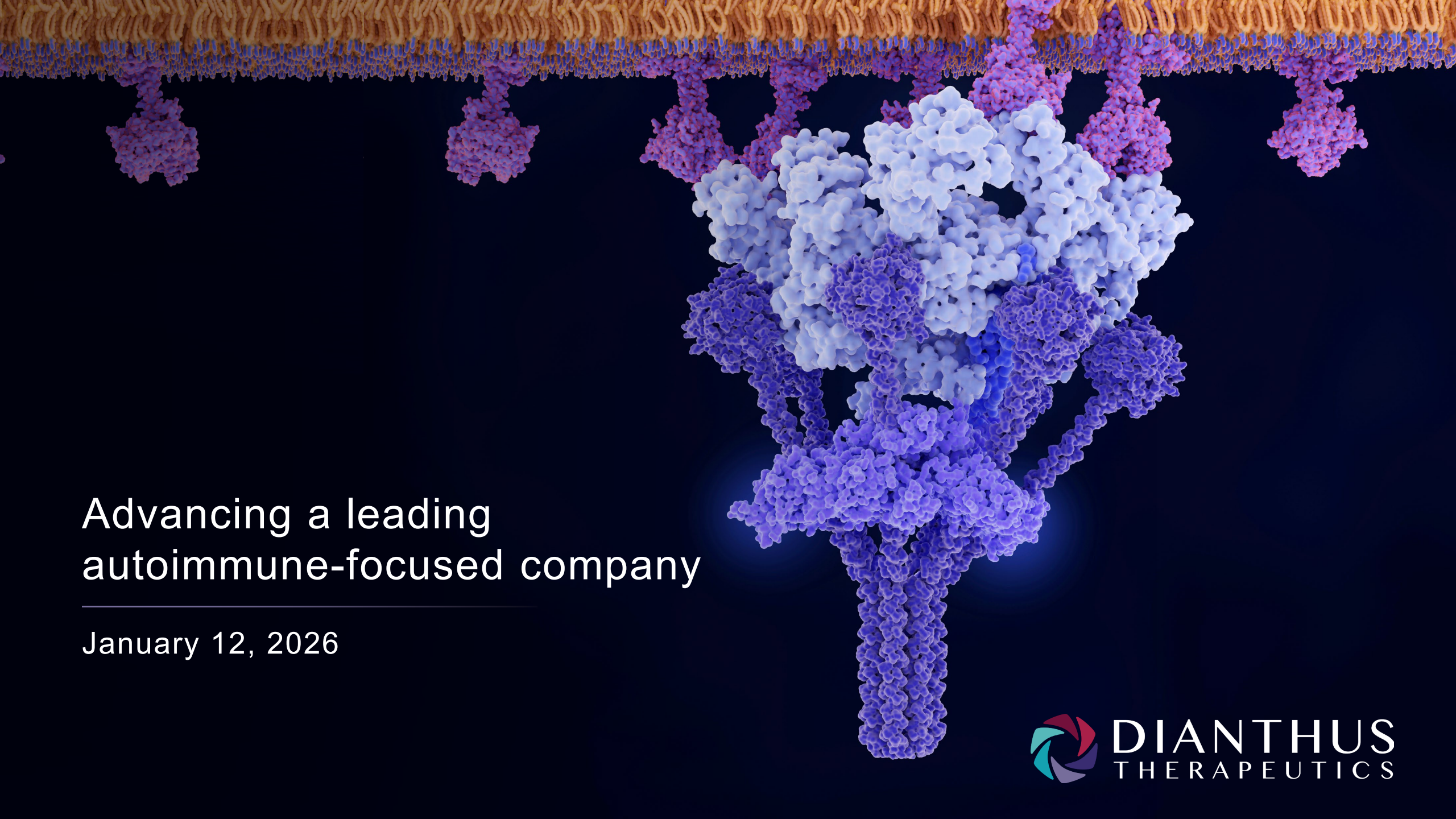


Advancing a leading autoimmune-focused company

January 12, 2026

DIANTHUS THERAPEUTICS

Forward-looking statements

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

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

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

Advancing a leading autoimmune-focused company

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

Claseprubart
(aC1s mAb)

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

DNTH212
(BDCA2 and BAFF/APRIL bifunctional fusion protein)

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

Claseprubart 2026 milestones:

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

DNTH212 2026 milestones:

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

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

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

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

claseprubart

CONFIDENCE

Aim for Potent, Rapid, Consistent Efficacy

Potential for Best-in-Class Profile

Broad Potential in Neuromuscular Diseases

CLASSICAL

Upstream Inhibition of Classical Pathway Only

Potential to Preserve Immune Function

Targeting No Boxed Warning or REMS

CONVENIENCE

<10-Second Autoinjector

One-Click Every 2 or 4 Weeks

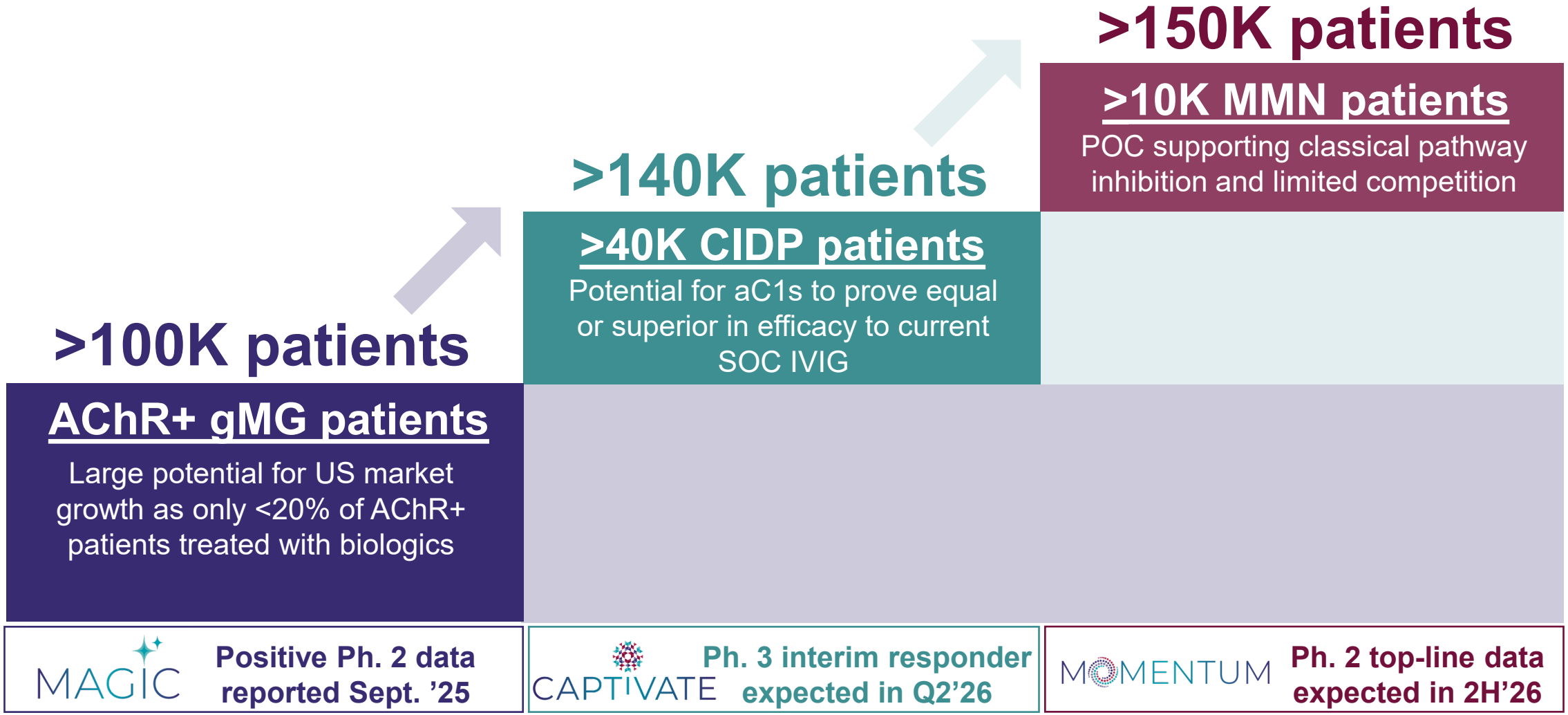
Self-Administered At Home *or* On-the-Go

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

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

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

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



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

CIDP & MMN: Komodo claims data 2013-2025, adjusted to account for 70% capture of real-world patient counts for biologic treated patients; CIDP adjusted to account for 27% misdiagnosed

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

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)*

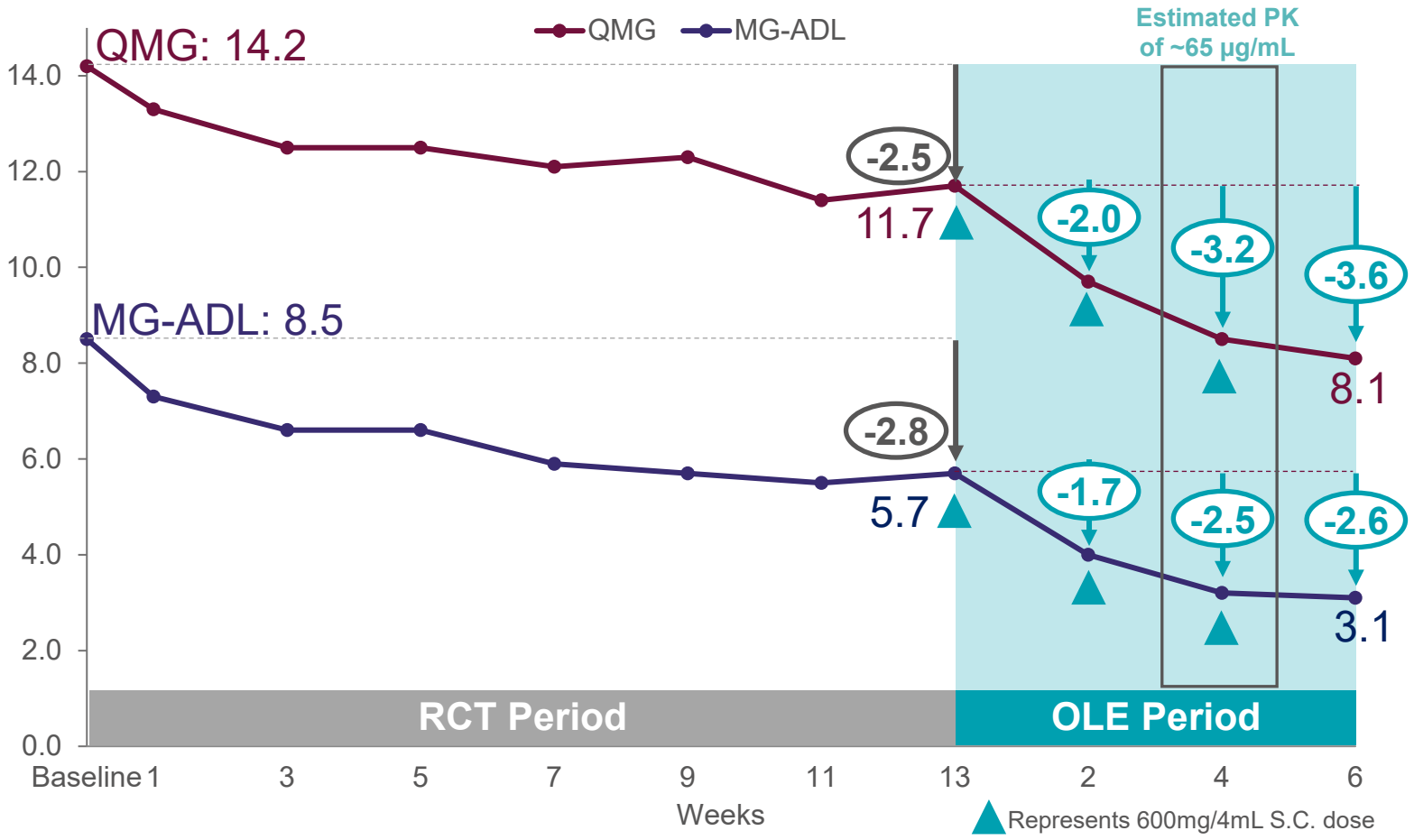
Claseprubart 300mg/2mL Q2W treatment arm achieved statistical significance vs. placebo across all five key efficacy measures

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

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

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

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



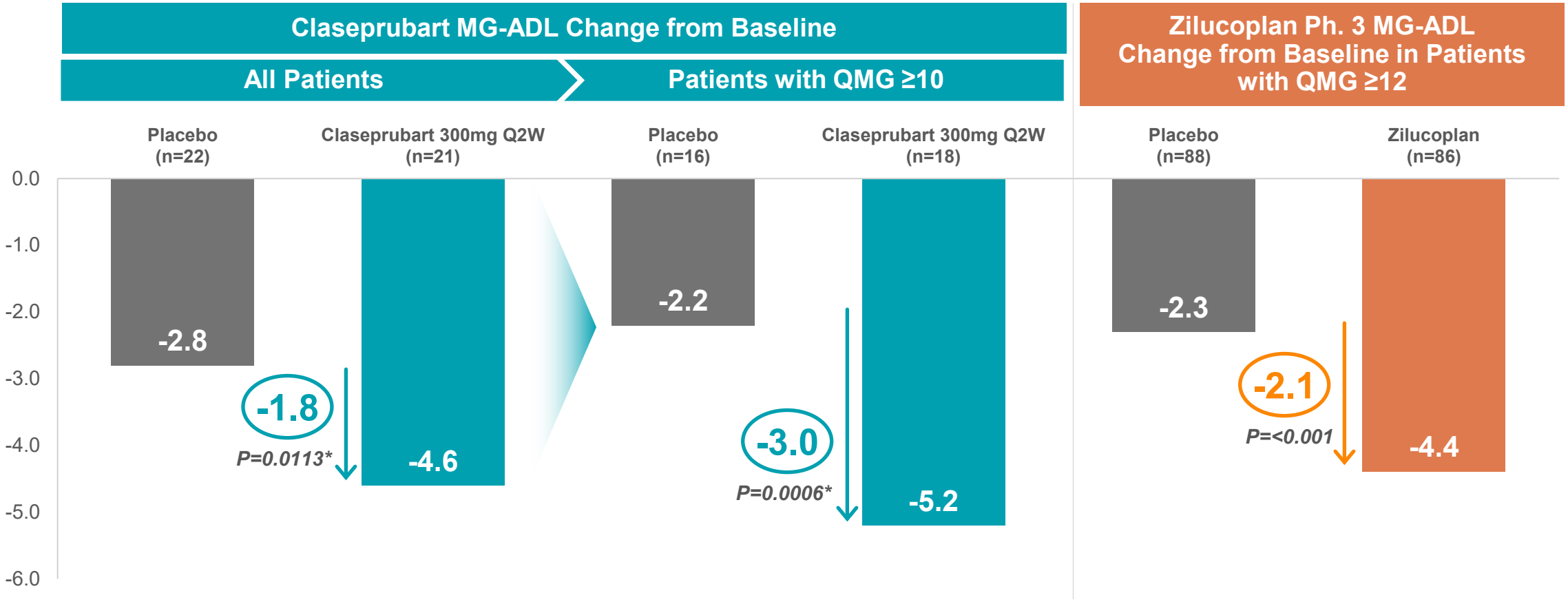
PBO Patients Entering OLE Received 600mg/4mL Q2W w/ No Loading Dose

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

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

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

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



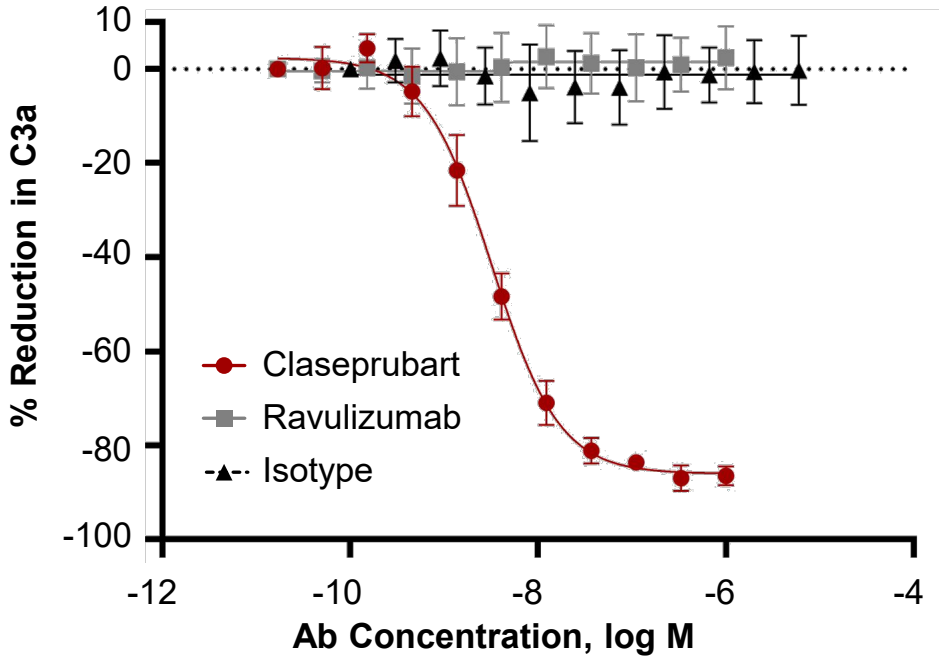
Ph. 2 study did not include QMG inclusion criteria, similar to ravulizumab Ph. 3; post-hoc analysis of MaGic data demonstrates potentially best-in-class MG-ADL improvement in patients with QMG ≥10

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

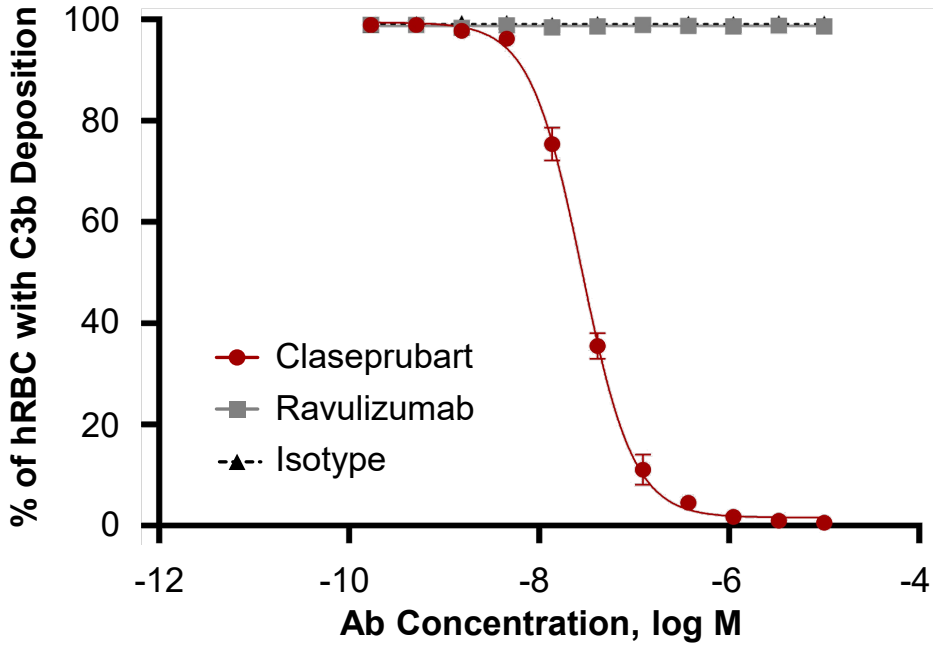
Potential for improved efficacy vs. C5 inhibitors with claseprubart may be due to upstream inhibition

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

C3a Formation[1]



C3b Deposition[2*]



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

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

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

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

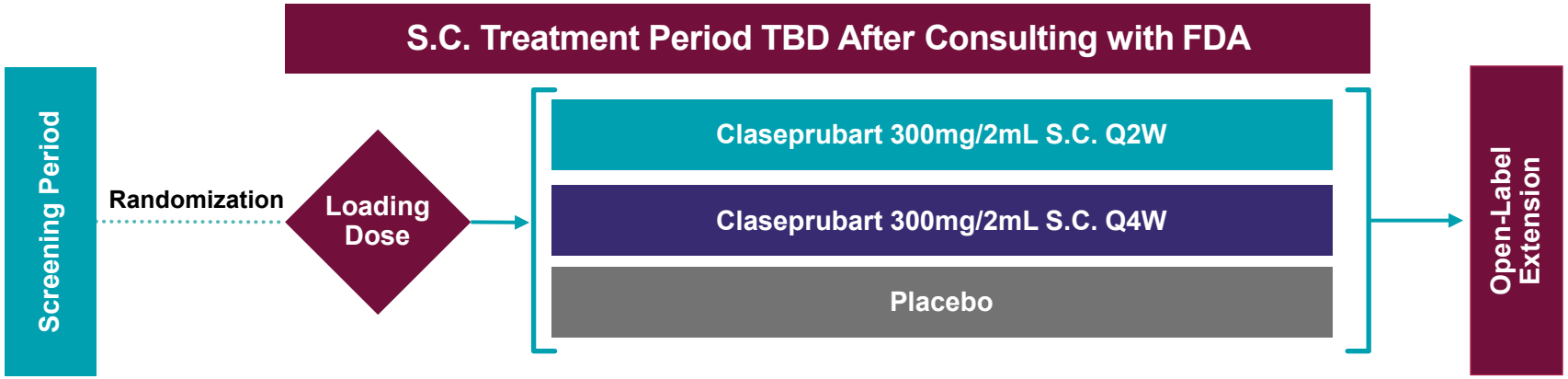
Final Ph. 3 trial design TBD after regulatory consultations

Highlights

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

Endpoints

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



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

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

C5 OR SUPERIOR EFFICACY
(ULTOMIRIS/SOLIRIS/ZILBRYSQ)

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

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

C1s SAFETY
(ENJAYMO)

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

Targeting no Boxed Warning & REMS

AUTOINJECTOR CONVENIENCE
(DUPIXENT)

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

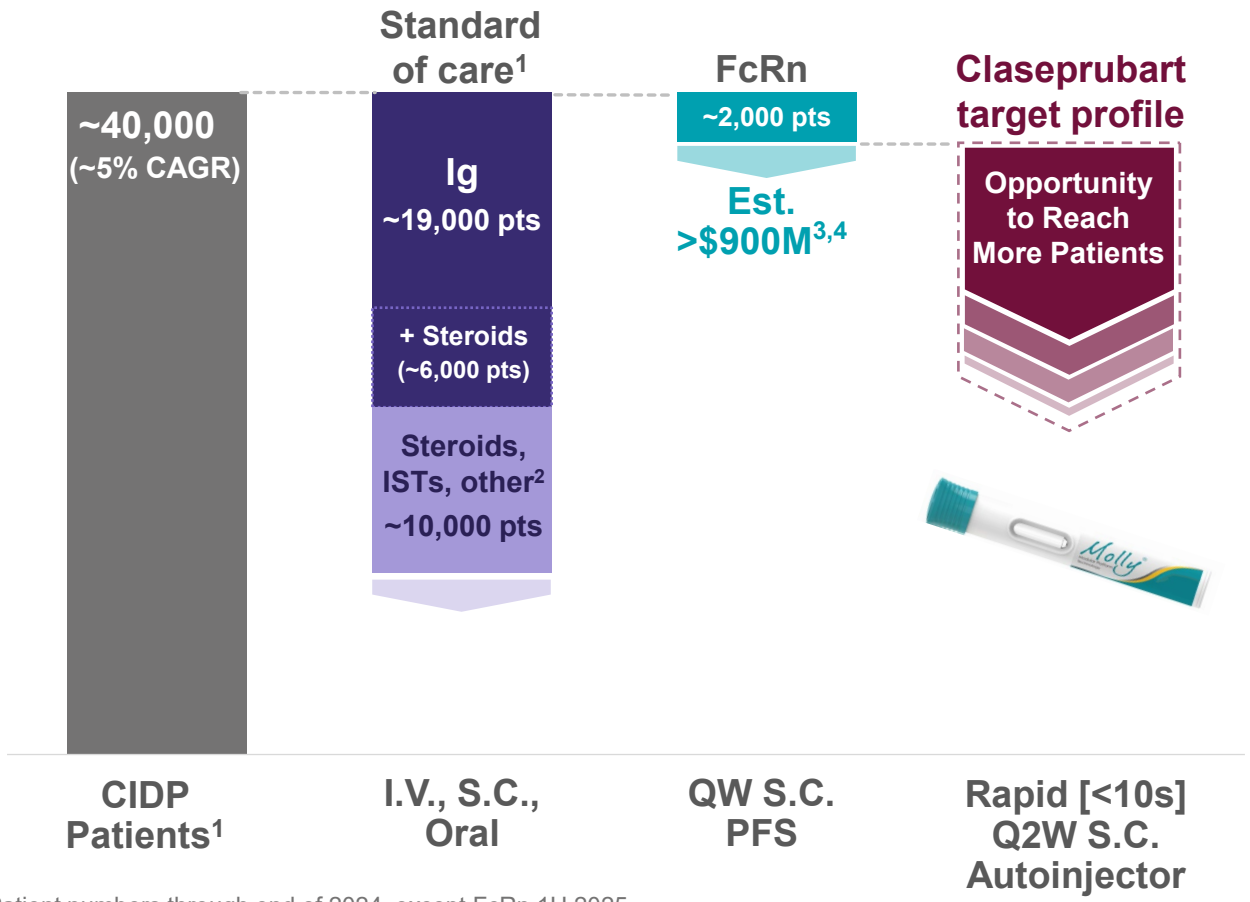
Targeting single 300mg/2mL S.C. Q2W or Q4W

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

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

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

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



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

US CIDP Market Opportunity

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

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

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

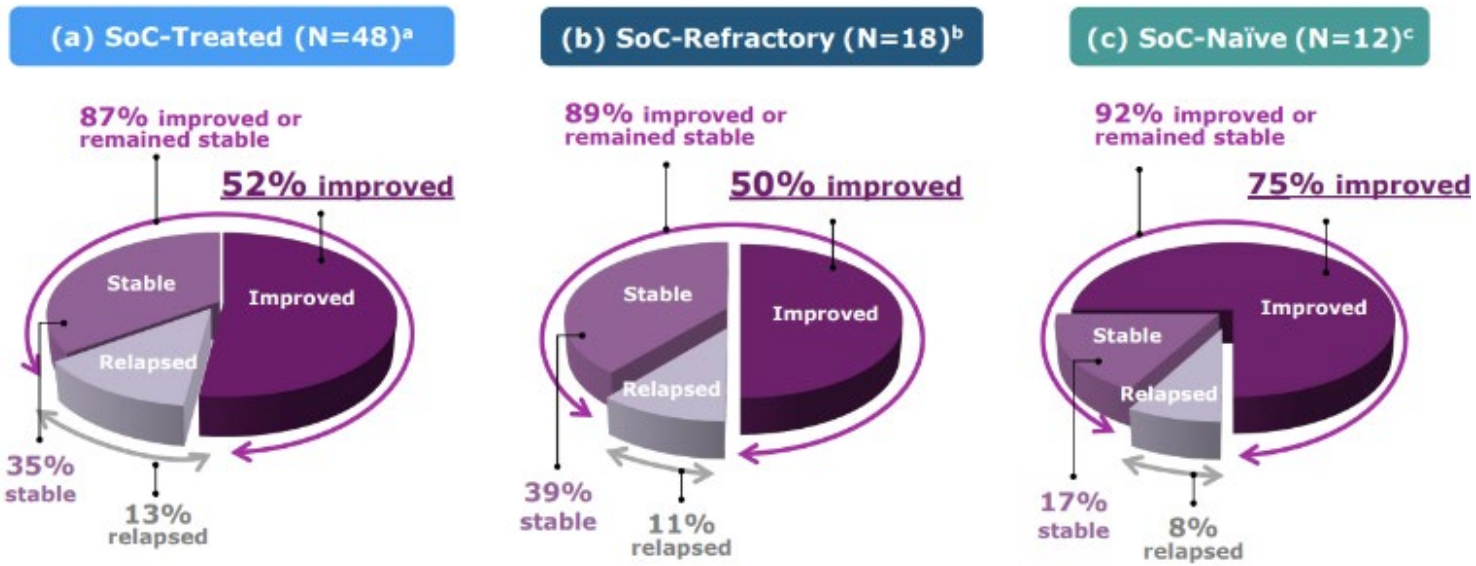
Neuromuscular indication with high unmet medical need

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

Evidence supports classical complement role in disease

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

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



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

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

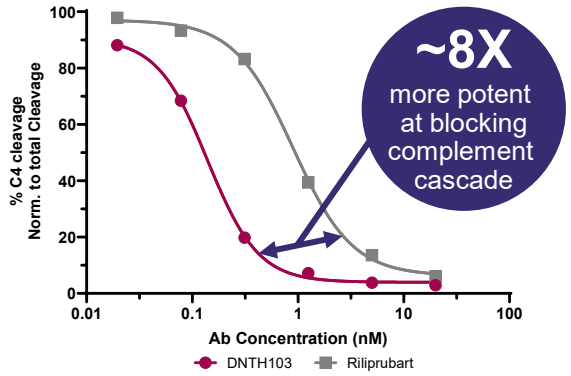
Claseprubart has superior affinity and potency vs. riliprubart

Affinity Assays

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

Enzymatic Assay

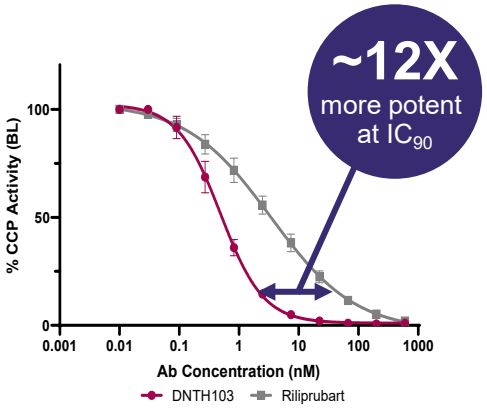
C4 cleavage by human active C1s[2]



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

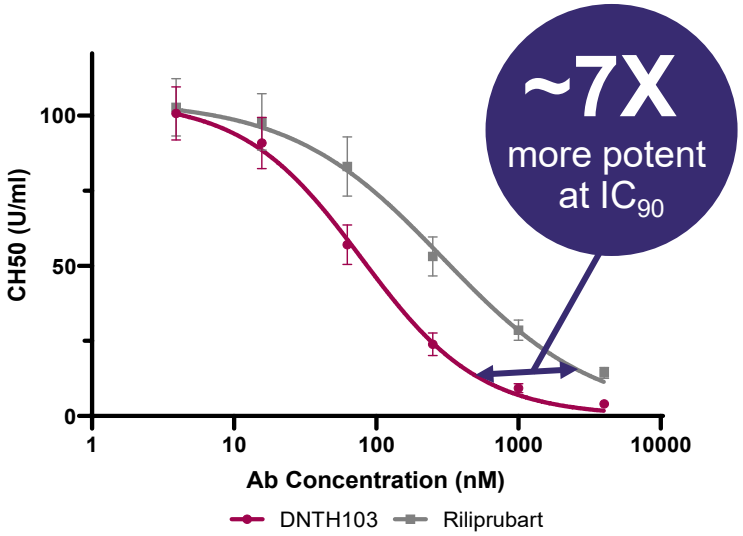
Functional Assays of Classical Pathway Inhibition

Wieslab classical pathway Assay in human serum[3]



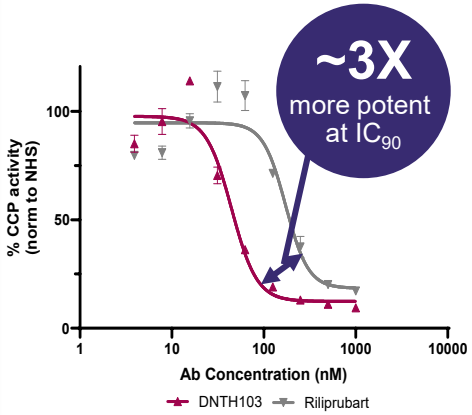
	DNTH103	riliprubart
IC_{90} (µg/mL)	**0.45**	5.4

CH50 assay of RBC lysis in human serum[3]



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

Liposome lysis in human serum[3]



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

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

Note: Riliprubart is produced using sequence from patent WO2018071676A1

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

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

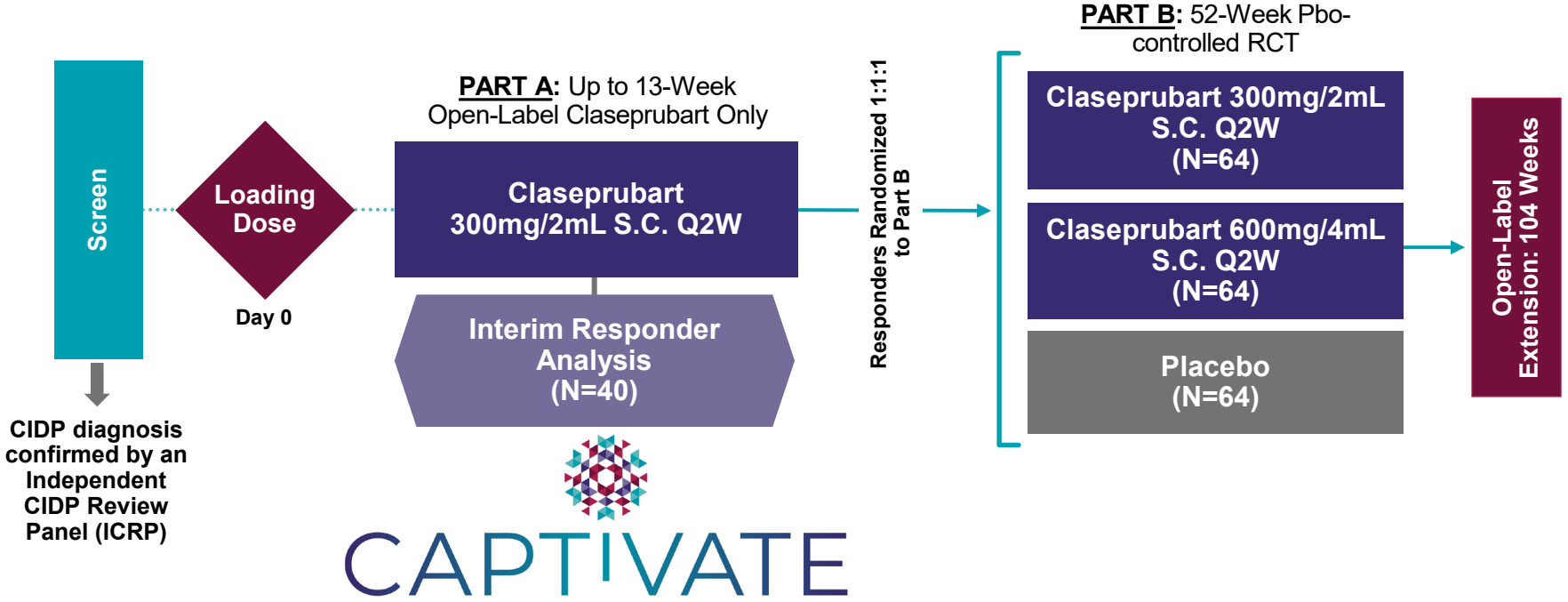
CIDP interim responder analysis with first 40 patients in Part A anticipated in Q2'26

Highlights

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

Endpoints

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



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

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

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

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

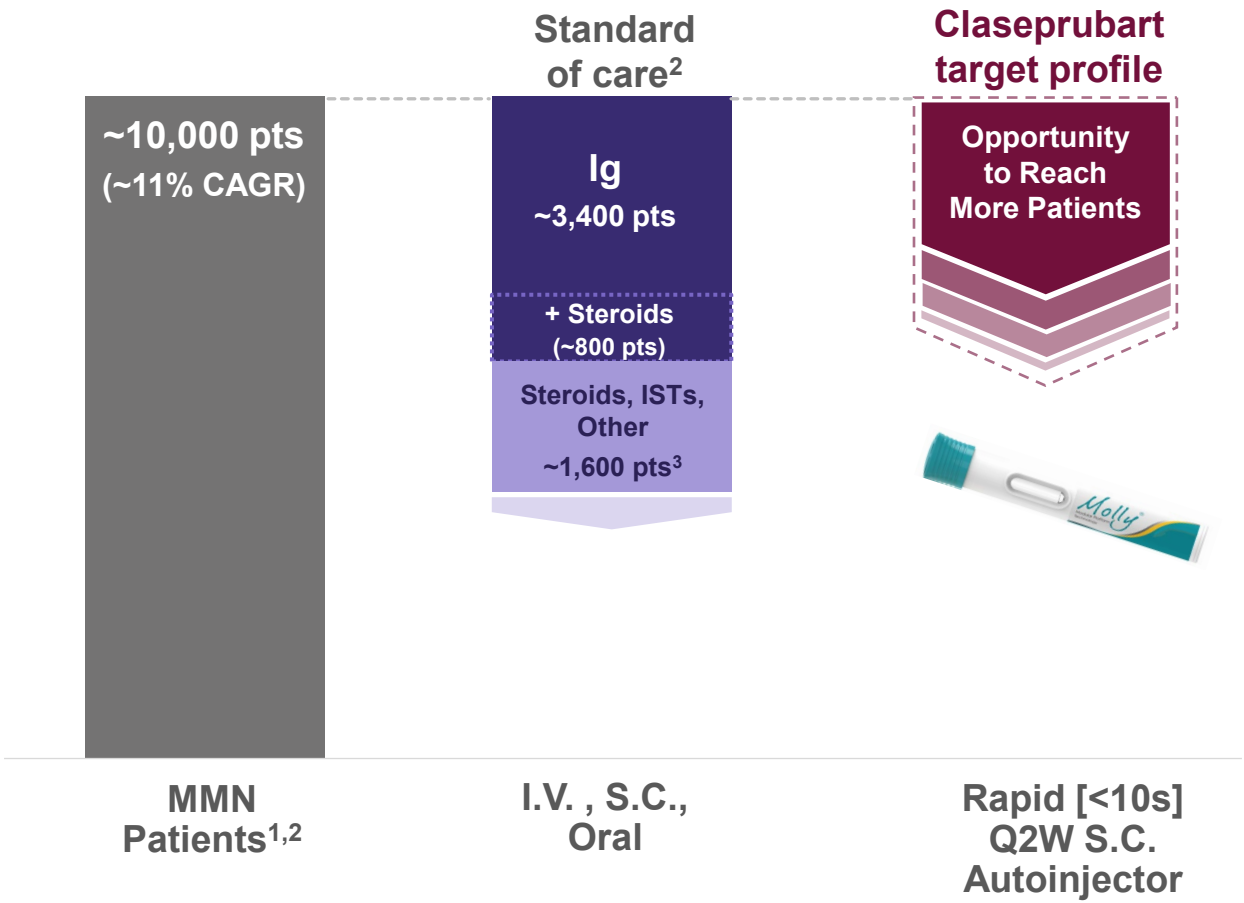
Source: Company filings, presentations and clinicaltrials.gov

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

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

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

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



*Patient numbers through end of 2024

US MMN Market Opportunity

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

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

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

Neuromuscular indication with high unmet medical need

>10,000
patients in the U.S.

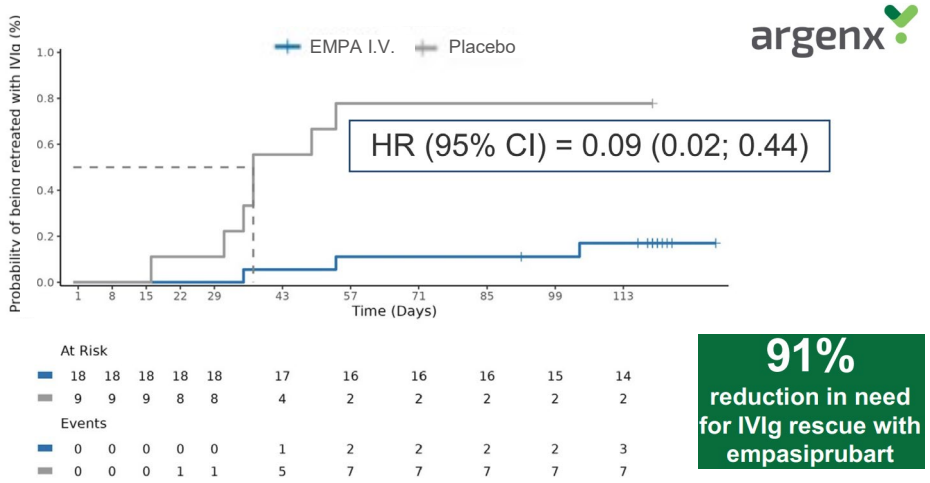
No approved targeted biologic therapies

Evidence supports classical complement role in disease

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

MMN patient sera has been confirmed to activate complement

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



91%
reduction in need for IVIg rescue with empasiprubart

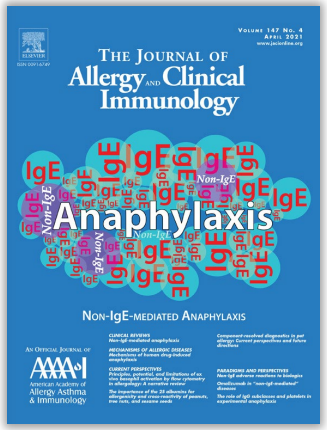
"We hypothesize that targeting the **classical complement pathway is a potential therapeutic approach in MMN**. We investigated the interaction of circulating anti-GM1 IgM from patients with MMN with complement in detail using iPSC-derived MNs. In this disease model for MMN, we evaluated the effects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

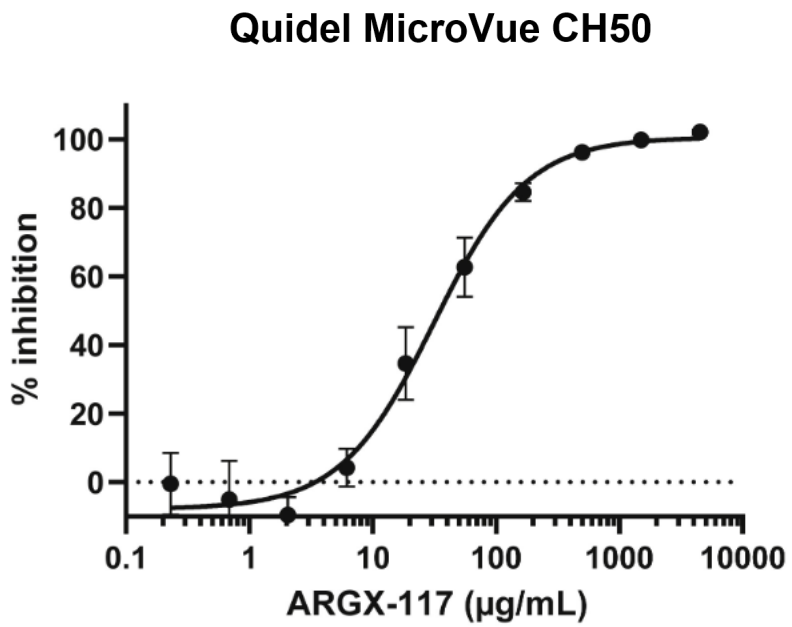
Phase 2 trial of claseprubart, a low-volume Q2W S.C., ongoing in MMN

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

Claseprubart demonstrates superior classical pathway potency vs. empasiprubart

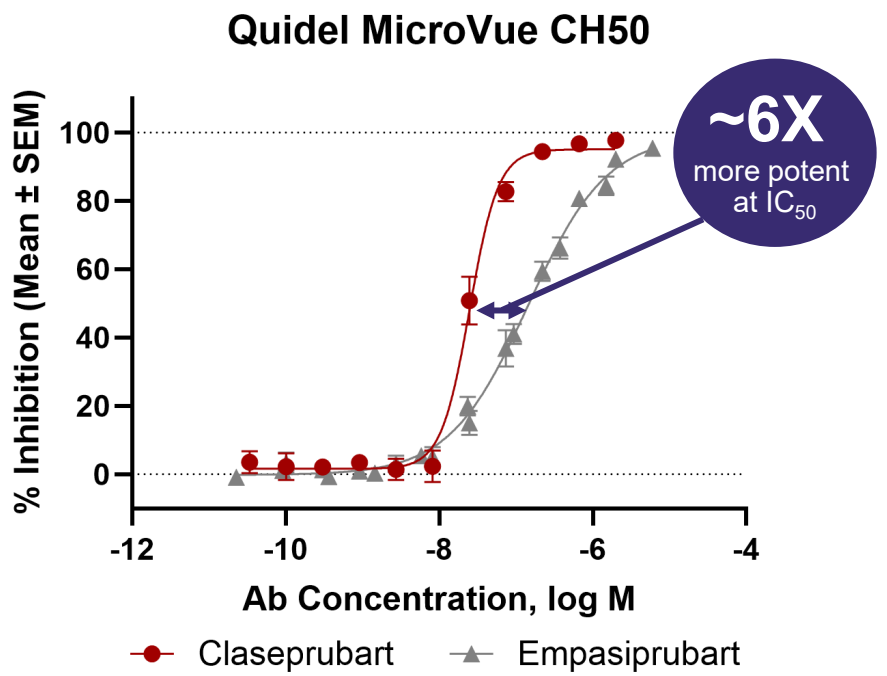
Empasiprubart Published Classical Pathway Potency Data Using the Quidel MicroVue CH50[1]





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

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



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

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

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

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

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

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

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

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

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

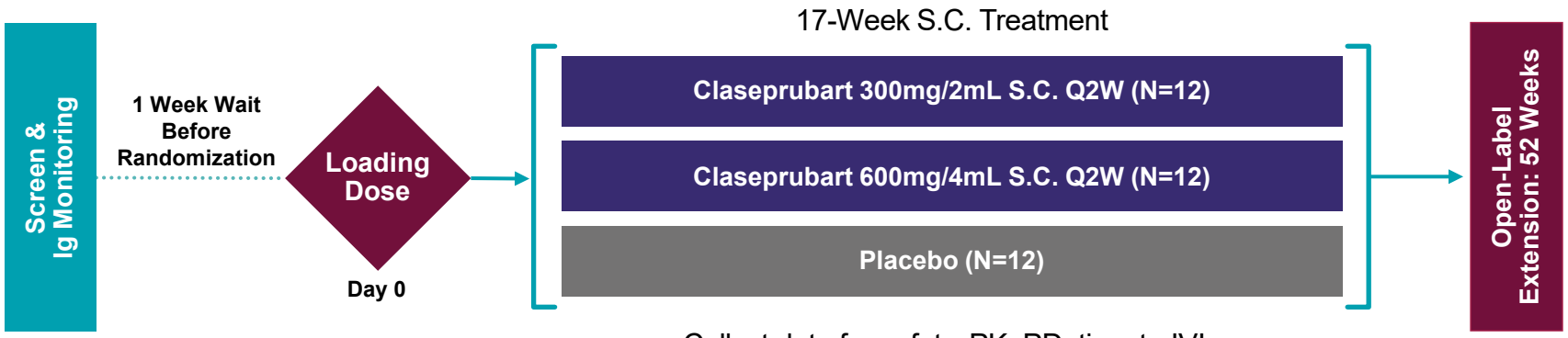
A global, multicenter, randomized, double-blind, placebo-controlled study to evaluate the safety, efficacy, and PK / PD of claseprubart administered S.C. following initial loading dose

Highlights

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

Endpoints

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



MOMENTUM

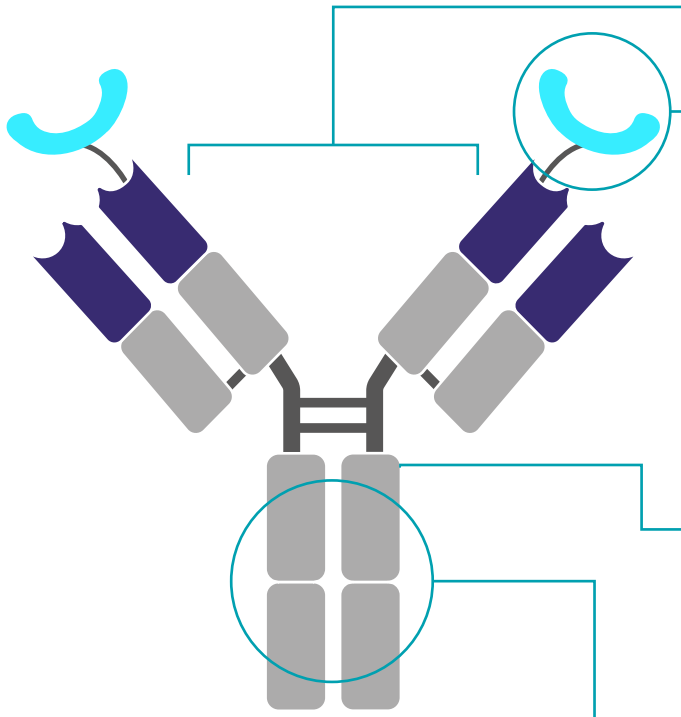
Top-line data expected in 2H’26

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

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

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

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



DNTH212

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

Anti-BDCA2 Antibody

Potent inhibition of Type 1 interferon

TACI-CRD2 Domain

Potent reduction of Ig levels via BAFF/APRIL inhibition

Afucosylated Bifunctional Ab

Enhanced ADCC on target cells leads to pDC depletion

YTE Mutation on Fc

Extended half-life

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

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

EFFICACY

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

SAFETY

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

CONVENIENCE

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

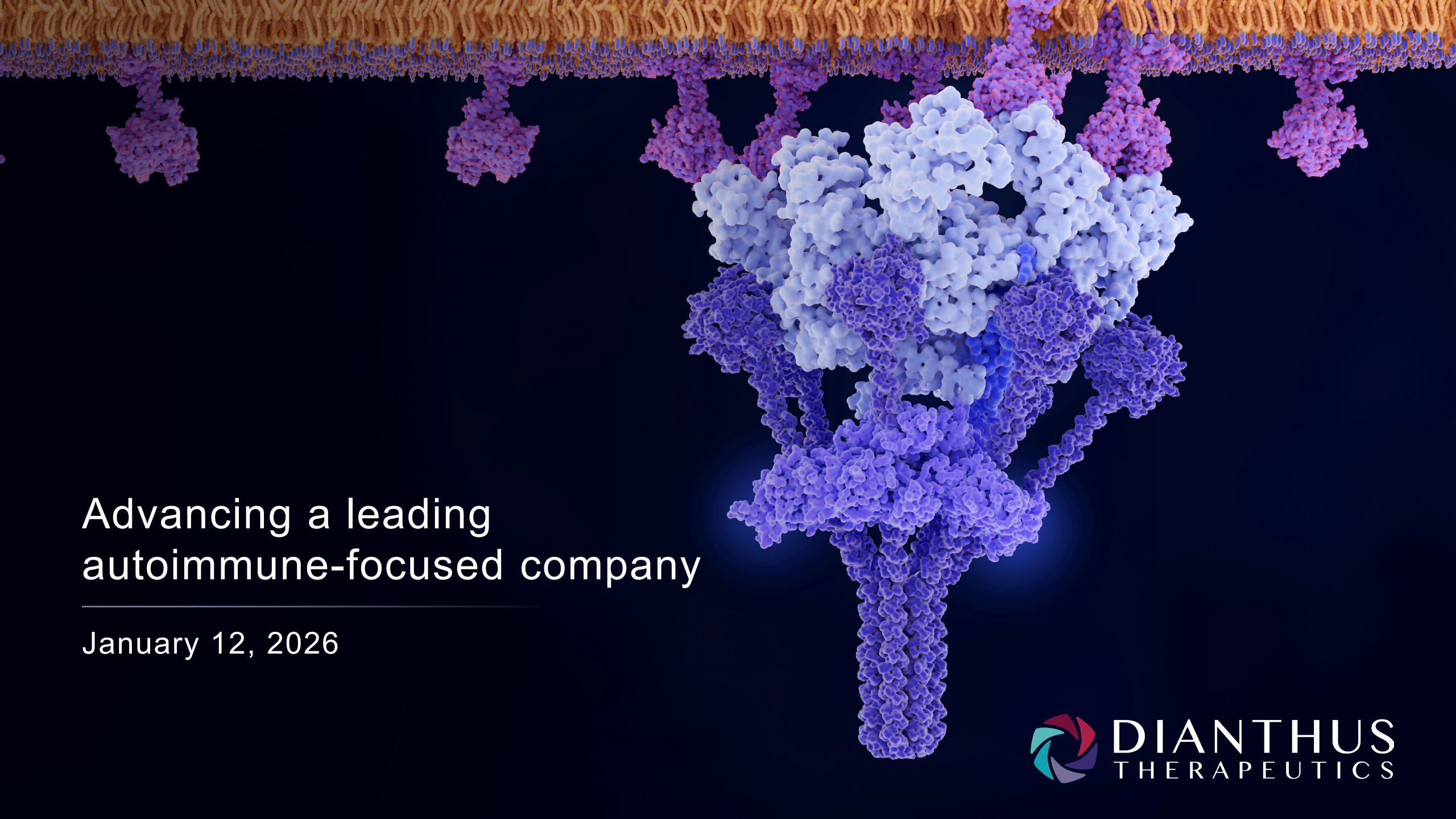
Achieving the TPP would position DNTH212 as a first-line biologic across a range of indications

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

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

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

1. Estimated cash includes preliminary and unaudited cash, cash equivalents and investments as of December 31, 2025
2. Shares outstanding on a pro forma basis, which assumes the exercise of all outstanding pre-funded warrants
3. Based on Sanofi Q3'25 financial results conference call transcript
4. Based on publicly available information: https://argenx.com/content/dam/argenx-corp/media-documents/Earnings_press_release_HY.pdf.coredownload.inline.pdf



Advancing a leading autoimmune-focused company

January 12, 2026

DIANTHUS THERAPEUTICS