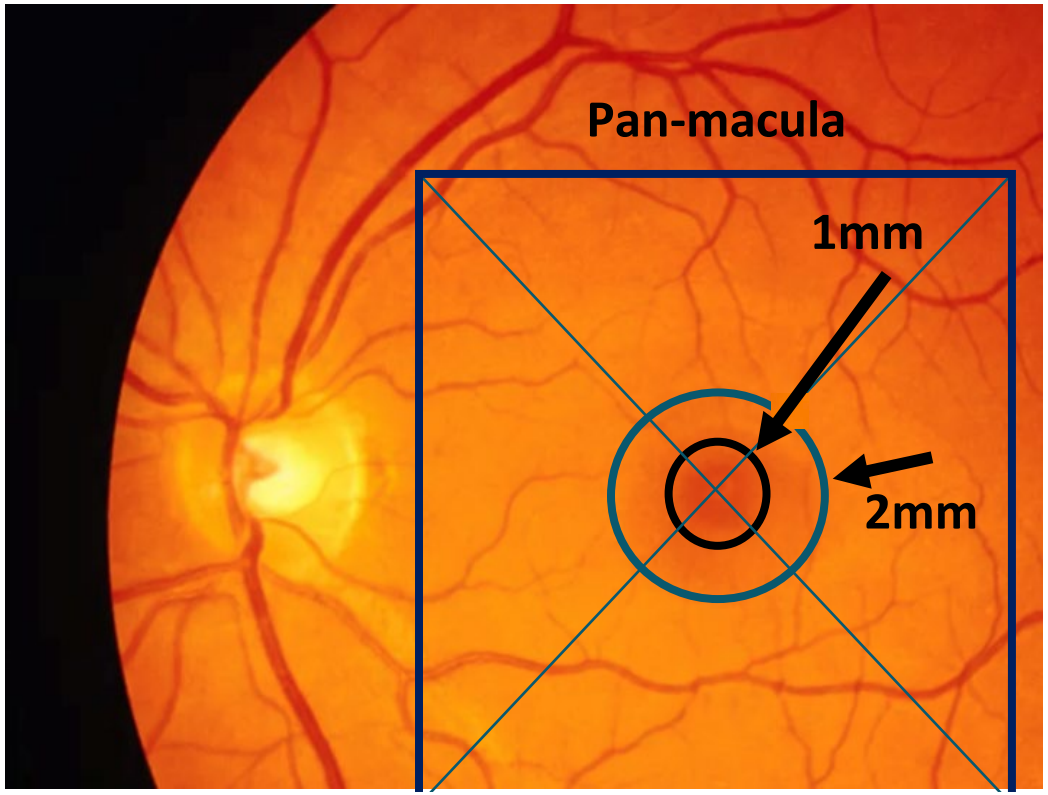
EZ ATTENUATION (EZ < 20 μm)*



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)

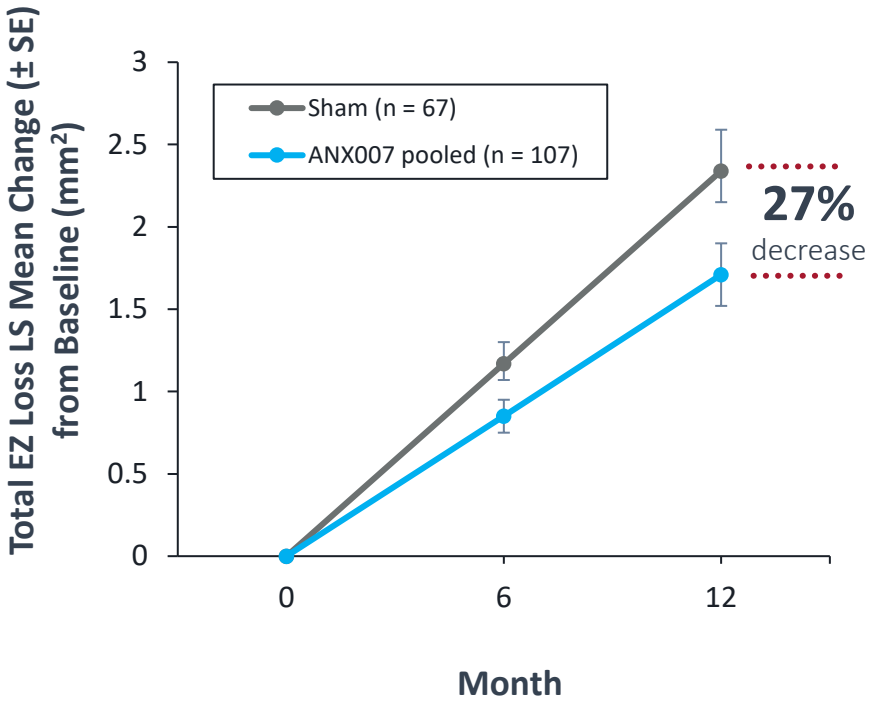
*p≤0.05

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

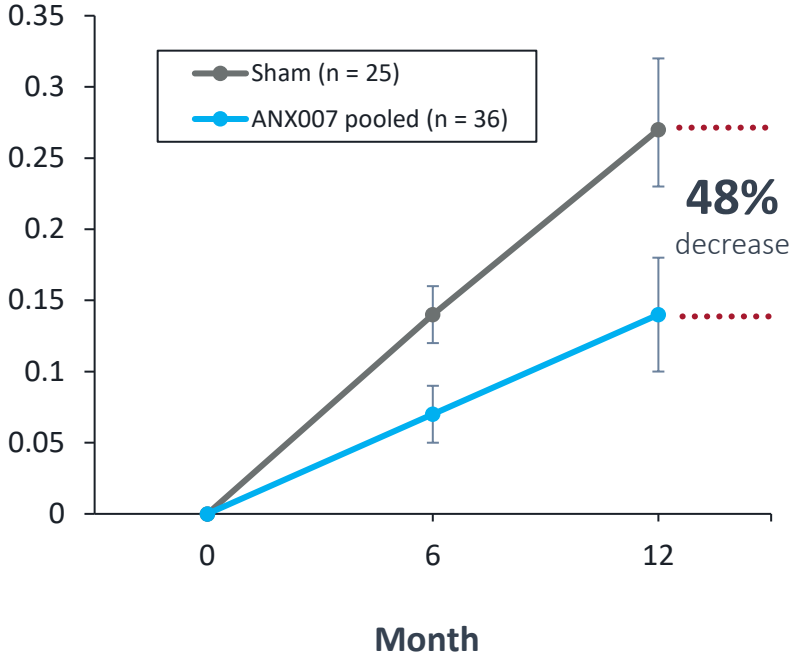
Photoreceptor Protection Through 12 Months in Central Fovea

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

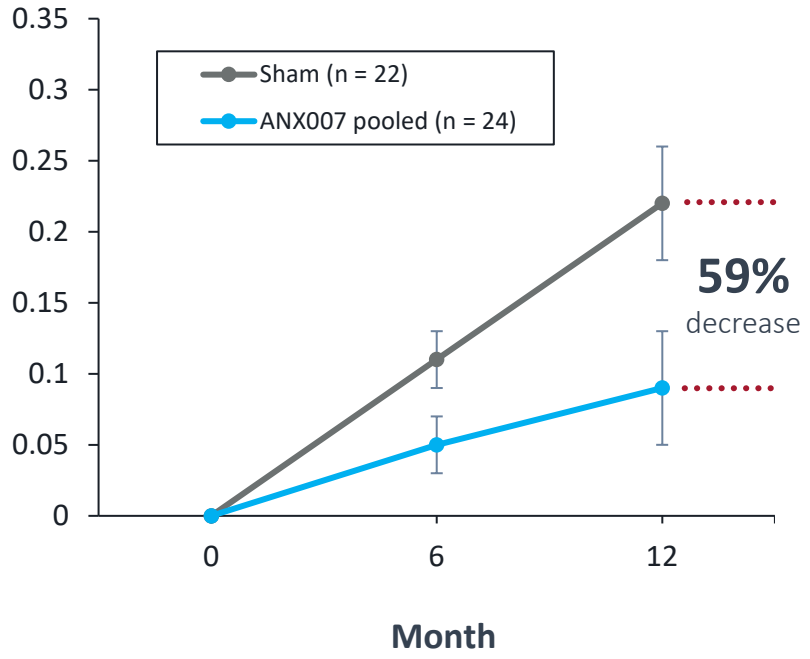
PAN-MACULA



CENTRAL 2.0 MM



CENTRAL 1.5 MM



Nominal p-value^

ANX007 Pooled vs Sham 0.0457

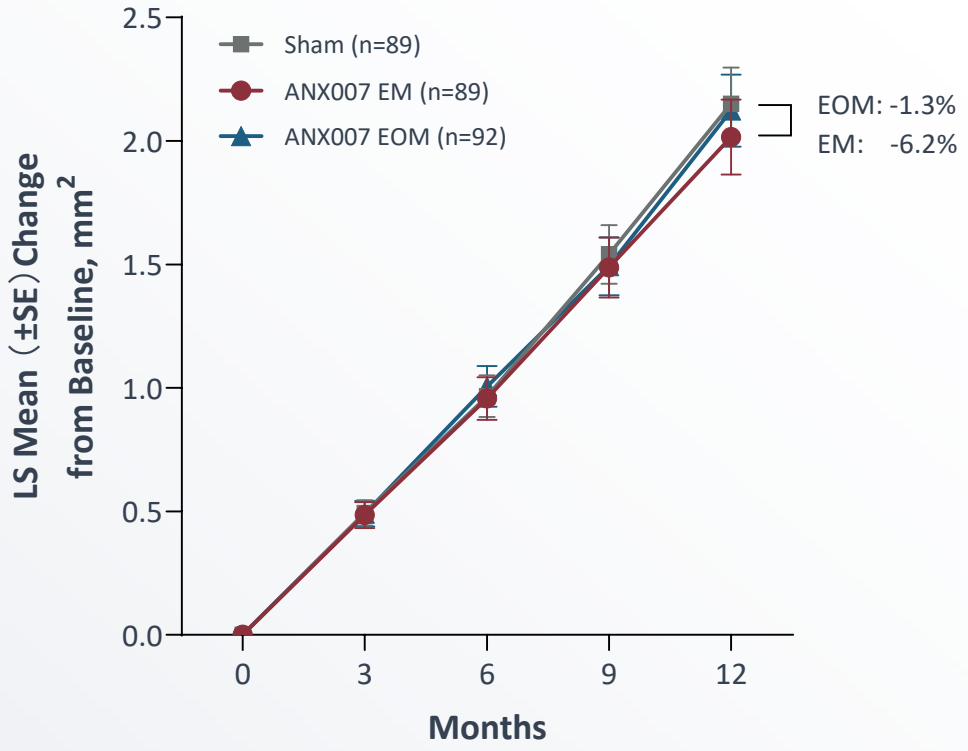
ANX007 Pooled vs Sham 0.0218

ANX007 Pooled vs Sham 0.0319

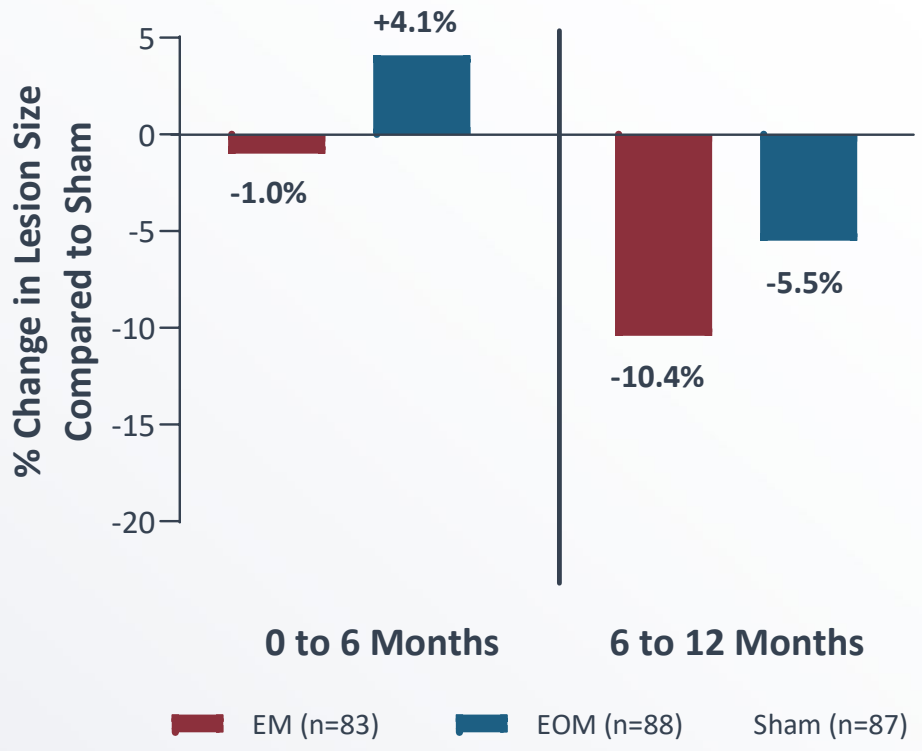
^Nominal p-values from a linear mixed model for repeated measures model (slope) analysis;
Heidelberg Spectralis OCT population with baseline OCT data, excludes patients with >98% atrophy/attenuation at baseline

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#

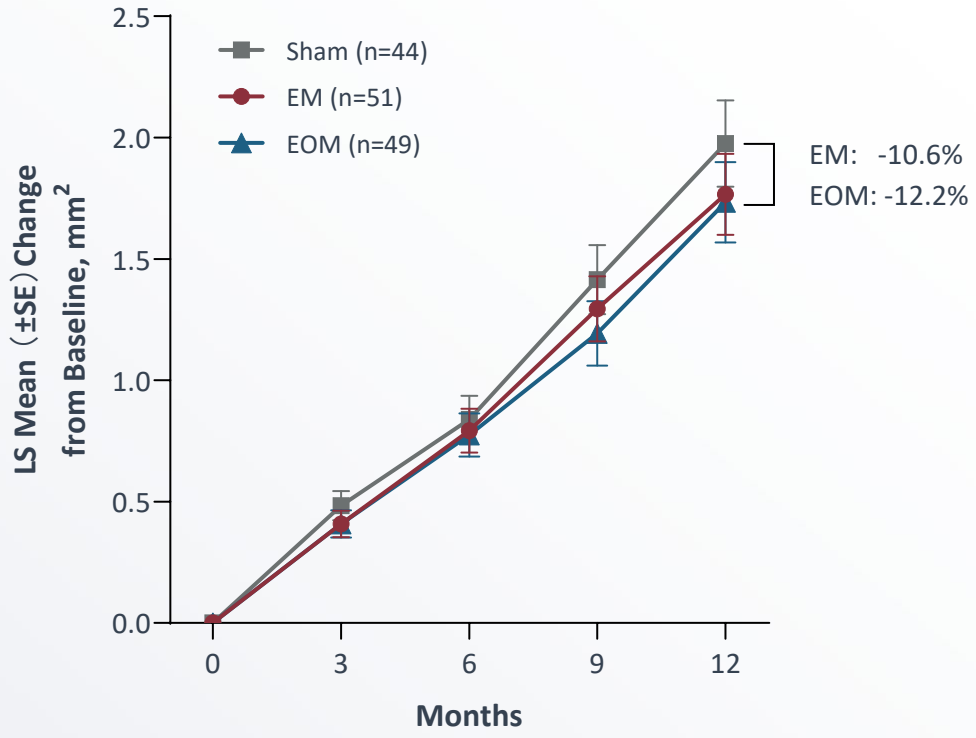


#Least-square (LS) mean and its standard error (SE) are based on a mixed-effect model for repeated measures (MMRM) adjusting for baseline lesion location, lesion focality, baseline GA lesion, and the baseline GA lesion by visit interaction.

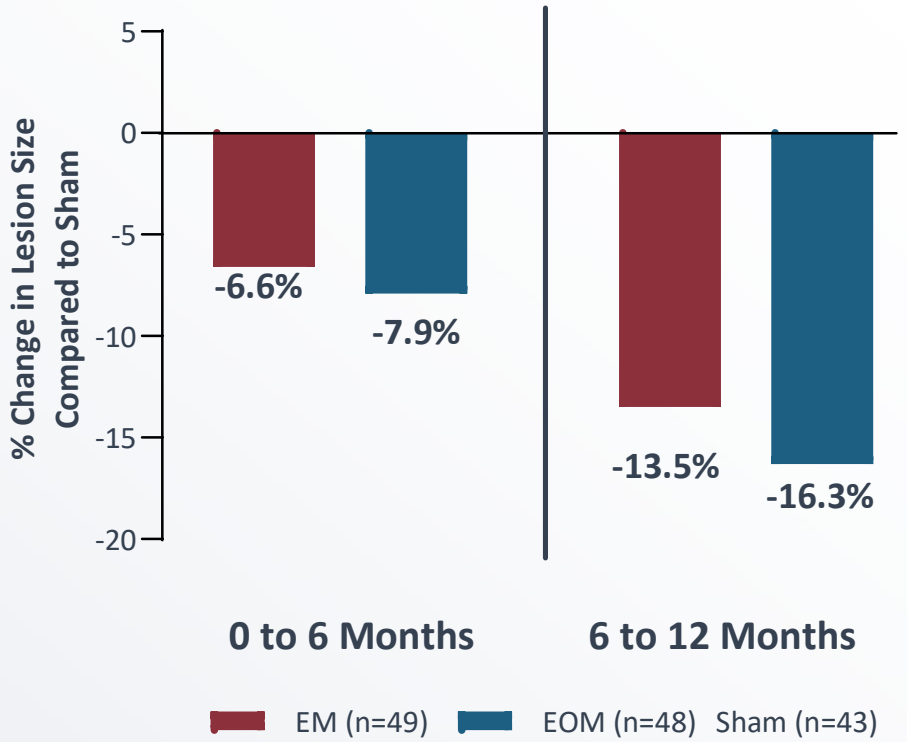
Stronger Impact on RPE Loss in Patients with Foveal Involvement at Baseline – Suggesting Differential ANX007 Effect in Foveal Center

Greater protection of RPE in region responsible for visual acuity

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



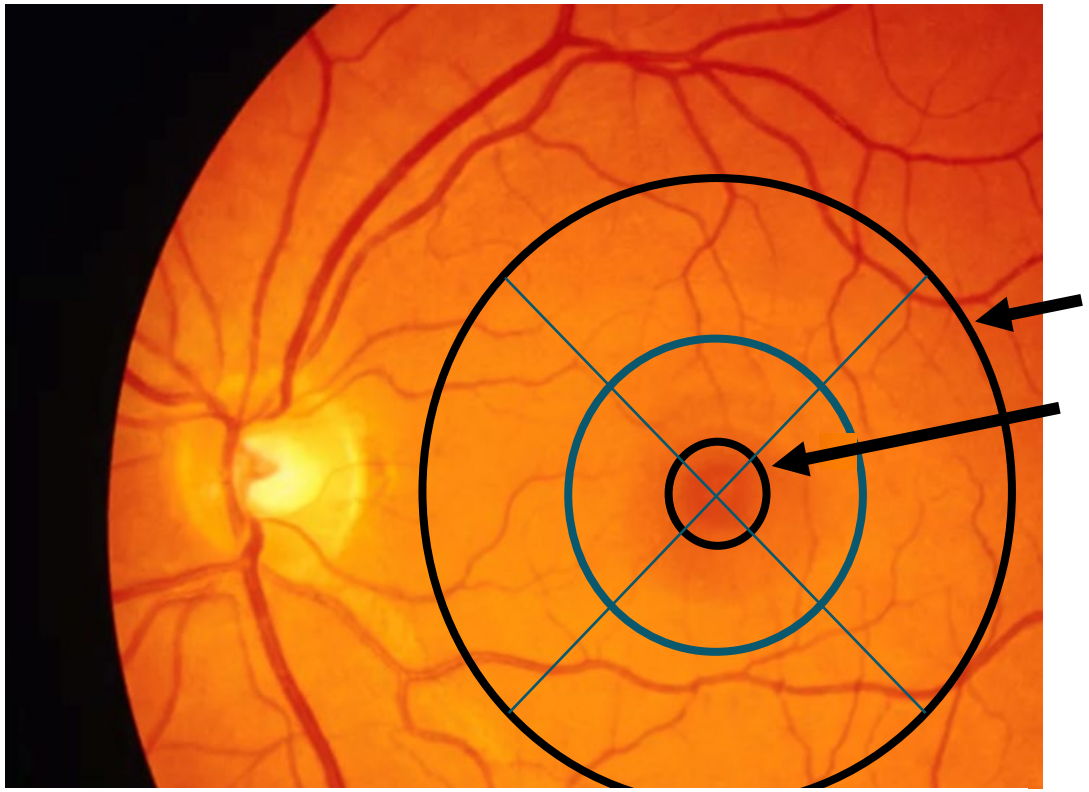
CHANGE IN RPE LOSS OVER TIME[#]
FOVEAL PATIENTS



[#]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

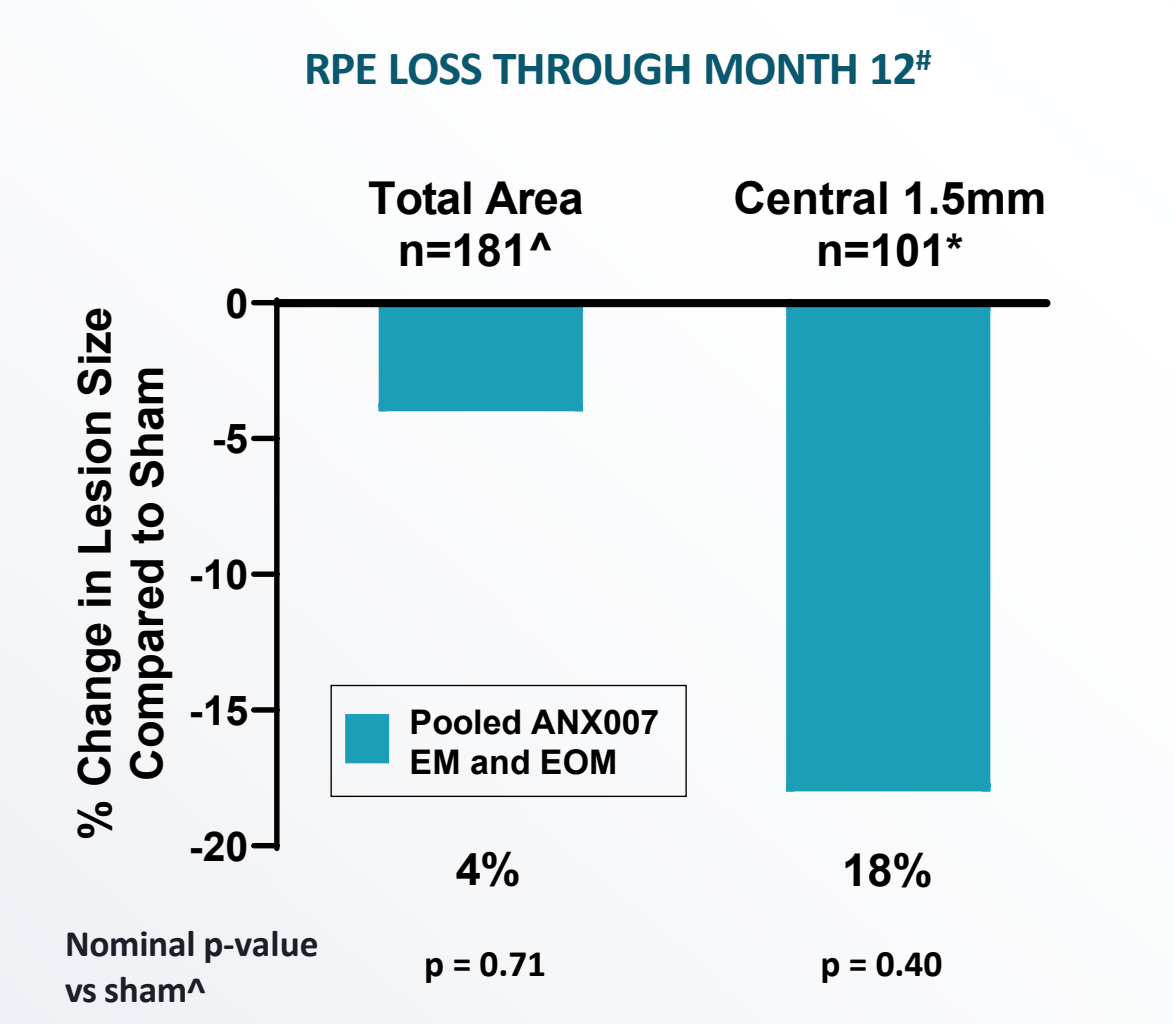
Location	Month 6	Month 12	Month 18
Full 6 mm diameter	p=0.59	p=0.15	**p=0.03**
1mm foveal center	**p=0.03**	**p=0.001**	**p<0.0001**

- 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

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

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**

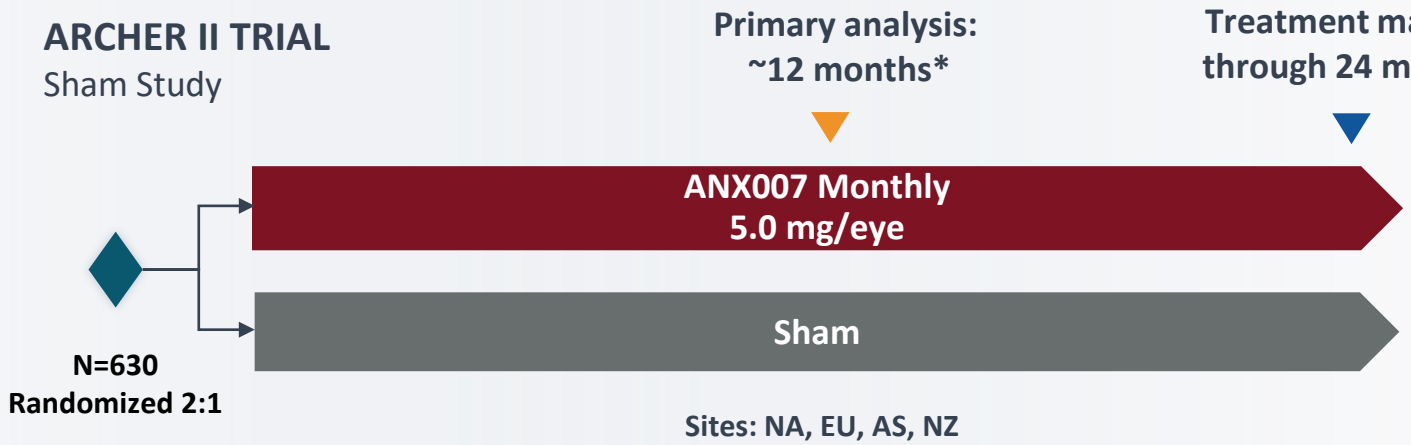
ANX007 treatment was **generally well-tolerated;** no CNV increase;
no reported cases of vasculitis

Regulatory-aligned Phase 3 program NOW ONGOING

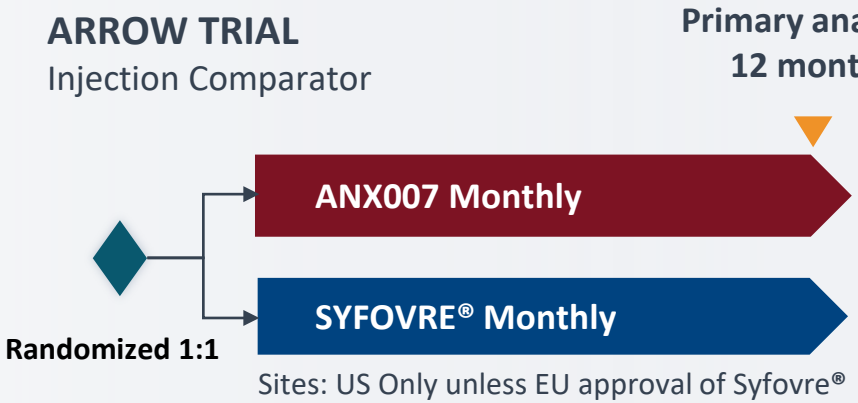
ANX007 Global GA Pivotal Program INITIATED

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

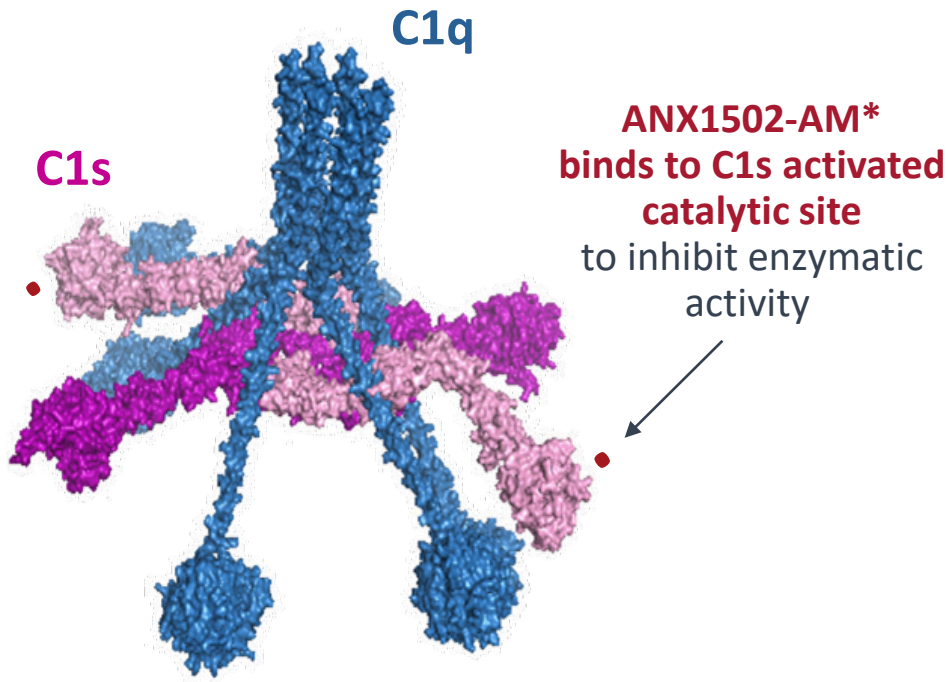
ANNEXON biosciences

ANX1502: First-in-Kind Oral Small Molecule Complement Therapy

Advancing for Complement-Mediated Autoimmune Diseases

Advancing ANX1502 as the First Oral, Small Molecule Inhibitor of Classical Complement Pathway in Development

- **Orally administered***
- **Targeting active form of C1s** responsible for transmitting classical pathway activation from C1q
- **Potent and selective inhibitor of C1s** (serine protease): selective over related proteases (200 – 50,000-fold)

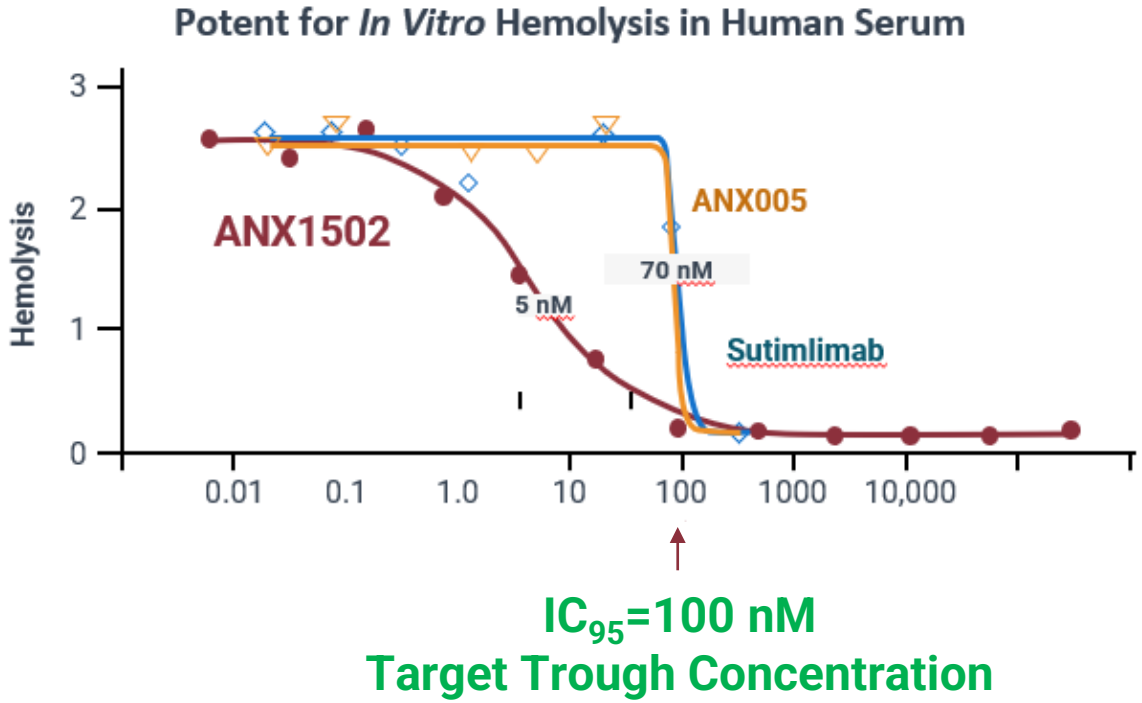


* ANX1502 prodrug administered; ANX1502-AM = Active Moiety

ANNEXON biosciences

Minimum Target Drug Level (100 nM) ANX1502-AM* for Robust Functional Inhibition of Classical Complement Pathway

- ANX1502-AM* demonstrated **robust functional inhibition of classical pathway** (IC_{50} = 5 nM)
  - Comparable to ANX005 and sutimlimab
  - *In vitro* hemolysis assay w/ high serum (30%)
- Normal sigmoidal dose response vs. antibodies likely due to rate-limiting concentrations of activated C1s
- **Minimum target drug levels for IC_{95}, desired at trough, set conservatively at 100 nM**



* ANX1502-AM: ANX1502 Active Moiety

ANNEXON biosciences

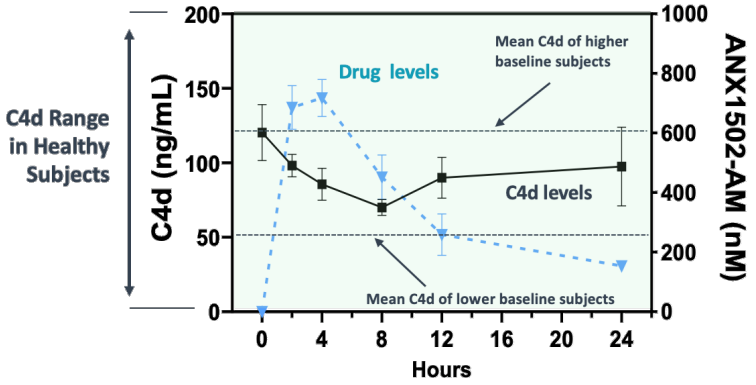
ANX1502 Ph 1 Program Well Tolerated and Achieved Dosing Objectives

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

SAFETY AND TOLERABILITY SHOWN WITH LIQUID SUSPENSION FORMULATION

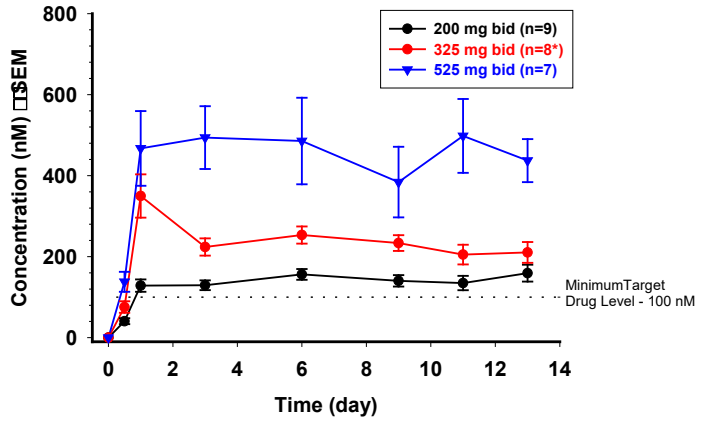
- All treatment-emergent adverse events (TEAEs) mild or moderate
- Most frequent TEAEs were GI related[1]
- **No serious adverse events (SAEs)**
- **No significant clinical/lab findings[2]**

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

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



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

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

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ANX1502 Suspension Formulation Generally Well-Tolerated Across SAD & MAD Cohorts in Healthy Volunteers

Manageable GI tolerability issues

Safety Results from Phase 1

- **ANX1502 generally safe and well tolerated through the highest dose level tested**
- All treatment-emergent adverse events (TEAEs) mild or moderate
- Most frequent TEAEs are gastro-intestinal and include nausea, emesis, and diarrhea
- **No serious adverse events (SAEs) observed**
- **No significant clinical/lab findings** (e.g., liver function enzymes, serum chemistry, hematology) observed

Subjects with TEAEs	SAD (Single Dose)						MAD (BID Dose)			
	25mg (N=6)	150mg (N=6)	450mg (N=6)	525mg (n=6)	1050mg (N=6)	**Placebo (N=10)**	200mg BID (N=9)	325mg BID (N=9)	525mg BID (N=9)	**Placebo BID (N=9)**
Subjects with any TEAE (%)	4 (66.6)	2 (33.3)	4 (66.6)	5 (83.3)	6 (100.0)	**6 (60.0)**	7 (77.7)	8 (88.9)	6 (66.6)	**7 (77.7.)**
Subjects with TEAE reported as related (%)	3 (50.0)	2 (33.3)	4 (66.6)	4 (66.6)	6 (100.0)	**4 (40.0)**	6 (66.6)	8 (88.9)	5 (55.5)	6 (66.6)
Subjects with any ≥ Grade 2 TEAE* (%)	1	0	0	0	0	**0**	0	2 (22.2)	1 (11.1)	**1 (12.5)**
Subjects with any Serious TEAE (%)	0	0	0	0	0	**0**	0	0	0	0

***No AEs higher than Grade 2**

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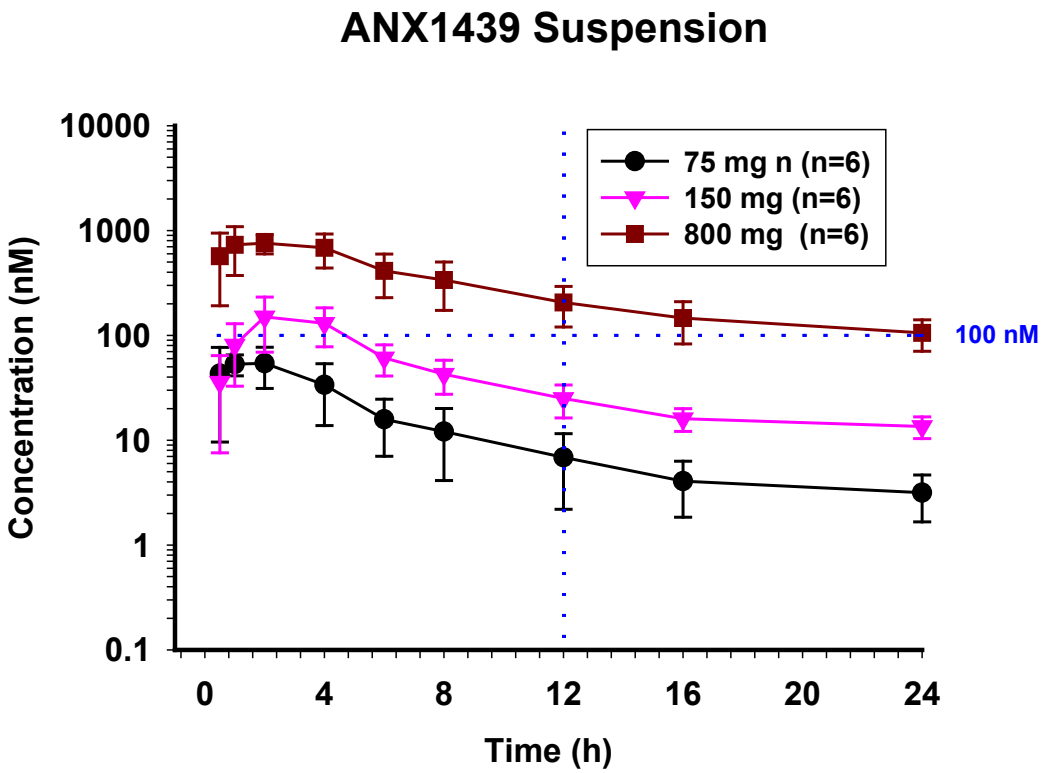
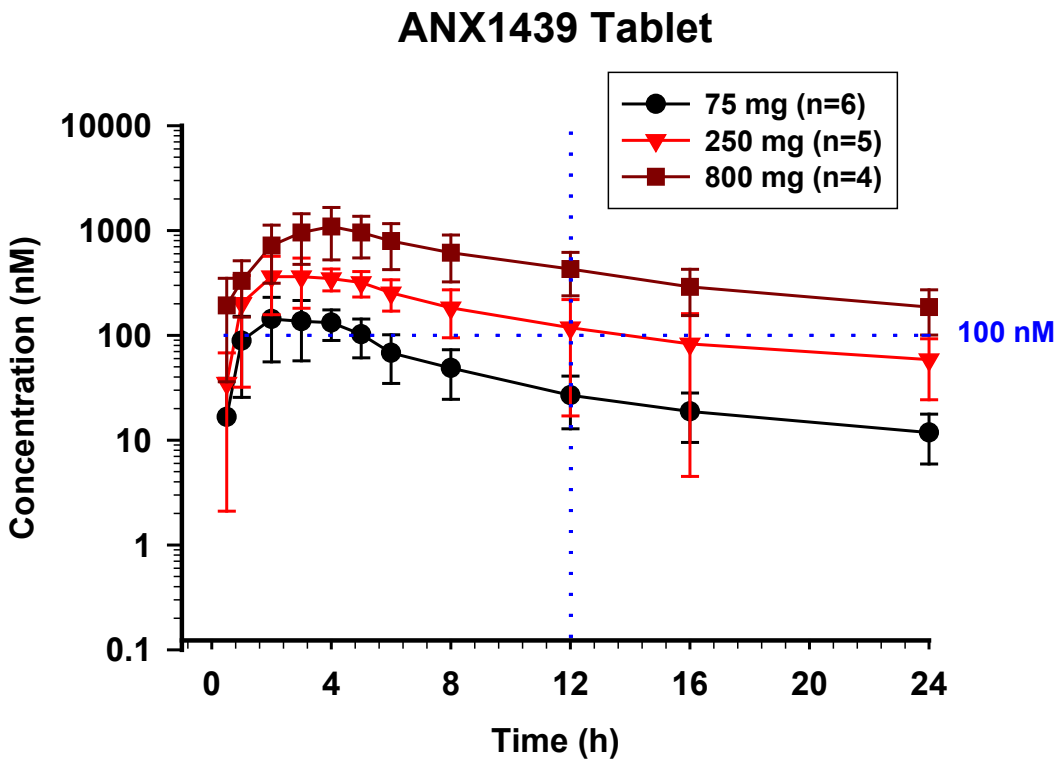
PK Comparable Between Suspension and Tablet

Observed results indicate ability to achieve target concentrations with BID dosing of tablet



Concentrations were BLQ post 36h for 75 mg dose and post 48h for 150mg and 800 mg



Concentrations were BLQ post 36h for 75 mg dose

ANNEXON biosciences

ANX1502 Transforms Administration in Chronic Autoimmune Disease

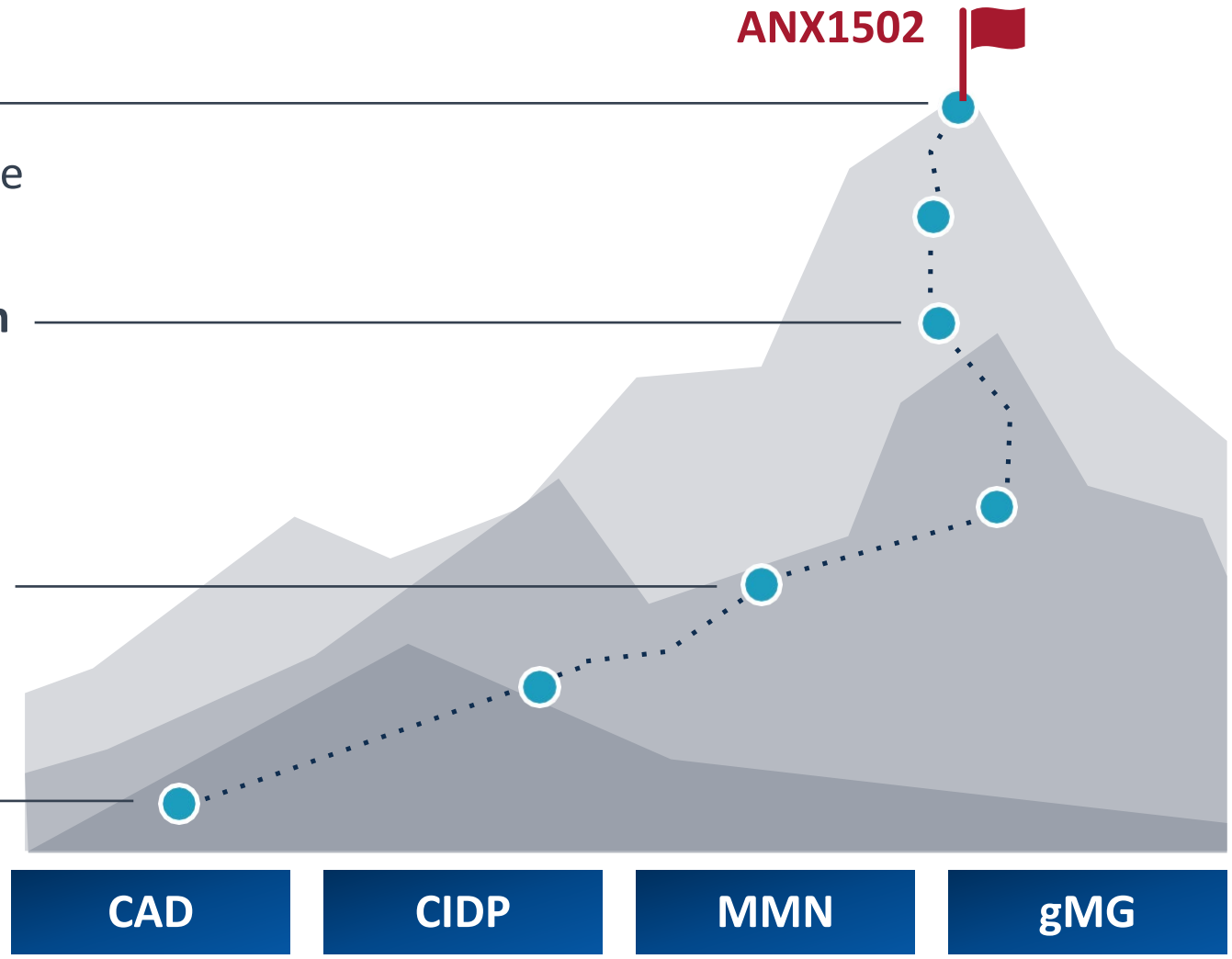
Oral dosing provides increased convenience and reduced patient burden

Oral dosing
Convenient with higher compliance

Subcutaneous self-administration
Self-administered needle-phobia and less flexibility

Daily self- or HCP-administration
Less convenience and flexibility

In clinic administration
Time consuming, expensive, and inconvenient



CAD: Cold agglutinin disease; CIDP: Chronic inflammatory demyelinating polyneuropathy; MMN: multifocal motor neuropathy; gMG: Myasthenia Gravis

ANNEXON biosciences

ANX1502 Clinical Development Plan Designed for Rapid Proof-of-Concept and Expansion

Oral tablet formulation provides significant market potential as a chronic treatment

FIRST-IN-HUMAN STUDY
in Healthy Volunteers

- ✓ Generally safe & well tolerated
- ✓ Targeted serum drug levels reached with suspension ***and tablet*** formulation
- ✓ Supportive PD data in participants with higher C4d baseline measures

PROOF-OF-CONCEPT TRIAL
in Patients

- Clinically validated indication
- Block complement activation triggered by cold agglutinins (CAD)
- **Rapid path to establish clinical POC on objective measures (e.g., hemoglobin) in small number of patients**
- POC readout expected 2H 2024

PROGRAM EXPANSION
upon Clinical POC

- **Autoimmune diseases with prior clinical validation and scientific rationale, including:**
- **CIDP**: Chronic inflammatory demyelinating polyradiculoneuropathy
- **MG**: Myasthenia gravis
- **MMN**: Multifocal motor neuropathy
- **Other** antibody-mediated autoimmune diseases

CAD = Cold agglutinin disease

ANNEXON
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A bold mission to enable MILLIONS of PATIENTS impacted by complement-mediated diseases of the body, brain and eye LIVE THEIR BEST LIVES

Annexon Bio: Intentionally and Rigorously Tackling an Array of Classical Complement-Mediated Diseases

Stopping the Start of Classical Pathway Neuroinflammation

Broad Therapeutic Application of Late-Stage Clinical Platform

Multiple Near-Term Clinical Catalysts

ON A JOURNEY TO HELP PATIENTS REGAIN THEIR INDEPENDENCE

Well-researched MOA demonstrated differentiated functional outcomes across GBS, CAD, GA and HD

Suite of fit-for-purpose drug candidates for diseases of the body, brain and eye

- ✓ *GBS pivotal Ph3 data readout (Q2)*
- ✓ *GA pivotal Ph3 initiation (mid-yr)*
- *Oral program POC data readout (2H)*

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Appendix

Complement Drives Nerve Damage Across all GBS GBS Subtypes

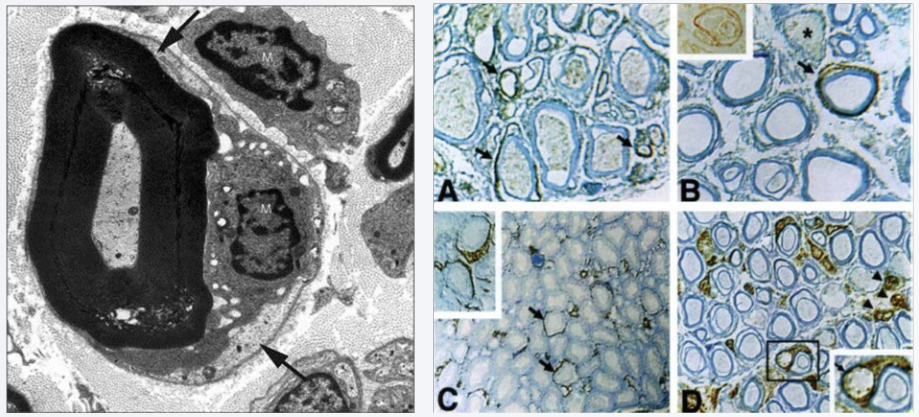
Complement is activated by autoantibodies on axon and myelin

C1q binds to IgM and IgG antibodies on nerve surface and activates the classical complement pathway which leads to….

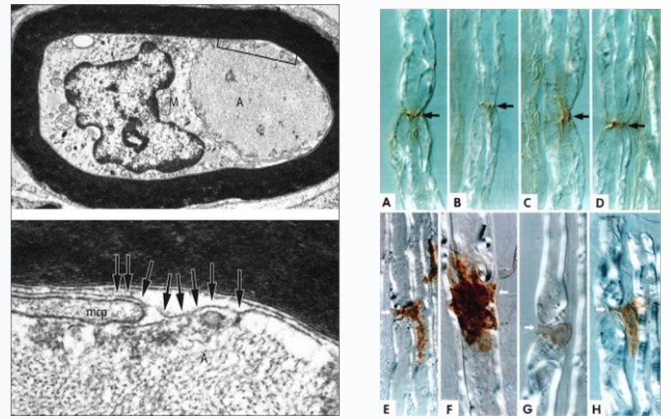
→ Neuroinflammation
→ Macrophage attack (C4b, C3b)
→ Direct membrane damage (C5b-9)
→ Sudden and prolonged loss of muscle strength

Activated Complement in AIDP and AMAN

Acute Inflammatory Demyelinating Polyneuropathy (AIDP)



Acute Motor Axonal Neuropathy (AMAN)



C3d

Griffin et al. Ann Neurol 1994, Hafer-Macko et al. Ann Neurol 1997

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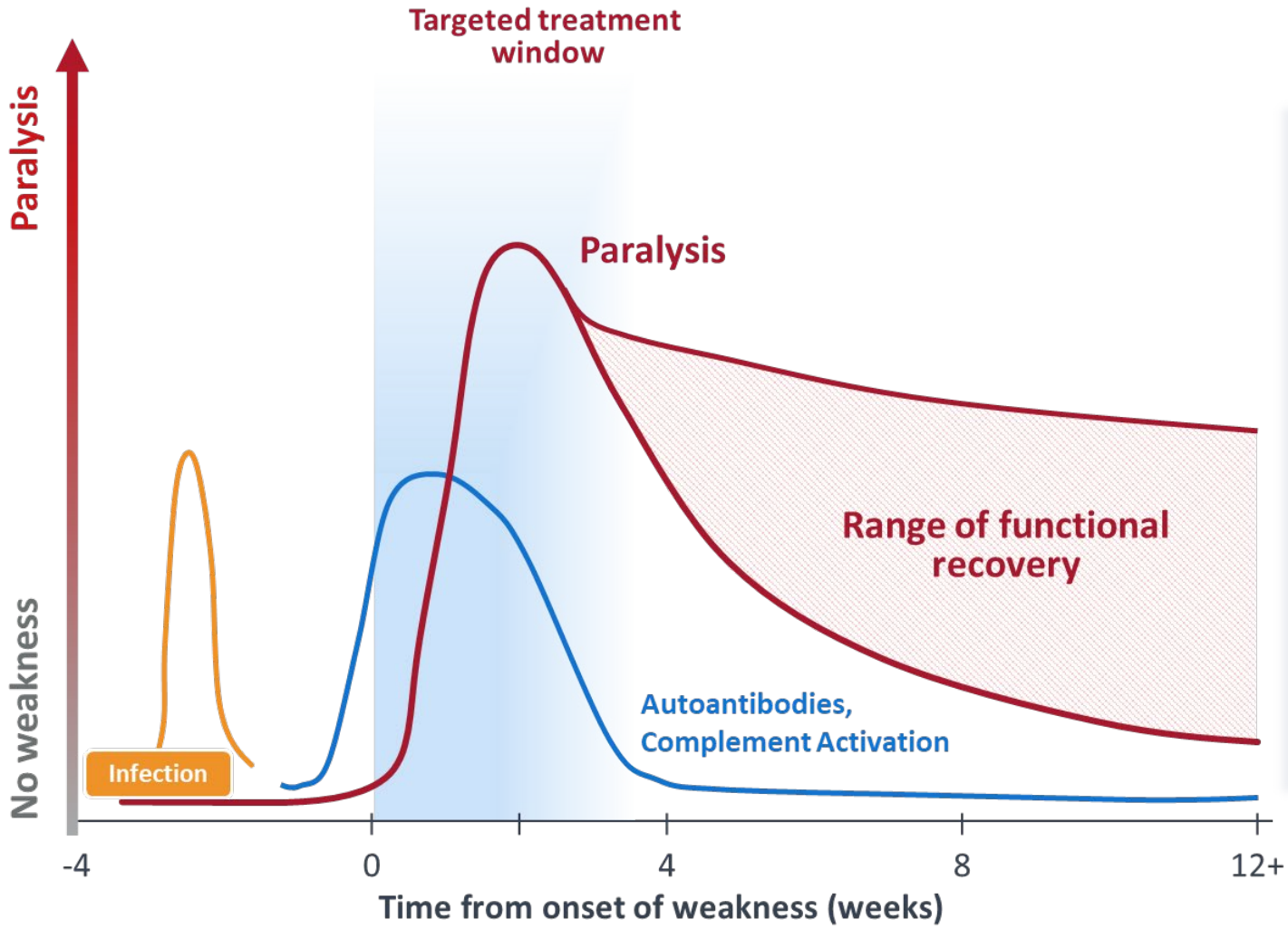
Complement Inhibition During the Active Disease Phase is Key

Acute disease phase of GBS is generally short and varies by patient



- GBS has an acute disease phase followed by spontaneous recovery
- Objectives of anti-C1q treatment in GBS
  - ✓ <u>Block</u> complement-mediated nerve damage during the acute disease
  - × <u>Do not block</u> complement-facilitated nerve repair during recovery phase
- Target treatment window is likely within first 2 weeks

1. van den Berg B et al. *Nat Rev Neurol*. 2014;10(8):469-482. 2. Leonhard SE et al. *Nat Rev Neurol*. 2019;15(11):671-683.

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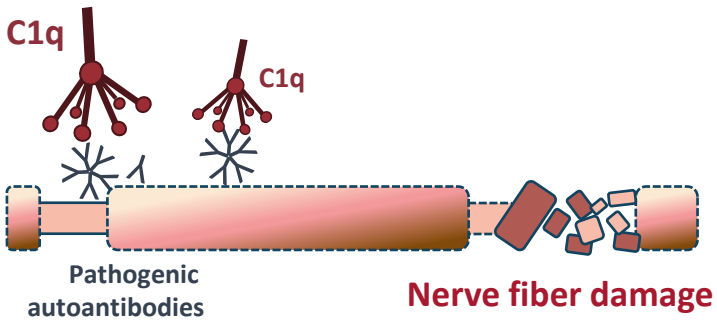
Conceptual Framework for Complement's Dual Role in GBS and how ANX005 is Intended to Work

GBS Active Disease Phase

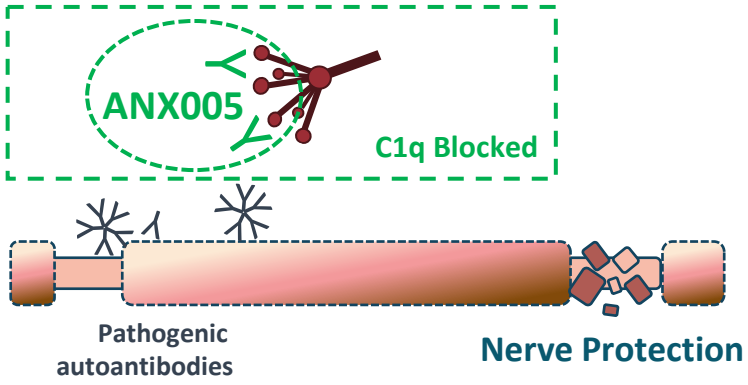
Complement mediates nerve damage

C1q ➡ • Classical complement activation
• Nerve fiber damage
• Tissue debris



ANX005 During Effective Treatment Window

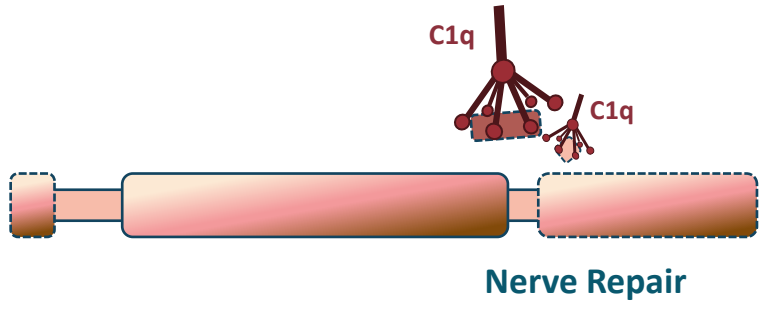
C1q ➡ • Entire cascade blocked by ANX005
• 30mg/kg leads to 1 week of C1q suppression during active disease



GBS Recovery Phase

Complement facilitates nerve repair

C1q ➡ • Clears debris
• Allows nerve regeneration
• Facilitates remyelination & repair



ANX005 protects nerves from complement-mediated nerve damage

Hafer-Macko C et al., (1996) Ann Neurol 39 & 40

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Complement Inhibition Stopped Nerve Damage During Acute Autoimmune Injury while Inhibition During Recovery Phase Slowed Repair in Rat Models

Complement inhibition blocks acute nerve damage in an autoimmune neuropathy model

- Animals developed autoantibodies that activated complement and damaged peripheral nerves
- Acute damage blocked by complement inhibition

Complement Inhibition Protects Against Acute Nerve Damage in Autoimmune Neuritis

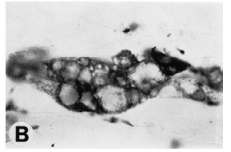
Complement inhibition during recovery phase slowed debris clearance and repair in an acute nerve injury model

- Wallerian degeneration with macrophage infiltration, myelin removal and axonal regrowth

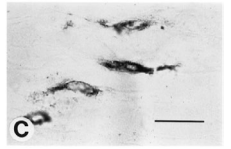
Clearance and Regrowth Slowed by Complement Inhibition

Debris Clearance by Macrophage **Axonal Repair**

Macrophage engorged with myelin debris



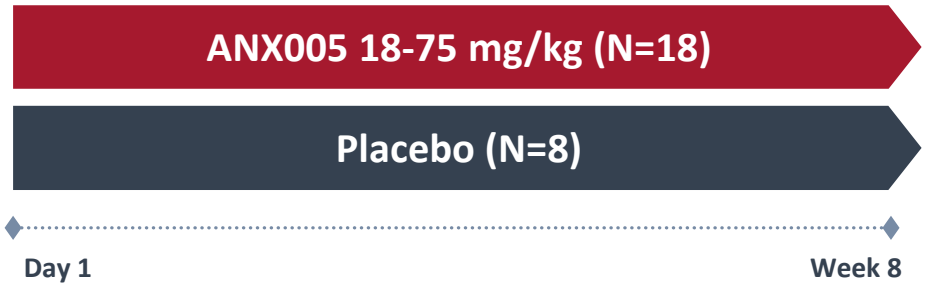
Resting macrophages w/ complement inhibition (cobra venom factor)



The Dose-Ranging Ph1b Study Laid Foundation for Phase 3 Design

Phase 1b Study Design

Study Schematic



- Randomized, double-blind, placebo-controlled study
- N=26[1] Adults with GBS in Bangladesh
- Mean time from onset of weakness: 8.1 days
- Mean GBS-DS at baseline: 4[1]

[1]18-75mg/kg double-blinded dose cohorts

Key Learnings Applied to Phase 3

- ✓ Rapid and full C1q inhibition observed at all doses
- ✓ Stratified by key prognostic factors
  - ✓ MRC
  - ✓ Time from onset of weakness
- ✓ Treat as early as possible (day of randomization)

Phase 3 Designed to Define the Appropriate Duration of Complement Inhibition in GBS

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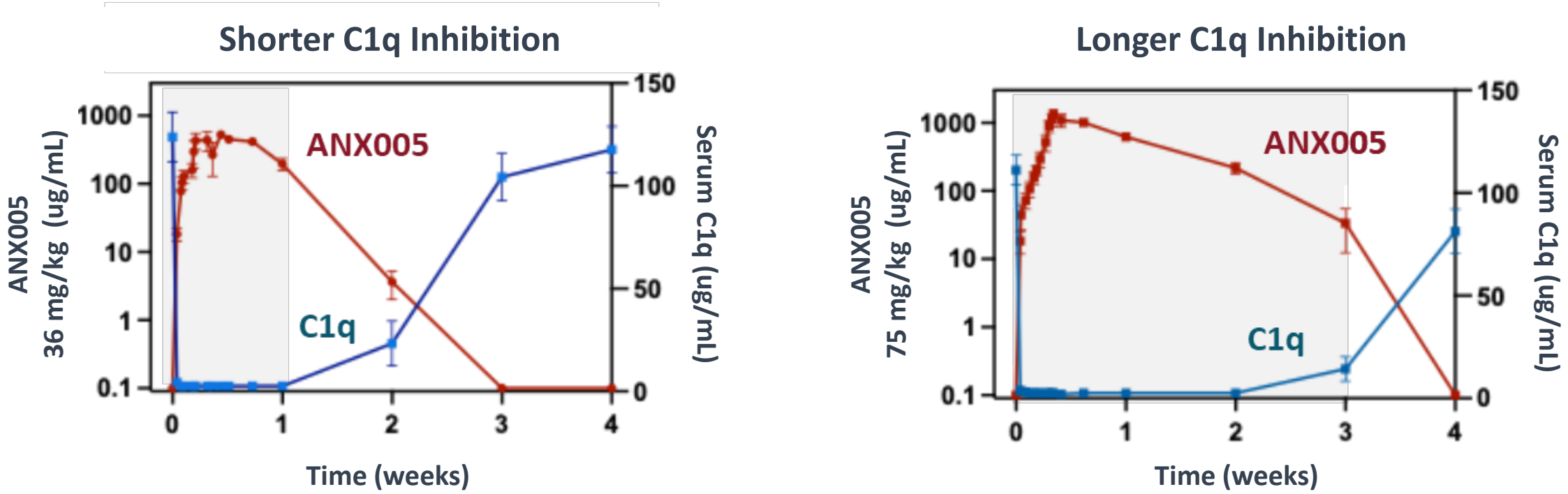
Phase 1b Evaluated Shorter & Longer Durations of Complement Inhibition



- ✓ Immediate full complement inhibition with single infusion
- ✓ C1q inhibition lasts 1-3 weeks with lower and higher dose

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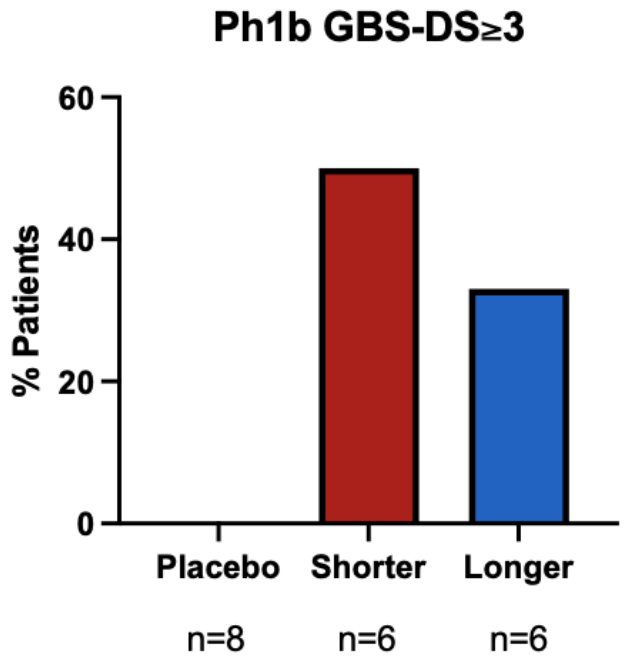
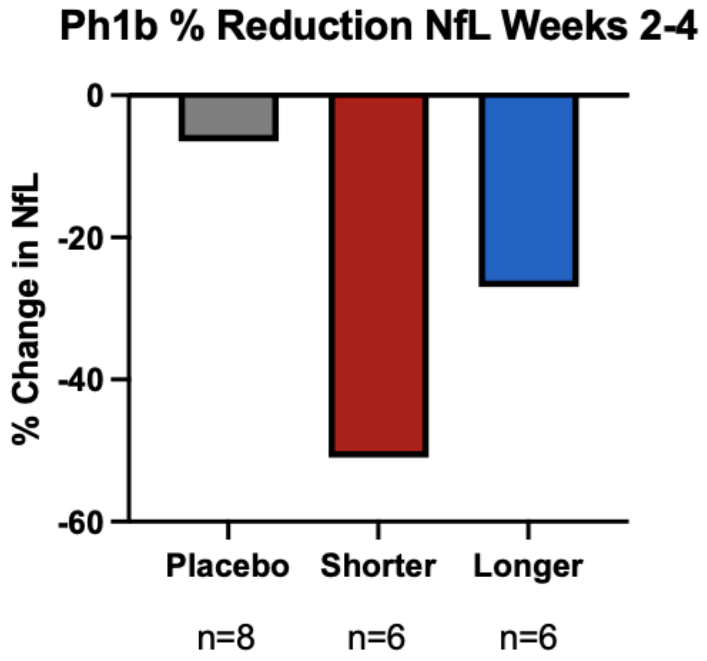
Phase 1b Suggested Shorter Duration of Complement Inhibition had a Greater Effect

Patients Gaining ≥ 3 Points on GBS-DS At Week 8



NfL Reduction Wks 2-4



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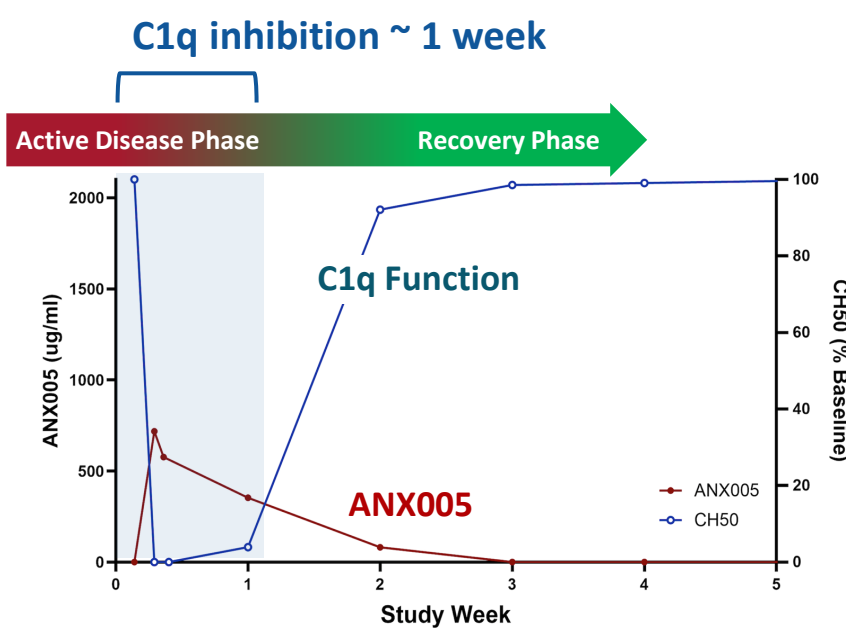
ANX005: Expected Pharmacokinetic and Dynamic Response for Both Doses

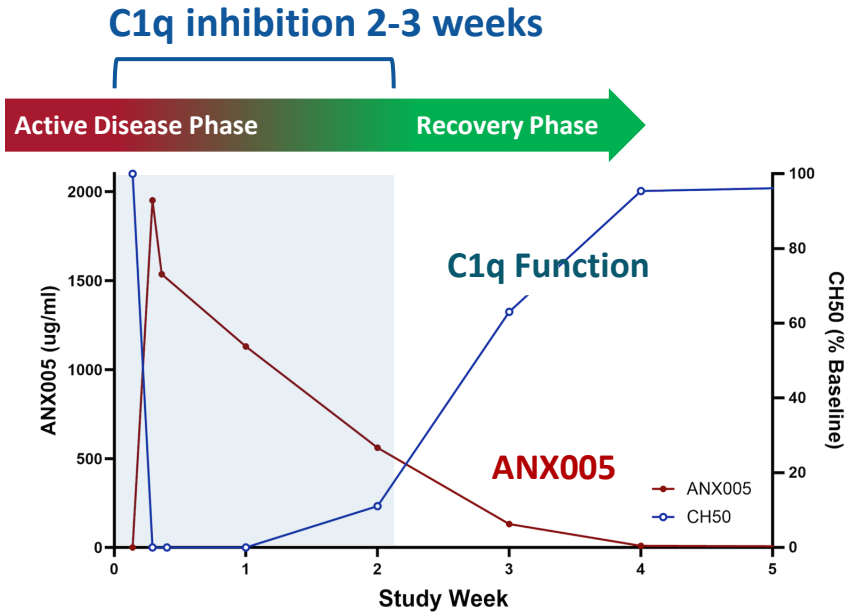
Duration of complement inhibition defines active treatment window

- **Rapid C1q engagement and functional inhibition** (CH50 assay)
  - 30 mg/kg provided: ~1 week duration of inhibition
  - 75 mg/kg provided: 2-3 weeks duration of inhibition

ANX005 30 mg/kg



ANX005 75 mg/kg



Topline Results Subject to Change

ANNEXON biosciences

Summary of Primary and Key Secondary Results

Statistical testing hierarchy of clinically relevant endpoints

Primary	Endpoint	Assesses	Timepoint	30 mg/kg Efficacy	P-value	75 mg/kg Efficacy	P-value
1	GBS-DS	GBS disability	Week 8	OR[1] = 2.41	0.0058	*OR[1] = 1.2*	*0.5548[3]*

Secondary Hierarchy	Endpoint	Assesses	Timepoint	30 mg/kg Efficacy	P-value	75 mg/kg Efficacy	P-value
2	Overall Neuropathy Limitations Scale (ONLS)	Functional disability	Week 8	-0.8[2]	0.0965[3]	*-0.3[2]*	*0.5033[3]*
3	MRC Sumscore	Muscle strength	Week 8	4.0[2]	0.0351[3] Nominal	*2.0[2]*	*0.2952[3]*
4			Day 8	10.0[2]	<0.0001[3] Nominal	*8.3[2]*	*<0.0001[3]*
5	Ventilation	Duration of ventilation[3]	Week 26	Median 28 fewer days	0.0356[4] Nominal	*Median 34 fewer days*	*0.0011[3]*

[1]Odds Ratio: Likelihood that a patient on ANX005 is in a better state of health relative to placebo

[2]LS mean point improvement relative to placebo

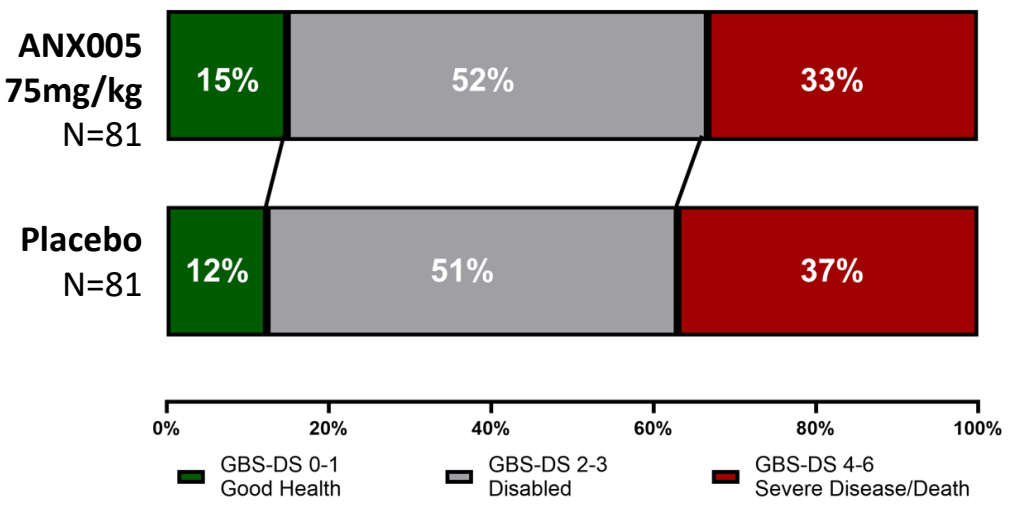
[3]P-values for nominal testing using 2-sided α=0.05

[4]For those requiring ventilation

ANX005 75 mg/kg Did Not Meet the Primary Endpoint with Inhibition Beyond Active Disease Process

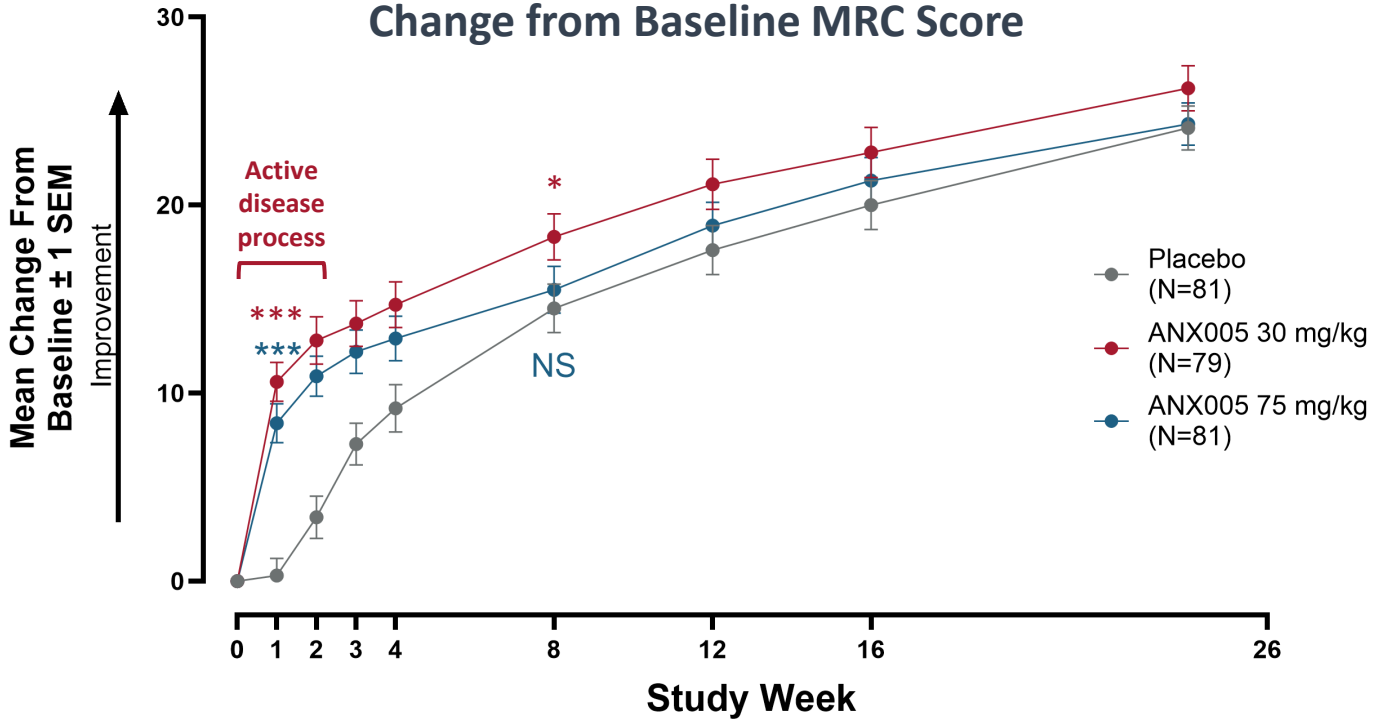
75 mg/kg improved muscle strength similar to 30 mg/kg during active disease process

Grotta Bar of GBS-DS at Week 8



OR 1.20, p=0.5548

Change from Baseline MRC Score



Nominal p-values

Topline Results Subject to Change

ANNEXON biosciences

Early and Durable Treatment Effects of ANX005 30 mg/kg vs. Placebo

Immediate impact to disease trajectory translated to improvements through week 26

		Early Impact on Disease Trajectory						Durable Benefits	
Pre-specified Analyses	**Unit**	**At Week 1**		**At Week 4**		**At Week 8**		**Through Week 26** (MMRM)	
GBS-DS	Odds Ratio	OR[1]: 7.22	p=<0.001[3]	OR[1]: 2.49	p=0.0073[3]	OR[1]: 2.41	p=0.0058	OR[1]: 1.49	p=0.0120[3]
MRC	Point Improvement	10 points[2]	p=<0.0001[3]	5.4 points[2]	p=0.0026[3]	4 points[2]	p=0.0351[3]	5.4[2]	p=0.0010[3]
ONLS	Point Improvement	-2.1 points[2]	p=<0.0001[3]	-1.1 points[2]	p=0.0154[3]	-0.8 points[2]	p=0.0965[3]	-1.1[2]	p=0.0063[3]
Ventilation	Median Days	N/A						28 days reduction[4]	p=0.0356[3]

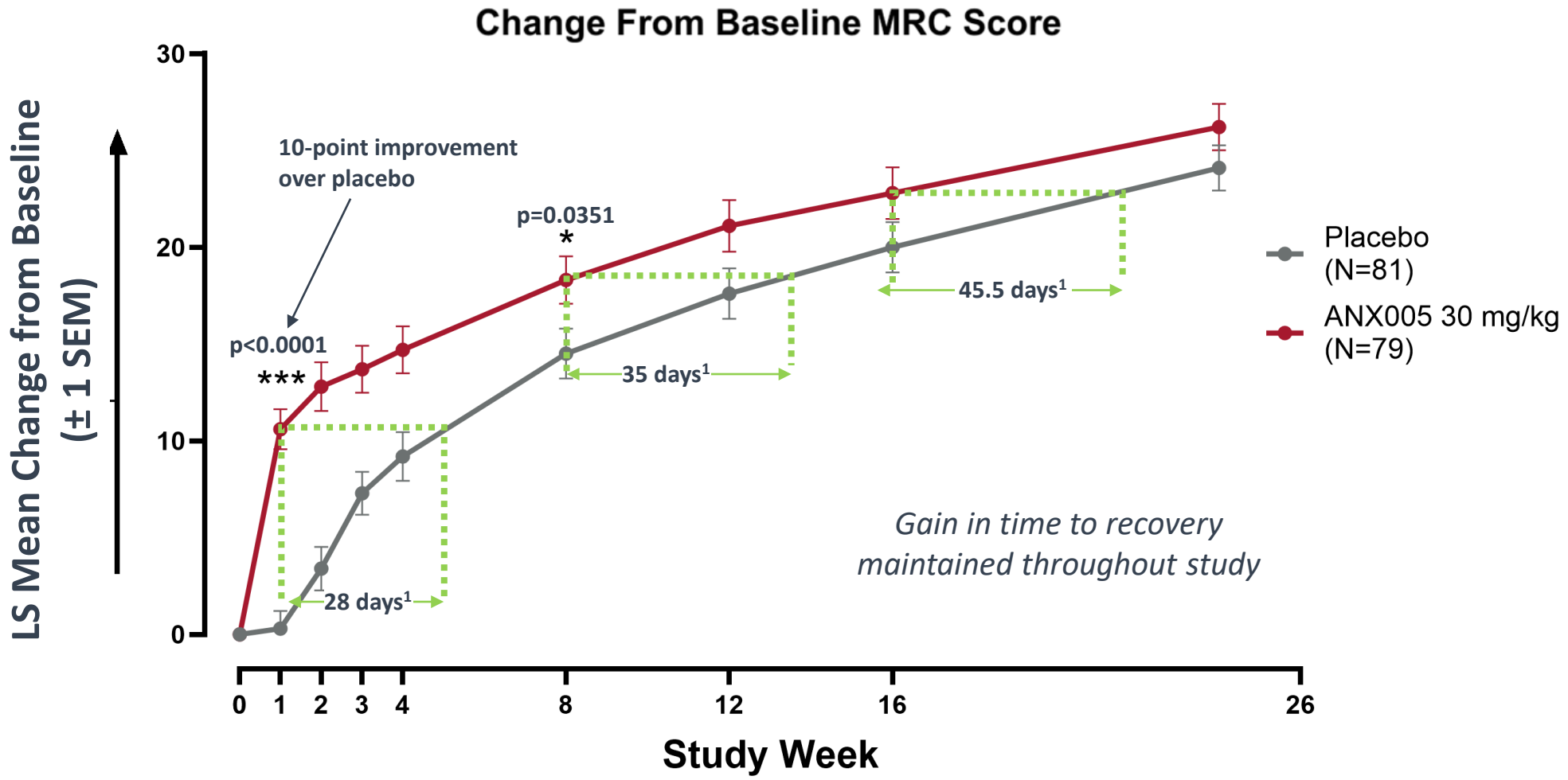
[1]Odds Ratio: Likelihood that a patient on ANX005 is in a better state of health relative to placebo
[2]LS mean difference relative to placebo
[3]P-values for nominal testing using 2-sided α=0.05
[4]For those requiring ventilation
[5]LS Mean percent reduction

Topline Results Subject to Change

ANNEXON biosciences

MRC: ANX005 30 mg/kg Increased Muscle Strength Earlier Relative to Placebo, and the Advantage Grew Over Time

Early muscle strength improvement maintained & increased through full 26-week study (p=0.0010)

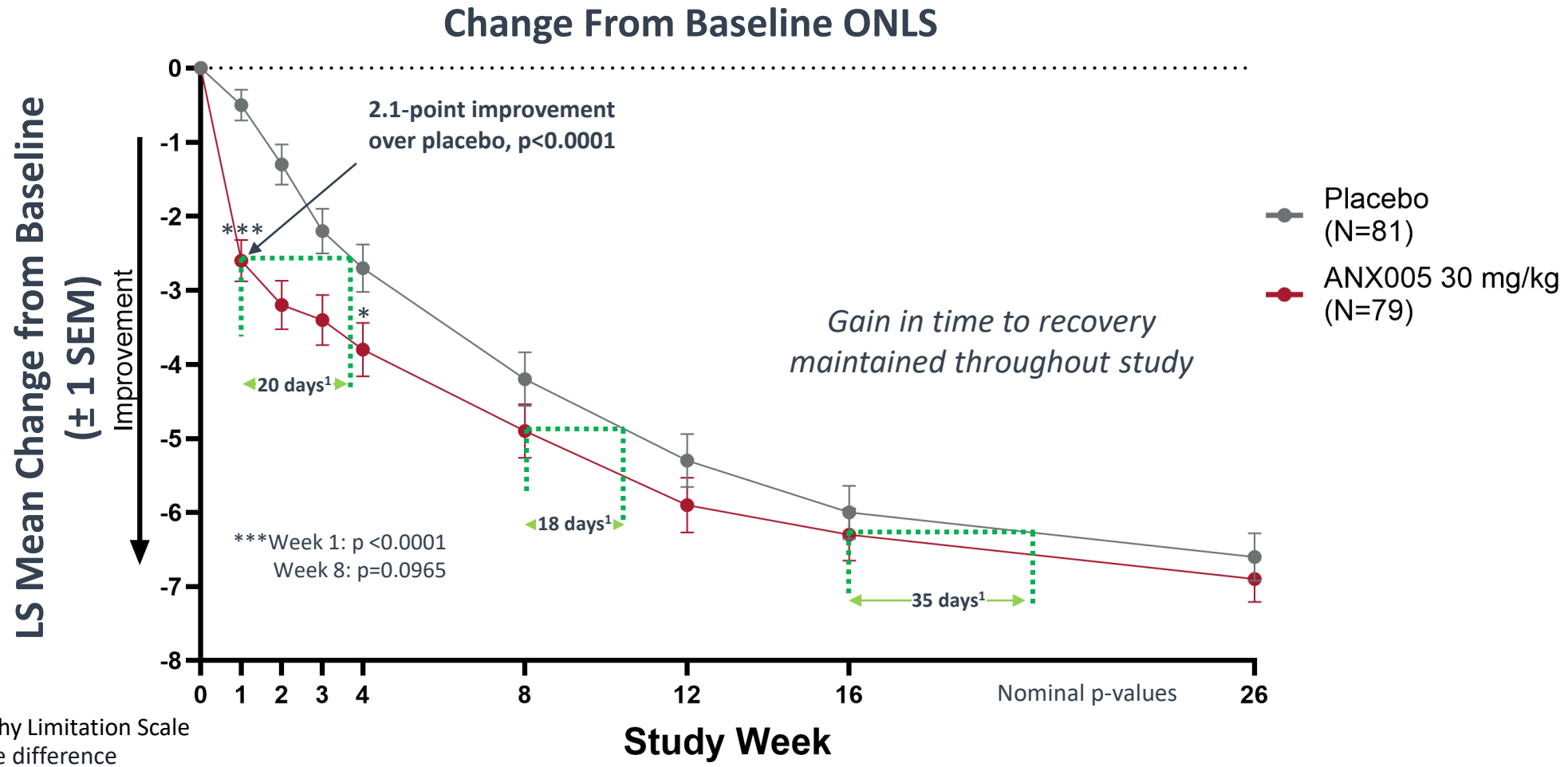


[1]Approximate

ANNEXON biosciences

ONLS: ANX005 30 mg/kg Showed Significant Early Improvement in Motor Disability vs. Placebo on the ONLS* Scale

Maintains ability to perform daily tasks through 26 weeks p=0.0063



*Overall Neuropathy Limitation Scale

[1]Approximate Time difference

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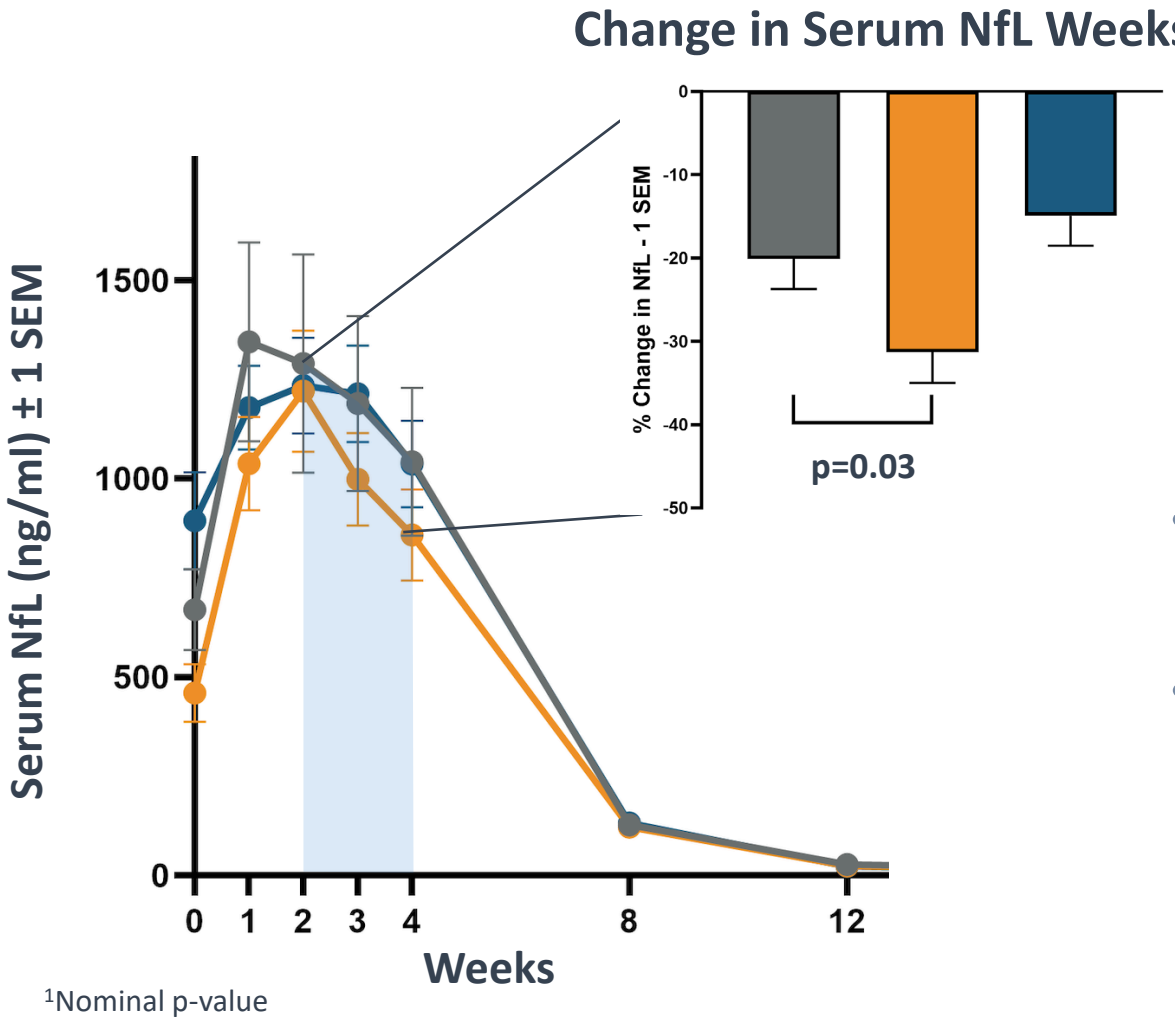
ANX005 30 mg/kg Demonstrated Significant Early Reduction in Prespecified Analysis of Neurofilament Light Chain (NfL) During Disease Recovery Phase



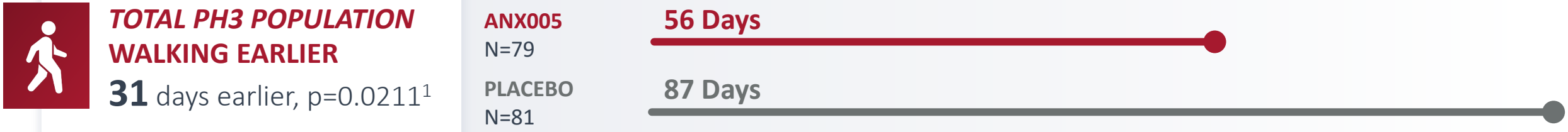
[1]Nominal p-value

Key Takeaways

- **Active disease phase (wks 1-2):** ANX005 associated with lower peak levels of NfL vs Placebo
- **Recovery phase (wks 2-4): ANX005 30 mg/kg provided significant early reduction in NfL vs Pbo** (31.3% vs. 20.1%, p=0.03[1])
  - Prespecified analysis, based on Phase 1b results

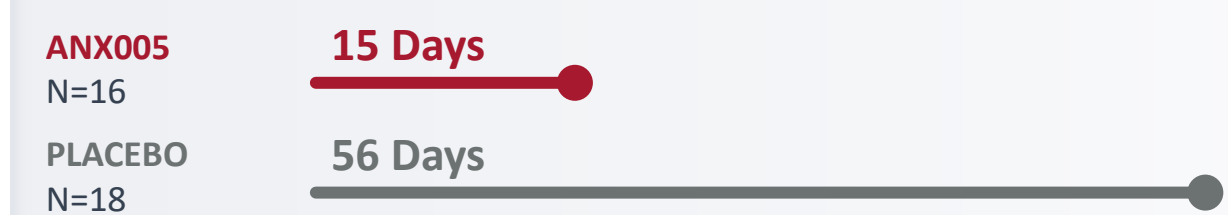
ANX005 Patients Resembling US and European Populations Got Better Sooner

TOTAL PH3 POPULATION
WALKING EARLIER
31 days earlier, p=0.0211[1]



PH3 SUB-GROUP: MRC >20
WALKING EARLIER
41 days earlier, p=0.0814[1]

PH3 SUB-GROUP: AIDP
WALKING EARLIER
41 days earlier, p=0.0048[1]



Topline Results Subject to Change

ANNEXON biosciences

Annexon + IGOS are Advancing a Real-World Evidence Study to Demonstrate Population Comparability Between Phase 3 and Western Patients in IGOS

- **Background**
  - FDA agreed that a single pivotal study could be sufficient for BLA assuming it demonstrates:
    - Substantial evidence of ANX005's treatment effect vs. placebo
    - Comparability between Ph3 population & Western patients (on-track)
  - Annexon has initiated a real-world evidence comparability protocol with IGOS (ANX005-GBS-04)
    - IGOS is a global, prospective, observational, multicenter cohort study
    - IGOS is led by global experts in GBS and has enrolled 2000 patients who were followed for 1-3 years

- **FDA precedent for approvals based on studies conducted entirely ex-US**
  - Radicava (edaravone), approved for ALS using N=137 study conducted in Japan in 2017
  - Brukinsa (zanubrutinib) approved for mantel cell lymphoma using N=86 study conducted in China in 2019

- **Current Status and Next Steps**
  - On pace to deliver full comparability data 1H25 in support of BLA submission
  - Annexon is also collaborating with IGOS on an outcomes comparison between ANX005 30mg/kg vs. IVIg

Summary of Primary, Key Secondary Results & Pre-Specified Sensitivity

Consistent & meaningful outcomes following 1 week of complement inhibition (30 mg/kg)

Primary Endpoint	GBS-DS at Week 8	**2.4x more** likely better state of health	p=0.0058
Secondary Endpoints	ONLS at Week 8	0.8-point improvement in daily activities	p=0.0965
	MRC Sumscore at Week 8	**4-point improvement** in muscle strength	p=0.0351[1]
	MRC Sumscore at Day 8	**10-point improvement** in muscle strength	p<0.0001[1]
	Duration of Ventilation	**28 fewer days** on ventilation	p=0.0356[1]
Pre-specified Sensitivity Analyses	GBS-DS Dichotomy at Week 8	**3.3x more** likely to run	p=0.0065[1]
	GBS-DS Responder at Week 8	**2x more** patients with ≥3-point improvement	p=0.0309[1]
	GBS-DS Through Week 26	**1.49x more** likely better state of health	p=0.0120[1]
Getting Better Sooner	Muscle Strength	**1-month sooner** to 10-point improvement	
	Activities of Daily Living	**20 days sooner** to 2-point improvement	
	Time to Walk	**1-month sooner** to walking independently	p=0.0211[1]
	Off Ventilation	**1-month sooner** to come of ventilator	p=0.0356[1]

[1]Nominal p-values

Topline Results Subject to Change

ANX005 GBS Phase 3 Summary of Key Results

First targeted therapy to demonstrate positive outcomes for GBS community

1 **Phase 3 Met Primary Endpoint**

Patients treated with ANX005 30 mg/kg were 2.4x more likely to be in a better health compared to placebo (p=0.0058)
Treatment during active disease phase was effective

2 **ANX005 Helped Patients with GBS Get Better Sooner**

Early, robust, and clinically meaningful benefit on multiple outcome measures
Walking earlier; Less time on mechanical ventilation

3 **Durable Treatment Effects Across Full Course of 26-Week Study**

More patients fully recovered at 26 weeks

4 **Generally Safe and Well Tolerated**

Safety profile similar to placebo – no increased rate of infections, convenient single dose

5 **Clear Path to BLA Submission and Launch**

Preparing to engage FDA later this year to support BLA submission 1H25
On-track to complete RWE study by 1H25 to support BLA timelines
Preparing clear launch strategy with focused commercial team

Topline Results Subject to Change

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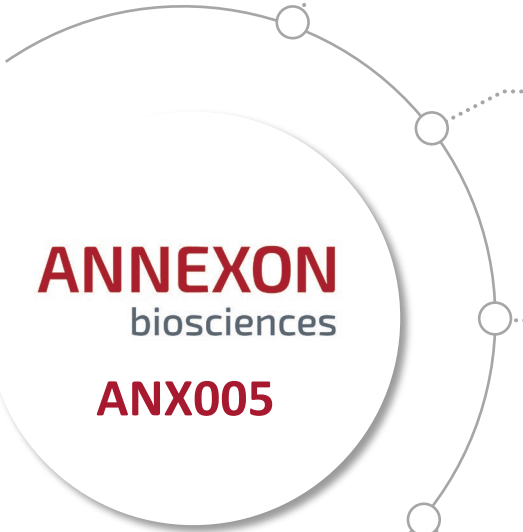
Commercial Strategy for ANX005 in GBS

Key Highlights



1	Potential First Approved Therapy for GBS	Pursuing first-line monotherapy single infusion treatment in GBS with strong registrational data to support BLA submission in 1H 2025
2	Robust Clinical Profile	Phase 3 data showed significant, rapid and sustained improvement across multiple GBS measures vs. placebo, and a well-tolerated safety profile consistent with placebo
3	Significant Commercial Opportunity	Estimated 22K patients hospitalized annually (7K in US / 15K in Europe) and annual peak sales potential of $650M-$1B+ in US and Europe. No approved treatments in US.
4	Focused Commercial Strategy	Focused and efficient commercialization strategy demonstrating a clear approach to customer targeting and market access
5	Indication Expansion Opportunity	Potential to be a pipeline-in-a-product and expand into other attractive large market opportunities (e.g. HD, TBI, ALS, CIDP, etc.)

Annexon's Commercial Approach to Launching ANX005 in GBS

Focused commercial footprint positions ANNX to capitalize on significant commercial opportunity

1 Significant Commercial Opportunity

GBS incidence numbers show **magnitude of disease** and **untapped market opportunity**

Significant disease burden despite available treatments

No FDA approved treatments – **opportunity for ANX005 to be the 1st approved therapy**

2 Addressing Unique Disease Dynamics

Lower referrals to large academic centers driven by **urgency to treat disease** and **high confidence in diagnosis**

Patients present at treatment centers **where symptom onset occurs**

Indiscriminate disease strikes patients at **same rate regardless of race, age, and sex**

3 Planning Focused Commercial Launch

Three-step targeted **customer engagement strategy** necessary for rapid adoption

Plan to start with **major metropolitan centers** where most GBS patients are treated

Building focused commercial team to **optimize education, support,** and **access**

4 Demonstrating Value-Based Benefits

Secure favorable formulary coverage through hospital P&T committees

GBS results in **significant cost burden** on patients, payers, caregivers and hospitals

ANX005 has **clear value-based benefits** that reduce cost to care for GBS patients

1 Significant Commercial Opportunity to Disrupt IVIg in GBS

"I was put on my hands and knees, and **I had to learn how to crawl just like a baby…**

I crawled for 8 or 9 months, and **it took about 2.5 years to learn how to walk…** Then I had 5 years in physical therapy."

Shane S.
53-year-old financial advisor and patient with GBS

SIGNIFICANT DISEASE BURDEN DESPITE CURRENT TREATMENTS [1,2,3,4,5,6,7]

~25%	~40%	~20%	~10%	~5%
require mechanical ventilation	admitted to ICU	can't walk at 1 year	permanently disabled and can no longer work	mortality

>$2B ANNUAL ECONOMIC COST OF GBS IN US[7]

~25% increase in daily cost of ICU care with mechanical ventilation[8]

GBS impacts patients' ability to work and places significant burden on caregivers[7]

ANX005 HAS POTENTIAL TO PROVIDE VALUE-BASED BENEFITS TO REDUCE COST TO CARE FOR GBS PATIENTS AND IMPACT OF DISEASE

1) ClearView Health market research analysis, 2) AAN Guidelines "Immunotherapy for GBS", 3) Hund EF et al (1993) Crit Care Med 21:433, 4) Doets, et al., Brain 2018, 141, 2866-2877 (2018), 5) Van den Berg, B. et al. Nat Rev. Neurol. 10, 469-482 (2014), 6) Leonhard, et al, Nature Reviews, Neurology (2019), 7) Inflation-adjusted from Frenzen, PD (2008) Neurology 71:21-27 7, 8) Kaier K, et al (2019). Epidemiology and Infection 147.

1 ANX005 is Poised to Replace IVIg as Standard of Care

Therapeutic-Specific Success Factors	Shortcomings of IVIg	Opportunities with ANX005
Directly Targeting Disease Mechanism	Mechanism of action in GBS **remains largely unknown**; in the era of targeted immunotherapy, IVIg is a **dated approach**	✓ **Directly targets mechanism** driving extensive nerve damage and paralysis via complement pathway, an **established GBS target**
Rapid Onset of Action	Full treatment course takes **5 days**, **allowing disease mechanisms to continue** and, in some cases, worsen	✓ **Single infusion** enabled patients to **get better sooner** for **significantly higher likelihood of full recovery** versus placebo
Clinical Benefit with Minimal Side-Effects	**Incomplete therapeutic effect**; patients often deteriorate and **recovery is slow and suboptimal;** **Black Box warning** for thrombosis, renal dysfunction, and acute renal failure	✓ ANX005 **generally well tolerated** with **safety similar to placebo** and no increased rate of infection; **prevents acute and ongoing nerve damage** to promote nerve repair

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2 Disease-Driven Commercial Dynamics Present a Unique Opportunity

Unlike other rare diseases, indiscriminate and urgent nature of GBS drives low referrals

Indiscriminate Disease

GBS can strike anyone of any age, race, or sex at any time

Typically present to nearest hospital / ER with neurologist vs. seeking out specific centers for treatment

Urgent, Acute Treatment

GBS is an acute, rapidly-developing disease

Requires urgent treatment within a critical window of time

Significant unmet medical need for treatment that can rapidly impact disease progression and prevent long-term complications

Low Referral Rates

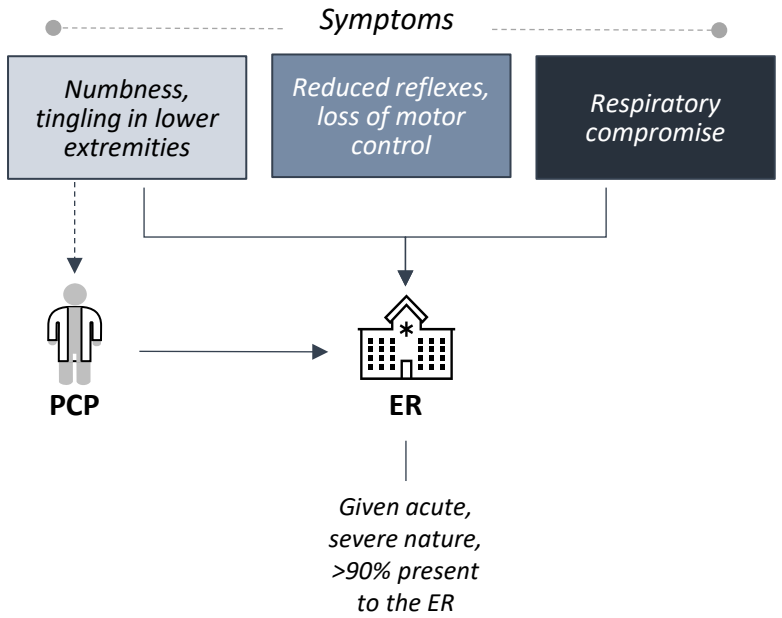
Physicians are hesitant to refer patients given confidence in diagnosis and urgency to treat

Patients geographically concentrated based on population enabling focused targeting

2 Illustrative Current GBS Patient Journey Prolonged By IVIg

Presentation

Patients most often seek care 1-3 days after symptom onset



Diagnosis and Workup

DIAGNOSING HCP*:
~95% Neurologists
~5% Emergency Med

~40% of EDs at non-COIs report use of telehealth to consult for GBS

Key Tests Conducted and Time to Results*

Physical Neuro Exam	Immediate results
Lumbar Puncture	Approx. 2+ hours
EMG (not widely used)	Approx. 48 hours

Confirmatory testing

Treatment and Management

Treatments Received

Outcomes Experienced During Inpatient Stay:

- **~45%** of patients are admitted to the **ICU****
- **~25%** of patients require **ventilation****

Outcomes Experienced Post-Discharge:

- **~85%** of patients require **rehabilitation***
- **~80%** of patients still experience **residual symptoms** 2-4 months after treatment**

Ongoing follow-ups every **~3-6 months during at least first year of recovery** with neurologist

Data is from patients charts unless otherwise noted (n=52); *From interviews; **Patient cases submitted by GBS primary treaters (i.e, neuros) only (n=30)
Source: GBS US Market Assessment. April-June 2024. N=35 HCPs and 10 Admins

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3 Planning Focused Launch Targeting Top Treatment Centers

Intend to target hospitals in three waves, starting with large metropolitan hospitals given significant proportion of total patients served

WAVE 1 — Large Metropolitan & Academic Hospitals

Develop Advocates At Leading Centers And Drive Referrals For Treatment

Initial Launch Targets Treat Majority of GBS Patients

Top 15 Hospitals Treat ~15% of All GBS Patients

Rapid Adoption From Less Cost Sensitive Hospitals

WAVE 2 — Large Community Hospitals

Broaden Outreach to Large Community Centers with High GBS Patient Volume

Leverage Experience and Endorsement of Physicians from Wave 1

Academic Advocates, KOLs, RWE to Encourage Uptake

WAVE 3 — Mid-Sized & Small Community Hospitals

Expand Penetration to Mid-Sized & Small Community Hospitals

Leverage Digital and Peer-to-Peer Platforms

Maximize Penetration by Expanding Outreach

Sources: 81qd and ClearView analysis

3 Building a Focused US Commercial Launch Team

Commercial team will provide education, support, and access to hospitals, neurologists, and care teams

Key Account Managers

- *Educate payers to secure formulary placement*
- *Cover top tier hospital systems*
- *Help facilitate P&T discussions*
- *Target GPOs, IDNs, National plans, Medicare*

Systems-level role to facilitate ANX005 access through education on clinical and value-based benefits

Medical Science Liaisons

- *Global and regional KOL development*
- *Drive awareness of clinical benefits*
- *Generate insights on GBS treatment paradigm*
- *Relay insights on KOL sentiment*

Physician interaction with greater scientific depth

Customer Support Reps

- *ANX005 promotion to label*
- *Active, ongoing customer support*
- *Cover Wave 1 targets at launch*
- *Manage interactions with physicians*

Customer support and sales interactions with physicians and care teams

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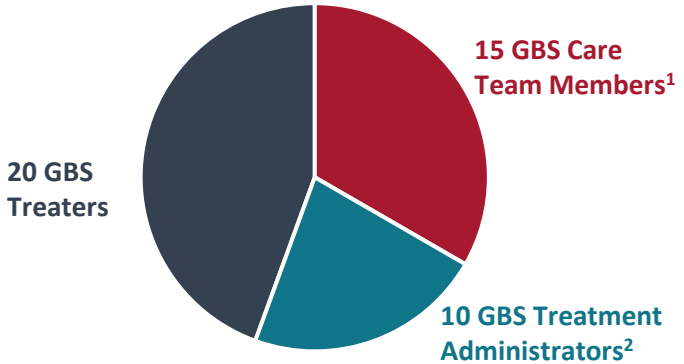
4 Market Access Research

METHODOLOGY

- 45 qualitative web-assisted telephone interviews (WATIs) conducted
- Respondents submitted 2 patient cases before interview to capture key patient details (e.g., outcomes on IVIg)
- Respondents recruited through claims identification, Magnolia's expert panel, and custom search and outreach
- Annexon team was able to listen and ask questions to the moderator during interviews
- Geographically focused in the U.S.

RESPONDENT SAMPLE



45 Total Respondents
(64% from academic centers)

PERSPECTIVES FROM KEY STAKEHOLDERS

*"**[A single, day-1 infusion] is extremely desirable** because you're getting your entire therapeutic dosage all at once as opposed to being spread out over 5 days. **You don't get the peak response until day 4 or 5 for IVIg and PE**."*

- Neuromuscular Specialist, Academic

*"**Efficacy is number one - it's number 1, 2 and 3 really**. We don't see a complete response or a response in all patients. The **response is variable and a bit underwhelming, especially in the serious ones**. The other thing is duration of response because we get that fluctuating disease… The third component would be residual deficits that are long-term or permanent."*

- Neuromuscular Specialist, Academic

*"IVIg and PE have been around for GBS for 30 years, but **neither improves outcomes**. They accelerate recovery, but when you look at the one-year mark, **the number of patients unable to walk, or disabled, are the same compared to placebo**."*

- Neuromuscular Specialist, Academic

*"IVIg is the best that we have for GBS, but **it doesn't do miracles and it's not 100% effective**."*

- Neuromuscular Specialist, Community

*"**We need a treatment that, realistically, can have a faster impact than the current treatments**. Now, treatments can take some time – so we assess what they do [outcomes] at 90 days, rather than right away."*

- Neuromuscular Specialist, Academic

*"**GBS is an expensive venture**. [Patients] are hospitalized a long time, treatment is expensive, staffing nurses, and rehab is all expensive."*

- Critical Care Specialist, Academic

1) HCPs who may diagnose and initiate or advise on treatment but not lead. 2) HCPs and non-HCPs involved in P&T and/or budgetary and hospital stocking decisions.

4 Early, Pre-Launch Community Education for ANX005 is Critical

Strategy	Tactics	Expected Impact
Highlight Clinical Benefit of ANX005	***Present at Neurology & Emergency Med Conferences*** ***Develop Medical Education Materials and Programs*** ***Publication Plan Execution***	• **Increased awareness** of ANX005's clinical value proposition through **tailored narratives delivered to key stakeholders** across settings of care • Willingness to prescribe driven by **increased education of GBS-DS and MRC Sum Score**
Develop ANX005 KOL Advocates	***KOL Outreach and Development*** ***Focused Patient Advocacy Strategy*** ***Sponsor KOL GBS Research and Speaker Series*** ***KOL Treater Visits to High-Volume Hospitals***	• Treatment paradigms developed and community centers educated through **early, pre-launch investment in KOLs** • Highest impact will likely be seen through **data-driven and KOL-leveraged** approaches
Support ANX005 Use by HCPs	***Provide Support for P&T Discussions*** ***Support Initiatives to Update Clinical Guidelines***	• **Ease of prescribing and use of ANX005** amongst HCPs driven by differentiated support systems

HCP: Healthcare Provider; KOL: Key Opinion Leader; RWE: Real World Evidence.
Source: Stakeholder Interviews; ClearView Analysis.

4 Strategies to Drive ANX005 Formulary Inclusion with Key Stakeholders

Strategy	Potential Tactics	Expected Impact
Highlight Clinical and Economic Benefit of ANX005	***Provide HEOR Data to P&T Committees*** ***Publish IVIg RWE***	• **Clear value narratives developed** illustrating clinical and economic benefit of ANX005 over IVIg • **Greater formulary inclusion and potential justification for premium price** relative to IVIg
Facilitate Neurologist and Payer Communication	***Utilize KAMs to Schedule Joint Meetings*** ***Leverage ANX005 Advocates***	• **Joint meetings conducted between neurologists and P&T committee** to help educate on clinical value of ANX005 • **Neurologist advocates play a role in P&T discussions** which are critical for formulary inclusion and favorable placement
Explore Methods to Mitigate Cost Burden to Hospitals	***Seek NTAP Designation*** ***Offer Consignment Stocking*** ***Targeted Contracting***	• **Higher formulary inclusion rates driving overall sales** prompted by Annexon seeking to **reduce cost burden to hospitals** • **Cost risks limited and rapid treatment allowed** due to leverging existing consignment process in hospitals

Illustrating clinical and economic benefit of ANX005, facilitating neurologist advocacy at P&T, and mitigating cost burden to hospitals will help drive formulary inclusion

HEOR: Health Economics and Outcomes Research; NTAP: New Technology Add-on Payment; P&T: Pharmacy and Therapeutics; RWE: Real World Evidence. Source: Stakeholder Interviews; ClearView Analysis.

4 ANX005 Has the Potential to Demonstrate Value-Based Benefits

Current GBS treatments do not address significant cost burden on patients, caregivers, hospitals, and payers

Most Important Outcomes for a New Treatment
(mentioned in top 3 by most respondents in market assessment study)

MINMIZING DAYS ON VENTILATION SUPPORT

INCREASE IN FUNCTIONAL ABILITY*

AVOIDANCE OF ICU STAY

Current Market Dynamics Supportive for Favorable ANX005 Pricing

- Currently, **GBS is not actively managed within hospitals** given its limited budget impact due to its rarity, urgency to treat, and mortality potential
- While IVIg treatments are at a lower price point of $25K per treatment in the US & EU, up to **50% of IVIg patients require a second-line treatment** of IVIg or plasma exchange **increasing time in the hospital and total cost of care**

US Pricing Assumptions for ANX005

Premium price point justified by efficacy and safety profile of ANX005 and its potential to demonstrate value-based benefits

$100K - $150K

Wall Street Analysts' Assumptions

Annexon is currently conducting a detailed pricing assessment which will be evaluated in conjunction with the RWE IVIg outcomes assessment and final label to inform launch price

Annexon will hone ANX005 value proposition through an evidence-generation strategy that leverages RWE and health economic modelling

Source: GBS US Market Assessment. April-June 2024. N=35 HCPs and 10 Admins

*Asked as "Increase in GBS-DS"
** Mentioned in top 3 most important outcomes for a new treatment by most respondents

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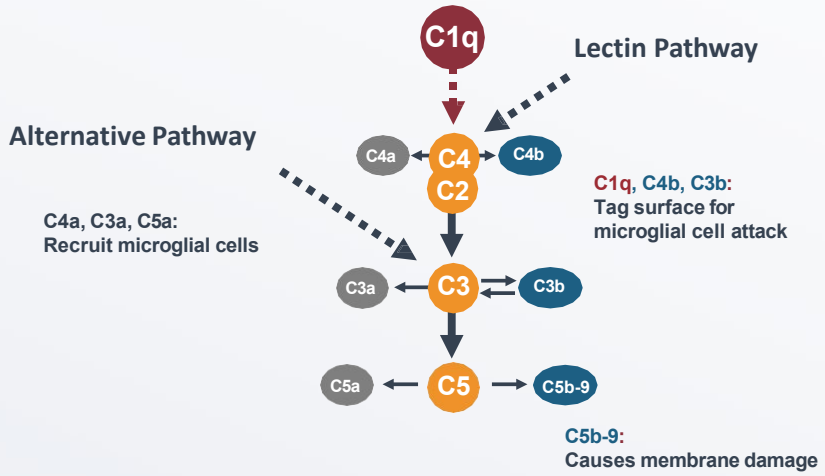
ANX007 Appendix

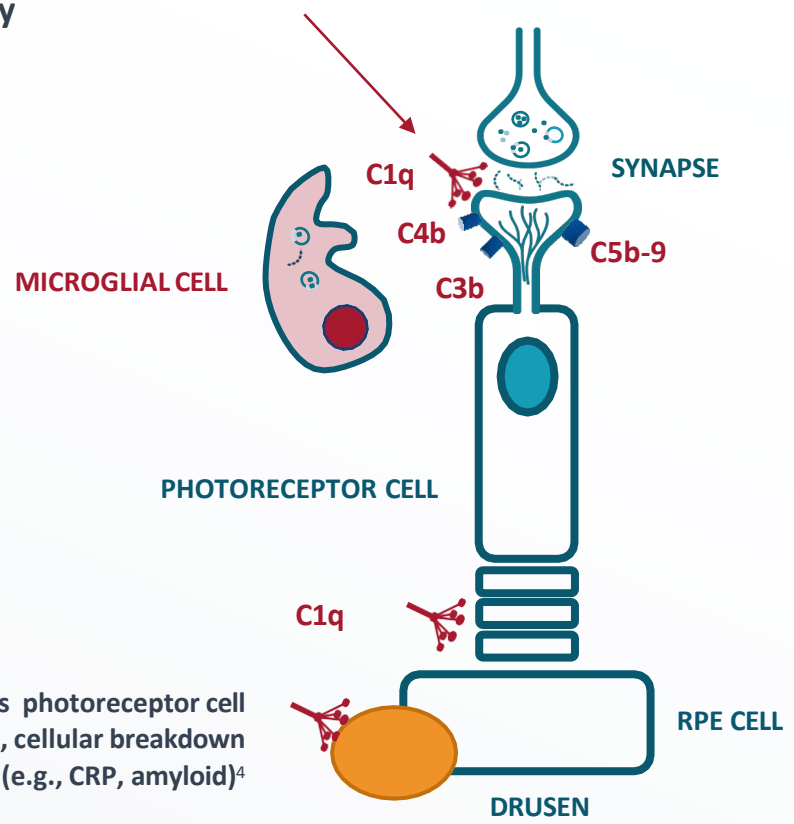
Anti-C1q: A Distinct Neuroprotective Mechanism

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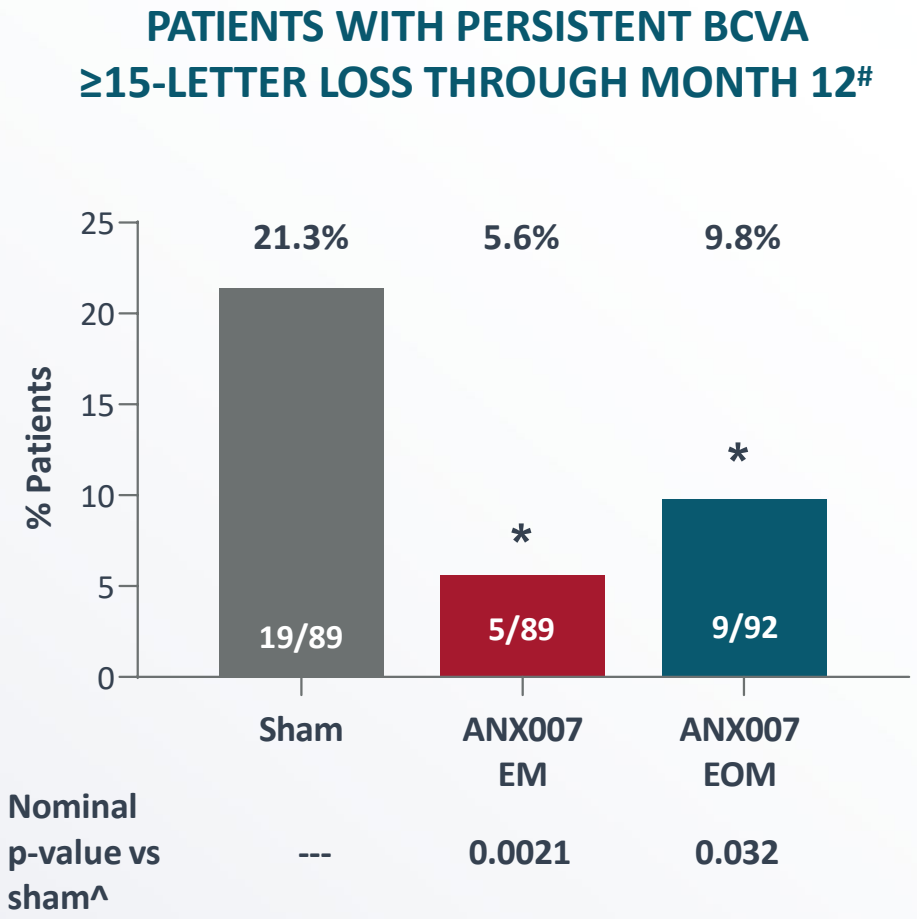
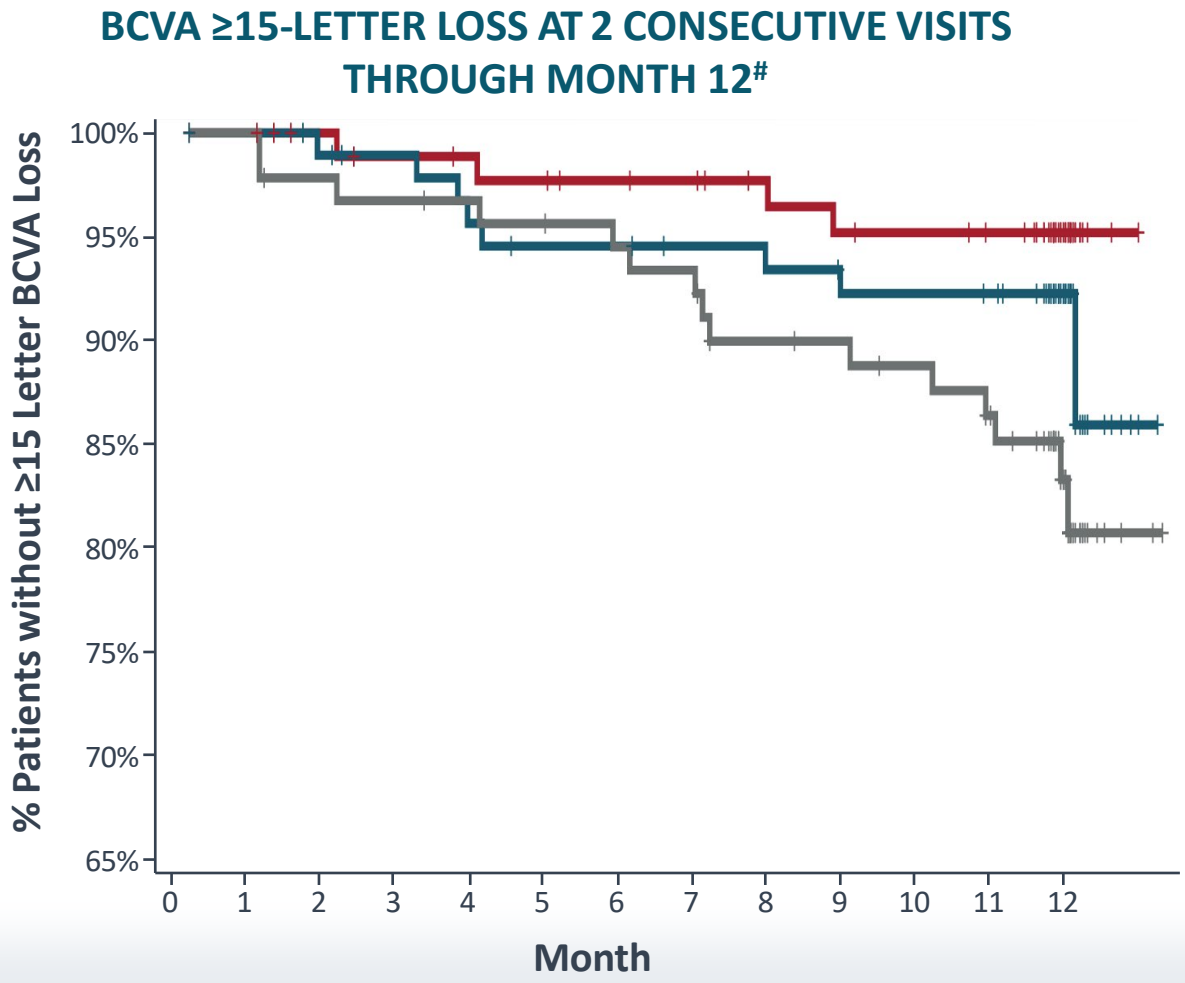
ANX007 Demonstrated Statistically Significant Protection From Vision Loss as Measured by BCVA ≥15-Letter Loss



#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)

53% 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

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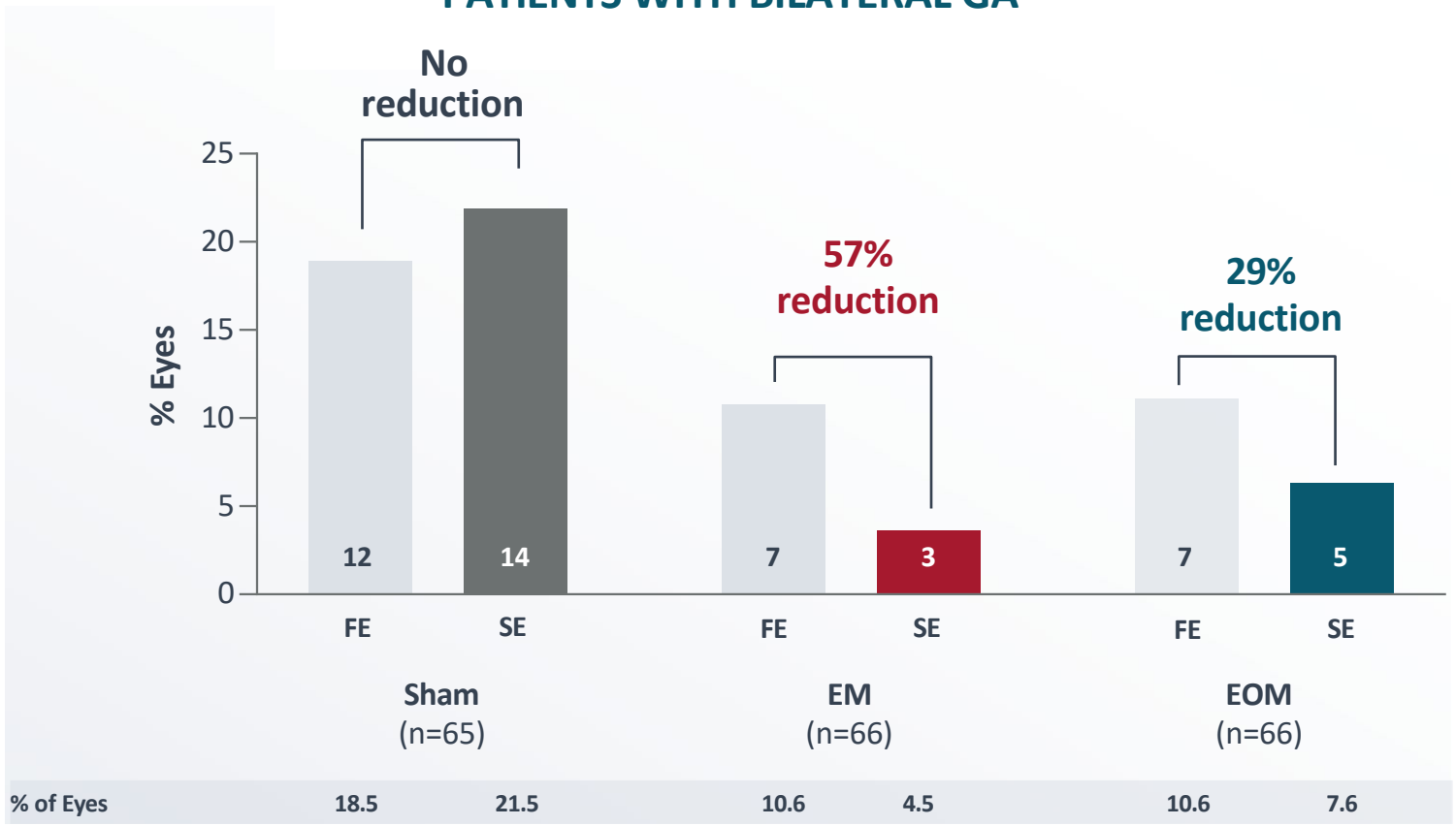
Protection From Vision Loss Supported by Fellow Eye Analysis

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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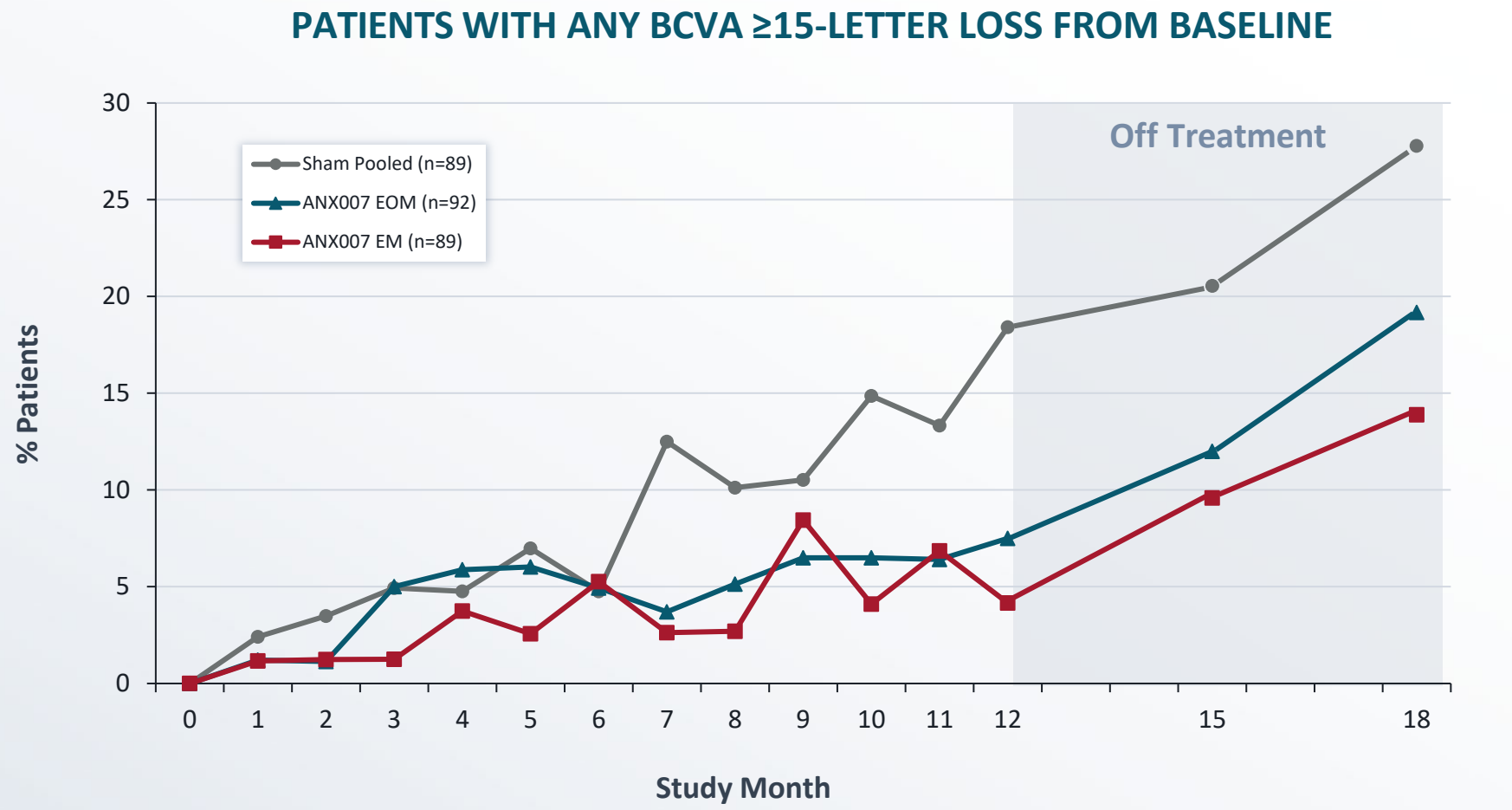
BCVA ≥15-Letter Loss Accelerated After Cessation of Treatment

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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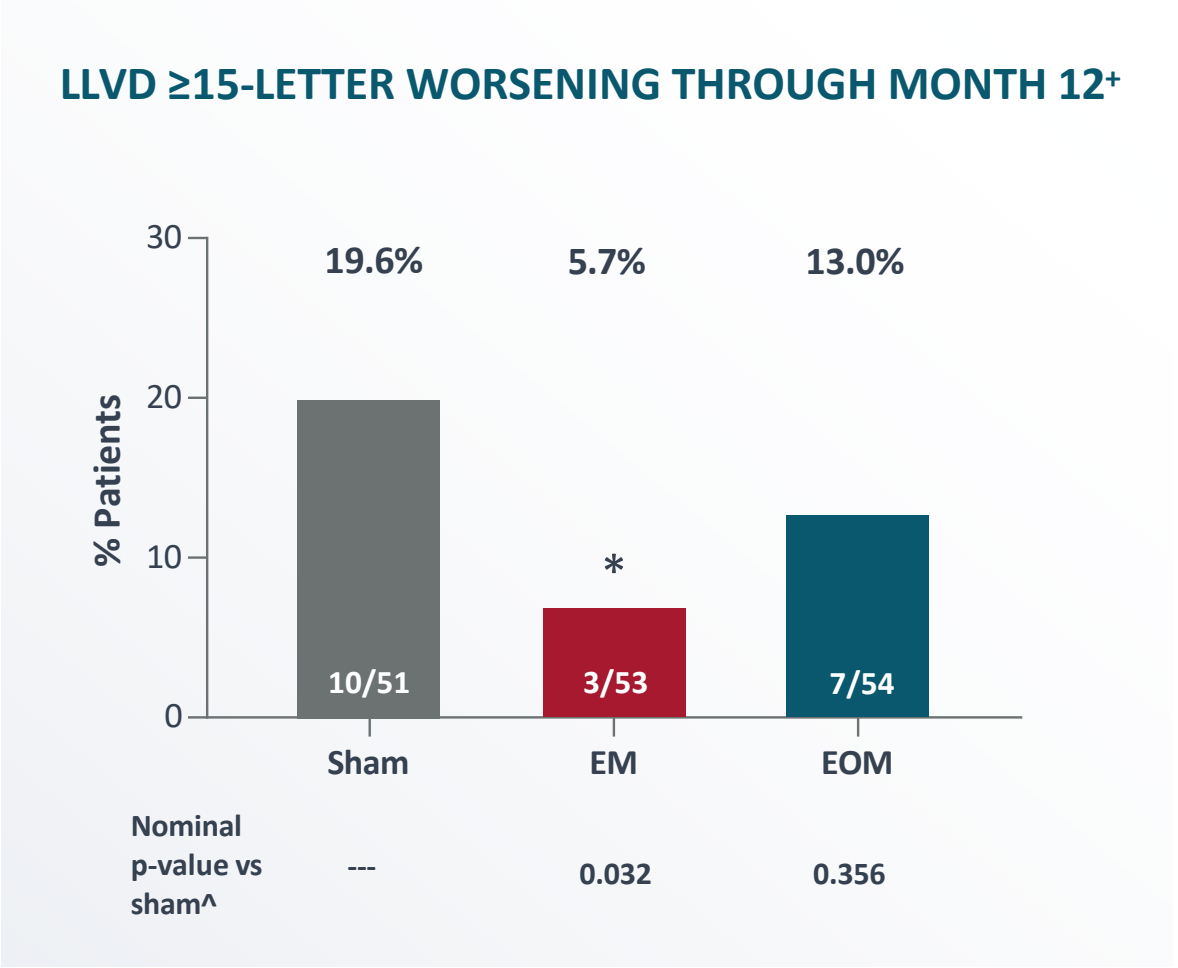
ANX007 Provided Consistent Protection from Vision Loss by LLVD



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

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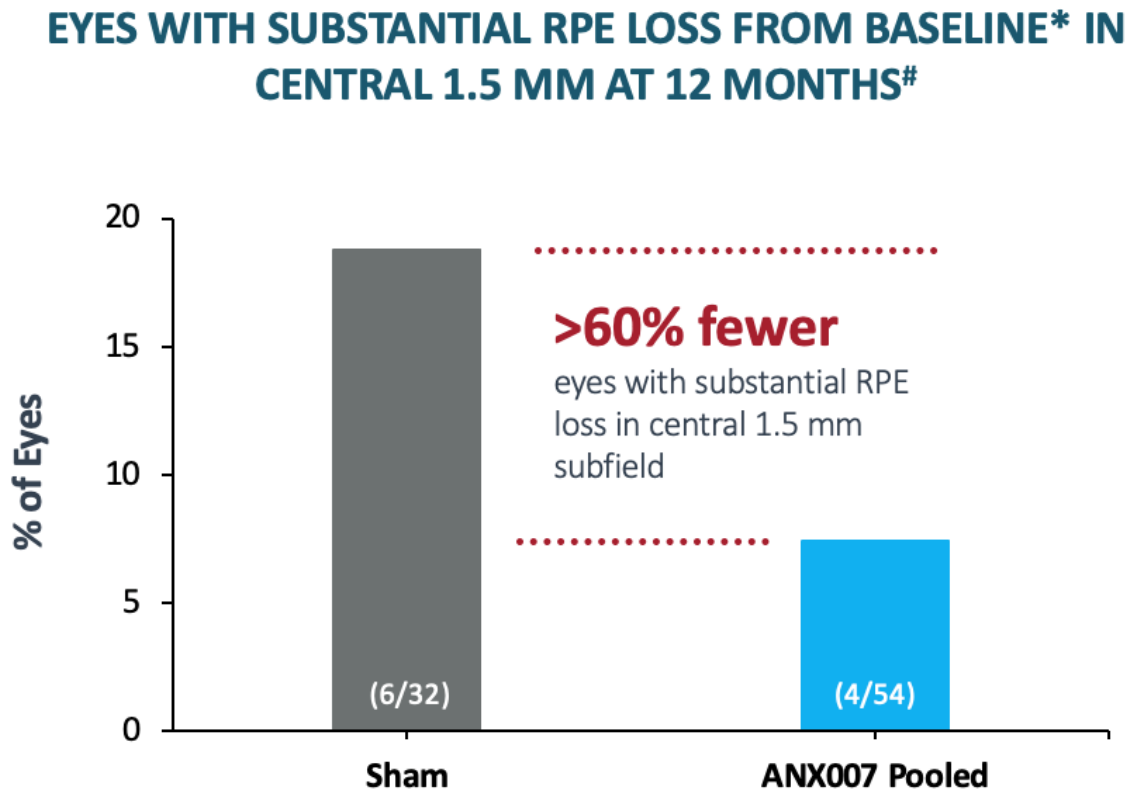
In Patients with Foveal Center RPE Remaining, ANX007 Reduced Patients Experiencing Substantial RPE Loss by 60%



#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

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ANX007 1st & Only Recipient of PRIME Designation - Best-in-Class Potential By Disconnecting Lesion Growth Surrogate from Vision Preservation

FDA Alignment on BCVA ≥15-Letter Loss as Primary Outcome Measure

No FDA requirement to study slowing of GA lesion growth by FAF

Program to include comparison to an injection agent of choice, consistent with trials across ophthalmic indications

PRIME Designation Granted in EU

"The unmet need in Geographic atrophy (GA) secondary to age-related macular degeneration (AMD) is agreed. The potential to address the unmet need relies on the Phase 2 clinical data and effects on visual function at 12 months…**the consistent effects on visual function across measures, analyses and subgroups indicated a potential to address the unmet need**."

– European Medicines Agency

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Next Wave Programs

Promising Next Wave Programs in Development Provide Optionality

HUNTINGTON'S DISEASE

80K patients globally

No approved treatments

ANX005 Ph2a Completed

- ✓ Rapid and sustained target engagement
- ✓ Reduction in markers of neuroinflammation
- ✓ Improved clinical function

Poised for late-stage Phase 2/3 development

ALS

~200K patients globally

Current approved treatments offer modest benefit or benefit in small patient segment (SOD1 - ~2%)

ANX005 Phase 2a Completed

- ✓ Generally well tolerated
- ✓ Rapid, sustained target engagement
- ✓ Reduced downstream PD complement markers
- ✓ Achieved better outcomes in patients with higher baseline classical complement activity

Poised for late-stage Phase 2/3 development

ALS: Amyotrophic Lateral Sclerosis