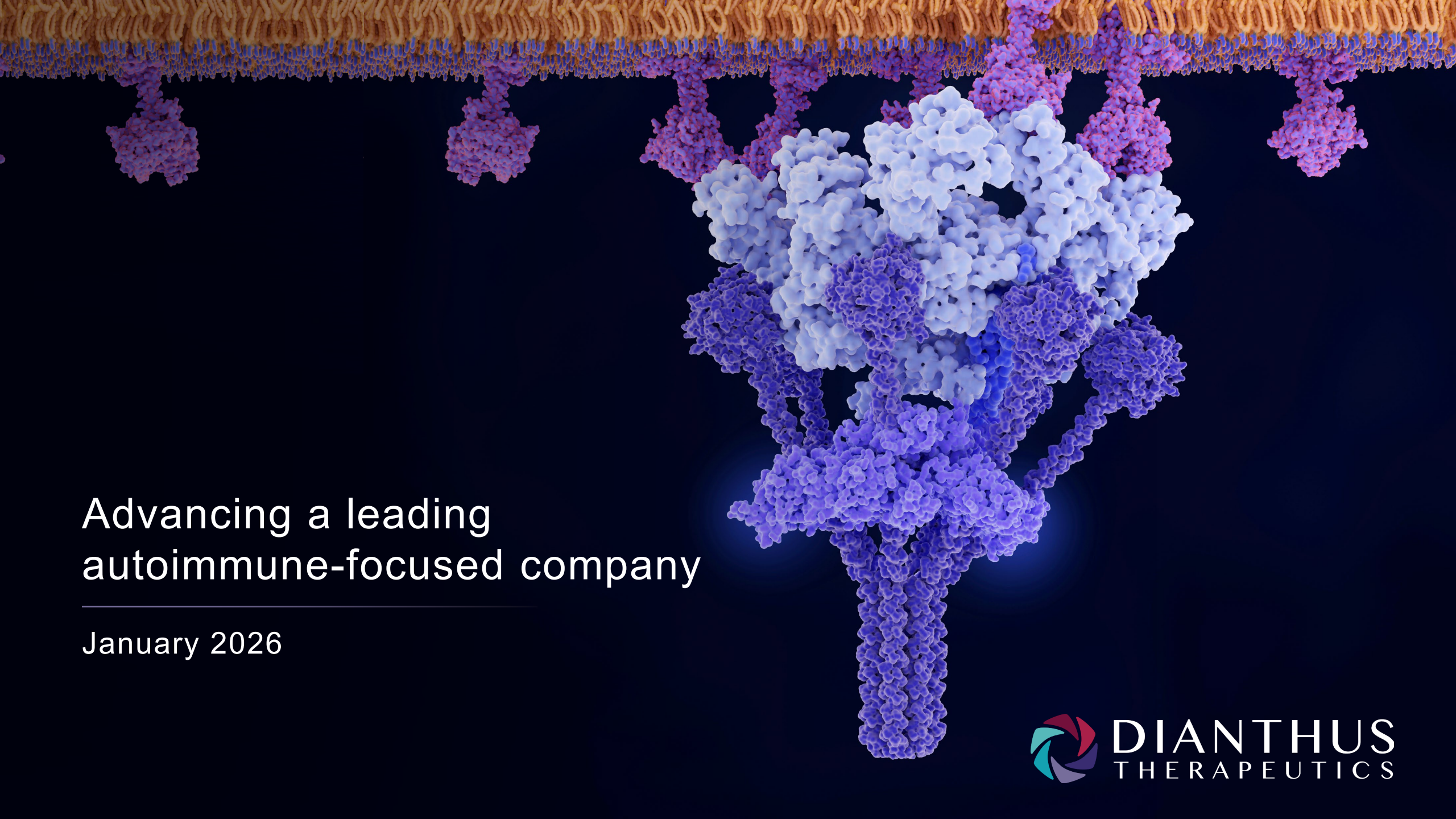


Advancing a leading autoimmune-focused company

January 2026

DIANTHUS THERAPEUTICS

Forward-looking statements

Certain statements in this presentation, other than purely historical information, may constitute “forward-looking statements” within the meaning of the federal securities laws, including for purposes of the safe harbor provisions under the United States Private Securities Litigation Reform Act of 1995, express or implied statements regarding future plans and prospects, including statements regarding the expectations or plans for discovery, preclinical studies, clinical trials and research and development programs, in particular with respect to claseprubart and DNTH212, and any developments or results in connection therewith, including the target product profile and administration of claseprubart and DNTH212; the anticipated timing of the initiation and results from those studies and trials; expectations regarding the clinical trial designs or indications; expectations regarding the time period over which the Company’s capital resources are expected to be sufficient to fund its anticipated operations; and expectations regarding market size, patient population size, and potential opportunities for complement therapies, in particular with respect to claseprubart and DNTH212. Claseprubart and DNTH212 are investigational agents that are not approved as therapies in any indication in any jurisdiction worldwide. The words “opportunity,” “potential,” “milestones,” “runway,” “will,” “anticipate,” “achieve,” “near-term,” “catalysts,” “pursue,” “pipeline,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “might,” “plan,” “possible,” “predict,” “project,” “should,” “strive,” “would,” “aim,” “target,” “commit,” and similar expressions (including the negatives of these terms or variations of them) generally identify forward-looking statements, but the absence of these words does not mean that statement is not forward looking.

Actual results could differ materially from those included in the forward-looking statements due to various factors, risks and uncertainties, including, but not limited to, that preclinical testing of claseprubart and DNTH212 and data from clinical trials may not be predictive of the results or success of ongoing or later clinical trials, that the development of claseprubart or DNTH212 may take longer and/or cost more than planned, that the Company or its partner may be unable to successfully complete the clinical development of the Company’s compounds, that the Company or its partner may be delayed in initiating, enrolling or completing its planned clinical trials, and that the Company's compounds may not receive regulatory approval or become commercially successful products. These and other risks and uncertainties are identified under the heading "Risk Factors" included in the Company’s Annual Report on Form 10-K for the period ended December 31, 2024, and other filings that the Company has made and may make with the SEC in the future. Nothing in this presentation should be regarded as a representation by any person that the forward-looking statements set forth herein will be achieved or that any of the contemplated results of such forward-looking statements will be achieved.

Nothing in this Presentation should be regarded as a representation by any person that the forward-looking statements set forth herein will be achieved or that any of the contemplated results of such forward-looking statements will be achieved. Dianthus undertakes no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events or otherwise, except as required by law.

Advancing a leading autoimmune-focused company

Developing two autoimmune therapeutics with best-in-class, pipeline-in-a-product potential and targeting patient-friendly, infrequent S.C. self-administration

Claseprubart
(aC1s mAb)

- **Highly potent, ~8-week half-life, classical pathway (CP) inhibitor targeting active C1s**
- **Validated pipeline-in-a-product** potential with **positive Ph. 2 gMG results** and clinical **PoC for CP inhibition in CIDP and MMN**
- **Clinical and *in vitro* head-to-head data** support potential for a more **effective and convenient** biologic with no boxed warning/REMS
- Targeting **convenience** of a **single, self-administered S.C. 300mg/2mL autoinjector dosed every 2 or 4 weeks**

DNTH212
(BDCA2 and BAFF/APRIL bifunctional fusion protein)

- **Bifunctional BDCA2 and BAFF/APRIL inhibitor** targeting two **validated** pathways
- Potential for **enhanced efficacy** from complementary mechanisms **targeting innate and adaptive immune systems**
- Demonstrated **superior *in vitro* pDC depletion** vs. litifilimab and **superior serum Ig inhibition** vs. povetacicept in NHPs
- **Pipeline-in-a-product opportunity across multiple diseases** with potential for **Q4W or less frequent S.C. self-administration**

Claseprubart 2026 milestones:

Ph. 3 gMG trial initiation (’26), Ph. 3 CIDP interim responder analysis (Q2’26) and Ph. 2 MMN top-line results (2H’26)

DNTH212 2026 milestones:

Update on indication prioritization (1H’26) and Ph. 1 healthy volunteer study top-line results (2H’26)

Strong financial position with cash of ~$514M[1] and runway into 2028 expected to fund multiple near-term catalysts

1. Estimated cash includes preliminary and unaudited cash, cash equivalents and investments as of December 31, 2025

Two clinical-stage candidates with best-in-class, pipeline-in-a-product potential

	Claseprubart			DNTH212
Indications	**Generalized Myasthenia Gravis**	**Chronic Inflammatory Demyelinating Polyneuropathy**	**Multifocal Motor Neuropathy**	**Update on Indication Prioritization in 1H'26**
	>100,000 U.S. patients	***>40,000 U.S. patients***	***>10,000 U.S. patients***	
Market Insight	Multi-billion $, growing market with opportunity for a best-in-class, convenient therapy to expand use of first-line biologics	Sanofi's riliprubart validated active C1s inhibition with robust efficacy in patients who were refractory, stable, as well as naïve to IVIG, the standard of care	Empasiprubart, a C2 inhibitor demonstrated impressive efficacy in MMN, validating classical pathway inhibition	Dual mechanism targeting innate and adaptive immune systems, with superior *in vitro* pDC depletion vs. litifilimab and superior serum Ig inhibition vs. povetacicept in NHPs
Our Opportunity	Ph. 2 data support potential for best-in-class efficacy with 300mg/2mL Q2W demonstrating rapid, robust, continuous symptom control with convenient, infrequent S.C. dosing and administration and a potentially differentiated safety profile	Demonstrated superiority vs. riliprubart in multiple head-to-head *in vitro* PD potency experiments, with potential to address unmet needs of CIDP patients including refractory to IVIG	Demonstrated superiority vs. empasiprubart in head-to-head *in vitro* classical pathway potency experiment, with potential to be a best-in-class therapeutic and capture majority of MMN market	Validation of both BDCA2 and BAFF/APRIL targeted therapies support bifunctional approach, with potential for best-in-class efficacy in various diseases vs. only targeting innate or adaptive immune system
Next Milestone	**Ph. 3 Initiation in 2026**	**Ph. 3 Interim Responder Analysis in Q2'26**	**Ph. 2 Data in 2H'26**	**Ph. 1 HV Top-line Results in 2H'26**

gMG: >100,000 gMG U.S. patients from Komodo claims data accessed 2013-2025; approx. 85% of gMG patients have AChR autoantibody-driven disease https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7033452/#
CIDP & MMN: Komodo claims data 2013-2025, adjusted to account for 70% capture of real-world patient counts for biologic treated patients; CIDP adjusted to account for 27% misdiagnosed

Claseprubart:
Building a Best-in-Class Neuromuscular Franchise

Pursuing the *power of consistent control*…with one-click!

claseprubart

CONFIDENCE

Aim for Potent, Rapid, Consistent Efficacy

Potential for Best-in-Class Profile

Broad Potential in Neuromuscular Diseases

CLASSICAL

Upstream Inhibition of Classical Pathway Only

Potential to Preserve Immune Function

Targeting No Boxed Warning or REMS

CONVENIENCE

<10-Second Autoinjector

One-Click Every 2 or 4 Weeks

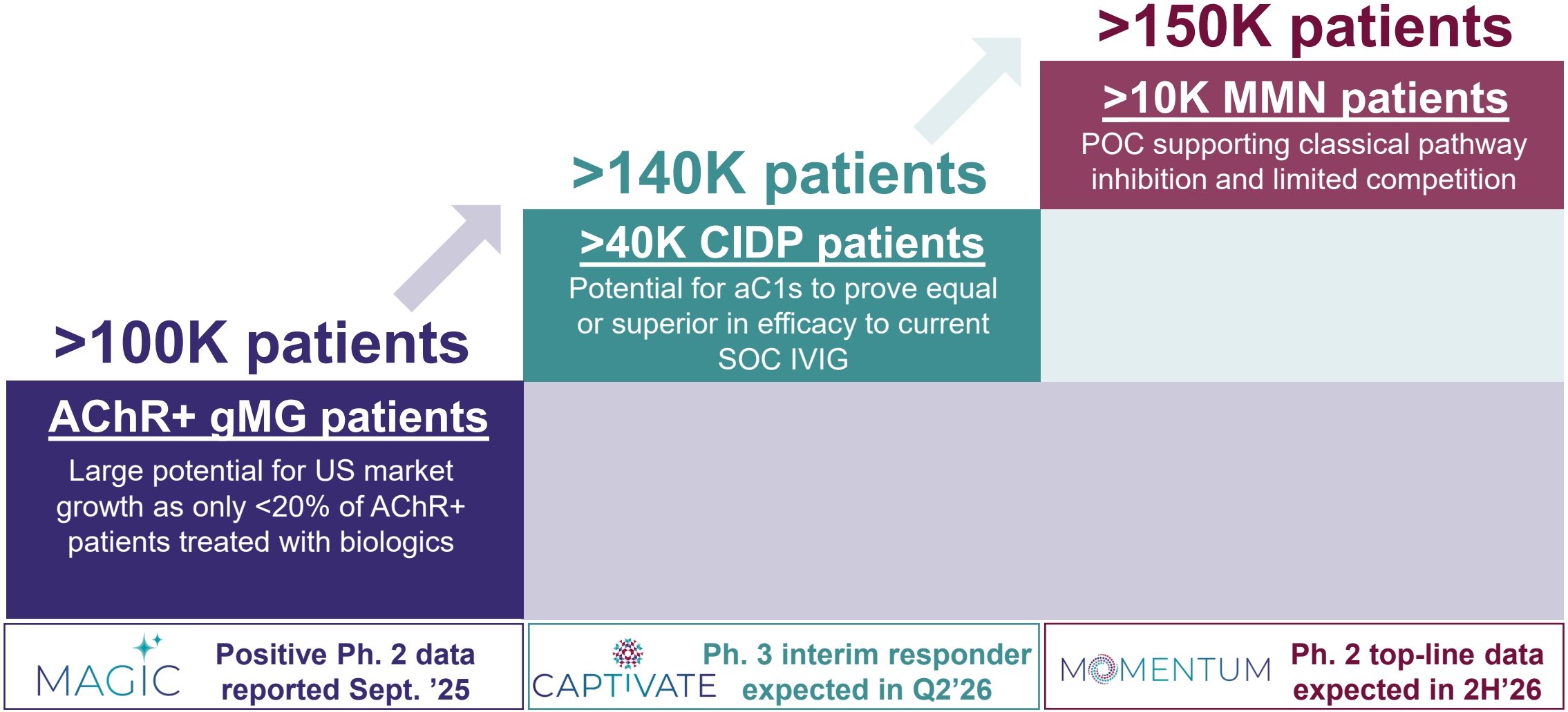
Self-Administered At Home *or* On-the-Go

Targeting a best-in-class, first-line biologic treatment for neuromuscular diseases

Autoinjector image for illustration purposes only. Autoinjector for claseprubart administration is anticipated to be SHL Medical's Molly technology, patented or patent pending in the US, China, India, Japan, Korea, Taiwan and at the European Patent Office. Claseprubart is an investigational agent that is not approved as a therapy in any indication in any jurisdiction worldwide.

Claseprubart has opportunity to compete as a first-line biologic in large and growing US neuromuscular market

gMG is just the first step in building a leading neuromuscular franchise with claseprubart



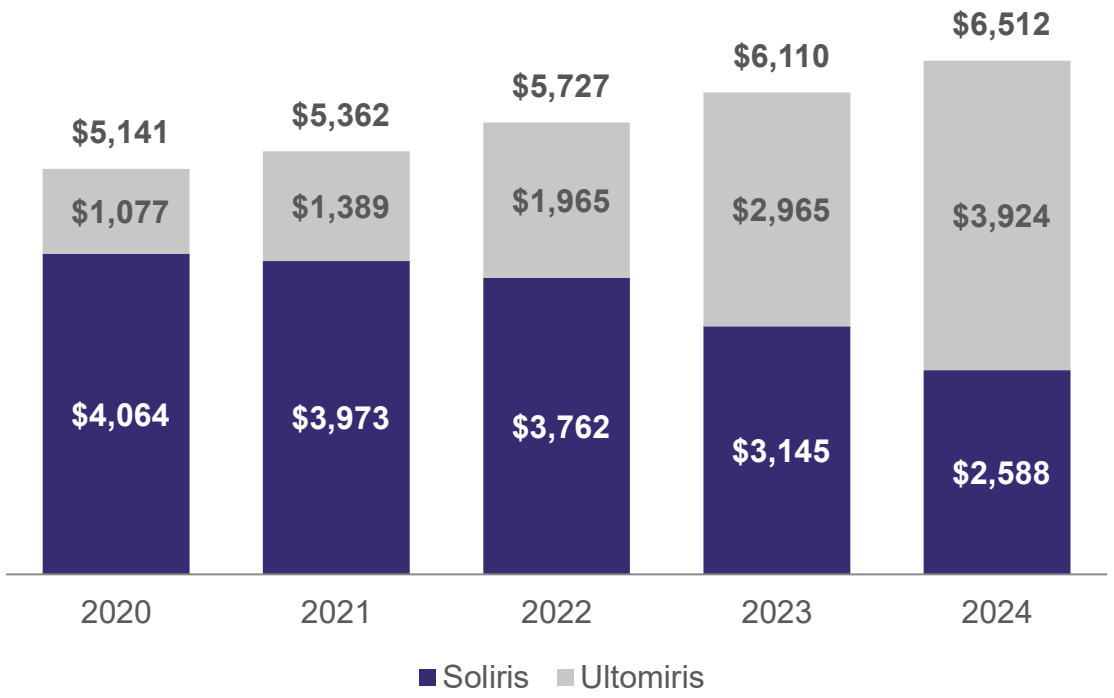
Figures represent U.S. estimated patients only. gMG: >100,000 gMG U.S. patients from Komodo claims data accessed 2013-2025; approx. 85% of gMG patients have AChR autoantibody-driven disease
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7033452/#
CIDP & MMN: Komodo claims data 2013-2025, adjusted to account for 70% capture of real-world patient counts for biologic treated patients; CIDP adjusted to account for 27% misdiagnosed

Claseprubart: Opportunity to be a Best-in-Class, First-Line Biologic for Generalized Myasthenia Gravis

Ultomiris is the leading blockbuster complement inhibitor, with continued growth driven by first-line biologic use in gMG

C5 Inhibitors (Ultomiris & Soliris) Global Sales ($M)

~1/3 of sales in gMG[1]; sales growth driven by U.S. biologic naïve gMG patients[2]



"Currently, **less than 20% of (gMG) patients are on branded treatments, and we expect this to increase to** approximately **50%** in the **next 3 years**. Additionally, **self-administered** medicine represent only a small part of this market today, and we expect this **segment to grow substantially**."

Q2 2025 financial results transcript

Ultomiris sales **grew 33%** in Q4'24… "driven by **neurology** indications, with the **vast majority of growth coming from generalized myasthenia gravis patients who are naive to branded treatments**."

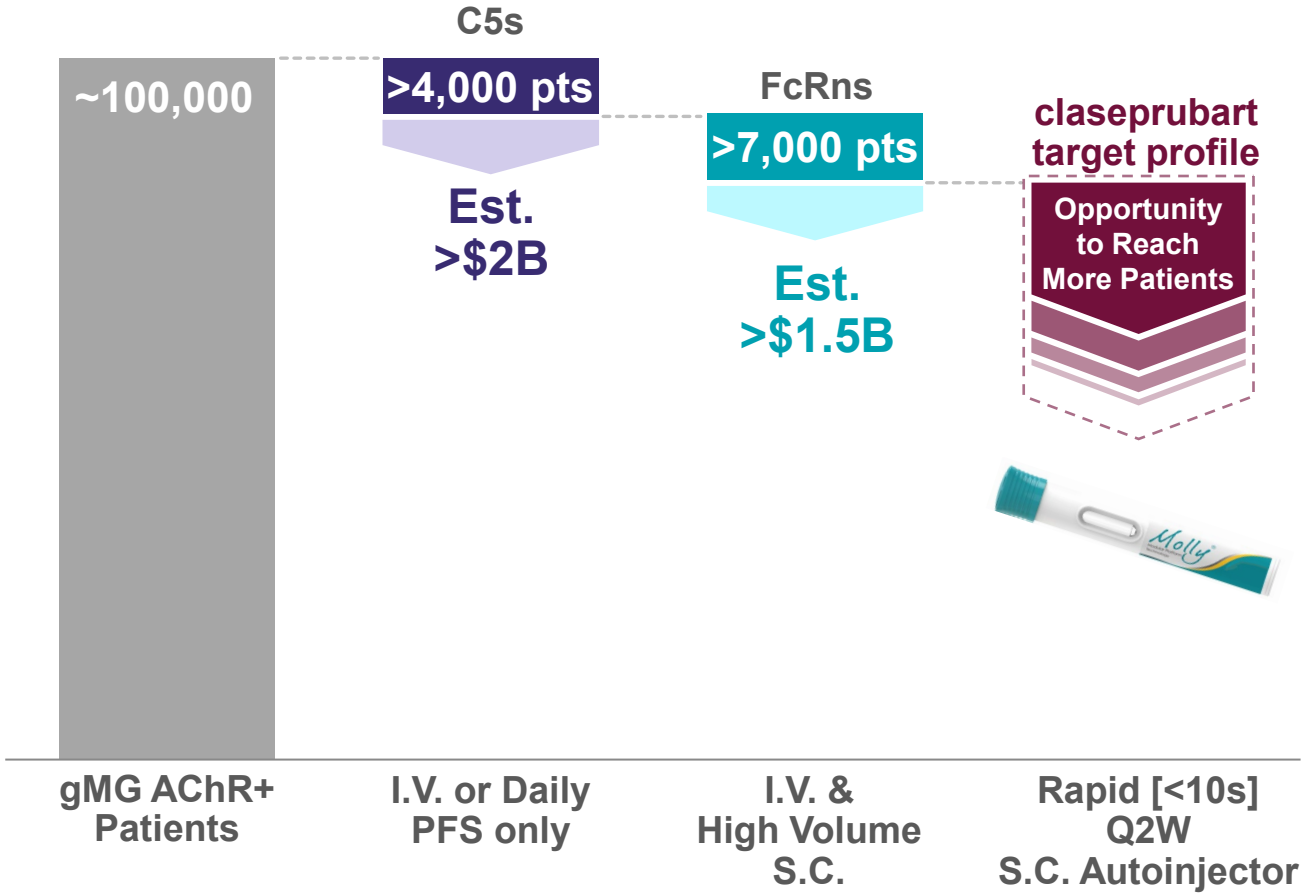
Q4 2024 financial results transcript

AstraZeneca

Soliris & Ultomiris 2021 sales account for 1/1 – 6/30 & 7/21 – 12/31. Evaluate Pharma
Soliris / Ultomiris are approved in gMG, aHUS, NMOSD and PNH
1. Wall Street research estimate; 2. Astra Zeneca Q4 2024 results

The multibillion-dollar US gMG market has significant potential to expand as <20% of AChR+ patients[1] currently treated with biologics

Significant opportunity for a highly differentiated, more patient-friendly biologic to expand use of biologics in gMG



US gMG Market Opportunity

- Current biologics market accounts for >$3.5B[2]
- Yet >80% of AChR+ patients remain untreated with a biologic[1]
- Majority of C5 and FcRn sales are via inconvenient I.V. or high-volume S.C. administration
- Opportunity to expand the market with a patient friendly and easier to use option

Note: Positioning of claseprubart's target profile is illustrative. (1) Komodo claims data accessed 2013-2025, adjusted to account for 70% capture of real-world patient counts; AChR+ 85% of gMG; (2) 2024 US financial reports on gMG drugs. Soliris/Ultomiris adjusted for relative size of MG based on claims. Vyvgart adusted for estimated CIDP sales.

Survey of US Neurologists confirms significant opportunity for differentiated biologic in gMG market

Claseprubart aims to address the significant unmet needs in the gMG market

Total Neurologists 81

Neuromuscular specialists	67%
Generalists	33%
Academic	40%
Community based	60%

Sample Demographics

~17 years in active clinical practice (post-residency), on average

~93% of professional time spent providing direct patient care, on average

~40 gMG patients seen in the past 12 months, on average

~81%

of Neurologists believe patients would benefit from treatment options with **greater durability of symptom relief**

~78%

of Neurologists believe patients would benefit **from a more convenient treatment option**

~67%

of Neurologists would start more patients on a complement inhibitor if they **do not have a boxed warning or REMS requirement**

~72%

of Neurologists **prefer low-volume autoinjector** over high-volume prefilled syringe **due to ease of use & faster injection (i.e. <10 seconds)**

Dianthus 2025 Neurologist MG quantitative survey (n=81, N = 54 Neuromuscular specialists, N = 27 General Neurologists), fielded Q2

MaGic Ph. 2 Results Support a Potentially Best-in-Class Treatment for gMG

MaGic Ph. 2 top-line results support a potentially best-in-class treatment for gMG

Efficacy Endpoints

Strong results support claseprubart potential as a best-in-class complement inhibitor

- Rapid, sustained, statistically significant symptom improvements as measured by MG-ADL, QMG, MSE, MGC, MG-QoL-15r

Safety Endpoints

Generally well tolerated, with a potentially differentiated safety profile

- No encapsulated bacterial infections
- No symptoms indicative of autoimmune activation
- Supports no Boxed Warning or REMS

Optimal Dose

Comparable efficacy & safety across both 300mg/2mL and 600mg/4mL doses

- Target dose of 300mg/2mL Q2W will be in Ph. 3 study
- Supports convenient, infrequent, self-administration with same autoinjector as Dupixent

MaGic Ph. 2 results support a profile with the potential to displace C5 complement inhibitors and compete effectively with FcRns as first-line biologic treatment in growing MG market

Autoinjector for claseprubart administration is anticipated to be SHL Medical's Molly technology, patented or patent pending in the US, China, India, Japan, Korea, Taiwan and at the European Patent Office.
C5 Efficacy data are derived from different clinical trials conducted at different times, with differences in trial design and patient populations.

MaGic is a global Ph. 2 trial in AChR+ gMG patients

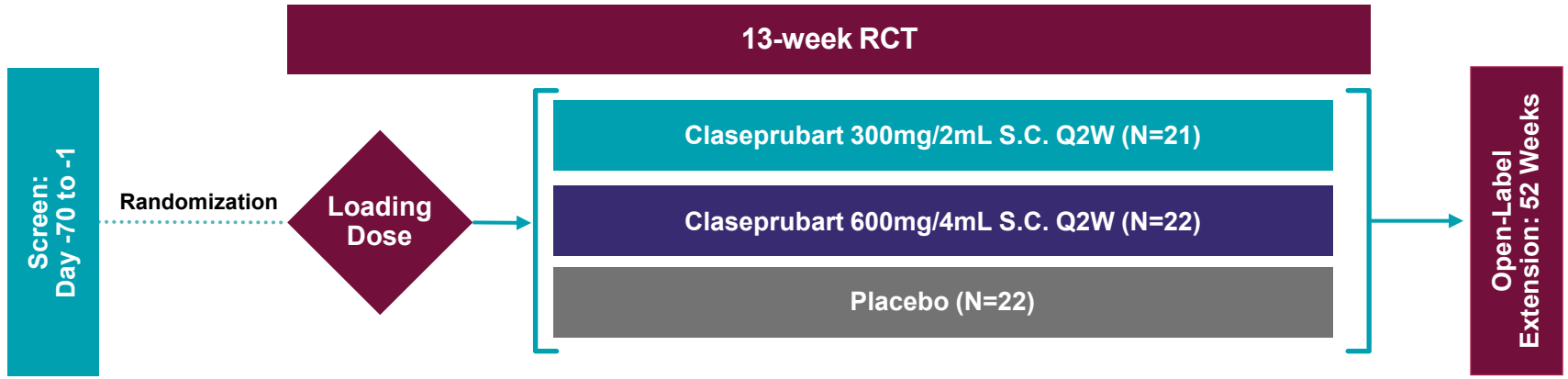
A global, multicenter, randomized, double-blind, placebo-controlled study to evaluate the safety, efficacy, and PK / PD of claseprubart administered S.C. following initial loading dose

Highlights

- **Design:** ~60 male and female subjects randomized to receive either claseprubart or placebo for 13 weeks
- **Inclusion:** ≥18 years old with AChR antibody + gMG
- **Dosing:** 15 or 20mg/kg I.V. Loading Dose followed by 300mg/2mL or 600mg/4mL S.C. Q2W starting Day 7

Endpoints

- **Primary:** Safety
- **Secondary / Exploratory:** Efficacy (MG-ADL, QMG, MSE, MGC, MG-QoL-15r)



MAGIC

Trial enrollment exceeded target, with 65 participants enrolled

MaGic Trial: https://clinicaltrials.gov/study/NCT06282159

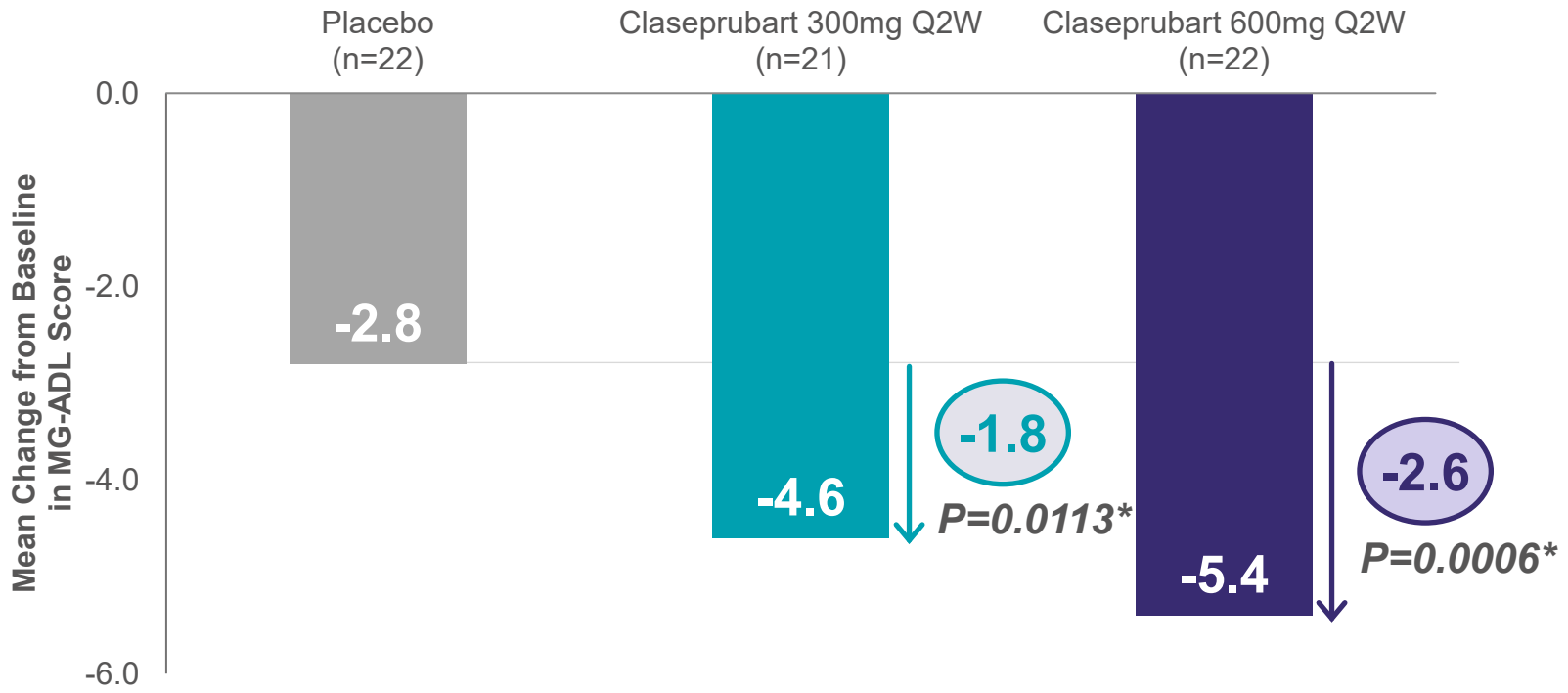
Participant baseline characteristics were generally well balanced across arms

AChR+ gMG participants	Placebo (N=22)	Claseprubart 300mg/2mL Q2W (N=21)	Claseprubart 600mg/4mL Q2W (N=22)
Age, mean (SD), years	52.2 (16.5)	57.1 (13.7)	55.3 (12.0)
Male, n (%)	13 (59%)	14 (67%)	10 (45%)
Weight, mean (SD), pounds	195.0 (48.0)	192.5 (35.5)	179.0 (35.4)
Duration of disease, median (range), years	7.7 (0.4 – 21.2)	3.0 (0.5 – 22.1)	7.6 (1.0 – 37.3)
MG-ADL score at baseline, mean (SD)	8.5 (2.9)	8.2 (2.2)	8.4 (2.6)
QMG score at baseline, mean (SD)	14.2 (5.8)	12.2 (2.7)	12.2 (3.6)
MG Composite score at baseline, mean (SD)	15.0 (7.8)	16.3 (4.5)	16.0 (5.3)
MG-QoL-15r score at baseline, mean (SD)	14.3 (7.0)	15.4 (6.6)	14.9 (5.9)
MGFA class at screening, n (%)			
II	7 (32%)	11 (52%)	12 (55%)
III	12 (55%)	10 (48%)	9 (41%)
IVa	3 (14%)	0 (0%)	1 (5%)
Prior thymectomy, n (%)	8 (36%)	6 (29%)	7 (32%)
Baseline corticosteroid use, n (%)	19 (86%)	17 (81%)	20 (91%)
Number of ISTs at baseline, n (%)			
1	11 (50%)	10 (48%)	12 (55%)
>1	11 (50%)	11 (52%)	10 (45%)
FcRn use in prior 24 months, n (%)	0 (0%)	1 (5%)	0 (0%)
Prior complement use, n (%)	0 (0%)	0 (0%)	0 (0%)

gMG, generalized Myasthenia Gravis; SD, standard deviation; N (n), number; MG-ADL, Myasthenia Gravis Activities of Daily Living scale; QMG, Quantitative Myasthenia Gravis score; MGFA, Myasthenia Gravis Foundation of America clinical classification; IST, immunosuppressive therapy; MGC, Myasthenia Gravis Composite; MG-QOL-15r, Myasthenia Gravis quality of life 15 revised.

Statistically significant improvement in MG-ADL score for both claseprubart arms vs. placebo at Week 13

Mean Change in MG-ADL Score from Baseline at Week 13



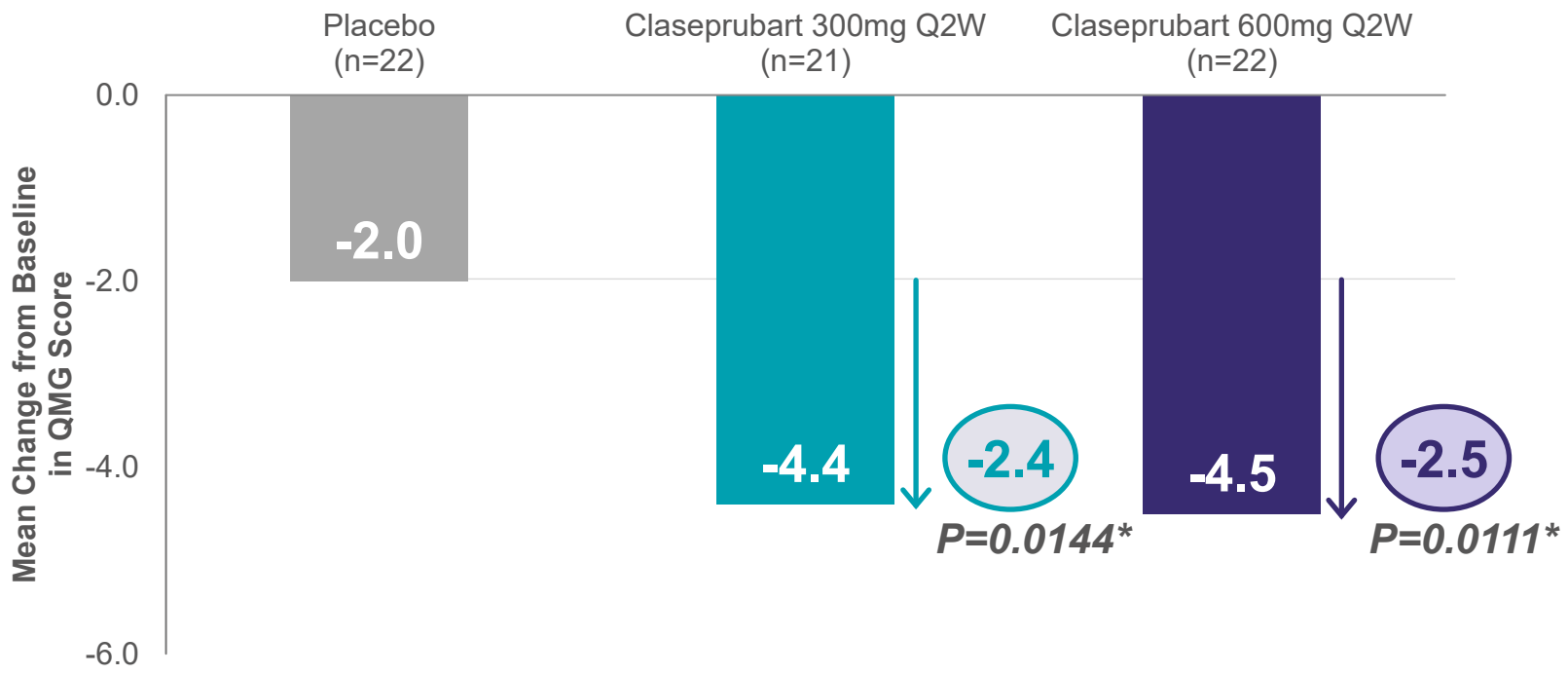
P-value	300mg/2mL	600mg/4mL
One-sided	P=0.0113*	P=0.0006*
Two-sided	P=0.0227**	P=0.0013**

Statistically significant and clinically meaningful reductions in MG-ADL across both treatment arms

The change from baseline in MG-ADL was analyzed using a mixed effect model for repeated measures (MMRM) with treatment group, visit, treatment by visit interaction, stratification factors, and baseline measure included.
*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically significant.
**Two-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.05 considered nominally statistically significant.

Statistically significant improvement in QMG score for both claseprubart arms vs. placebo at Week 13

Mean Change in QMG Score from Baseline at Week 13

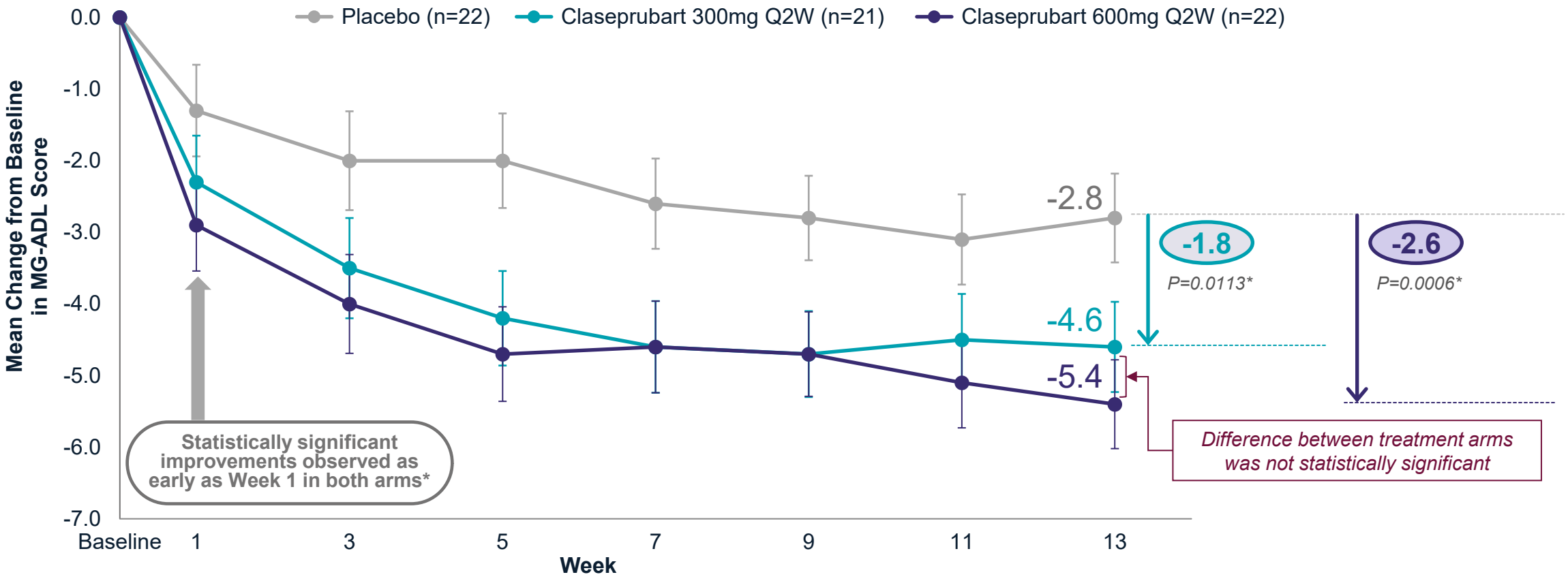


P-value	300mg/2mL	600mg/4mL
One-sided	P=0.0144*	P=0.0111*
Two-sided	P=0.0288**	P=0.0222**

Statistically significant and clinically meaningful reductions in QMG across both treatment arms

The change from baseline in QMG was analyzed using a mixed effect model for repeated measures (MMRM) with treatment group, visit, treatment by visit interaction, stratification factors, and baseline measure included.
*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically significant.
**Two-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.05 considered nominally statistically significant.

Claseprubart arms demonstrated rapid, sustained, and clinically meaningful improvements in MG-ADL score

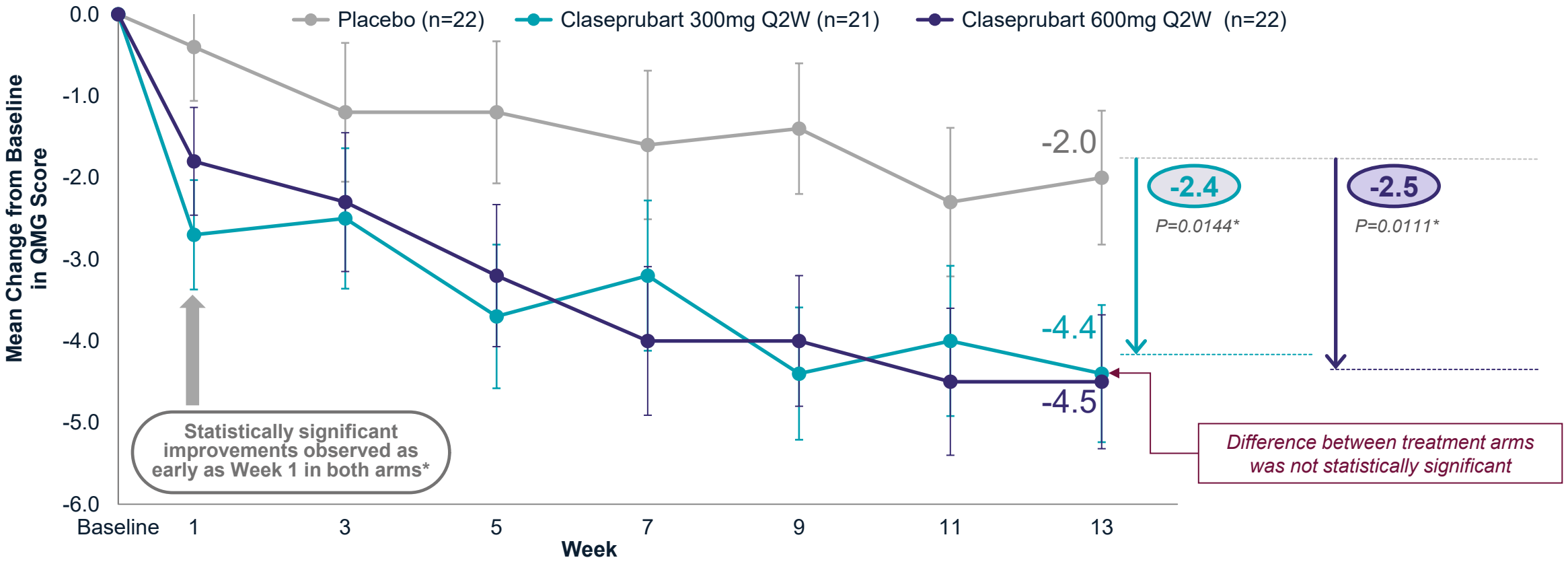


MG-ADL improvements for participants treated with claseprubart were rapid, sustained, clinically meaningful and statistically significant as early as Week 1

The change from baseline in MG-ADL was analyzed using a mixed effect model for repeated measures (MMRM) with treatment group, visit, treatment by visit interaction, stratification factors, and baseline measure included. Bars represent standard error of the mean.

*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically significant.

Claseprubart arms demonstrated rapid, sustained, and clinically meaningful improvements in QMG score



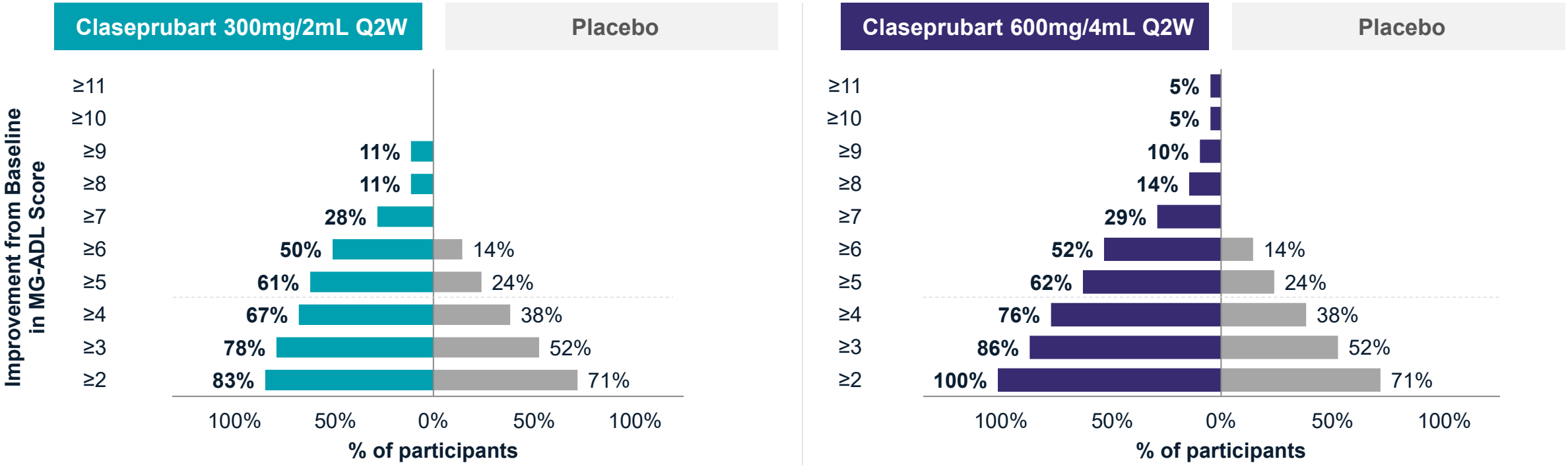
QMG improvements for participants treated with claseprubart were rapid, sustained, clinically meaningful and statistically significant as early as Week 1

The change from baseline in QMG was analyzed using a mixed effect model for repeated measures (MMRM) with treatment group, visit, treatment by visit interaction, stratification factors, and baseline measure included. Bars represent standard error of the mean.

*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically significant.

>60% of participants on claseprubart 300mg/2mL achieved ≥5 point improvement in MG-ADL

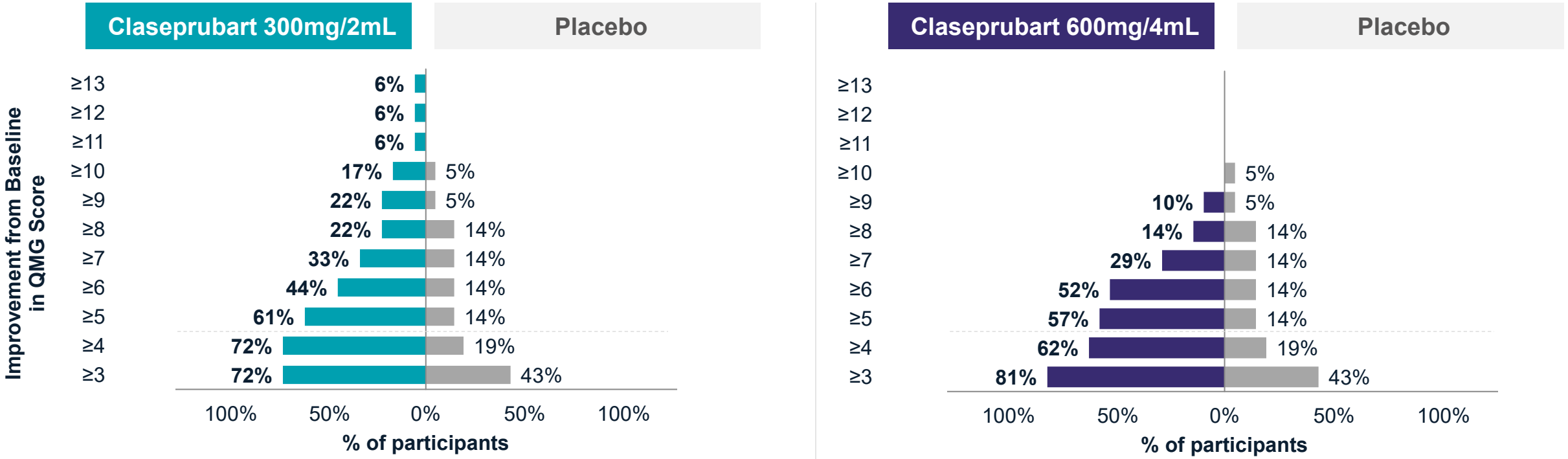
Improvement in MG-ADL Total Score



Participants across both treatment arms achieved robust improvements in MG-ADL at Week 13

>60% of participants on claseprubart 300mg/2mL achieved ≥5 point improvement in QMG

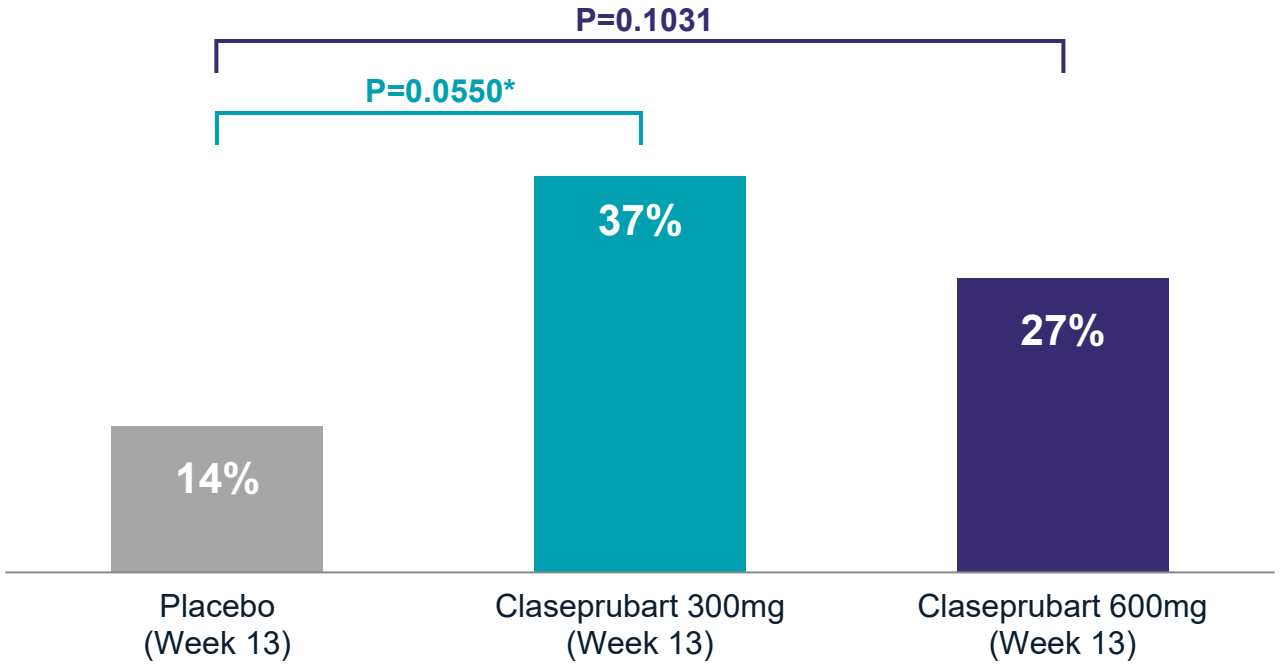
Improvement in QMG Total Score



Participants across both treatment arms achieved robust improvements in QMG at Week 13

37% of 300mg/2mL claseprubart-treated participants achieved Minimal Symptom Expression on MG-ADL at Week 13

Minimal Symptom Expression (MSE)
% of Participants Achieving MG-ADL Score of 0 or 1 at Week 13

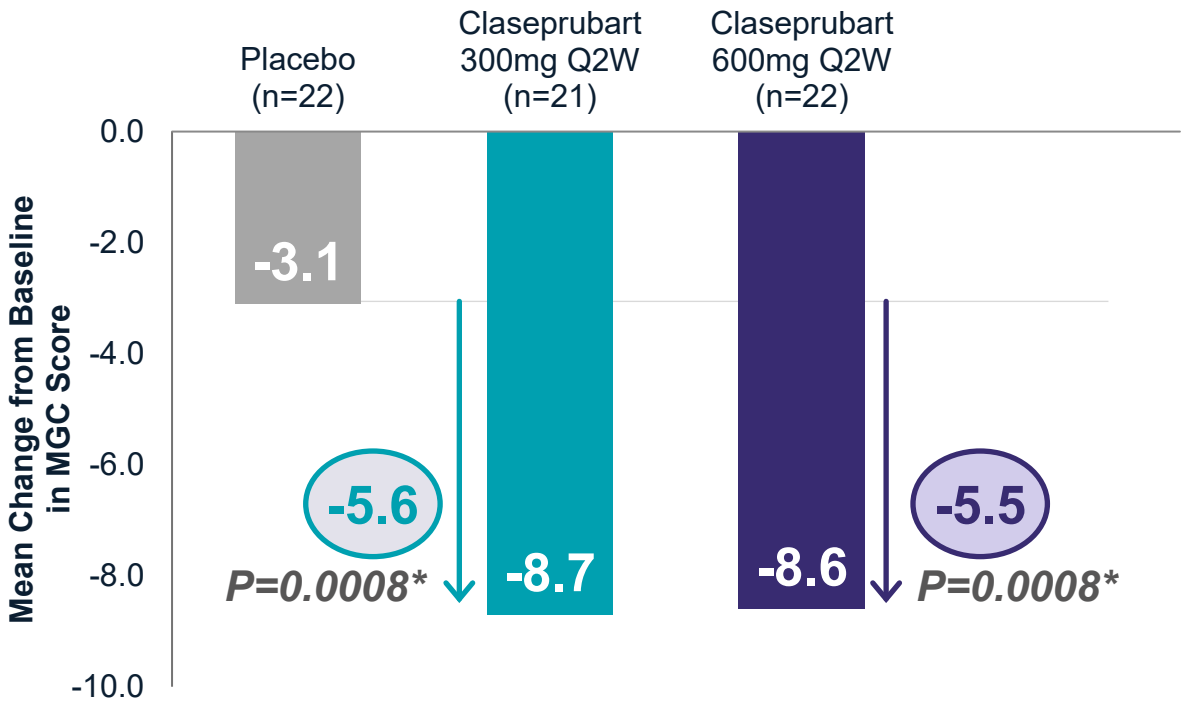


MSE supports potential best-in-class profile

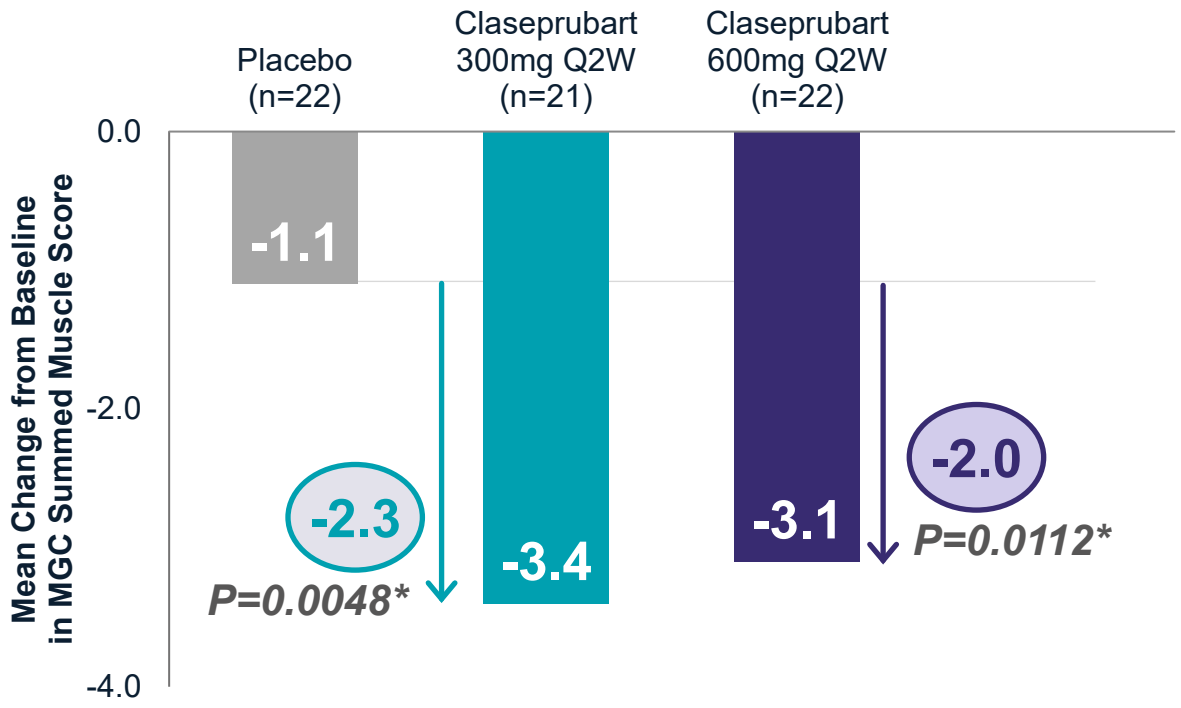
The proportion of participants who achieve MSE was analyzed using a logistic regression with terms for treatment group, stratification factors, and baseline MG-ADL included.
*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically significant.

Statistically significant improvement in MGC for both claseprubart arms vs. placebo at Week 13

Mean Change in MGC Score from Baseline at Week 13



Mean Change in MGC Summed Muscle Score from Baseline at Week 13 (post-hoc analysis)



The change from baseline in MGC and MGC Summed Muscle Score were separately analyzed using a mixed effect model for repeated measures (MMRM) with treatment group, visit, treatment by visit interaction, stratification factors, and baseline measure included.

*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically significant.

Statistically significant improvement in MG-QoL-15r score for 300mg/2mL vs. placebo at Week 13

Mean Change in MG-QoL-15r Score from Baseline at Week 13

The change from baseline in MG-QoL 15r was analyzed using a general linear model with treatment group, stratification factors, and baseline measure included.
*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically significant.

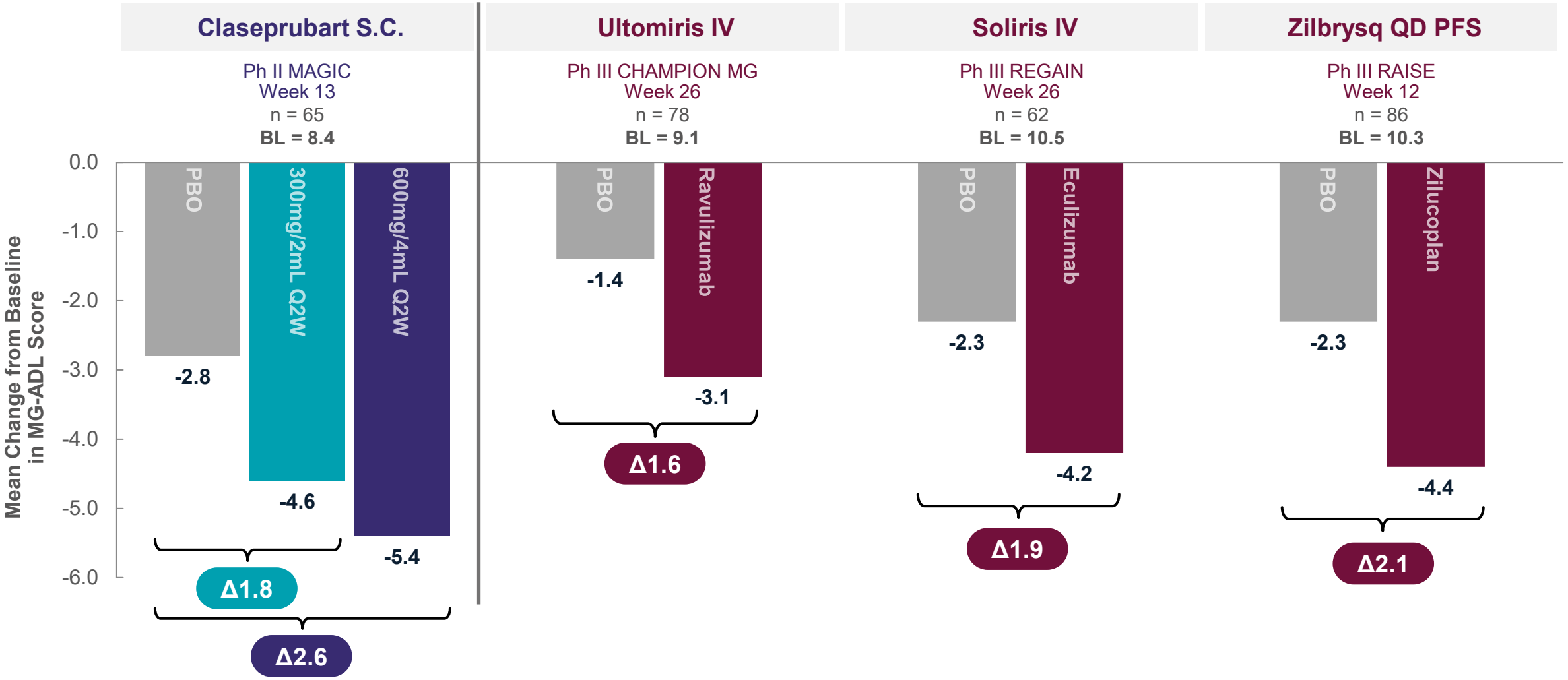
Across key efficacy measures, claseprubart demonstrated robust and clinically meaningful responses

	Placebo	Claseprubart 300mg/2mL Q2W		Claseprubart 600mg/4mL Q2W	
		Absolute	Placebo-adjusted	Absolute	Placebo-adjusted
MG-ADL mean change from baseline at Week 13	-2.8	-4.6	-1.8 *(P=0.0113)**	-5.4	-2.6 *(P=0.0006)**
QMG mean change from baseline at Week 13	-2.0	-4.4	-2.4 *(P=0.0144)**	-4.5	-2.5 *(P=0.0111)**
MSE at Week 13	14%	37%	23% *(P=0.0550)**	27%	13% *(P=0.1031)*
MGC mean change from baseline at Week 13	-3.1	-8.7	-5.6 *(P=0.0008)**	-8.6	-5.5 *(P=0.0008)**
MG-QoL-15r mean change from baseline at Week 13	-3.9	-6.1	-2.2 *(P=0.0414)**	-5.4	-1.5 *(P=0.1122)*

Claseprubart 300mg/2mL Q2W treatment arm achieved statistical significance vs. placebo across all five key efficacy measures

*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically significant.

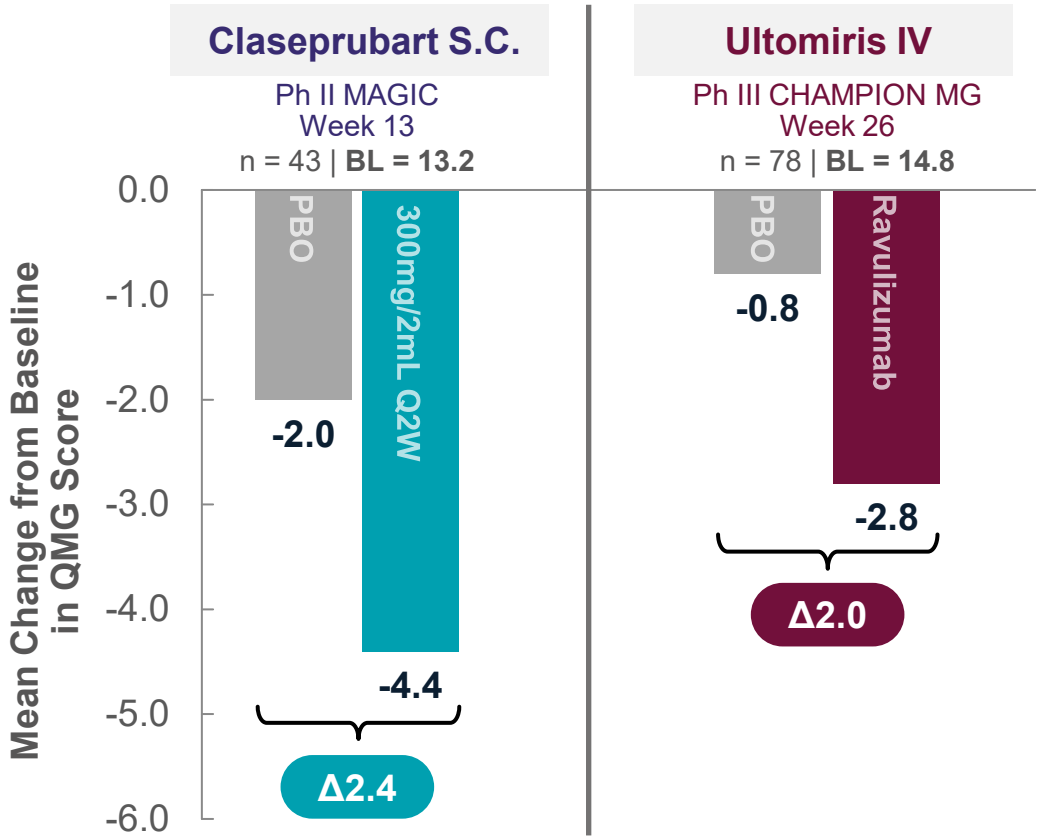
Claseprubart demonstrated statistically significant and clinically meaningful improvements in MG-ADL



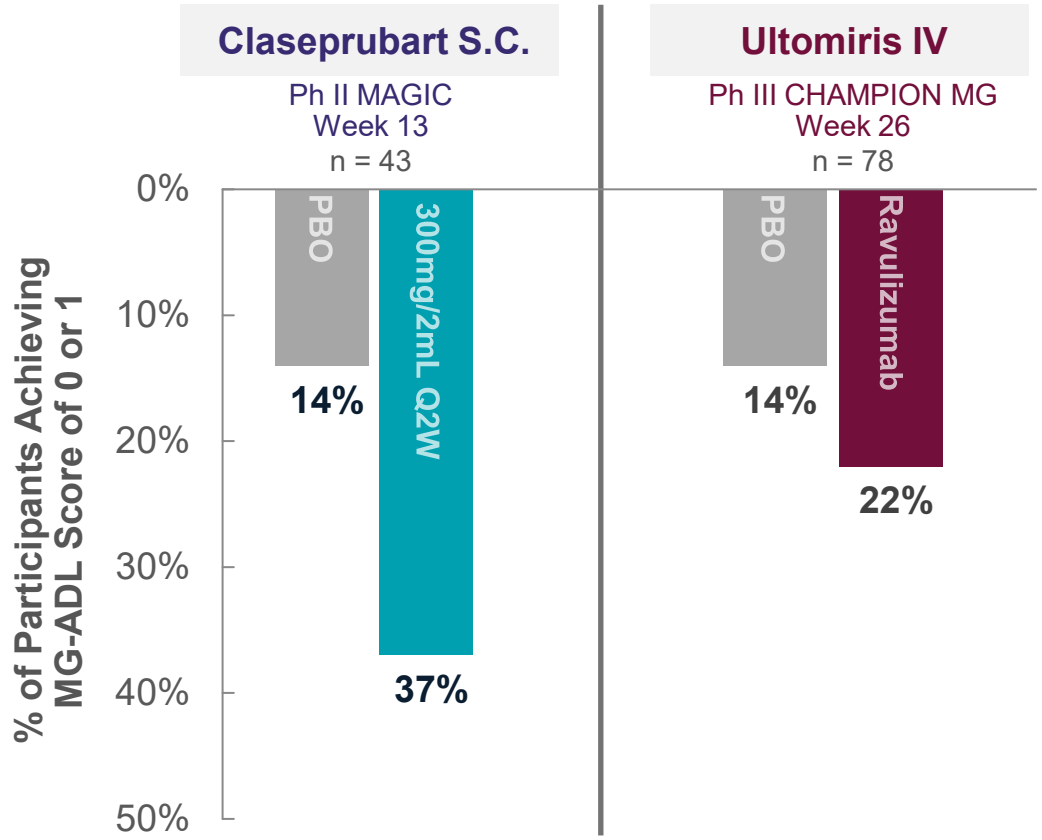
Note: For illustrative purposes only. Efficacy data are derived from different clinical trials conducted at different times, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted. Statistical treatment of missing data may vary across studies shown. Source: SOLIRIS (Ph3 REGAIN; 1200mg Q2W regimen; worst-rank ANCOVA). ULTOMIRIS (Ph3 CHAMPION-MG; weight-based Q8W regimen with maintenance doses 3000-3600mg; MMRM ANCOVA with no imputation of missing data). ZILBRYSQ (Ph3 RAISE; 0.3mg/kg QD regimen, MMRM ANCOVA with no data censorship).

Additional secondary efficacy measures support claseprubart potential as best-in-class complement inhibitor

QMG Score



Minimal Symptom Expression (MSE)



Note: For illustrative purposes only. Efficacy data are derived from different clinical trials conducted at different times, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted. Statistical treatment of missing data may vary across studies shown. Source: ULTOMIRIS (Ph3 CHAMPION-MG; weight-based Q8W regimen with maintenance doses 3000-3600mg; MMRM ANCOVA with no imputation of missing data).

Claseprubart was generally well tolerated, with a favorable, potentially differentiated safety profile in Phase 2

	Placebo (N=22)	Claseprubart 300mg/2mL Q2W (n=21)	Claseprubart 600mg/4mL Q2W (n=22)
Clinical adverse events (AEs)[1]	11 (50.0%)	13 (61.9%)	15 (68.2%)
Related serious AEs	1 (4.5%)	0 (0%)	0 (0%)
RCT discontinuation due to related AE	0 (0%)	0 (0%)	0 (0%)
Infections	10 (45.5%)	5 (23.8%)	6 (27.3%)
Related serious infections	1 (4.5%)	0 (0%)	0 (0%)
Injection site reactions[2]	0 (0%)	2 (9.5%)	2 (9.1%)
Newly positive for anti-nuclear antibodies (ANA)[3]	0 (0%)	1 (5.9%)	8 (36.4%)
Rashes	0 (0%)	0 (0%)	0 (0%)
Arthralgia	1 (4.5%)	1 (4.8%)	0 (0%)

Comparable clinical safety profile to placebo with remarkably benign administration, no infection signal and no symptoms indicative of autoimmune activation

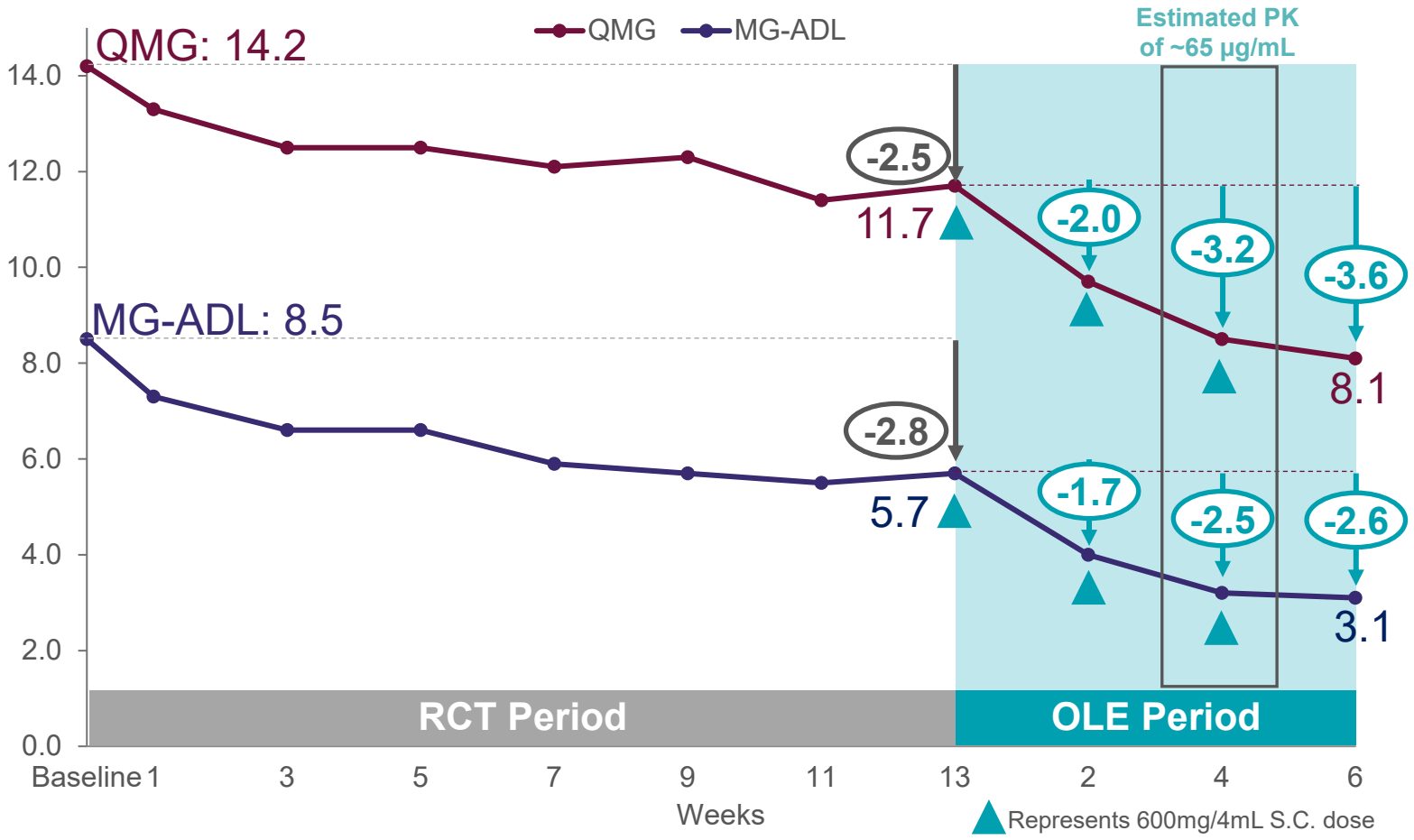
(1) Excludes events in the investigations System Organ Class (MedDRA).
(2) All injection site reactions were mild to moderate.
(3) Represents participants who were ANA negative at baseline and tested positive at ≥1:320 at any point during RCT (percentages calculated from n=17 for 300mg arm and n=22 for 600mg arm). An ANA titer of ≥1:320 was an exclusion criterion for the clinical trial protocol. At end of RCT (Week 13), 2 of the 8 patients in 600mg arm tested negative for ANA, 2 of the 8 patients in 600mg arm remained positive but at <1:320.

Rationale for Q4W 300mg/2mL Dosing & Potential for Enhanced, Best-in-Class Efficacy in gMG

OLE data support addition of 300mg/2mL Q4W in Ph. 3

PK levels approximately half of 300mg/2mL Q2W steady state resulted in robust reductions on MG-ADL & QMG

Mean Change in PBO Patients' MG-ADL and QMG Score from RCT Baseline to OLE Week 6



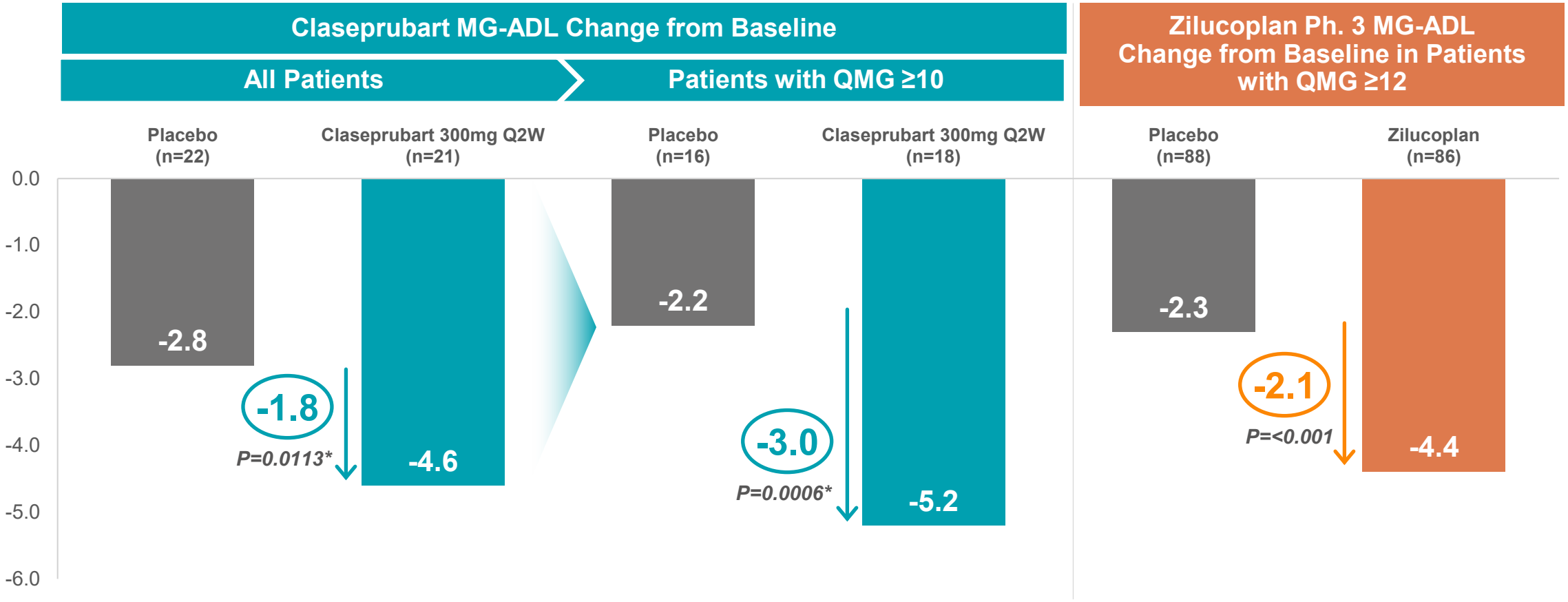
PBO Patients Entering OLE Received 600mg/4mL Q2W w/ No Loading Dose

- PK of ~65 µg/mL at week 4 after only two 600mg/4mL doses is substantially lower than steady state seen with 300mg/2mL dosing of ~100-120 µg/mL
- Robust reductions in MG-ADL and QMG are achieved by week 4, after just two 600mg/4mL doses and remain stable in subsequent weeks
- Growing external evidence further supports that lower levels of complement inhibition (<90%) may be sufficient for efficacy in gMG[1]

The change from RCT baseline in MG-ADL and QMG were separately analyzed using a mixed effect model for repeated measures (MMRM) with randomized treatment group, visit, randomized treatment by visit interaction, stratification factors, and baseline measure included. All patients received claseprubart in OLE.

1. https://newsroom.regeneron.com/node/31216/pdf

Adding QMG screening criteria in Ph. 3, similar to zilucoplan Ph. 3[1], may better control for placebo response



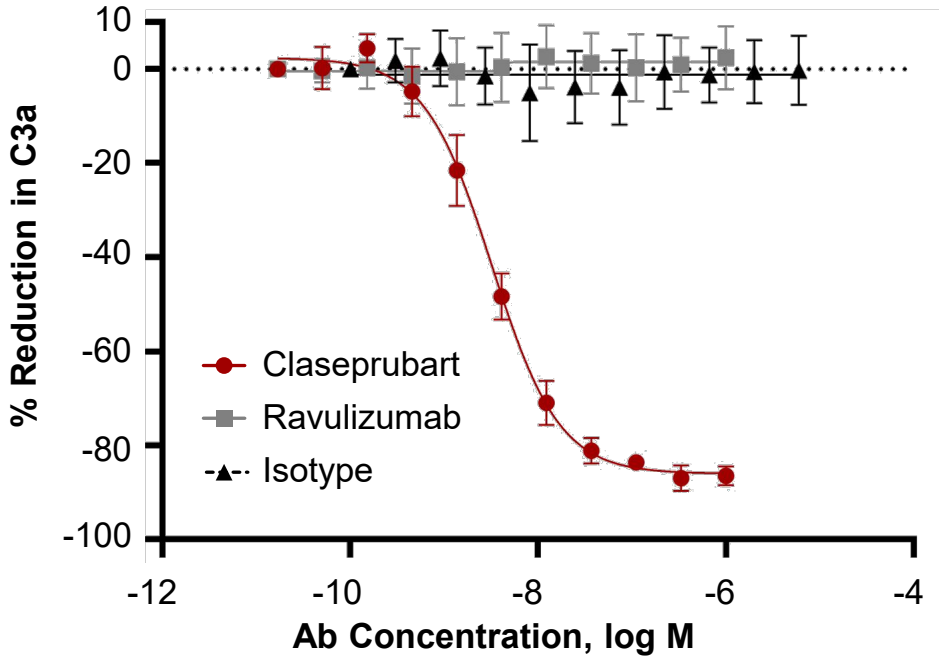
Ph. 2 study did not include QMG inclusion criteria, similar to ravulizumab Ph. 3; post-hoc analysis of MaGic data demonstrates potentially best-in-class MG-ADL improvement in patients with QMG ≥10

The change from baseline in ADL was analyzed using a mixed effect model for repeated measures (MMRM) with treatment group, visit, treatment by visit interaction, stratification factors, and baseline measure included.
*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically significant.
1. Zilucoplan Ph. 3 MG trial had screening criteria of QMG ≥ 12 and MG-ADL ≥ 6 (https://clinicaltrials.gov/study/NCT04115293)

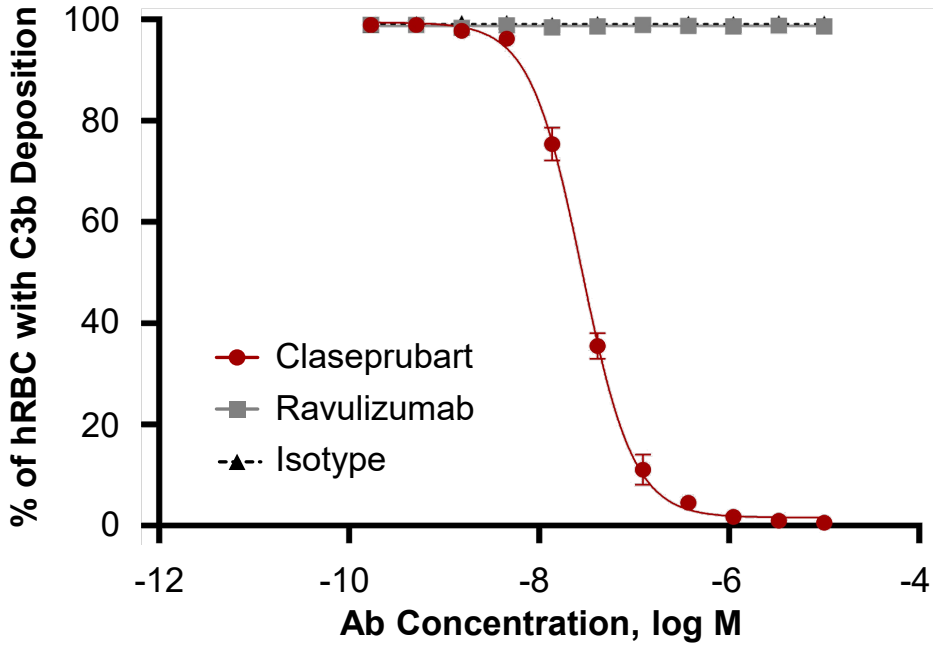
Potential for improved efficacy vs. C5 inhibitors with claseprubart may be due to upstream inhibition

Claseprubart Prevents the Creation of Pro-inflammatory Split Products C3a and C3b vs. Ravulizumab

C3a Formation[1]



C3b Deposition[2*]



Upstream inhibition prevents the creation of pro-inflammatory C3a and C3b as well as MAC, potentially providing additional efficacy benefits for AChR+ gMG patients

1. C3a Formation Assay: Human C3a ELISA specific to C3a-desArg with no cross-reactivity to C3 (N=3)
2. C3b Deposition Assay: Ab-sensitized hRBC triggered by complement-positive sera to deposit C3b on the hRBC surface, measured by flow cytometry (N=3)

*Enjaymo (sutimlimab) targets the C1s complement protein, which prevents C3b deposition on red blood cells, thereby stopping hemolysis and improving anemia in patients with cold agglutinin disease (Jager U, et al. Blood 2019;133:893–901)

Ph. 3 trial design to include additional Q4W arm, and new screening criteria of QMG ≥10 as well as MG-ADL ≥6

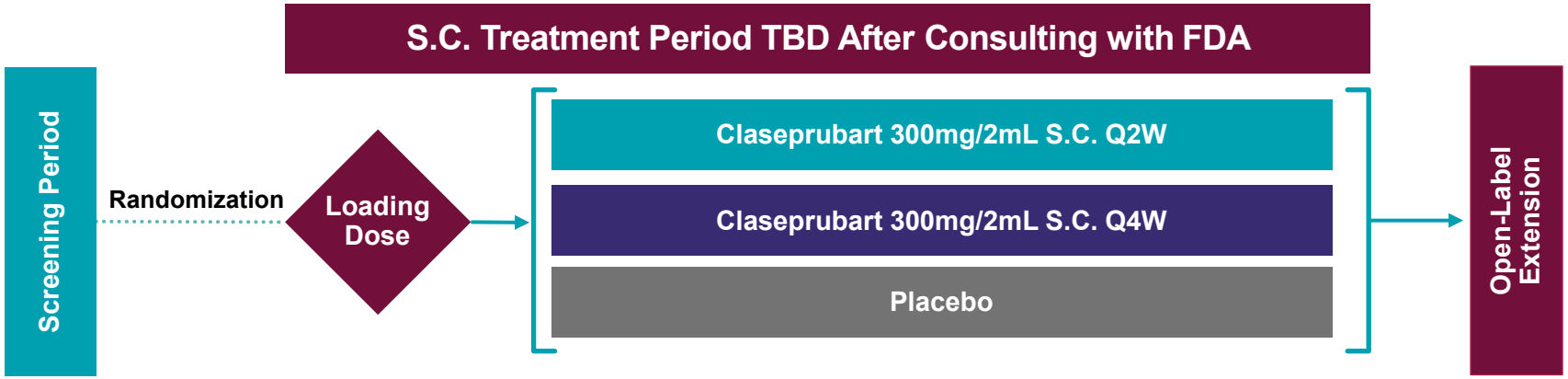
Final Ph. 3 trial design TBD after regulatory consultations

Highlights

- **Design:** Male and female subjects randomized to receive either claseprubart or placebo for TBD weeks
- **Inclusion:** ≥18 years old with AChR antibody + gMG, **MG-ADL of ≥6 and QMG of ≥10**
- **Dosing:** I.V. Loading Dose followed by 300mg/2mL S.C. Q2W or **Q4W** starting Day 7

Endpoints

- **Primary:** MG-ADL change from baseline
- **Secondary / Exploratory:** Efficacy (QMG, MSE, MGC, MG-QoL-15r)



Potential to further enhance best-in-class differentiation on efficacy and dosing convenience with QMG screening criteria and 300mg/2mL Q4W dosing

Achieving this profile could position claseprubart as a potential best-in-disease treatment for gMG

C5 OR SUPERIOR EFFICACY
(ULTOMIRIS/SOLIRIS/ZILBRYSQ)

Similar or superior MG-ADL to FDA-approved C5 inhibitors with continuous, effective symptom control

Targeting >2-point MG-ADL improvement vs. placebo

C1s SAFETY
(ENJAYMO)

Comparable *safety* to FDA-approved C1s & Classical Pathway inhibitor, leaving the lectin and alternative pathways intact

Targeting no Boxed Warning & REMS

AUTOINJECTOR CONVENIENCE
(DUPIXENT)

Comparable *convenience* to DUPIXENT with one-click, self-administered SHL-Molly autoinjector

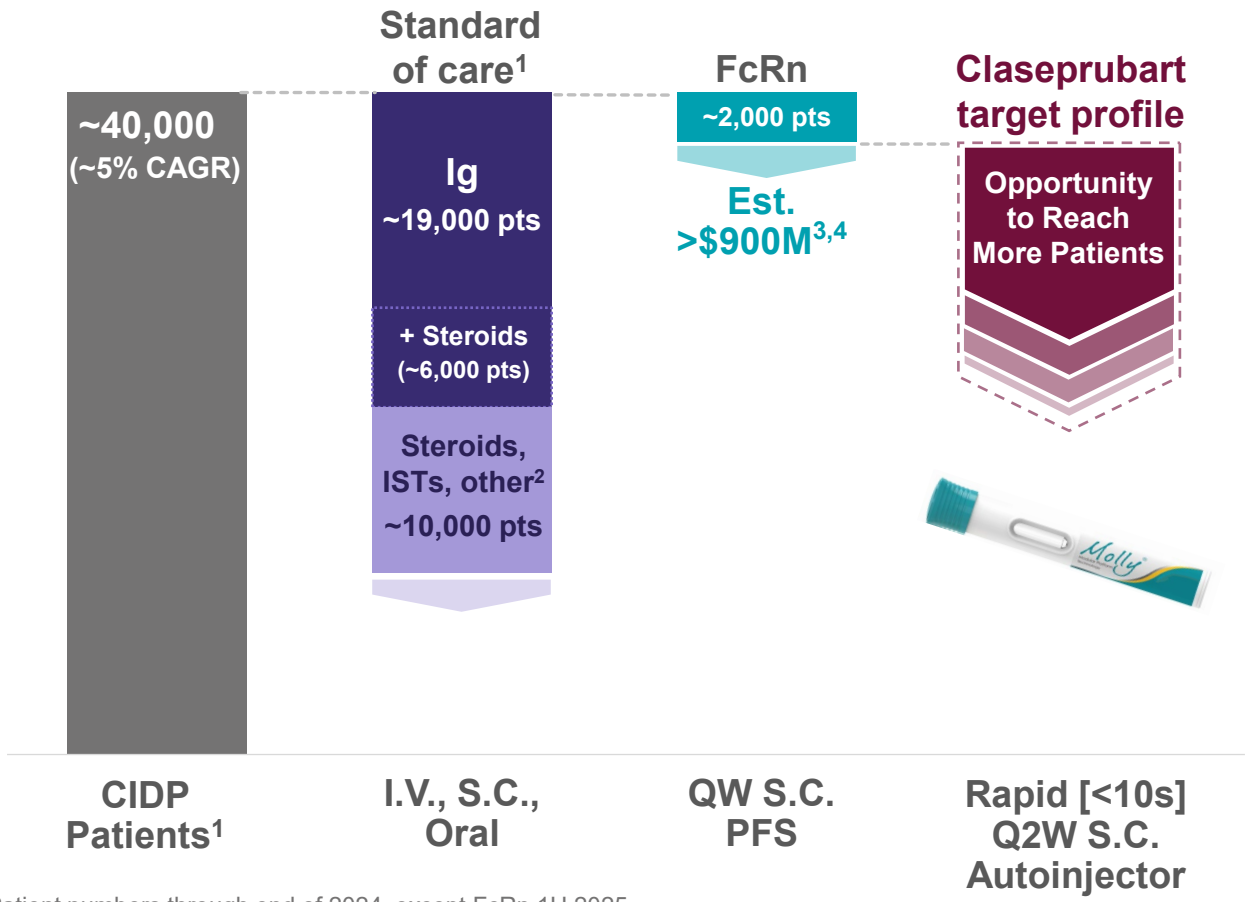
Targeting single 300mg/2mL S.C. Q2W or Q4W

Claseprubart is an investigational agent that is not approved as a therapy in any indication in any jurisdiction worldwide. Safety and efficacy for claseprubart has not been evaluated in head-to-head comparative clinical studies.
Autoinjector for claseprubart administration is anticipated to be SHL Medical's Molly technology, patented or patent pending in the US, China, India, Japan, Korea, Taiwan and at the European Patent Office

Claseprubart: Opportunity to Change the Treatment Paradigm in Chronic Inflammatory Demyelinating Polyneuropathy

The US CIDP market offers substantial growth given the high unmet need and limitations of the current standard of care

Opportunity for an active C1s inhibitor with the target profile of claseprubart to replace the standard of care



*Patient numbers through end of 2024, except FcRn 1H 2025

US CIDP Market Opportunity

- Current Ig and biologics account for >$3.5B[3,4,5]
- Despite SoC, many (30-50%) patients are refractory, face risk of relapse, and confront adverse effects of long-term treatment[6-8]
- FcRn is considered more of an alternative than improvement over IVIg[9]
- Active C1s inhibition has demonstrated ~50% improvement in both SoC treated and SoC refractory patients[10]
- Opportunity to replace SoC with a patient friendly and easy-to-use active C1s inhibitor

1. Komodo claims data 2013-2025, adjusted to account for 70% capture of real-world patient counts for biologic treated patients, adjusted to account for 27% misdiagnosed; 2. Other Tx: PLEX/Splenectomy/Thymectomy, Rituximab, Biologic; 3. Argenx Corp Pres – July 2025; 4. Fierce Pharma, CIDP Pricing; 5. CIDP - Intravenous Immunoglobulin Market Statistics. Grand View Horizon. 6. Mair D, et al. Novel therapies in CIDP. Journal of Neurology, Neurosurgery & Psychiatry 2025;96:38-46.; 7. Gogia B, et al. Chronic Inflammatory Demyelinating Polyradiculoneuropathy, StatPearls Publishing.; 8. Bus, S.R.M., et al. Clinical outcome of CIDP one year after start of treatment. J Neurol.; 9. Levine T, et al. Early deterioration of CIDP following transition from IVIG to FcRn inhibitor, Journal of the Neurological Sciences; 10. Novel therapies in CIDP, Journal of Neurology, Neurosurgery, and Psychiatry (2024).

Active C1s inhibition has demonstrated clinical POC with potential for equal or superior efficacy to current SOC IVIG

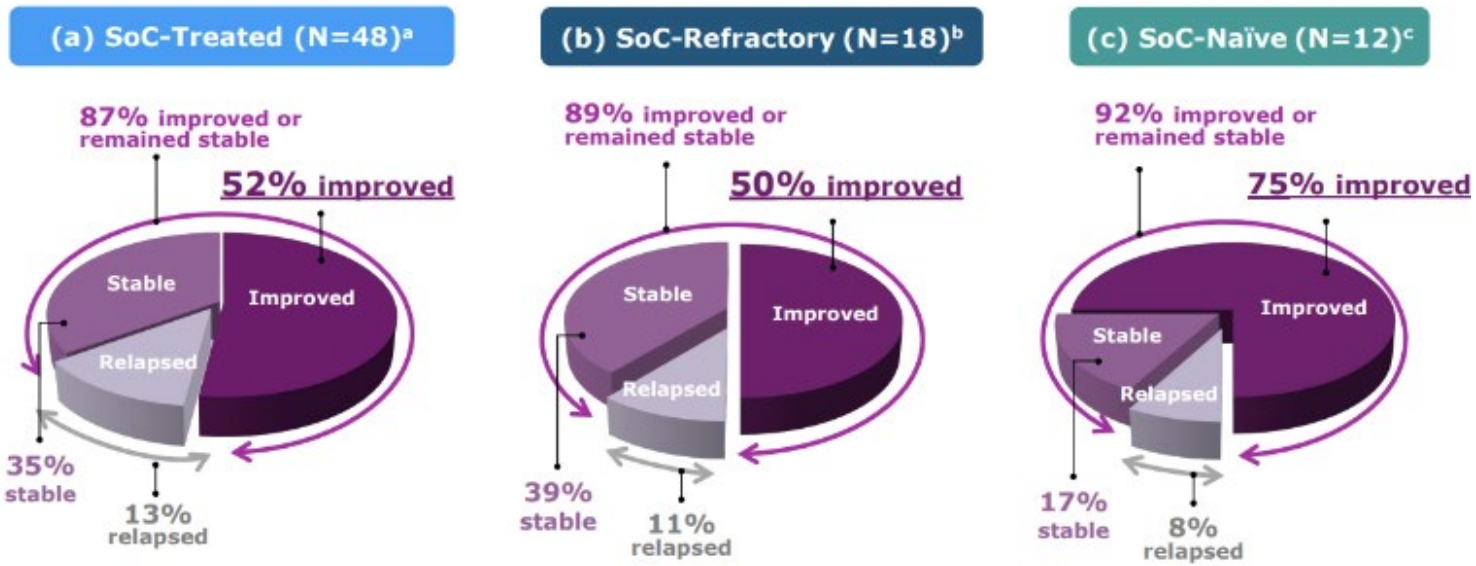
Neuromuscular indication with high unmet medical need

>40,000
patients in the U.S. and **no approved targeted complement therapies**

Evidence supports classical complement role in disease

riliprubart (active C1s inhibitor) recently reported positive interim efficacy results[1]

Ph. 2 Riliprubart Data Validates Active C1s in CIDP[1] but with High Volume, Weekly Dosing of 600mg/4mL[2]



Claseprubart target dose of 300mg/2mL S.C. every two weeks may offer more convenient, lower volume dosing for CIDP patients vs. riliprubart

1 Riliprubart Phase 2 at PNS 2024
2 Pg 76: riliprubart patent filing

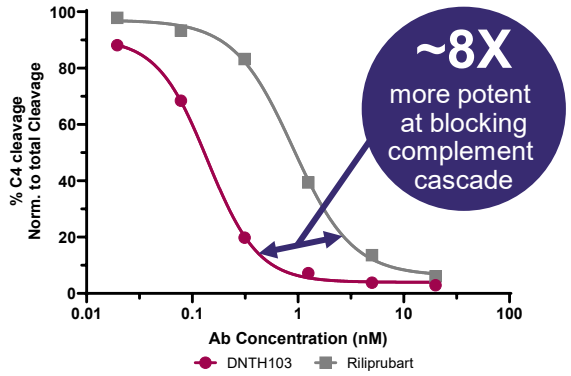
Claseprubart has superior affinity and potency vs. riliprubart

Affinity Assays

		DNTH103	riliprubart	Fold Improvement
Binding Affinity to human active C1s (K_D)[1]	KinExa	**9pM**	75pM	**~8X**
	SPR	**8pM**	35pM	**~4X**

Enzymatic Assay

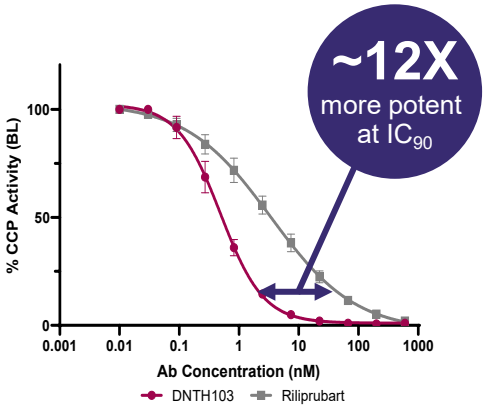
C4 cleavage by human active C1s[2]



	DNTH103	riliprubart
IC_{90} (nM)	**0.47**	3.69

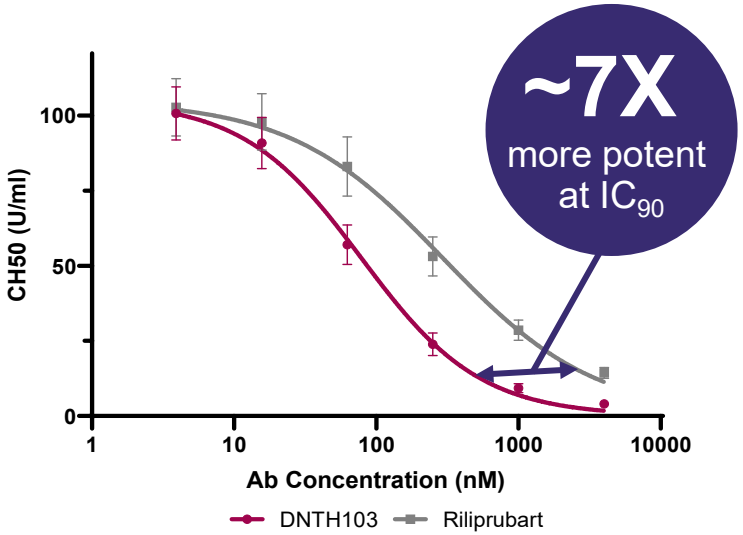
Functional Assays of Classical Pathway Inhibition

Wieslab classical pathway Assay in human serum[3]



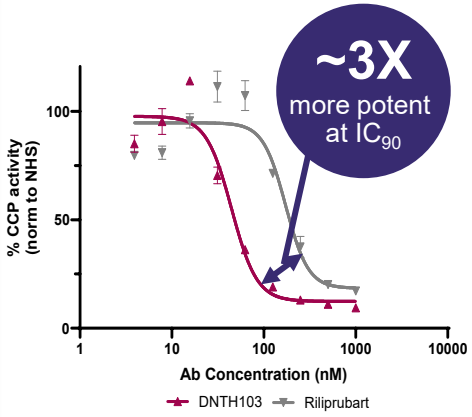
	DNTH103	riliprubart
IC_{90} (µg/mL)	**0.45**	5.4

CH50 assay of RBC lysis in human serum[3]



	DNTH103	riliprubart
IC_{90} (µg/mL)	**98**	668

Liposome lysis in human serum[3]



	DNTH103	riliprubart
IC_{90} (ug/mL)	**14.7**	39.8

Claseprubart consistently outperforms riliprubart in affinity and potency when compared head-to-head in multiple *in vitro* experiments

Note: Riliprubart is produced using sequence from patent WO2018071676A1

1. Data shown is dissociation constant (K_D) and the average of 3 different experiments performed at independent laboratories
2. Data is quantitative analysis of active C1s protease inhibition of cleaved C4 fragments in the presence of claseprubart or riliprubart
3. Data shown are the average of 3 experiments conducted for each of the functional assays (CH50 hemolysis, Wieslab and Liposome). CH50 and Wieslab were confirmed at independent laboratories

CIDP Ph. 3 pivotal trial includes open-label Part A testing the target dose of 300mg/2mL S.C. Q2W

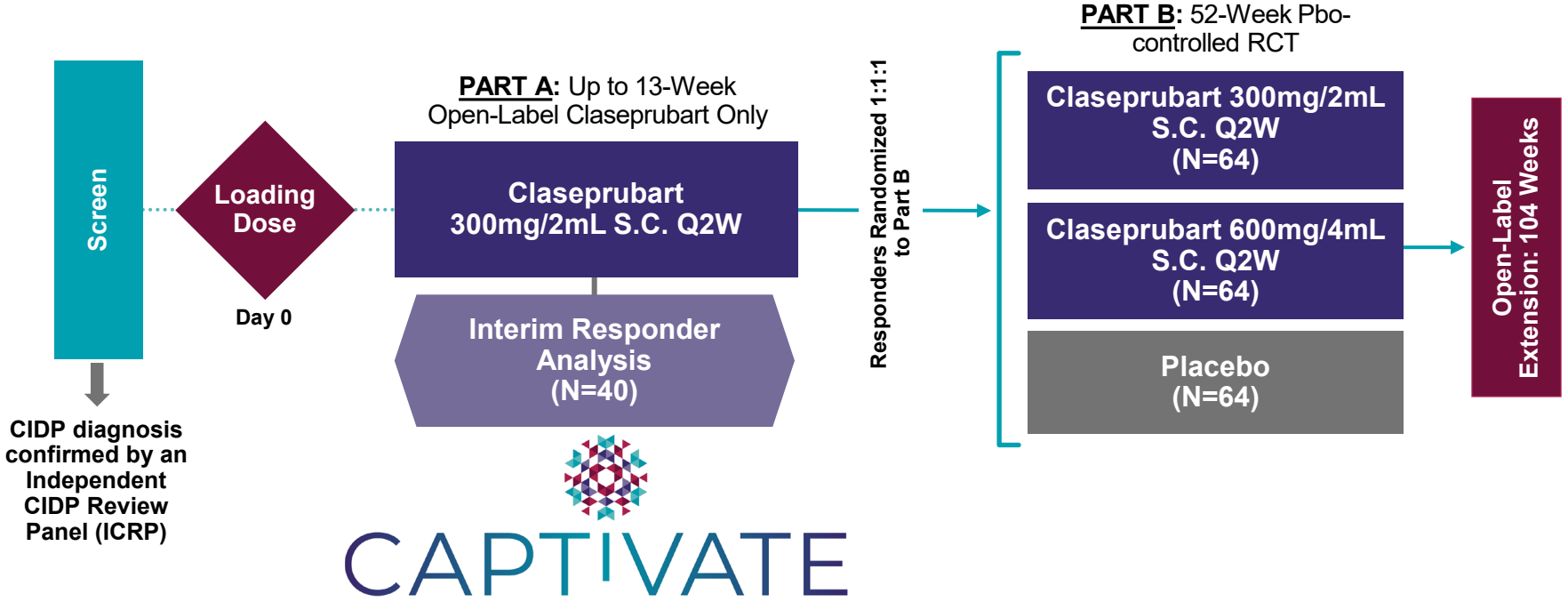
CIDP interim responder analysis with first 40 patients in Part A anticipated in Q2'26

Highlights

- **Design:** All subjects receive claseprubart in Part A for up to 13 weeks. Only responders randomized to Part B for 52 weeks
- **Inclusion:** ≥18 years old with confirmed CIDP, including SOC-refractory, SOC-stable or SOC-naïve
- **Dosing:** I.V. loading dose followed by 300mg/2mL S.C. Q2W in Part A; followed by 300mg/2mL or 600mg/4mL or placebo in Part B

Endpoints

- **Part A:** Response as measured as ≥1 point decrease (improvement) in adjusted INCAT score compared to Part A baseline
- **Part B Primary:** Efficacy (time to relapse) as measured as ≥1 point increase in adjusted INCAT



- **Single pivotal** two-part, randomized withdrawal, double-blind, placebo-controlled trial designed to **support BLA in adult patients with CIDP**
- Enrolling a **broad patient population including SOC-refractory patients**, in addition to SOC-stable and SOC-naïve patients
- **All confirmed CIDP patients receive convenient 300mg/2mL S.C. Q2W** dosing of claseprubart in Part A
- **Only responders from Part A** randomized into the double-blind, placebo-controlled Part B

CAPTIVATE Trial: https://clinicaltrials.gov/study/NCT06858579

Key differences between ADHERE and CAPTIVATE trials make cross-trial comparisons challenging

Considerations	Efgartigimod (FcRn) S.C. QW	Claseprubart (aC1s) 300mg/2mL S.C. Q2W	Key Differentiators of CAPTIVATE
Ph. 3 Study Populations	adhere **SoC-Treated** **Off Treatment**	CAPTIVATE **SoC-Treated** **SoC-Refractory** **SoC-Naïve**	Enrolling a **broad population of CIDP patients**, including SOC-Refractory
IVIG Withdrawal Required Prior to Part A of Study[1]	*No SOC-Refractory patients* **YES** *Patients must relapse before enrolling into Part A*	**NO**	**No requirement for IVIG withdrawal and disease worsening**, consistent with ongoing FcRn and complement CIDP studies[3]
Study Endpoints / Results	• **Confirmed ECI[2]** • **Ph. 3 Stage A results:** –66.5% ECI (wk 12) *~1/3 of pts* ***did not return*** *to pre-IVIG washout baseline*	• **≥1-point aINCAT improvement** • **Part A expectations:** –Targeting similar response in open-label Part A to riliprubart open-label Ph. 2 in SOC-Treated and SOC-Refractory arms	Potential to show **clinically meaningful improvement** (similar to riliprubart) from baseline **without first requiring IVIG withdrawal and disease worsening**

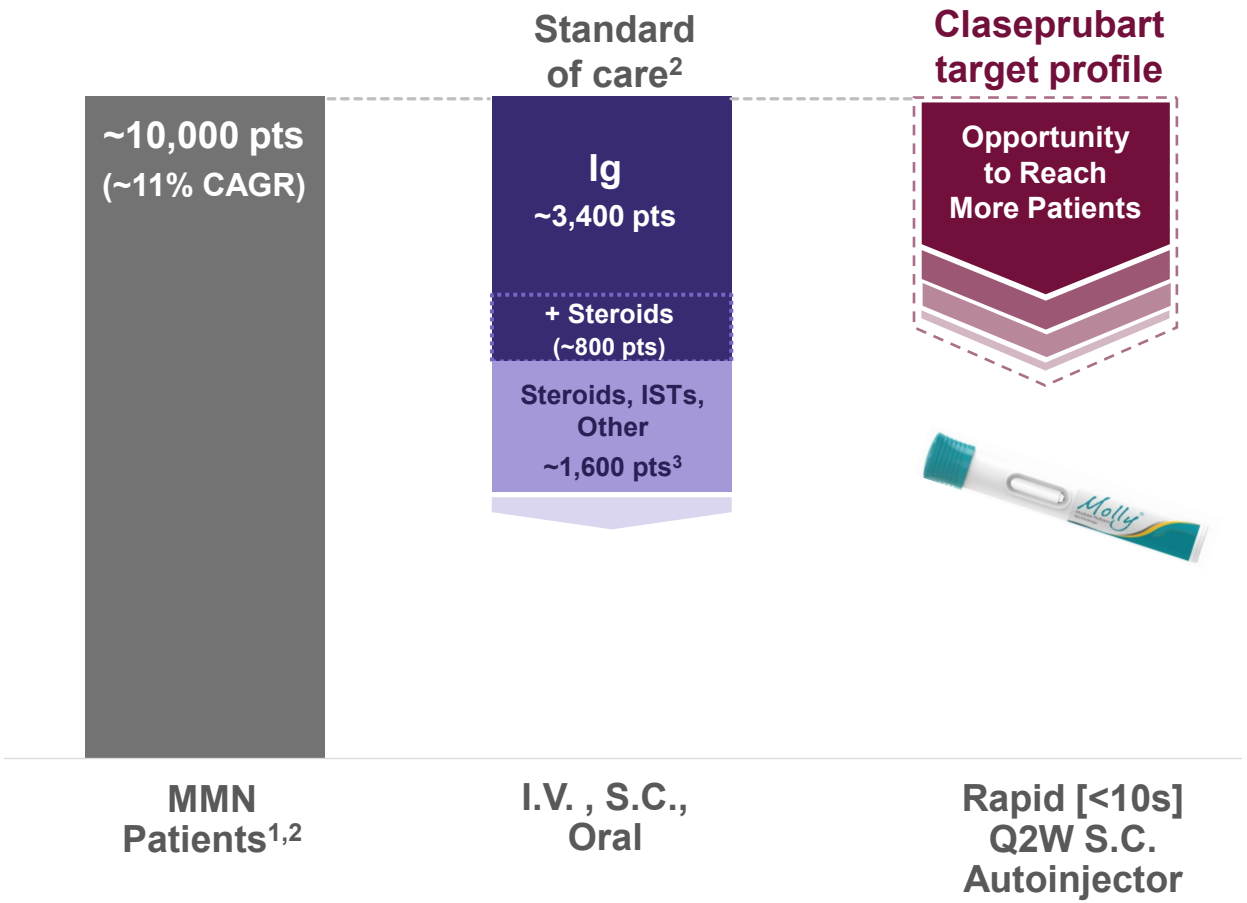
Source: Company filings, presentations and clinicaltrials.gov

1. ADHERE required removal of IVIG and evidence of clinically meaningful deterioration before dosing in Part A
2. Defined as a clinical improvement on the parameters that the participant worsened in during run-in (≥4-point increase in I-RODS and/or ≥8-kPa increase in mean grip strength) or clinical improvement (≥1-point decrease) in INCAT
3. Represents IMVT-1402, empasiprubart and riliprubart studies

Claseprubart:
Opportunity to be Best-in-Class in Multifocal Motor Neuropathy

The US MMN market is underdiagnosed with a need for more effective and convenient treatment options

Opportunity for the target profile of claseprubart to become the new standard of care in MMN



*Patient numbers through end of 2024

US MMN Market Opportunity

- Market is growing ~11% per year with ~2K newly diagnosed patients each year[2]
- Despite standard of care, patients face progressive and disabling weakness[4]
- Patients also supplement Ig treatment with steroids despite guidelines against use[5]
- Opportunity for an effective and easy-to-use active C1s inhibitor to become the new SoC

1. 2024 patients projected from 2023 count due to unreliable 2024 data from the Change Healthcare cyber-attack; 2. Komodo claims data 2013-2025, adjusted to account for 70% capture of real-world patient counts; 3. Other Tx: CS, NSISTs, PLEX/Splenectomy/Thymectomy, RTX, Biologic; 4. MMN. National Organization for Rare Diseases (2025); 5. Schaik et al., Intravenous immunoglobulin for MMN. Cochrane Library (2005)

MMN is an attractive opportunity with clinical PoC demonstrated via classical pathway inhibition

Neuromuscular indication with high unmet medical need

>10,000
patients in the U.S.

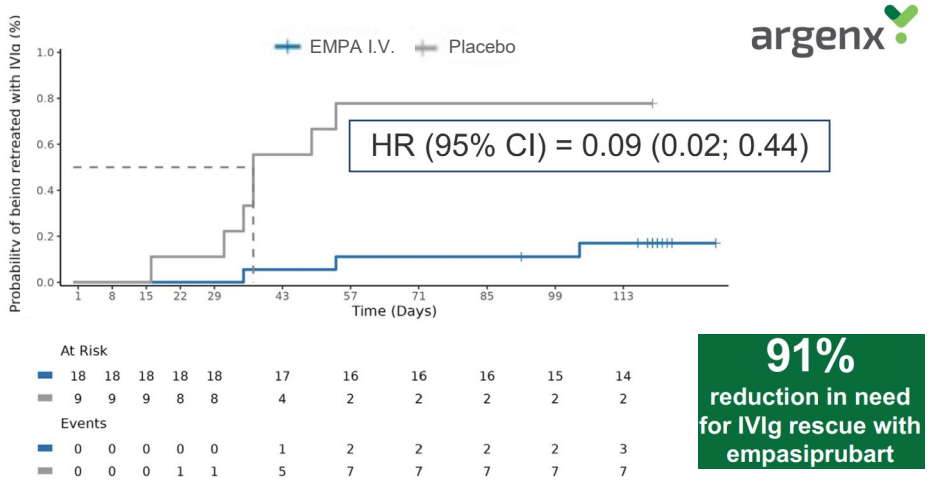
No approved targeted biologic therapies

Evidence supports classical complement role in disease

Empasiprubart (I.V., C2 inhibitor) reported efficacy signals[1]

MMN patient sera has been confirmed to activate complement

Empasiprubart (Q1-2W I.V., C2 inhibitor) Ph. 2 Data Demonstrating Efficacy Signals[1]



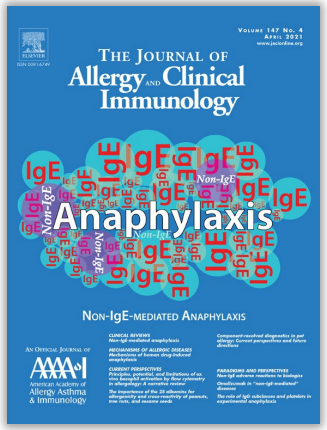
"We hypothesize that targeting the **classical complement pathway is a potential therapeutic approach in MMN**. We investigated the interaction of circulating anti-GM1 IgM from patients with MMN with complement in detail using iPSC-derived MNs. In this disease model for MMN, we evaluated the effects of ARGX-117, a novel monoclonal antibody that inhibits complement factor C2." - *Neurol Neuroimmunol Neuroinflamm. 2022 Jan; 9(1): e1107*

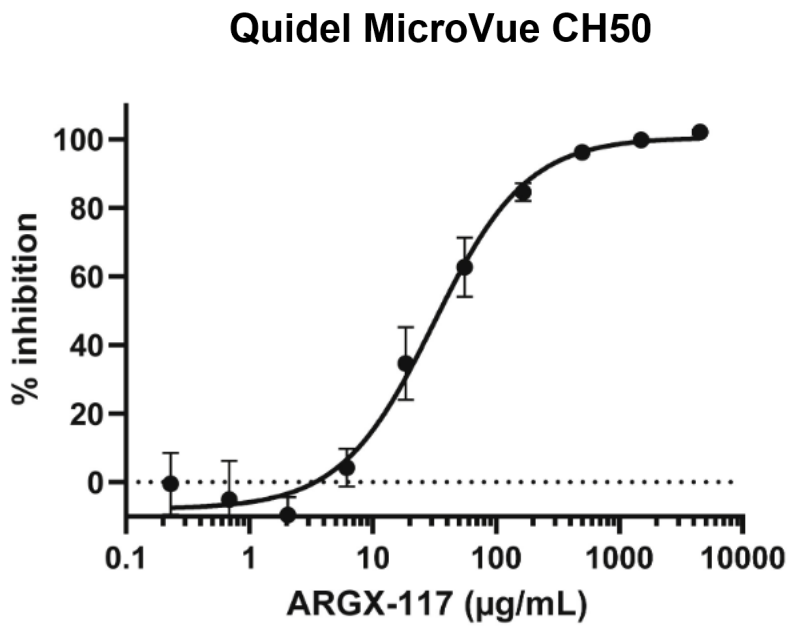
Phase 2 trial of claseprubart, a low-volume Q2W S.C., ongoing in MMN

1 https://argenx.com/content/dam/argenx-corp/events-presentations/argenx_RnD_Day_2024_Slides.pdf#/page=127

Claseprubart demonstrates superior classical pathway potency vs. empasiprubart

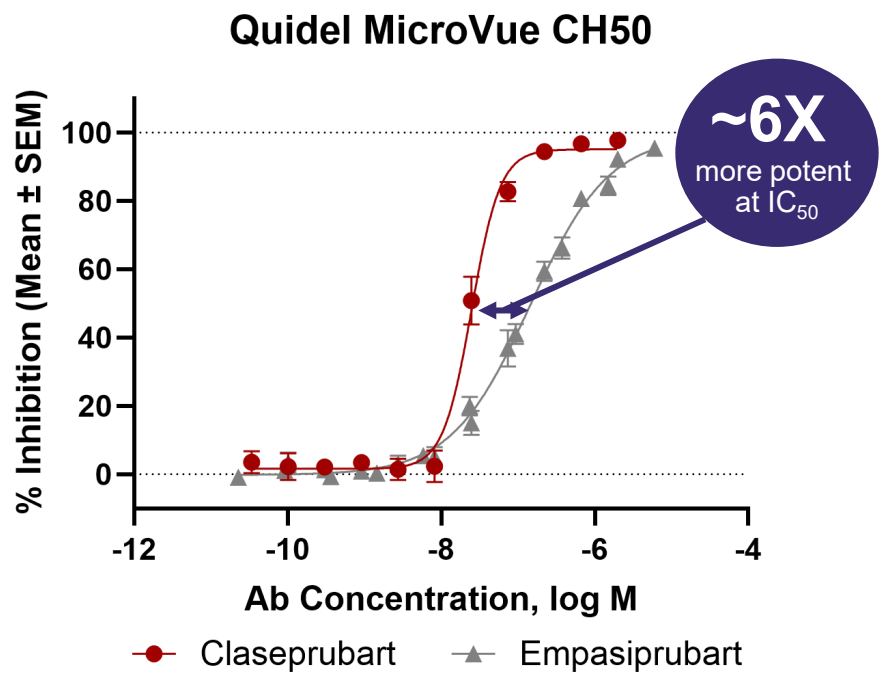
Empasiprubart Published Classical Pathway Potency Data Using the Quidel MicroVue CH50[1]





"**ARGX-117 potently inhibited CP** and LP (half-maximal effective concentration [EC_{50}] = **30.5 ± 4.5** and 93.4 ± 10.4 µg/mL, respectively) in a concentration-dependent manner" – *Journal of Allergy and Clinical Immunology*

Claseprubart Demonstrates Superior Classical Pathway Potency Head-to-head vs. Empasiprubart Using Same Assay



	claseprubart	empasiprubart
IC_{50} (µg/mL)	**3.8 ± 0.8**	22.1 ± 5.7

Claseprubart and empasiprubart are investigational agents that are not approved as therapies for MMN or any indication in any jurisdiction worldwide. Head-to-head data shown are the average of 3 experiments conducted for claseprubart and 8 experiments conducted for empasiprubart. Empasiprubart in the head-to-head experiment is produced using the sequence published in the IMGT database (DB card 12277). EC_{50} and IC_{50} can be considered as interchangeable for this analysis

1. Journal of Allergy and Clinical Immunology, Volume 147, Issue 4, 1420 - 1429.e7

Claseprubart has the potential to dominate the MMN market with its best-in-class target product profile

Considerations	Empasiprubart (C2)*	Claseprubart (active C1s)*	Key Differentiators of Claseprubart
MMN is an IgM and classical pathway driven disease[1]	Published classical pathway[3] EC_{50} = 30.5 ±4.5 µg/mL using Quidel MicroVue CH50	**Claseprubart has demonstrated potent inhibition of classical pathway in multiple assays**	**~6x more potent than empasiprubart** on IC_{50} in head-to-head in-vitro experiment using Quidel MicroVue CH50
Lectin pathway inhibition not required for efficacy in MMN	Published lectin pathway[3] inhibition of EC_{50} = 93.4 ±10.4 µg/mL	**Does not inhibit lectin pathway**	Claseprubart **preserves key bacterial killing role** of lectin pathway[2]
Patients prefer convenient therapies	I.V. Q4W	**Targeting Q2W self-administration via 300mg/2mL S.C. autoinjector**	**More convenient** by targeting infrequent, low volume, self-administered S.C. autoinjector

Claseprubart has the potential to be the first-line targeted biologic treatment given its unique combination of classical pathway potency, preservation of the lectin pathway, and dosing convenience

* Claseprubart and empasiprubart are investigational agents that are not approved as therapies for MMN or any indication in any jurisdiction worldwide. EC_{50} and IC_{50} can be considered as interchangeable for this analysis

1. Budding et al., (2021). Neurol Neuroimmunol Neuroinflamm.9(1):e1107; Vlam et al., (2015). Neurol Neuroimmunol Neuroinflamm. 2015;2(4):e119; 2. Ali et al., (2012). PLoS Pathog 8(7):e1002793 3. Journal of Allergy and Clinical Immunology, Volume 147, Issue 4, 1420 - 1429.e7.

MMN Phase 2 top-line data anticipated 2H’26

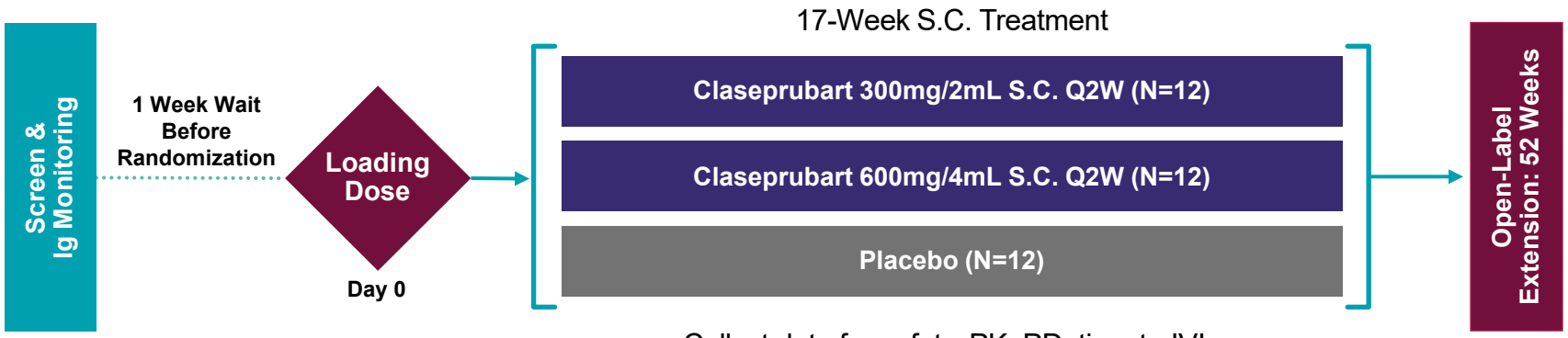
A global, multicenter, randomized, double-blind, placebo-controlled study to evaluate the safety, efficacy, and PK / PD of claseprubart administered S.C. following initial loading dose

Highlights

- **Design:** 36 participants randomized to receive either claseprubart or placebo for 17 weeks
- **Inclusion:** ≥18 years old with MMN who are immunoglobulin responsive and dependent
- **Dosing:** I.V. Loading Dose followed by 300mg/2mL or 600mg/4mL S.C. Q2W starting Day 7

Endpoints

- **Primary:** Safety
- **Secondary:** Efficacy (time to IVIg retreatment, time to relapse, grip strength and other muscle strength and motor function measurements)



MOMENTUM

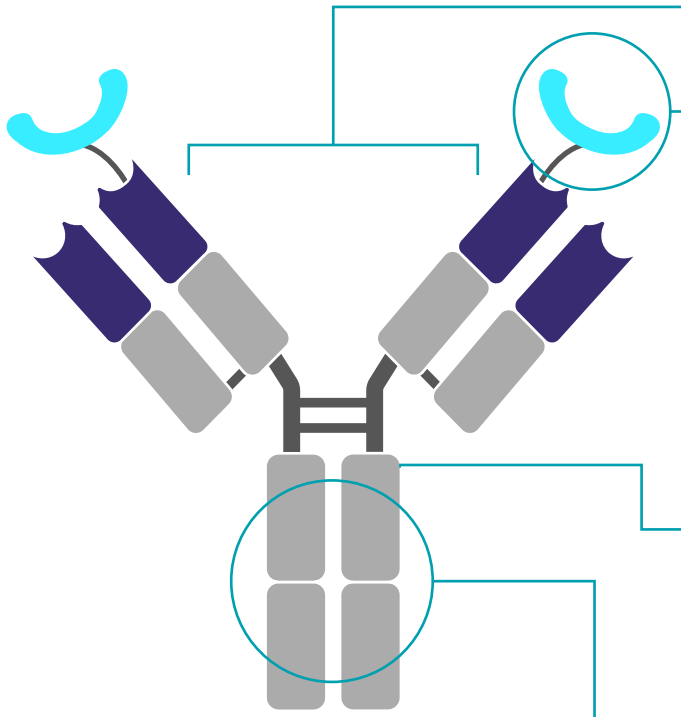
Top-line data expected in 2H’26

MoMeNtum Trial: https://clinicaltrials.gov/study/NCT06537999

DNTH212: Potential Best-in-Class Bispecific Fusion Protein for Multiple Autoimmune Indications

DNTH212 is a bifunctional BDCA2 and BAFF/APRIL inhibitor targeting two validated pathways

- Inhibiting BDCA2 **reduces Type 1 interferon production from plasmacytoid dendritic cells (pDCs)**
- Single CRD2 domain of TACI designed to deliver **robust B cell modulation via BAFF/APRIL inhibition**



DNTH212
Humanized monoclonal antibody targeting **BDCA2** fused with **TACI-CRD2**

Anti-BDCA2 Antibody
Potent inhibition of Type 1 interferon

TACI-CRD2 Domain
Potent reduction of Ig levels via BAFF/APRIL inhibition

Afucosylated Bifunctional Ab
Enhanced ADCC on target cells leads to pDC depletion

YTE Mutation on Fc
Extended half-life

DNTH212 targets both the innate and adaptive immune systems with complementary disease modifying mechanisms enabling potential best-in-class efficacy

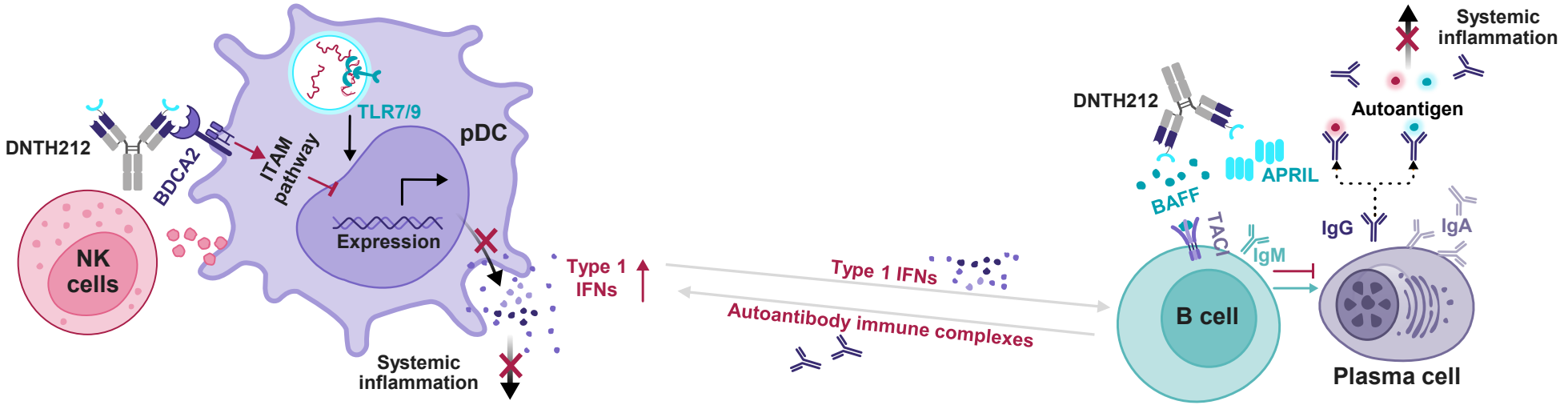
Potential to drive superior clinical efficacy by targeting both the innate and adaptive immune systems

Innate Immune System: Plasmacytoid Dendritic Cells (pDCs)

- Key cell type producing Type 1 interferon
- Promote B cell proliferation and Ig secretion through antigen presentation and production of BAFF
- Direct and indirect activation of other innate and adaptive immune cells
- Type 1 interferon inhibition has been shown effective in multiple autoimmune diseases

Adaptive Immune System: B Cells

- Generate autoantibodies, forming immune complexes that trigger inflammation and tissue damage
- Inhibiting BAFF/APRIL has been shown effective in multiple autoimmune diseases

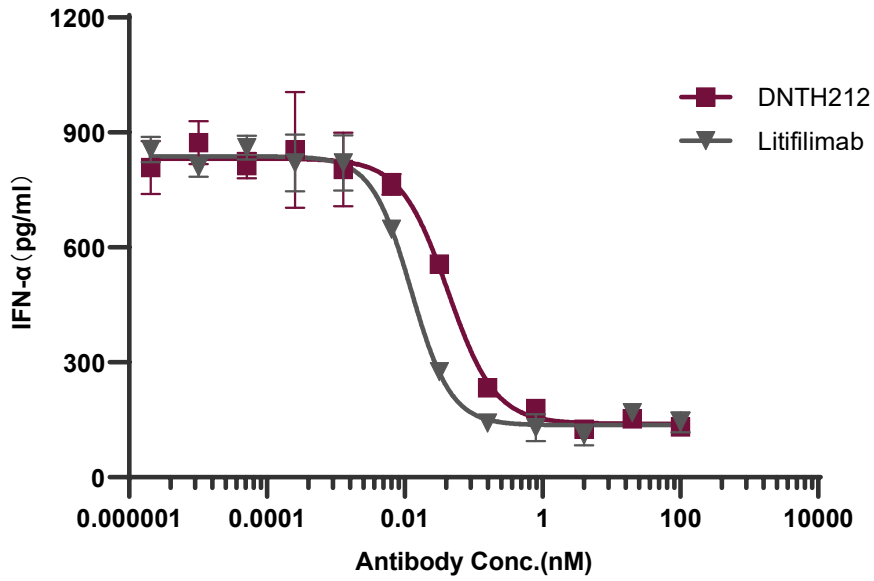


Bifunctional approach addressing autoimmune diseases where both Type 1 interferon and B Cells are implicated has strong mechanistic rationale for potential best-in-class efficacy

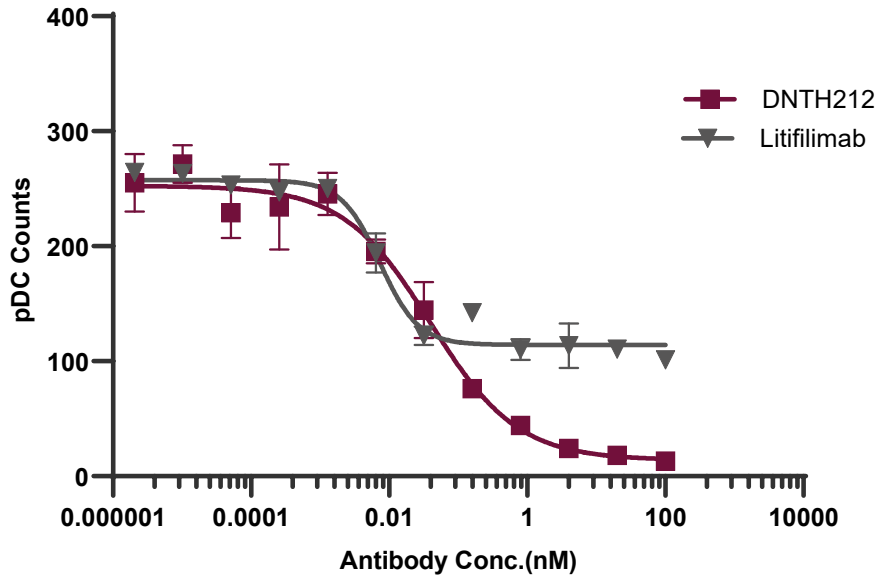
Panda SK, et al., Plasmacytoid dendritic cells in autoimmunity. Curr Opin Immunol. 2017 Feb;44:20-25.
Minaga K, et al., Plasmacytoid Dendritic Cells as a New Therapeutic Target for Autoimmune. Front Immunol. 2021 Jul 23;12:713779

DNTH212 achieves superior pDC depletion compared to litifilimab *in vitro*

Comparable Suppression of Pro-Inflammatory Cytokine IFN-α[1]



Deeper Depletion of pDC[2]

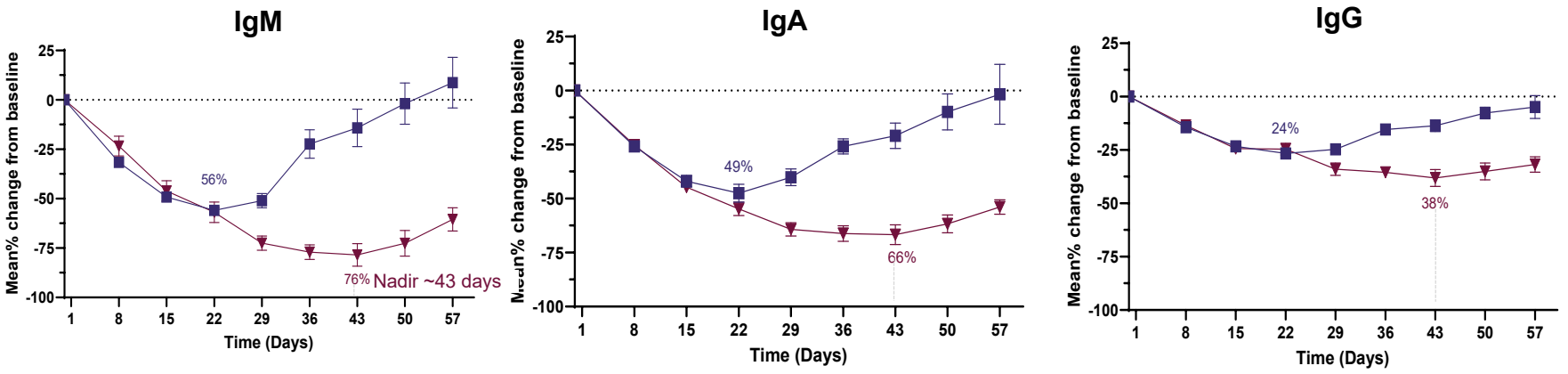


pDC depletion removes a key cell type involved in Type 1 interferon production and activation of other immune cells which contribute to disease

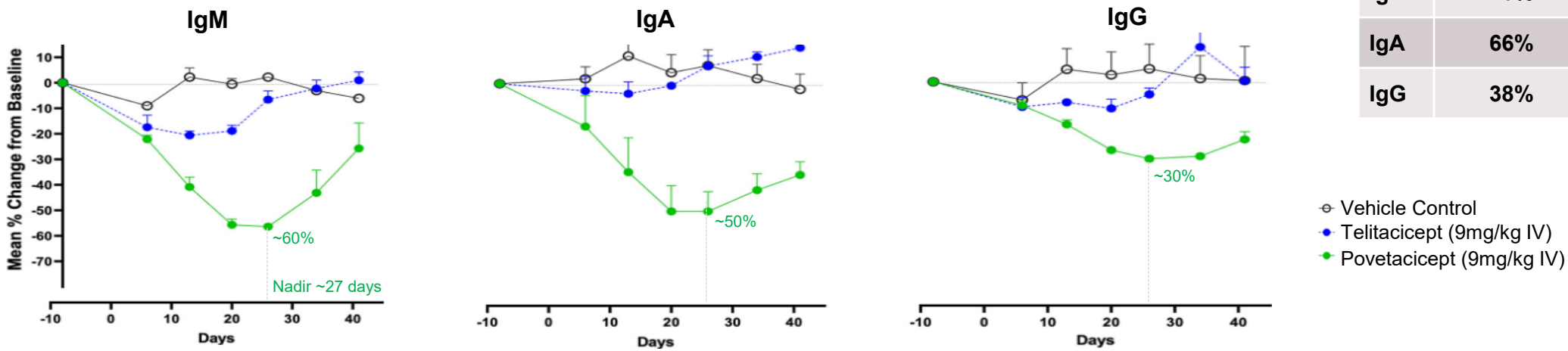
1. Method: Human PBMCs from a healthy donor were co-cultured with a TLR9 agonist and serially diluted antibodies for 24 hours. IFN-α release in the supernatant was measured using an HTRF kit
2. Method: Human PBMCs from a healthy donor were co-cultured with serially diluted antibodies for 24 hours. pDC counts were assessed via flow cytometry

DNTH212 shows superior inhibition of IgM, IgA, and IgG compared to povetacicept following single dose in NHPs

S.C. DNTH212



IV povetacicept and telitacicept[1]



Maximal Ig Inhibition		
	DNTH212 10mg/kg	Povetacicept[1] 9mg/kg
IgM	76%	~60%
IgA	66%	~50%
IgG	38%	~30%

Deeper Ig reductions have potential to drive superior clinical efficacy while maintaining at least Q4W dosing

Note: These data are derived from different studies at different points in time, with differences in methodology, design and populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials of DNTH212 and other agents have been conducted

1. Arthritis Rheumatol.2023 Jul;75(7):1187-1202. Note: WT TACI (13-118) Fc:Telitacicept

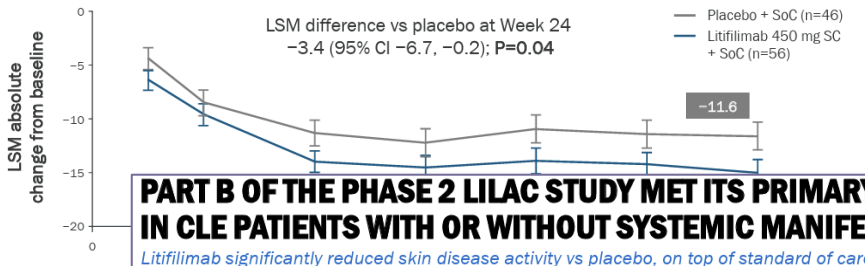
Validation of both BDCA2 and BAFF/APRIL targeted therapies support DNTH212 bifunctional approach

Positive Litifilimab (BDCA2) Data in SLE / CLE

PART A OF THE PHASE 2 LILAC STUDY MET ITS PRIMARY ENDPOINT

Improved Joint Activity: Litifilimab significantly reduced the mean total number of active joints vs placebo

Total active joint count*† in patients with SLE and active skin disease and joint involvement (N=132‡) (primary endpoint at Week 24)[1]

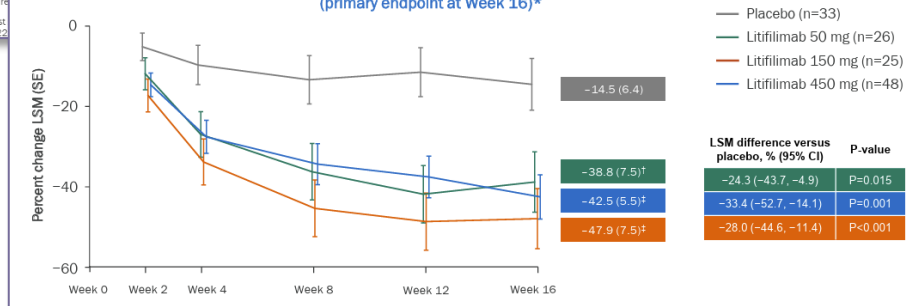


*Sum of swollen and tender joints, as
patients between the study groups pr
made. ‡Data from 12 patients who re
CI = confidence interval; LSM = least
1. Furie RA, et al. N Engl J Med 2022

PART B OF THE PHASE 2 LILAC STUDY MET ITS PRIMARY ENDPOINT IN CLE PATIENTS WITH OR WITHOUT SYSTEMIC MANIFESTATIONS

Litifilimab significantly reduced skin disease activity vs placebo, on top of standard of care

Percentage change in CLASI-A score from baseline over time (primary endpoint at Week 16)*



	LSM difference versus placebo, % (95% CI)	P-value
−38.8 (7.5)†	−24.3 (−43.7, −4.9)	P=0.015
−42.5 (5.5)‡	−33.4 (−52.7, −14.1)	P=0.001
−47.9 (7.5)‡	−28.0 (−44.6, −11.4)	P<0.001

Observed consistent safety profile with no new safety signals

*Mixed-effects model for repeated measurements; †P<0.05 versus placebo; ‡P≤0.001 versus placebo
CLASI-A = Cutaneous Lupus Erythematosus Disease Area and Severity Index-Activity; CLE = cutaneous lupus erythematosus; CI = confidence interval; LSM = least squares mean; SE = standard error
1. Werth VP, et al. N Engl J Med 2022;387:321–331

Biogen

BAFF/APRIL Validation Across Multiple Autoimmune Indications and Strategic Activity

VOR

Commercial Approvals	BLA Submissions
Validated Commercial Therapy in China Across Diverse Autoimmune Diseases	*Poised to Further Expand Telitacicept Footprint in Large, Underserved Diseases in China*
2021 - Systemic Lupus Erythematosus (SLE)†	*Est. 2026* - Primary Sjögrens Disease (pSD)
2024 - Rheumatoid Arthritis (RA)	*Est. 2026* - IgA Nephropathy (IgAN)*
2025 - Myasthenia Gravis (MG)	

Vor Bio Enters into Exclusive Global License Agreement with RemeGen for Late-Stage Autoimmune Asset

June 25, 2025

-Vor Bio receive
development for

VOR

Telitacicept Achieved Primary Endpoint in Phase 3 Clinical Study for Primary Sjögren's Disease

August 13, 2025

Phase 3 results position telitacicept as potential best-in-disease profile in primary Sjögren's disease

Telitacicept demonstrated a favorable safety profile

VERTEX

Vertex Enters Into Agreement to Acquire Alpine Immune Sciences

April 10, 2024

- Alpine is a clinical stage biotechnology company focused on discovering and developing innovative, protein-based immunotherapies -

- Alpine's lead product, povetacicept, demonstrated best-in-class potential in patients with IgA nephropathy (IgAN); Phase 3 to initiate in H2 2024 -

- Povetacicept holds promise as a pipeline-in-a-product, with clinical studies in additional serious diseases underway -

- Alpine's protein engineering and immunotherapy expertise augments Vertex's toolbox and capabilities -

- Vertex to host investor call today, April 10, at 4:30 pm ET -

BOSTON & SEATTLE--(BUSINESS WIRE)--Apr. 10, 2024-- Vertex Pharmaceuticals Incorporated (Nasdaq: VRTX) and Alpine Immune Sciences, Inc. (Nasdaq: ALPN), a biotechnology company focused on discovering and developing innovative, protein-based immunotherapies, today announced that the companies have entered into a definitive agreement under which Vertex will acquire Alpine for $65 per share or approximately $4.9 billion in cash. The transaction was unanimously approved by both the Vertex and Alpine Boards of Directors and is anticipated to close later this quarter.

Note: Company press releases and investor presentations

Broad opportunity for DNTH212 across multiple diseases where Type 1 interferon and B Cells are implicated

Indications with biological rationale and supportive clinical data

	Biological Rationale	Clinical Evidence
Primary Sjögren's Syndrome *~350,000 U.S. Patients*	✓	• *B Cell:* ianalumab positive Ph. 3; telitacicept positive Ph. 3
Cutaneous Lupus Erythematosus *~300,000 U.S. Patients*	✓	• *Type 1 interferon:* litifilimab positive Ph. 2
Systemic Lupus Erythematosus *~225,000 U.S. Patients*	✓	• *Type 1 interferon:* anifrolumab approved; litifilimab positive Ph. 2 • *B Cell:* belimumab approved; telitacicept approved (CN); ianalumab positive Ph. 2
Lupus Nephritis *~120,000 U.S. Patients*	✓	• *B Cell:* belimumab approved
Dermatomyositis *~50,000 U.S. Patients*	✓	• *Type 1 interferon:* dazukibart positive Ph. 2

Indications with biological rationale

	Biological Rationale
Hidradenitis Suppurativa *~330,000 U.S. Patients*	✓
Scleroderma *~75,000 U.S. Patients*	✓
Pemphigus Vulgaris *~32,000 U.S. Patients*	✓

Dianthus to provide update on indication prioritization in 1H 2026

Type 1 interferon targeting Ph. 3 studies currently ongoing in: SLE, CLE, LN, Scleroderma, DM. B Cell (BAFF/APRIL) targeting Ph. 3 studies currently ongoing in: LN, SLE
Estimated U.S. patients per Dianthus meta-analysis and estimates

Ph. 1 study initiated in China in Dec. ’25 with top-line Part A HV results in 2H’26

Healthy Volunteers (Part A)

~46 HVs enrolled into seven cohorts:

- Treated (N= up to 6)
- Placebo (N= up to 2)

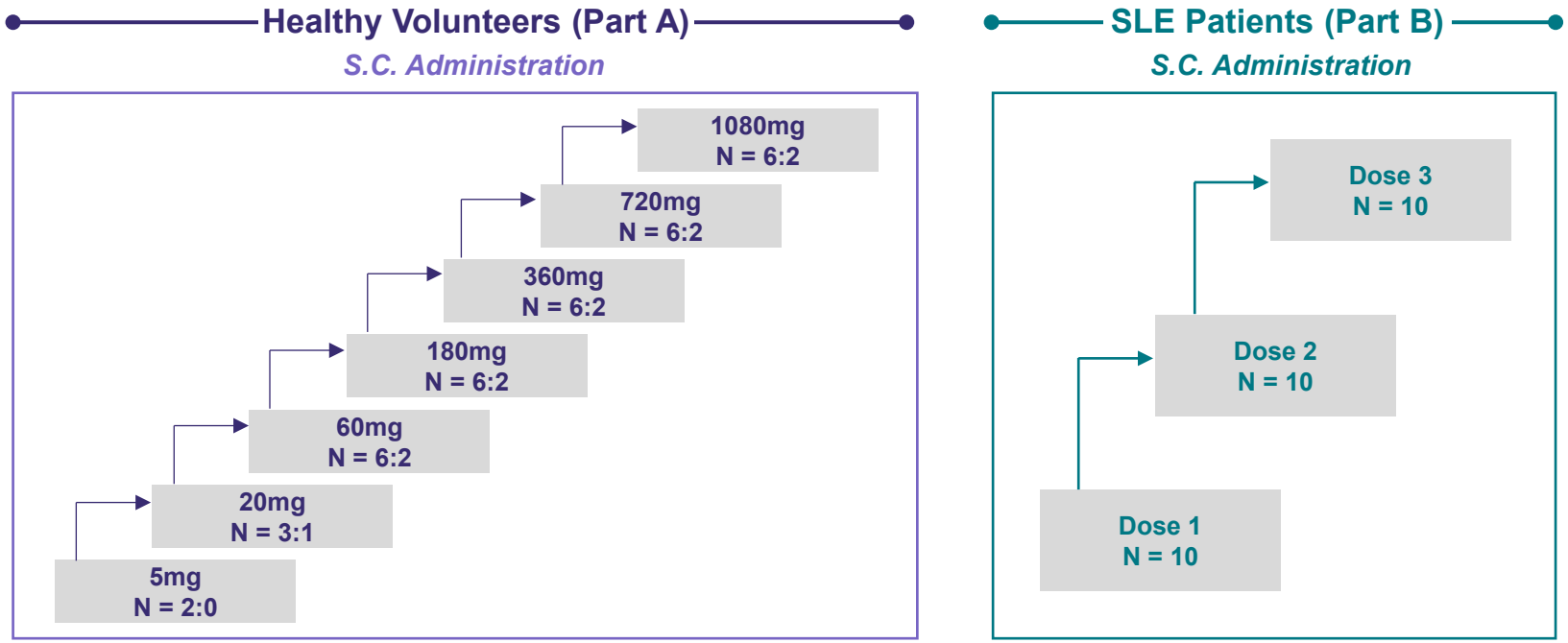
SLE Patients (Part B)

~30 patients enrolled into three cohorts:

- Treated (N= up to 10)

Key Parameters

- **Safety, PK, and PD as well as other biomarkers and preliminary efficacy**



Phase 1 trial designed to evaluate safety, tolerability and PK/PD

DNTH212 TPP aims to deliver superior efficacy in a safe and well-tolerated therapy with patient friendly convenience

EFFICACY

Bifunctional approach has potential for *superior* efficacy in various disease states versus only targeting innate or adaptive immune system

SAFETY

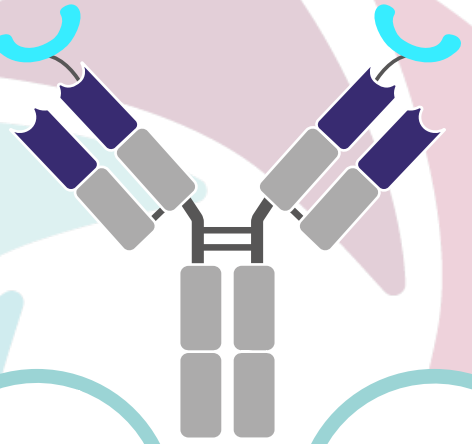
Inhibiting Type 1 interferon or BAFF/APRIL has been generally safe and well tolerated

CONVENIENCE

Targeting patient friendly S.C. self-administration with Q4W or less frequent dosing

Achieving the TPP would position DNTH212 as a first-line biologic across a range of indications

Achieving DNTH212 TPP would position DNTH212 as a first-line, best-in-class therapy across multiple indications



Clinically Validated MoA	Superior Efficacy	Patient-friendly Convenience	Favorable Safety Potential	Pipeline-in-a-Product	Robust IP Protection
Type 1 interferon and BAFF/APRIL are known drivers of autoimmune disease	Targeting innate and adaptive immune systems has potential for superior efficacy	Potential S.C. self-administration Q4W or less frequent	Inhibiting Type 1 interferon or BAFF/APRIL has been generally safe and well tolerated	Broad opportunity across multiple diseases where Type 1 interferon and B-cells are implicated	Composition of matter patent expected to expire no earlier than 2044

DNTH212 is an investigational agent that is not approved as a therapy in any indication in any jurisdiction worldwide

Recap of Dianthus Leadership in Severe Autoimmune Diseases

Advancing a leading autoimmune-focused biotech with two clinical-stage programs

Program	Indication	Ph. 1	Ph. 2	Ph. 3	Upcoming Milestones
Claseprubart *aC1s*	**gMG** >100,000 U.S. Patients				• Expect to initiate Ph. 3 study in 2026
	CIDP >40,000 U.S. Patients				• Interim responder analysis expected in Q2'26 • *Peer Catalyst: riliprubart Ph. 3 MOBILIZE and VITALIZE (H2H vs. IVIG) data expected by early '27*[3]
	MMN >10,000 U.S. Patients				• Ph. 2 top-line results expected in 2H'26 • *Peer Catalyst: empasiprubart Ph. 3 data in Q4'26*[4]
DNTH212 *BDCA2 and BAFF/APRIL*	**Multiple Autoimmune Diseases**	*Healthy volunteers (Part A)* *SLE patients (Part B)*			• Update on indication prioritization in 1H'26 • Ph. 1 HV top-line results expected in 2H'26

Strong balance sheet with ~$514M[1] of cash & runway into 2028
~44.8M shares outstanding[2]

1. Estimated cash includes preliminary and unaudited cash, cash equivalents and investments as of December 31, 2025
2. Shares outstanding on a pro forma basis, which assumes the exercise of all outstanding pre-funded warrants
3. Based on Sanofi Q3'25 financial results conference call transcript
4. Based on publicly available information: https://argenx.com/news/2026/press-release-3216531

Appendix

C1s is a clinically validated target in the classical complement pathway with an FDA approved therapy

1

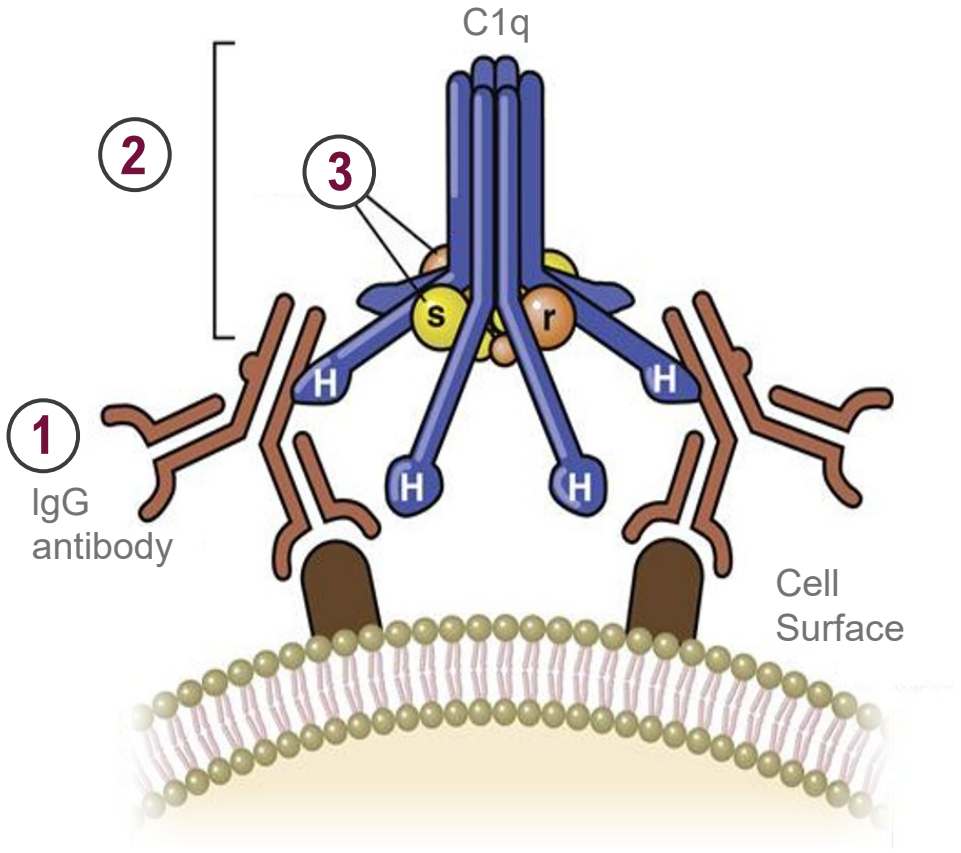
Classical pathway
The only pathway activated by the presence of IgG and IgM, which bind to the **C1 complex**

2

The C1 complex
The initial component of the classical complement pathway consisting of C1q, C1r and C1s

3

Active C1s
A serine protease that executes catalytic function of the C1 complex, leading to MAC formation



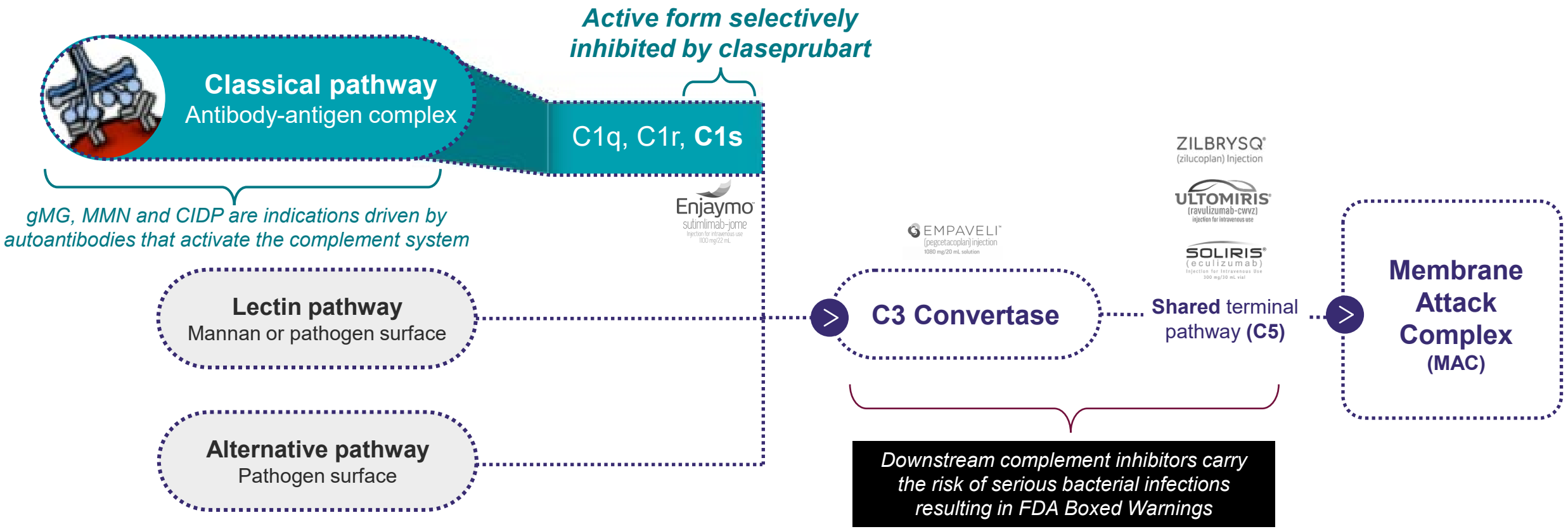
C1s is the only target of the C1 complex with an FDA approved therapy
Enjaymo, FDA approved in 2022 for CAD, is a C1s inhibitor but is not selective to the active form and dosed I.V. at 6,500-7,500mg every two weeks

Active C1s inhibition has recently demonstrated clinical benefit in CIDP
Riliprubart results show clinical PoC for inhibiting active C1s in autoimmune neuromuscular diseases

Enjaymo information sourced from prescribing information

Selectively targeting classical pathway aims to provide effective but safer complement inhibitor

Targeting aC1s aims to deliver efficacy demonstrated with terminal inhibitors while preserving the critical immune activity of lectin and alternative pathways, leading to a lower risk of infection and no FDA boxed warning/REMS



Unlike C5 inhibitors, ENJAYMO® has no FDA boxed warning and REMS, or prophylactic antibiotic requirement prior to vaccination

ULTOMIRIS® (ravulizumab-cwvz) injection for intravenous use 300 mg/3 mL vial

C5 inhibitor

✗ Boxed Warning and REMS

ULTOMIRIS® (ravulizumab-cwvz) injection, for intravenous or subcutaneous use
Initial U.S. Approval: 2018

WARNING: SERIOUS MENINGOCOCCAL INFECTIONS
See full prescribing information for complete boxed warning.

ULTOMIRIS increases the risk of serious and life-threatening infections caused by *Neisseria meningitidis*.

- **Complete or update meningococcal vaccination at least 2 weeks prior to the first dose of ULTOMIRIS, unless the risks of delaying ULTOMIRIS outweigh the risks of developing a serious infection. Comply with the most current Advisory Committee on Immunization Practices (ACIP) recommendations for meningococcal vaccination in patients receiving a complement inhibitor. (5.1)**
- **Patients receiving ULTOMIRIS are at increased risk for invasive disease caused by *N. meningitidis*, even if they develop antibodies following vaccination. Monitor patients for early signs and symptoms of meningococcal infections and evaluate immediately if infection is suspected. (5.1)**

ULTOMIRIS is available only through a restricted program called ULTOMIRIS and SOLIRIS REMS. (5.2)

✗ Antibiotic Prophylaxis Required if not Fully Vaccinated

2.2 Recommended Vaccination and Prophylaxis for Meningococcal Infection

Vaccinate patients against meningococcal infection (serogroups A, C, W, Y and B) according to current ACIP recommendations at least 2 weeks prior to initiation of ULTOMIRIS *[see Warnings and Precautions (5.1)]*.

If urgent ULTOMIRIS therapy is indicated in a patient who is not up to date with meningococcal vaccines according to ACIP recommendations, provide the patient with antibacterial drug prophylaxis and administer these vaccines as soon as possible.

Healthcare providers who prescribe ULTOMIRIS must enroll in the ULTOMIRIS and SOLIRIS REMS *[see Warnings and Precautions (5.2)]*.

Enjaymo sutimlimab-jome Injection for intravenous use 1100 mg/22 mL

C1s inhibitor

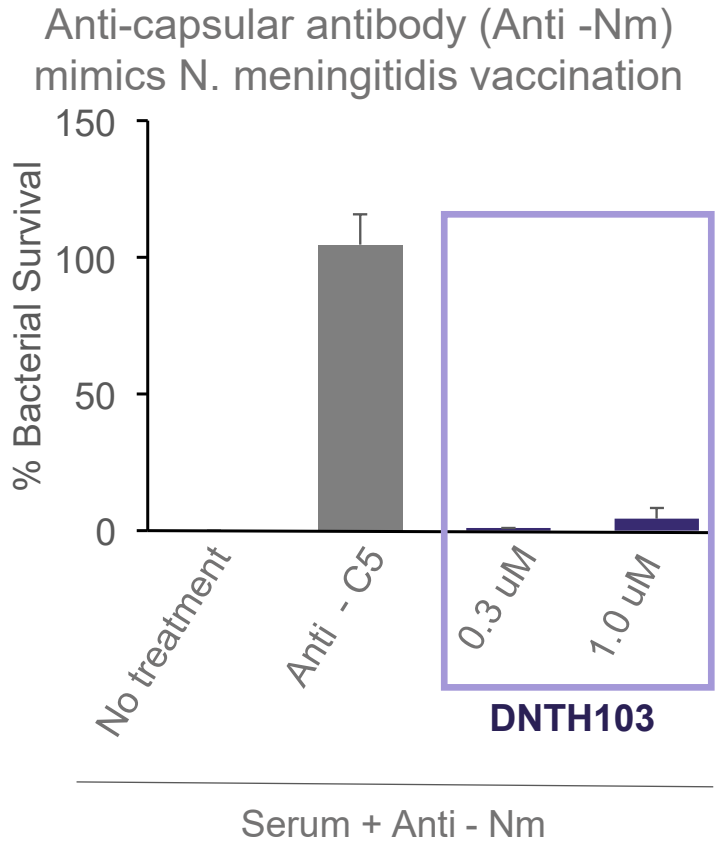
✓ No Boxed Warning or REMS

✓ No Antibiotic Prophylaxis Required

Source: ULTOMIRIS prescribing information & ENJAYMO prescribing information

Claseprubart *in vitro* study indicates lower risk of *Neisseria meningitidis* infections

- Protection against infection is a critical function of the complement pathway
- **DNTH103 selectively inhibits** the **classical pathway**, leaving the alternative and lectin-activated defense pathways intact
- An *in vitro* assay measured **antibody-dependent complement-mediated killing of *N. meningitidis*** in the presence of **DNTH103** and **anti-C5 (ravulizumab*)**
- In this assay, **DNTH103 maintained bacterial killing**, potentially leading to a decreased risk of infection vs. C5 inhibitors



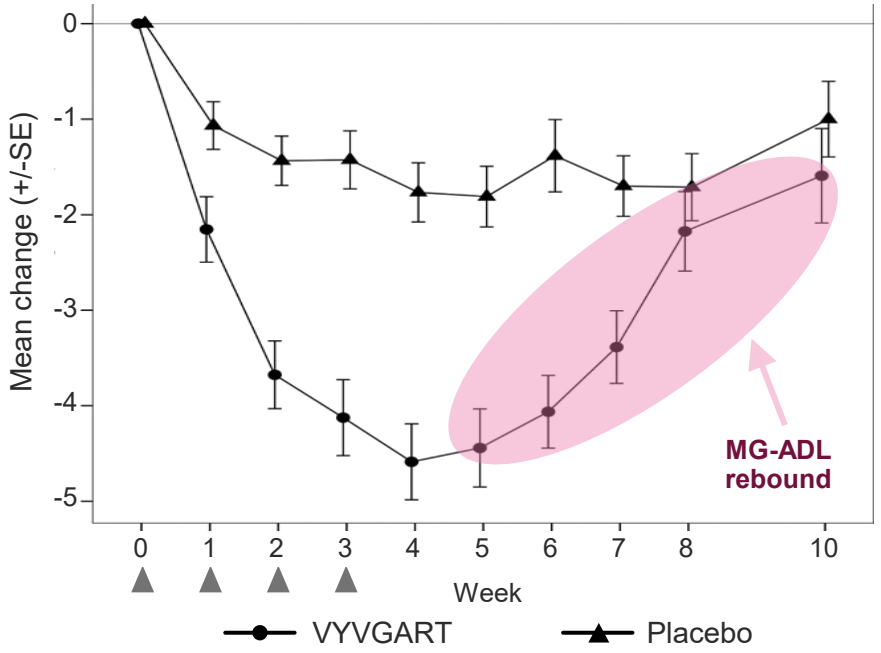
Results further validate the potential differentiated safety profile for DNTH103 as a selective classical pathway inhibitor consistent with ENJAYMO, an approved C1s inhibitor without an FDA Boxed Warning or REMS

* Engineered using patent sequence

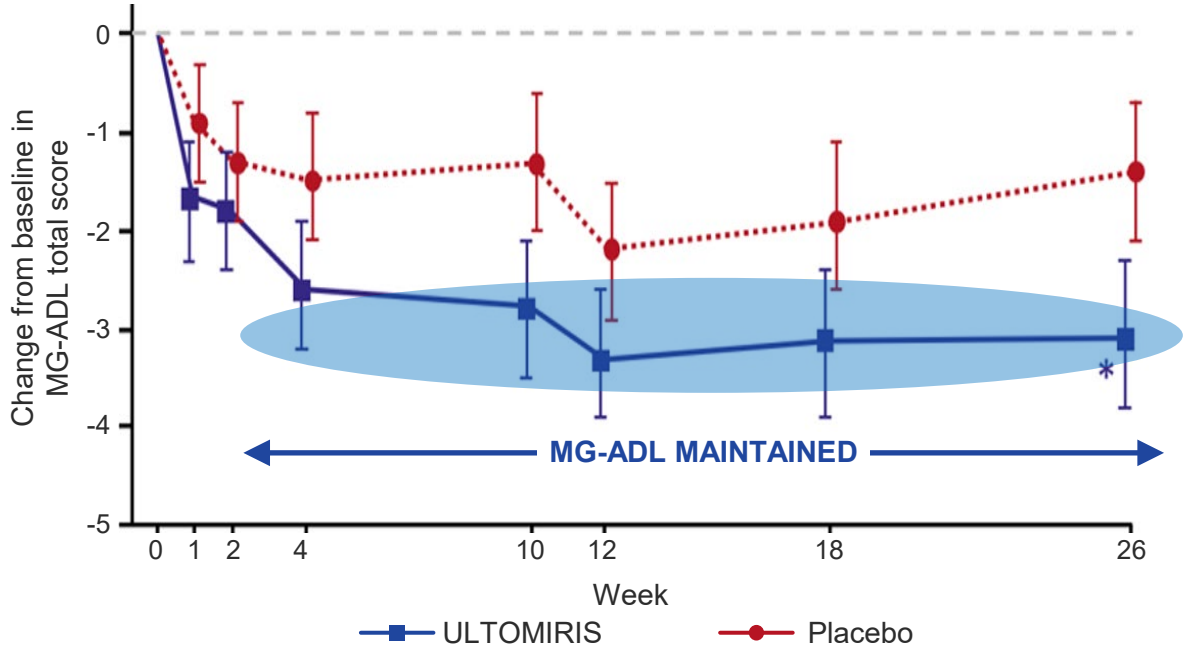
Claseprubart aims to provide consistent symptom control with convenient Q2W S.C. dosing

- Chronic diseases like MG benefit from **consistent treatment and symptom control**
- When patients are required to take a drug holiday on FcRns, **MG-ADL scores immediately begin to rebound** as patients self-report symptoms and disease worsening
- Real-world evidence[1] suggests **sustained inhibition / treatment with complement is better over time**

VYVGART®: Cyclic Dosing Leads to MG-ADL Rebound



ULTOMIRIS®: Complement Inhibition Provides Consistent MG-ADL Symptom Control



▲ Indicates VYVGART® dose administered

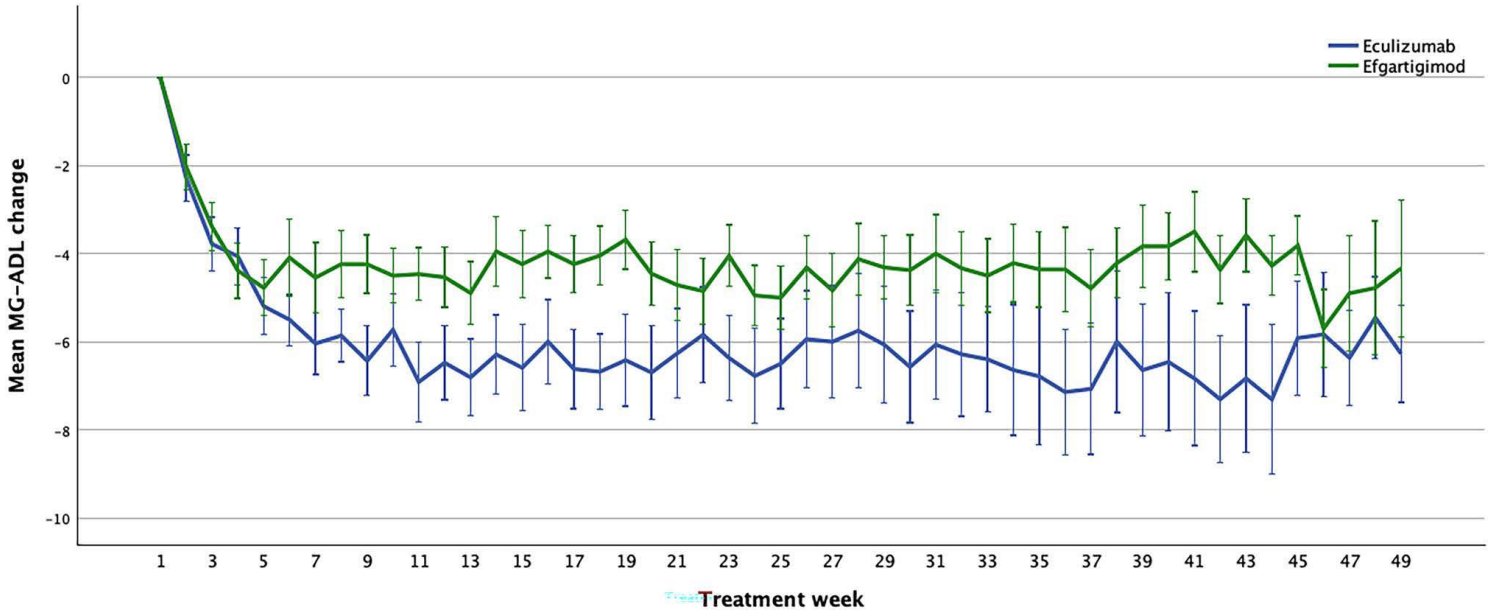
Source: VYVGART® prescribing information and ULTOMIRIS® prescribing information

1. Pane et al. A real-life experience with eculizumab and efgartigimod in generalized myasthenia gravis patients. *J Neurol* 271, 6209–6219 (2024)

Real-world evidence demonstrates clinical advantages for complement vs. FcRns

Real-world Evidence Indicates Sustained MG-ADL Reductions for Complement Inhibitors vs. FcRns, Among Other Clinical Benefits

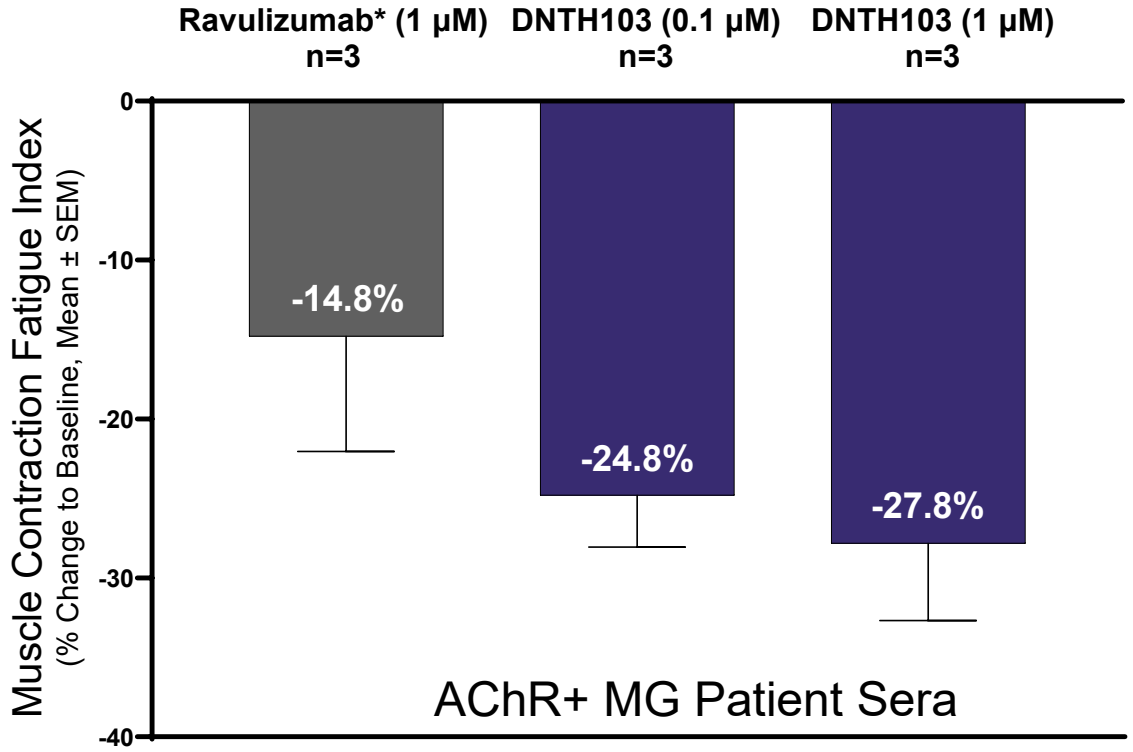
- **Patients on eculizumab showed a greater reduction in MG-ADL** over time than those on efgartigimod
- **Eculizumab showed statistically significant, deeper QMG reduction** and higher responder rate than efgartigimod
- **Eculizumab patients had a statistically significant greater reduction in rate of clinical events** (deteriorations, crisis or hospitalization) than efgartigimod
- **Patients on efgartigimod were more likely to suspend treatment** (p = 0.015 for AChR+ patients) with the "main reason for discontinuation was MG deterioration"
- **Eculizumab patients also had a statistically significant greater reduction in steroid dose** coming from a higher baseline dose vs. efgartigimod patients, but reaching a numerically lower dose during the treatment period



Source: Pane et al., J Neurol 271, 6209–6219 (2024)

Claseprubart improves neurotransmission and muscle contraction in an AChR+ MG model

- **Serum from MG patients** used in a validated in vitro MG model[1,2,3]
- **Assessed improvement in neurotransmission and muscle contraction** of ravulizumab* and DNTH103, as measured by decrease in muscle contraction fatigue
- **Results confirm DNTH103 improved neurotransmission and muscle contraction**

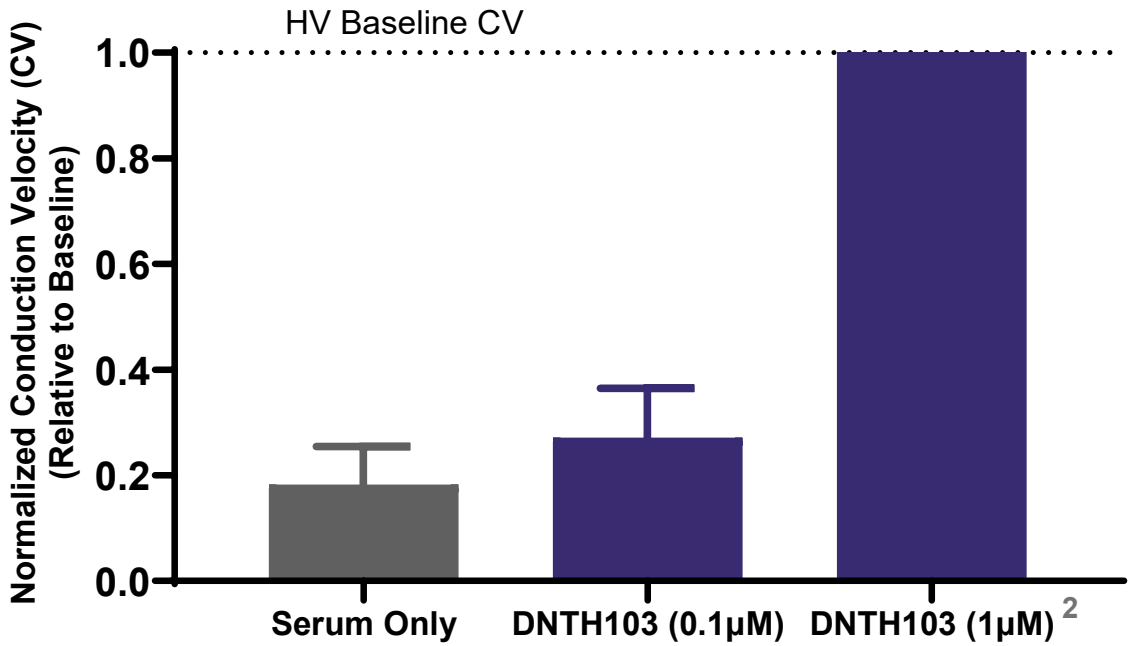


Results provide further scientific rationale for DNTH103 in gMG

1. Smith et al., Front Cell Dev Biol. 2021;9:745897; 2. Vila et al., Expert Opin Drug Discov. 2020;15(3):307-317; 3. Vila et al., Theranostics. 2019;9(5):1232-1246.
* Engineered using patent sequence

Claseprubart restores neuronal conduction velocity in an *in vitro* CIDP model

- **Serum from 3 CIDP patients** was evaluated in a , commercially available *in vitro* CIDP model[1]
- **Assessed improvement in neuronal conduction velocity** of two doses of DNTH103 as compared to baseline conduction velocity determined in sera from healthy volunteers (N=3)
- **Results confirm DNTH103 completely restored conduction velocity** across the axons of human motor neurons in the presence of autoantibodies from CIDP patient sera



Results provide further scientific rationale for DNTH103 in CIDP

1 Rumsey et al., *Adv. Therap.*, 2022, 5(6): 2200030
2 Results for DNTH103 (1uM) include data from multiple conduction velocity recordings that exceed 1.0. For the purposes of this illustration, results are shown up to the baseline value

Claseprubart Phase 1 healthy volunteer study was designed to validate extended half-life, potency and safety

SAD

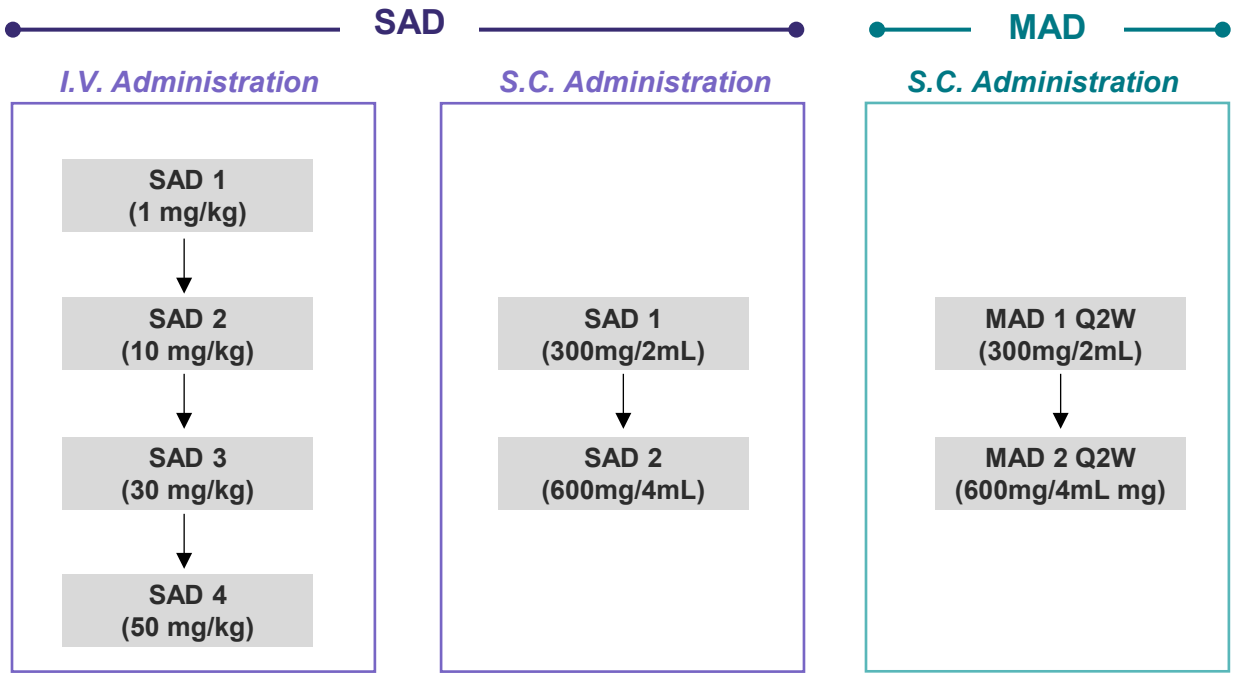
44 HVs enrolled into six cohorts:
- Placebo (N= up to 2)
- Treated (N= up to 6)

MAD

16 HVs enrolled into two cohorts:
- Placebo (N= up to 2)
- Treated (N= up to 6)

Key Parameters

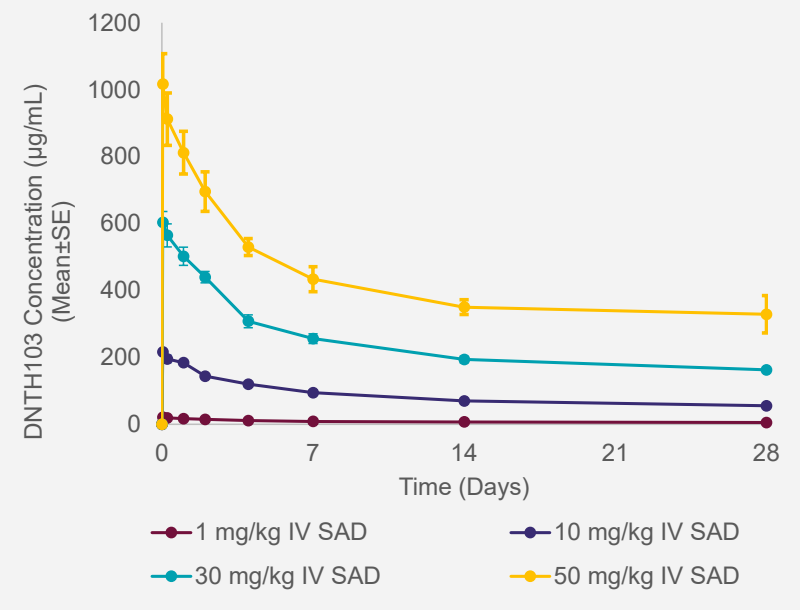
- **Safety, PK, and PD measured by percent classical pathway inhibition quantified in each cohort**



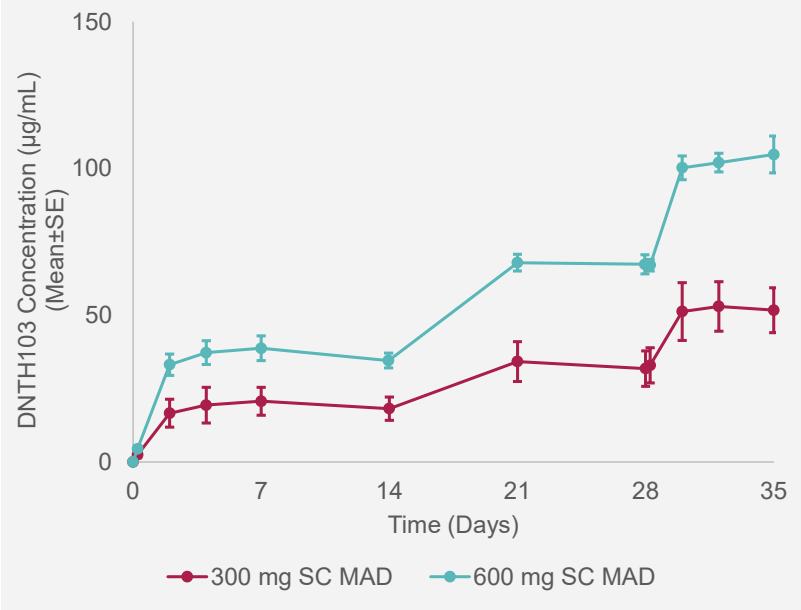
60 healthy volunteers completed dosing as of December 2023 across these eight cohorts

Claseprubart has demonstrated deep and sustained complement inhibition in healthy volunteers

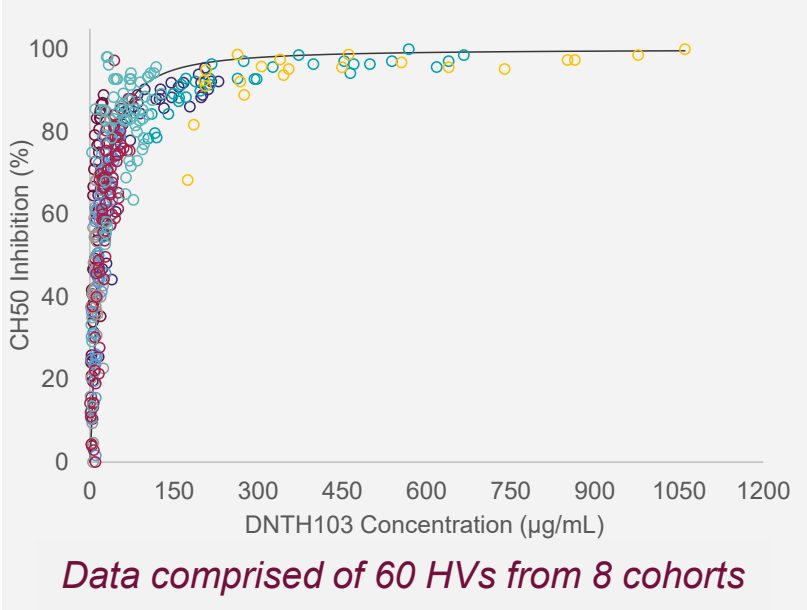
I.V. SAD:
Linear PK with Exposure Proportional Across Doses



S.C. MAD:
Strong Accumulation with Q2W Dosing



PK/PD:
Analysis Demonstrates IC90 of 87 µg/mL



Data comprised of 60 HVs from 8 cohorts

Claseprubart demonstrated a ~60-day half-life and IC90 of 87 µg/mL

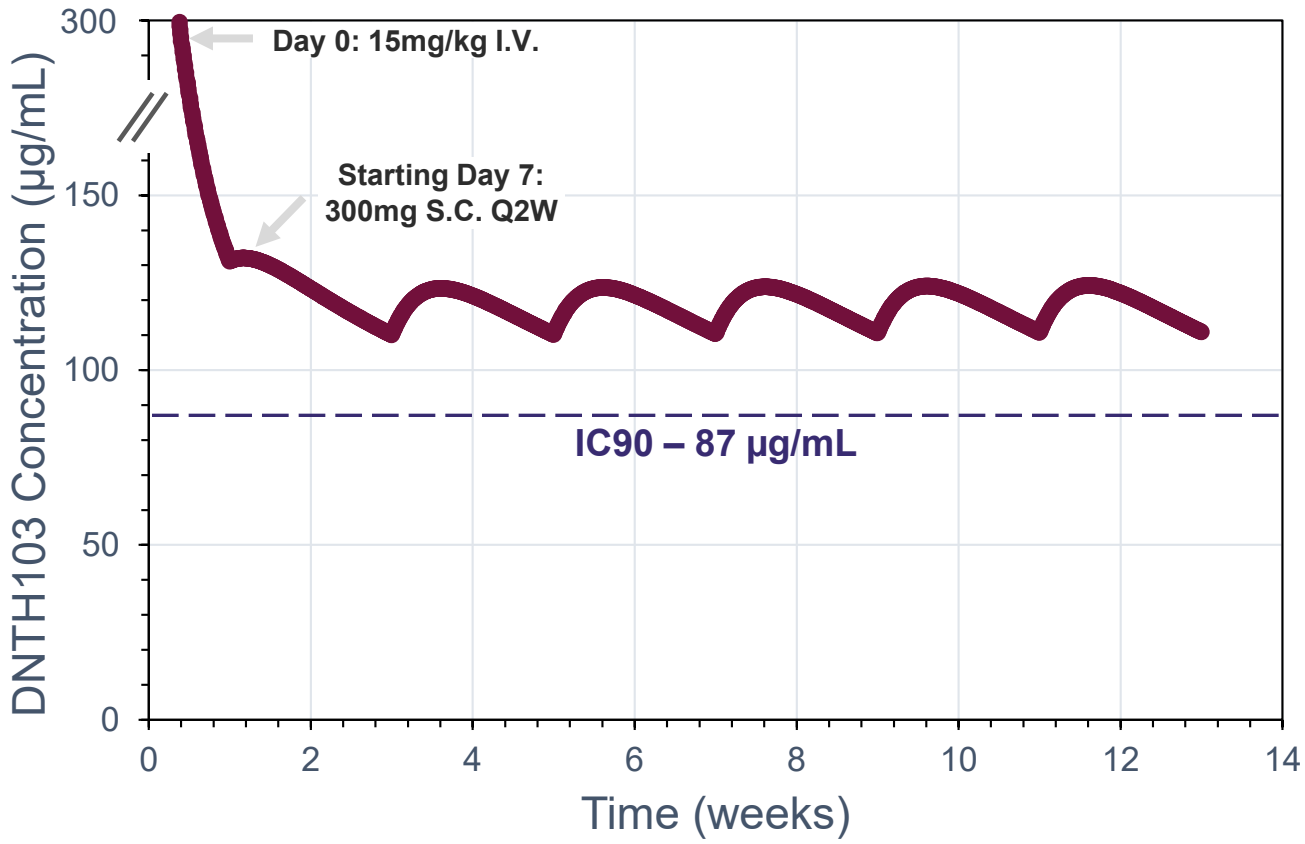
Phase 1 data estimated IC90 at ~87 µg/mL, leading to target dose of 300mg/2mL in Ph. 2 to achieve steady state >IC90

Ph. 1 Data Confirms

- ~60-day half-life
- IC90 calculated at 87 µg/mL

Dosing Modeled

- 15mg/kg I.V. on Day 0
- 300mg S.C. Q2W starting Day 7



Simulation using data from 60 healthy volunteers dosed across multiple cohorts demonstrates 300mg/2ml Q2W achieves steady state above target inhibition of IC90 at 87 µg/mL

Claseprubart was generally well tolerated, with a favorable safety profile in Phase 1

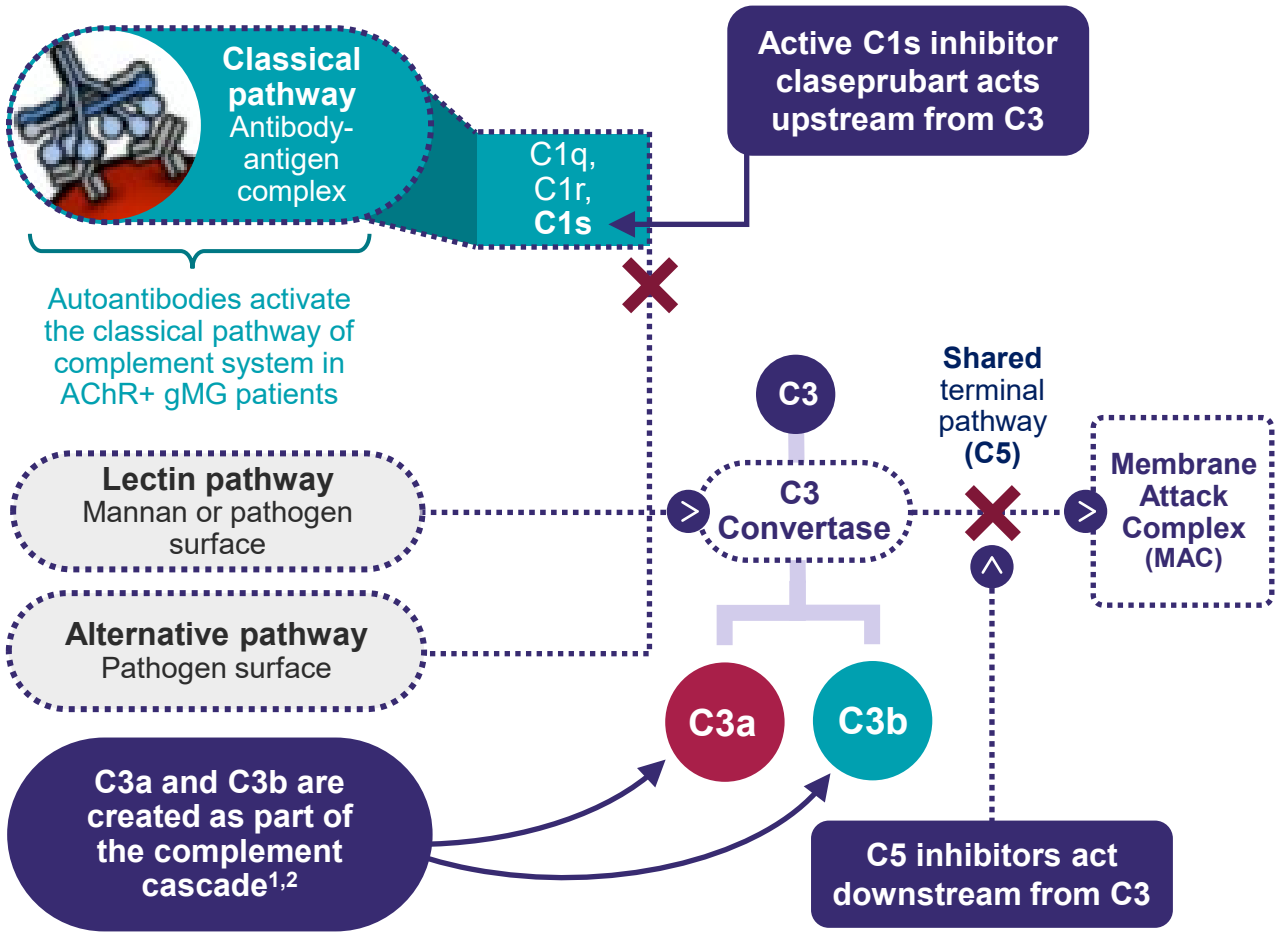
- **No standard safety lab findings (hematology, chemistry, coagulation LFTS and renal function)**
- **No serious adverse events**
- **No infection adverse event signal and no infections related to encapsulated bacteria**

	I.V. & S.C. SAD (n=44)			S.C. MAD (n=16)	
	Pooled DNTH103 I.V. (n=21)	Pooled DNTH103 S.C. (n=12)	Pooled Placebo I.V. / S.C. (n=11)	Pooled DNTH103 S.C. (n=12)	Pooled Placebo S.C. (n=4)
Participant with:					
Any AEs	13 (62%)	9 (75%)	7 (64%)	8 (67%)	4 (100%)
Any SAEs	0	0	0	0	0
Grade 3 / 4 AEs	0	0	0	0	0
Treatment Related AEs	2 (10%)	1 (8%)	0	2 (17%)	0

- Five participants experienced mild/moderate Treatment Related AEs
  - Two participants (one in each 300mg/2mL and 600mg/4mL S.C. MAD cohorts) had a mild or moderate injection site reactions (ISRs); no intervention was required and both participants completed treatment
  - One participant experienced several non-specific AEs during infusion; infusion was paused for 8 minutes and restarted at the same rate without sequelae
  - Two participants in 50mg/kg SAD I.V.[1] cohort became ANA[2] positive at Day 57; both participants had no evidence of SLE and both tested negative for dsDNA[3]
  - One participant in 600mg/4mL S.C. SAD reported vomiting on Day 1, which resolved on same day

1 Highest dose used in Phase 2 trial was single I.V. loading dose of 20mg/kg
2 Non-specific indicator of autoimmune disease present in up to 25% of healthy individuals: https://www.labcorp.com/assets-media/2785
3 Anti-double-stranded deoxyribonucleic acid antibodies are highly specific markers of systemic lupus erythematosus or SLE

Pro-inflammatory split products C3a and C3b have potential negative effects on NMJ pathology beyond MAC in gMG



C3a and C3b Are Elevated in AChR+ gMG – Both Cause Inflammatory Damage[1,2]

C3a	C3b
• Anaphylotoxin[2,3] • Initiates mast cell degranulation[4] • Promotes neutrophil-mediated acute phase reactions[5] driven by pro-inflammatory cytokines[6] – particularly IL-6 in gMG[7] • Has the potential to directly act on NMJ via smooth muscle contraction and endothelial permeability[8]	• Opsonin[3] • Forms part of an amplification loop that perpetuates a cycle of complement activation[3] • Facilitates integrin-mediated phagocytois[3]

Ab, antibody; gMG, generalized myasthenia gravis; IL, interleukin; MAC, membrane attack complex; NMJ, neuromuscular junction

1. Stascheit F, et al. *Eur J Neurol* 2023;30:1409–16; 2. Iacomino N, et al. *Biomedicines* 2022;10; 3. Watanabe-Kusunoki K, Anders HJ. *J Autoimmun* 2024;145:103216; 4. Nilsson G, et al. *J Immunol* 1996;157:1693–8; 5. Riaz B, Sohn S. Cells 2023;12; 6. Wang Y, et al. J Neurol 2025;272:489; 7. Uzawa A, et al. J Neuroimmunol 2021;358:577634; 8. Drouin SM, et al. *J Immunol* 2001;166:2025–32; 2015;2:e119

Classical pathway / aC1s inhibition has the potential to change the CIDP landscape

	FcRn	Complement Inhibitors		
	Efgartigimod S.C. QW	**Empasiprubart (C2) I.V. Q4W**	**Riliprubart (aC1s) 600mg/4mL S.C. QW**	**Claseprubart (aC1s) 300mg/2mL S.C. Q2W**
Ph. 3 Study Populations	adhere SoC-Treated Off Treatment	emnergize SoC-Treated Off Treatment emvigorate H2H vs IVIG Treated	Mobilize Clinical Trial SoC Refractory Vitalize Clinical Trial H2H vs IVIG Treated	CAPTIVATE SoC-Treated SoC-Refractory SoC-Naïve
IVIG Withdrawal Required Prior to Entering Study[1]	**YES**	**NO**	**NO**	**NO**
Endpoints / Results	• **Confirmed ECI[2]** • **Ph. 3 Stage A results:** – 66.5% ECI (wk 12) *~1/3 pts **did not return** to pre-washout baseline*	• **≥1-point aINCAT improvement**	• **≥1-point aINCAT improvement** • **Ph. 2 PoC response rates:** – SOC-Treated: 52% – SOC-Refractory: 50%	• **≥1-point aINCAT improvement** • **Part A expectations:** – Targeting similar response in open-label Part A to riliprubart open-label Ph. 2

- **aC1s inhibitors enroll a broad patient population including SOC-refractory patients**
- **FcRns are not being evaluated H2H vs. IVIG**
- **No requirement for disease worsening in ongoing complement trials**
- **≥1-point aINCAT improvement used as efficacy measure in ongoing studies**

Source: Company filings, presentations and clinicaltrials.gov

1. ADHERE required removal of IVIG and evidence of clinically meaningful deterioration before dosing in Part A
2. Defined as a clinical improvement on the parameters that the participant worsened in during run-in (≥4-point increase in I-RODS and/or ≥8-kPa increase in mean grip strength) or clinical improvement (≥1-point decrease) in INCAT

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