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STOP THE START of classical complement-driven diseases

Topline Results from Phase 3 Study of ANX005 in Guillain-Barré Syndrome

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.

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 early stages of clinical development of our product candidates; the effects of COVID-19 or other public health crises on our clinical programs and business operations; our ability to obtain regulatory approval of and successfully commercialize our product candidates; any undesirable side effects or other properties of our product candidates; our reliance on third-party suppliers and manufacturers; the outcomes of any future collaboration agreements; and our ability to adequately maintain intellectual property rights for our product candidates. These and other risks are described in greater detail under the section titled “Risk Factors” contained in our Quarterly Report on Form 10-Q filed with the Securities Exchange Commission (SEC) on May 13, 2024 and our other filings with the SEC from time to time. All forward-looking statements in this presentation speak only as of the date of this presentation. Except as required by law, we undertake no obligation to publicly update any forward-looking statements, whether as a result of new information, future events or otherwise.

This presentation concerns drug candidates that are under clinical investigation, and which have not yet been approved for marketing by the U.S. Food and Drug Administration (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.

Pursuing an Intentional and Rigorous Approach to Tackling an Array of Classical Complement-Mediated Diseases

Poised for a transformational 2024 and beyond

Stopping the Start of Classical Pathway Neuroinflammation

Broad Therapeutic Potential of Late-Stage Clinical Platform

Significant Near Term Value Creating Catalysts

ON A PATH TO HELPING PATIENTS GET THEIR LIVES BACK

Well-supported MOA demonstrated differentiated functional outcomes across GBS, GA, HD and ALS

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)

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

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ANX005 Achieved a Breakthrough Phase 3 Win for GBS Patients Worldwide

A single infusion demonstrated consistent benefit across multiple endpoints meaningful to patients

Met Primary Endpoint P=0.0058	Expedited Recovery Patients Got Better Sooner	Durable Treatment Effect	Generally Well Tolerated
2.4-fold higher likelihood of being in a better state of health on GBS-DS at Week 8	**Early, robust & clinically meaningful benefit on multiple outcome measures @ Week 8**	**Maintained improvement over placebo at all timepoints across multiple measures**	**Safety data was similar to placebo**
✓ FDA-agreed primary endpoint ✓ Consistent, significant results from multiple pre-specified analyses ✓ Larger effect in sub-group with western baseline characteristics	✓ Able to walk earlier vs placebo ✓ Able to run earlier vs placebo ✓ Less nerve damage vs placebo	✓ Less time on ventilation ✓ Less overall disability	✓ No new safety signals ✓ No increased infection rate ✓ No difference in all-cause mortality

Topline Results Subject to Change

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GBS: Neurological Emergency with Long-Term Disability; Requires an Immediately Targeted and Effective Intervention

POST-INFECTIOUS COMPLEMENT-MEDIATED DISEASE

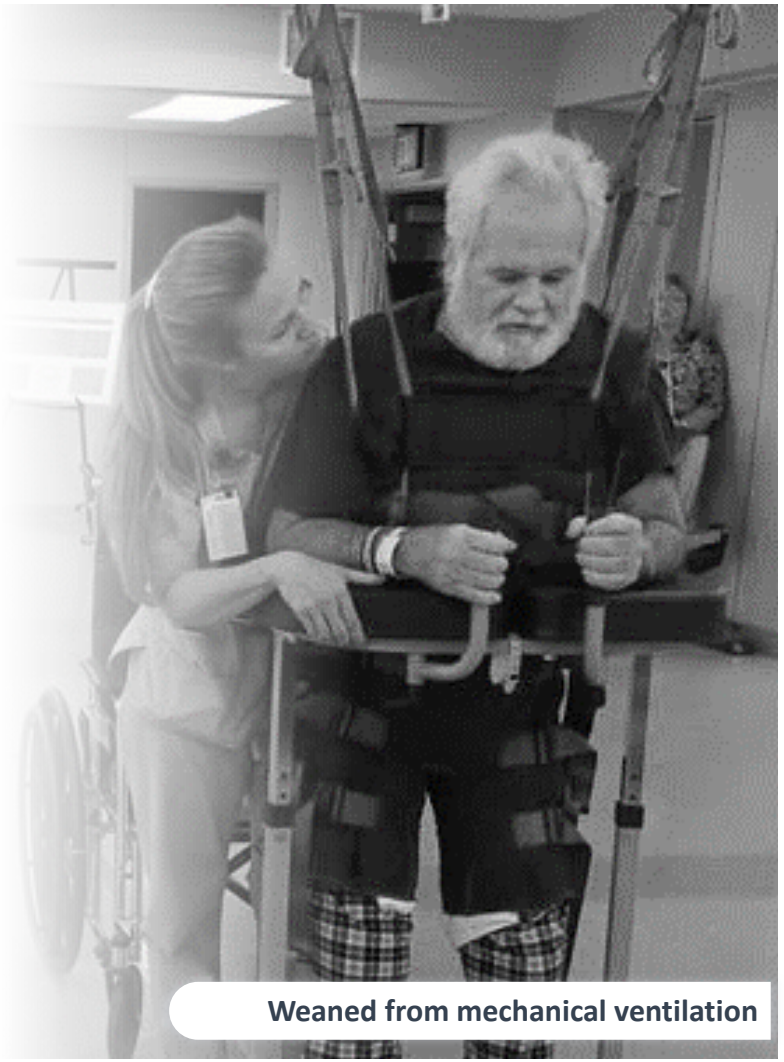
- Following infection, complement-activating autoantibodies attack nerves leading to nerve damage & acute paralysis
- Can happen to anyone, anytime, anywhere

HIGH UNMET MEDICAL NEED

- 22,000 patients hospitalized in US & Europe every year
- IVIg not FDA approved, unknown MOA, requires 5-day treatment

SIGNIFICANT MORBIDITY

- Notwithstanding IVIg treatment, GBS results in:
  - Severe weakness and paralysis
  - Mechanical ventilation in 25% of patients
  - Extensive nerve damage causing uncertain and incomplete recovery



MOA: mechanism of action

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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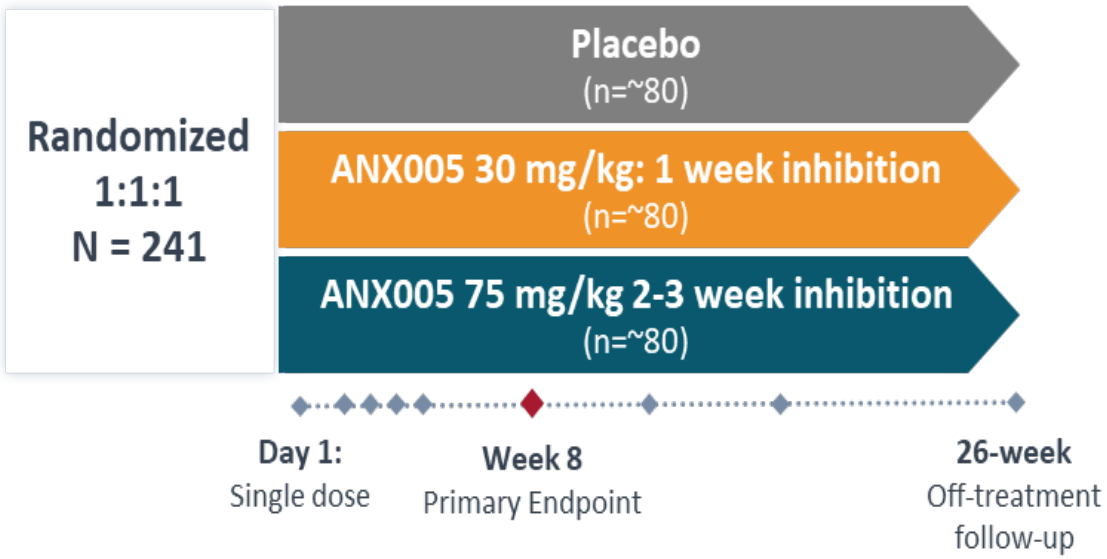
Well Designed & Executed Pivotal Trial Showed Clear Results

ANX005 for GBS Granted FDA Fast Track and FDA / EMA Orphan Drug Designation



TWO DOSES SELECTED TO DETERMINE MOST EFFECTIVE DURATION OF INHIBITION

STUDY DESIGN

- Baseline GBS-DS score 3-5
- GBS diagnosed <10 days from onset of weakness
- Patients stratified for baseline prognostic factors: muscle strength and time from onset of weakness
- Conducted in Bangladesh and Philippines given high prevalence of GBS of all types, scientific leadership in GBS, and limited access to IVIg

KEY ENDPOINTS

- **Primary Outcome Measure:** GBS-DS[1] at week 8: well-accepted regulatory endpoint assessing functional status
- **Secondary Endpoints:** Muscle strength (MRC sumscore), Motor Disability (ONLS), Duration of Ventilation, and others

[1]Analyzed using a proportional odds model, a common statistical analysis method (van Leeuwen, et al., 2019, doi.org/10.1371/journal.pone.0211404)

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Baseline Characteristics Generally 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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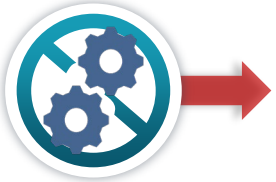
ANX005 30 mg/kg Treated Patients Had Significant, Rapid and Sustained Improvement Across Multiple GBS Measures

Achieved Goal of Helping Patients GET BETTER SOONER

✓ Rapid & Complete Inhibition of classical complement cascade	✓ Statistically Significant[1] Early Reduction in NfL	✓ Statistically Significant Higher Odds of Being Better at Week 8 (Primary Endpoint)	✓ Statistically Significant[1] Early & Sustained Improvement in Muscle Strength	✓ Statistically Significant[1] Less Time on Ventilator over entire study period
				
C1q	NfL	GBS-DS	MRC Sumscore	Ventilation

[1]nominal p-value

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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

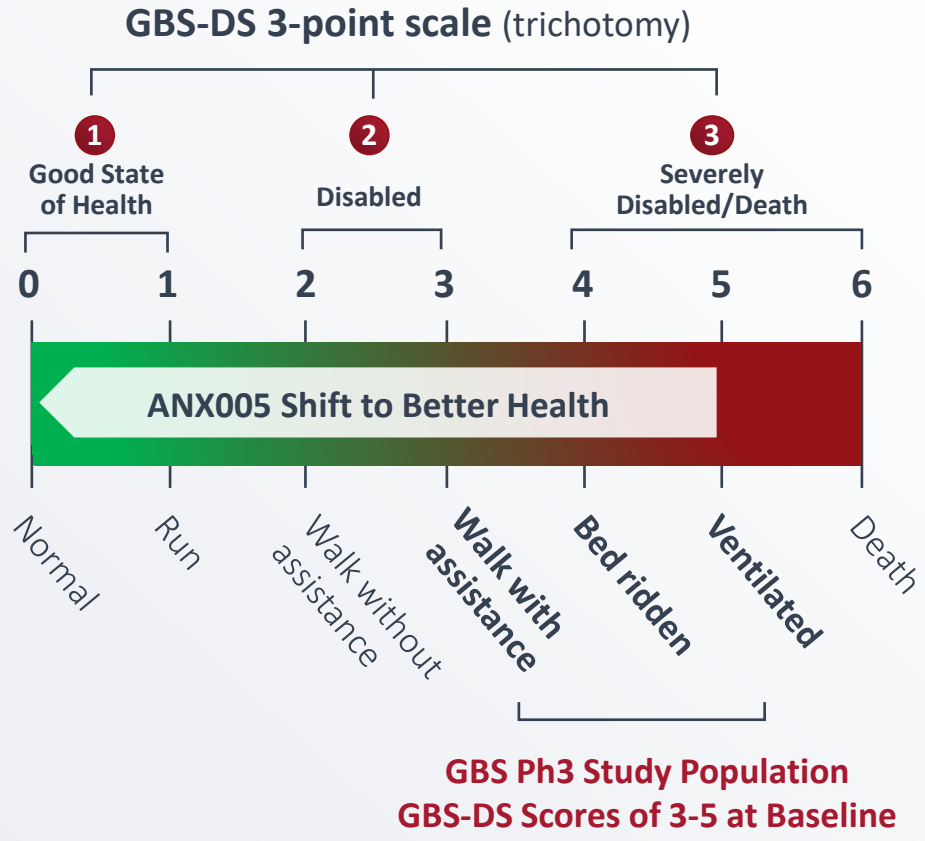
Approach: Collapse 7-grade scale to a 3-grade scale (trichotomy)

- **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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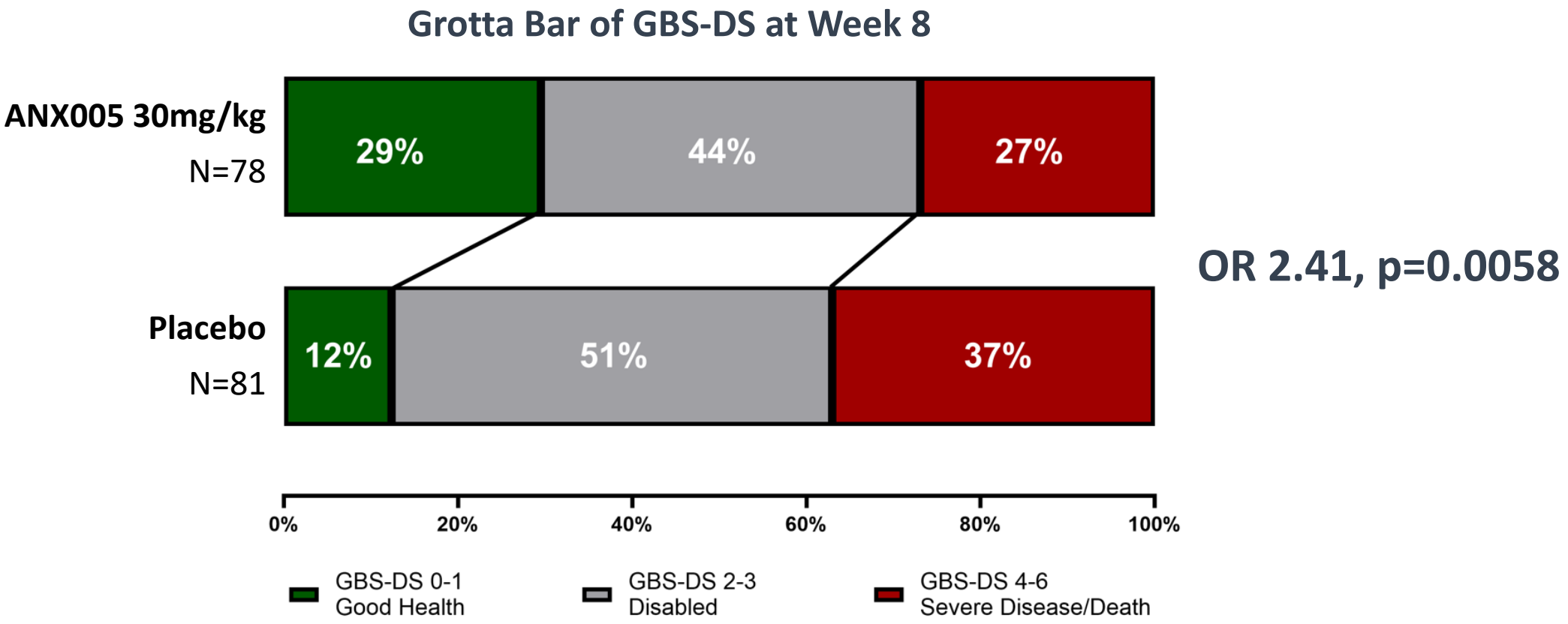
ANX005 30 mg/kg Showed Highly Significant and Clinically Meaningful Treatment Effect on GBS-DS at Week 8 (Primary Endpoint)

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



OR 2.41, p=0.0058

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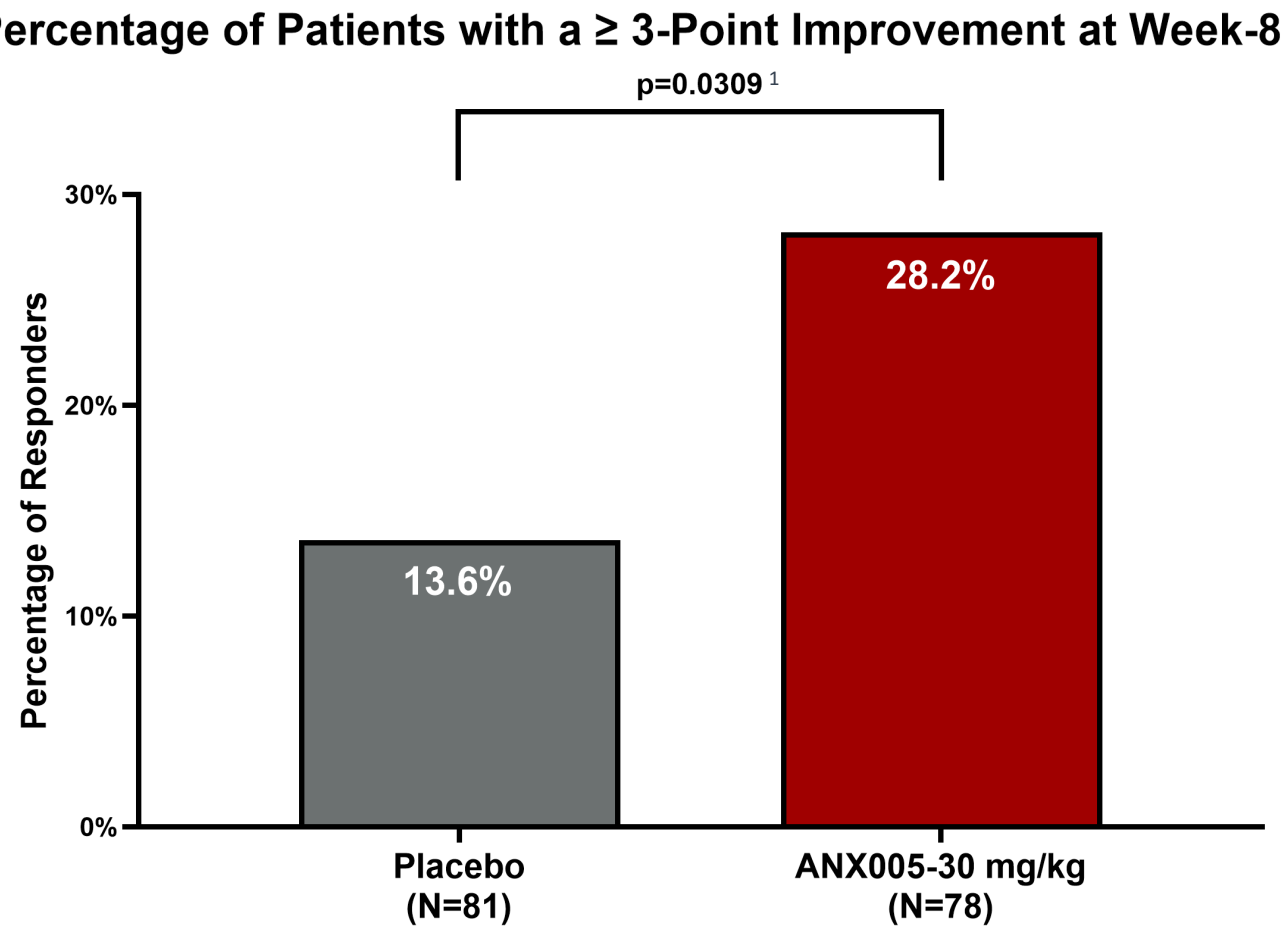
Pre-Specified GBS-DS Responder Analysis at Week 8: ANX005 30 mg/kg Demonstrated a Significant ≥3-Point Improvement vs. Placebo

Substantial treatment effect at week 8, further supporting primary analysis



[1]nominal p-value

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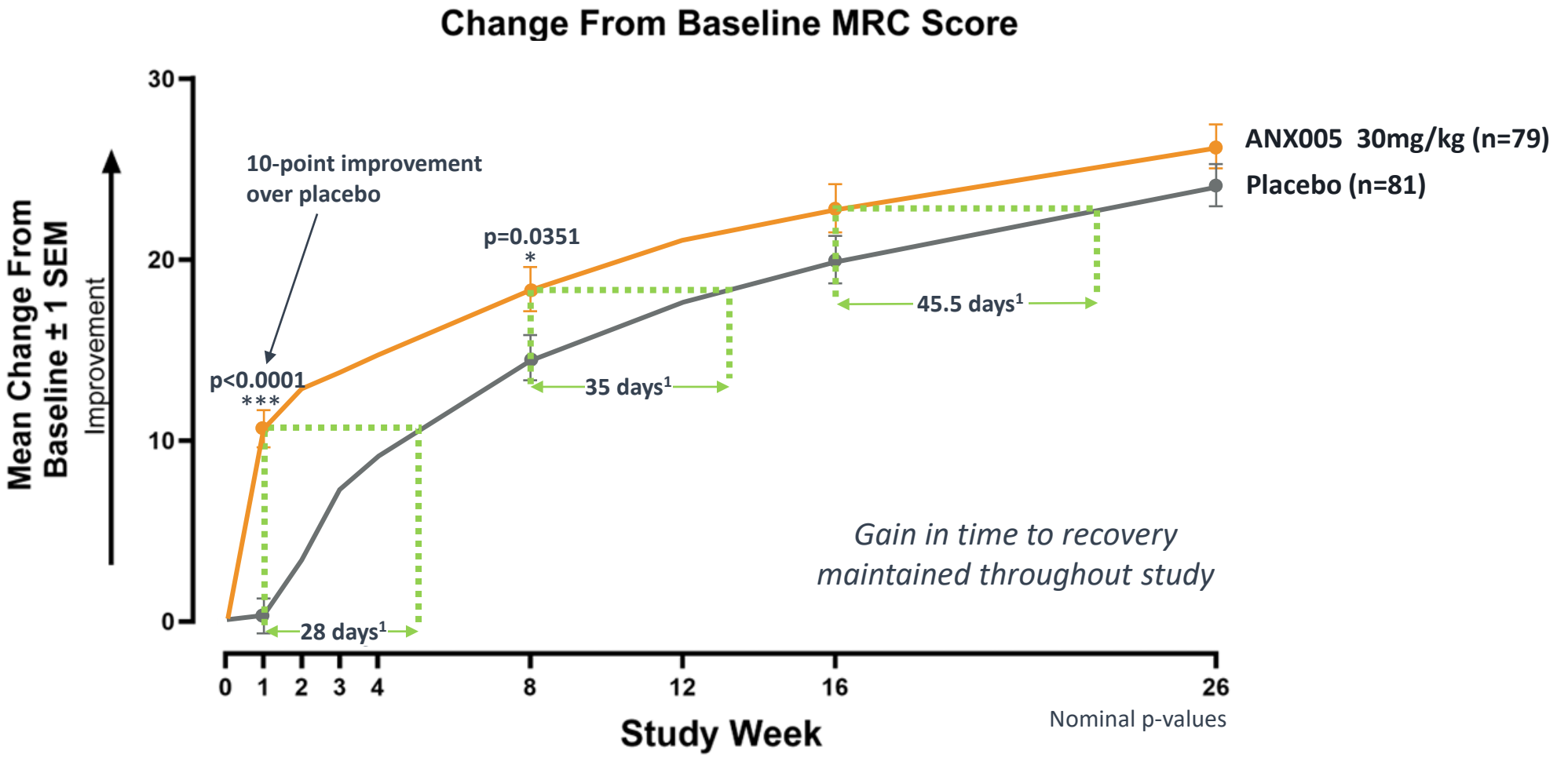
Getting Better Sooner: ANX005 30 mg/kg Increased Muscle Strength Earlier Relative to Placebo, and the Advantage Grew Over Time

Early muscle strength improvement maintained & increased through full 26-week study (p=0.001)



Modified Intent-to-Treat (N=241)

[1]Approximate Time difference

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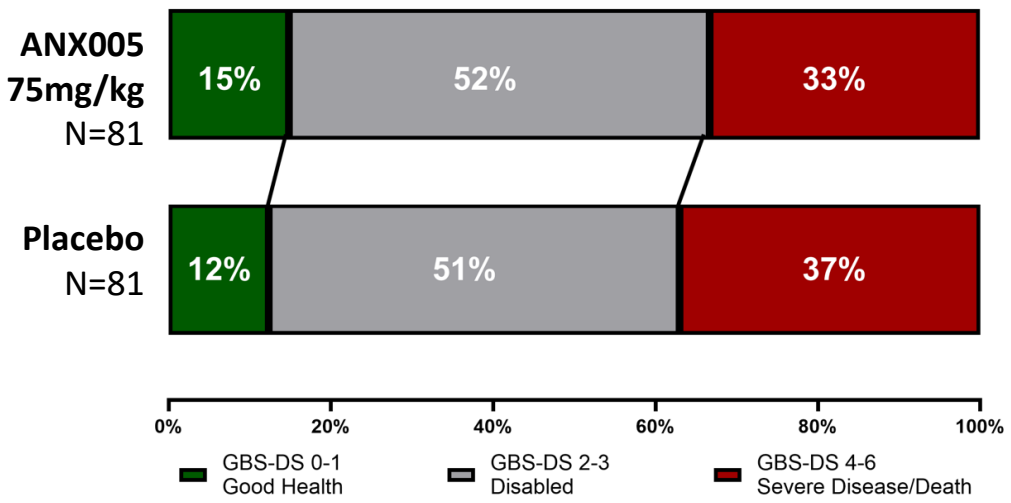
ANX005 75 mg/kg Did Not Meet the Primary Endpoint with Inhibition Beyond Active Disease Process

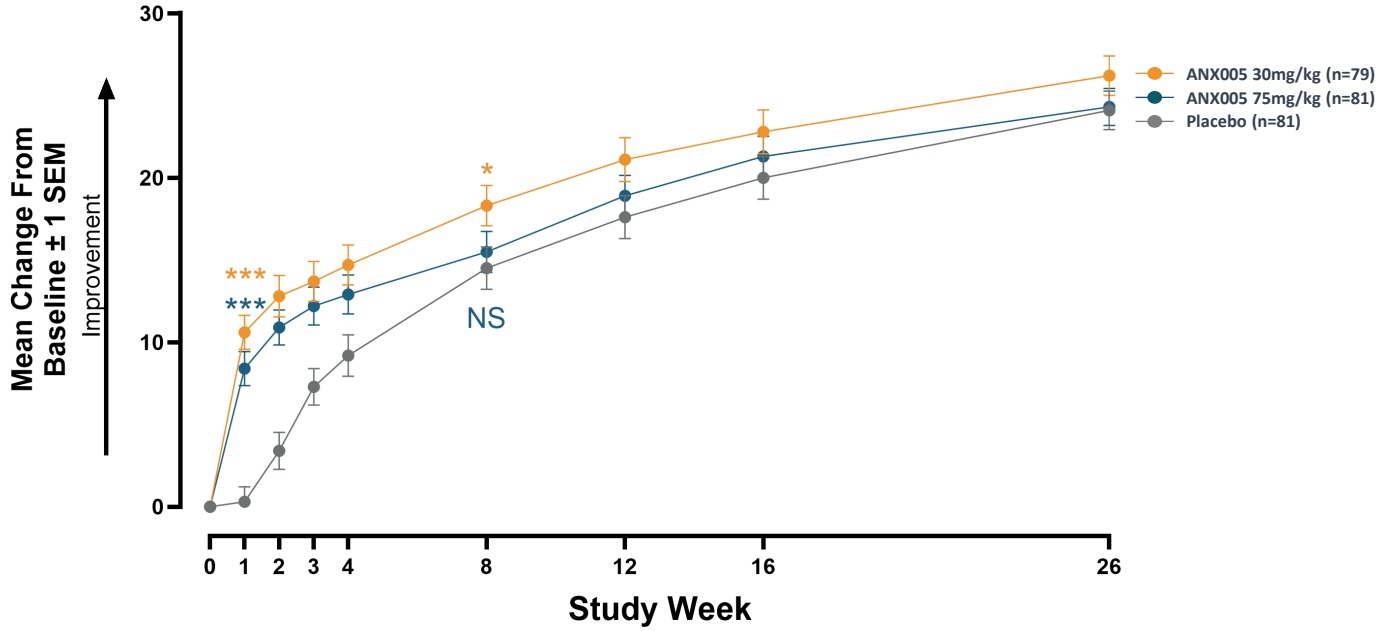
75mg/kg did improve muscle strength similar to 30 mg/kg at early timepoints to week 4

Grotta Bar of GBS-DS at Week 8



OR 1.20, p=0.5548

Change from Baseline MRC Score



Modified Intent-to-Treat (N=241) Nominal p-values

Topline Results Subject to Change

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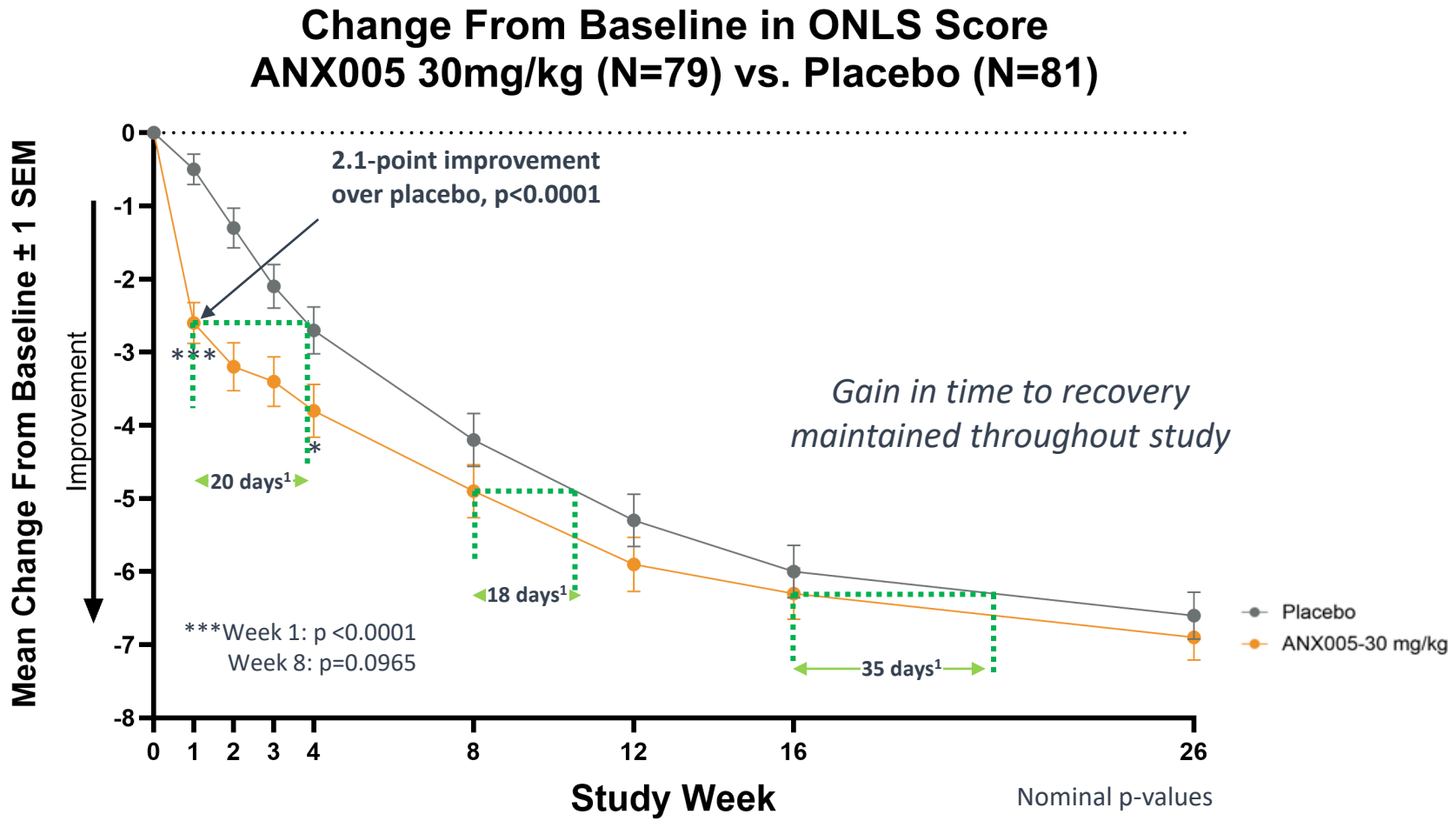
Getting Better Sooner: ANX005 30 mg/kg Showed Significant Early Improvement in Motor Disability vs. Placebo on the ONLS* Scale

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



*Overall Neuropathy Limitation Scale
[1]Approximate Time difference

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Getting Better Sooner: ANX005 30 mg/kg Consistently Showed Faster Recovery Across Clinically Important Measures Relative to Placebo

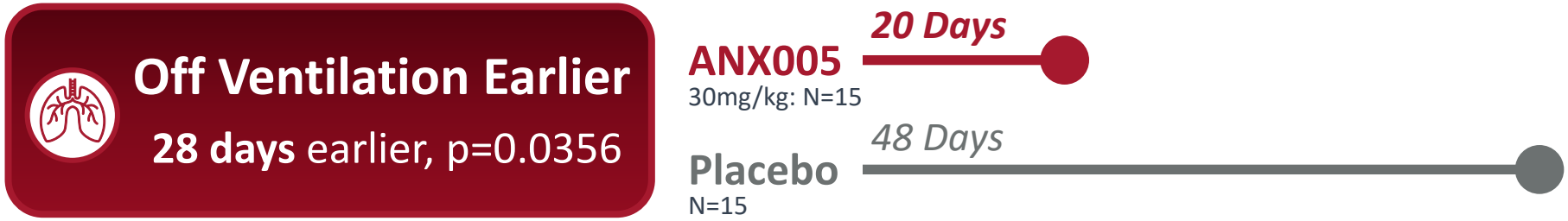
Helping patients achieve their independence sooner

Walking Earlier

31 days earlier, p=0.0211

Off Ventilation Earlier

28 days earlier, p=0.0356



Nominal p-values

Topline Results Subject to Change

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Through Week 26, ANX005 30mg/kg-Treated Patients had Better Outcomes Relative to Placebo

ANX005 treatment demonstrated better outcomes than placebo at all time points in the study

Through Week 26, Patients Treated with ANX005 30mg/kg Were More Likely to:

Be in a better state of health	**GBS-DS: 1.49x** more likely of being better p=0.012
Have more muscle strength	**MRC: 5.4** mean point improvement p=0.0010
Perform daily tasks better	**ONLS: 1.1** mean point improvement p=0.0063

Nominal p-values

GBS Phase 3 Results are Highly Relevant to Western Populations

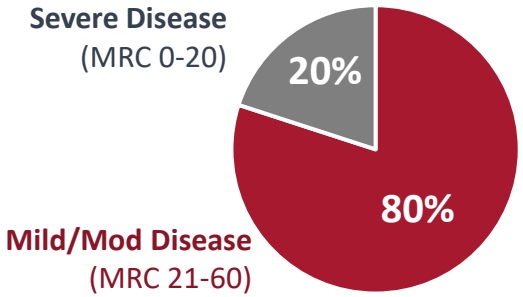
ANX005 30 mg/kg treatment effect more pronounced in Western World-type patients

Western patients tend to have less severe disease and an AIDP neurotype

ANX005 30 mg/kg impact on patients with these characteristics

Disease Severity

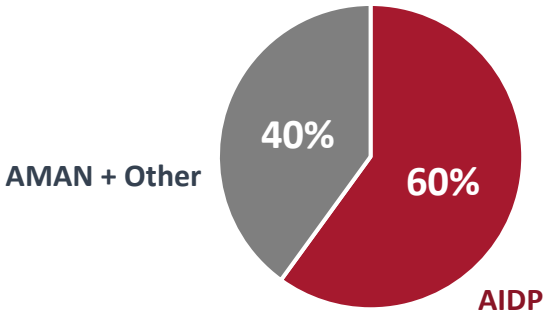
US/Europe



80% of US/European patients have baseline **MRC of >20 points**

3.03x more likely to be in a better state of health at **Week 8**
p=0.0102
(47% of patients had milder disease)

Neurotype



60% of US/European patients have AIDP

5.31x more likely to be in a better state of health at **Week 8**
p=0.0130
(21% of patients had AIDP)

Nominal p-values

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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
- Infection rates were comparable across dose groups and consistent with typical hospital acquired infections
- 3 patients had treatment discontinuations
  - 1 in each dose group

Deaths

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

	Placebo N=81		ANX005 30mg/kg N=79		ANX005 75mg/kg N=81	
	All Grades	Grade ≥ 3	All Grades	Grade ≥ 3	All Grades	Grade ≥ 3
Number of Subjects Reporting TEAEs, n (%)	79 (97.5)	35 (43.2)	79 (100.0)	33 (41.8)	80 (98.8)	36 (44.4)
Number of Subjects with Infusion Related Reaction	4 (4.9)	1 (1.2)	24 (30.4)	4 (5.1)	32 (39.5)	7 (8.6)
Rash (most common with IRR)	2 (2.5)	0	20 (25.3)	1 (1.3)	25 (30.9)	2 (2.5)
Most Common TEAEs (non-IRR), n (%)						
Blood CPK Increased	46 (56.8)	16 (19.8)	44 (55.7)	14 (17.7)	35 (43.2)	12 (14.8)
Musculoskeletal Pain	35 (43.2)	0	36 (45.6)	0	26 (32.1)	1 (1.2)
ALT Increased	23 (28.4)	6 (7.4)	21 (26.6)	2 (2.5)	23 (28.4)	6 (7.4)
Urinary Tract Infection	18 (22.2)	6 (7.4)	19 (24.1)	5 (6.3)	18 (22.2)	1 (1.2)
Hypokalemia	24 (29.6)	8 (9.9)	16 (20.3)	4 (5.1)	11 (13.6)	3 (3.7)
Constipation	10 (12.3)	0	15 (19.0)	0	17 (21.0)	0
AST Increased	16 (19.8)	3 (3.7)	11(13.9)	1 (1.3)	17 (21.0)	3 (3.7)

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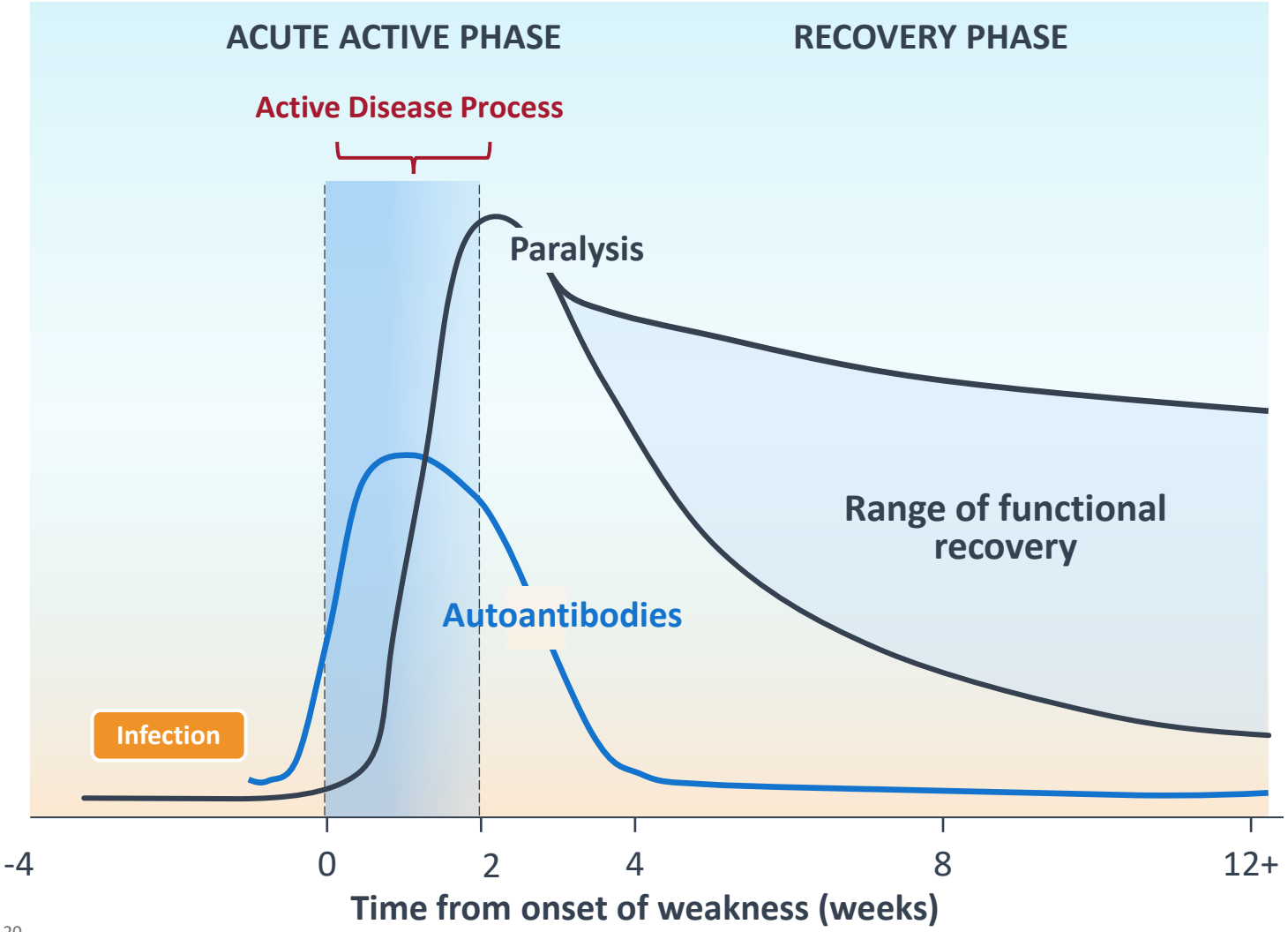
GBS Pathophysiology and the Targeted MOA of ANX005

GBS is a Neurological Emergency Requiring Urgent Intervention

Limited time window to stop the active disease process and achieve a therapeutic effect



ACUTE ACTIVE PHASE

- Rapidly progressive bilateral muscle weakness peaking by 1 – 2 weeks in most cases
- Paralysis in legs, arms and potentially breathing muscles
- Extended periods of ventilation in ICU, and intensive supportive care

RECOVERY PHASE

- Gradual muscle strength and functional improvement over months to years as nerve regeneration takes place
- ~20% unable to walk or dead at 1 year and additional 20% continue to experience symptoms

*Adapted from van den Berg, et al. (2014) Nat Rev Neurol **10**, 469–482*

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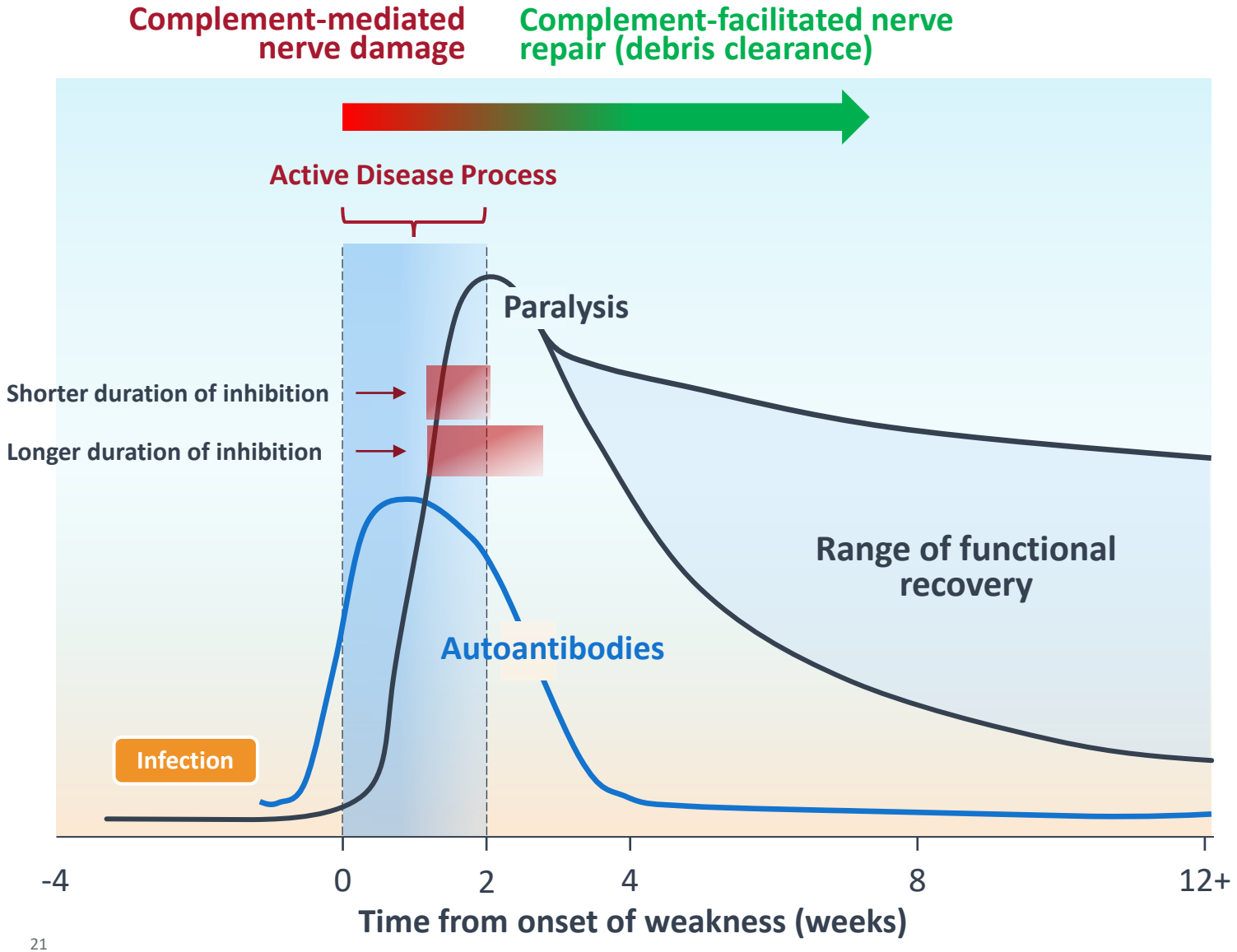
GBS Time Course: Autoimmune Complement-Mediated Nerve Damage Followed by Normal Complement-Facilitated Repair



IDENTIFY MOST EFFECTIVE TREATMENT WINDOW

BLOCK AUTOIMMUNE COMPLEMENT-MEDIATED NERVE DAMAGE DURING ACTIVE DISEASE

ALLOW NORMAL COMPLEMENT FACILITATED NERVE REPAIR DURING RECOVERY PHASE

*Adapted from van den Berg, et al. (2014) Nat Rev Neurol **10**, 469–482*

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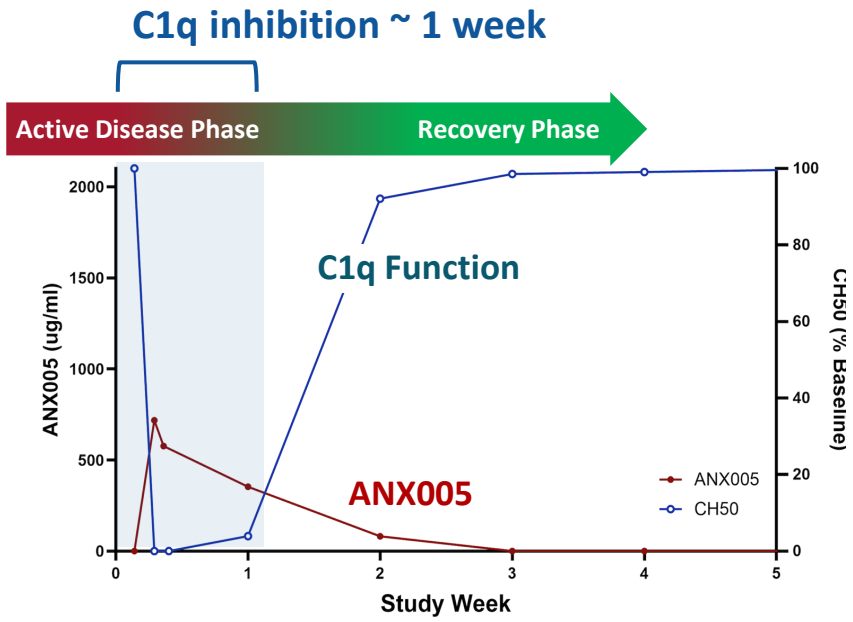
ANX005: Expected Pharmacokinetic and Dynamic Response for Both Doses

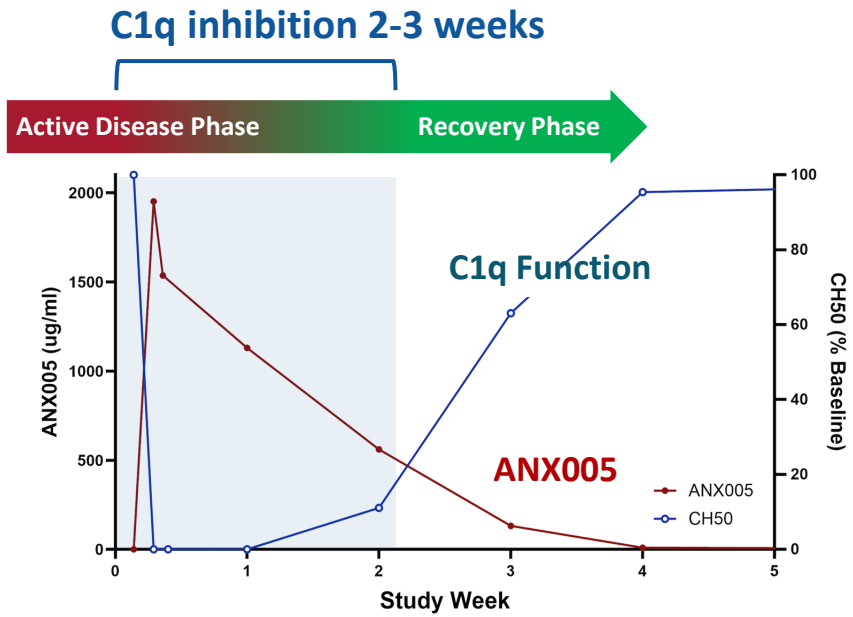
Duration of complement inhibition defines active treatment window

- **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



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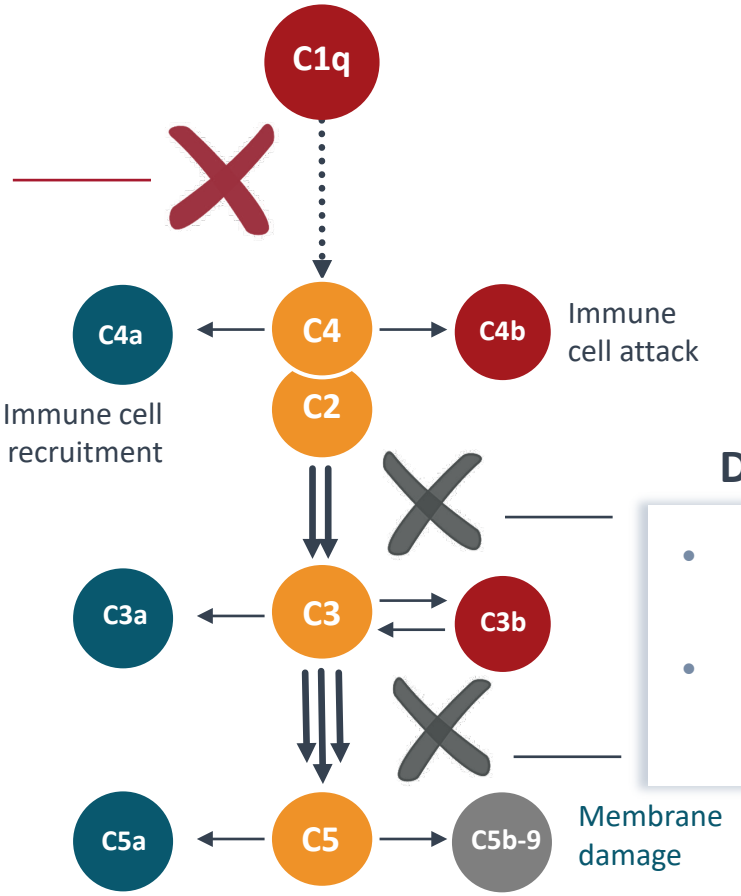
ANX005 Rapidly Shuts Down Activation of the *ENTIRE* Classical Complement Cascade on the Nerve to Prevent Acute Injury

Classical Complement Drives Harmful Inflammation and Tissue Destruction

STOPPING AT THE START

- Blocks upstream and downstream 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

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

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ANX005 GBS Phase 3 Trial Summary and Path Forward

Douglas Love, President & CEO
Annexon Biosciences

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 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**
- ✓ **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

ANX005 GBS Phase 3 Summary of Key Results

A profound moment for the GBS community – first targeted therapy to demonstrate positive outcomes

1 **Phase 3 Met Primary Endpoint, confirming earlier study**

GBS-DS at Week 8: Patients treated with ANX005 were 2.4 times more likely to be in a better state of health compared to placebo, p=0.0058

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

Early, robust, and clinically meaningful benefit on multiple outcome measures by week 8 including ability to walk earlier and less nerve damage vs. placebo

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

Maintained improvement over placebo at all timepoints across multiple measures including less time on ventilation and less overall disability

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

To our employees, collaborators and advisors,
thank you for your WARRIOR SPIRIT AND ALL FOR ONE COMMITMENT!

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Thank You!
Q&A

Annexon Biosciences sincerely thanks all the patients, families, and study staff who are helping make the ANX005 Ph3 GBS study possible.

GBS|CIDP Foundation International

IGOS

icddr,b

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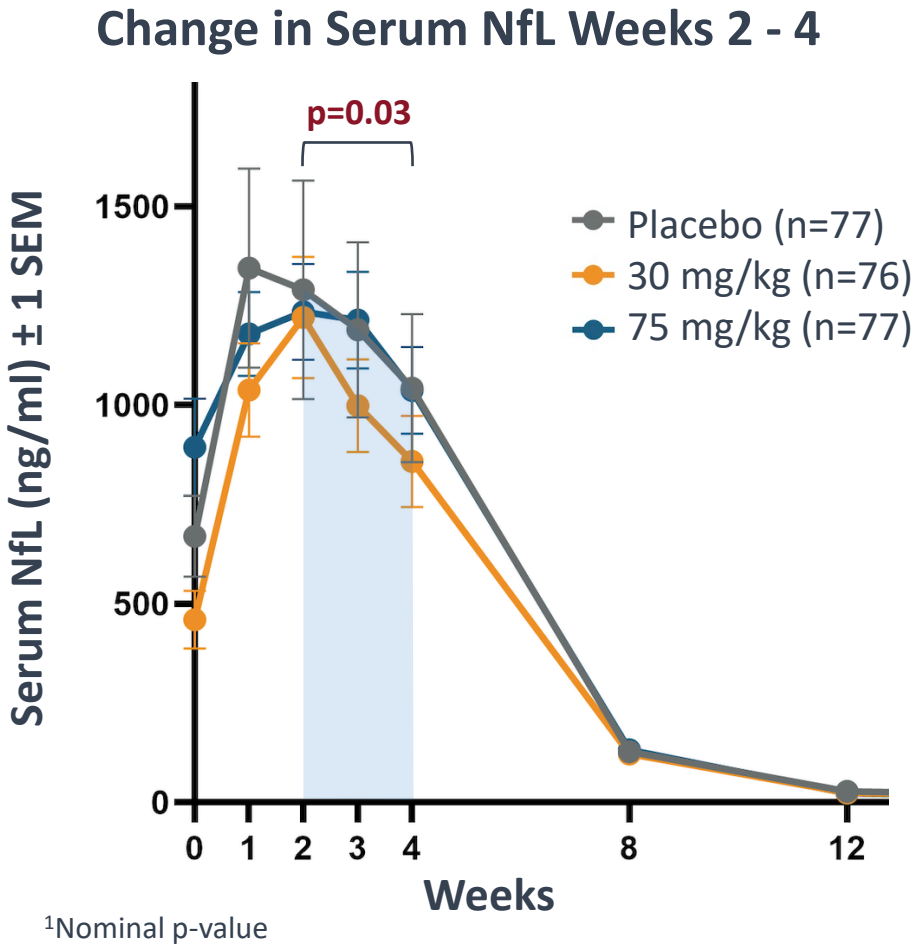
Appendix

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

Assessment of reduction of neuronal damage



[1]Nominal p-value

Key Takeaways

- Prespecified assessment of NfL reduction during weeks 2-4 consistent with Ph1b
- Captures transition from acute progressive to recovery phase of disease
- **ANX005 30 mg/kg achieved significant early reduction in NfL between weeks 2 – 4 vs. pbo** (31.3% vs. 20.1%, p=0.03[1])

Complement is Pivotal Force in Driving Nerve Damage in GBS

ANX005 is a Targeted Immunotherapy which Rapidly Blocks Complement

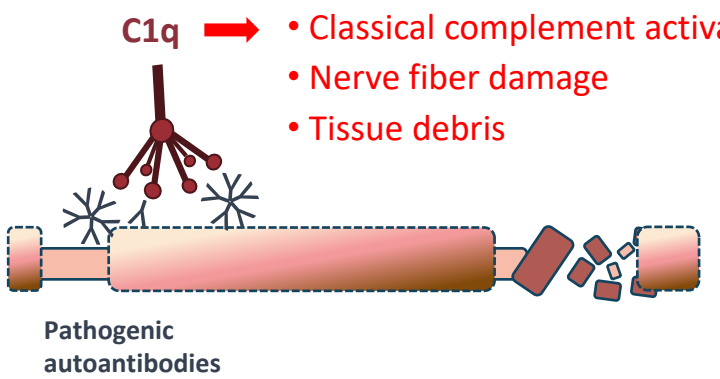
GBS

- C1q binds to autoantibodies on nerve surface
- Activates classical complement pathway
- Results in neuroinflammation, nerve damage, tissue debris and paralysis

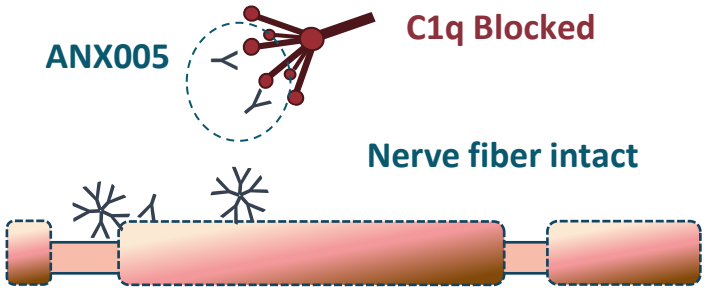
ANX005

- One dose of ANX005 rapidly blocks C1q
- Stops activation of entire classical pathway
- Blocks nerve fiber damage during the active disease phase

Peripheral Nerve Fiber Under Attack in GBS



Peripheral Nerve Fiber in GBS with ANX005



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

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Phase 3 Comparison of Eculizumab vs. ANX005

	Eculizumab Ph3 GBS Trial	ANX005 GBS Ph3 Trial
MOA	Targets downstream complement (C5) - misses important upstream complement drivers of nerve damage	**Blocks entire classical complement cascade**
Mean time from onset of weakness to treatment	>7 days	**< 7 days***
N	57	**241**
Stratification by prognostic factors	Not stratified leading to imbalance	**Stratified**

*Stratified for days since onset of weakness (<7 days, ≥7 days)

ANX005 Has Demonstrated Characteristics Required to Combat GBS

✓ Directly targets mechanism driving extensive nerve damage and paralysis

- Complement is an established target in GBS
- C1q binds to autoantibodies on nerve components initiating local activation of complement leading to inflammation, recruitment of immune cells, and damage to nerves

✓ Rapid onset of action

- ANX005 has demonstrated rapid target engagement in blood & CSF across multiple central and peripheral neurological disorders
- A single dose of ANX005 inhibits classical complement pathway on day 1
- Prevents acute and ongoing nerve damage to promote nerve repair

✓ Provides clinical benefit across entire disease spectrum

- Complement-mediated nerve destruction present in all neurotypes of GBS
- ANX005 mechanism of action is agnostic to neurotype or disease severity
- Early improvement in MRC seen across disease spectrum

✓ Minimal side-effects

- ANX005 has been safely administered in > 250 patients with GBS
- Generally well-tolerated
- No drug-related deaths & no serious infections observed

The Phase 3 Study Embodies Key Characteristics of a Smart, Data-Driven, & Patient-Centric Design

HOW I WOULD DESIGN A PH3 GBS STUDY	HOW ANNEXON DESIGNED THE PHASE 3 PIVOTAL STUDY
Use all available global data and routinely seek expert input	✓ Data-driven by Ph1b, IGOS, and multiple external IVIg/PE datasets ✓ Routinely engaged with leading experts in GBS
Measures all meaningful outcomes through all phases of disease	✓ Proportional odds uses full GBS-DS scale, includes all patients, increases power ✓ Efficacy assessments cover all GBS symptoms & signs at all important timepoints
Control for disease heterogeneity	✓ Patients stratified by baseline MRC and days since onset of GBS symptoms ✓ Using MRC, time of onset of weakness, baseline NfL and age as covariates
Rigorous execution	✓ Streamlined time from onset to treatment increasing likelihood of better outcomes ✓ Conducted at sites with internationally recognized GBS clinical experience

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