

Global Science. One Purpose.

Corporate Presentation

August 2026



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Immune Remodulation Through BAFF/APRIL Inhibition

OUR AMBITION

To Transform the Approach to B Cell-Driven Autoimmune Disease

TELITACICEPT

Selective BAFF/APRIL inhibitor designed to reduce pathogenic B cells and antibodies while preserving immune protection

Clinically validated in 8+ autoimmune indications in China

Manageable safety and tolerability in tens of thousands of patients

LEAD AUTOIMMUNE PROGRAMS

Myasthenia Gravis: Global Phase 3 topline data in 1H27

Sjögren's Disease: Global Phase 3 enrollment ongoing; first patient dosed in 1Q26

NEAR-TERM EXPANSION OPPORTUNITIES

Broad applicability across B cell-mediated autoimmune diseases

~\$515M WITH RUNWAY INTO EARLY 2029*, FUNDED THROUGH KEY MILESTONES



Dual BAFF/APRIL Inhibition Targets Autoreactive B Cells and Plasma Cells

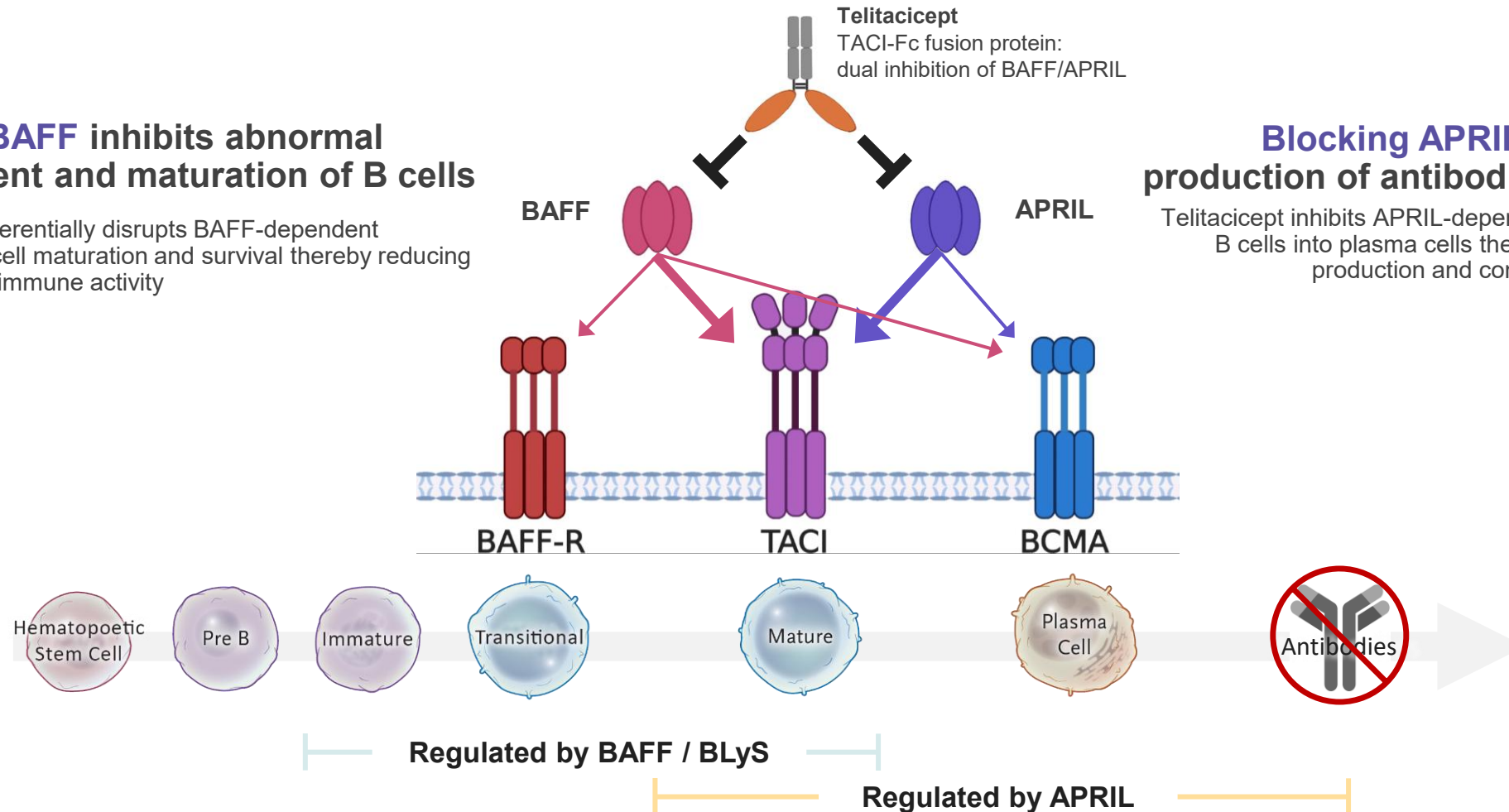
Disease modification through upstream control of B cell survival and downstream antibody production

Blocking BAFF inhibits abnormal development and maturation of B cells

Telitacicept preferentially disrupts BAFF-dependent autoreactive B-cell maturation and survival thereby reducing disease-driving immune activity

Blocking APRIL inhibits abnormal production of antibodies by plasma cells

Telitacicept inhibits APRIL-dependent differentiation of mature B cells into plasma cells thereby minimizing autoantibody production and contributing to control of disease



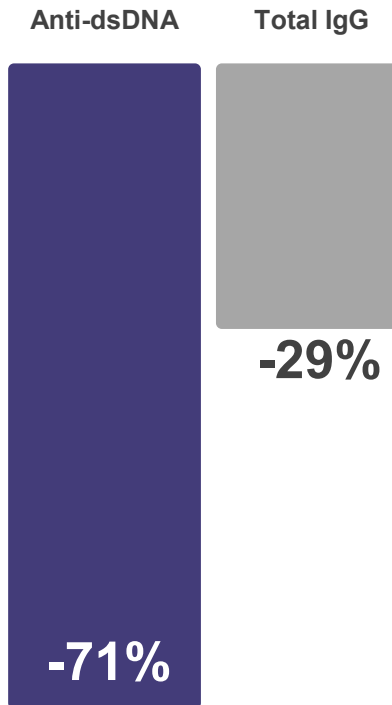
TELITACICEPT PREFERENTIALLY DEPLETES PATHOGENIC CELLS WHILE PRESERVING HUMORAL IMMUNITY

Pathogenic autoantibodies fell ~1.9-2.4x more than total IgG pool in SLE and MG patients treated with telitacicept^{1,2}

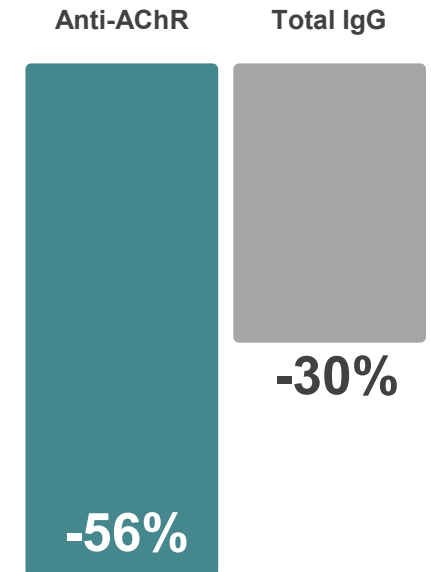
Telitacicept removes BAFF/APRIL survival signals that pathogenic long-lived plasma cells depend on without depleting the immune system^{1,3}

Functional B cell reservoir were retained with CD4 T cell immunity unaffected, protective IgG pool largely spared, and a proportion of regulatory B cells restored to healthy-control levels^{1,3}

SYSTEMIC LUPUS
ERYTHEMATOSUS (SLE)
(n= 20 | 24 Weeks)¹



MYASTHENIA
GRAVIS (MG)
(n= 22 | 24 Weeks)²



Established Efficacy in China Across Autoimmune Diseases

5

Commercial Approvals

Validated Commercial Therapy in China Across Diverse Autoimmune Diseases

2021 - Systemic Lupus Erythematosus (SLE)[†]

2024 - Rheumatoid Arthritis (RA)

2025 - Myasthenia Gravis (MG)

2026 – Sjögren's Disease (SjD)

2026 – IgA Nephropathy (IgAN)*

3

Best-In-Disease

Unique Dual BAFF/APRIL Inhibition Drives Superior Clinical Benefit

Systemic Lupus Erythematosus

Myasthenia Gravis

Sjögren's Disease



Favorable Safety At Scale

10s of
Thousands

Patients Treated
Commercially in China

Favorable and Predictable Safety Profile Observed
Among ~2,400* Patients Studied in Clinical Trials



**No Burdensome
Vaccination Requirements**



**No Signature B Cell
Depletion Associated SAEs**



Mild to Moderate AEs

Frequency (%) of safety events reported in clinical trials

	Telitacicept (n=1669)	Placebo (n=711)
Upper respiratory tract infection	39	36
Injection site reaction	21	2
Urinary tract infection	10	8
Cough	5	3
Diarrhea	5	5

*Pooled safety analysis across 14 completed telitacicept clinical studies; data cutoff December 1, 2025
AEs, adverse events; SAEs, serious adverse events.



Advancing the Leading BAFF/APRIL Inhibitor

Strong cash position of ~\$515M* with runway into early 2029 expected to cover key milestones

Vor Indications	Preclinical	Phase 1	Phase 2	Phase 3	Marketing Approval	Milestones
Generalized Myasthenia Gravis (gMG)			Phase 3			Global Phase 3 Topline Data (1H27)
Primary Sjögren's Disease (pSjD)			Phase 3			FPD (1Q26); Enrollment Ongoing
RemeGen Indications	Preclinical	Phase 1	Phase 2	Phase 3	Marketing Approval	Milestones
Generalized Myasthenia Gravis (gMG)					China Marketed	
Primary Sjögren's Disease (pSjD)					China Marketed	
IgA Nephropathy (IgAN)					Conditional Approval	
Systemic Lupus Erythematosus (SLE)					China Marketed	
Rheumatoid Arthritis (RA)					China Marketed	
Connective Tissue Disease-Associated Interstitial Lung Disease (CTD-ILD)			Phase 3			
Ocular Myasthenia Gravis (oMG)			Phase 3			
Membranous Nephritis (MN)			Phase 3 Planned			
Pediatric Systemic Lupus Erythematosus (pSLE)			Phase 3 Planned			
Autoimmune Encephalitis (AE)			Phase 3 Planned			
Pediatric IgA Nephropathy (pIgAN)			Phase 3 Planned			





Myasthenia Gravis

Moving Beyond Complement and
IgG Therapies

A Large and Growing Global Opportunity in Myasthenia Gravis

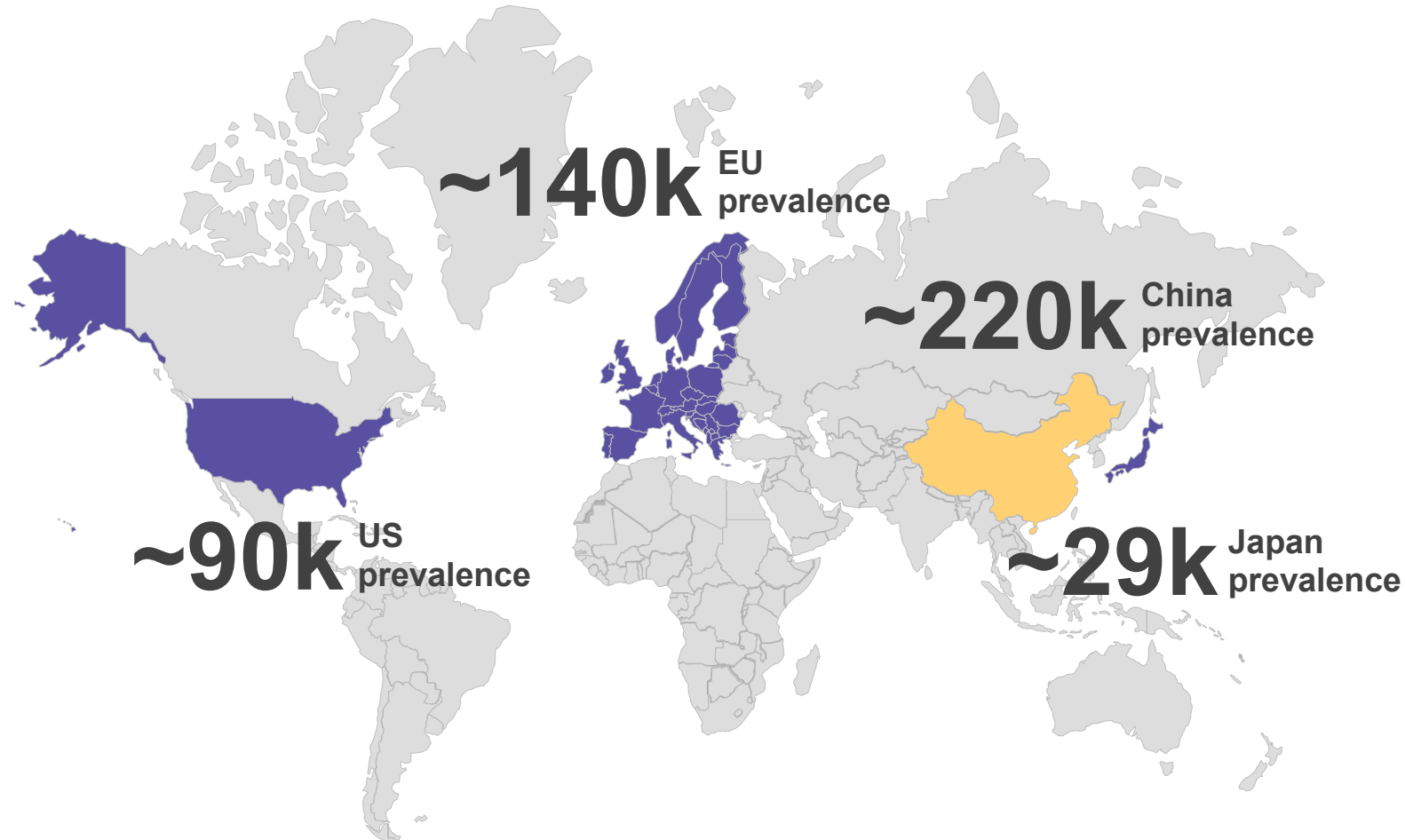
~260,000 diagnosed MG patients across key markets¹

Significant Burden

- A large, diagnosed patient population across key markets establishes a substantial initial opportunity

Favorable Growth Drivers

- Prevalence is growing due to increased awareness, improved diagnostics, and an aging population



MG Market Rapidly Expanding But Lacks Disease-Modifying Treatments

High growth market with underserved patient segments

gMG US Biologic Sales

~90,000

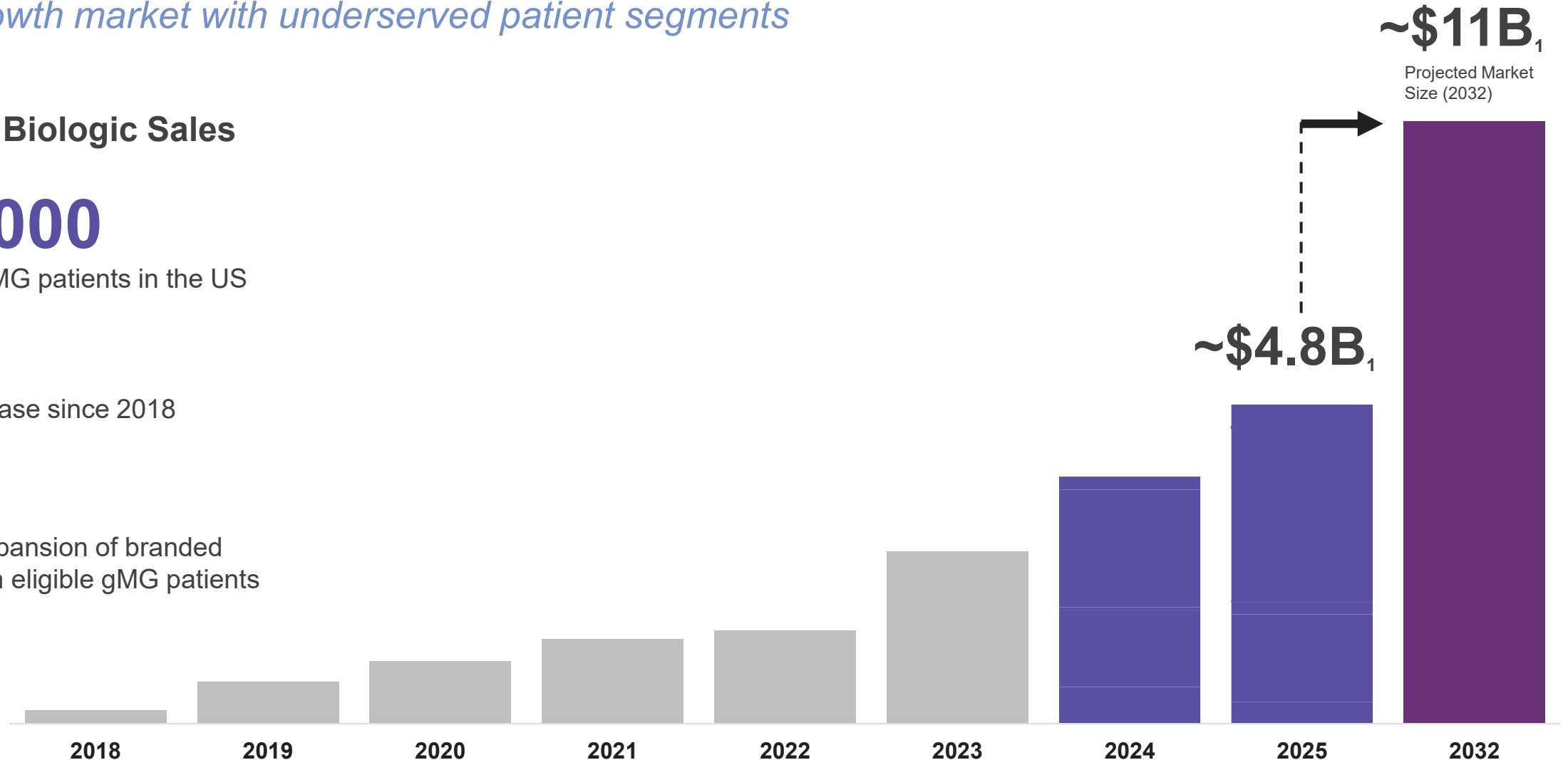
Diagnosed MG patients in the US

57%

CAGR increase since 2018

60%

Potential expansion of branded medicines in eligible gMG patients



Current Myasthenia Gravis Therapies Target Symptoms, Not Disease

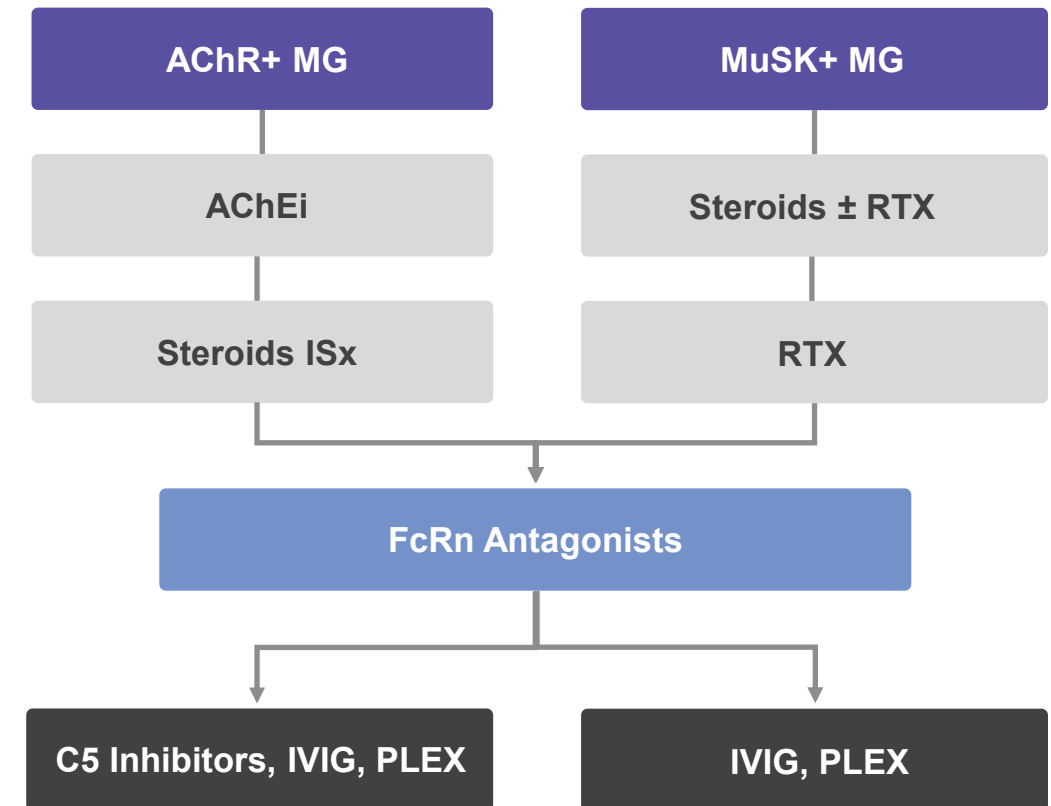
Even with the availability of biologics, unmet need for new therapies remain

Biologic use rising (~35% of gMG patients¹) with choices driven by efficacy, convenience, phenotype, and coverage

FcRn inhibitors dominate but ~20-50% of patients experience insufficient treatment responses²

Complement inhibitors limited by safety, black box warning, and vaccination requirements³

B cell depleting and cell therapies face challenges with efficacy, safety, and logistical limits despite disease-modifying potential⁴



1. Wedbush Neurologist Surveys
2. doi: 10.1136/jnnp-2024-334404
3. ULTOMIRIS, SOLIRIS label
4. doi: 10.1007/s40259-020-00443-w

AChEi, acetylcholinesterase inhibitors; AChR, acetylcholine receptor; FcRn, neonatal Fc receptor; IVIG, intravenous immune globulin; MuSK, muscle-specific tyrosine kinase; PLEX, plasma exchange; RTX, rituximab; ISx, immunosuppressants.



The Need for Therapies to Go Beyond IgG in Myasthenia Gravis

New evidence shows IgA and IgM drive pathology, demanding broader therapeutic strategies

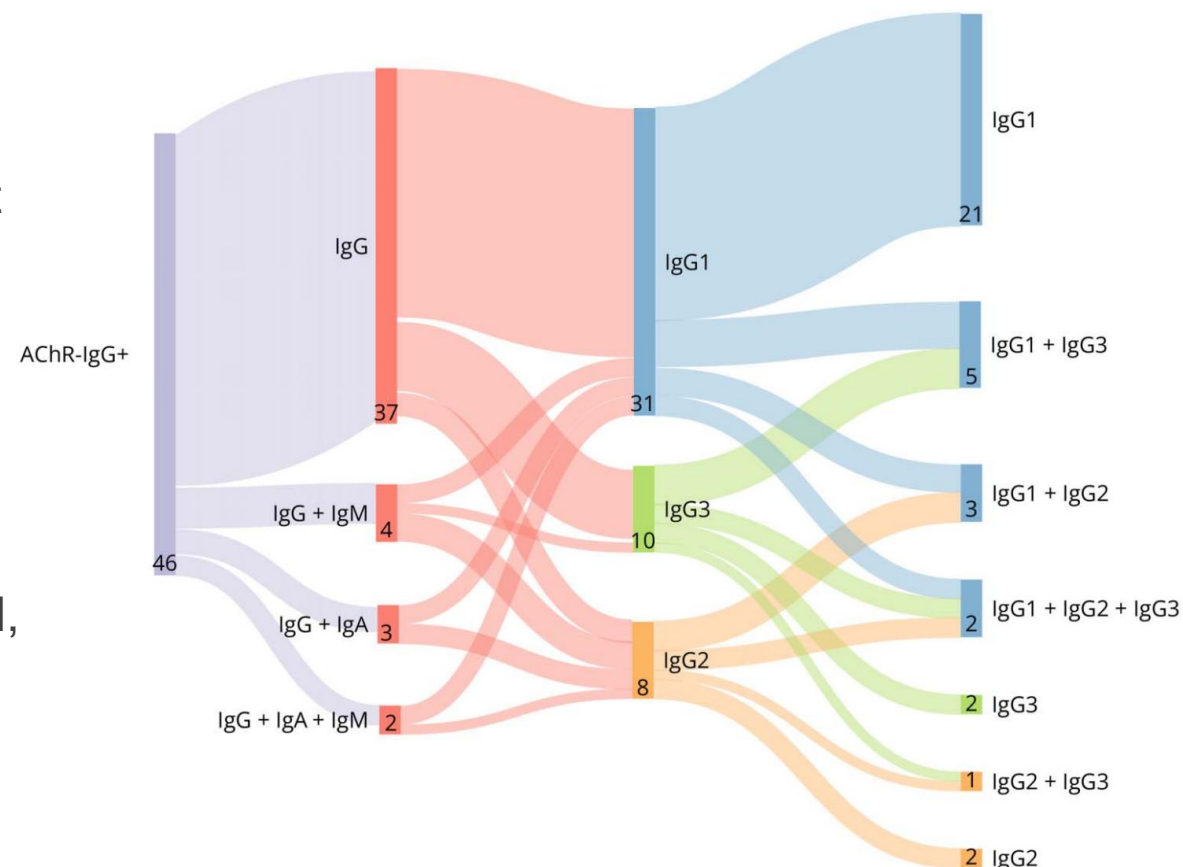
Patients carry different blends of IgG, IgA, and IgM,
driving overlapping mechanisms

Greater than 1 in 5 patients have IgA and/or IgM as part
of their autoantibody profile

MG patient autoantibody profiles shift over time,
sometimes tied to relapse

Current MG therapies only hit IgG, missing IgA and IgM,
which drive disease and are present in a meaningful
subset of patients

Distribution of AChR Autoantibody Isotypes and IgG Subclasses in MG



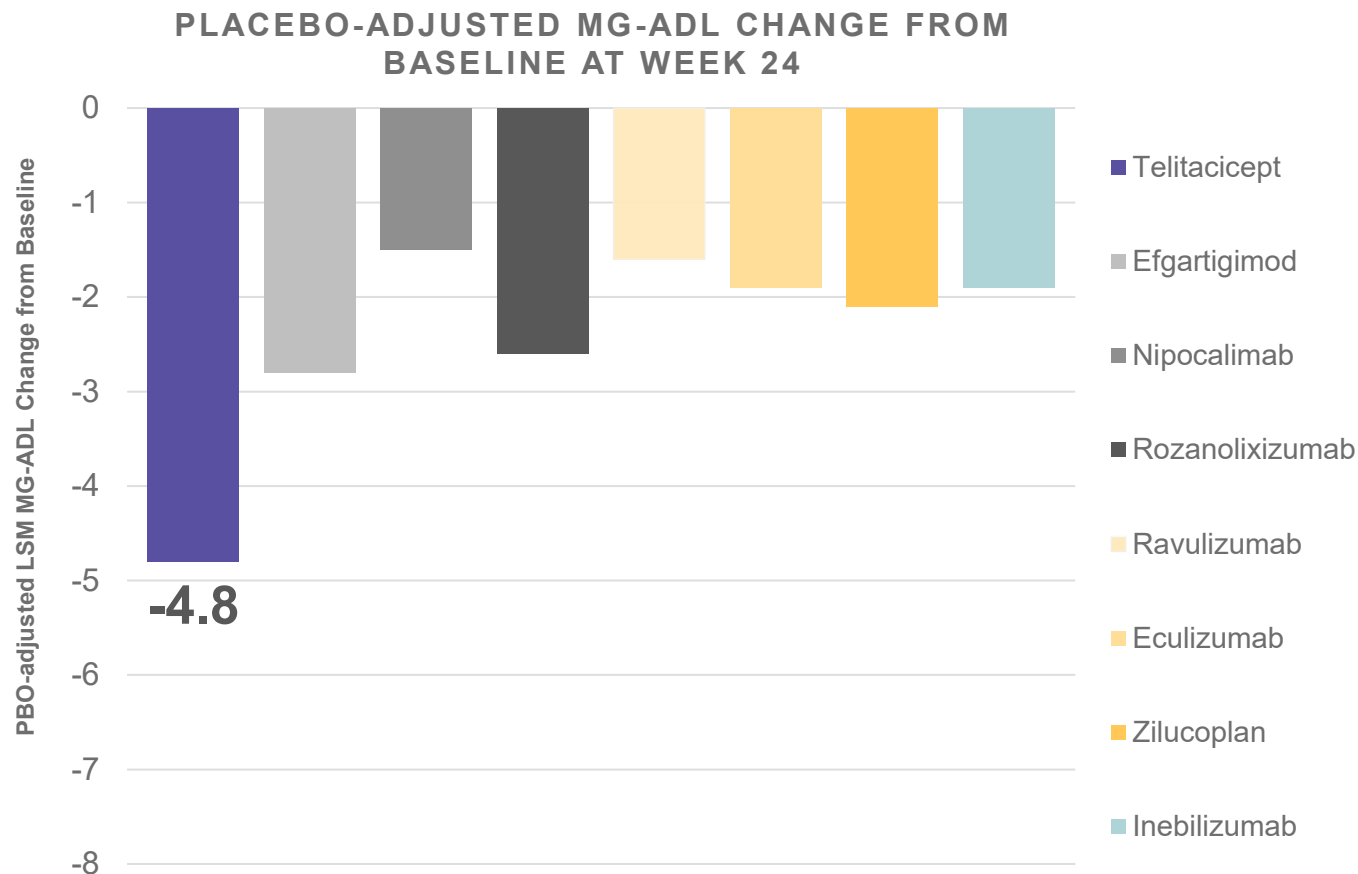
Myasthenia Gravis: The Beachhead Indication

Telitacicept demonstrated depth, durability, and a differentiated upstream mechanism

Across leading mechanisms, telitacicept demonstrates **largest placebo-adjusted MG-ADL improvement**

24- and 48-week data show continued improvement, suggesting patient benefit deepens over time

Upstream disease control with consistent safety and tolerability over one year of therapy



Based on historical clinical data; not a head-to-head trial



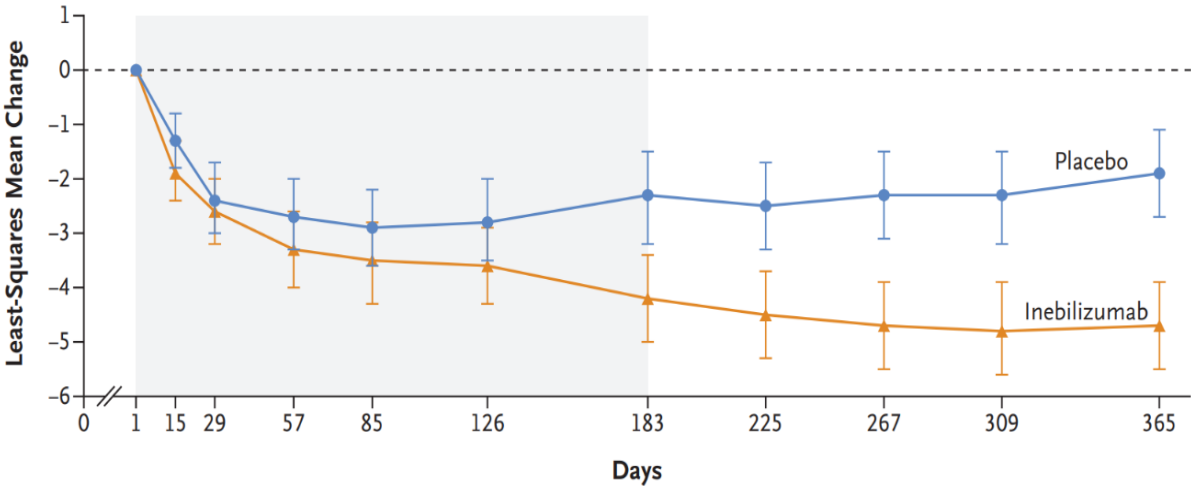
Durability in Myasthenia Gravis | Upstream vs Downstream Agents

How a therapy acts on antibody production and clearance yields different durability profiles

UPSTREAM

AMGEN PHASE 3 MINT TRIAL

UPLIZNA | Least Square Mean Change From Baseline in MG-ADL Score, AChR-Ab+ Population



Breadth:

- ⊗ Indiscriminately, broadly depletes CD19+ B cell lineage
- ⊗ Slightly reduces IgG

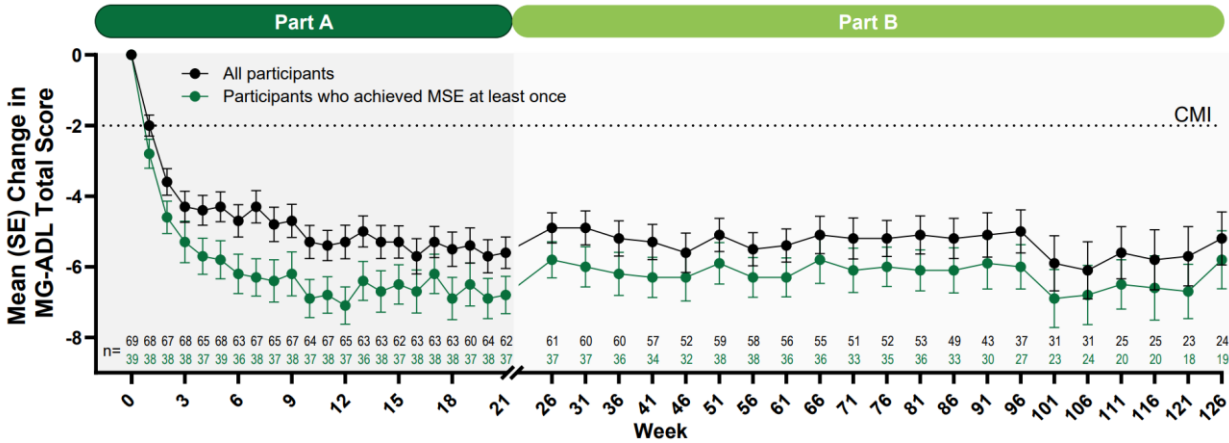
Durability:

- ☑ MG-ADL response deepens over time
- ⊗ Depth of response accrues slowly

DOWNSTREAM

ARGENX PHASE 3B ADAPT NXT TRIAL

VYVGART | Mean (SE) Change From in MG-ADL Total Score AChR-Ab+ Population



Breadth:

- ⊗ Pathogenic B cells left intact
- ⊗ Indiscriminately, broadly depletes circulating IgG only; no effect on IgM, IgA

Durability:

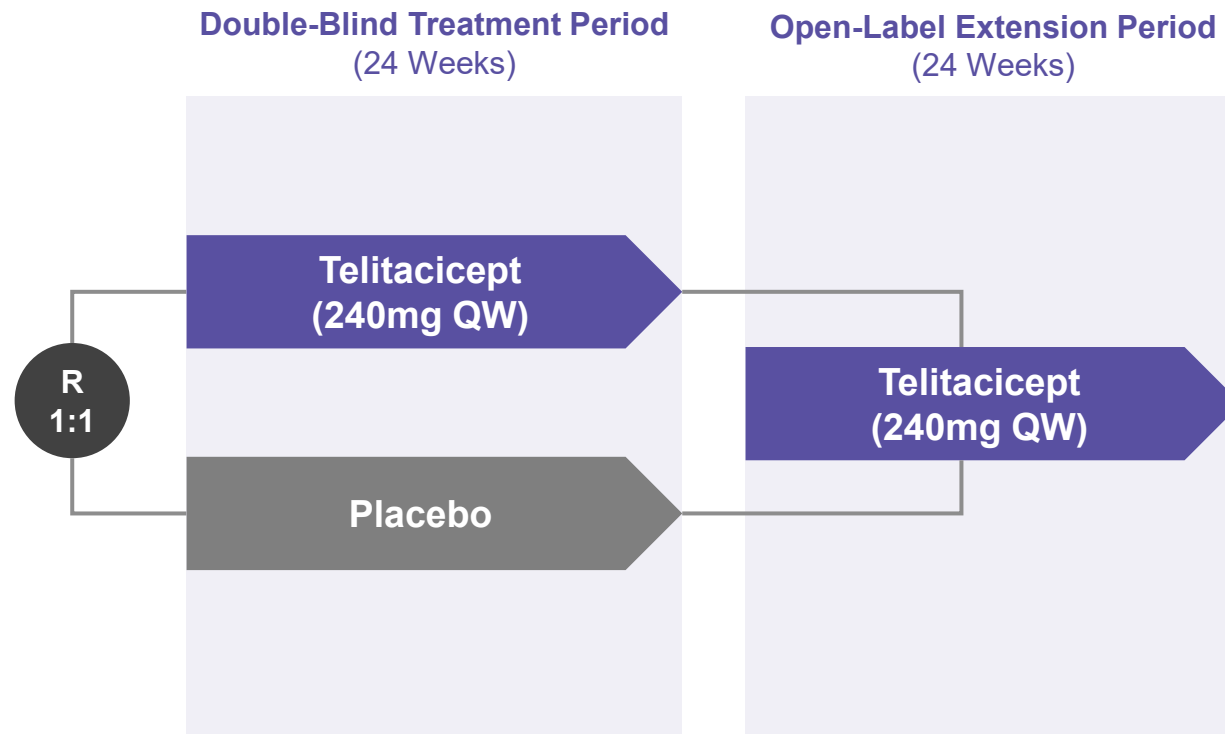
- ⊗ MG-ADL plateaus within the first 12 weeks; no further deepening with time
- ☑ Rapid onset by targeting IgG



Phase 3 Trial in Generalized Myasthenia Gravis Completed in China

Best-in-disease profile in China; randomized, double-blind, placebo-controlled study

114
Adults
With gMG



Primary Endpoint

- Change from baseline in MG-ADL at 24 weeks

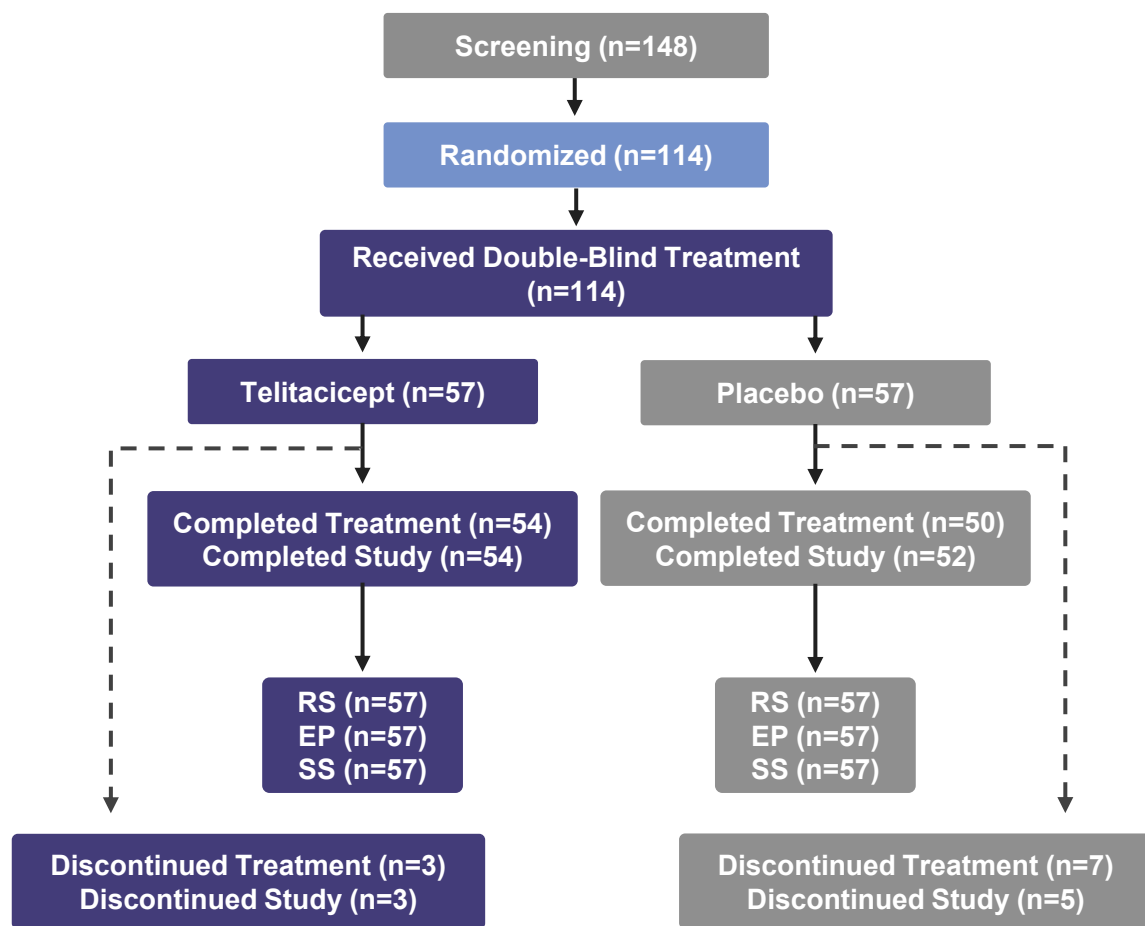
Secondary Endpoints

- Change from baseline in MG-ADL at 12, 36, and 48 weeks
- Change from baseline in QMG at 12, 24, 36, and 48 weeks
- Number of patients with ≥ 3 point decrease in MG-ADL, ≥ 5 point decrease in QMG at 24 and 48 weeks



Well Balanced Baseline Characteristics

Consistent with recent global Phase 3 populations

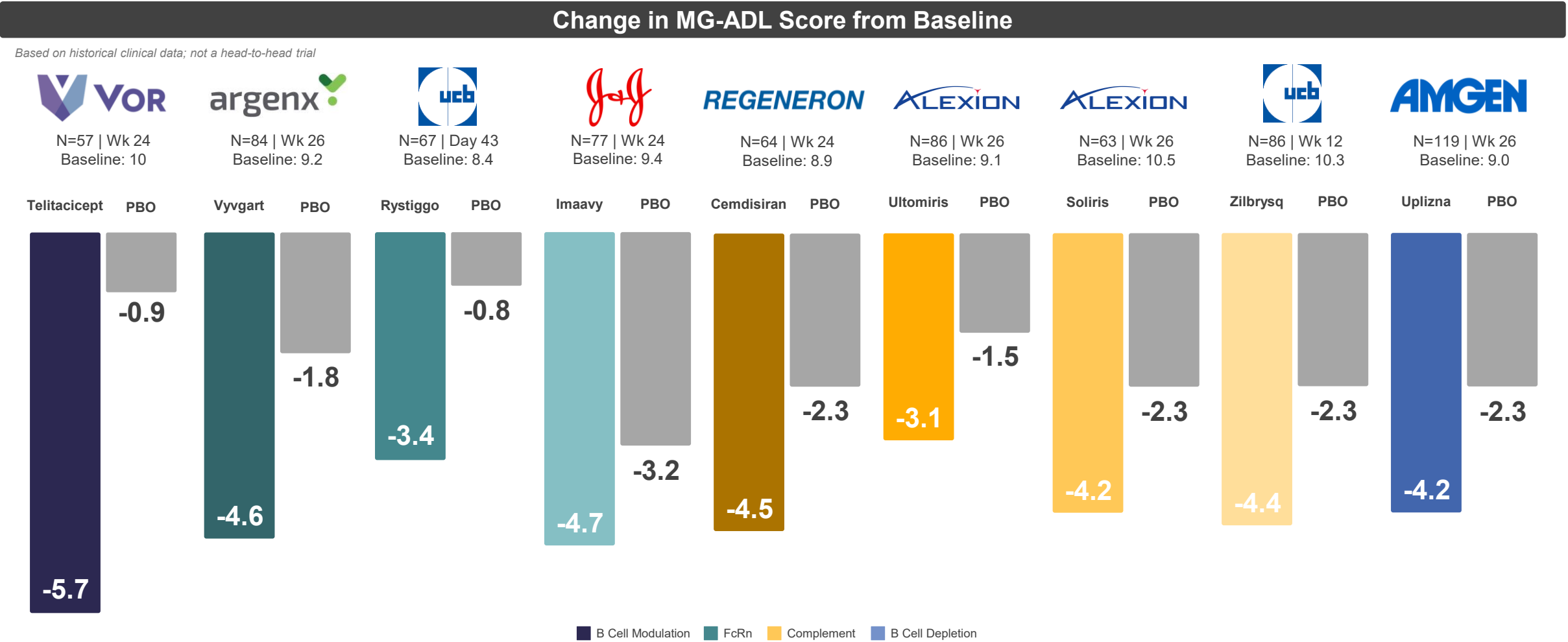


	Telitacicept (N=57)	Placebo (N=57)
Age (yr), mean ± SD	49.1 ± 14.69	49.6 ± 15.03
Sex		
Male, n (%)	30 (52.6)	21 (36.8)
Female, n (%)	27 (47.4)	36 (63.2)
Disease duration (month), mean ± SD	83.09 ± 84.507	76.05 ± 87.817
MGFA classification		
Class IIa, n (%)	3 (5.3)	12 (21.1)
Class IIb, n (%)	14 (24.6)	9 (15.8)
Class IIIa, n (%)	25 (43.9)	23 (40.4)
Class IIIb, n (%)	11 (19.3)	12 (21.1)
Class IVa, n (%)	4 (7.0)	1 (1.8)
Baseline MG-ADL score, mean ± SD	10.0 ± 2.60	9.9 ± 2.62
Baseline QMG score, mean ± SD	17.9 ± 3.43	18.8 ± 3.65
Antibody-positive at screening		
AChR, n (%)	55 (96.5)	55 (96.5)
MuSK, n (%)	2 (3.6)	2 (3.5)
Standard-of-care therapy		
Anticholinesterase inhibitors, n (%)	53 (93.0)	52 (91.2)
Steroids, n (%)	36 (63.2)	34 (59.6)
Immunosuppressants, n (%)	34 (59.6)	34 (59.6)



Telitacept Shows Potential Best-In-Disease Profile in gMG

Telitacept delivered 4.8-point MG-ADL separation from placebo, the largest reported in gMG

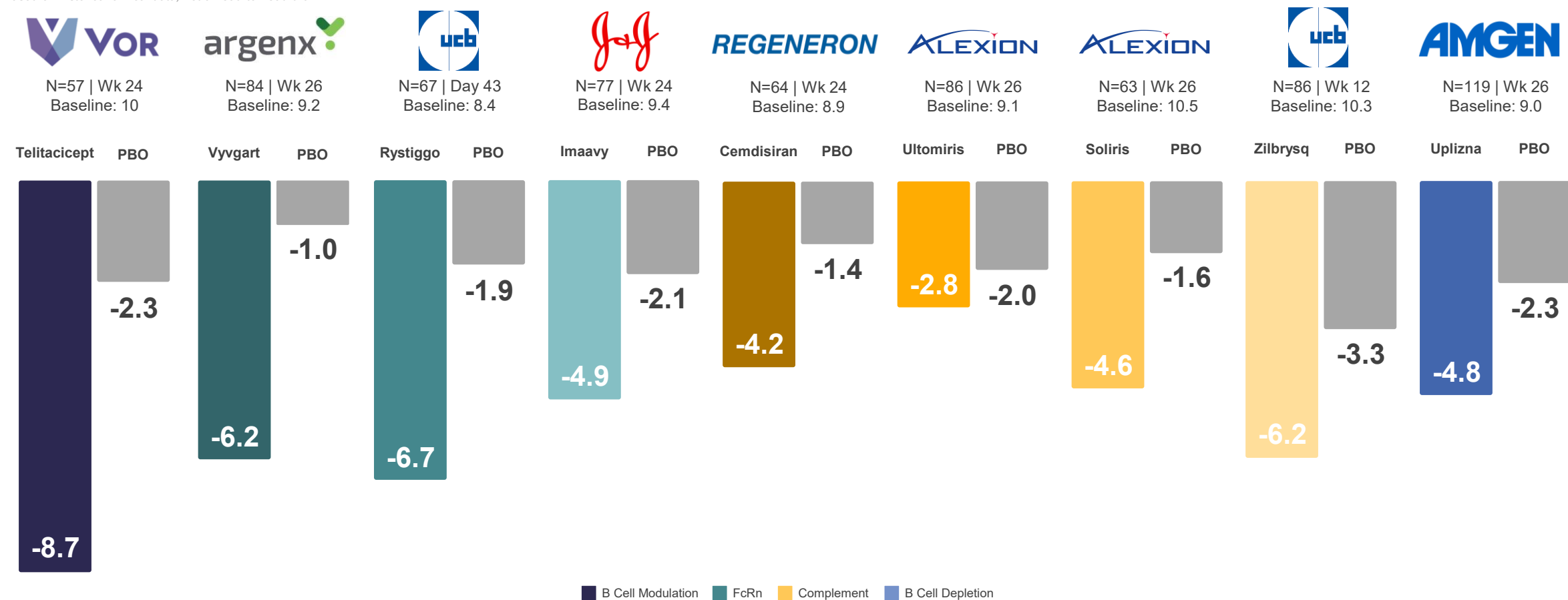


Telitacicept Shows Potential Best-In-Disease Profile in gMG

Telitacicept delivered 6.4-point QMG separation from placebo, the largest reported in gMG

Change in QMG Score from Baseline

Based on historical clinical data; not a head-to-head trial



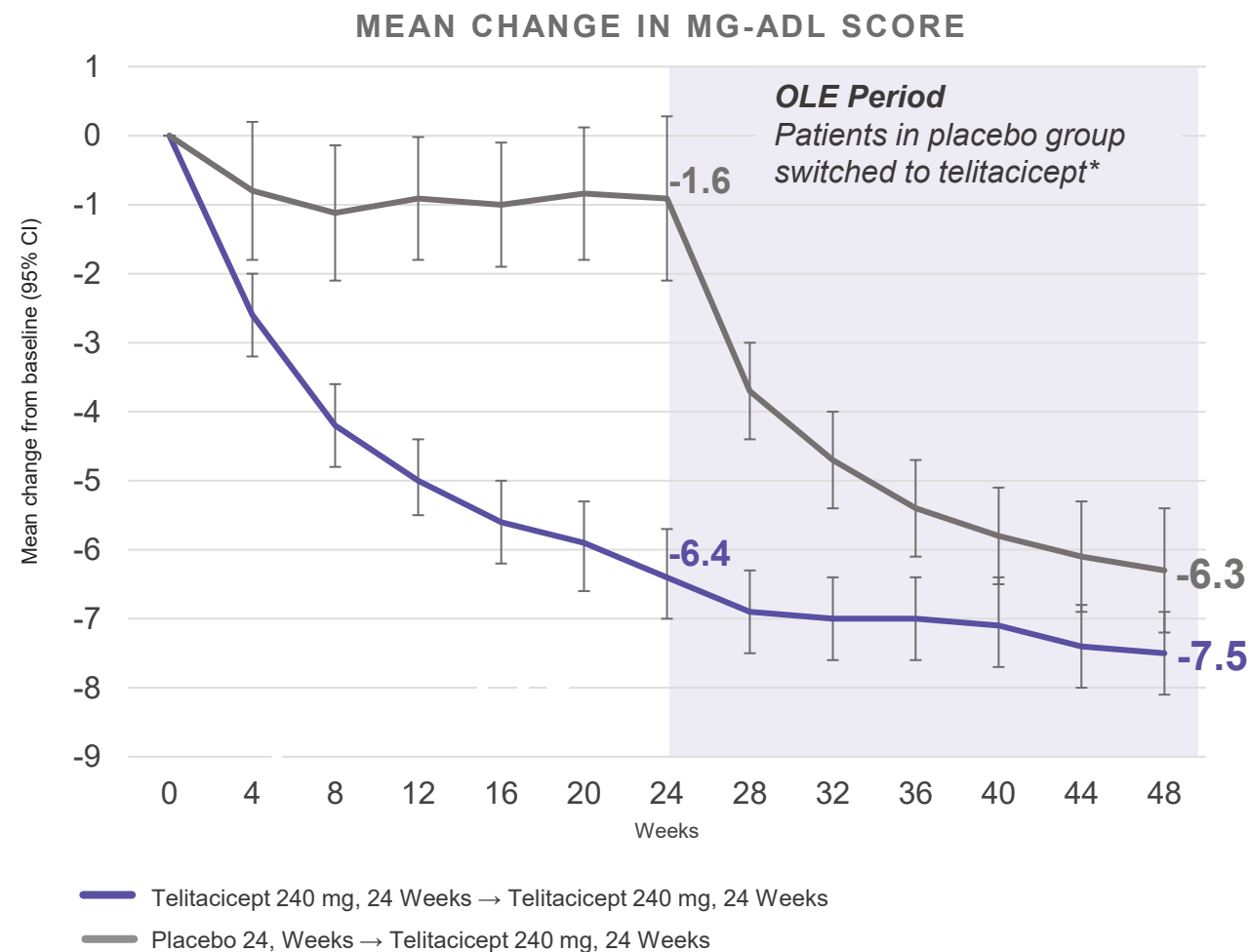
■ B Cell Modulation ■ FcRn ■ Complement ■ B Cell Depletion

QMG, Quantitative Myasthenia Gravis; gMG, generalized myasthenia gravis; Efgartigimod - ADAPT; Nipocalimab - Vivacity-MG3; Rozanolixizumab - MycarinG; Cemdisiran - NIMBLE; Ravulizumab - CHAMPION-MG; Eculizumab - REGAIN; Zilucoplan - RAISE; Inebilizumab - MINT

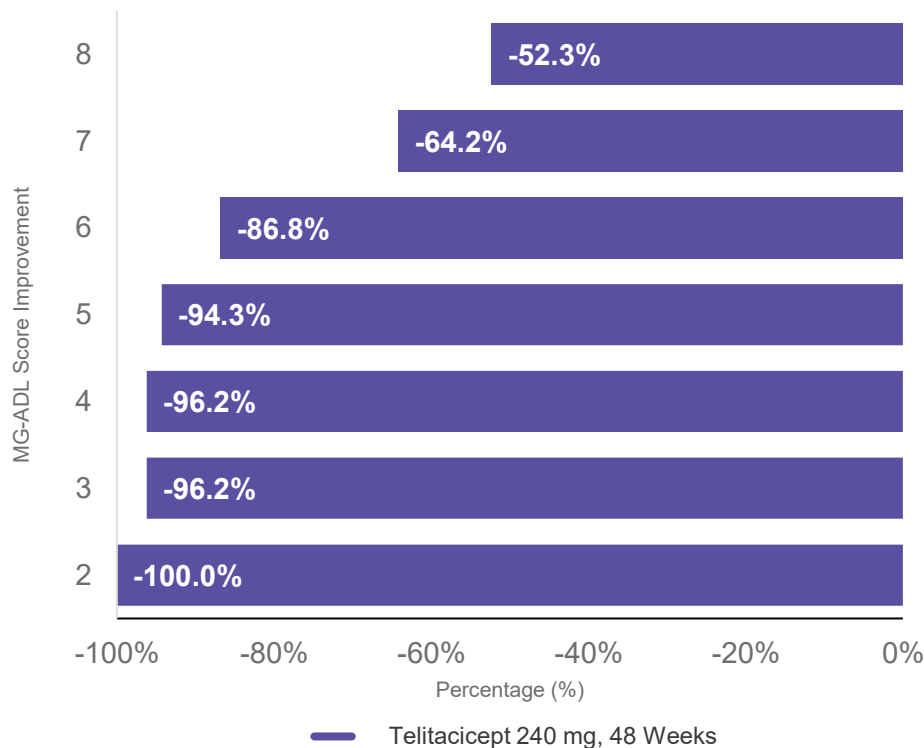


Telitacicept Demonstrated Durable MG-ADL Over Time

Sustained and deepening functional improvement through 48 weeks



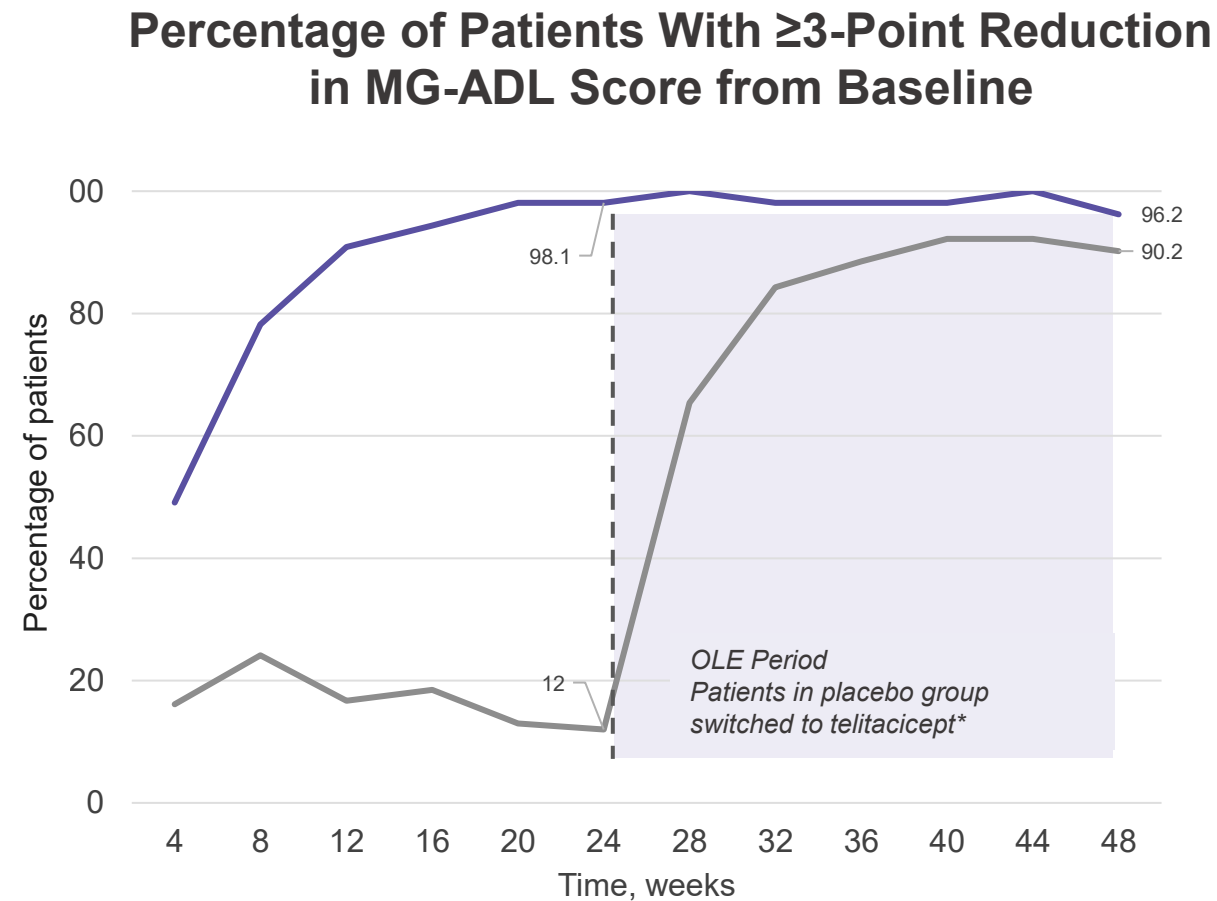
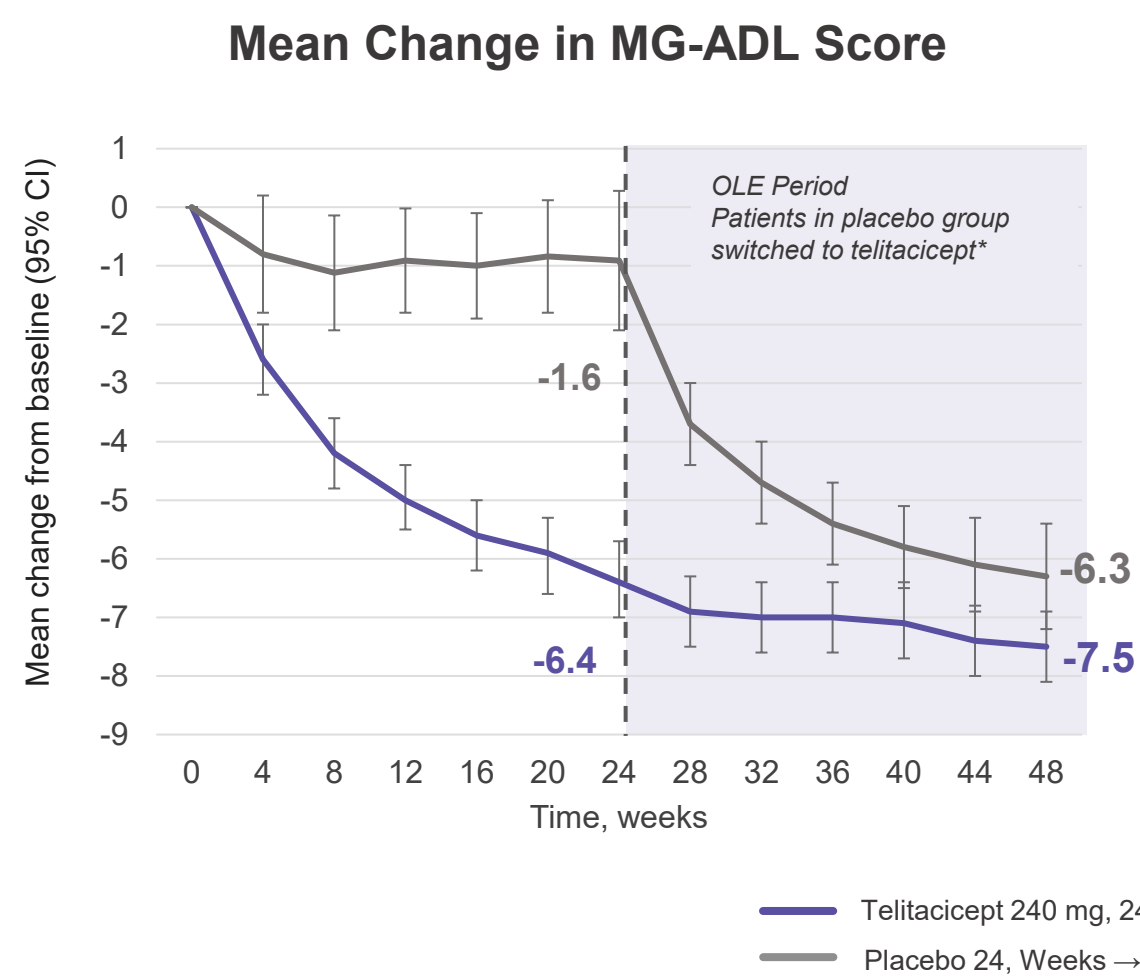
PERCENTAGE OF PATIENTS ACHIEVING IMPROVEMENT IN MG-ADL BY SCORE THRESHOLD AT WEEK 48



* Telitacicept arm continue with the same treatment, Placebo arm switched to Telitacicept during OLE period. The efficacy analysis was based on descriptive statistical analysis of the actual data in the full analysis set (FAS), and missing data were not filled.



OLE Data Highlight the Significant Change in MG-ADL Scores Seen With Telitacicept vs Placebo



Non-adjusted data presented. The efficacy analysis was based on descriptive statistical analysis of the actual data in the full analysis set (FAS), and missing data were not filled.

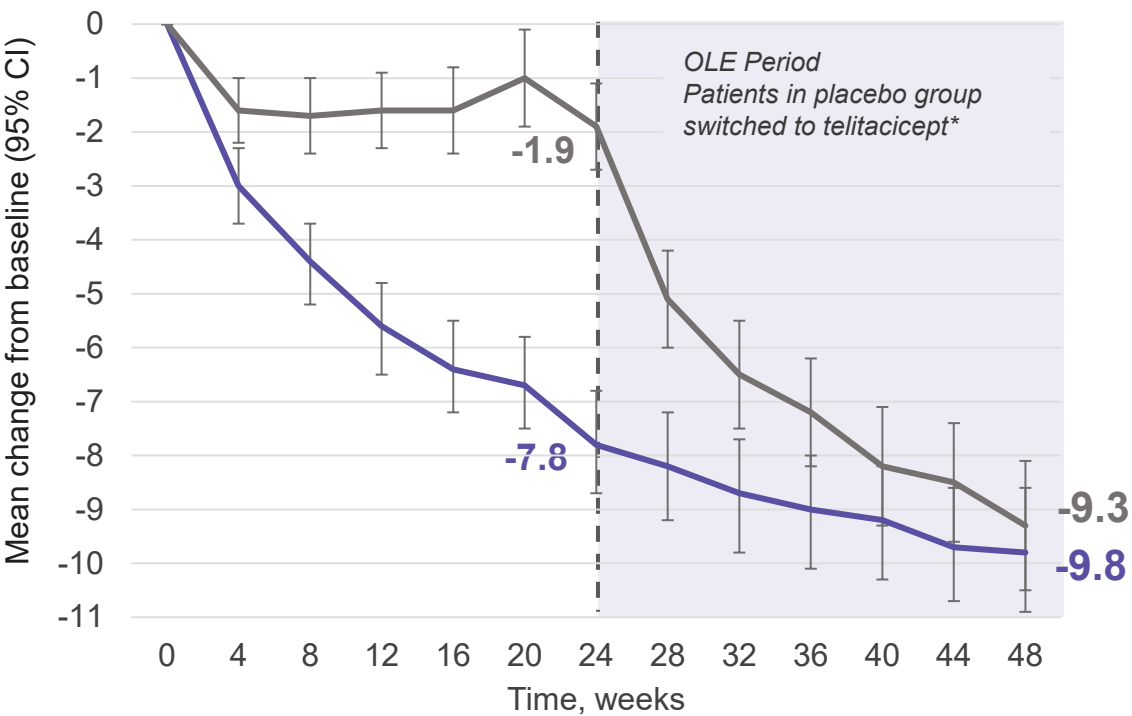
*Patients in placebo group switched to telitacicept 240 mg. Patients in telitacicept arm continued with telitacicept 240 mg QW.

Li G, et al. Presented at: American Association of Neuromuscular & Electrodiagnostic Medicine Annual Meeting; October 29-November 1, 2025; San Francisco, CA.

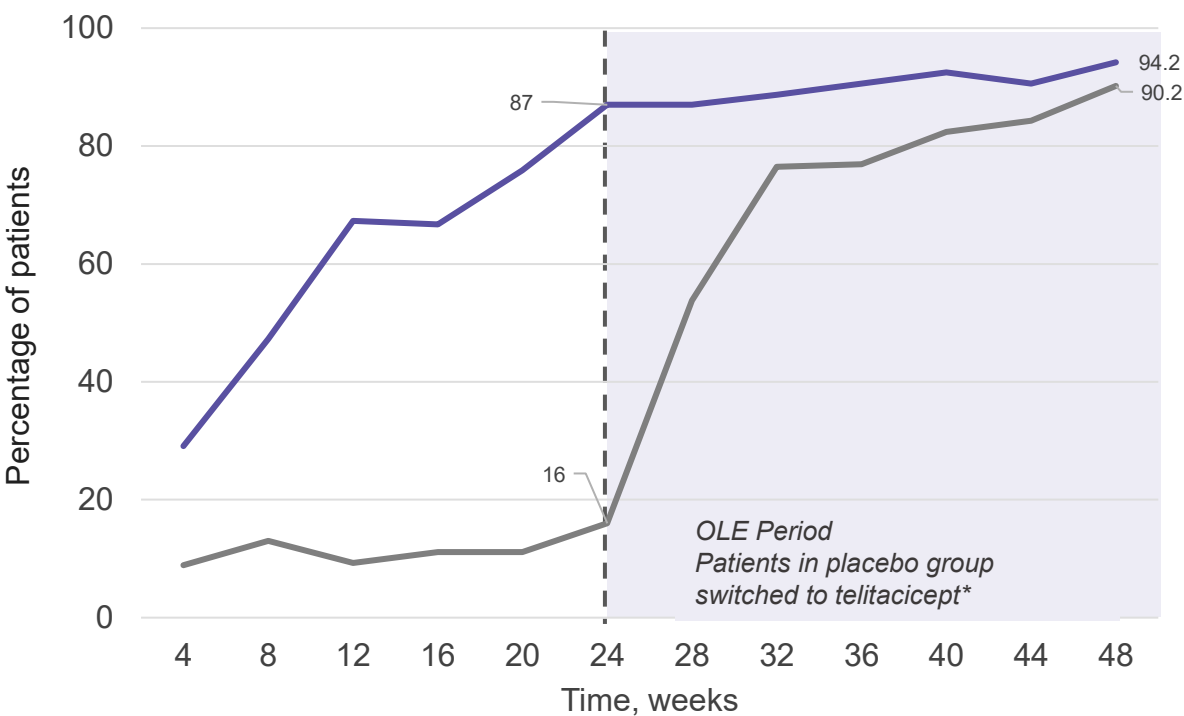


OLE Data Highlight the Significant Change in QMG Scores Seen With Telitacicept vs Placebo

Mean Change in QMG Score



Percentage of Patients With ≥ 5 -Point Reduction in QMG Score from Baseline

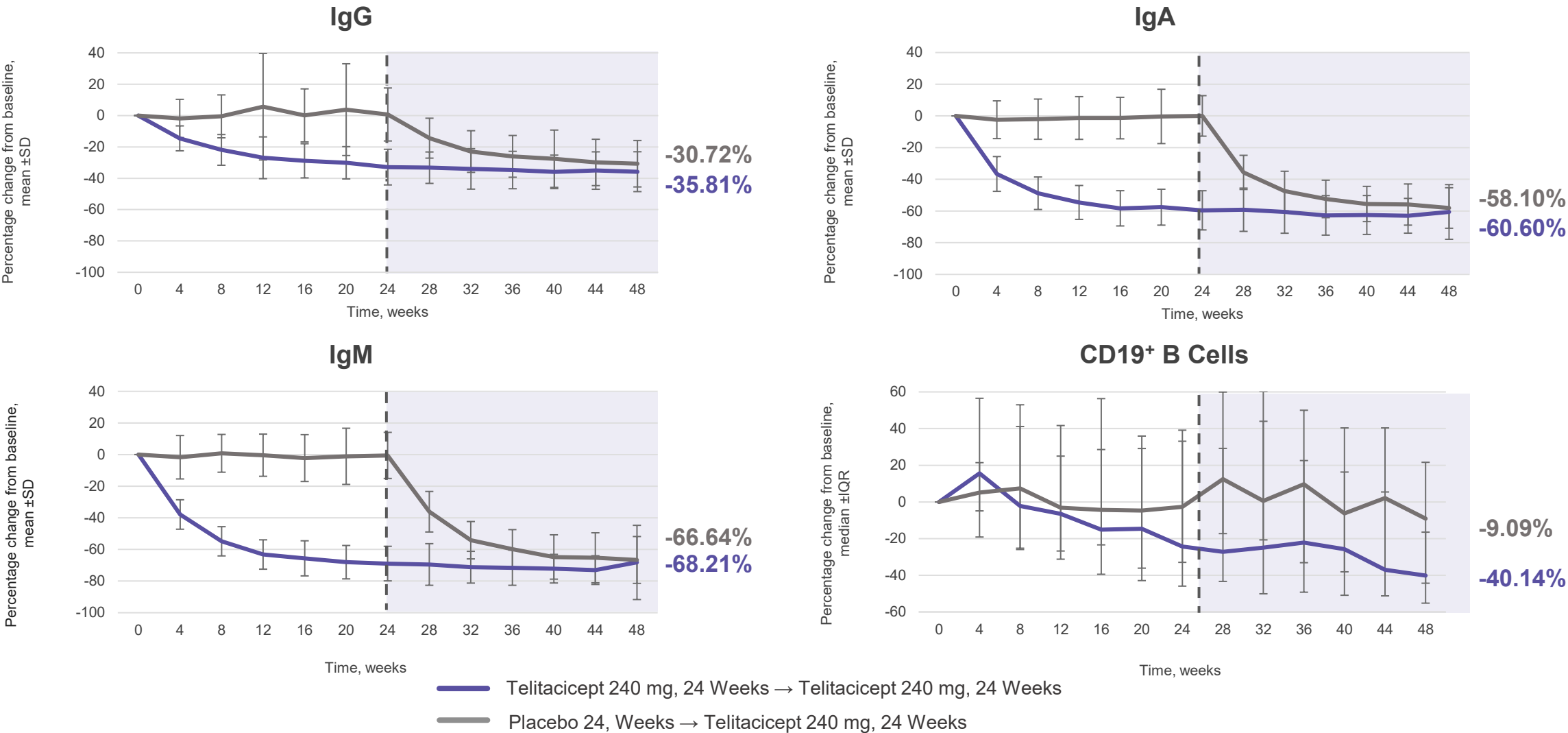


— Telitacicept 240 mg, 24 Weeks → Telitacicept 240 mg, 24 Weeks
— Placebo 24, Weeks → Telitacicept 240 mg, 24 Weeks

Non-adjusted data presented. The efficacy analysis was based on descriptive statistical analysis of the actual data in the full analysis set (FAS), and missing data were not filled.
*Patients in placebo group switched to telitacicept 240 mg. Patients in telitacicept arm continued with telitacicept 240 mg QW.
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Consistent Reduction in IgG, IgA, IgM, and B Cells



*Patients in placebo group switched to telitacicept. Patients in telitacicept arm continued with telitacicept 240 mg QW.



Favorable Safety Profile in gMG

Consistent with data from clinical trials in SLE, RA, SjD, and IgAN, and post-marketing data

	Randomized Control Period (Baseline to Week 24) ¹				OLE Period (Weeks 24-48) ²			
	Telitacicept (n=57)		Placebo (n=57)		Telitacicept 240 mg (n=54)		Placebo → Telitacicept 240 mg (n=53)	
	n (%)	Events	n (%)	Events	n (%) [†]	Events [‡]	n (%) [†]	Events [‡]
Infections and infestations	26 (45.6)	46	34 (59.6)	50	Infections and infestations	27 (50.0)	33	21 (39.6)
Upper respiratory tract infection	12 (21.1)	17	20 (35.1)	24	Upper respiratory tract infection	14 (25.9)	15	11 (20.8)
Urinary tract infection	9 (15.8)	11	6 (10.5)	6	Urinary tract infection	8 (14.8)	10	5 (9.4)
Pneumonia	1 (1.8)	1	6 (10.5)	6	Pneumonia	1 (1.9)	1	0 (0)
Respiratory tract infection	1 (1.8)	1	2 (3.5)	2	Respiratory tract infection	1 (1.9)	1	0 (0)
Influenza	0 (0)	0	3 (5.3)	3	Nasopharyngitis	1 (1.9)	1	1 (1.9)
					Gastroenteritis	1 (1.9)	1	1 (1.9)
					Herpes zoster	0 (0)	0	3 (5.7)
					COVID-19	0 (0)	0	2 (3.8)
					Pharyngitis	0 (0)	0	0 (0)
					Vaginal infection	0 (0)	0	2 (3.8)
					Oral herpes	1 (1.9)	1	0 (0)

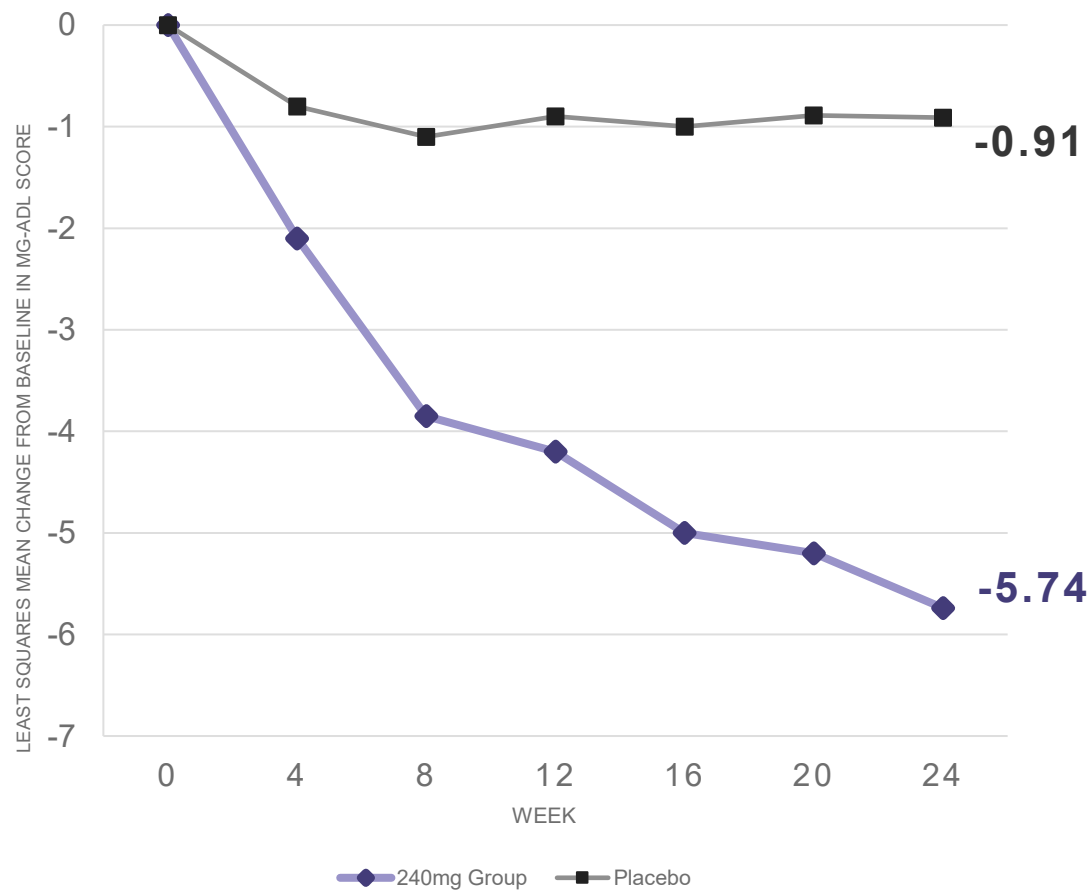
AE, adverse event; gMG, generalized myasthenia gravis; SjD, Sjogren's disease; SLE, systemic lupus erythematosus; RA, rheumatoid arthritis; IgAN, IgA nephropathy



Telitacicept Delivers Lasting Disease Control vs. Cyclic Relapse from FcRn Inhibitors

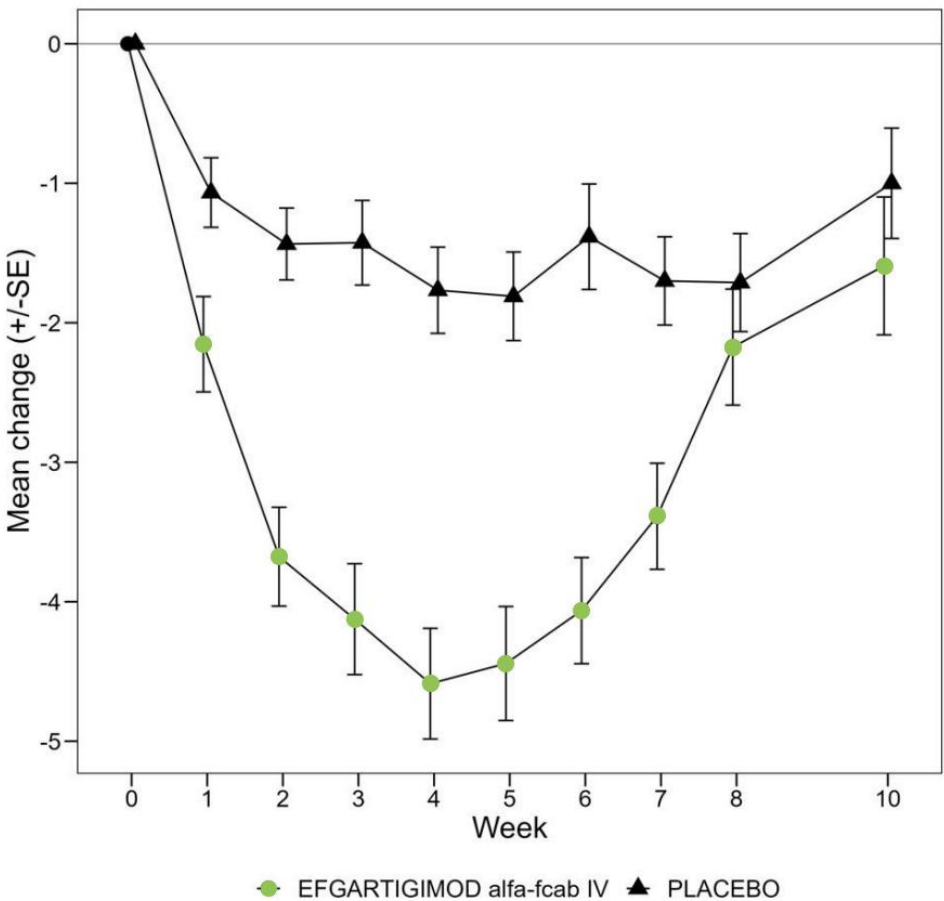
REMEGEN PHASE 3 CHINA TRIAL

Mean Change in MG-ADL Score†



ARGENX PHASE 3 ADAPT TRIAL

Mean Change in Total MG-ADL From Cycle 1 Baseline Over Time in AChR-Ab Positive Patients (mITT Analysis Set)



Based on historical clinical data; not a head-to-head trial

†Missing data imputed as non-response.



BAFF/APRIL Inhibition Creates the Potential for a Better Clinical Profile

Telitacicept avoiding extreme IgG lowering and B cell depletion supporting depth, breadth and durability

	Telitacicept	VYVGART™ (efgartigimod alfa-fcab)	UPLIZNA® inebilizumab-cdon
MoA	TACI-Fc	Anti-FcRn	Anti-CD19
IgG Reduction	25-35%	60-65%	N/A
CD19+ B cell reduction	20-40%	NA	100%
ΔMG-ADL vs. PBO	-4.8	-2.8	-1.9
ΔQMG vs. PBO	-6.4	-5.2	-2.5
Data readout	Week 24	Week 4	Week 24

Based on historical clinical data; not a head-to-head trial

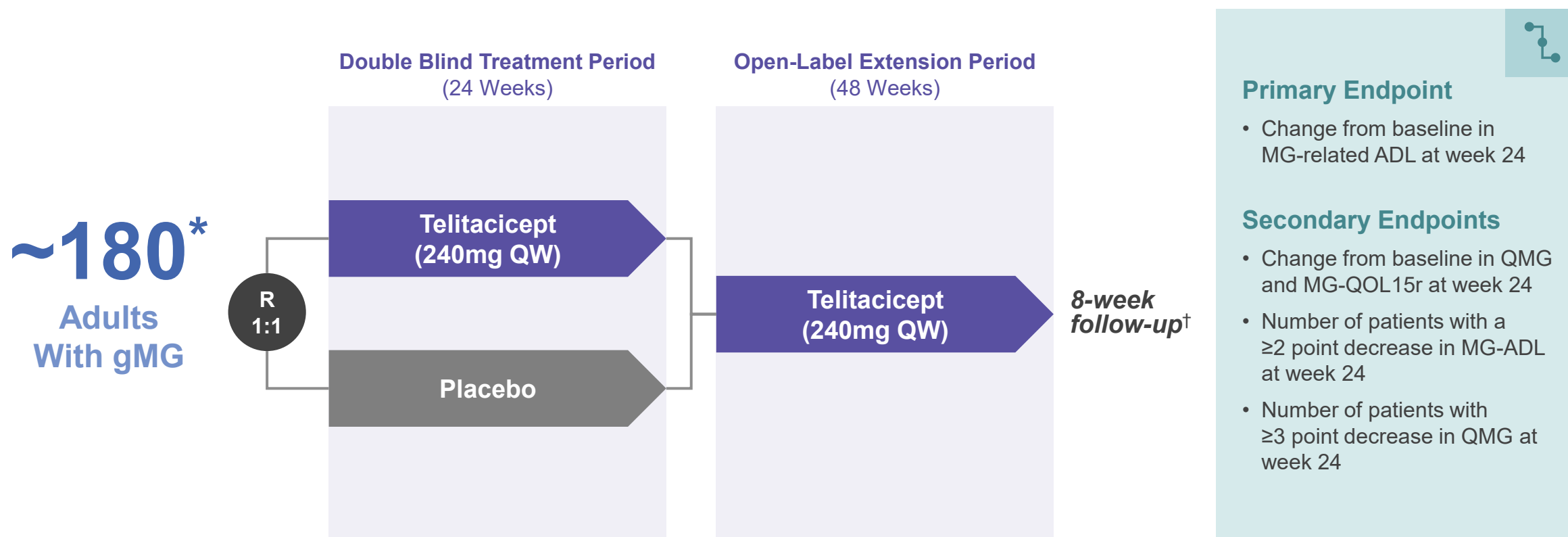
- 1. Argenx Phase 3 ADAPT Trial
- 2. Amgen Phase 3 MINT Trial
- 3. RemeGen China Phase 3 Trial

MoA, mechanism of action; MG-ADL, myasthenia gravis-activities of daily living; QMG, quantitative myasthenia gravis; PBO, placebo



UPSTREAM MG: Global Phase 3 in Generalized Myasthenia Gravis

Potential best- and first-in-class BAFF/APRIL inhibitor; randomized, double-blind, placebo-controlled study



Topline Global Phase 3 Data Anticipated in 1H27

Batoclimab Showed Consistent MG-ADL Improvement Independent of Region

PBO-adjusted MG-ADL Δ: −1.9 China vs −2.0 global

Metric	China n=131 6 weeks (JAMA Neurol 2024)	Global n=164 12 weeks (Immunovant 2025)	What It Means
Baseline MG-ADL (PBO)	8.4 (8.5)	8.8 (8.7)	Similar disease severity
PBO MG-ADL Absolute Δ	-1.7	-3.6	Global placebo more active
Drug MG-ADL Absolute Δ	-3.6	-5.6	Meaningful performance in both
PBO-Adjusted MG-ADL Δ	-1.9	-2.0	Similar contribution
IgG Reduction	~75% (max)	74% (mean)	Similar pharmacodynamics
Sustained Responder Rate (PBO)	58.2% (31.3%)*	93%†	Consistent or stronger globally
MSE Rate	25.4% (4.7%)	42% (7.0%)	MSE numerically higher globally

Based on historical clinical data; not a head-to-head trial

*58.2% in China uses a ≥3-point MG-ADL threshold; †93% globally uses a ≥2-point threshold.; PBO, placebo; MSE, minimal symptom expression





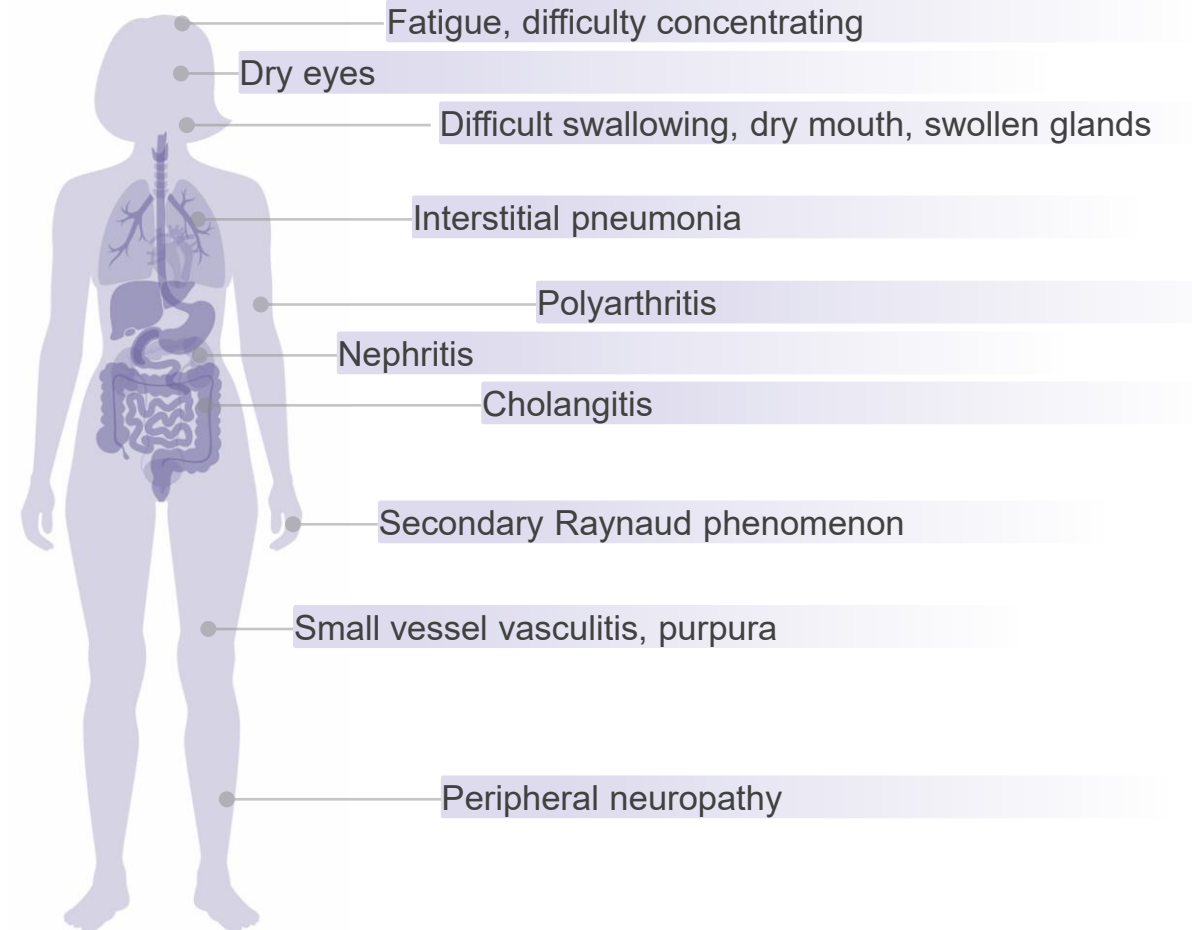
Sjögren's Disease

Moving Beyond Symptom Management

Sjögren's: A Large, Vastly Underserved Autoimmune Disease

Targeting ~100,000¹ addressable patient US opportunity with a potential best-in-disease profile

1	Symptom-Directed Local/topical therapies for dryness	ESSDAI 0
2	Mild-to-Moderate (No Major Organ Involvement) DMARDs (hydroxychloroquine, methotrexate)	ESSDAI 0-4
3	Moderate-to-Severe (Non-Life Threatening) Immunosuppressants (Methotrexate, azathioprine, or cyclosporine)	ESSDAI 5-13
4	Severe Disease (Major Organ Involvement) High potency immunosuppressants, biologics	ESSDAI ≥14



Sjögren's: One of the Most Common Systemic Autoimmune Diseases Globally

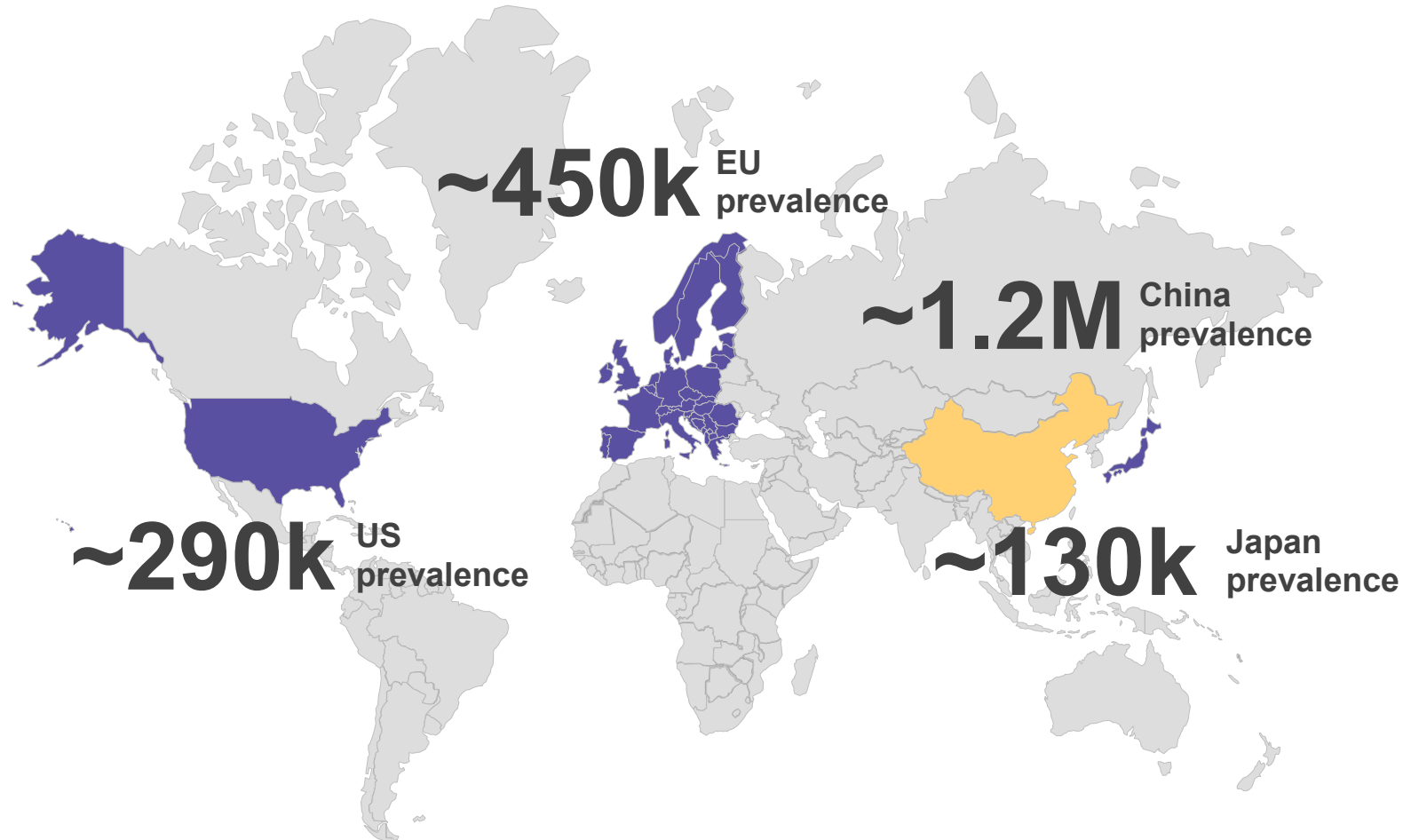
~870,000 diagnosed Sjögren's patients across key markets¹

Favorable Growth Drivers

- Rising diagnosis rates from better awareness and testing

Underpenetrated Market

- No approved disease-modifying systemic therapies
- Patients managed with symptomatic treatments



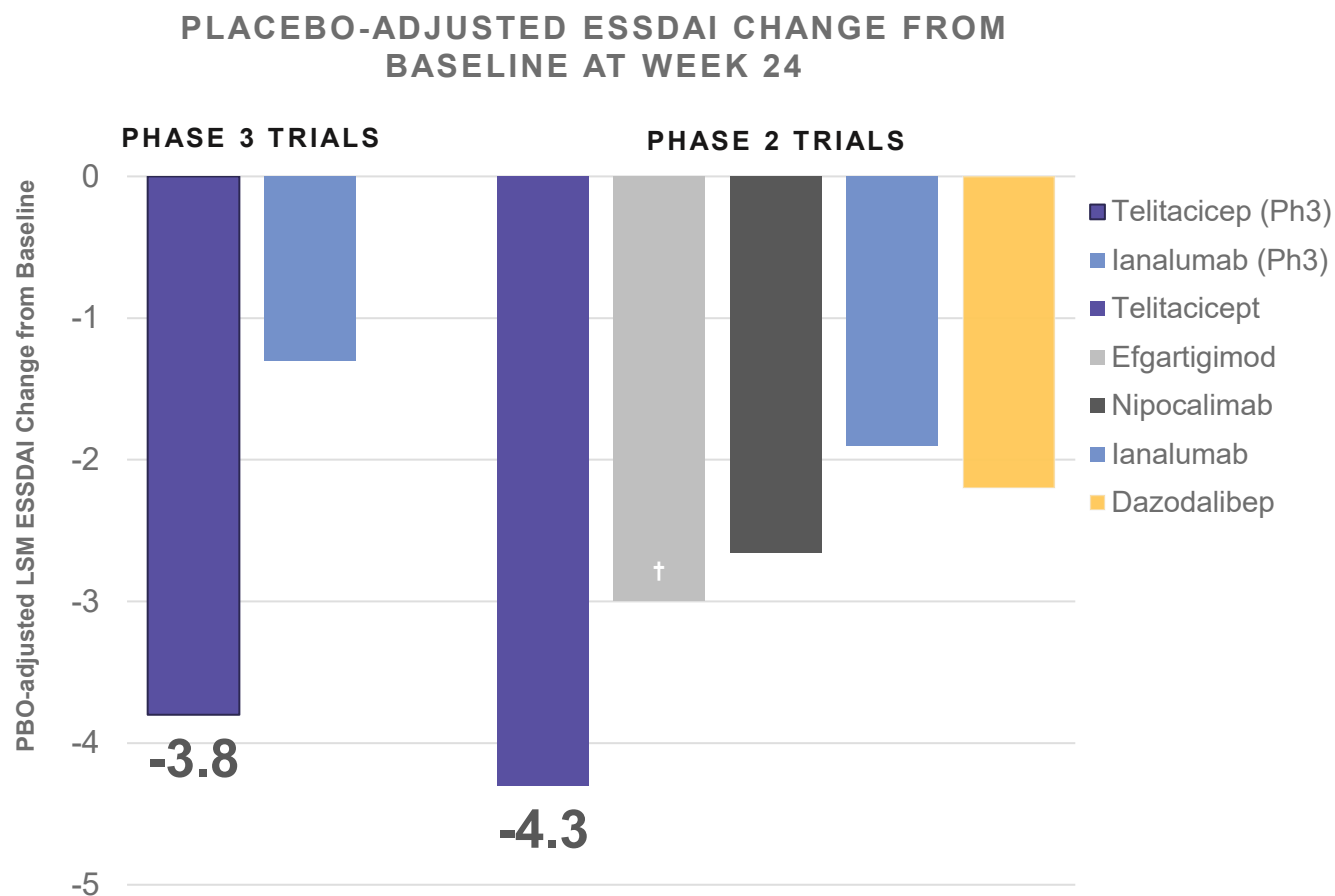
Sjögren's Disease: The White Space Expansion Opportunity

Telitacicept demonstrated depth, durability, and a differentiated upstream mechanism

Across leading mechanisms, telitacicept demonstrates **largest placebo-adjusted ESSDAI improvement**

Response deepens through week 48, with 73% of patients achieving ≥ 3 -point ESSDAI reduction vs 16.5% for placebo

Upstream disease control with consistent safety and tolerability over one year of therapy



Based on historical clinical data; not a head-to-head trial



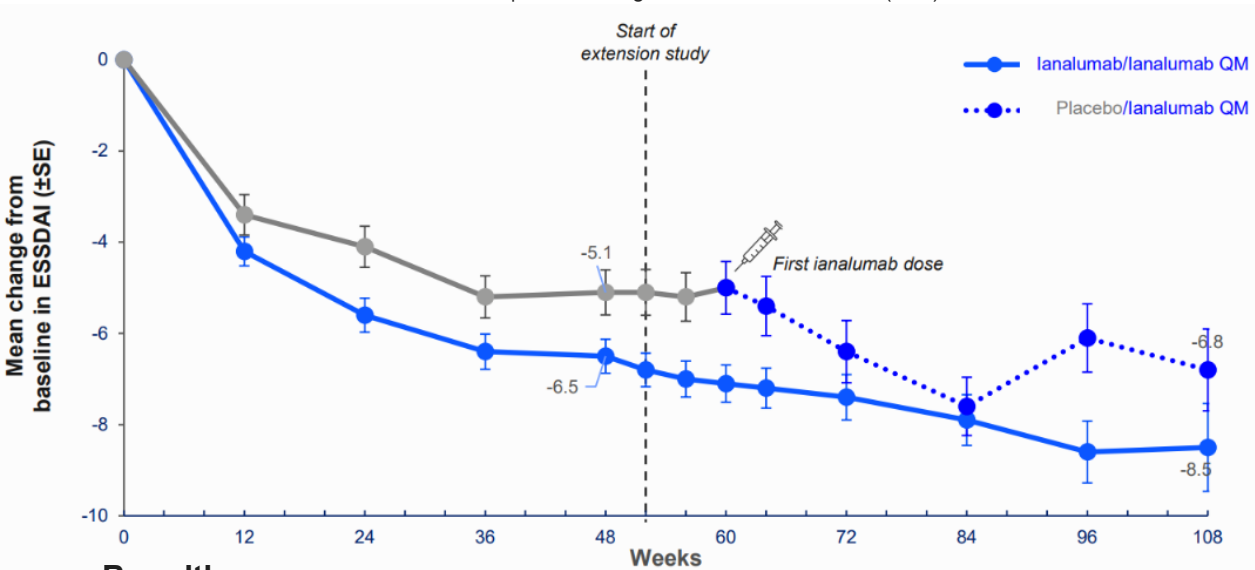
Durability in Sjögren's Disease | Upstream vs Downstream Agents

How a therapy acts on antibody production and clearance yields different durability profiles

UPSTREAM

NOVARTIS PHASE 3 NEPTUNUS TRIAL

Ianalumab | Mean Change from Baseline ESSDAI (±SE)



Breadth:

- ⊗ Indiscriminately, broadly depletes BAFF-R+ B cell lineage
- ⊗ Modestly reduces IgG; no reported effect on IgA or IgM

Durability:

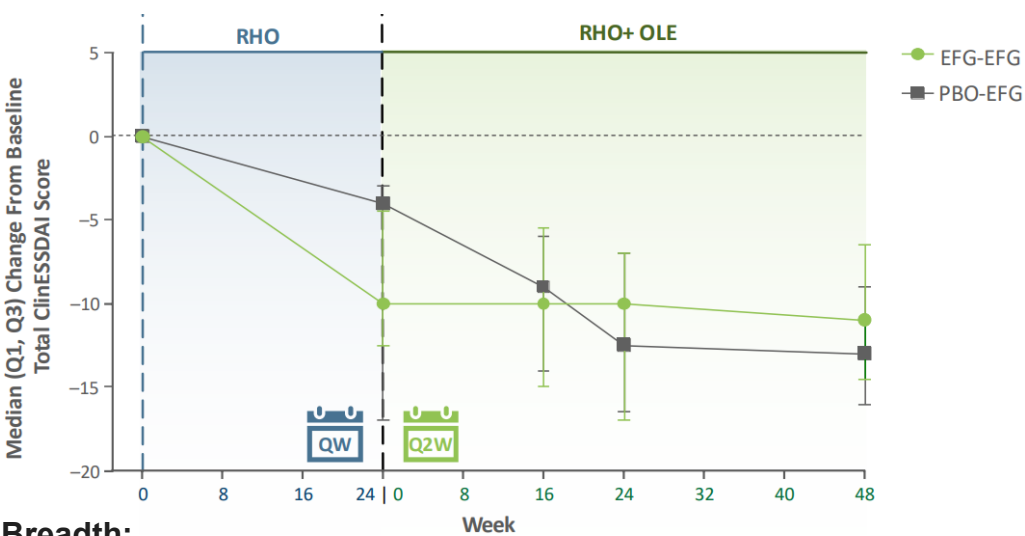
- ☑ ESSDAI response deepens over time
- ⊗ Depth of response accrues slowly

DOWNSTREAM

ARGENX PHASE 2 RHO+ OPEN-LABEL EXTENSION TRIAL

VYVGART | Median (Q1,Q3) Change* From Baseline Total ClinESSDAI Score

*Calculated relative to RHO study baseline (post hoc analyses); participants that rolled over into the OLE study



Breadth:

- ⊗ Pathogenic B cells left intact
- ⊗ Indiscriminately, broadly depletes circulating IgG only; no effect on IgM, IgA

Durability:

- ⊗ Plateaued clinESSDAI
- ☑ Benefit maintained after responders moved to Q2W dosing

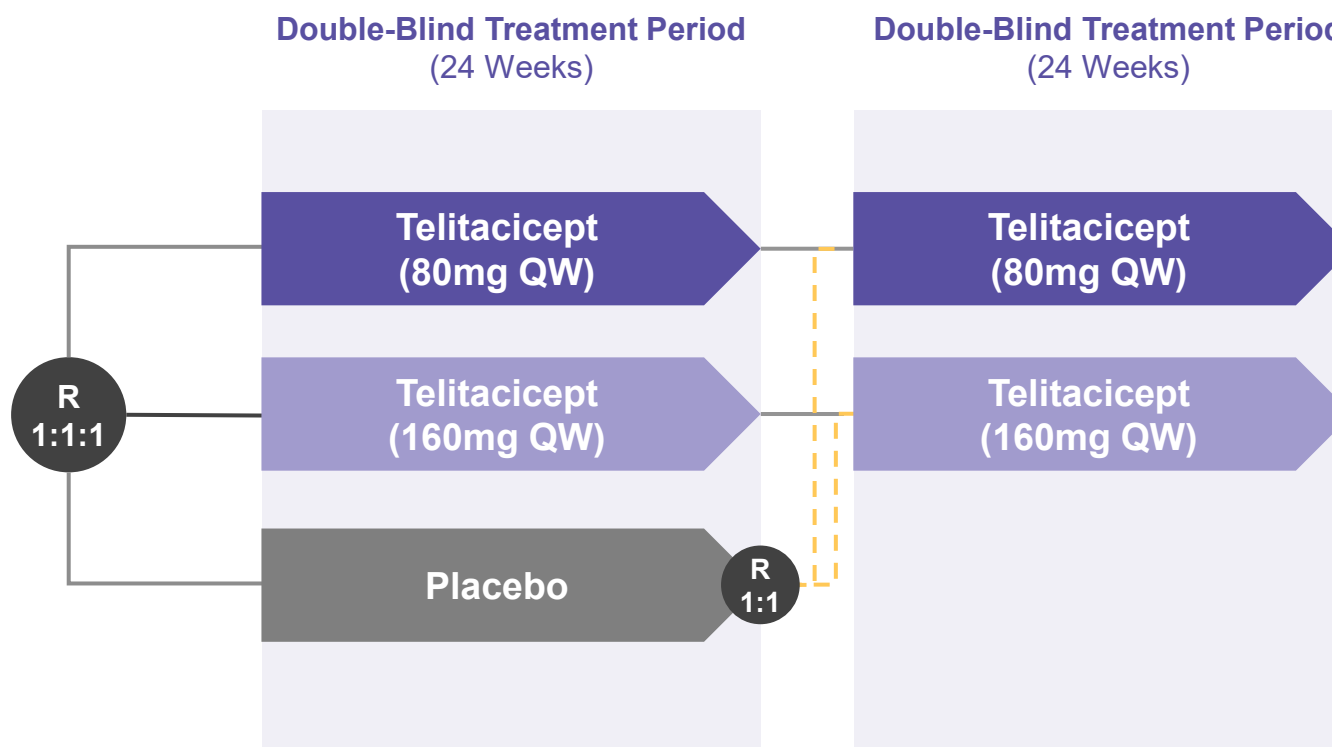


Phase 3 Trial in Primary Sjögren's Disease Completed in China

Best-in-disease profile in China; randomized, double-blind, placebo-controlled study

380*

Adults
With pSjD



Primary Endpoint

- Change from baseline in ESSDAI at 24 weeks

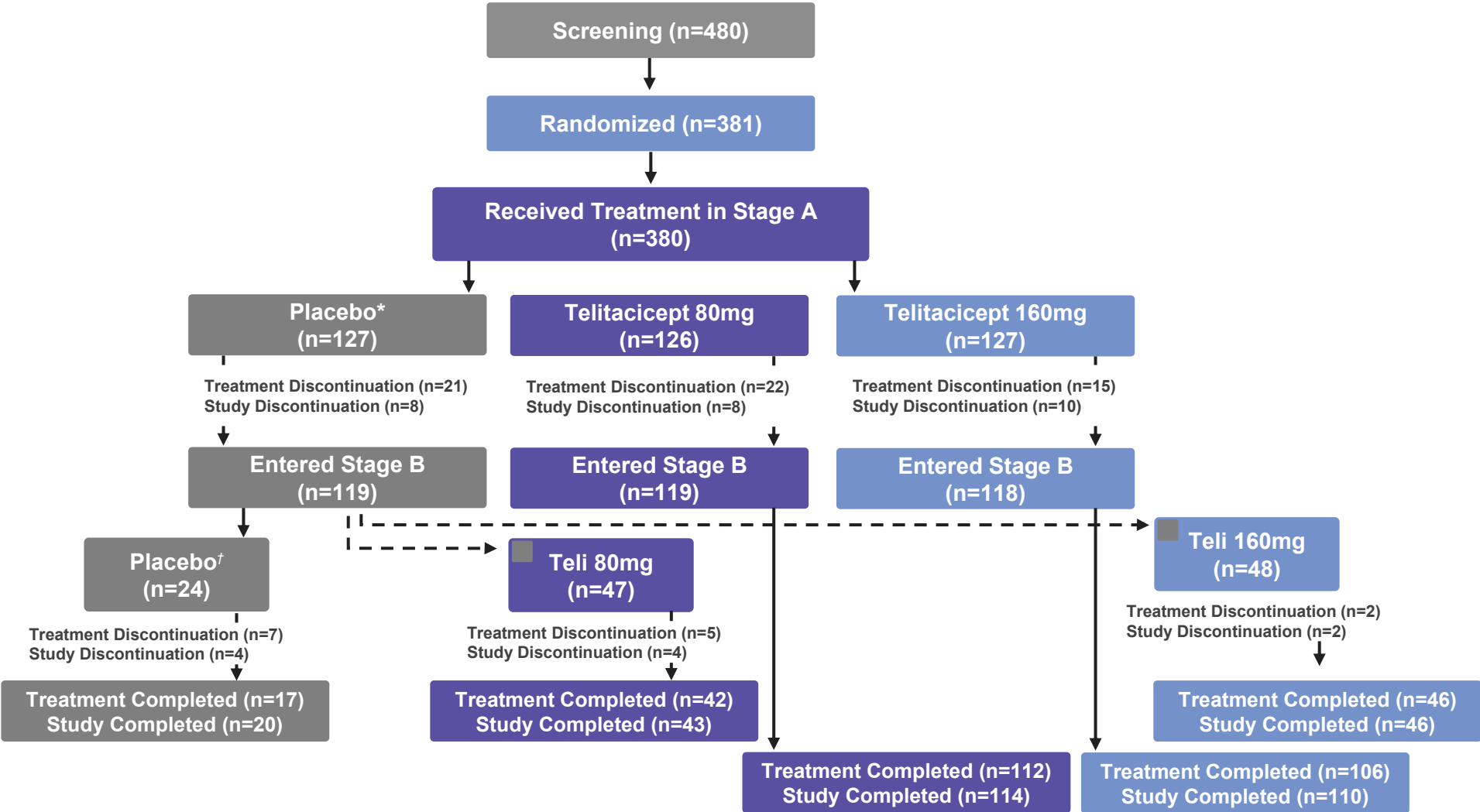
Secondary Endpoints

- Change from baseline in ESSDAI at 12 weeks
- Changes from baseline in ESSPRI, PGA, PaGA, SF-36 and MFI-20 at 12 and 24 weeks

Primary Endpoint Achieved in August 2025

Patient Disposition

Strong study execution across 79 sites in China



*Participants randomized to the placebo group; †Participants who did not switch treatment throughout the trial; Teli, telitacicept



Baseline Characteristics

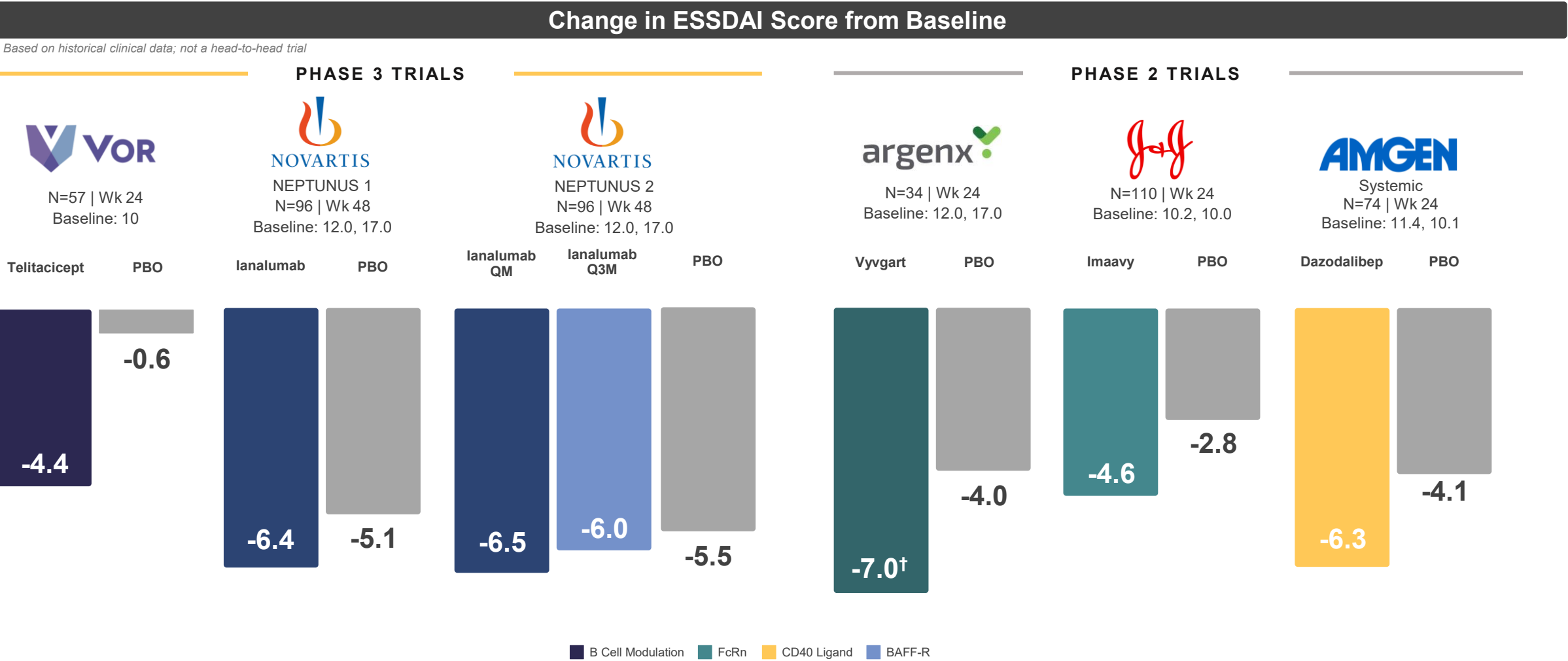
Well-balanced, representative patient population across treatment arms

	Telitacicept 160mg (n=127)	Telitacicept 80mg (n=127)	Placebo (n=127)	Total (n=381)
Age (yr), Mean (SD)	45.9 (12.29)	44.6 (12.06)	47.3 (12.75)	46.0 (12.39)
Body Weight (kg), Mean (SD)	55.89 (9.04)	56.88 (8.71)	56.99 (11.08)	56.58 (9.65)
BMI (kg/m ²), Mean (SD)	22.03 (3.16)	22.31 (3.19)	22.24 (3.58)	22.19 (3.31)
Sex, n (%), Female	124 (97.6)	124 (97.6)	123 (96.9)	371 (97.4)
pSD Duration (mon), Mean (SD)	21.388 (36.32)	25.831 (46.90)	19.930 (39.57)	22.383 (41.14)
ESSDAI Score, Mean (SD)	10.0 (3.77)	9.8 (3.52)	10.2 (4.17)	10.0 (3.82)
ESSDAI ≥10 points, n (%)	63 (49.6)	61 (48.0)	63 (49.6)	187 (49.1)
ESSPRI Score, Mean (SD)	5.07 (1.60)	4.91 (1.72)	5.08 (1.76)	5.02 (1.69)
MFI-20 Total Score, Mean (SD)	56.8 (12.08)	56.8 (12.50)	58.1 (13.07)	57.2 (12.54)
Baseline Hydroxychloroquine Use, n (%)	87 (68.5)	97 (76.4)	99 (78.0)	283 (74.3)
IgG (g/L), Mean (SD)	21.459 (7.43)	22.429 (7.95)	22.297 (7.21)	22.062 (7.53)
IgA (g/L), Mean (SD)	3.435 (1.63)	3.637 (1.86)	3.154 (1.68)	3.409 (1.73)
IgM (g/L), Mean (SD)	1.458 (0.64)	1.372 (0.92)	1.274 (0.70)	1.368 (0.77)
CD19 ⁺ B Cell (cells/μL), Mean (SD)	209.412 (129.10)	183.993 (109.52)	266.902 (726.52)	220.102 (430.96)



Telitacept Shows Potential Best-In-Disease Profile in pSjD

Telitacept delivered 3.8-point ESSDAI separation from placebo, the largest reported in SjD



Telitacicept Shows Potential Best-In-Disease Profile in pSjD

Telitacicept delivered 1.5-point ESSPRI separation from placebo, the largest reported in SjD

Change in ESSPRI Score from Baseline

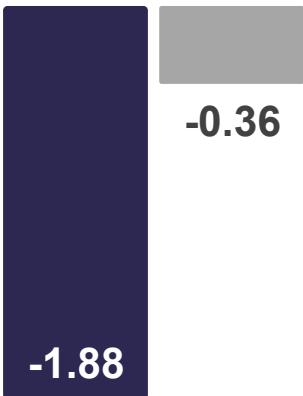
Based on historical clinical data; not a head-to-head trial

PHASE 3 TRIALS



N=57 | Wk 24
Baseline: 10

Telitacicept PBO



NOVARTIS

NEPTUNUS 1
N=96 | Wk 48
Baseline: 12.0, 17.0

Ianalumab PBO

Not Reported



NOVARTIS

NEPTUNUS 2
N=96 | Wk 48
Baseline: 12.0, 17.0

Ianalumab QM Ianalumab Q3M PBO

Not Reported



N=34 | Wk 24
Baseline: 12.0, 17.0

Vyvgart PBO

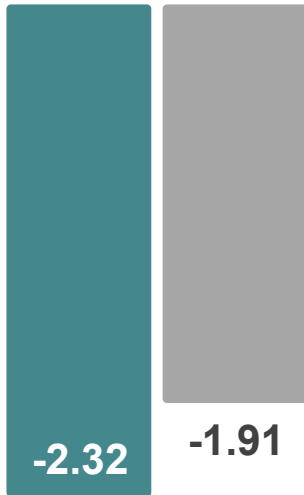
Not Reported

PHASE 2 TRIALS



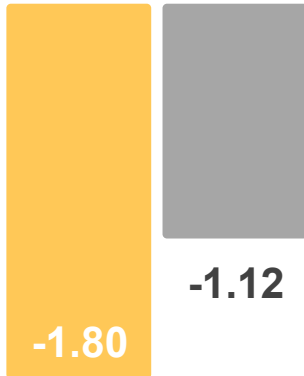
N=110 | Wk 24
Baseline: 10.2, 10.0

Imaavy PBO



Systemic
N=74 | Wk 24
Baseline: 11.4, 10.1

Dazodalibep PBO



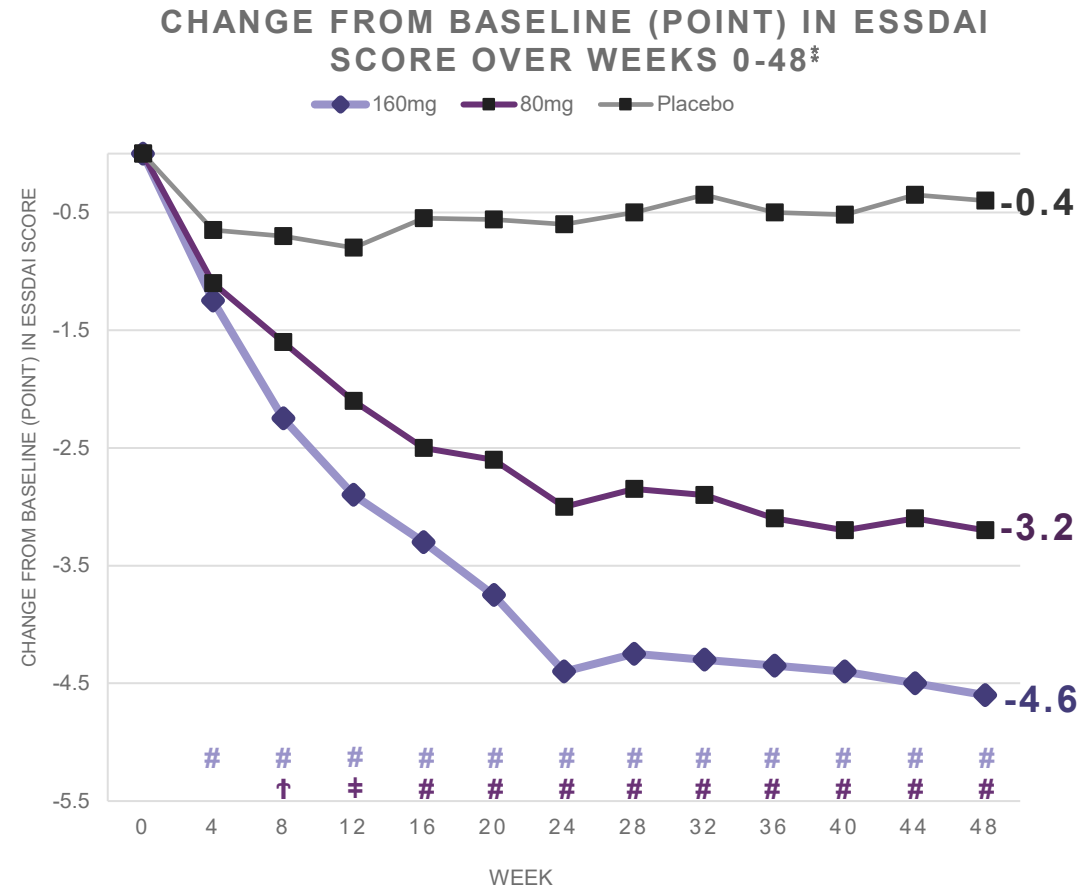
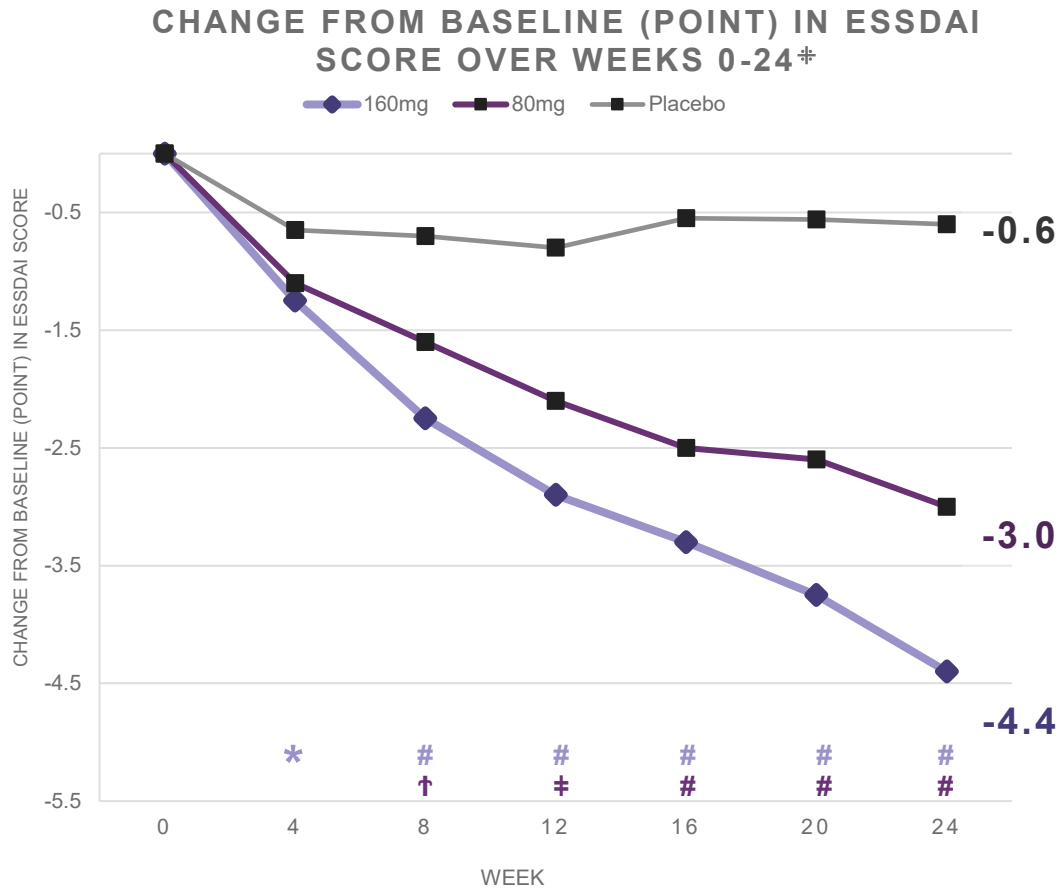
■ B Cell Modulation ■ FcRn ■ CD40 Ligand ■ BAFF-R

ESSPRI, EULAR Sjögren's Syndrome Patient Reported Index; pSjD, primary Sjögren's disease; Efgartigimod - RHO; Nipocalimab - Bowman 2022, Lancet; Ianalumab - St. Clair 2024, Nature and Grader-Beck 2025 ACR; Dazodalibep - Xu 2024 Rheumatology; Ianalumab Phase 3 – ACR 2025; *Median clinESSDAI reported



Deep, Consistent ESSDAI Reduction Through 48 Weeks

7x greater improvement means fewer active symptoms and broader systemic relief for patients



(* $P<0.05$, $\uparrow P<0.01$, $\pm P<0.001$, $\# P<0.0001$)

Placebo*: Participants randomized to the placebo group. *The analysis of change from baseline in ESSDAI score over Weeks 0-24 was based on the estimate population (EP). The MMRM method was used and missing data were not imputed. *The analysis of change from baseline in ESSDAI score over Weeks 0-48 was based on the estimate population (EP). The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.

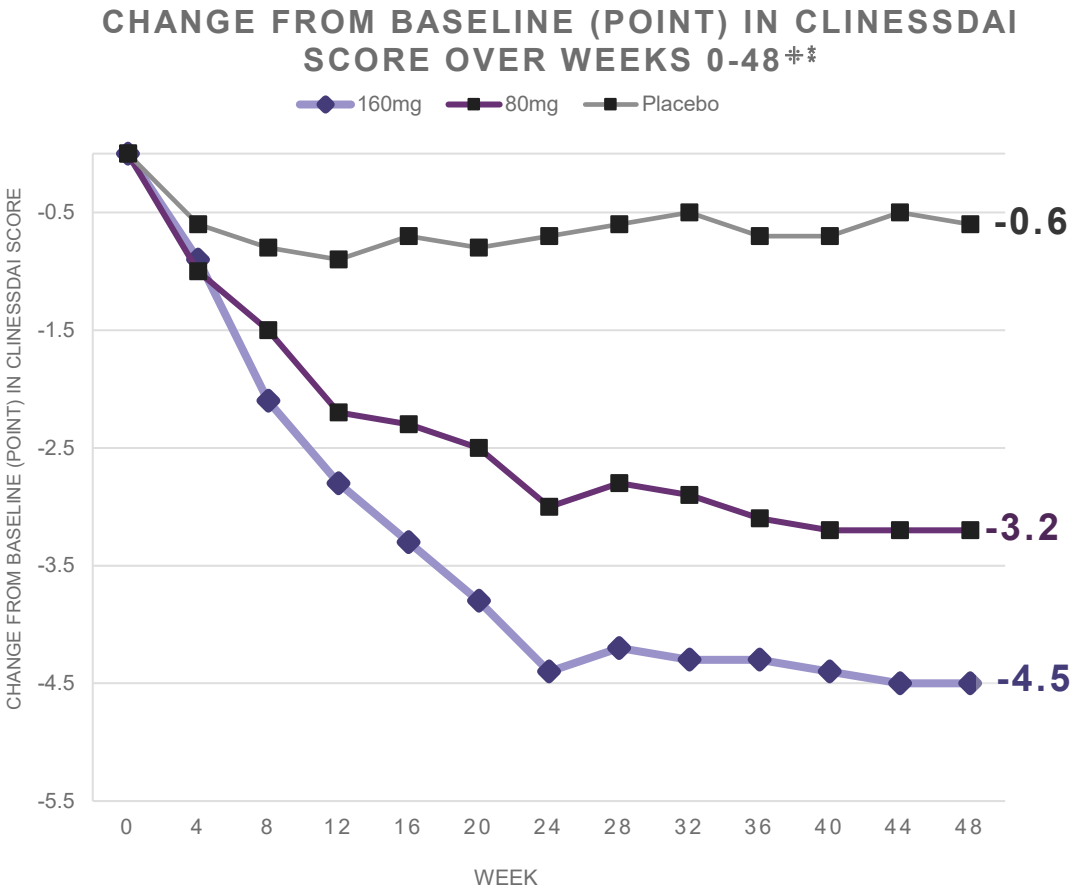


Deep, Consistent ClinESSDAI Reduction Through 48 Weeks

Sustained improvement in clinical benefit beyond serologic change

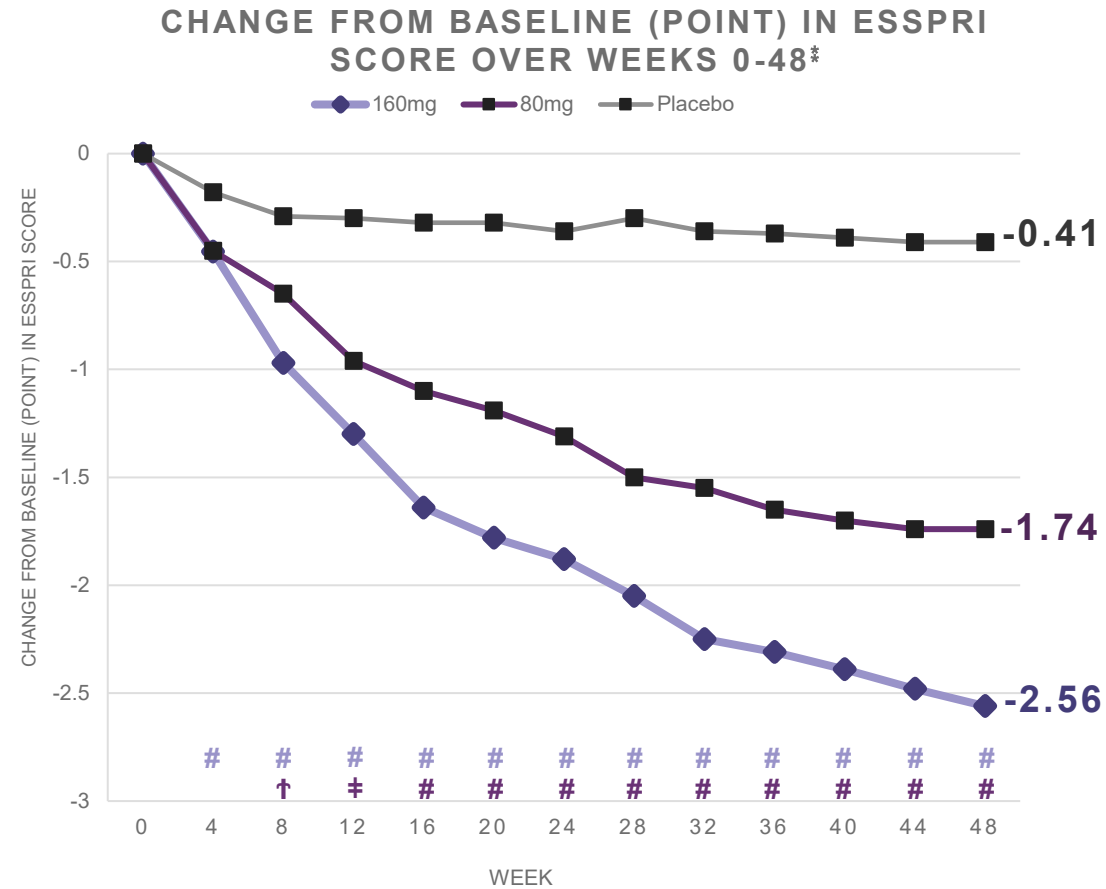
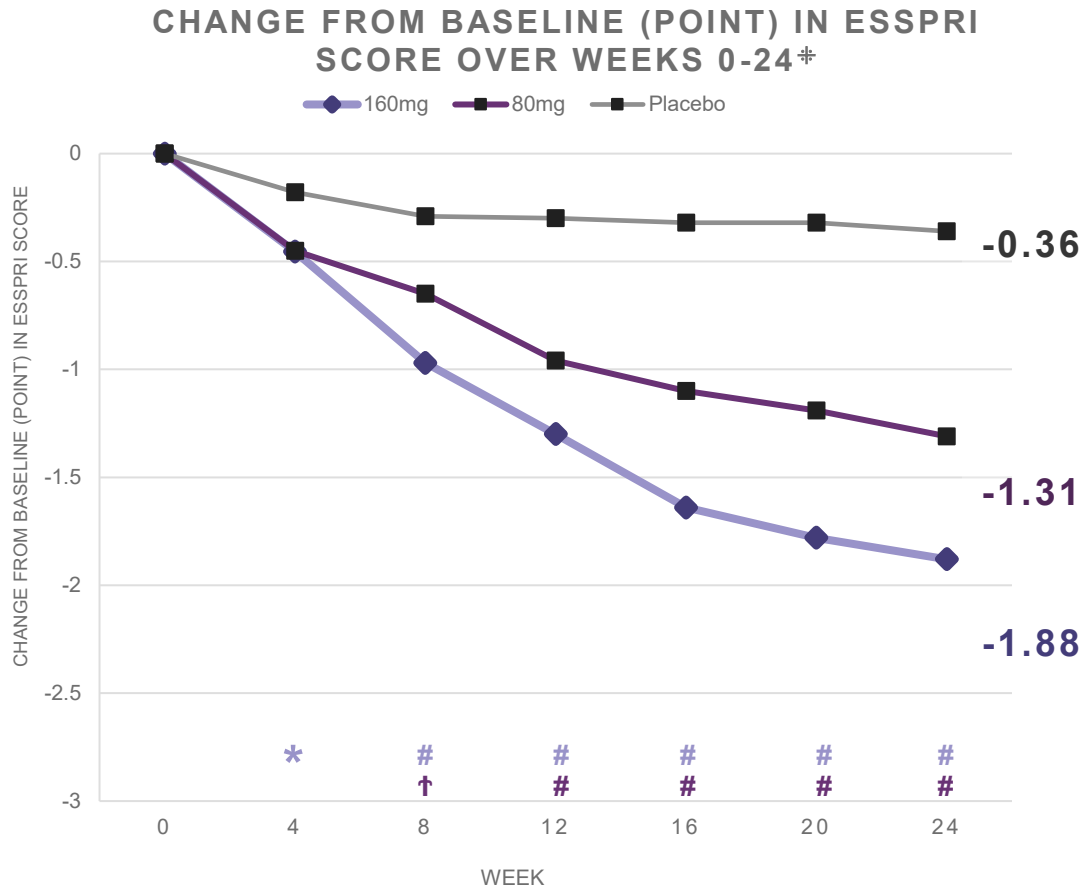
**ESSDAI and ClinESSDAI
remain nearly identical at week 48**
-4.6 vs -4.5

ClinESSDAI excludes biological domain (IgG, complement), representing a more sensitive measure of pure clinical disease activity



Sustained Improvement in ESSPRI Through 48 Weeks

Reduction in patient-reported fatigue, pain, and dryness by ~2.6 points at one year



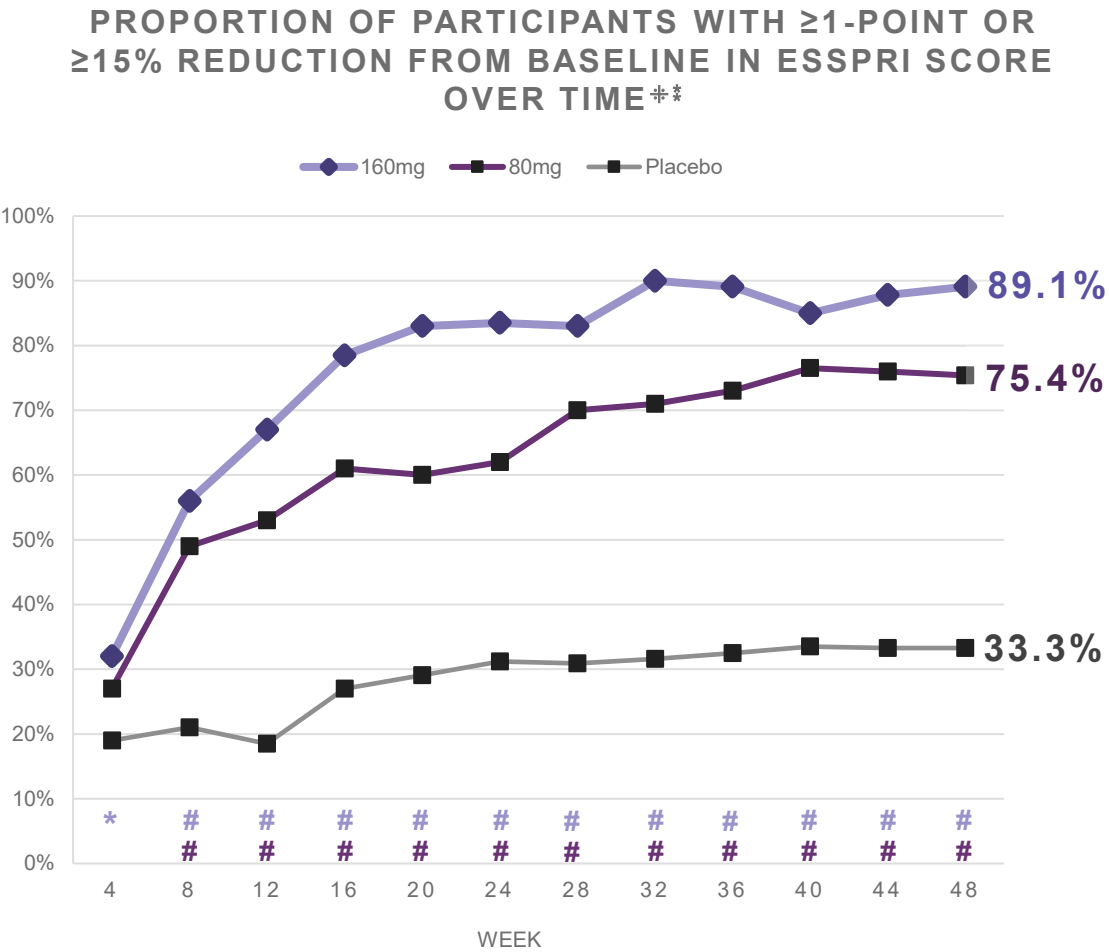
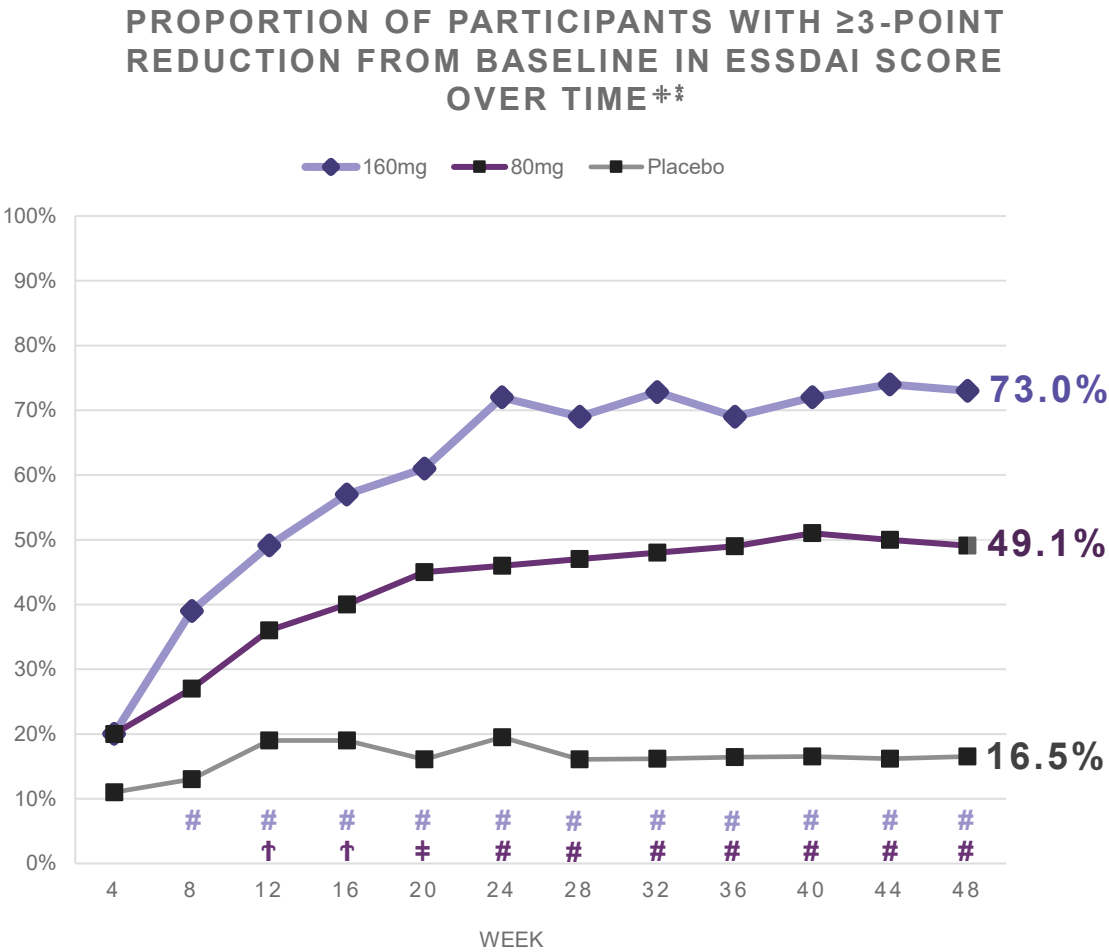
(* $P < 0.05$, † $P < 0.01$, ‡ $P < 0.001$, # $P < 0.0001$)

Placebo*: Participants randomized to the placebo group. ‡ The analysis of change from baseline in ESSPRI score over Weeks 0-24 was based on the estimate population (EP). The MMRM method was used and missing data were not imputed. ‡ The analysis of change from baseline in ESSPRI score over Weeks 0-48 was based on the estimate population (EP). The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.



Early, Broad Symptom Improvements Observed with Telitacept

Nearly 90% of patients report improvement as physicians confirm disease control in 3 out of 4 patients



(* $P<0.05$, † $P<0.01$, ‡ $P<0.001$, # $P<0.0001$)

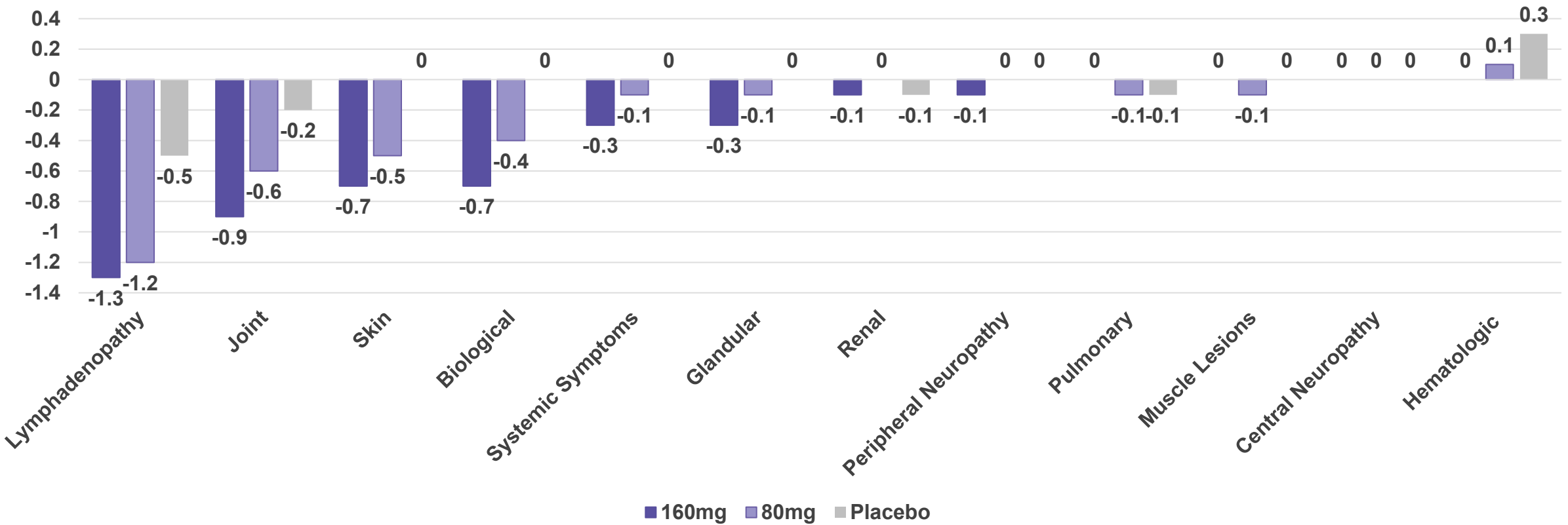
Placebo*: Participants randomized to the placebo group. ‡The analysis of change from baseline in ESSDAI and ESSPRI score over Weeks 0-24 was based on the estimate population (EP). The MMRM method was used and missing data were not imputed. ‡The analysis of change from baseline in ESSDAI and ESSPRI score over Weeks 0-48 was based on the estimate population (EP). The post-switching data for the two telitacept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.



BAFF/APRIL MOA Yields Improvement Across Key ESSDAI Domains

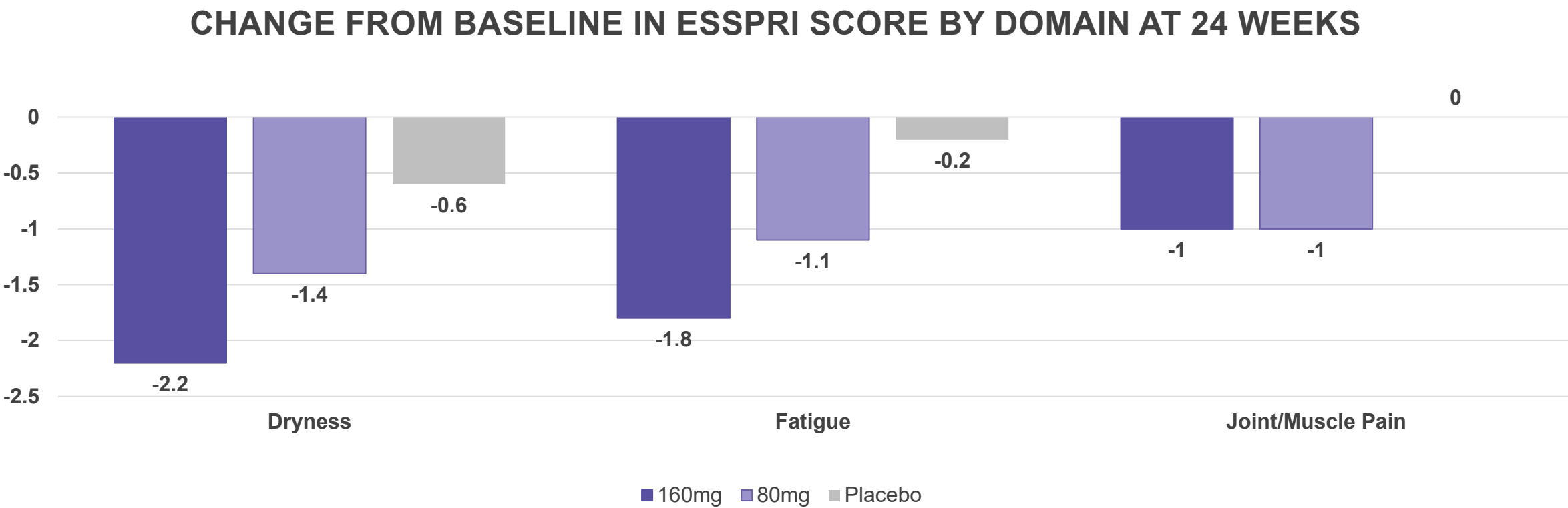
Lack of domain worsening supports balanced B cell modulation without over-suppression

CHANGE FROM BASELINE IN ESSDAI SCORE BY DOMAIN AT 24 WEEKS



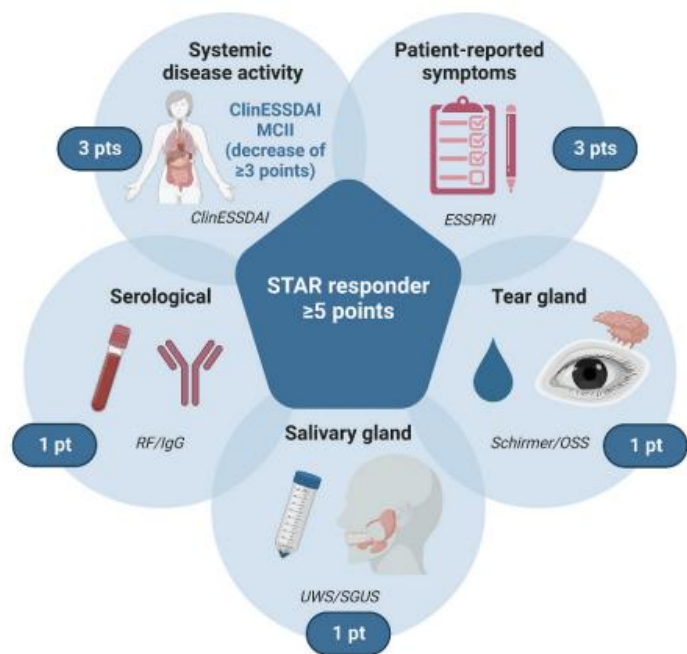
BAFF/APRIL MOA Associated With Improvement Across All ESSPRI Domains

Meaningful improvement across dryness, fatigue, and pain – symptoms that define daily patient life



STAR: Exploratory Endpoint Demonstrates Multi-Domain Improvement

Nearly 3 in 4 patients achieved ≥ 5 point response



- STAR integrates systemic disease activity (ClinESDAI), symptoms (ESSPRI), and glandular function (Schirmer's / salivary flow)
- Systemic disease activity and patient-reported symptoms are considered as major items (3 points per item) and the rest as minor items (1 point per item)
- Patients are classified as STAR responders when they reach ≥ 5 of 9 points

STAR Responders (%) at Week 24

Telitacicept 160mg

74.8%

Telitacicept 80mg

53.2%

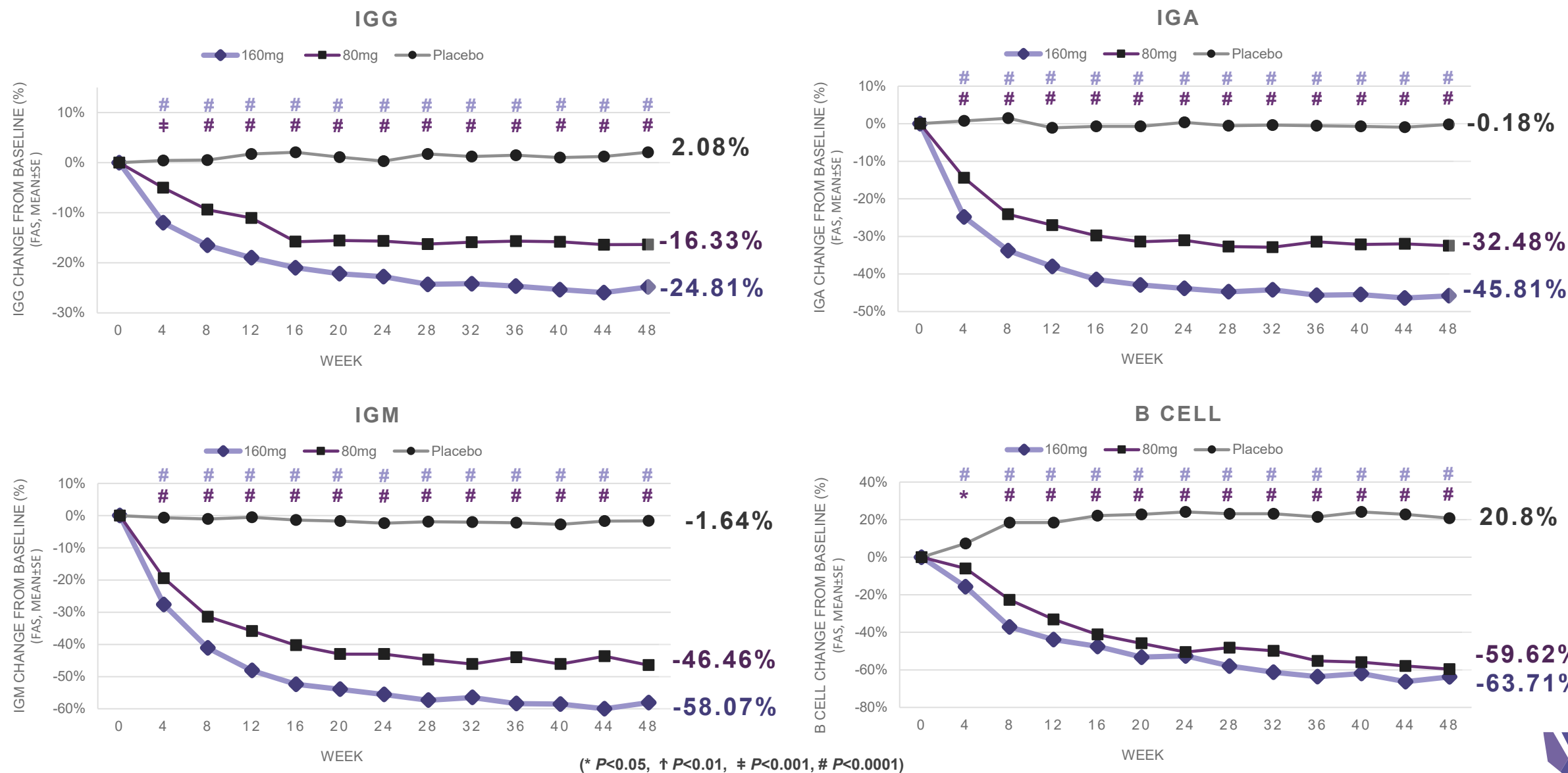
Placebo Group

21.3%

Placebo*: Participants randomized to the placebo group. A STAR responder was defined as a participant with a total STAR score of ≥ 5 points. Participants with missing scores in any domain (except those with a total STAR score of ≥ 5 points despite missing scores in some domains) were imputed as "non-responders". The responder proportion analysis for Weeks 0-24 was based on estimate population (EP). The stratum-adjusted between-group difference was tested with the CMH method. The responder proportion analysis for Weeks 28-48 was based on the estimate population (EP). The stratum-adjusted between-group difference was tested with the CMH method. The post-switching data for the two telitacicept groups and the placebo group were handled with the LOCF method, i.e. imputing all the post-switching values with the most recent pre-switching results.



Consistent Reduction in IgG, IgA, IgM, and B Cells



Favorable Safety Profile in pSjD

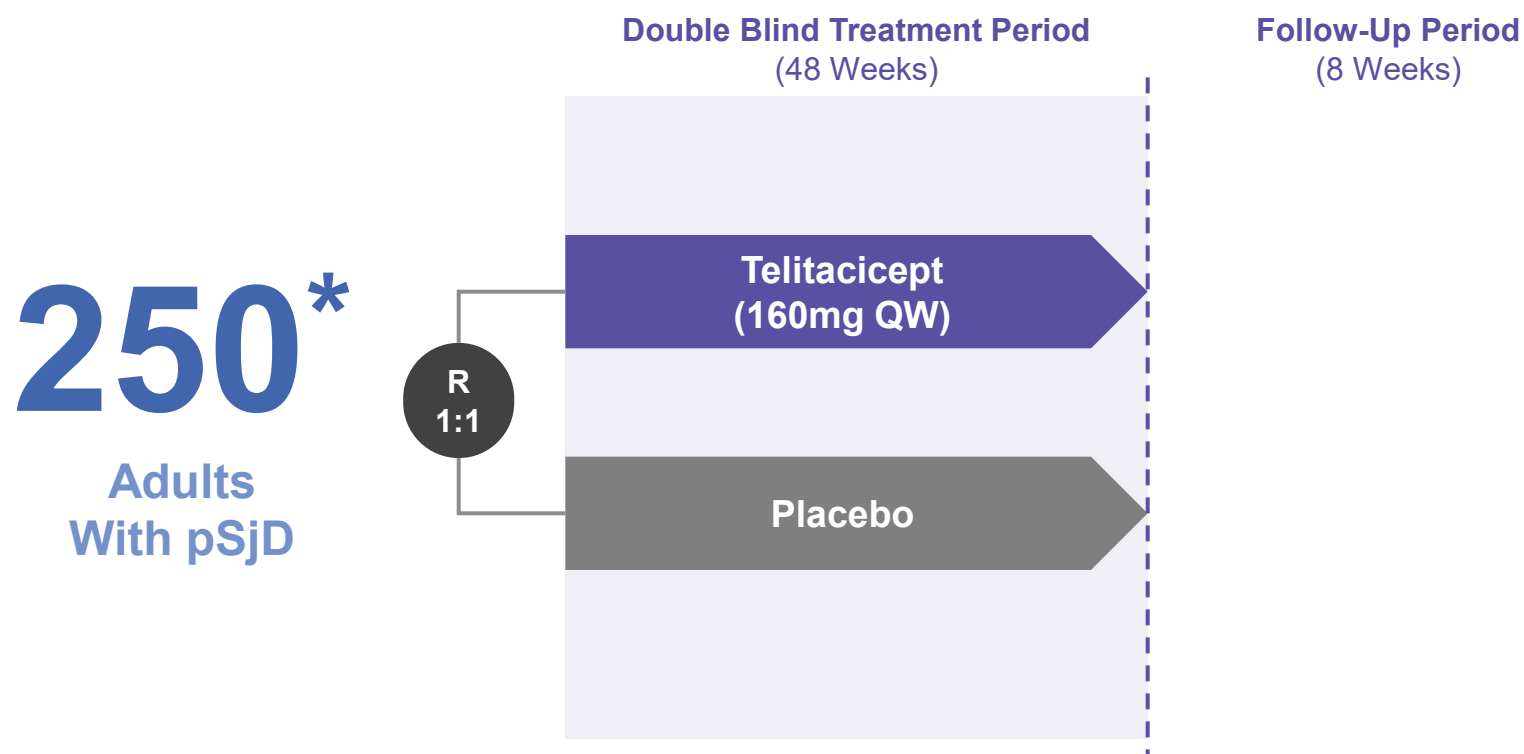
Consistent with data from clinical trials in SLE, RA, gMG, and IgAN, and post-marketing data

	Telitacicept 160mg (N=127)	Telitacicept 80 mg (N=126)	Placebo Group (N=127)
TEAE, n(%)	122 (96.1)	119 (94.4)	112 (88.2)
TRAE, n(%)	107 (84.3)	106 (84.1)	74 (58.3)
TESAE, n(%)	11 (8.7)	14 (11.1)	10 (7.9)
TRSAE, n(%)	2 (1.6)	5 (4.0)	4 (3.1)
Severe TEAE, n(%)	3 (2.4)	5 (4.0)	3 (2.4)
Severe TRAE, n(%)	0	1 (0.8)	1 (0.8)
Death, n(%)	0(0)	0(0)	0(0)
Common TEAE (incidence ≥10% in any group)			
Upper respiratory tract infections, n(%)	80 (63.0)	85 (67.5)	74 (58.3)
Urinary tract infection, n(%)	8 (6.3)	15 (11.9)	7 (5.5)
Cough, n(%)	11 (8.7)	14 (11.1)	8 (6.3)
Hepatic function abnormal, n(%)	7 (5.5)	16 (12.7)	7 (5.5)
Injection site reaction, n(%)	53 (41.7)	51 (40.5)	5 (3.9)
Pyrexia, n(%)	4 (3.1)	14 (11.1)	4 (3.1)



UPSTREAM SjD: Global Phase 3 Trial in Primary Sjögren's Disease

Potential best- and first-in-class BAFF/APRIL inhibitor; randomized, double-blind, placebo-controlled study



Primary Endpoint

- Change from baseline in ESSDAI at 48 weeks

Secondary Endpoints

- Changes from baseline in ESSPRI, Whole Salivary Flow, Unstimulated Whole Salivary Flow, Schirmer's Test, SF-36, FACIT-F, and MFI-20 at 48 weeks
- Proportion of patients who achieve >3 domains of CRESS and ≥5 domains of STAR

Enrollment Ongoing; First Patient Dosed in 1Q26

*Estimated. ESSDAI, EULAR Sjögren's syndrome disease activity index; ESSPRI, EULAR Sjögren's Syndrome Patient Reported Index; MFI-20, multidimensional fatigue inventory; PGA, physician's global assessment; PaGA, patient's global assessment; SF-36, 36-item short-form; pSjD, primary Sjögren's disease; QW, per week.

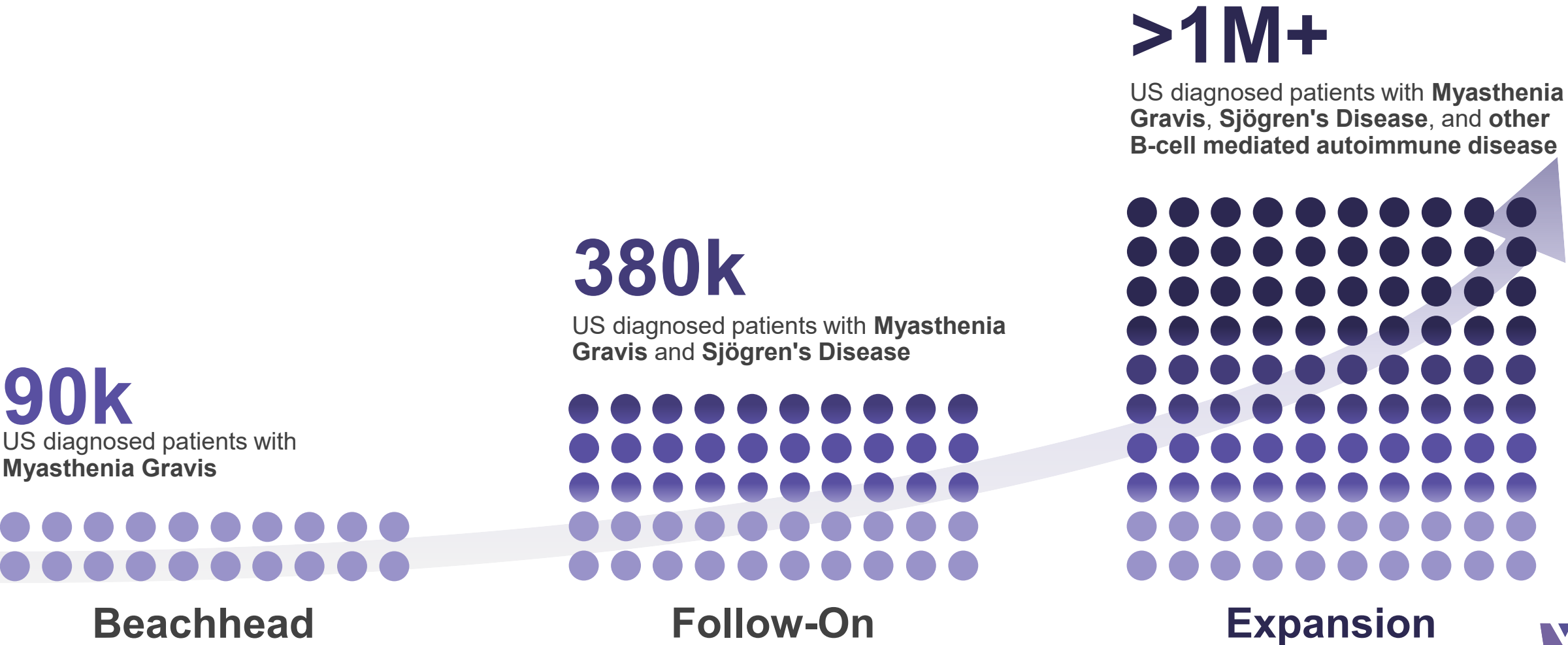
Sjögren's: Ianalumab Shows Consistent Activity Independent of Region

No signal of reduced effect in Novartis NEPTUNUS China subgroup

		Ianalumab				Telitacicept	
		Global		China Subgroup		China Phase 3	
		300mg QM N=305	Placebo N=307	300mg QM N=29	Placebo N=36	160mg N=127	Placebo N=127
ESSDAI	Mean Change from Baseline	-6.5	-5.3				
	Δ vs Placebo	-1.2		-1.4		-3.8	
	ESSDAI <5	53.4%	45.1%	42.7%	30.3%	49.6%	10.9%
ESSPRI	Mean Change from Baseline	-1.73	-1.47	-0.62	-0.83	-1.88	-0.36
	Δ vs Placebo	-0.26		0.21		-1.52	
SSSD	Mean Change from Baseline	-1.52	-1.29	-0.75	-0.71		
	Δ vs Placebo	-0.23		-0.04			
PaGA	Mean Change from Baseline	-13.5	-8.7	-7.9	-4		
	Δ vs Placebo	-4.9		-3.9			
PhGA	Mean Change from Baseline	-29.2	-25.9	-17.1	-16.5		
	Δ vs Placebo	-3.3		-0.6			
Safety (Week 52)	Any Adverse Event (%)	85.9%	83.4%	100.0%	97.2%	96.1%	88.2%
	Series Adverse Events (%)	6.9%	9.8%	6.9%	11.1%	2.4%	2.4%
	AEs Leading to Discontinuation	6.2%	3.6%	3.4%	2.8%	3.1%	3.9%
	Deaths	0	1	0	0		



Significant Near-Term Expansion Opportunities



2026:

Advancing The Leading BAFF/APRIL Inhibitor

~\$515M

Runway into Early 2029*

01

MYASTHENIA GRAVIS

Global Phase 3 Topline Data in 1H27

Best-in-Disease, Commercially Approved in China

02

SJÖGREN'S DISEASE

Global Phase 3 Initiated; FPD in 1Q26

Best-in-Disease, Commercially Approved in China

03

EXPANSION OPPORTUNITIES

Broad Potential Across B Cell-Driven Immune Diseases

Indication Focus on High Unmet Need And Clinical Value



Thank You.



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