UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): June 12, 2026

DIANTHUS THERAPEUTICS, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-38541**	**81-0724163**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

7 Times Square **43rd Floor** **New York, New York**	**10036**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (929) 999-4055

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, $0.001 Par Value	DNTH	The Nasdaq Capital Market

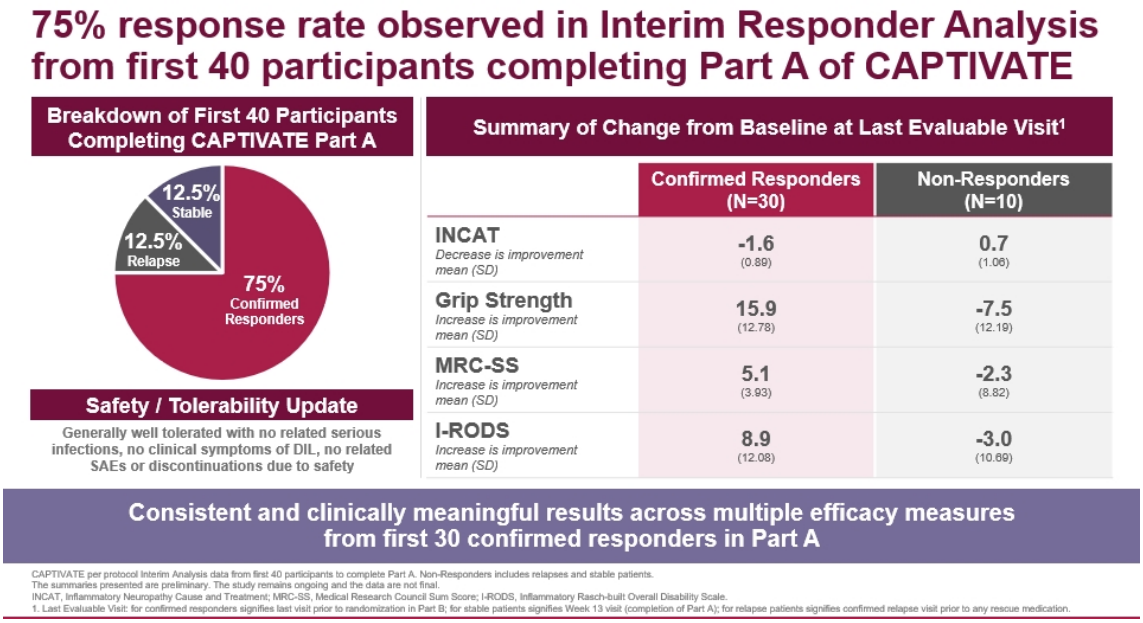
Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 8.01 Other Events.

On June 11, 2026, Dianthus Therapeutics, Inc. (the "Company") posted an updated corporate presentation (the "Presentation") on the investor relations section of its website, which included the following interim responder analysis data for the first 40 patients that completed Part A of the Phase 3 CAPTIVATE trial of claseprubart in chronic inflammatory demyelinating polyneuropathy:



The Presentation is filed as Exhibit 99.1 and is incorporated by reference into this Item 8.01.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Corporate Presentation of Dianthus Therapeutics, Inc., dated June 2026
104	Cover Page Interactive Data File (embedded within the inline XBRL document)

SIGNATURES

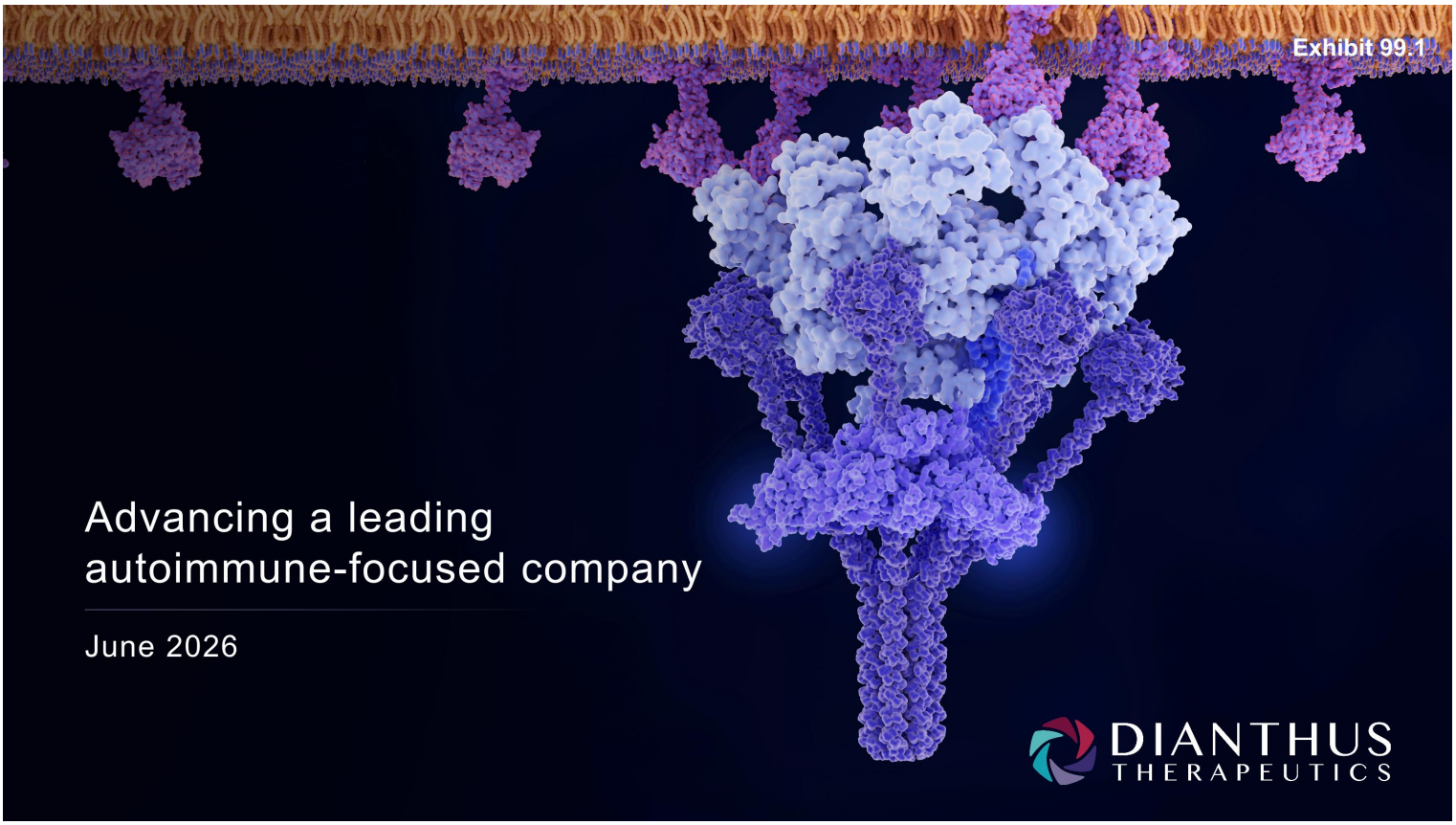
Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

DIANTHUS THERAPEUTICS, INC.

Date: June 12, 2026

By: /s/ Adam M. Veness, Esq.
Adam M. Veness, Esq.
SVP, General Counsel and Secretary

Exhibit 99.1



Advancing a leading autoimmune-focused company

June 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 preliminary interim analysis based on a limited number of patients from the Part A open label portion of the claseprubart CAPTIVATE study in patients with chronic inflammatory demyelinating polyneuropathy may not be predictive of the results or success of the remaining patients treated in Part A or patients treated in Part B of the CAPTIVATE study, 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, 2025, 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.

The forward-looking statements in this presentation speak only as of the date they are made and are qualified in their entirety by reference to the cautionary statements herein. 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 with neuromuscular and rheumatology franchises

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

Claseprubart: Neuromuscular Franchise
(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, early** Interim Responder Analysis **GO decision** in **Ph. 3 CIDP,** and clinical **PoC for CP inhibition in 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: Rheumatology Franchise
(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**
- **Initial priority indications selected: SjD, SLE and DM**

Claseprubart 2026 milestones:
Ph. 3 gMG trial initiation (mid'26), Ph. 2 MMN top-line data (Q4'26) and Ph. 3 CIDP Part B top-line guidance by YE'26

DNTH212 2026 milestones:
Ph. 1 healthy volunteer study top-line data (2H'26)

Strong financial position with cash of ~$1.2B[1] and runway expected into 2030 to fund multiple near and long-term catalysts

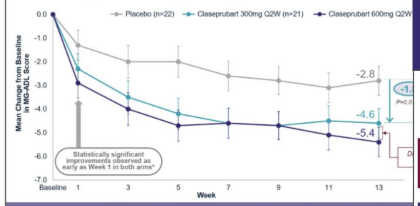
SjD: Sjögren's Disease; SLE: Systemic Lupus Erythematosus; DM: Dermatomyositis
1. Cash, cash equivalents and investments as of March 31, 2026

Building on the promise of claseprubart to be a potential pipeline-in-a-product and best-in-disease therapy in growing and underserved markets

Ph.2 MaGic Showed Rapid & Robust Improvements in gMG

Q3'25

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



MG-ADL improvements for participants treated with cl were rapid, sustained, clinically meaningful and statistically signifi

The change from baseline at MG-ADL was analyzed using a mixed effect model for repeated measures (MMRM) with treatment group, visit, treatment by visit interaction, stratification fac minimal 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.

CAPTIVATE Part A Early GO Decision in CIDP

Q1'26

Announced early GO decision reached with less than 40 planned participants completing Part A in M

CAPTIVATE Interim Analysis Objective	Targeting response rate of 50% or greater (≥20 patien Part A) based on precedent set with aC1s inhibition
GO Decision	GO decision reached early after 20 confirmed respon than 40 planned participants completing Part A
Safety / Tolerability Update	Independent DSMB reviewed the data to date and cor no related serious infections, no clinical symptoms o no related SAEs or discontinuations

GO decision supports continued development of claseprubart at 30 targeting a potentially best-in-disease biologic

CAPTIVATE interim analysis data cut off as of March 4, 2026
Drug-induced Lupus (DIL) is an autoimmune syndrome triggered by specific medications, such as statins, TNF-alpha inhibitors, and ACE inhibitors/beta-blockers. A key distinguishing typically resolve once the offending medication is withdrawn.

Ph. 2 MoMeNtum Top-line Data Anticipated Q4'26

Q4'26

Phase 2 MoMeNtum top-line data in MMN anticipated Q4'26

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
- No **ANA screening exclusion criteria or routine ANA testing** during the RCT or OLE

Endpoints

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

Collect data for safety, PK, PD, time to IVIg retreatment, time to relapse, grip strength and other muscle strength and motor function measurements

MOMENTUM

Top-line data expected in Q4'26

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

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

	Claseprubart			DNTH212
Indications	**gMG**	**CIDP**	**MMN**	**SjD, SLE and DM**
	>100,000 U.S. patients	***>40,000 U.S. patients***	***>10,000 U.S. patients***	**~625,000 U.S. patients**
Market Insight	Multi-billion $, growing market with opportunity for a best-in-disease, convenient therapy to expand first-line biologics use	Active C1s inhibition has shown robust efficacy in patients who were refractory, stable, and naïve to IVIg	Empasiprubart, a C2 inhibitor, demonstrated impressive efficacy in MMN, validating classical pathway inhibition	Targets both 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-disease profile demonstrating rapid, robust, continuous symptom control with convenient, Q2W or Q4W S.C. dosing and a potentially differentiated safety profile	Observed 75% response rate from first 40 participants to complete CAPTIVATE Part A, supporting best-in-disease potential	Demonstrated superiority vs. empasiprubart in head-to-head *in vitro* classical pathway potency experiment, with potential to be a best-in-disease therapeutic with limited competition	Validation of both BDCA2 and BAFF/APRIL targeted therapies support bifunctional approach, with potential for best-in-disease efficacy
Next Milestone	**Ph. 3 Top-line Data in 2H'28**	**Part B Top-line Guidance by YE'26**	**Ph. 2 Data in Q4'26**	**Ph. 1 HV Top-line Data 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
Estimated SjD, SLE and DM U.S. patients per Dianthus meta-analysis and estimates

Claseprubart: Building a Best-in-Disease Neuromuscular Franchise

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

claseprubart

CONFIDENCE

Aim for Potent, Rapid, Consistent Efficacy

Potential for Best-in-Disease Efficacy

Broad Potential in Autoimmune Diseases

CLASSICAL

Upstream Inhibition Prevents Pro-inflammatory C3a/C3b

Potential to Preserve Immune Function

Targeting No Boxed Warning or REMS

CONVENIENCE

<10-Second Autoinjector

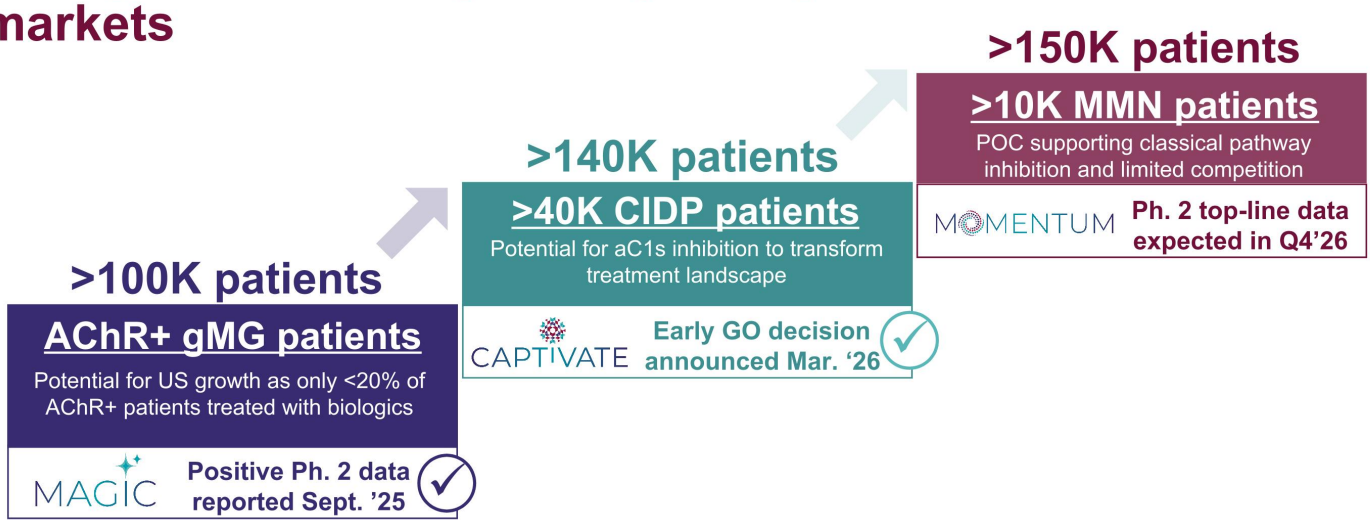
One-Click Every 2 or 4 Weeks

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

Targeting a best-in-disease, 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 potential 1L biologic in three large and growing US neuromuscular markets



Claseprubart has potential to capture meaningful market share across three synergistic multi-billion dollar markets

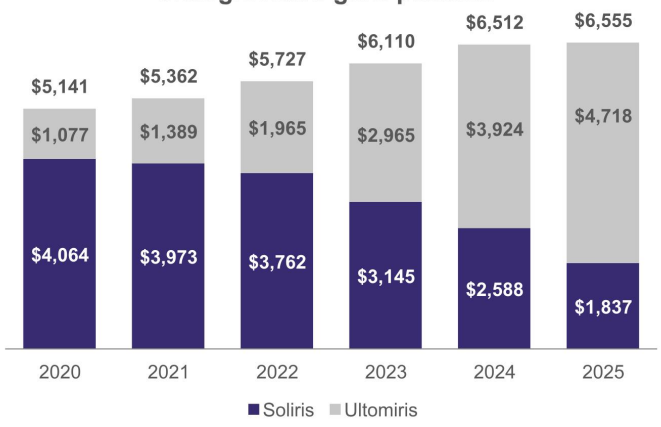
Figures represent U.S. estimated patients only. gMG: >100,000 gMG U.S. patients from Komodo claims data accessed 2013-2025; approx. 85% of gMG patients have AChR autoantibody-driven disease https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7033452/#
CIDP & MMN: Komodo claims data 2013-2025, adjusted to account 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-Disease, 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]



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

Ultomiris sales **grew 18%** in Q1'26**... "driven by** patient demand across indications, including the competitive **myasthenia gravis** and PNH **markets.**"

Q1 2026 financial results transcript

Ultomiris sales **grew 15%** in Q4'25**... "driven by patient demand across indications, including the competitive gMG** and PNH **markets.** In 2026, **we expect Ultomiris to continue to grow, driven primarily by neurology indication,** including **new-to-brand patients** and those switching from Soliris**,** as well as further market expansions. **We indicated peak year sales for Ultomiris to be above $5 billion**, with contribution from both existing and new indications..."

Q4 2025 financial results transcript

"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**."

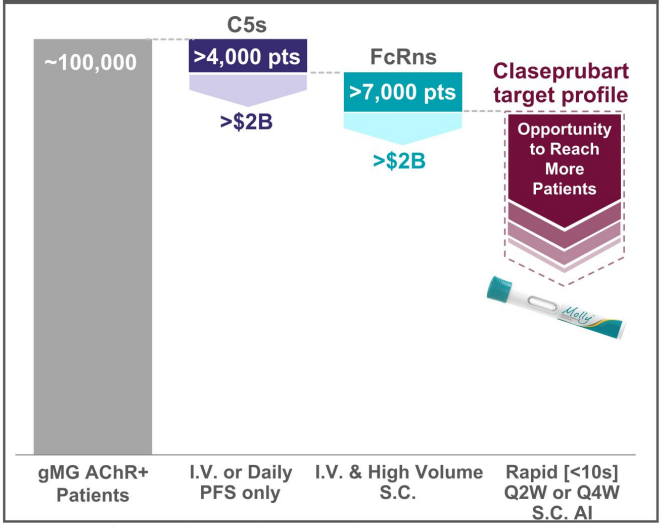
Q2 2025 financial results transcript

AstraZeneca

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

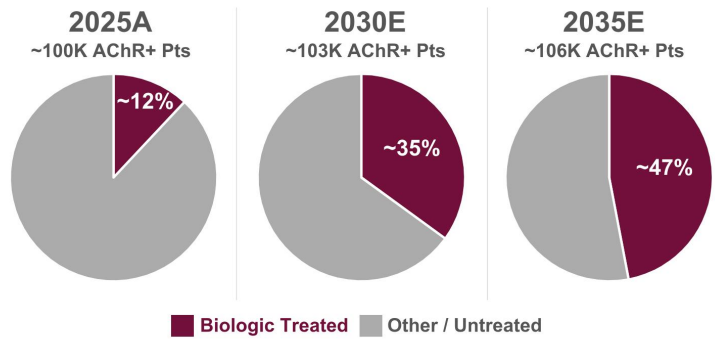
Significant opportunity for a highly differentiated, more patient-friendly biologic to expand use of biologics in gMG

Current US gMG Market



>$8B gMG Opportunity for Complement in 2035[2]

Estimated US biologic penetration[3,4]



Estimated US biologic sales[3,4]

~$4B	~$14B	~$20B
~12K pts	~36K pts	~50K pts

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 countsAChR+ 85% of gMG. 2. Based on Dianthus market research and estimates. 3. 2025 US financial reports on gMG drugs. Soliris/Ultomiris adjusted for relative size of MG based on claims. Vyvgart adusted for estimated CIDP sales.4. Based on EvaluatePharma (Jan '26) and Dianthus market research and estimates. Assumes average biologic net price per patient of ~$400,000 per year. AstraZeneca 2024 Investor Day estimated >36,000 AChR+ treated patients in future state. AstraZeneca Q2'25 earnings transcript expected 50% of gMG patients to be on branded treatments by 2028.

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-Disease Treatment for gMG

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

Efficacy Endpoints

Strong results support claseprubart potential as a best-in-disease 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 or DIL
- 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.
Drug-Induced Lupus (DIL) is an autoimmune syndrome triggered by specific medications, such as statins, TNF-alpha inhibitors, and ACE inhibitors/beta-blockers. A key distinguishing feature of DIL is its reversibility, as symptoms typically resolve once the offending medication is withdrawn.

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

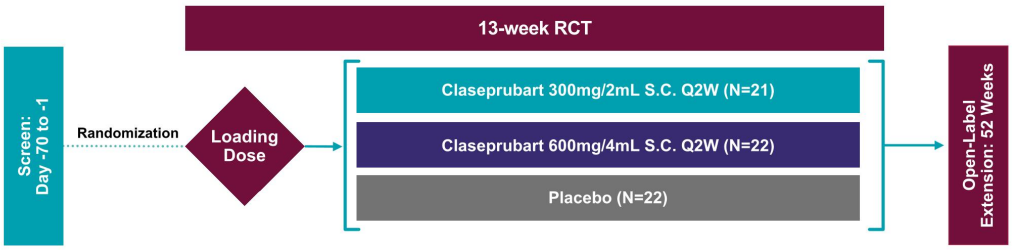
A global, multicenter, randomized, double-blind, placebo-controlled study to evaluate the safety, efficacy, and PK / PD of claseprubart administered S.C. following initial loading dose

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

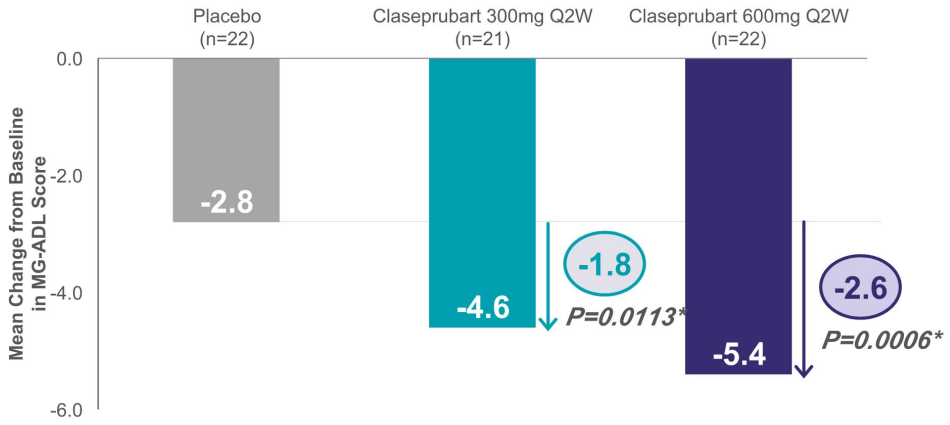
Participant baseline characteristics were generally well balanced across arms

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



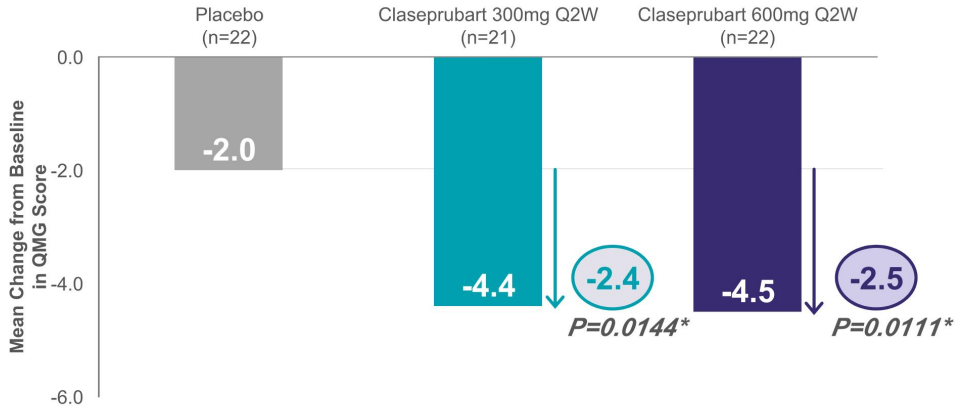
P-value	300mg/2mL	600mg/4mL
One-sided	P=0.0113*	P=0.0006*
Two-sided	P=0.0227**	P=0.0013**

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

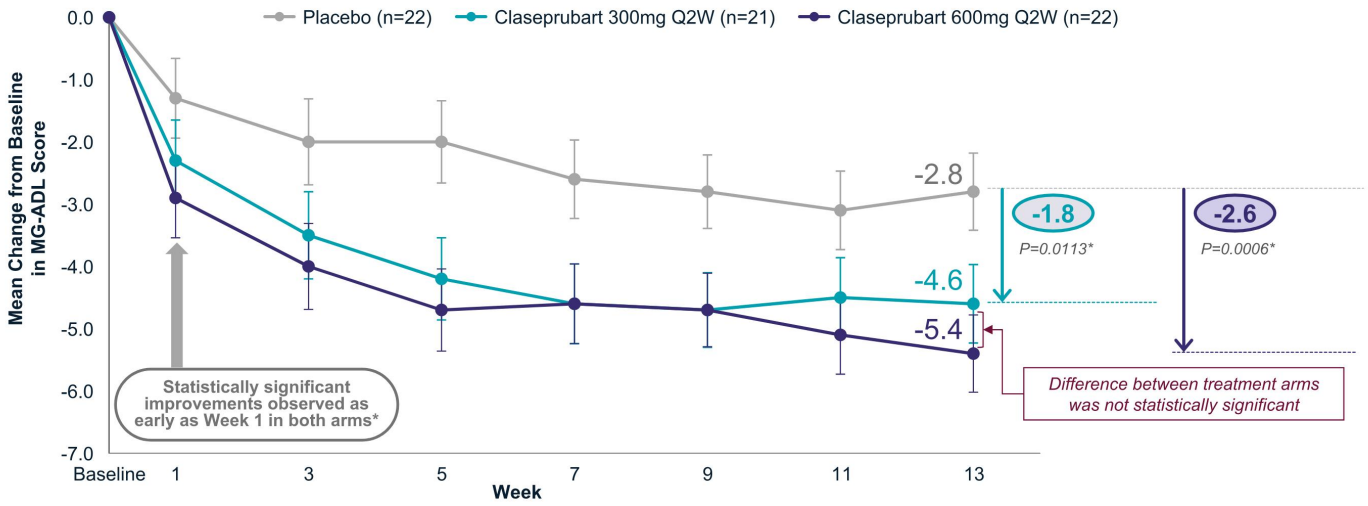


P-value	300mg/2mL	600mg/4mL
One-sided	P=0.0144*	P=0.0111*
Two-sided	P=0.0288**	P=0.0222**

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.

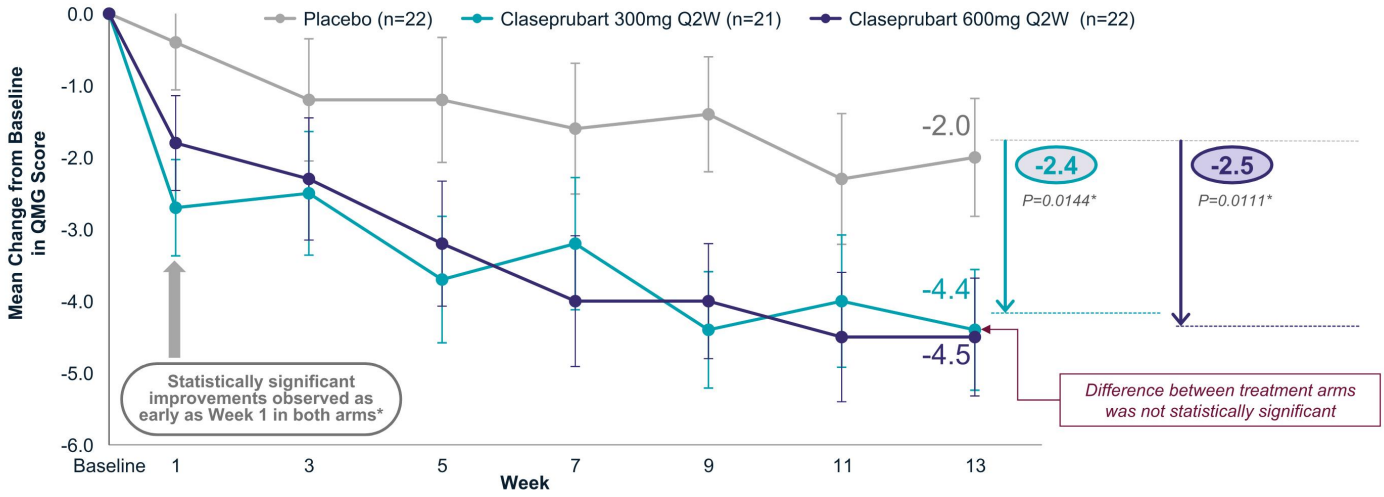
Claseprubart arms demonstrated rapid, sustained, and clinically meaningful improvements in MG-ADL score



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.

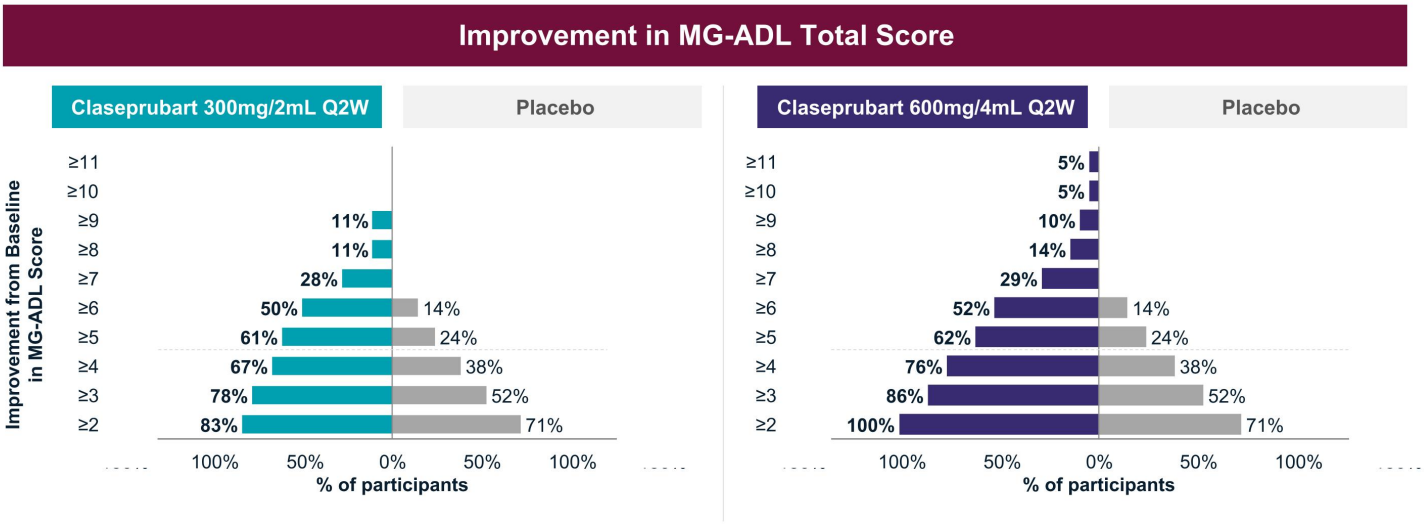
Claseprubart arms demonstrated rapid, sustained, and clinically meaningful improvements in QMG score



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.

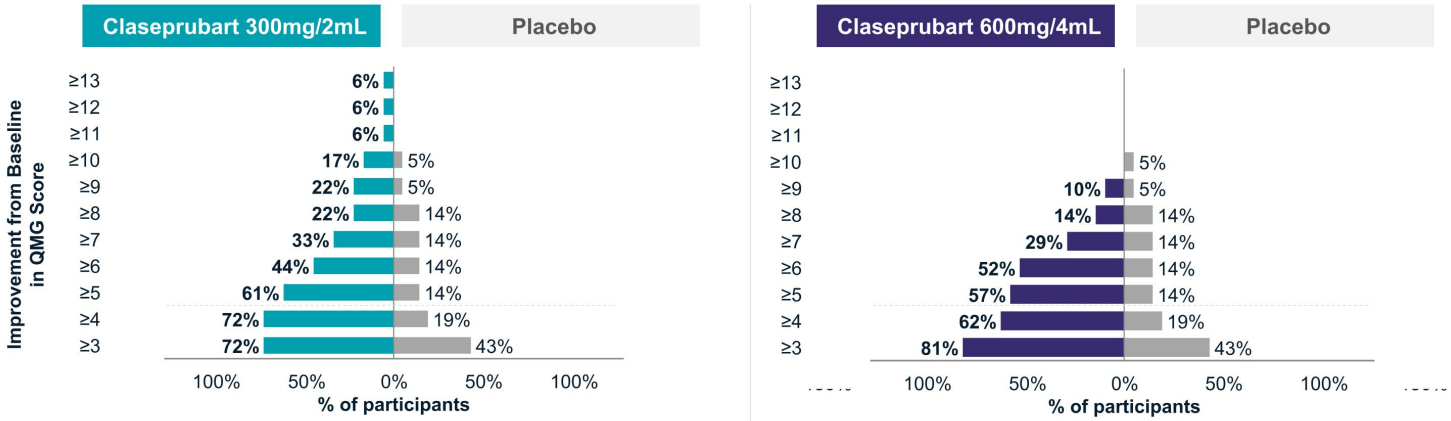
>60% of participants on claseprubart 300mg/2mL achieved ≥5 point improvement in MG-ADL



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

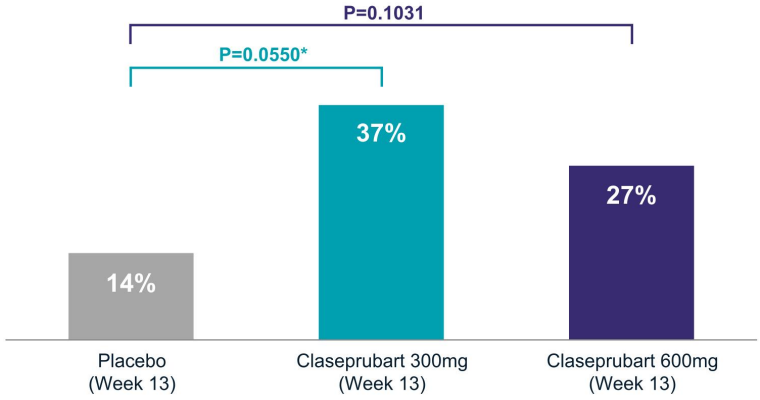
Improvement in QMG Total Score



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

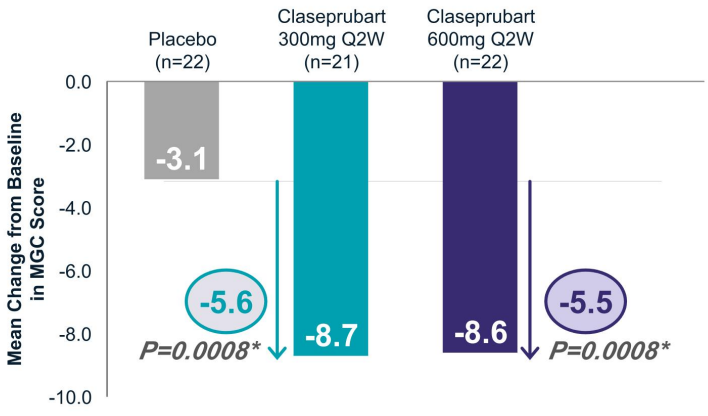


MSE supports potential best-in-class profile

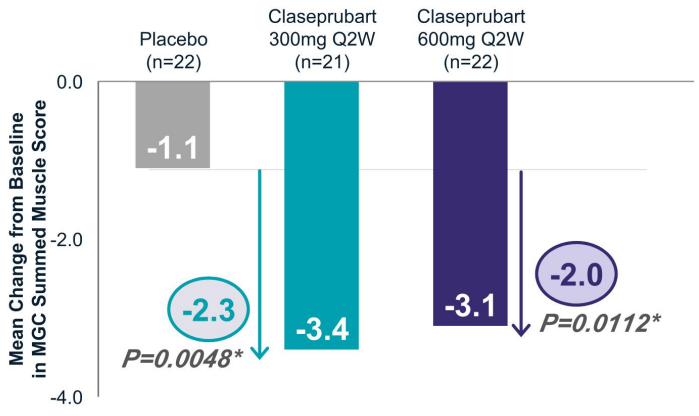
The proportion of participants who achieve MSE was analyzed using a logistic regression with terms for treatment group, stratification factors, and baseline MG-ADL included.
*One-sided p-values are presented for comparisons of claseprubart vs placebo, with any p-value below 0.1 considered nominally statistically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

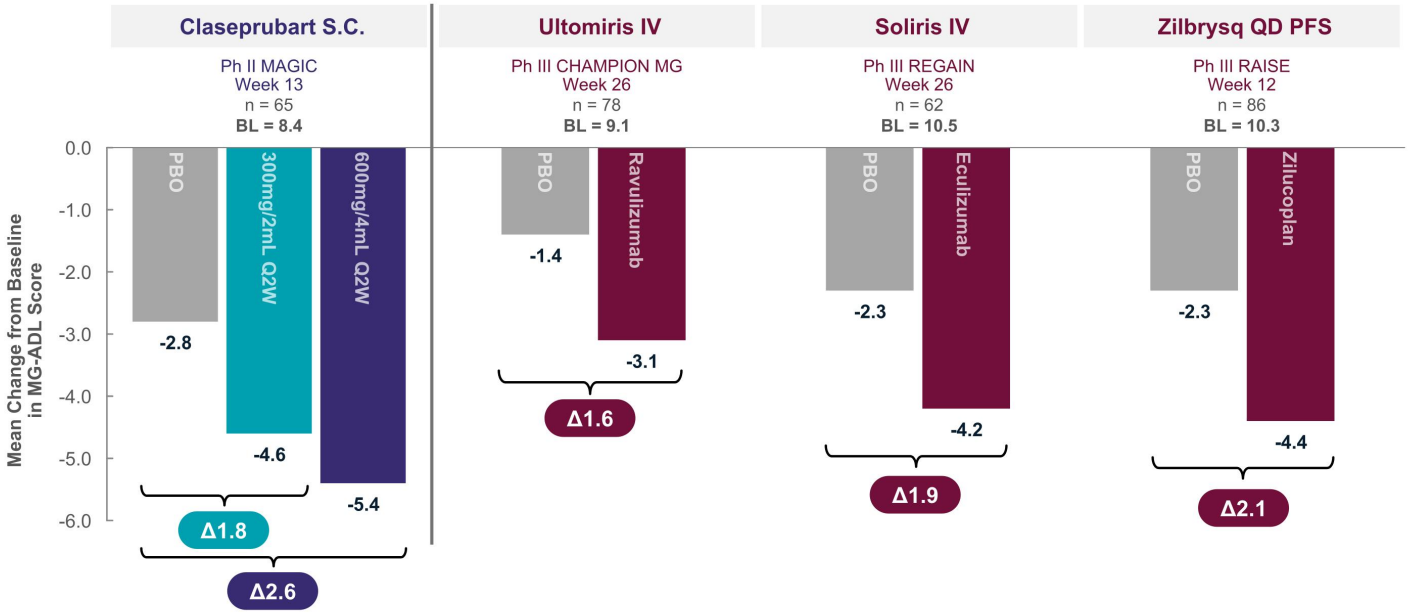
Across key efficacy measures, claseprubart demonstrated robust and clinically meaningful responses

	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.

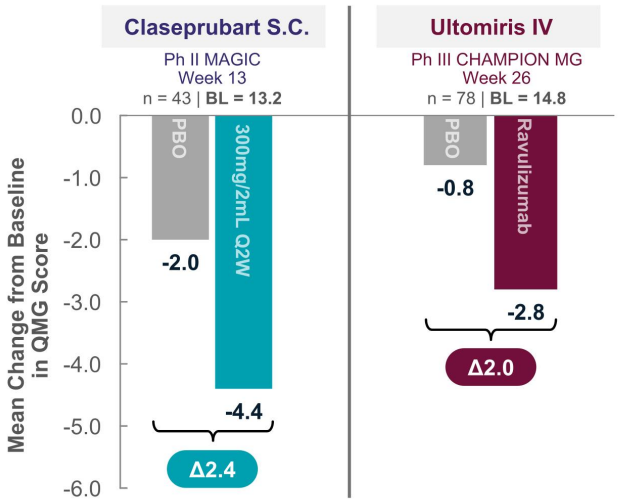
Claseprubart demonstrated statistically significant and clinically meaningful improvements in MG-ADL



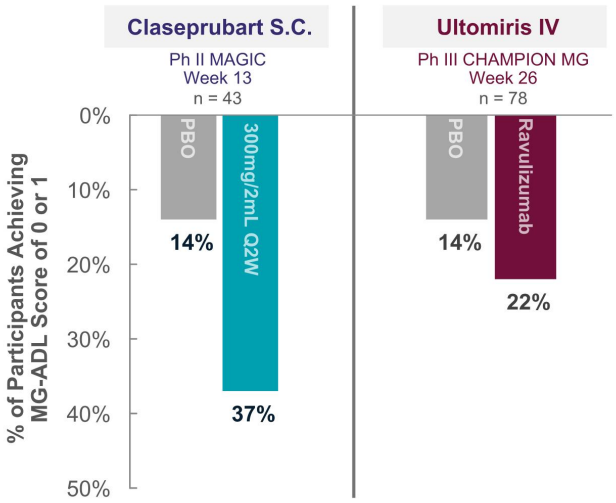
Note: For illustrative purposes only. Efficacy data are derived from different clinical trials conducted at different times, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted. Statistical 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 or DIL[4]

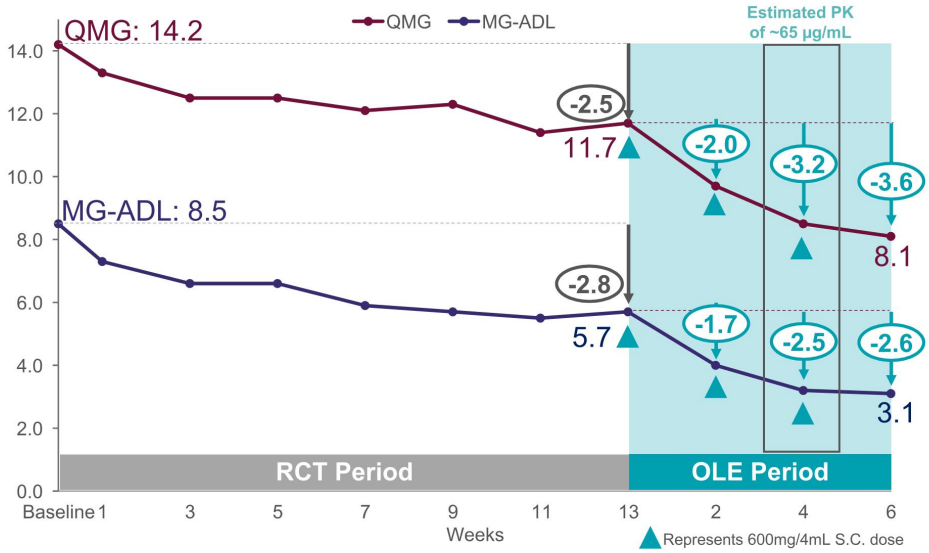
1. Excludes events in the investigations System Organ Class (MedDRA).
2. All injection site reactions were mild to moderate.
3. Represents participants who were ANA negative at baseline and tested positive at ≥1:320 at any point during RCT (percentages calculated from n=17 for 300mg arm and n=22 for 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.
4. Drug-Induced Lupus (DIL) is an autoimmune syndrome triggered by specific medications, such as statins, TNF-alpha inhibitors, and ACE inhibitors/beta-blockers. A key distinguishing feature of DIL is its reversibility, as symptoms typically resolve once the offending medication is withdrawn.

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



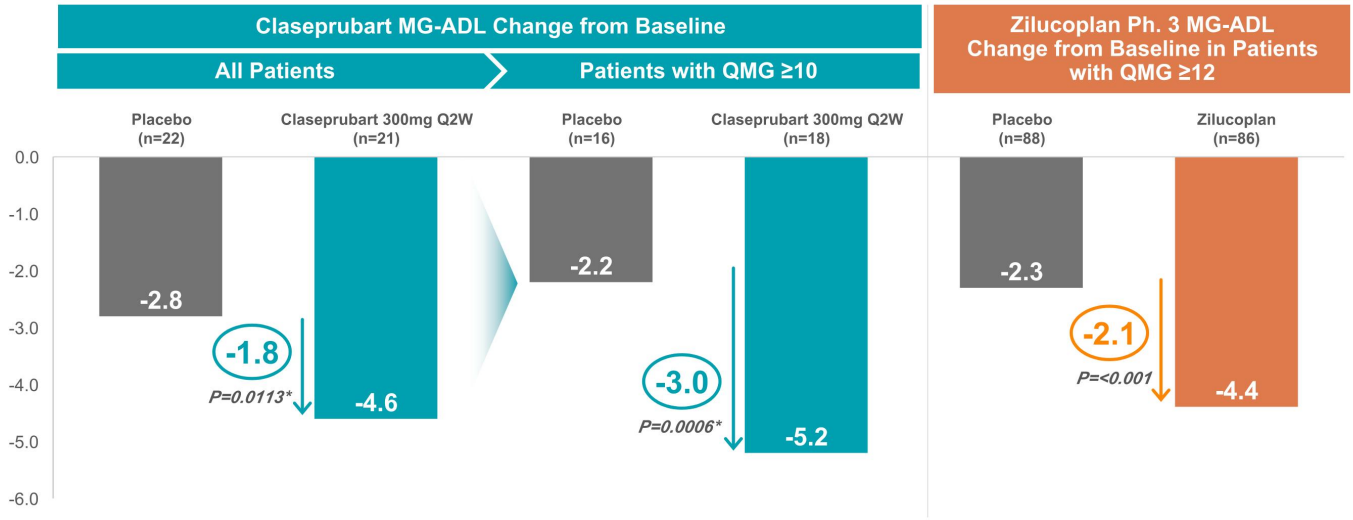
PBO Patients Entering OLE Received 600mg/4mL Q2W w/ No Loading Dose

- 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



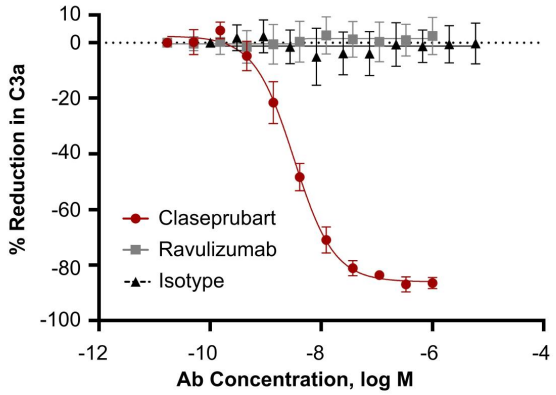
Ph. 2 study did not include QMG inclusion criteria, similar to ravulizumab Ph. 3; post-hoc analysis of MaGic data demonstrates potentially best-in-class MG-ADL improvement in patients with QMG ≥10

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)

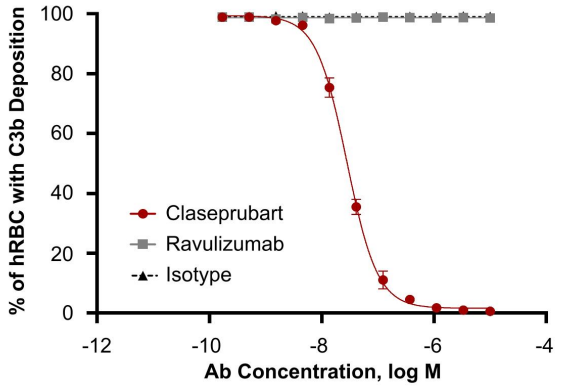
Potential for improved efficacy vs. C5 inhibitors with claseprubart may be due to upstream inhibition

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)

EMERGE is a global Ph. 3 trial evaluating Q4W and Q2W S.C. claseprubart in AChR+ gMG; top-line data 2H'28

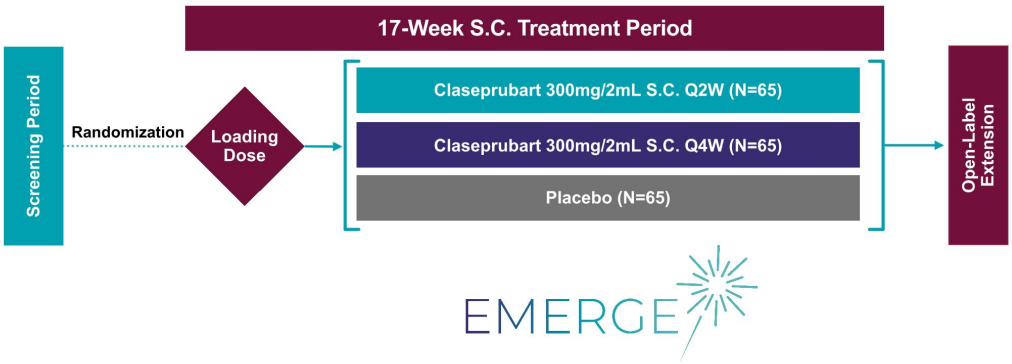
No ANA screening exclusion criteria or routine ANA testing during RCT or OLE, inclusion criteria of MG-ADL of ≥6 and QMG of ≥10, and 17-week S.C. treatment period per alignment with FDA

Highlights

- **Design:** Male and female subjects randomized to receive either claseprubart or placebo for 17 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
- **No ANA screening exclusion criteria or routine ANA testing** during the RCT or OLE

Endpoints

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



EMERGE

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

Study design pending regulatory approval

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 self-administered S.C. Q2W or Q4W

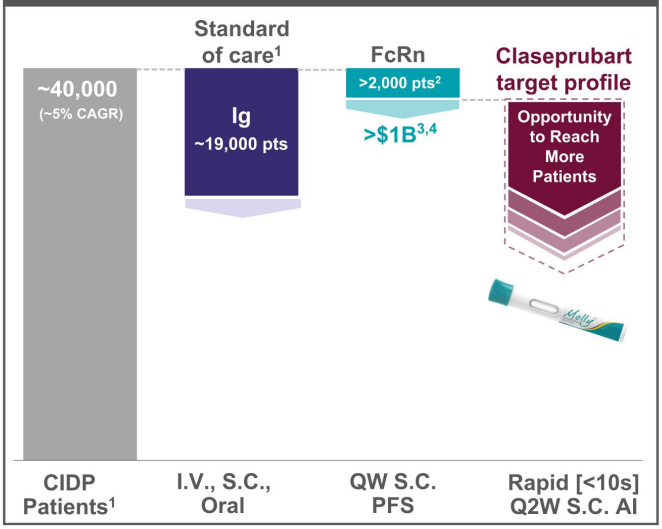
Claseprubart is an investigational agent that is not approved as a therapy in any indication in any jurisdiction worldwide. Safety and efficacy for claseprubart has not been evaluated in head-to-head comparative clinical studies.
Autoinjector for claseprubart administration is anticipated to be SHL Medical's 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 potential given high unmet need and limitations of current standard of care

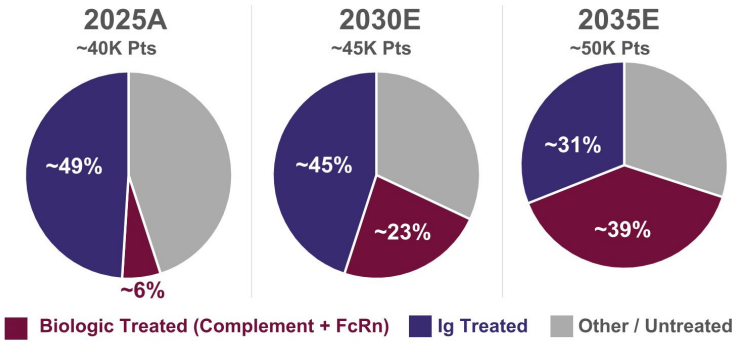
Opportunity for an active C1s inhibitor with the target profile of claseprubart to replace the standard of care

Current US CIDP Market



>$8B CIDP Opportunity for Complement in 2035[5]

Estimated US biologic and Ig penetration[6]



Estimated US biologic and Ig sales[6]

~$4B	~$10B	~$16B
~22K pts	~31K pts	~35K pts

Note: Positioning of claseprubart's target profile is illustrative. 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. Argenx Vyvgart Hytrulo HCP website. 3. Fierce Pharma, CIDP Pricing. 4. Argenx 4Q 2025 Financial Results, Feb 26, 2026. 5.Based on Dianthus market research and estimates. 6. Based on EvaluatePharma (Jan '26), Immunoglobulin – Global Market Analysis, Fortune Business Insights, and Dianthus market research and estimates. Assumes Ig price per patient of ~$150,000 per year and average biologic net price per patient of ~$700,000 per year.

Survey of US Neurologists supports potential transformative opportunity in CIDP

Claseprubart aims to differentiate and effectively address the significant unmet needs in the CIDP market

Total Neurologists 80

Neuromuscular Specialist	81%
Generalist	19%
Academic	58%
Community based	42%

Sample Demographics

- **~13** years in active clinical practice (post-residency), on average
- **~90%** of professional time spent providing direct patient care, on average
- **~60** CIDP patients seen in the past 12 months, on average

~79%

of Neurologists strongly believe patients prefer treatments with **more consistent and sustained symptom control**

~66%

of Neurologists strongly believe there is high unmet **need for therapies with greater efficacy**

~54%

of Neurologists strongly prefer treatment options **without a boxed warning or REMS program**

~75%

of Neurologists strongly believe patients prefer therapies that are **more convenient and easier to administer**

Dianthus 2026 Neurologist CIDP and MMN quantitative survey (n=80, N = 65 Neuromuscular specialists, N = 15 General Neurologists), fielded Q1
Data represents % of Neurologists selecting 7-9 on a 9-pt. agreement scale

Surveyed Neurologists want safer, more effective and convenient treatment options than IVIg for CIDP patients

Overall, surveyed Neurologists believe ~50% patients on IVIg have partial or no response to treatment

% of Surveyed Neurologists Who Strongly Believe…	
	Significant unmet **need for effective therapies** for patients who don't respond to IVIg
	Patients on IVIg commonly report **waning efficacy prior to their next dose**
	IVIg imposes a **high treatment burden** due to frequent and time-consuming infusions
	IVIg **boxed warning is concerning** due to potential risk of kidney failure or thrombosis

Dianthus 2026 Neurologist CIDP and MMN quantitative survey (n=80, N = 65 Neuromuscular specialists, N = 15 General Neurologists), fielded Q1
Data represents % of Neurologists selecting 7-9 on a 9-pt. agreement scale

Interim Responder Analysis in CAPTIVATE was planned with first 40 patients completing Open-Label Part A

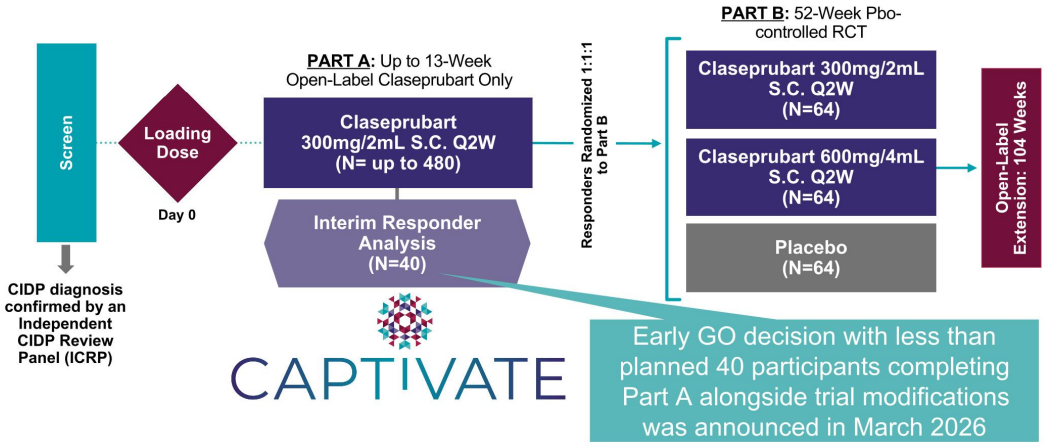
Highlights

- **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-Treated 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

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



- **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-Treated and SoC-Naïve patients
- **No requirement for IVIg withdrawal and disease worsening,** consistent with ongoing FcRn and complement CIDP studies
- **Only responders from Part A** randomized into the double-blind, placebo-controlled Part B

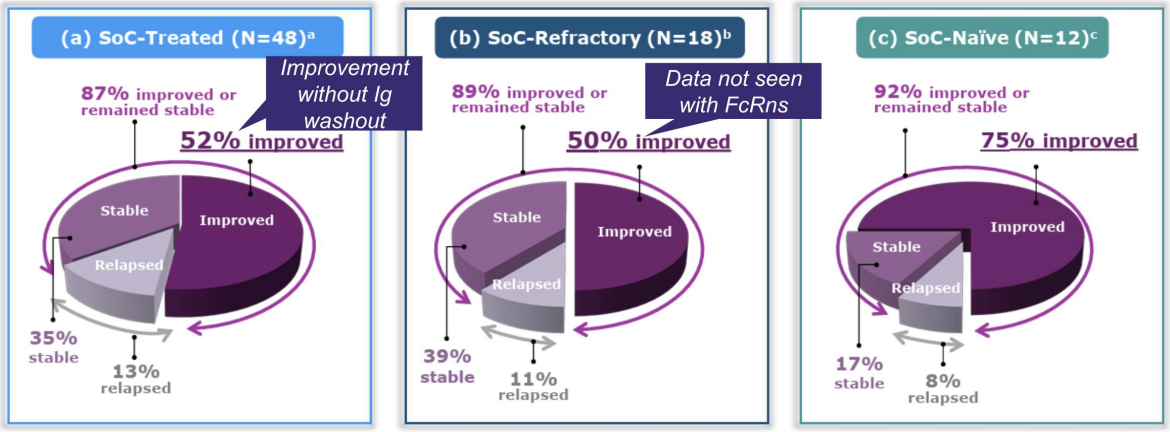
Significant differences between CAPTIVATE and ADHERE

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-Naïve SoC-Refractory	**Evaluating claseprubart in SoC-Refractory CIDP patients**, in addition to a broader CIDP patient population including SoC-Treated and SoC-Naïve
Require IVIg or SCIg Withdrawal and Relapse Prior to Enrolling in Part A of Study[1]	YES	NO	**Immediate switch 7 days from last Ig dose to claseprubart**; consistent with other ongoing 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-Ig washout baseline*	• Switching Ig patients to claseprubart 7 days after last dose • Aiming for ≥1-point adj. INCAT improvement OVER SoC/Ig in ≥50% of patients in Part A	Part A designed to evaluate **clinically meaningful improvement over Ig after immediate switch 7 days after last dose**

Source: Company filings, presentations and clinicaltrials.gov.
Ig refers to IVIg and SCIg
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.
1. ADHERE required discontinuation of IVIg or SCIg 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. Empasiprubart and riliprubart studies

Active C1s inhibition with riliprubart has demonstrated clinical proof-of-concept across broad patient groups

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



Claseprubart is being evaluated with a convenient, low volume dose of 300mg/2mL Q2W

1. Riliprubart Phase 2 at PNS 2024
2. Based on riliprubart patent filing (Pg 76)

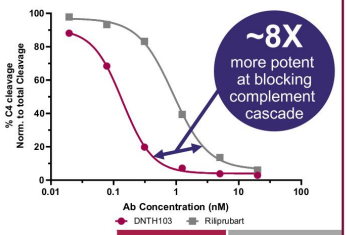
Claseprubart *in vitro* affinity and potency

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

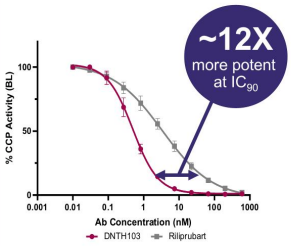
C4 cleavage by human active C1s[2]



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

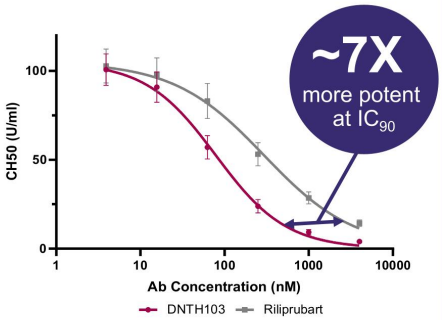
Functional assays of classical pathway inhibition

Wieslab classical pathway Assay in human serum[3]



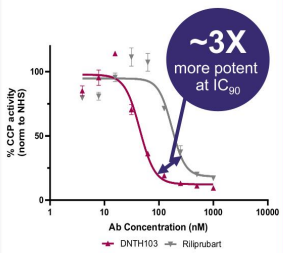
	DNTH103	riliprubart
IC_{90} (µg/mL)	**0.45**	5.4

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 has consistently higher affinity and stronger potency in multiple head-to-head *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.

Announced early GO decision reached with less than 40 planned participants completing Part A in March'26

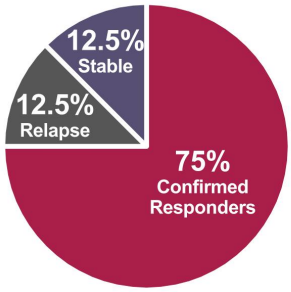
CAPTIVATE Interim Analysis Objective	Targeting response rate of 50% or greater (≥20 patients out of first 40 participants in Part A) based on precedent set with aC1s inhibition
GO Decision	GO decision reached early after 20 confirmed responders were achieved with less than 40 planned participants completing Part A
Safety / Tolerability Update	Independent DSMB reviewed the data to date and confirmed GO decision; no related serious infections, no clinical symptoms of autoimmune activation or DIL, no related SAEs or discontinuations

GO decision supports continued development of claseprubart at 300mg/2mL Q2W S.C. in CIDP targeting a potentially best-in-disease biologic profile

CAPTIVATE Interim Analysis data cut off as of March 4, 2026
Drug-Induced Lupus (DIL) is an autoimmune syndrome triggered by specific medications, such as statins, TNF-alpha inhibitors, and ACE inhibitors/beta-blockers. A key distinguishing feature of DIL is its reversibility, as symptoms typically resolve once the offending medication is withdrawn.

75% response rate observed in Interim Responder Analysis from first 40 participants completing Part A of CAPTIVATE

Breakdown of First 40 Participants Completing CAPTIVATE Part A



Safety / Tolerability Update

Generally well tolerated with no related serious infections, no clinical symptoms of DIL, no related SAEs or discontinuations due to safety

Summary of Change from Baseline at Last Evaluable Visit[1]

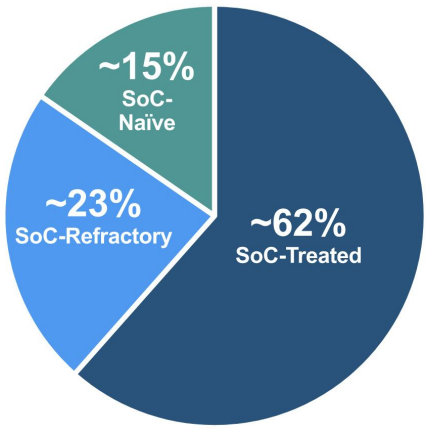
	Confirmed Responders (N=30)	Non-Responders (N=10)
INCAT *Decrease is improvement* *mean (SD)*	-1.6 (0.89)	0.7 (1.06)
Grip Strength *Increase is improvement* *mean (SD)*	15.9 (12.78)	-7.5 (12.19)
MRC-SS *Increase is improvement* *mean (SD)*	5.1 (3.93)	-2.3 (8.82)
I-RODS *Increase is improvement* *mean (SD)*	8.9 (12.08)	-3.0 (10.69)

Consistent and clinically meaningful results across multiple efficacy measures from first 30 confirmed responders in Part A

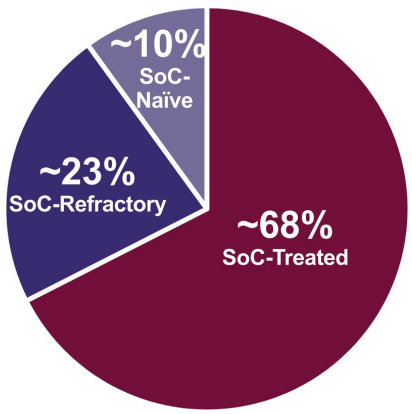
CAPTIVATE per protocol Interim Analysis data from first 40 participants to complete Part A. Non-Responders includes relapses and stable patients.
The summaries presented are preliminary. The study remains ongoing and the data are not final.
INCAT, Inflammatory Neuropathy Cause and Treatment; MRC-SS, Medical Research Council Sum Score; I-RODS, Inflammatory Rasch-built Overall Disability Scale.
1. Last Evaluable Visit: for confirmed responders signifies last visit prior to randomization in Part B; for stable patients signifies Week 13 visit (completion of Part A); for relapse patients signifies confirmed relapse visit prior to any rescue medication.

CAPTIVATE Part A baseline patient group characteristics are similar to precedent aC1s Ph. 2 study[1]

Riliprubart Ph. 2[1]
Baseline Patient Group Characteristics (N=78)



CAPTIVATE Part A[2]
Baseline Patient Group Characteristics (N=40)



Broad representation of patients across North America, Europe, and Asia in CAPTIVATE Part A

Data from CAPTIVATE and riliprubart Ph. 2 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.

1. Riliprubart Phase 2 at PNS 2024
2. Includes only first 40 participants who have completed Part A

Early responder rates support 300mg/2mL Q2W dose and updates to CAPTIVATE

Trial Design Element	Original Design	Anticipated New Design	Claseprubart CAPTIVATE Implications
Part A Dose	• 300mg/2mL S.C. Q2W	• 300mg/2mL S.C. Q2W	No change to Part A dose given results observed to date
Study Arms in Part B	1. 300mg/2mL Q2W (N=64) 2. 600mg/4mL Q2W (N=64) 3. Placebo (N=64) • *Total Part B Patients (N=192)*	1. 300mg/2mL Q2W (N=64) 2. Placebo (N=64) • *Total Part B Patients (N=128)*	~1/3 fewer total patients anticipated in Part B and potential faster execution to top-line results
Estimated Enrollment in Part A	Up to 480 patients, conservative 40% minimum responder rates	Up to 256 patients, conservative 50% minimum responder rates	Ratio from Part A to Part B changed due to responder rates seen across all patient groups

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

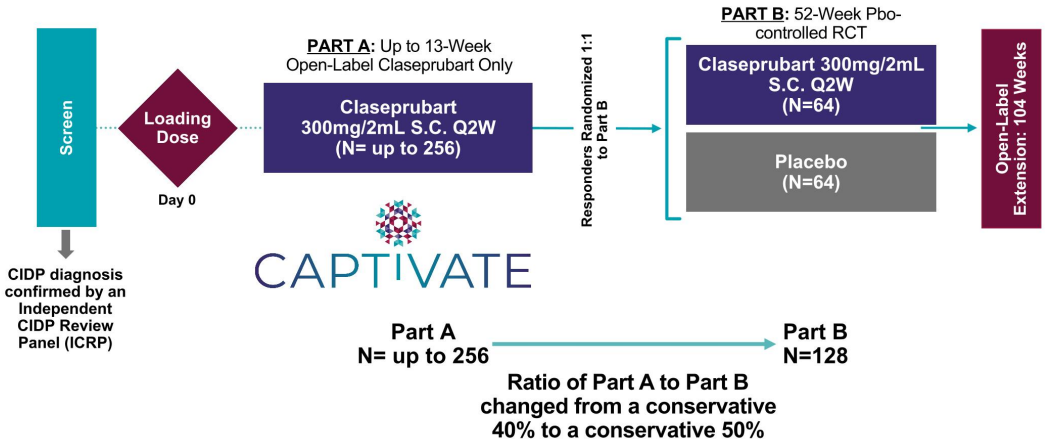
Revised CAPTIVATE study design going forward

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-Treated or SoC-Naïve
- **Dosing:** I.V. Loading Dose followed by 300mg/2mL S.C. Q2W in Part A; followed by 300mg/2mL or placebo in Part B
- **No ANA screening exclusion criteria or routine ANA testing** during the RCT or OLE

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



Part B top-line guidance expected by YE'26

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

Achieving this target profile could position claseprubart as a potential blockbuster treatment for CIDP

EFFICACY
(IVIg/SCIg)

Improvement over SoC (i.e. Ig) with continuous, effective symptom control

Targeting Best-in-Disease Efficacy

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)

Most *convenient* therapy with self-administered SHL-Molly autoinjector

Targeting single 300mg/2mL self-administered S.C. Q2W

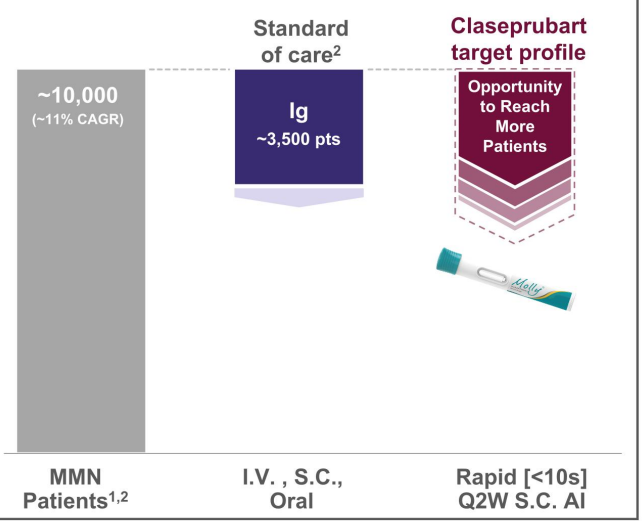
Claseprubart is an investigational agent that is not approved as a therapy in any indication in any jurisdiction worldwide. Safety and efficacy for claseprubart has not been evaluated in head-to-head comparative clinical studies.
Autoinjector for claseprubart administration is anticipated to be SHL Medical's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

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

The US MMN market is a growing blockbuster market opportunity for claseprubart

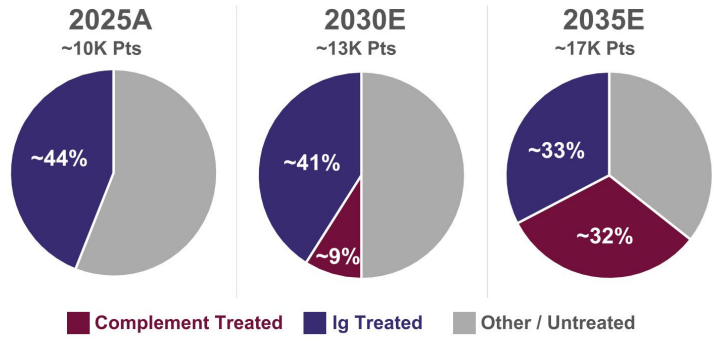
Opportunity for the target profile of claseprubart to become the new standard of care in MMN

Current US MMN Market



~$4B MMN Opportunity for Complement in 2035[3]

Estimated US complement and Ig penetration[4]



Estimated US complement and Ig sales[4]

<$1B	~$2B	~$5B
~4K pts	~7K pts	~11K pts

Note: Positioning of claseprubart's target profile is illustrative. 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. Dianthus market research and estimates. 4. Based on EvaluatePharma (Jan '26), Immunoglobulin – Global Market Analysis, Fortune Business Insights, and Dianthus market research and estimates. Assumes Ig price per patient of ~$150,000 per year and average biologic net price per patient of ~$700,000 per year.

Survey of US Neurologists supports MMN underdiagnosis and large unmet need

Claseprubart aims to offer a uniquely effective, safe, convenient classical pathway inhibitor

Total Neurologists 80

Neuromuscular Specialist	81%
Generalist	19%
Academic	58%
Community based	42%

Sample Demographics

~13 years in active clinical practice (post-residency), on average

~90% of professional time spent providing direct patient care, on average

~25 MMN patients seen in the past 12 months (median)

~81%
of Neurologists strongly believe that MMN is an **underdiagnosed condition**

~73%
of Neurologists strongly believe there is a high unmet need for **therapies with meaningful clinical improvement**

~68%
of Neurologists strongly believe that treatment options **without boxed warnings or REMS are preferred**

~63%
of Neurologists strongly believe there is a high unmet need **for therapies with a more favorable dosing schedule**

Dianthus 2026 Neurologist CIDP and MMN quantitative survey (n=80, N = 65 Neuromuscular specialists, N = 15 General Neurologists), fielded Q1
Data represents % of Neurologists selecting 7-9 on a 9-pt. agreement scale

Over half of surveyed US Neurologists agree IVIg is a suboptimal treatment option for MMN patients

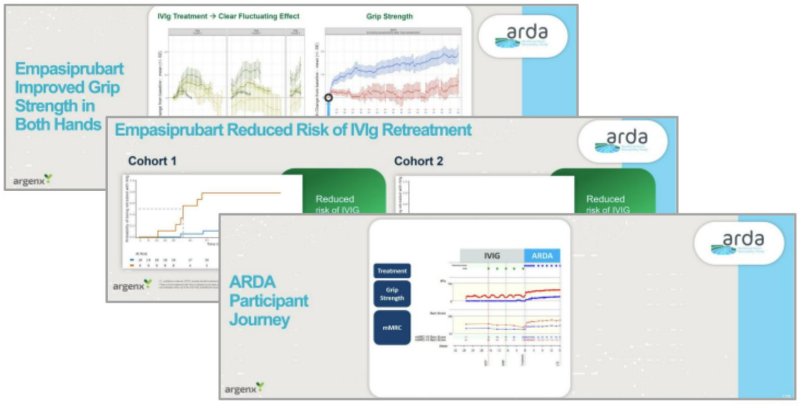
The lack of satisfaction with IVIg efficacy in MMN presents a significant opportunity for claseprubart

% of Surveyed Neurologists Who Strongly Believe…	
58%	Patients on IVIg often report **residual or unaddressed symptoms**
51%	**Clinical efficacy of IVIg** remains is **incomplete, inconsistent, and/or short-lived**
51%	Despite SoC, **1/3 of patients remain refractory** and face **risk of clinical deterioration**
55%	Current **label warnings/risks** for IVIg are **important in their treatment decisions**

Dianthus 2026 Neurologist CIDP and MMN quantitative survey (n=80, N = 65 Neuromuscular specialists, N = 15 General Neurologists), fielded Q1
Data represents % of Neurologists selecting 7-9 on a 9-pt. agreement scale

Classical Pathway inhibition has demonstrated PoC in MMN

Empasiprubart (an inhibitor of both the Classical and Lectin Pathways)
Ph. 2 Data Demonstrating Efficacy Signals[1]



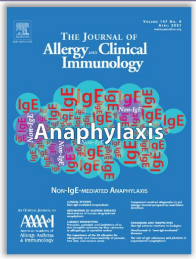
"We hypothesize that targeting the **classical complement pathway is a potential therapeutic approach in MMN**. We investigated the interaction of circulating anti-GM1 IgM from patients with MMN with complement in detail using iPSC-derived MNs. In this disease model for MMN, we evaluated the effects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

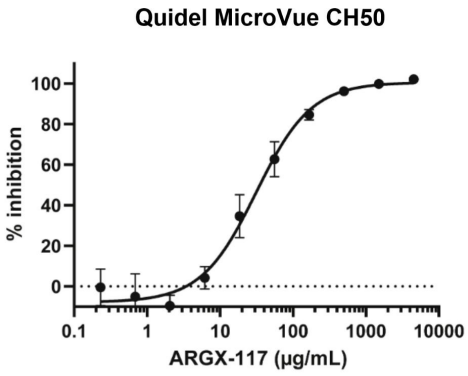
Phase 2 MoMeNtum trial of claseprubart, a potent & selective Classical Pathway inhibitor, is ongoing in MMN

1. https://argenx.com/content/dam/argenx-corp/events-presentations/argenx_RnD_Day_2024_Slides.pdf

Claseprubart demonstrates superior classical pathway potency vs. empasiprubart

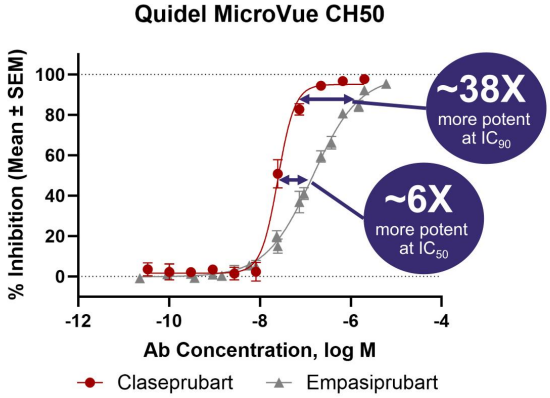
Empasiprubart Published Classical Pathway Potency Data Using the Quidel MicroVue CH50[1]





"**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
IC_{90} (µg/mL)	**9.9 ± 2.5**	375 ± 266

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

Phase 2 MoMeNtum top-line data in MMN anticipated Q4’26

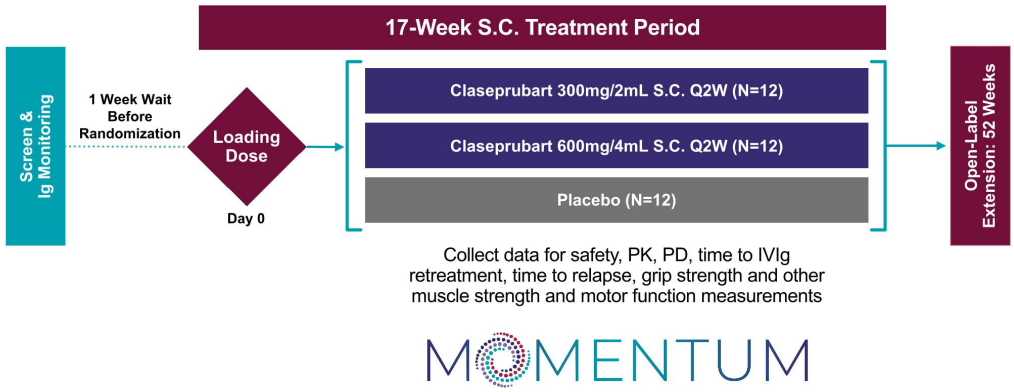
A global, multicenter, randomized, double-blind, placebo-controlled study to evaluate the safety, efficacy, and PK / PD of claseprubart administered S.C. following initial loading dose

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
- **No ANA screening exclusion criteria or routine ANA testing** during the RCT or OLE

Endpoints

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



Top-line data expected in Q4’26

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

Key differences between ARDA and MoMeNtum trials make cross-trial comparisons challenging

Considerations	Empasiprubart (C2) ARDA Trial	Claseprubart (aC1s) MoMeNtum Trial	Key Differentiators of MoMeNtum
Study Size & Treatment Duration	Placebo N = 18 10mg/kg Q2W IV, N = 18 5mg/kg Q4W IV, N = 18 Length of Trial = 16 weeks	**Placebo N = 12** **300mg/2mL S.C., N = 12** **600mg/4mL S.C., N = 12** **Length of Trial = 17 weeks**	**Small study designed to demonstrate PoC** and support initiation of Ph. 3 as quickly as possible. **Aims to show consistent trends** in key efficacy measures. **Not powered for statistical significance** on any efficacy endpoints
Allowed IVIG Rescue Treatment Upon Patient Request	YES	**NO**	**MoMeNtum trial only allows IVIG rescue if** participant experienced **clinical deterioration** defined by MRC10 ≥2 points or GS decrease >30%

Note: Empasiprubart maintenance dosing from clinicaltrials.gov
Source: Company filings, presentations and clinicaltrials.gov.

Claseprubart has the potential to address the unmet need in the MMN market with its unique 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 best-in-disease biologic treatment given its unique combination of classical pathway potency, preservation of the lectin pathway, convenience and potential for no boxed warning

* 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.

Achieving this target profile could position claseprubart as a potential blockbuster treatment for MMN

EFFICACY
(IVIg/SCIg)

Improvement over SoC (i.e., Ig) with continuous, effective symptom control

Targeting Best-in-Disease Efficacy

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)

Most *convenient* therapy with self-administered SHL-Molly autoinjector

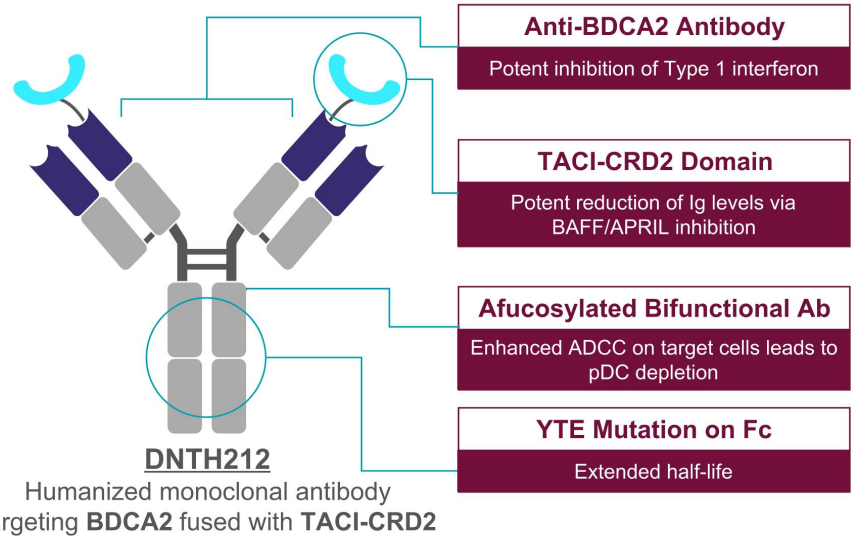
Targeting single 300mg/2mL self-administered S.C. Q2W

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

DNTH212:
Potential Best-in-Disease 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**

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

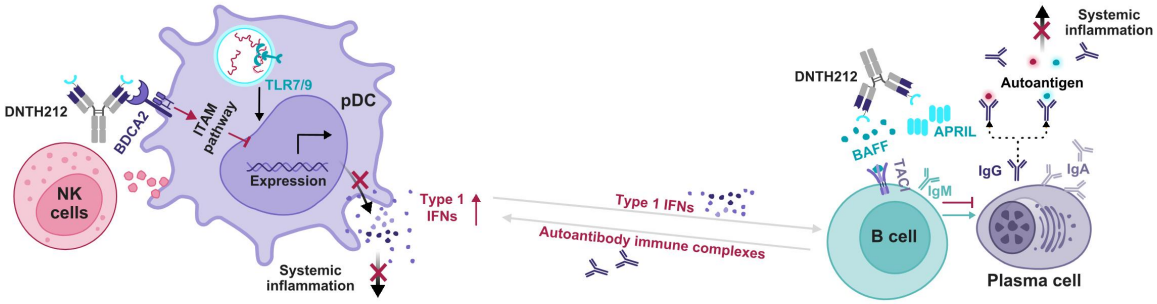
Potential to drive superior clinical efficacy by targeting both the innate and adaptive immune systems

Innate Immune System: Plasmacytoid Dendritic Cells (pDCs)

- Key cell type producing Type 1 interferon
- 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

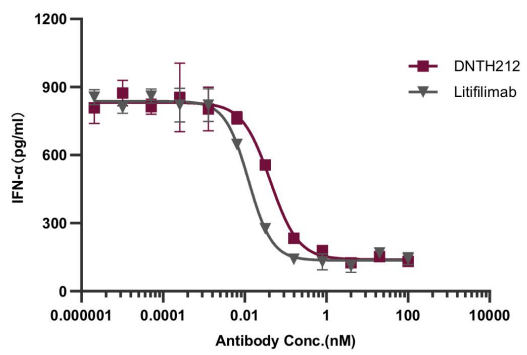


Bifunctional approach addressing autoimmune diseases where both Type 1 interferon and B Cells are implicated has strong mechanistic rationale for potential best-in-class efficacy

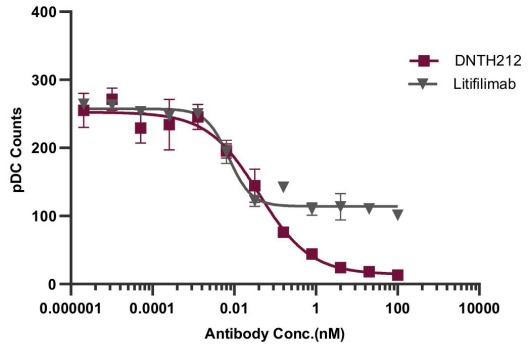
Panda SK, et al., Plasmacytoid dendritic cells in autoimmunity. Curr Opin Immunol. 2017 Feb;44:20-25.
Minaga K, et al., Plasmacytoid Dendritic Cells as a New Therapeutic Target for Autoimmune. Front Immunol. 2021 Jul 23;12:713779

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

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



Deeper Depletion of pDC[2]

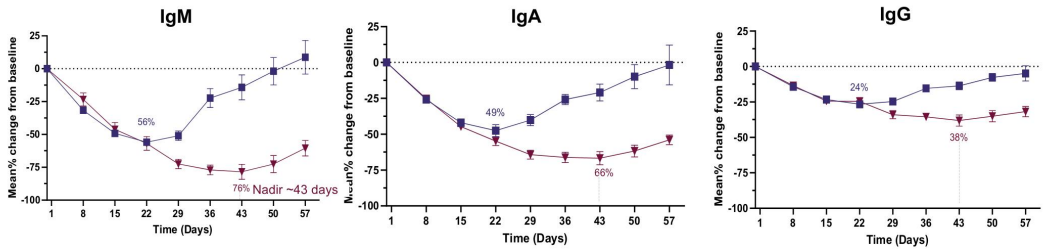


pDC depletion removes a key cell type involved in Type 1 interferon production and activation of other immune cells which contribute to disease

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

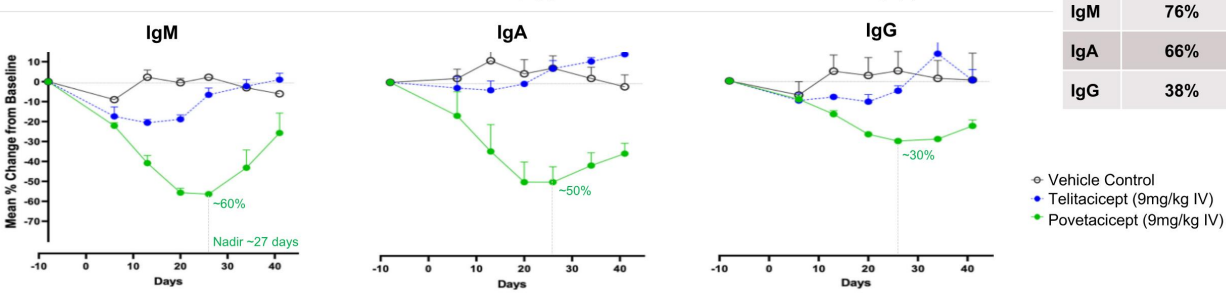
DNTH212 shows superior inhibition of IgM, IgA, and IgG compared to povetacicept following single dose in NHPs

S.C. DNTH212



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

IV povetacicept and telitacicept[1]



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

Priority indications with high unmet need and dual involvement of both innate and adaptive immune systems

Prioritization Considerations

- Clinical evidence or biological rationale of both interferon / pDC and B-cell involvement
- Potential for improved efficacy from dual-mechanism approach
- High unmet need with current SoC and future treatments
- Well-established development pathways

DNTH212 Priority Rheumatology Indications

Sjögren's disease (SjD)
~350,000 U.S. patients

Systemic lupus erythematosus (SLE)
~225,000 U.S. patients

Dermatomyositis (DM)
~50,000 U.S. patients

Targeting both innate and adaptive immune systems simultaneously may offer potential for meaningful differentiation

Building a beachhead in rheumatology with clear clinical development and commercial synergies across priority indications

Estimated U.S. patients per Dianthus meta-analysis and estimates

SLE: Strong BDCA2 and BAFF / APRIL validation with a proven development path support DNTH212 opportunity

Disease Overview

SLE is a systemic autoimmune disease affecting multiple organs such as the kidneys, skin, and joints and may lead to significant or life-threatening end-organ damage

Supportive Evidence

BDCA2/Type 1 IFN (Innate)

- Saphnelo approved
- Litifilimab Ph. 2 data

BAFF/APRIL (Adaptive)

- Benlysta approved
- Telitacicept Ph. 3 data (CN) (BAFF/APRIL)

Mechanism Strength

Proven skin and joint efficacy

Multi-organ responses (i.e., joints and kidney)

DNTH212 Target Opportunity

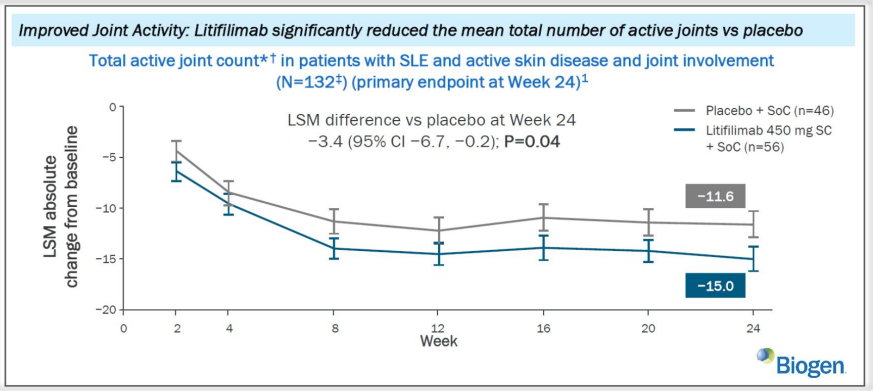
Potential to more effectively address multiple disease manifestations with lower corticosteroid burden

~225K U.S. Patients

Source: Company filings, presentations and clinicaltrials.gov
Estimated U.S. patients per Dianthus meta-analysis and estimates
ClearView analysis

SLE: Potential for dual mechanism to address key unmet needs despite approved treatments

Litfilimab Ph. 2 data[1] and multiple approvals (Saphnelo, Benlysta) support dual-mechanism approach in SLE



Benlysta (belimumab) Saphnelo (anifrolumab-fnia) **Utilized concurrently with steroids**

Significant Unmet Need

- SLE is a heterogenous disease - potential for dual-mechanism approach to impact multiple organs vs. single mechanism alone
- ~45% of SLE patients categorized as moderate-to-severe[2]

DNTH212 Target Opportunity

Potential to more effectively address multiple disease manifestations with lower corticosteroid burden

1. Biogen Lupus Virtual Investor Seminar (2025)
2. Lupus Sci Med. 2025 Dec 23;12(2):e001766

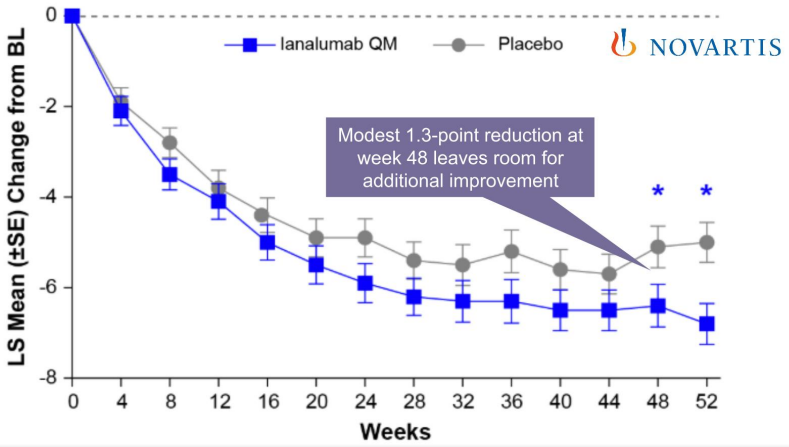
SjD: High unmet need for a single therapy that can address multiple disease manifestations

Disease Overview	Sjögren's destroys mucosal exocrine glands causing extreme dry eyes and mouth (sicca) and leads to debilitating systemic symptoms such as fatigue and joint pain	
Supportive Evidence	**BDCA2/Type 1 IFN (Innate)** ✓ Evidence for glandular pDC infiltration	**BAFF/APRIL (Adaptive)** ✓ Telitacicept Ph. 3 data (CN) (BAFF/APRIL) ✓ Ianalumab Ph. 3 data (BAFF)
Mechanism Strength	Potential for improved patient reported outcomes, including systemic disease manifestations	Proven impact on primary endpoint (i.e., ESSDAI)
DNTH212 Target Opportunity	**More effectively address multiple disease manifestations including dry eye, dry mouth and other systemic symptoms (i.e. pain, fatigue)**	**~350K U.S. Patients**

Source: Company filings, presentations and clinicaltrials.gov
Estimated U.S. patients per Dianthus meta-analysis and estimates
ClearView analysis

SjD: Potential for increased efficacy from dual-mechanism approach vs. targeting BAFF receptor only

Ianalumab Ph. 3[1] highlights potential for B-cell approaches to lower ESSDAI



"Additional secondary endpoints showed a trend towards improvement in both studies" – Ianalumab ACR 2025[1]

Significant Unmet Need

- Fatigue, pain and cognitive dysfunction are significant drivers of patient disability[2]
- 70% increased mortality compared with the general population due to infections, cancer and cardiovascular diseases[2]

DNTH212 Target Opportunity

More effectively address multiple disease manifestations including dry eye, dry mouth and other systemic symptoms (i.e., pain, fatigue)

1. Ianalumab ACR 2025 abstract
2. Pagnoni et al. J Rheum 2026, 53(2)

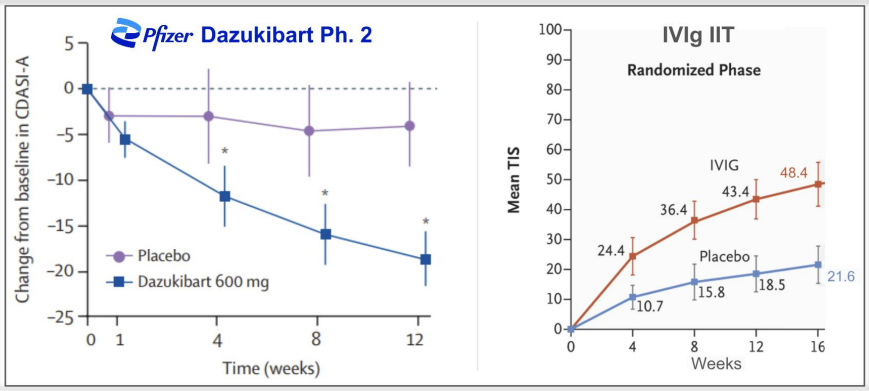
DM: High unmet need for a single therapy that can more effectively address the disease

Disease Overview	DM causes progressive muscle weakness, painful rash, pruritus and interstitial lung disease; limited therapeutic options beyond corticosteroids (i.e. IVIg)	
Supportive Evidence	**BDCA2/Type 1 IFN (Innate)** ✓ Dazukibart (IFN-β) Ph. 2 data ✓ Positive Saphnelo case reports	**BAFF/APRIL (Adaptive)** ✓ IVIg approved, Vyvgart Ph. 2 ✓ Benlysta small studies / case reports
Mechanism Strength	Improvement in skin symptoms and TIS (Total Improvement Score)	Clear evidence of antibody driven pathology and improvement in TIS
DNTH212 Target Opportunity	**More effectively address the disease and prevent damage over its course with lower corticosteroid burden**	**~50K U.S. Patients**

Source: Company filings, presentations and clinicaltrials.gov
Estimated U.S. patients per Dianthus meta-analysis and estimates
ClearView analysis
Dazukibart binds and neutralizes IFN-β. Saphnelo is a Type 1 receptor antagonist

DM: Dual-mechanism approach may more effectively address the disease

Dazukibart Ph. 2 data[1] and IVIg[2] lays foundation for multi-organ efficacy in DM



"This study showed that IFNβ is an important mediator of disease activity in skin-predominant dermatomyositis" - Lancet 2025; 405; 137–46

Significant Unmet Need

- Muscle weakness, fatigue and pain remain leading causes of morbidity[3]
- Limited therapeutic treatment options
- Majority of patients are managed with systemic steroids, which can carry long-term safety concerns[4]

DNTH212 Target Opportunity

More effectively address the disease and prevent damage over its course with lower corticosteroid burden

1. Dazukibart Phase 2 publication
2. Trial of Intravenous Immune Globulin in Dermatomyositis
3. Christopher-Stine; BMC Rheumatology 2025
4. George et al. RMD Open 2026

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

Healthy Volunteers (Part A)

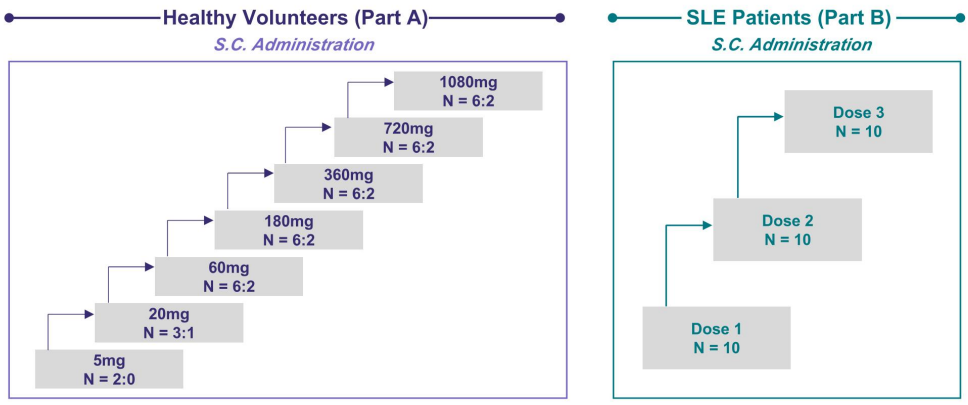
~46 HVs enrolled into seven cohorts:
- Treated (N= up to 6)
- Placebo (N= up to 2)

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

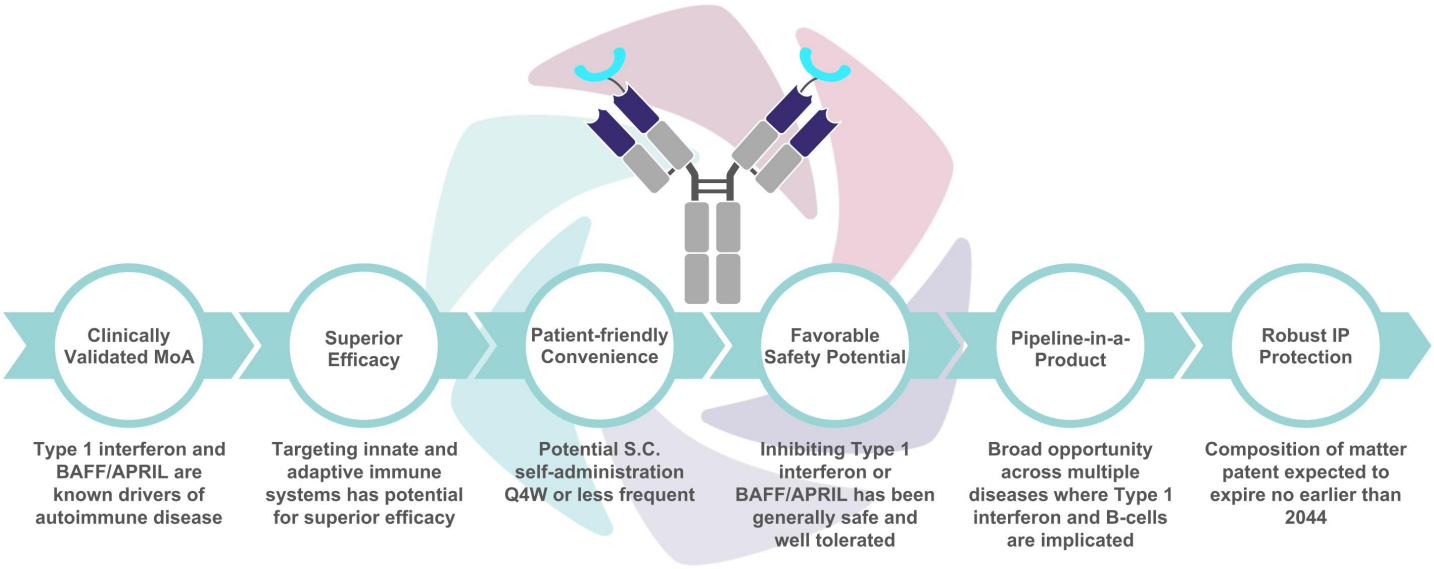
Inhibiting Type 1 interferon or BAFF/APRIL has been generally safe and well tolerated

CONVENIENCE

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

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

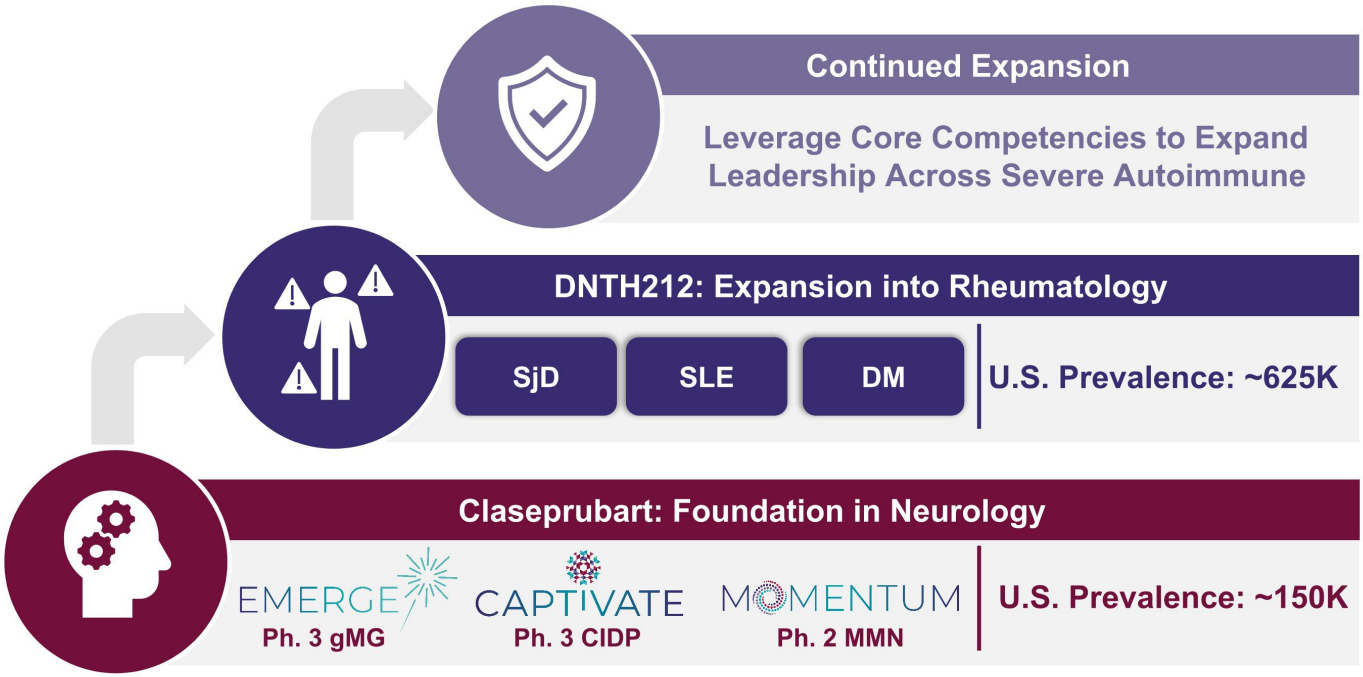
Achieving DNTH212 TPP could position DNTH212 as a first-line, best-in-disease therapy across multiple indications



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

Establishing our vision as the leading autoimmune focused biotech through potential best-in-disease innovation



Estimated U.S. patients per Dianthus meta-analysis and estimates

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				• Initiation of Ph. 3 study expected in mid-26 • Ph. 3 top-line data expected in 2H'28
	CIDP >40,000 U.S. Patients				• Part B top-line guidance expected by YE'26 • *Peer Milestone: riliprubart Ph. 3 VITALIZE (H2H vs. IVIg) data expected in 2027*[3]
	MMN >10,000 U.S. Patients				• Ph. 2 top-line data expected in Q4'26 • *Peer Milestone: empasiprubart Ph. 3 data expected in Q4'26*[4]
DNTH212 *BDCA2 and BAFF/APRIL*	SjD, SLE, DM *~625,000 U.S. Patients (Combined)*	*Healthy volunteers (Part A)* *SLE patients (Part B)*			• Ph. 1 HV top-line data expected in 2H'26

Strong balance sheet with ~$1.2B[1] of cash & runway expected into 2030
~56.0M shares outstanding[2]

* FDA Orphan Drug Designation received in May 2026 for claseprubart in Myasthenia Gravis
1. Cash includes cash, cash equivalents and investments as of 3/31/26
2. Pro forma shares includes 54.7M shares outstanding as of May 1, 2026 and assumes the exercise of all outstanding pre-funded warrants
3. Based on Sanofi Q1'26 financial results presentation
4. Based on publicly available information: https://argenx.com/news/2026/press-release-3289577

Appendix

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

1

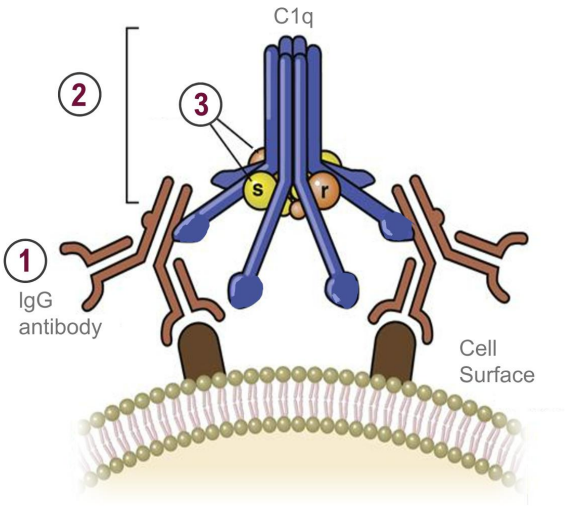
Classical pathway
The only pathway activated by the presence of IgG and IgM, which bind to the **C1 complex**

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



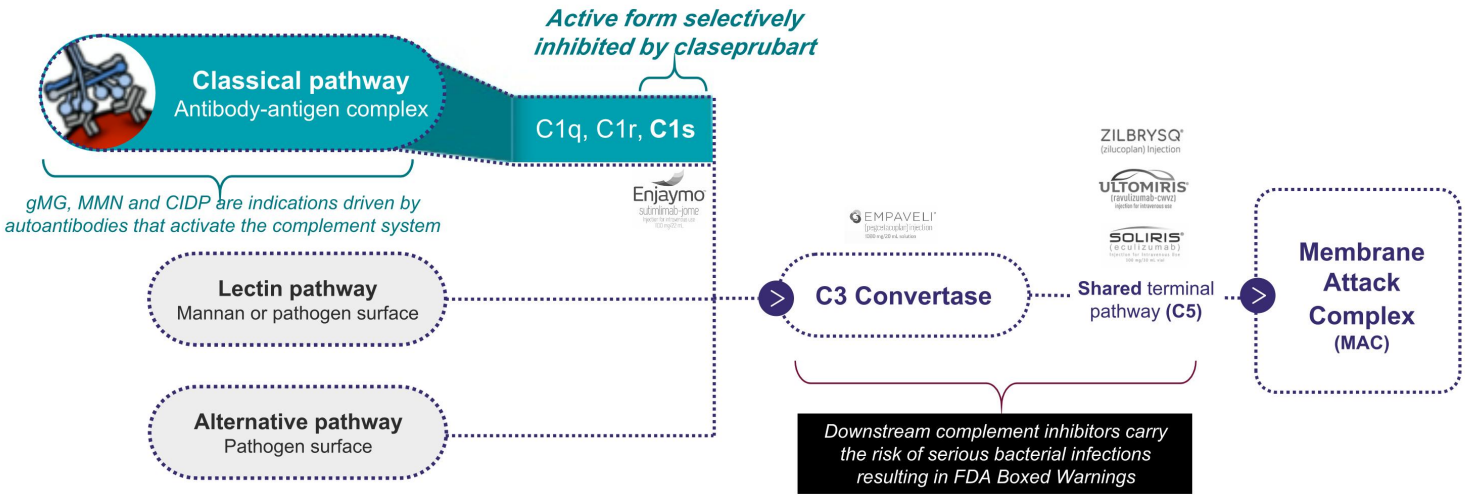
C1s is the only target of the C1 complex with an FDA approved therapy
Enjaymo, FDA approved in 2022 for CAD, is a C1s inhibitor but is not selective to the active form and dosed I.V. at 6,500-7,500mg every two weeks

Active C1s inhibition has demonstrated clinical benefit in MG and CIDP
Claseprubart MaGic and CAPTIVATE results provide clinical PoC for aC1s in 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

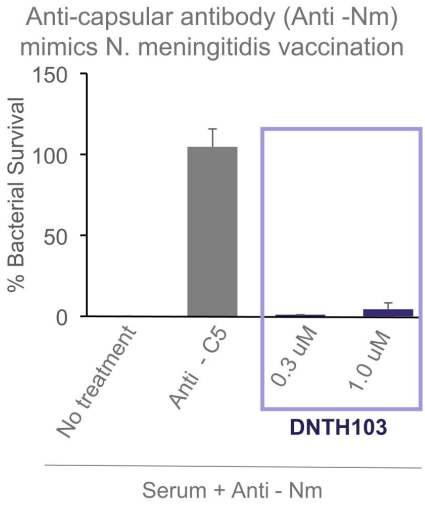
✓ No Boxed Warning or REMS

✓ 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



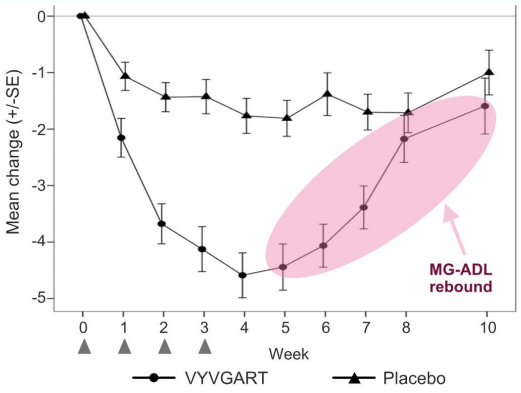
Results further validate the potential differentiated safety profile for DNTH103 as a selective classical pathway inhibitor consistent with ENJAYMO, an approved C1s inhibitor without an FDA Boxed Warning or REMS

*Engineered using patent sequence

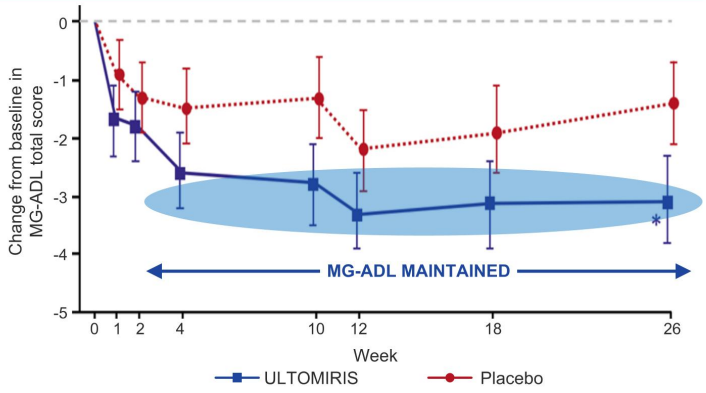
Claseprubart aims to provide consistent symptom control with convenient Q2W S.C. dosing

- 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

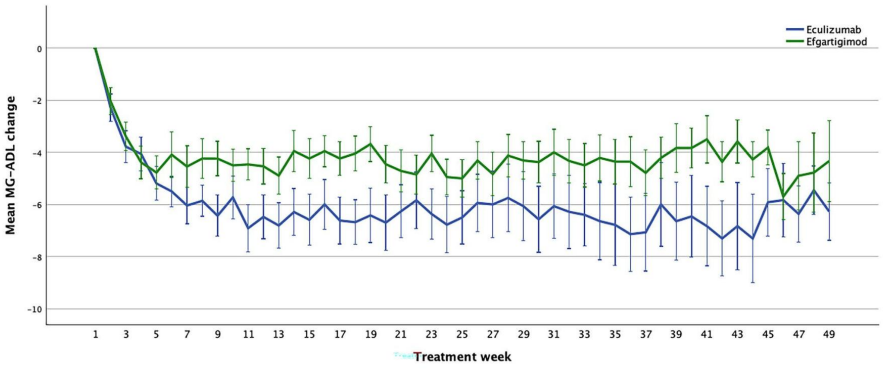


▲ Indicates VYVGART® dose administered
Source: VYVGART® prescribing information and ULTOMIRIS® prescribing information
1. Pane et al. A real-life experience with eculizumab and efgartigimod in generalized myasthenia gravis patients. *J Neurol* 271, 6209–6219 (2024)

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

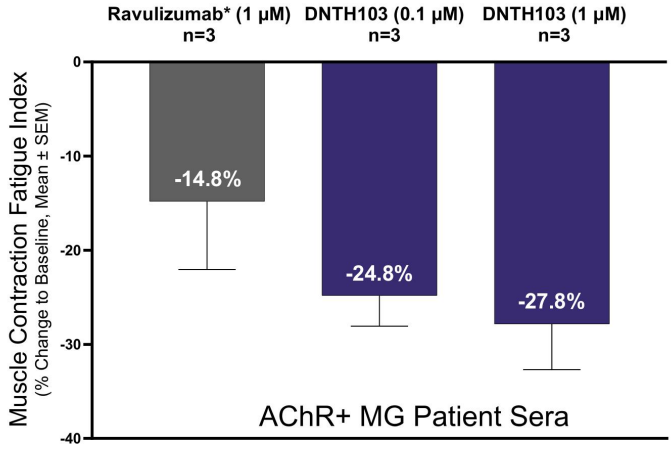
- **Patients on eculizumab showed a greater reduction in MG-ADL** over time than those on efgartigimod
- **Eculizumab showed statistically significant, deeper QMG reduction** and higher responder rate than efgartigimod
- **Eculizumab patients had a statistically significant greater reduction in rate 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**

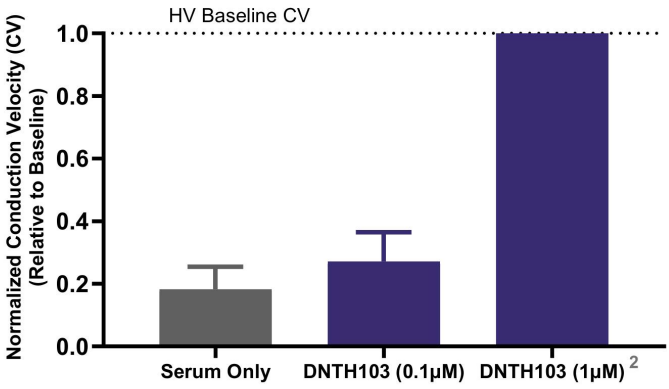


Results provide further scientific rationale for DNTH103 in gMG

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

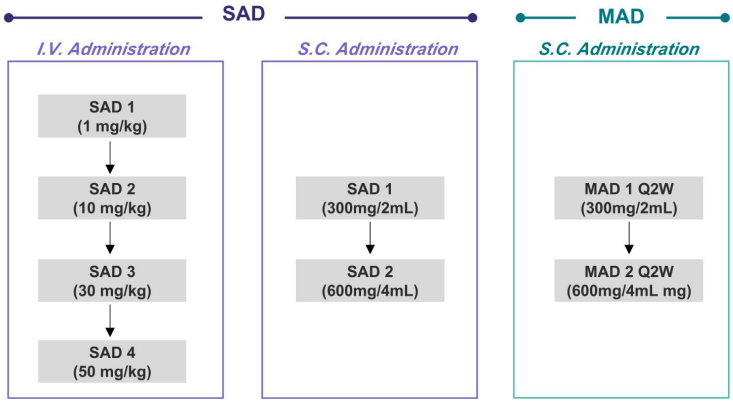
44 HVs enrolled into six cohorts:
- Placebo (N= up to 2)
- Treated (N= up to 6)

MAD

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

Key Parameters

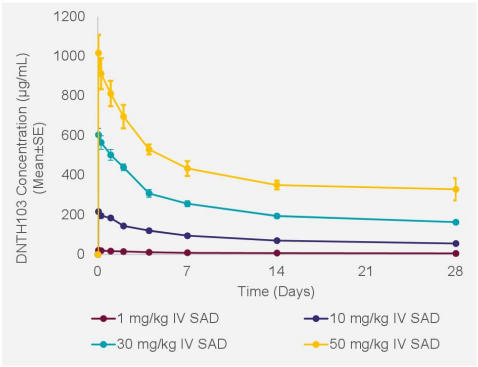
- **Safety, PK, and PD measured by percent classical pathway inhibition quantified in each cohort**



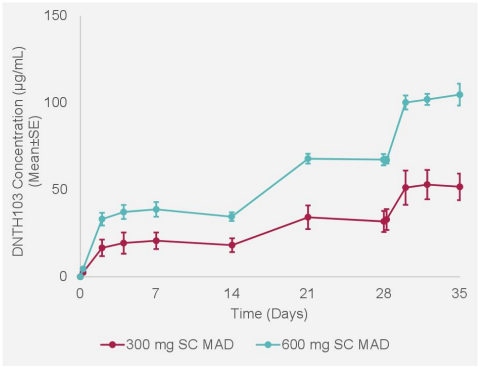
60 healthy volunteers completed dosing as of December 2023 across these eight cohorts

Claseprubart has demonstrated deep and sustained complement inhibition in healthy volunteers

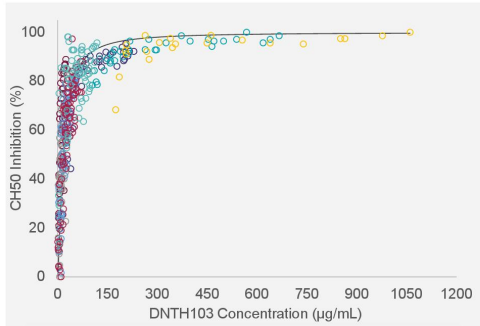
I.V. SAD:
Linear PK with Exposure Proportional Across Doses



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

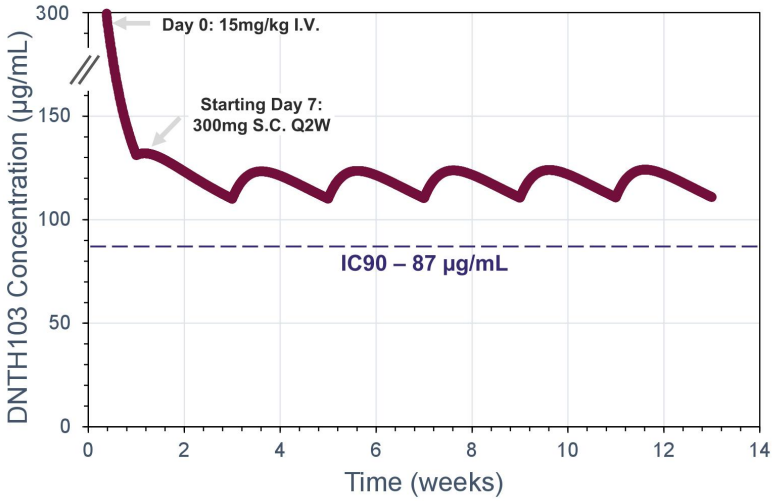
Phase 1 data estimated IC90 at ~87 µg/mL, leading to target dose of 300mg/2mL in Ph. 2 to achieve steady state >IC90

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

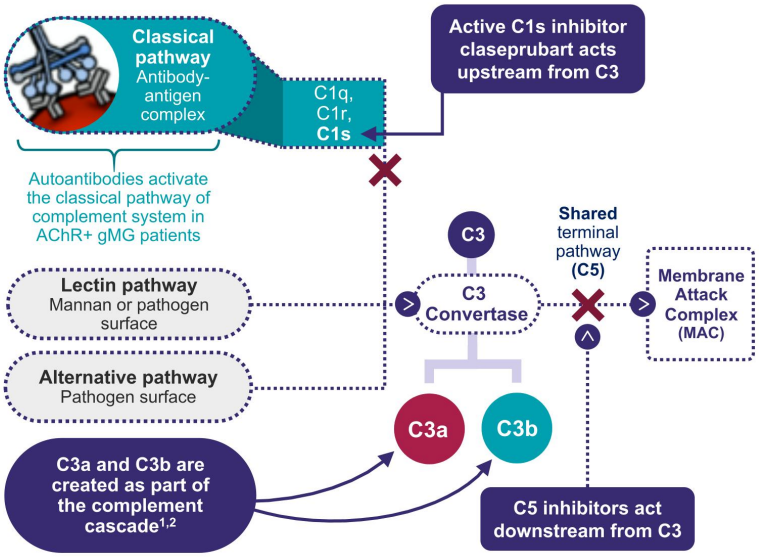
- **No standard safety lab findings (hematology, chemistry, coagulation LFTS and renal function)**
- **No serious adverse events**
- **No infection adverse event signal and no infections related to encapsulated bacteria**

	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
Ig Withdrawal Required Prior to Entering Study[1]	**YES**	**NO**	**NO**	**NO**
Study Endpoints / Results	• Confirmed ECI[2] • Ph. 3 Stage A results: □66.5% ECI (wk 12) *~1/3 pts **did not return** to pre-Ig washout baseline*	• ≥1-point aINCAT improvement	• ≥1-point aINCAT improvement • Ph. 2 PoC response rates: □SOC-Treated: 52% □SOC-Refractory: 50%	• Switching Ig patients to claseprubart 7 days after last dose • Aiming for ≥1-point aINCAT improvement OVER SoC/Ig in ≥50% of patients in Part A

- 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
Ig refers to IVIG and SCIg
1. ADHERE required discontinuation of IVIg or SCIg 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

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 Disease *~350,000 U.S. Patients*	✓	• *B Cell:* ianalumab positive Ph. 3; telitacicept positive Ph. 3
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
Dermatomyositis *~50,000 U.S. Patients*	✓	• *Type 1 interferon:* dazukibart positive Ph. 2
Cutaneous Lupus Erythematosus *~300,000 U.S. Patients*	✓	• *Type 1 interferon:* litifilimab positive Ph. 2
Lupus Nephritis *~120,000 U.S. Patients*	✓	• *B Cell:* belimumab approved

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 identified first three priority indications of Sjögren's Disease, Systemic Lupus Erythematosus, and Dermatomyositis (DM) to build a rheumatology franchise

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

Accomplished team of biotech industry veterans and scientists committed to bringing innovation to market

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Select Autoimmune Drugs Developed by Dianthus Team

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