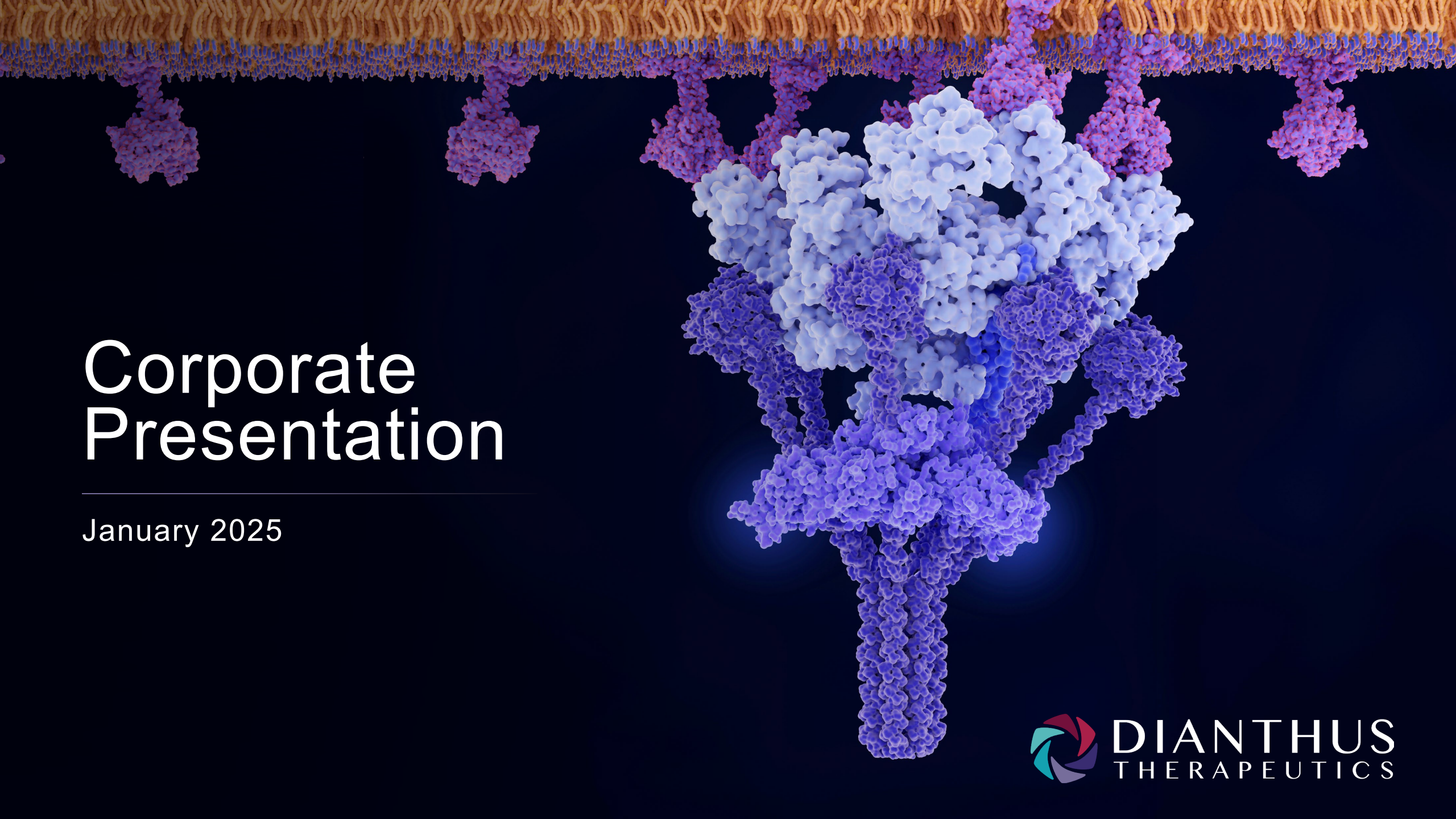


Corporate Presentation

January 2025

DIANTHUS THERAPEUTICS

FORWARD-LOOKING STATEMENTS

Certain statements in this presentation (“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 Stated Private Securities Litigation Reform Act of 1995, concerning Dianthus Therapeutics, Inc. (the “Company”). These forward-looking statements include statements regarding the Company’s 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 DNTH103, and any developments or results in connection therewith, including the target product profile of DNTH103; the anticipated timing of the results from those studies and trials; expectations regarding the use of proceeds and the time period over which the Company’s capital resources will be sufficient to fund its anticipated operations; and expectations regarding the market and potential opportunities for complement therapies, in particular with respect to DNTH103. DNTH103 is an investigational agent that is not approved as a therapy 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 DNTH103 and data from clinical trials may not be predictive of the results or success of ongoing or later clinical trials, that the development of DNTH103 or the Company's compounds may take longer and/or cost more than planned, that the Company may be unable to successfully complete the clinical development of the Company’s compounds, that the Company may be delayed in initiating, enrolling or completing any 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, 2023, 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. 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.

Building a leading autoimmune-focused company by advancing best-in-class complement therapeutics

- Rapidly advancing **DNTH103, a highly potent and differentiated** investigational complement inhibitor **selectively targeting active C1s and the classical pathway**
- DNTH103 intended to be conveniently delivered via a **patient friendly**, **self-administered, subcutaneous auto-injector dosed once-every-two-weeks** to treat severe autoimmune diseases
- **Best-in-class** complement inhibitor profile supported by a **~60-day half-life from Ph. 1,** a potentially **differentiated safety profile,** and **superior *in vitro* potency demonstrated vs. riliprubart**
- **Pipeline-in-a-product potential** validated with clinical proof-of-concept for **classical pathway** inhibition in gMG, CIDP and MMN
- **Near-term data catalysts** with **top-line gMG Ph. 2 results** in **2H'25, interim responder analysis for pivotal Ph. 3 CIDP trial** in **2H'26,** and **top-line MMN Ph. 2 results** in **2H'26**
- **Strong financial position** with **cash runway into 2H'27** expected to fund multiple near-term catalysts

DNTH103 is a pipeline-in-a-product with potential to be a first-line, best-in-class biologic treatment

	Generalized Myasthenia Gravis	Chronic Inflammatory Demyelinating Polyneuropathy	Multifocal Motor Neuropathy
Indications			
Market size	***~60,000 U.S. patients***	***>40,000 U.S. patients***	***~5,000–10,000 U.S. patients***
Market insight	Multi-billion $ and growing market with opportunity for a best-in-class and convenient classical pathway inhibitor to expand use of first-line biologics	Sanofi's riliprubart validated active C1s inhibition with robust efficacy in patients who were refractory, stable, as well as naïve to IVIG, the standard of care	Empasiprubart, a C2 inhibitor demonstrated impressive efficacy in MMN, validating classical pathway inhibition
	DNTH103 Aims to be the First-line Biologic Choice Across All Three Indications		
DNTH103 opportunity	Unique potential to combine robust, continuous symptom control with convenient uninterrupted dosing and administration and a differentiated safety profile	Demonstrated superiority vs. riliprubart in multiple head-to-head *in vitro* PD potency experiments, with potential to address unmet needs of CIDP patients including refractory to IVIG	Potent and selective classical pathway inhibition could capture majority of MMN market with limited competition and no other MOAs in development
Data timing	**Ph. 2 data in 2H'25**	**Ph. 3 interim responder analysis in 2H'26**	**Ph. 2 data in 2H'26**

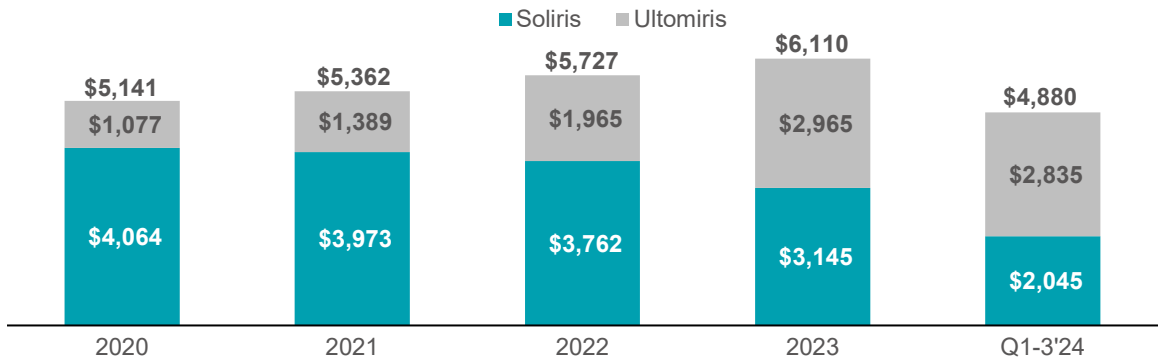
Neuromuscular Franchise: Clinical Development & Commercial Synergies

gMG: https://www.mgregistry.org/, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7033452/#; CIDP: IQVIA Claims Data, Riliprubart Phase 2 at EAN 2024; MMN: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3983019/

gMG represents a multi billion-dollar opportunity with only two first-line biologics, each with room to improve

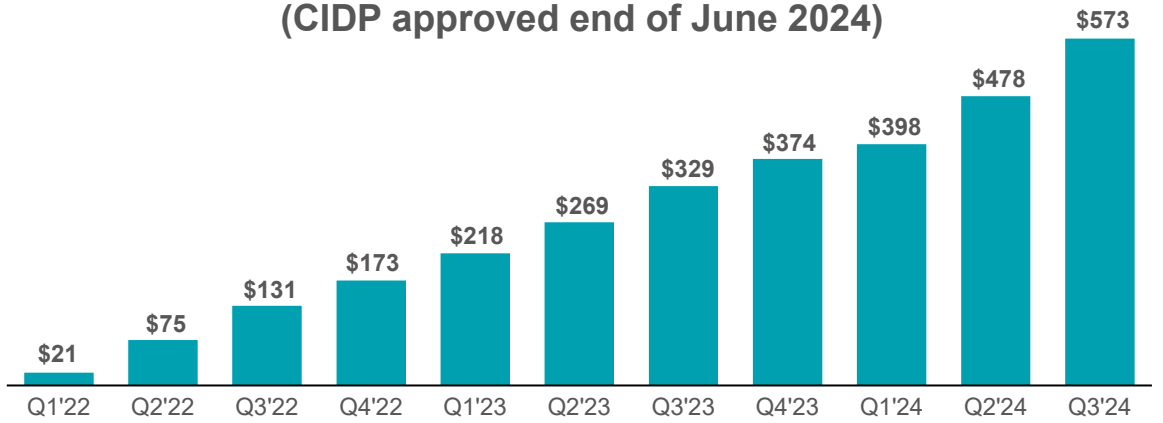
Complement Class Sales ($M)
I.V. C5 inhibitors[1]

~1/3 of sales in gMG[2] (only I.V.); sales growth driven by U.S. naïve gMG patients[3]



FcRn Class Sales ($M)
Efgartigimod

Majority of sales in gMG (CIDP approved end of June 2024)



Considerations	Ravulizumab	Efgartigimod	Key Differentiators of DNTH103
gMG is a chronic, classical pathway-driven disease[4]	C5s validate classical pathway inhibition as SOC for continuous and effective symptom control Increased risk of infections from encapsulated bacteria due to inhibition of lectin and alternative pathways	Discontinuous dosing leads to clinical deterioration between cycles beginning in week 5 as measured by the MG-ADL and QMG	**Targeting continuous symptom control with a classical pathway inhibitor dosed at 300mg Q2W achieving >IC90 on CH50** **Potentially differentiated safety profile by leaving lectin and alternative pathways intact**
Convenient dosing and administration	I.V. infusion administered every 8 weeks	I.V. or S.C. infusion by healthcare professional every week for 4 weeks followed by interruptions in dosing, leading to rebound in symptoms as measured by MG-ADL and QMG	**Patient-friendly convenience by targeting Q2W self-administered S.C. autoinjector**

Soliris & Ultomiris 2021 sales account for 1/1 – 6/30 & 7/21 – 12/31. Evaluate Pharma
https://www.mgregistry.org/, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7033452/#
1 Soliris / Ultomiris are approved in gMG, aHUS, NMOSD, PNH; 2 Wall Street research estimate; 3 Astra Zeneca Q1 2024 results; 4 AChR antibody positive gMG: https://nyaspubs.onlinelibrary.wiley.com/doi/10.1111/nyas.13522

DNTH103 targeting superior convenience and reduced patient burden vs. approved biologics for gMG

Lower volume, less frequent, self-administered **S.C. autoinjector**

DNTH103

Daily self-administration or higher-volume **S.C.** administered by health care professional

VYVGART® Hytrulo (efgartigimod alfa and hyaluronidase-qvfc) Subcutaneous Injection 180 mg/mL and 2000 U/mL vial

RYSTIGGO® (rozanolixizumab-noli) Injection For Subcutaneous Use 280 mg/2 mL vial

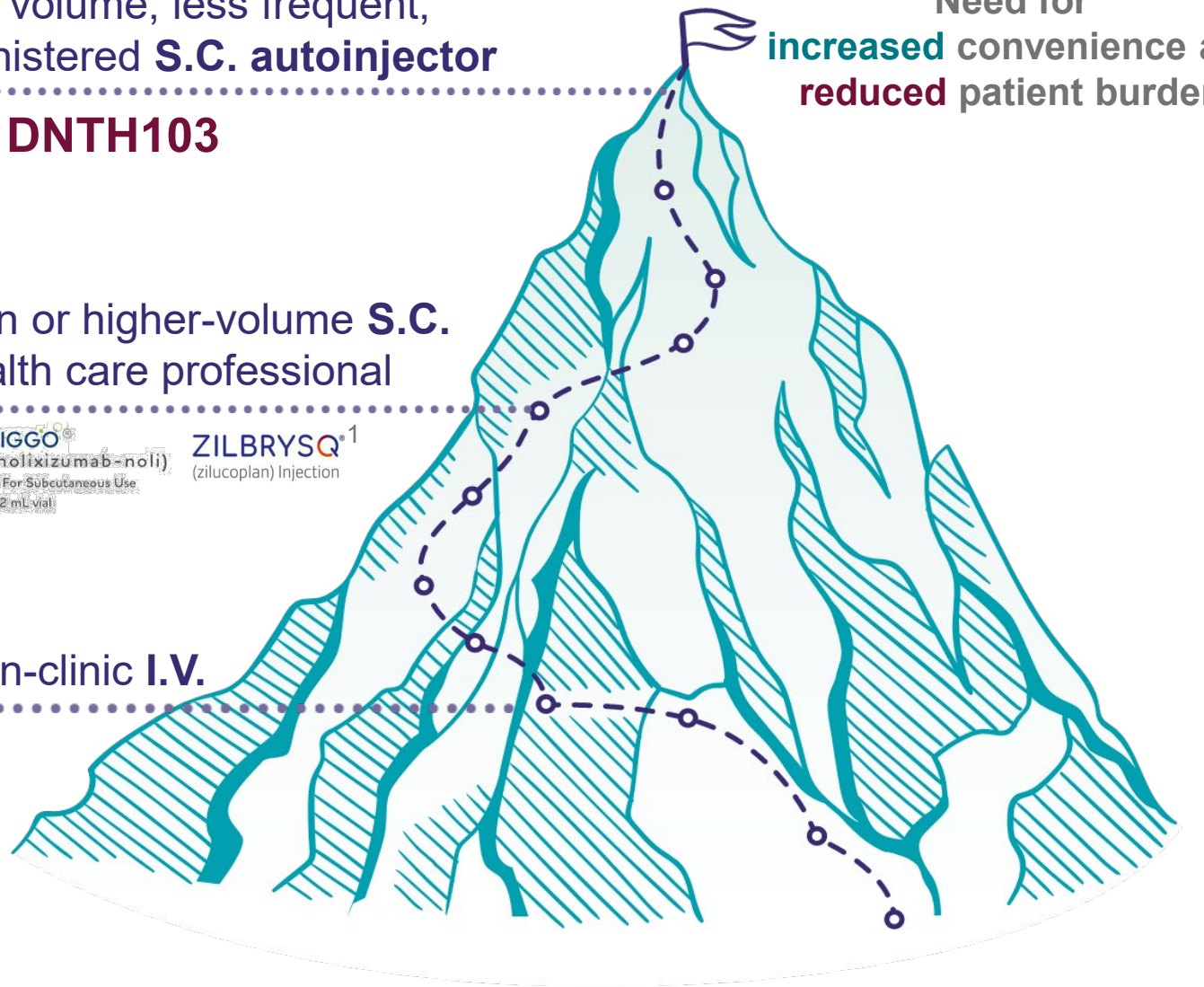
ZILBRYSQ®[1] (zilucoplan) Injection

More frequent and/or higher-volume, in-clinic **I.V.**

SOLIRIS® (eculizumab) Injection for Intravenous Use 300 mg/30 mL vial

VYVGART® (efgartigimod alfa-fcab) Injection for Intravenous Use 400 mg/20 mL vial

ULTOMIRIS® (ravulizumab-cwvz) injection for intravenous use

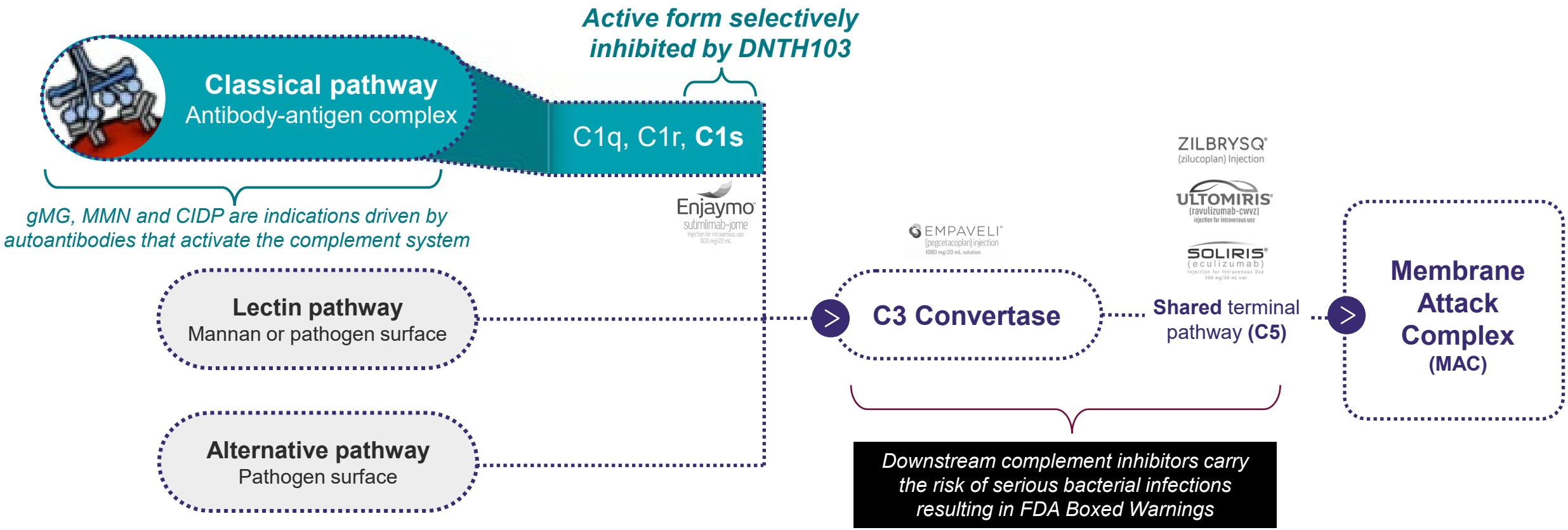


Need for **increased** convenience and **reduced** patient burden

1 Can be self-administered daily via pre-filled syringe

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



CIDP is an attractive opportunity with clinical PoC demonstrated via active C1s inhibition

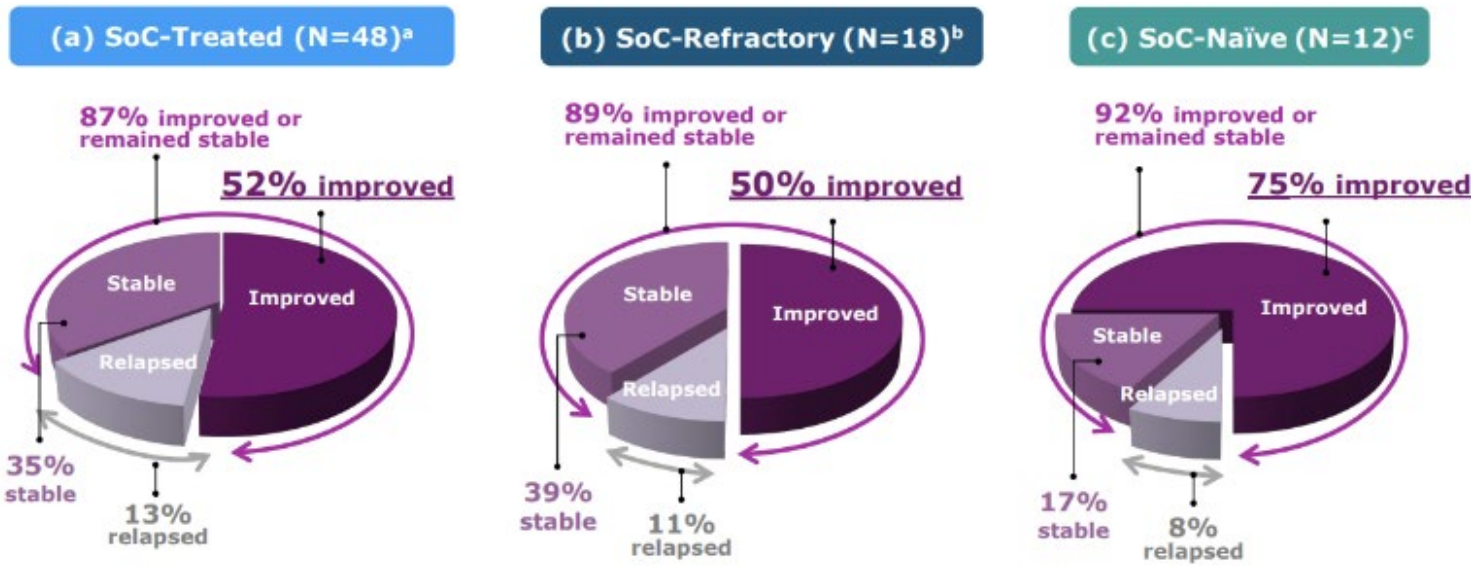
Neuromuscular indication with high unmet medical need

>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]

Sanofi Ph. 2 riliprubart (SAR445088) data validates active C1s in CIDP[1]; Maintenance regimen of 600mg/4mL S.C. weekly[2]



DNTH103 target dose of 300mg/2mL S.C. every two weeks may offer more convenient, lower volume dosing for CIDP patients

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

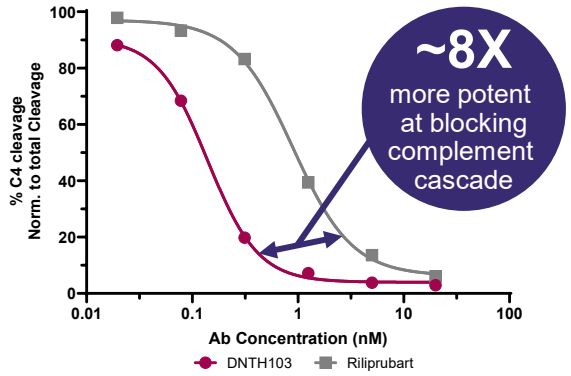
DNTH103 has superior affinity and potency vs. riliprubart

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

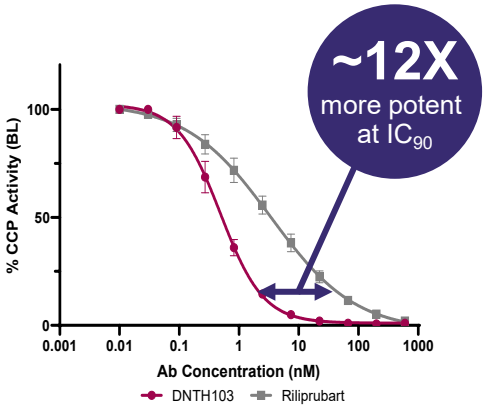
C4 cleavage by human active C1s[2]



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

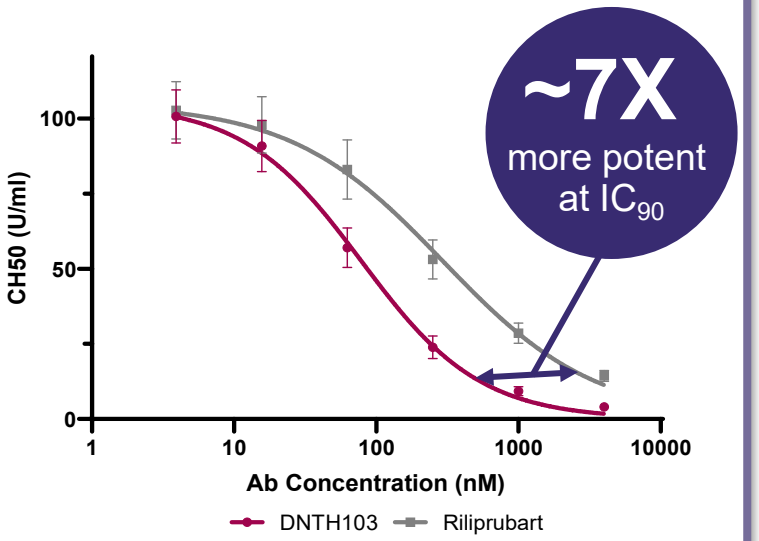
Functional assays of classical pathway inhibition

Wieslab classical pathway Assay in human serum[3]



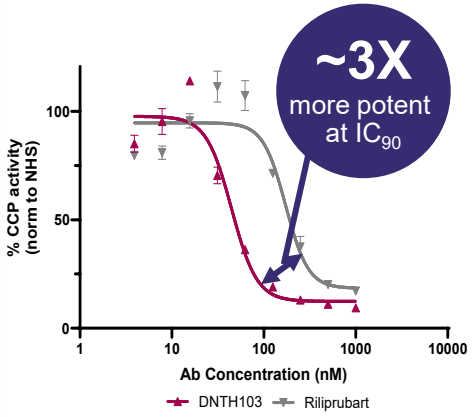
	DNTH103	riliprubart
IC_{90} (ug/mL)	**0.45**	5.4

CH50 assay of RBC lysis in human serum[3]



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

Liposome lysis in human serum[3]



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

DNTH103 consistently outperforms riliprubart in affinity and potency when compared head-to-head across 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 DNTH103 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

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

Neuromuscular indication with high unmet medical need

~5,000 - 10,000
patients in the U.S.

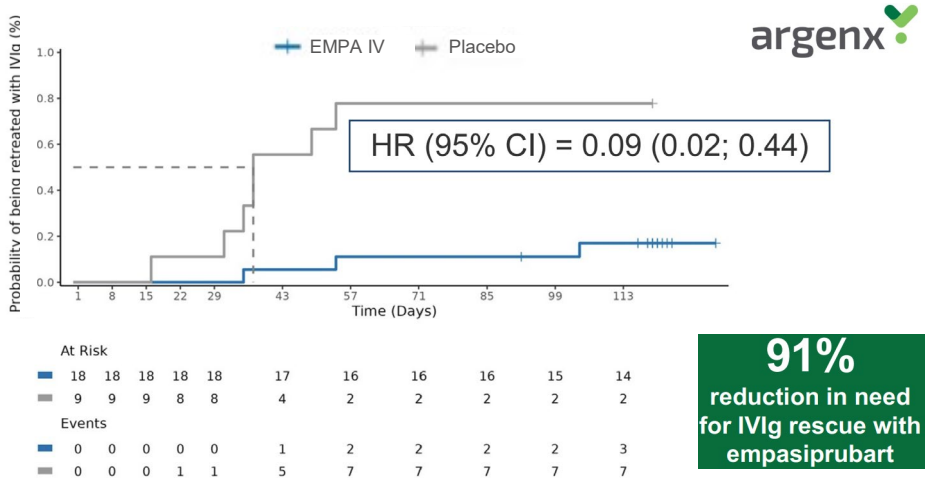
No approved targeted biologic therapies

Evidence supports classical complement role in disease

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

MMN patient sera has been confirmed to activate complement

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



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

Phase 2 trial of DNTH103, 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

DNTH103 has the potential to dominate the MMN market with its best-in-class target profile

Considerations	Empasiprubart (C2)*	DNTH103 (active C1s)*	Key Differentiators of DNTH103
MMN is an IgM and classical pathway driven disease[1]	C2 inhibitor that targets classical and lectin pathways	**Active C1s inhibitor targeting classical pathway only**	Demonstrated potent classical pathway inhibition with target dose achieving >IC90 on CH50 hemolytic assay
Lectin pathway critical to fight against bacterial infections[2]	Targets classical and lectin pathways	**Selective for classical pathway only, leaving lectin and alternative pathways intact**	Preserves key bacterial killing role of lectin pathway[2]
Convenient dosing & administration	I.V. QW or I.V Q2W	**Target dose of 300mg/2mL S.C. Q2W**	More convenient by targeting infrequent, low volume, self-administered S.C. autoinjector

DNTH103 has the potential to be the first-line targeted biologic treatment given its unique combination of classical pathway potency, safety and dosing convenience

* DNTH103 and empasiprubart are investigational agents that are not approved as therapies for MMN or any indication in any jurisdiction worldwide

1 Budding K, et al. (2021). Neurol Neuroimmunol Neuroinflamm.9(1):e1107; Vlam L, et al. (2015). Neurol Neuroimmunol Neuroinflamm. 2015;2(4):e119; 2 Ali YM, et al. (2012). PLoS Pathog 8(7):e1002793

DNTH103 rapidly advancing in three clinical trials with near-term data catalysts on track beginning in 2H'25

Program (Target)	Target Population	2024	2025	2026
DNTH103 (S.C. Active C1s antibody)	Generalized Myasthenia Gravis (gMG) ~60,000 U.S. patients	Initiated Ph. 2 trial Q1'24	Top-line Ph. 2 data 2H'25 ★	
	Multifocal Motor Neuropathy (MMN) ~5,000-10,000 U.S. patients	Initiated Ph. 2 trial Q3'24		Top-line Ph. 2 data 2H'26 ★
	Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) >40,000 U.S. patients	IND cleared in Q4'24 for Ph. 3 trial		Interim Responder Analysis 2H'26 ★

DNTH103 has potential to expand into multiple classical pathway-driven diseases with its best-in-class profile

gMG: https://www.mgregistry.org/, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7033452/#; CIDP: IQVIA Claims Data; MMN: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3983019/

DNTH103 Clinical Development

DNTH103 Phase 1 healthy volunteer study was designed to validate extended half-life, potency and safety

SAD

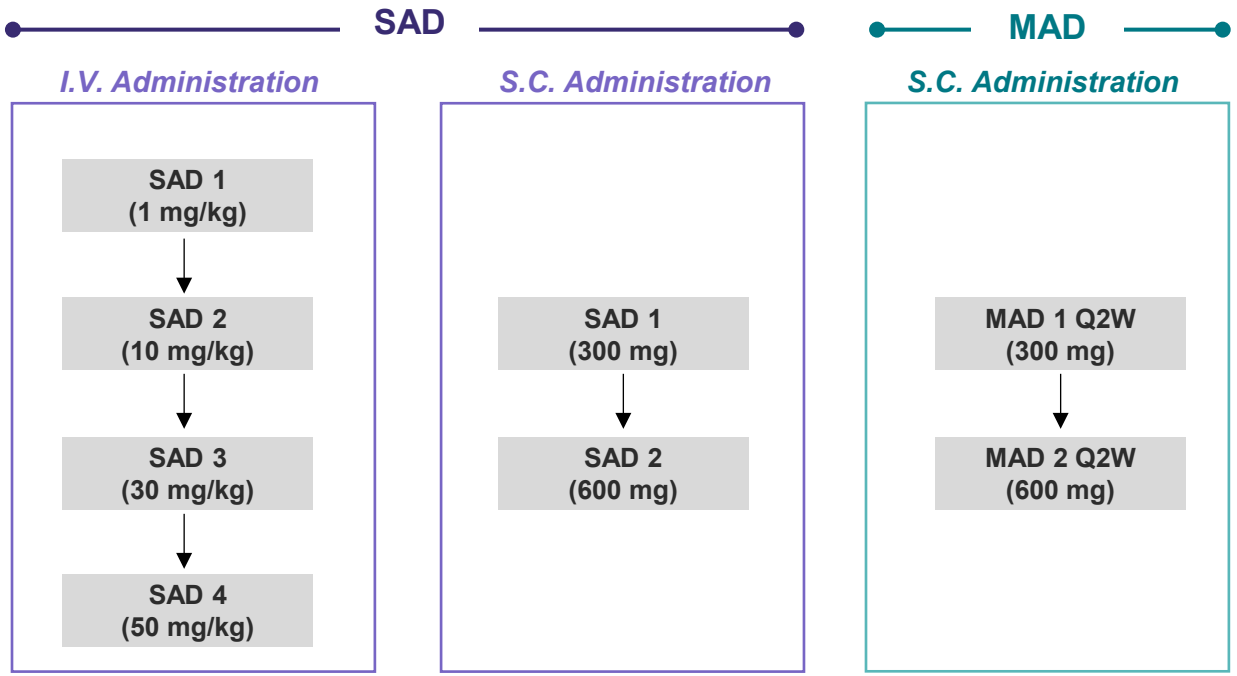
44 HVs enrolled into six cohorts:
- Placebo (N= up to 2)
- Treated (N= up to 6)

MAD

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

Key Parameters

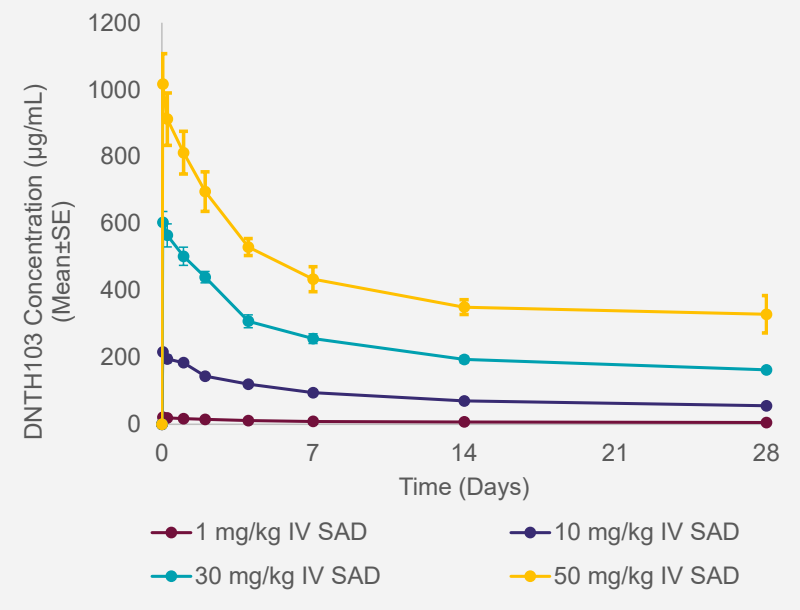
- **Safety, PK, and PD measured by percent classical pathway inhibition quantified in each cohort**



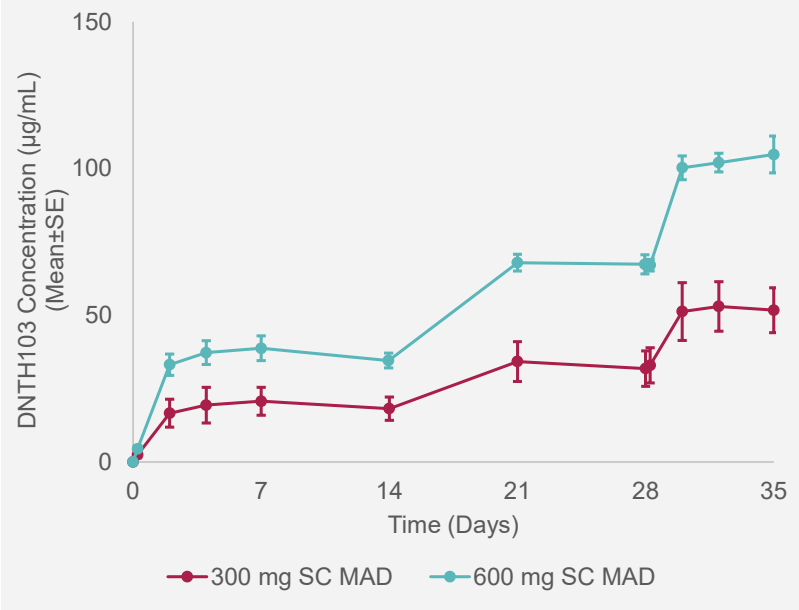
In completed cohorts, 60 healthy volunteers completed dosing as of December 2023

DNTH103 has demonstrated deep and sustained complement inhibition in healthy volunteers

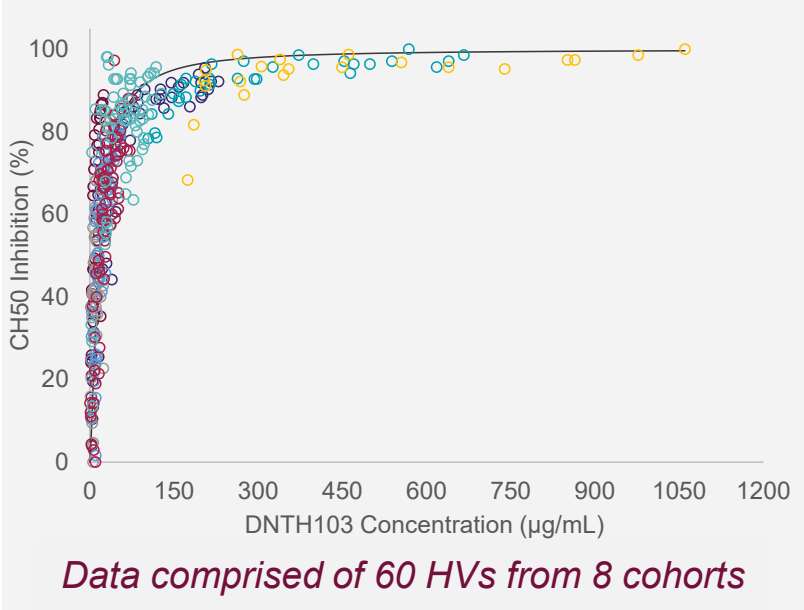
I.V. SAD:
Linear PK with Exposure Proportional Across Doses



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

DNTH103 demonstrated a ~60-day half-life and IC90 of 87 µg/mL

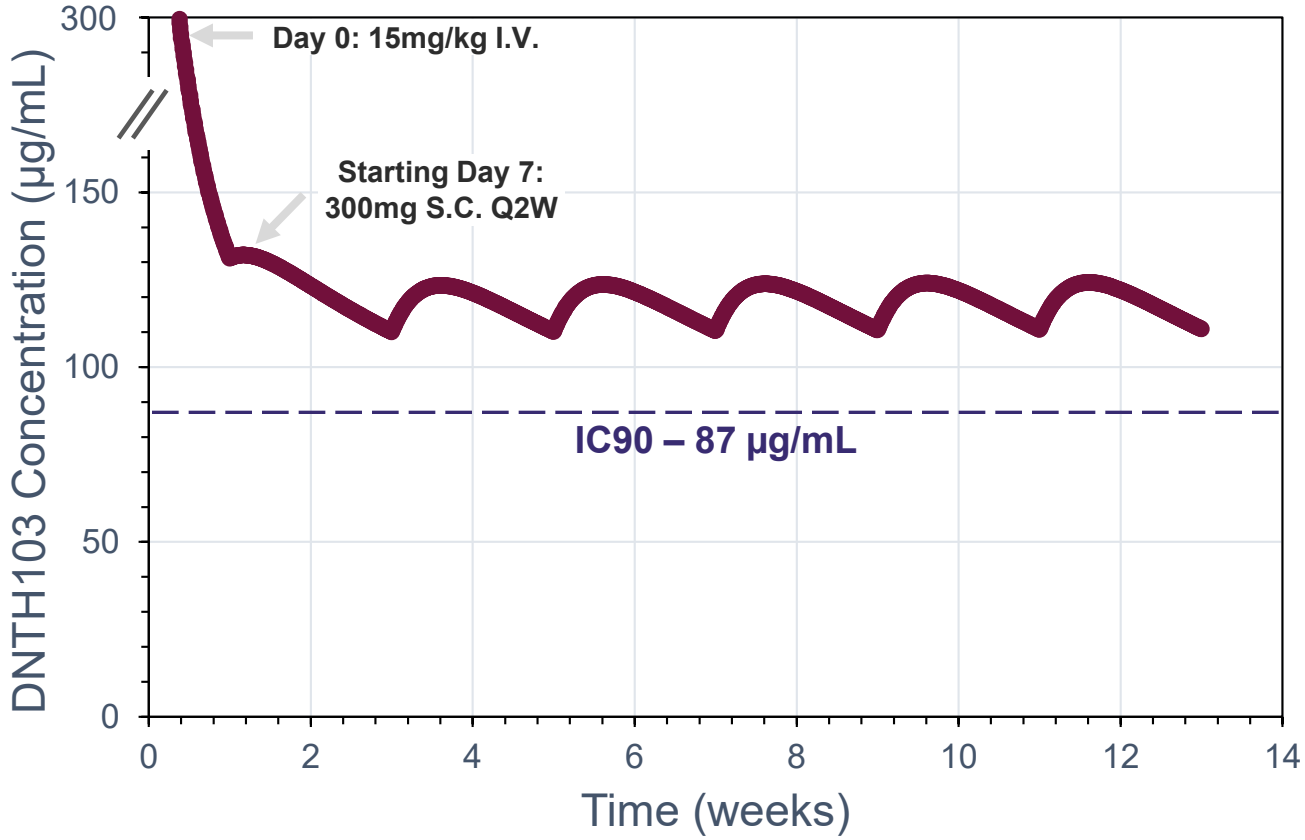
DNTH103 Phase 1 data confirms potent inhibition of the classical pathway as a Q2W S.C. injection

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 potent inhibition with infrequent S.C. dosing

DNTH103 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 and 600mg 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 S.C. SAD reported vomiting on Day 1, which resolved on same day

1 Highest dose to be used in Phase 2 trials is 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

gMG Phase 2 top-line data on-track for this year in 2H'25

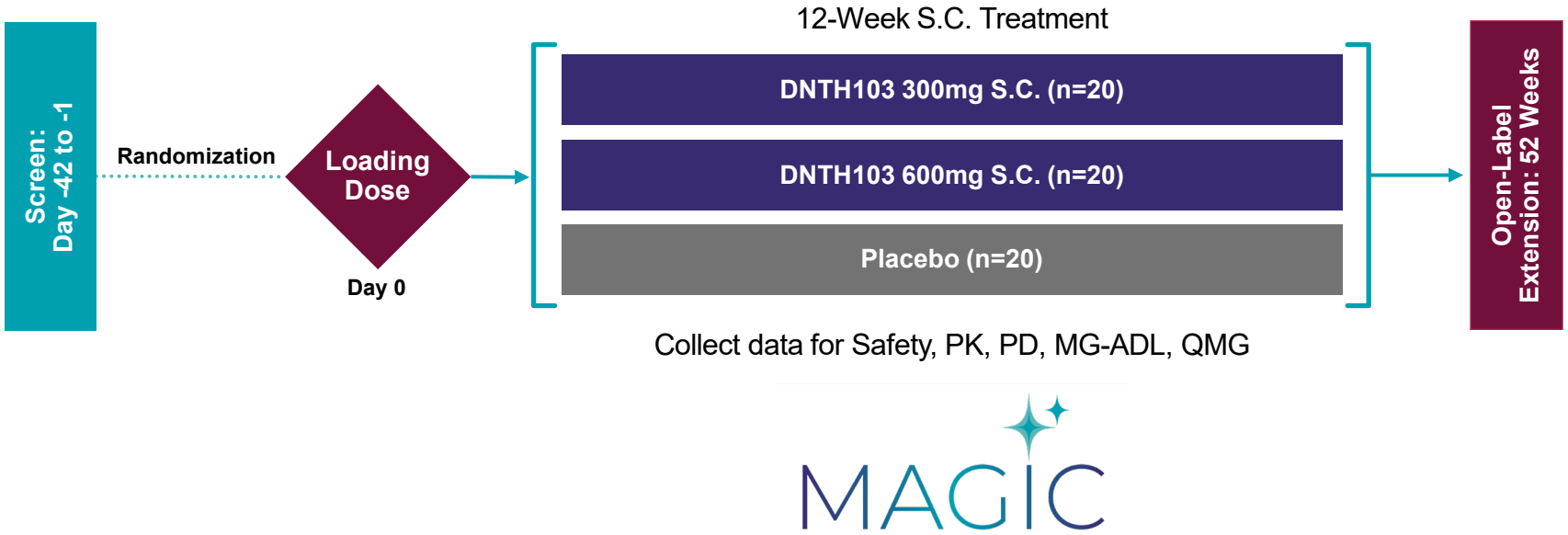
A global, multicenter, randomized, double-blind, placebo-controlled study to evaluate the safety, efficacy, and PK / PD of DNTH103 administered S.C. following initial loading dose

Highlights

- **Design:** 60 male and female subjects randomized to receive either DNTH103 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 or 600mg[1] S.C. Q2W starting Day 7

Endpoints

- **Primary:** Safety
- **Secondary:** Efficacy (MG-ADL and QMG)



If successful, path to BLA expected to require only one additional Phase 3 of similar design with more patients

300mg and 600mg S.C. Q2W dosing surpasses IC90 (87 µg/mL) and IC95 (149 µg/mL), respectively
MaGic Trial: https://clinicaltrials.gov/study/NCT06282159

CIDP interim responder analysis anticipated 2H'26

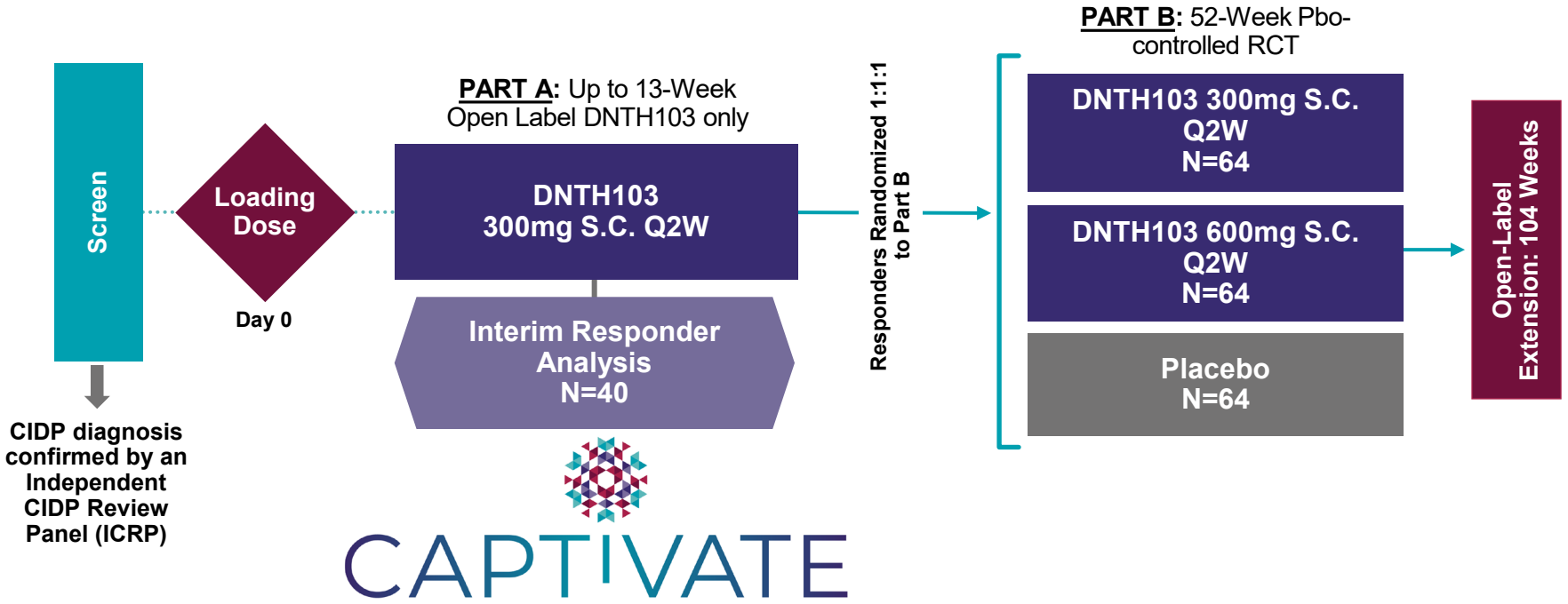
CIDP Phase 3 trial designed as a two-part, randomized withdrawal, double-blind, placebo-controlled trial to evaluate the efficacy and safety of DNTH103 300mg and 600mg administered S.C. Q2W

Highlights

- **Design:** All subjects receive DNTH103 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 S.C. Q2W in Part A; followed by 300mg or 600mg 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 trial** designed to support BLA filing 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 S.C. Q2W dosing of DNTH103 in Part A
- **Only responders from Part A** randomized into the double-blind, placebo-controlled Part B

300mg and 600mg S.C. Q2W dosing surpasses IC90 (87 µg/mL) and IC95 (149 µg/mL), respectively

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

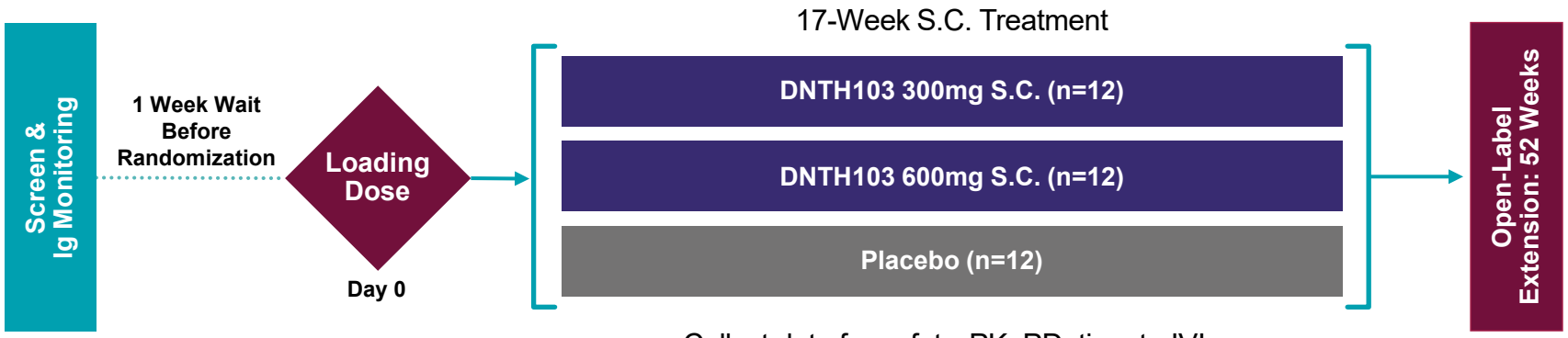
A global, multicenter, randomized, double-blind, placebo-controlled study to evaluate the safety, efficacy, and PK / PD of DNTH103 administered S.C. following initial loading dose

Highlights

- **Design:** 36 participants randomized to receive either DNTH103 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 or 600mg 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)



Top-line data expected in 2H’26

300mg and 600mg S.C. Q2W dosing surpasses IC90 (87 µg/mL) and IC95 (149 µg/mL), respectively
MoMeNtum Trial: https://clinicaltrials.gov/study/NCT06537999

Corporate

DNTH103 targets best-in-class properties by exploiting validated classical pathway inhibition and C1s biology

Potent classical pathway inhibition

Picomolar binding affinity and >IC90 on CH50 at target dose

Convenient S.C. self-administration

Targeting aC1s supports low-volume, 300mg / 2mL autoinjector

Infrequent Q2W dosing

Extended ~60-day half-life from Ph. 1 through validated YTE technology

Differentiated safety profile

Aims to lower risk of infection and avoid FDA boxed warning/REMS

Pipeline-in-a-product potential

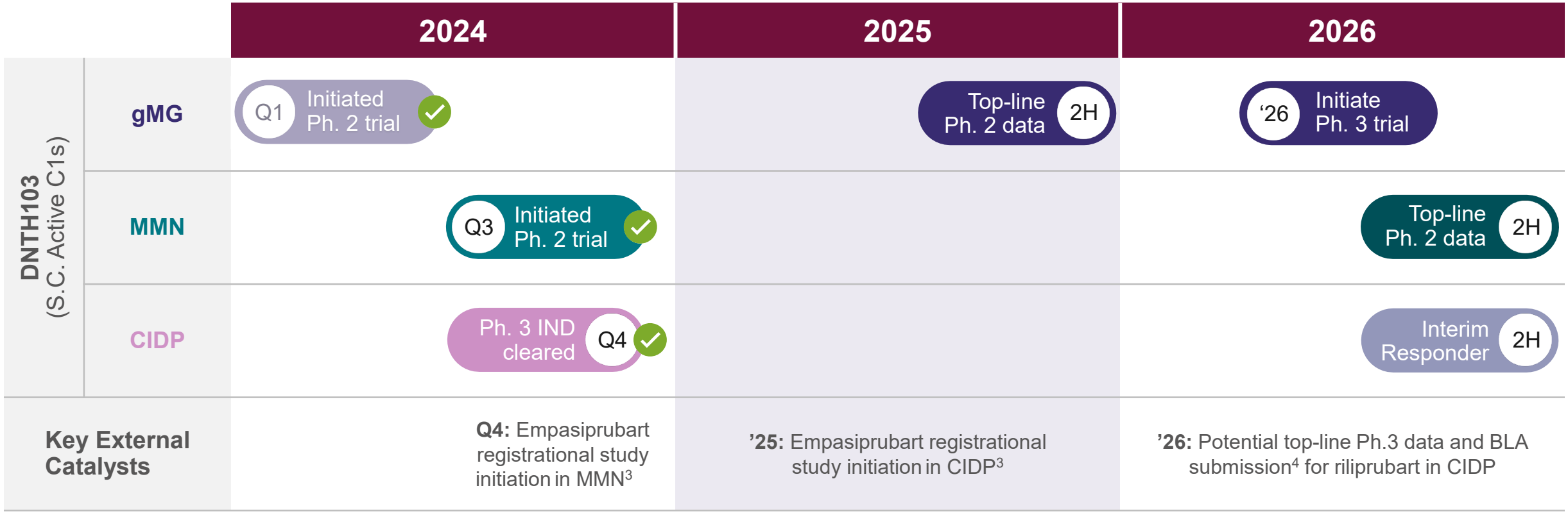
Currently in Ph. 2 trials in gMG and MMN and a pivotal Ph. 3 CIDP trial

Robust IP protection

Composition of matter and method of use patents expected to expire no earlier than 2043

DNTH103 has the potential to offer best-in-class combination of efficacy and safety with convenient S.C. Q2W self-administration

Three clinical trials for DNTH103 ahead of transformative Phase 2 gMG readout



Strong balance sheet with ~$343M[1] of cash & runway into the second half of 2027
~34.3M shares outstanding[2]

1 Includes unaudited cash, cash equivalents and investments as of 9/30/24
2 Shares outstanding on a pro forma basis, which assumes the exercise of all outstanding pre-funded warrants
3 https://argenx.com/news/2024/argenx-reports-third-quarter-2024-financial-results-and-provides-business-update.html
4 https://www.sanofi.com/assets/dotcom/content-app/events/investor-presentation/2023/r-and-d-day-2023/Presentation.pdf#page=91

Appendix

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

SENIOR MANAGEMENT

Marino Garcia
President & CEO

Simrat Randhawa, M.D.
Chief Medical Officer

Ryan Savitz
Chief Financial Officer & Chief Business Officer

Jeffrey Stavenhagen, Ph.D.
Chief Scientific Officer

Adam Veness, Esq.
General Counsel

Kristina Maximenko
Chief People Officer

Rivka Gluck, R.N.
Head of Clinical Development, Operations

Debra Segal
Head of Regulatory Affairs

Edward Carr
Chief Accounting Officer

Jud Taylor
Head of Technical Operations

Jennifer Davis Ruff
Head of Investor Relations & Corporate Affairs

Ronny Hashmonay, M.D.
Head of Medical Affairs

Polly Hanff
Head of Quality

Scott Nogi
Head of Business Operations

Select Experience Includes:

BOARD OF DIRECTORS

Alison Lawton
Chair of the Board
Board Member, ProQR and X4, Prior Chair of Board, Magenta

Tomas Kiselak
Managing Member, Fairmount

Anne McGeorge
Board Member, The Oncology Institute, Board Member, Be the Match

Lonnie Moulder
Managing Member, Tellus BioVentures, Founder, CEO & Chairman, Zenas BioPharma

Steven Romano, M.D.
EVP, Chief Research & Development Officer, Silence Therapeutics, Inc.

Paula Soteropoulos
Venture Partner, 5AM Ventures

Jonathan Violin, Ph.D.
Venture Partner, Fairmount, Co-founder of Dianthus, Board Member, Astria Therapeutics, and former President/CEO of Viridian Therapeutics

Marino Garcia
President & CEO, Dianthus

Select Auto-Immune Drugs Developed by Dianthus Team

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

1

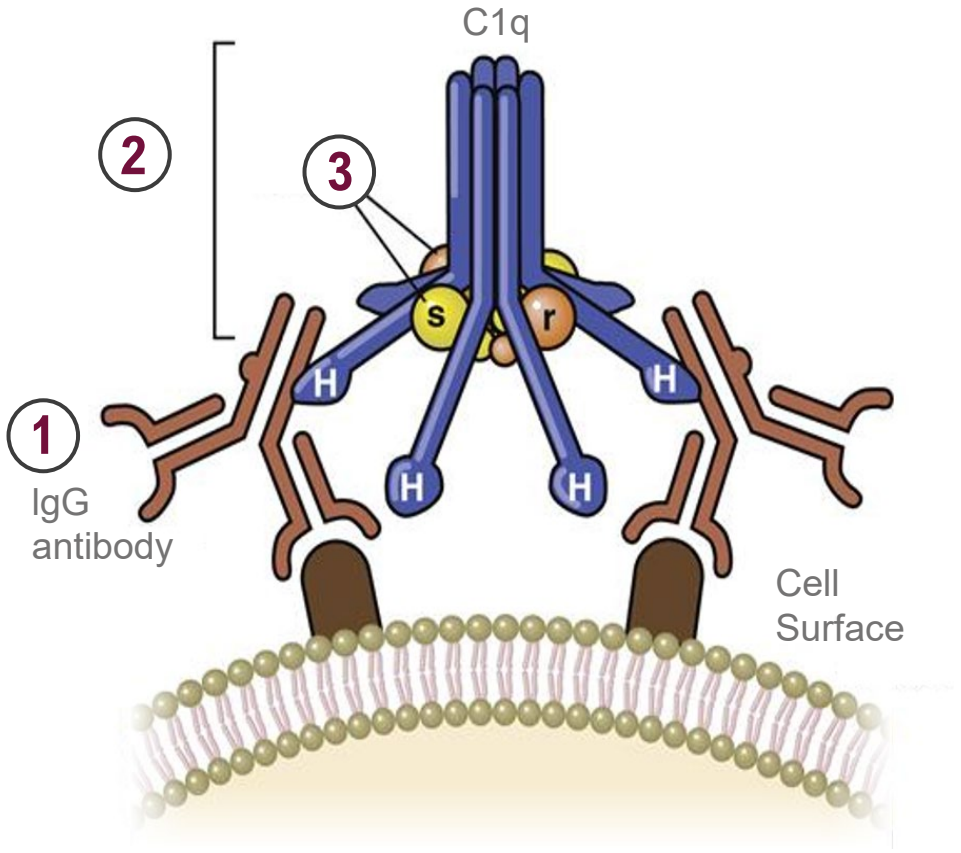
Classical pathway
The only pathway activated by the presence of IgG and IgM, which bind to the **C1 complex**

2

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

3

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



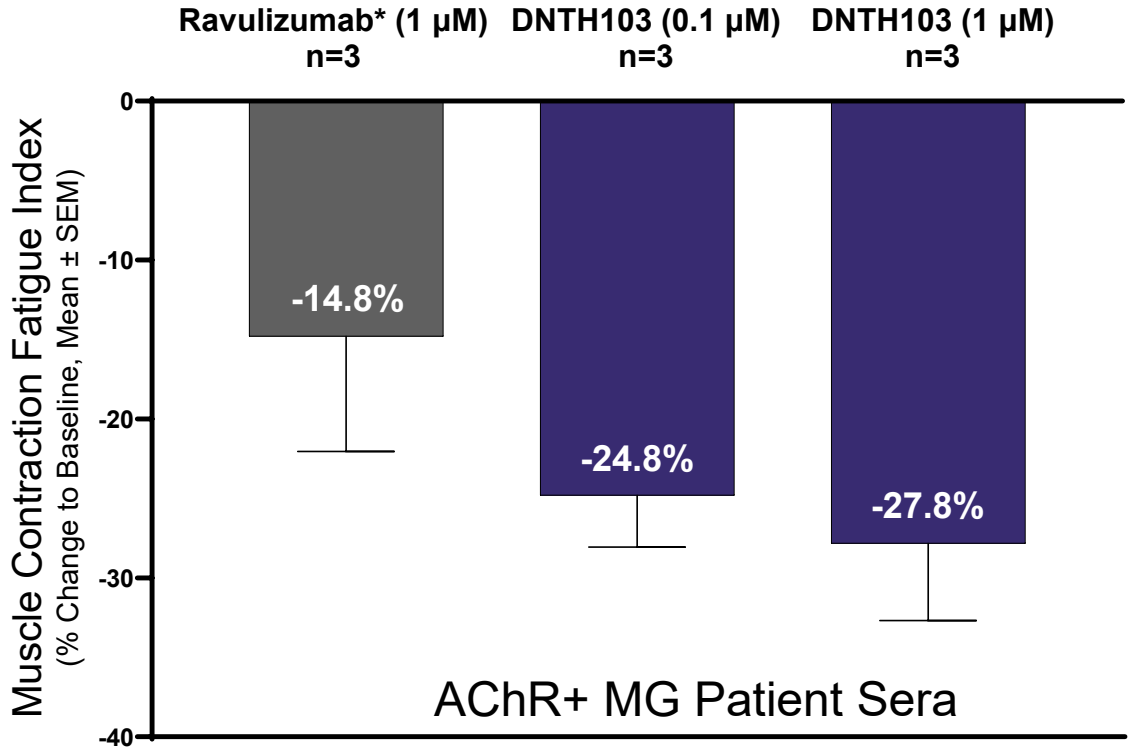
C1s is the only target of the C1 complex with an FDA approved therapy
Enjaymo®, FDA approved in 2022 for CAD, is a C1s inhibitor but is not selective to the active form and dosed I.V. at 6,500-7,500mg every two weeks

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

Enjaymo® information sourced from prescribing information

DNTH103 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**

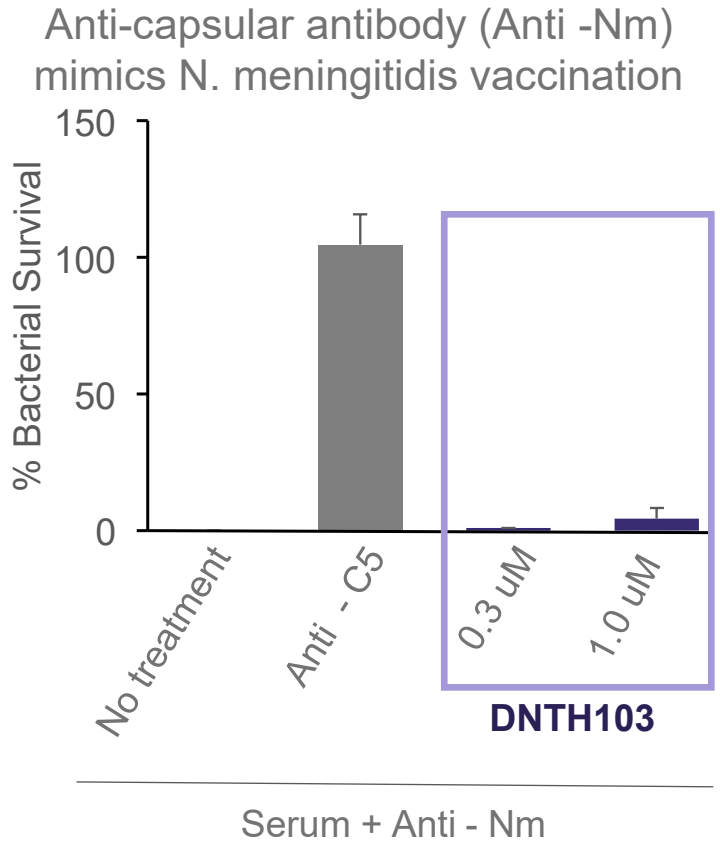


Results provide further scientific rationale for DNTH103 in gMG

1 https://pubmed.ncbi.nlm.nih.gov/34881241/, 2 - https://pubmed.ncbi.nlm.nih.gov/31846349/, 3 - https://pubmed.ncbi.nlm.nih.gov/30867827/
* Engineered using patent sequence

DNTH103 *in vitro* study demonstrates 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 differentiated safety profile of 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

C5 inhibitor Ultomiris carries FDA Boxed Warning and REMS requirement

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)

PATIENT SAFETY CARD

Important Safety Information for Patients Taking ULTOMIRIS (ravulizumab-cwvz) or SOLIRIS (eculizumab)

ULTOMIRIS® and SOLIRIS® can increase your chance of getting **serious meningococcal infections. These infections may quickly become life-threatening or cause death if not recognized and treated early.** If you experience any of the following signs and symptoms of serious meningococcal infection, you should immediately call your healthcare provider or seek emergency medical care, preferably in a major emergency medical care center:

- fever
- fever and a rash
- fever with high heart rate
- headache with nausea or vomiting
- headache and fever
- headache with stiff neck or stiff back
- confusion
- eyes sensitive to light
- muscle aches with flu-like symptoms

Get emergency medical care right away if you have any of these signs and symptoms and show this card to any healthcare provider who treats you.

Your risk of meningococcal infection may continue for several months after your last dose of ULTOMIRIS or SOLIRIS.

For **ULTOMIRIS**, keep this card with you at all times during your treatment and for 8 months after your last dose.

For **SOLIRIS**, keep this card with you at all times during your treatment and for 3 months after your last dose.

ULTOMIRIS (ravulizumab-cwvz) SOLIRIS (eculizumab)

PATIENT SAFETY CARD

Information for the Treating Healthcare Provider

This patient has been prescribed ULTOMIRIS (ravulizumab-cwvz) or SOLIRIS (eculizumab) therapy, which increases the patient's susceptibility to meningococcal infections (*Neisseria meningitidis*) or other general infections.

- Meningococcal infections may become rapidly life-threatening or fatal if not recognized and treated early.
- **Closely monitor patients for early signs and symptoms of serious meningococcal infections and evaluate immediately if infection is suspected. Promptly treat known infections.**
- Contact the healthcare provider who prescribed ULTOMIRIS or SOLIRIS (listed below) as soon as possible if the patient has signs or symptoms of serious meningococcal infection.

For more information about ULTOMIRIS or SOLIRIS, please refer to the Prescribing Information. Report adverse events suggestive of serious meningococcal infections at 1-844-259-6783.

Patients receiving ULTOMIRIS or SOLIRIS should carry this card at all times. ULTOMIRIS patients should carry for 8 months after the last dose of treatment and SOLIRIS patients should carry for 3 months after the last dose of treatment. Show this card to any healthcare provider involved in your health care.

Patient Name _____

Prescriber Name _____

Prescriber Phone _____

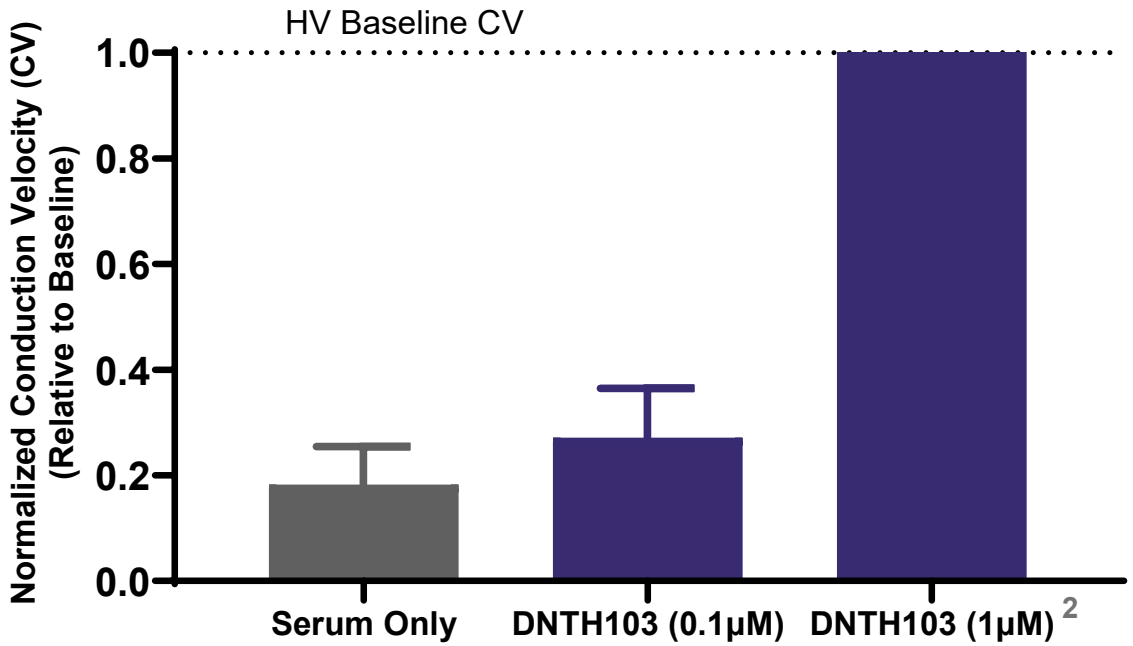
Phone: 1-888-765-4747 www.UltSolREMS.com Fax: 1-866-750-0481

ULTOMIRIS (ravulizumab-cwvz) SOLIRIS (eculizumab)

Source: ULTOMIRIS prescribing information & https://ultomirishcp.com/-/media/Ultomiris_NMOSD_hcp/full-site/pdfs/nmosd-ultomiris-rems-patient-safety-card

DNTH103 restores neuronal conduction velocity in an *in vitro* CIDP model

- **Serum from 3 CIDP patients** was evaluated in a validated, 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