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Welcome & Opening Remarks

Beth DelGiacco /// Vice President, Corporate Communications & Investor Relations

Forward Looking Statements

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Certain statements contained in this presentation, other than present and historical facts and conditions independently verifiable at the date hereof, may constitute forward-looking statements. Certain statements contained in this presentation, other than present and historical facts and conditions independently verifiable at the date hereof, may constitute forward-looking statements. These forward-looking statements can be identified by the use of forward-looking terminology, including the terms “advance,” “broaden,” “build,” “develop,” “expand,” “grow,” “predict,” “potential,” “reach,” “start,” “seek,” “vision,” and “will,” and include statements argenx makes regarding the potential of its new pipeline candidates, including ARGX-213 and ARGX-121; future phases of ongoing product candidate development; the anticipated timing of argenx’s clinical trials, including the anticipated timing of the end of the Phase 2 ARDA clinical trial and the initiation of the Phase 3 clinical trial for empasiprubart in MMN, the anticipated timing of the Phase 2 EMPACIFIC clinical trial, the anticipated timing of the analysis for the Phase 2 ALKIVIA clinical trial, the anticipated timing of the initiation of the Phase 3 clinical trial for efgartigimod in SjD, the anticipated timing of the initiation of the Phase 1b and Phase 2a clinical trials for ARGX-119 in CMS and ALS, respectively, the anticipated timing of the initiation of the Phase 1 clinical trial for ARGX-213, and the anticipated timing of the initiation of the Phase 1 clinical trial for ARGX-121; the timing and outcome of regulatory filings and regulatory approvals, including the anticipated timing of the clinical trial applications for ARGX-121 and ARGX-213; the number of patients that its products will reach in 2030; the size and growth of the market for its products, including the growing MG opportunity, the in-market opportunity when evaluating ocular and seronegative MG, and the MMN, SjD and TED opportunities; its future position as a market leader among branded biologics; its thought leadership in the scientific community; its trajectory to be a leading autoimmune franchise; the outcome and findings of its various studies, including the findings of the iMMersioN clinical trial; its goals and visions for its future advancement, including its vision for 2025 and 2030; its capabilities to scale; and the number of its products and the number of indications those products will have. By their nature, forward-looking statements involve risks and uncertainties and readers are cautioned that any such forward-looking statements are not guarantees of future performance. argenx’s actual results may differ materially from those predicted by the forward-looking statements as a result of various important factors, including the results of argenx's clinical trials; expectations regarding the inherent uncertainties associated with the development of novel drug therapies; preclinical and clinical trial and product development activities and regulatory approval requirements in products and product candidates; the acceptance of argenx's products and product candidates by patients as safe, effective and cost-effective; the impact of governmental laws and regulations on our business; disruptions caused on our reliance of third parties suppliers, service provides and manufacturing; inflation and deflation and the corresponding fluctuations in interest rates; and regional instability and conflicts. A further list and description of these risks, uncertainties and other risks can be found in argenx’s U.S. Securities and Exchange Commission (the “SEC”) filings and reports, including in argenx’s most recent annual report on Form 20-F filed with the SEC as well as subsequent filings and reports filed by argenx with the SEC. Given these uncertainties, the reader is advised not to place any undue reliance on such forward-looking statements. These forward-looking statements speak only as of the date of publication of this document. argenx undertakes no obligation to publicly update or revise the information in this presentation, including any forward-looking statements, except as may be required by law.

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Agenda

Welcome and Opening Remarks	·····	Beth DelGiacco
argenx Vision 2030	·····	Tim Van Hauwermeiren
Immunology Innovation	·····	Peter Ulrichts, Karen Silence
Clinical Development	·····	Luc Truyen
Myositis	·····	Leentje De Ceuninck
Sjögren's Disease	·····	Julie Jacobs
Sjögren's Disease KOL Panel	·····	Julie Jacobs, Prof. Simon Bowman (Moderated by Luc Truyen)
Q&A Session 1	·····	argenx Management Team
	BREAK	
Phase 2 ARDA Study (MMN)	·····	Inge Van de Walle, Jeff Guptill
MMN KOL Panel	·····	Dr. Patrick Kwon, Jeff Guptill (Moderated by Luc Truyen)
Sustainable Commercial Engine	·····	Karen Massey
Q&A Session 2	·····	argenx Management Team

argenx Leadership Here Today

Tim Van Hauwermeiren
Chief Executive Officer

Karen Silence Ph.D.
Head Preclinical Product Development

Beth DelGiacco
Vice President, Corporate Communications Investor Relations

Luc Truyen M.D., Ph.D.
Chief Medical Officer

Peter Ulrichts Ph.D.
Chief Scientific Officer

Karen Massey
Chief Operating Officer

Leentje DeCeuninck Ph.D.
Senior Clinical Scientist

Jeff Guptill, M.D.
Neuromuscular Franchise Lead, Clinical Development

Julie Jacobs Ph.D.
Principal Scientist

Inge Van de Walle Ph.D.
Research Fellow

Thought Leaders Here Today

Simon Bowman, Ph.D., M.B.B.S., F.R.C.P.
Institute of Inflammation and Ageing,
University of Birmingham

Patrick Kwon, M.D.
Clinical Associate Professor, Neurology,
New York University Grossman School of Medicine

Key Themes For Today

- Our innovation model
- Leadership in FcRn
- Expansion of our immunology pipeline
- Setting a new standard in MG and CIDP
- Next wave of efgartigimod indications
- Building weight behind empasiprubart
- Vision 2030 - path to 50,000 patients

Vision 2030

Tim Van Hauwermeiren /// Chief Executive Officer

Vision 2030

5 New Molecules in Phase 3

10 Labeled Indications

50k Patients on Treatment

COMMITMENT TO OUR TRANSFORMATION MISSION

- Continuous Pipeline of Innovation
- Leadership in FcRn
- Disciplined Scaling

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Est. 2008

argenx

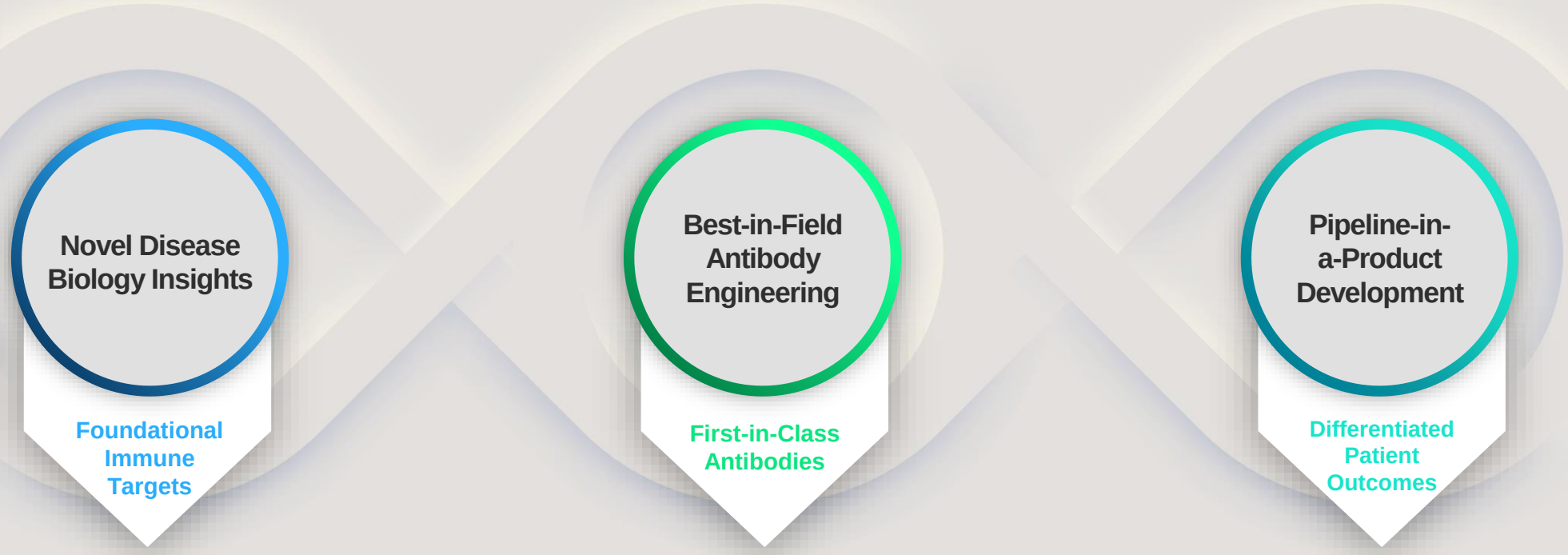
Entrepreneurial spirit – calculated risk based on data

Immunology innovation through model of **co-creation**

Execution excellence

Our Understanding of Human Immunology is Growing Exponentially

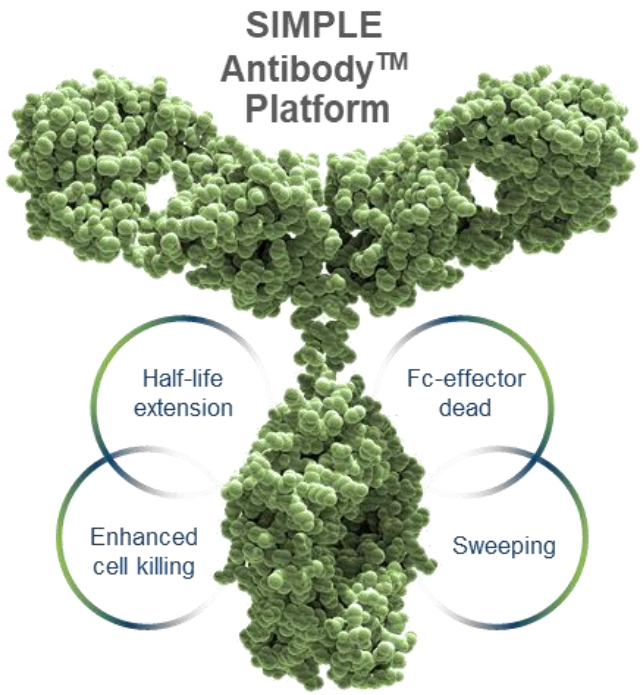
Our Innovation Playbook

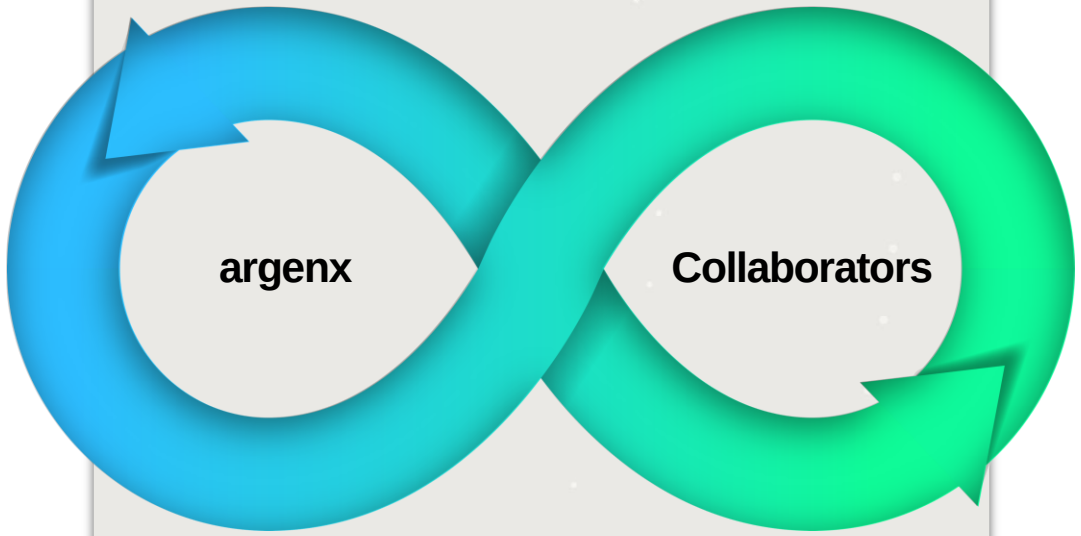


Co-Creation is Our Innovation Formula

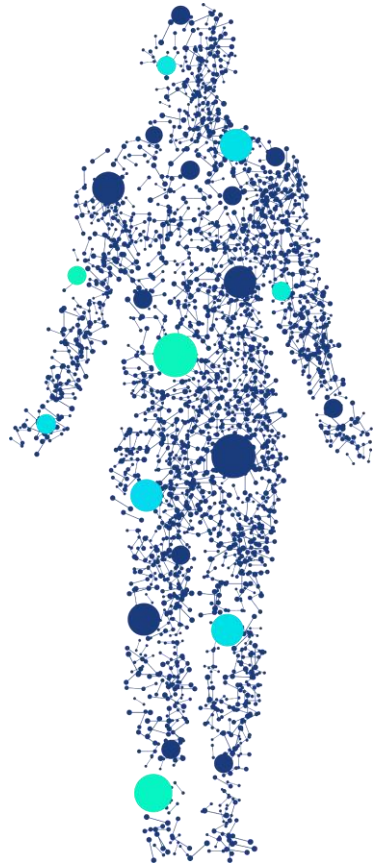
WORLD CLASS ANTIBODY ENGINEERING CAPABILITY

V-REGION CAPABILITIES





DISEASE BIOLOGY INSIGHTS



IL6

CD70

FcRn

GARP

Galectin-10

MuSK

CMET

IL22R

ApoC3

C2

IgA

‘ARGX-220’

Our Innovation Model Has a Strong Track Record

8/12 demonstrated human POC

9 first-in-class targets

5 partnered

Broad applicability **across 35+ indications**

Innovation Through Co-Creation Exists Across argenx

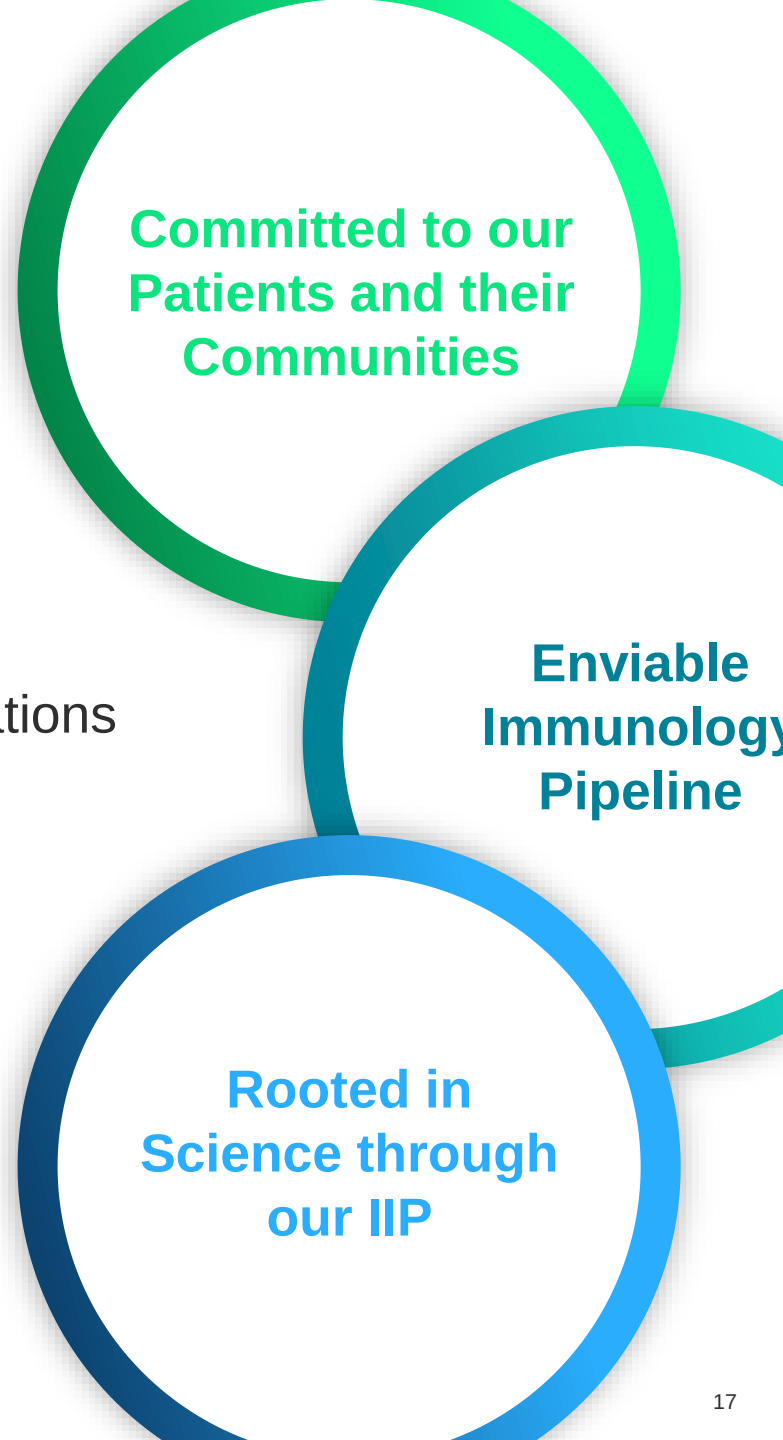
DISCOVERY

DEVELOPMENT

COMMERCIAL

Successful Execution of Our Vision 2025

- Growing autoimmune market
- Efgartigimod available globally
- Vibrant franchises
- Efgartigimod in development in 15 indications
- ARGX-117 in late-stage trials
- Proof-of-concept in ARGX-119
- New asset each year from IIP



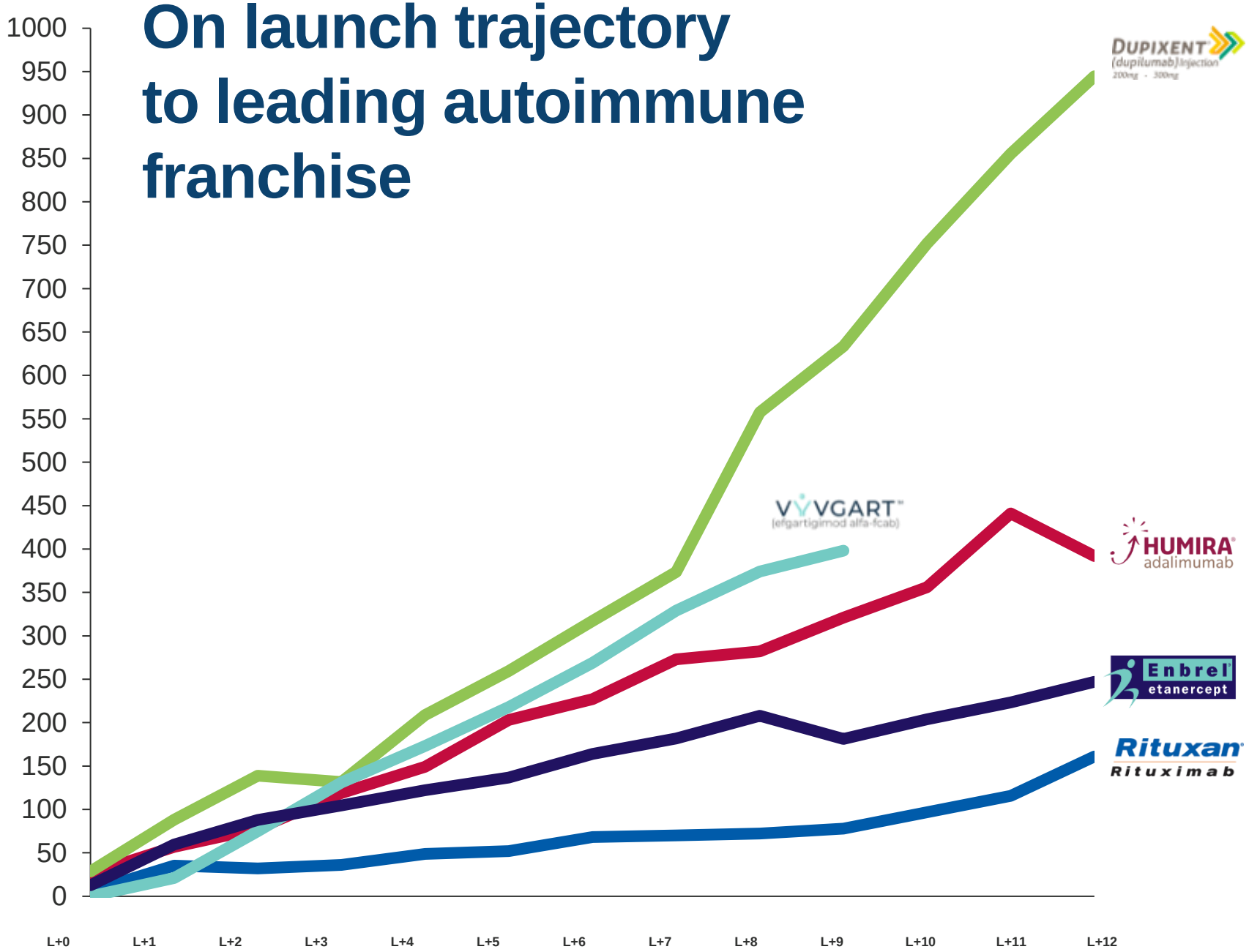
VYVGART is a Global Blockbuster

VYVGART generated >$1B in second year of launch

Approved in 3 indications globally

Leading market share among MG branded biologics

On launch trajectory to leading autoimmune franchise



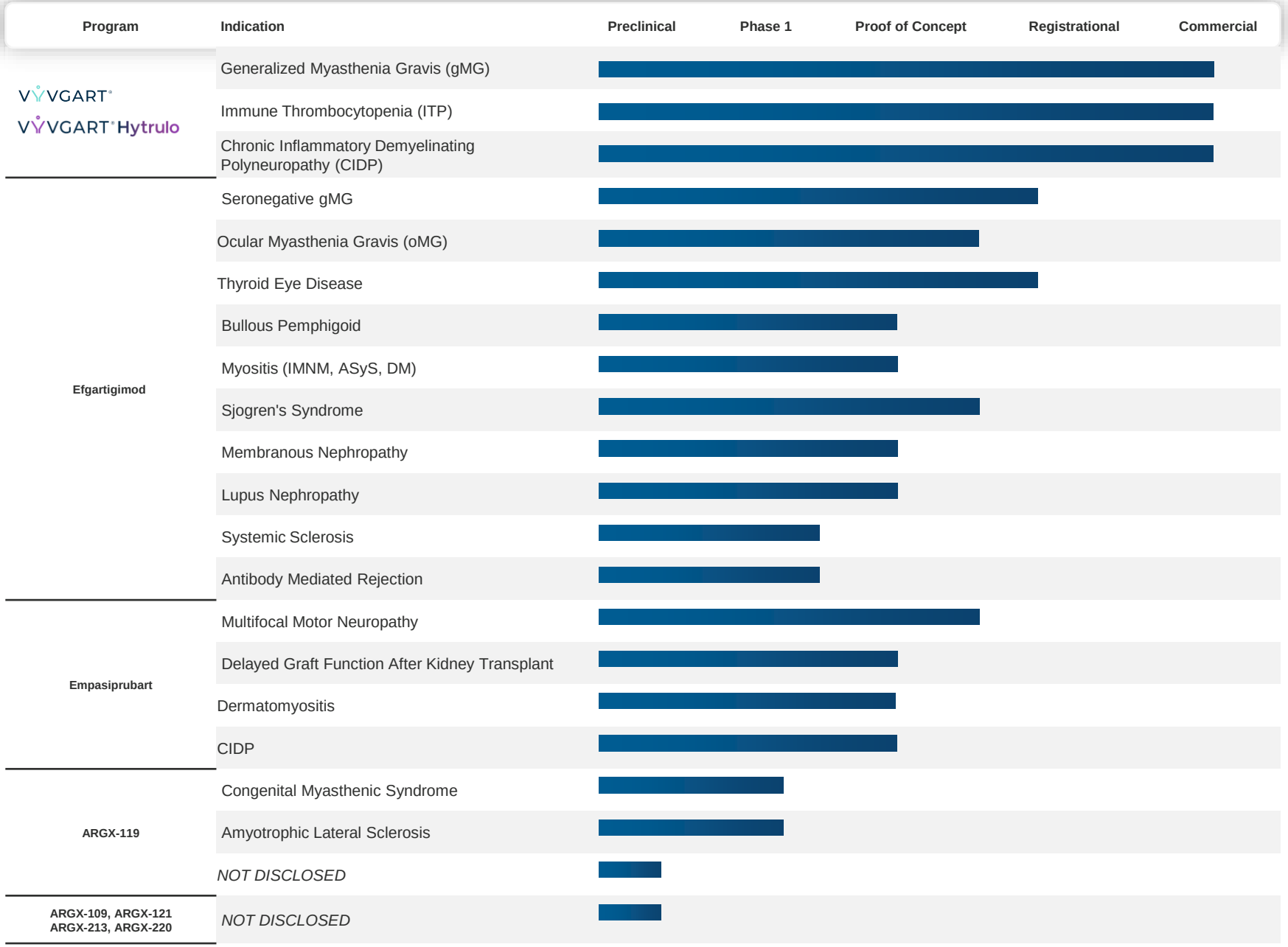
Robust Pipeline of Multi-Indication Assets

Efgartigimod in 15 indications

Empasiprubart in 4 indications

ARGX-119 in CMS and ALS

4 new INDs by end of 2025

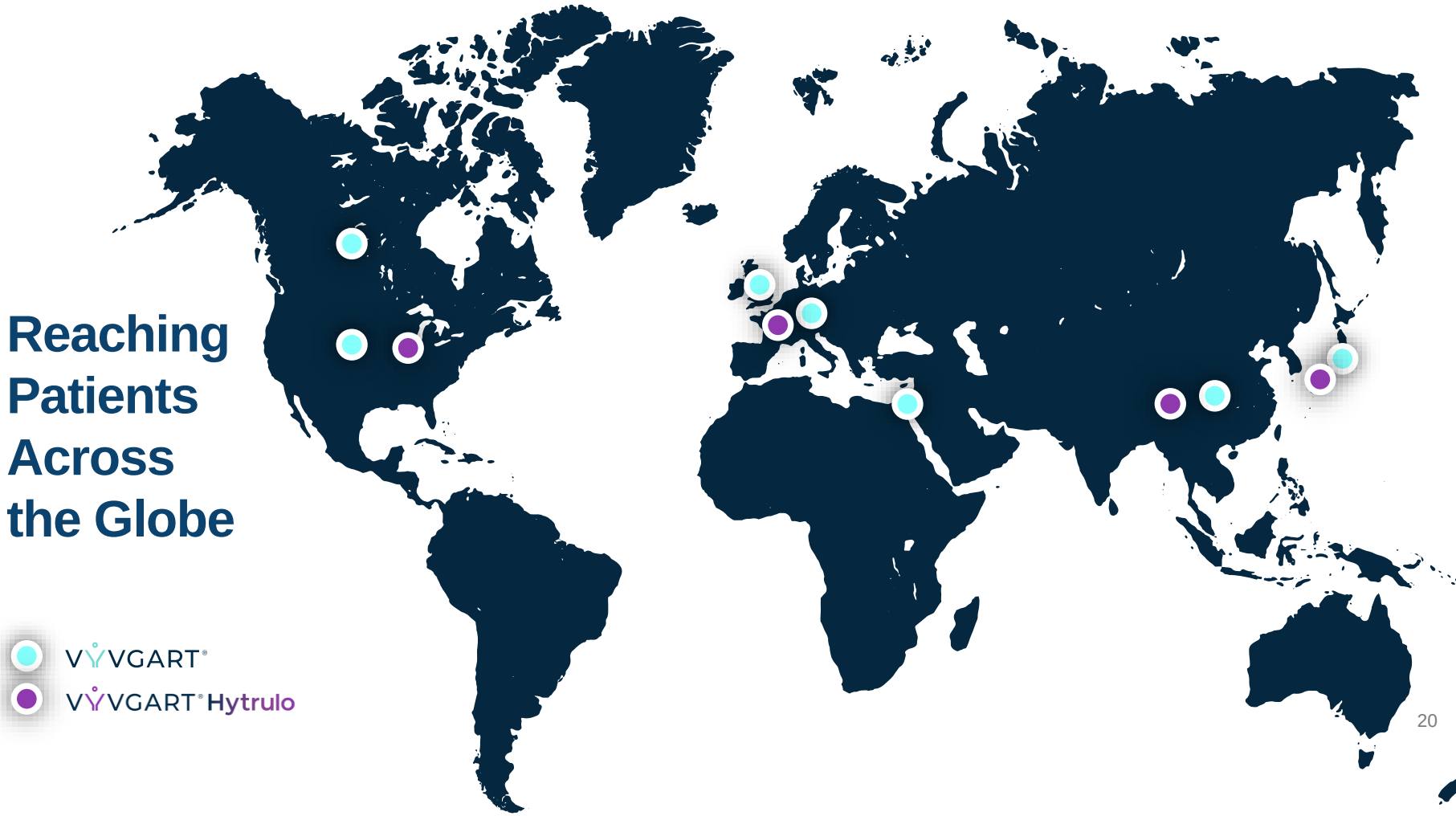


Reaching Patients Globally with VYVGART Franchise

>10,000 patients on treatment[1]

VYVGART and VYVGART Hytrulo[2] approved across 3 continents within one calendar year

Reaching Patients Across the Globe



1. Patients on treatment globally as of 1Q 2024
2. VYVGART Hytrulo is marketed as VYVGART-SC in Europe and VYVDURA® in Japan

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Staying True to our Scientific Roots

Robust patent portfolio

From IIP to marketplace, science is our common language

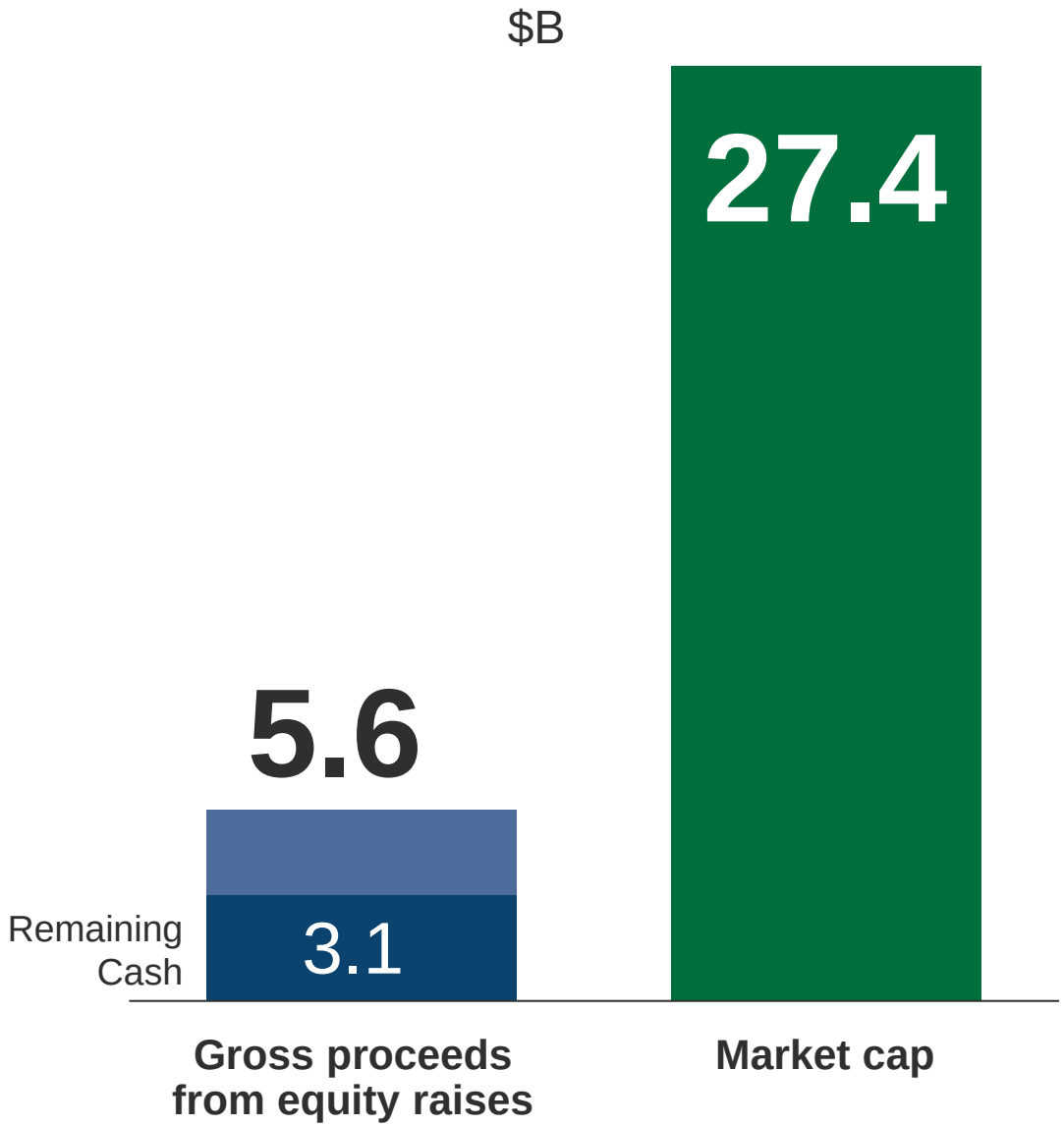
Advanced our scientific expertise with peer reviewed publications in top medical journals

Creating Superior Shareholder Value on our Path to Self-Sustainability

Rapid transition to sustainable company

Disciplined scaling

Total Shareholder Return since IPO in 2014



argenx TSR
~5,000%

Vision 2030

5 New Molecules in Phase 3

10 Labeled Indications

50K Patients on Treatment

COMMITMENT TO OUR TRANSFORMATION MISSION

- Continuous Pipeline of Innovation
- Leadership in FcRn
- Disciplined Scaling

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Our Horizons

ARGX-213
(Anti-FcRn)

ARGX-109
(Anti-IL-6)

New Molecule

ARGX-121
(Anti-IgA)

New Molecule

ARGX-220

New Molecule

ARGX-XXX

5 Additional Molecules in Phase 3

iip immunology innovation program

EARLY PIPELINE

LATE PIPELINE

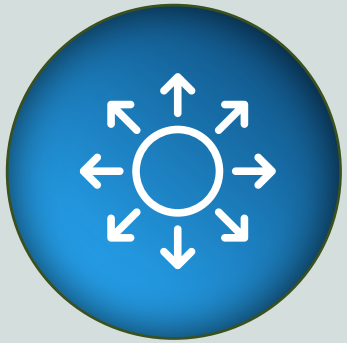
10 Labeled Indications

In-Market Expansion

- gMG ✓
- Sn MG
- ITP ✓
- Ocular MG
- CIDP ✓

Next Wave of Potential Launches

- TED
- DM (empa)
- SjD
- MMN (empa)
- Myositis [3]
- CIDP (empa)
- CMS (119)
- BP



✓ Currently approved indications ● VYVGART ● Empasiprubart ● ARGX-119

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Innovation has no meaning unless it reaches patients and provides real benefit

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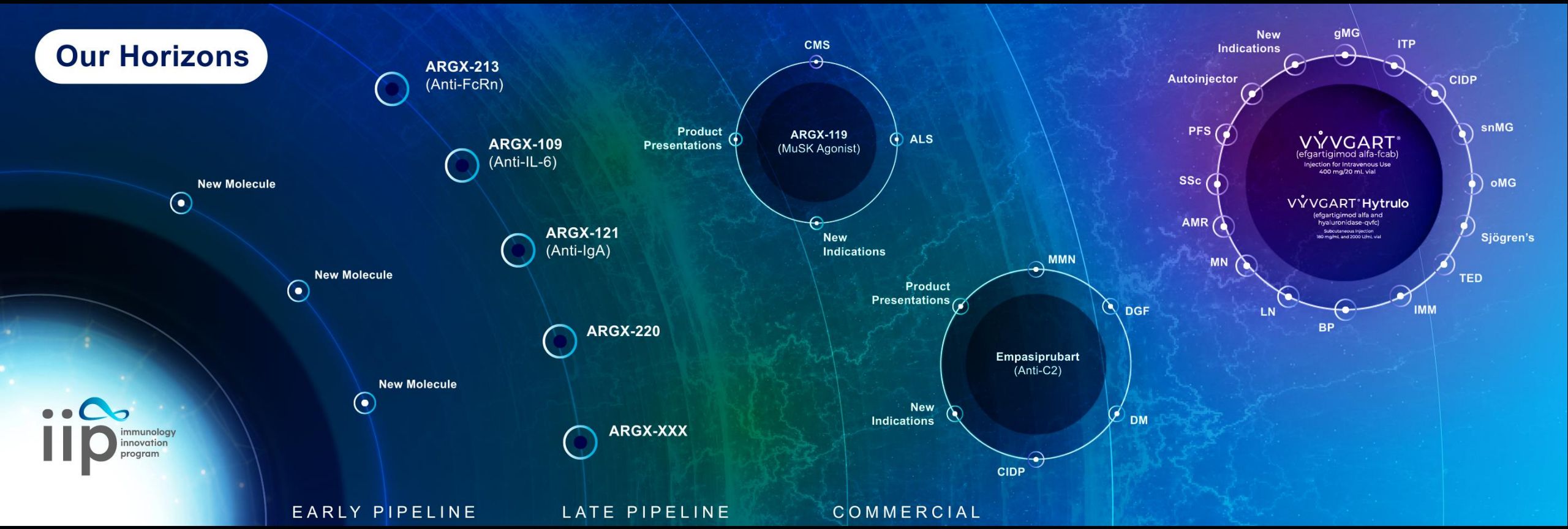

Our Horizons
ARGX-213
(Anti-FcRn)
ARGX-109
(Anti-IL-6)
New Molecule
ARGX-121
(Anti-IgA)
New Molecule
ARGX-220
New Molecule
ARGX-XXX
CMS
Product Presentations
ARGX-119
(MuSK Agonist)
ALS
New Indications
MMN
Product Presentations
DGF
Empasiprubart
(Anti-C2)
New Indications
DM
CIDP
New Indications
gMG
ITP
Autoinjector
CIDP
PFS
snMG
VYVGART
(efgartigimod alfa-fcab)
Injection for Intravenous Use
400 mg/20 mL vial
SSc
oMG
VYVGART Hytrulo
(efgartigimod alfa and hyaluronidase-qvfc)
Subcutaneous Injection
180 mg/mL and 2000 U/mL vial
AMR
Sjögren's
MN
TED
LN
IMM
BP
immunology innovation program
EARLY PIPELINE
LATE PIPELINE
COMMERCIAL

Blueprint for Innovation
Next Wave of First-in-Class Immunology Targets

Peter Ulrichts /// Chief Scientific Officer

Immunology Innovation Program: Model of Co-Creation

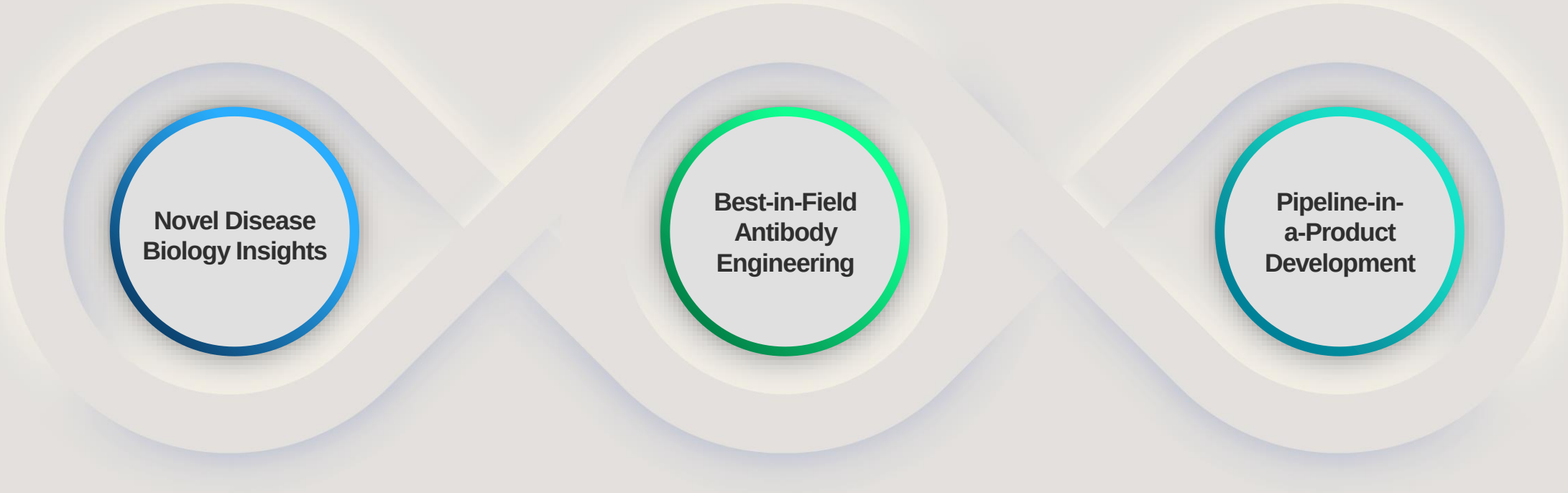
ARGX-113
Efgartigimod

ARGX-117
Empasiprubart

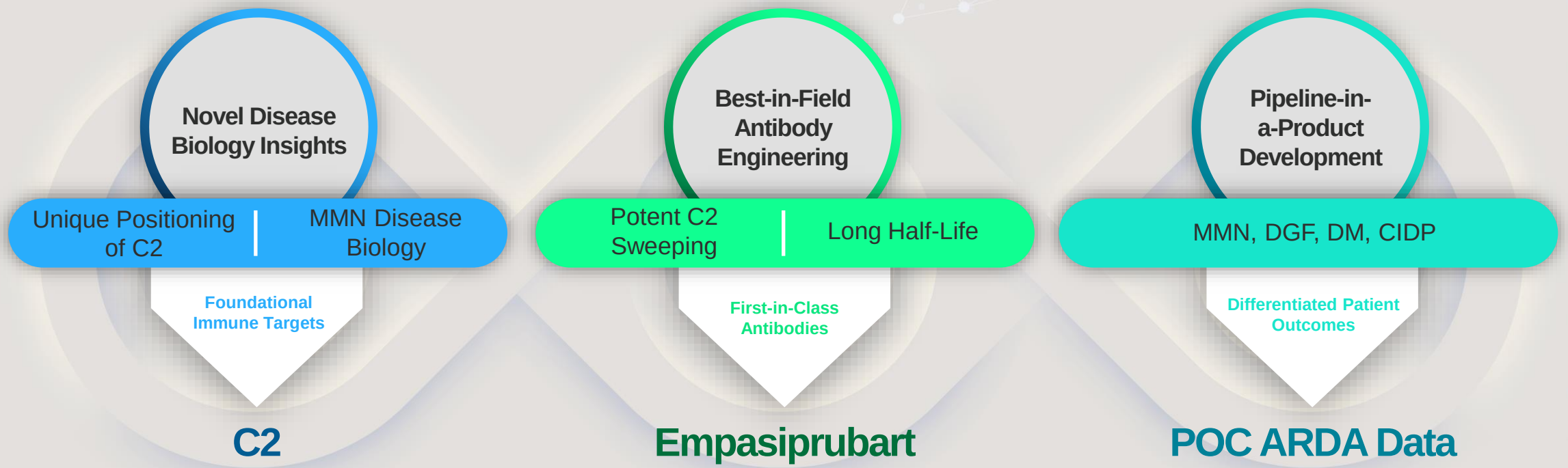
ARGX-119

ARGX-117

Unraveling Central Role of C2 in Complement Cascade



Unraveling Central Role of C2 in Complement Cascade



Unique complement toolkit

C2-KO and human C2-transgenic mice

>30 complement assays in house across different species

Various species cross-reactive anti-C2 mAbs for translational models

EMPASIPRUBART

INNOVATION ECOSYSTEM

Extensive network of experts

University of Glasgow
Cedars Sinai
UMC Utrecht
AARHUS UNIVERSITY
KU LEUVEN
Leids Universitair Medisch Centrum
UNIVERSITY OF LEICESTER
VIB

Advancing science

JACI The Journal of Allergy and Clinical Immunology
BRAIN COMMUNICATIONS
AMERICAN ACADEMY OF NEUROLOGY

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Empasiprubart in Action

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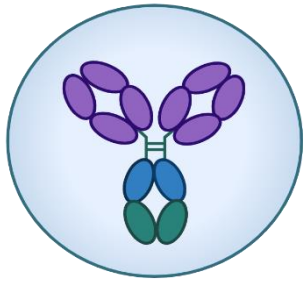
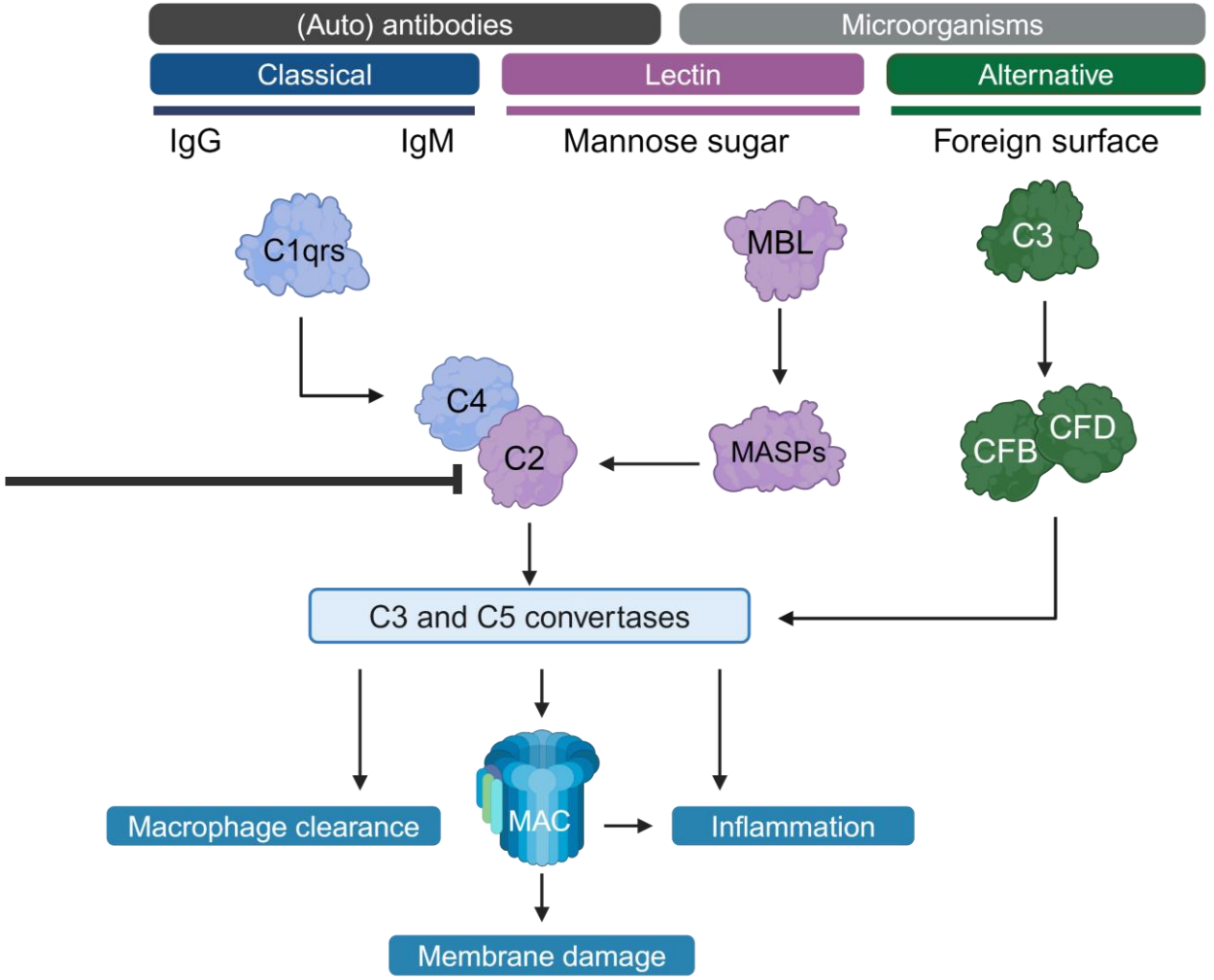
C2 is Uniquely Positioned in Complement Cascade

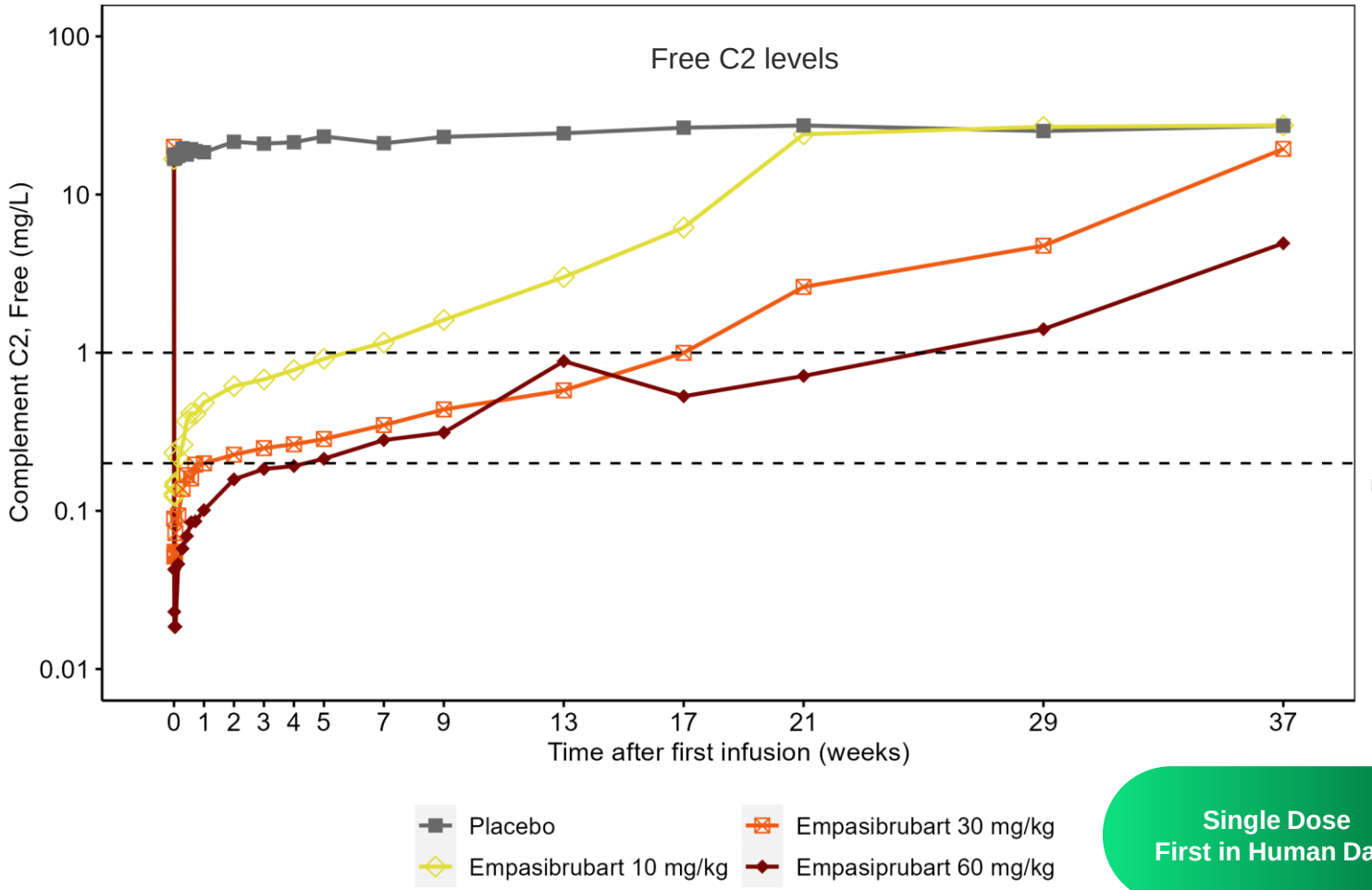
Empasiprubart





Empasiprubart Demonstrates Long Half-life and Sustained Pharmacodynamic Effect

Sustained reduction in free C2 levels by 95% for > 100 days as of 30 mg/kg dose

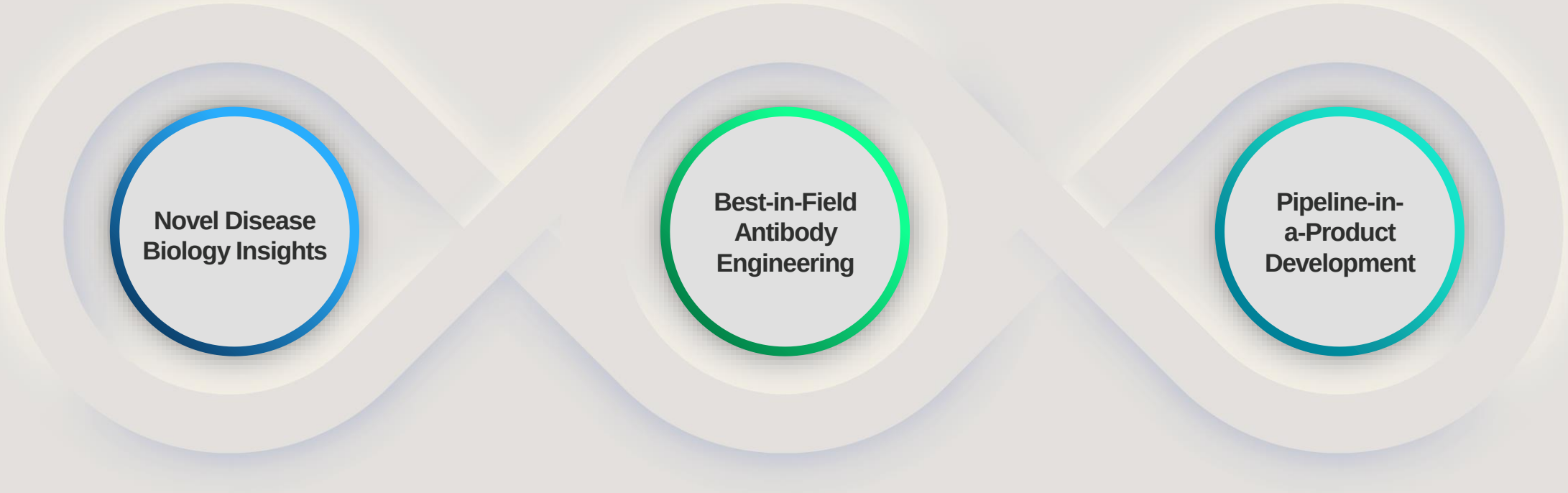


Single Dose First in Human Data

36

ARGX-119

Strengthening the Neuromuscular Junction through MuSK Activation



Strengthening the Neuromuscular Junction through MuSK Activation

MuSK

ARGX-119

Phase 1 Data Support POC Studies

Unique MuSK toolkit

MuSK phosphorylation assays

AChR clustering assays

Various binding assays

Human in vitro ALS NMJ co-cultures

Various in vivo neuromuscular disease models including DOK7 CMS mice

ARGX-119

INNOVATION ECOSYSTEM

Extensive network of experts

Leids Universitair Medisch Centrum

NYU Langone Health

University of Oxford

TRICALS The highway towards a cure

Université de Montréal

neuro Montreal Neurological Institute-Hospital

CHEO

Massachusetts General Hospital

RAYA Therapeutic

Advancing science

nature

Science Translational Medicine

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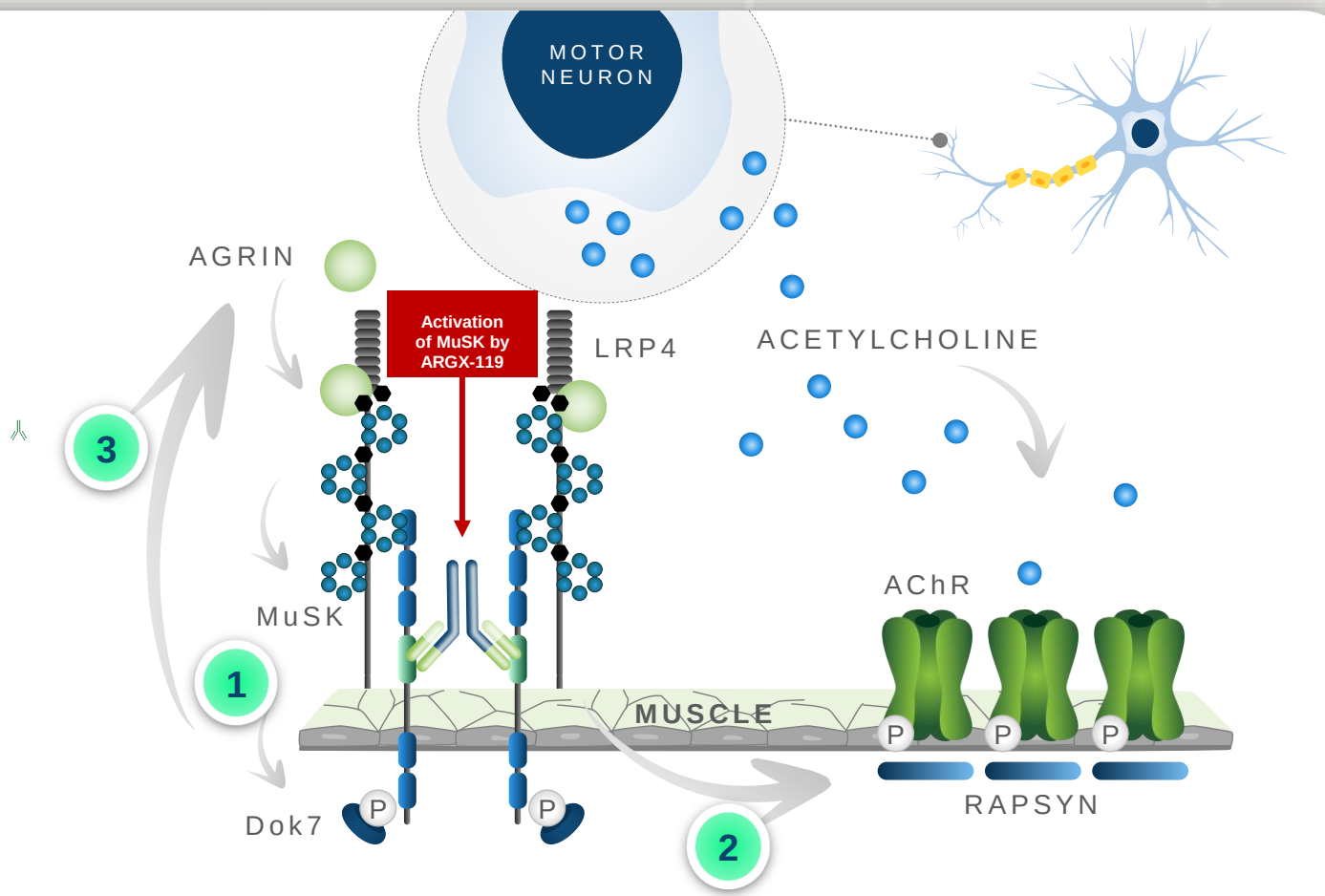
ARGX-119 Boosts Functioning of NMJs by Improving AChR Clustering

1. MuSK phosphorylates downstream signaling components
2. MuSK-induced clustering of AChRs and muscle contraction upon Acetylcholine binding
3. Retrograde/signal to stimulate presynaptic differentiation



AGRIN MuSK ARGX-119 Acetylcholine AChR

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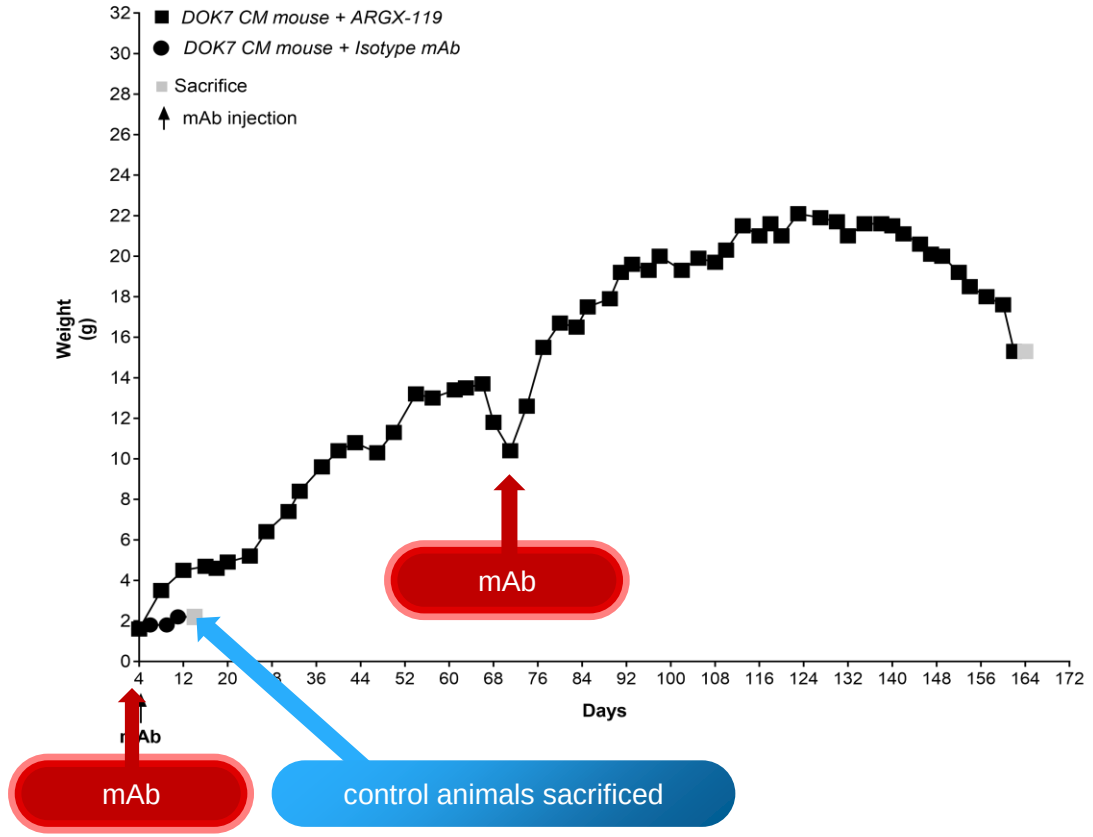
CMS Rationale: Early Neonatal Lethality and Disease Relapse are Rescued by ARGX-119 in DOK7 CMS mice

1. Diminished MuSK phosphorylation in DOK7 CMS

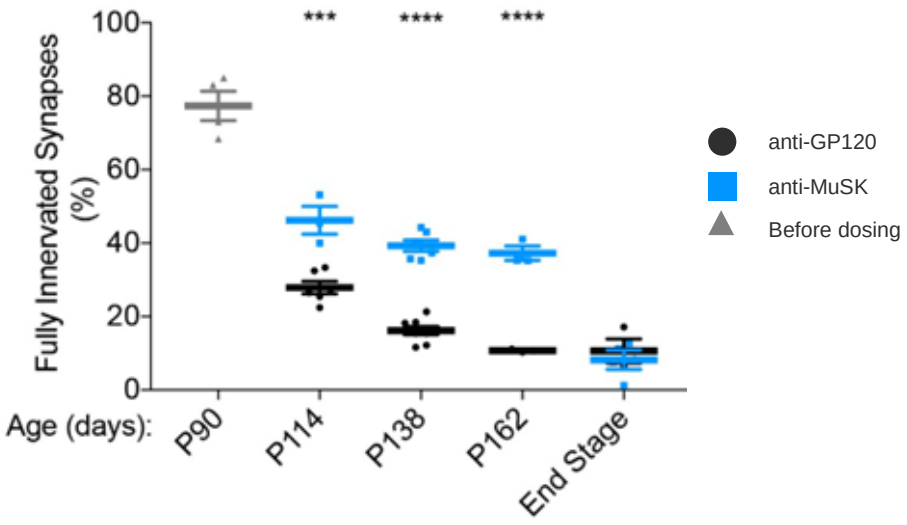
2. Leads to lethal weakness of diaphragm muscles

3. MuSK activation by ARGX-119 rescues phenotype

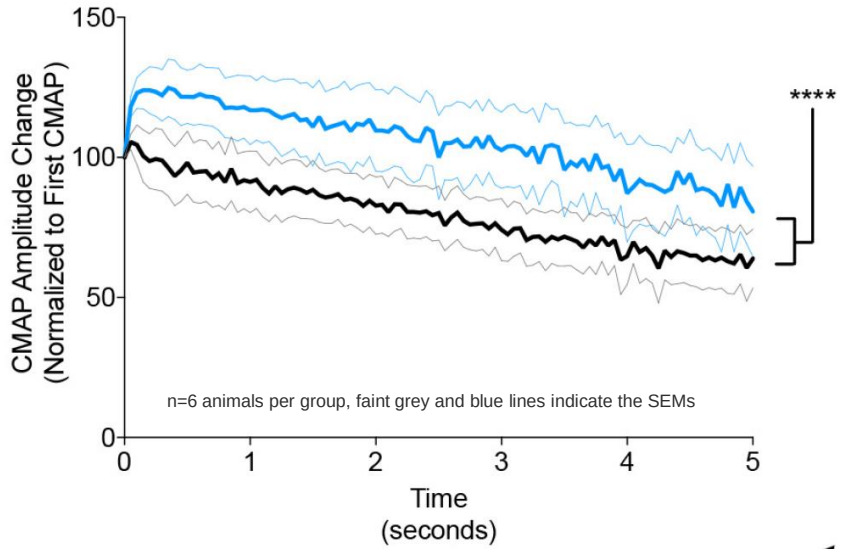


ALS Rationale: Activation of MuSK Signaling Slows Muscle Denervation and Improves Motor Function

Slowdown of muscle denervation



Improving motor system output



In vivo model show: Delayed disease onset | Improvement in survival

Reference: Cantor et al. 2018; Pérez-García et al. 2012; argenx internal data;

Path Forward for ARGX-119

CMS

Phase 1b
to start in 4Q24

Proof of Biology
Intra-Patient Dosing

ALS

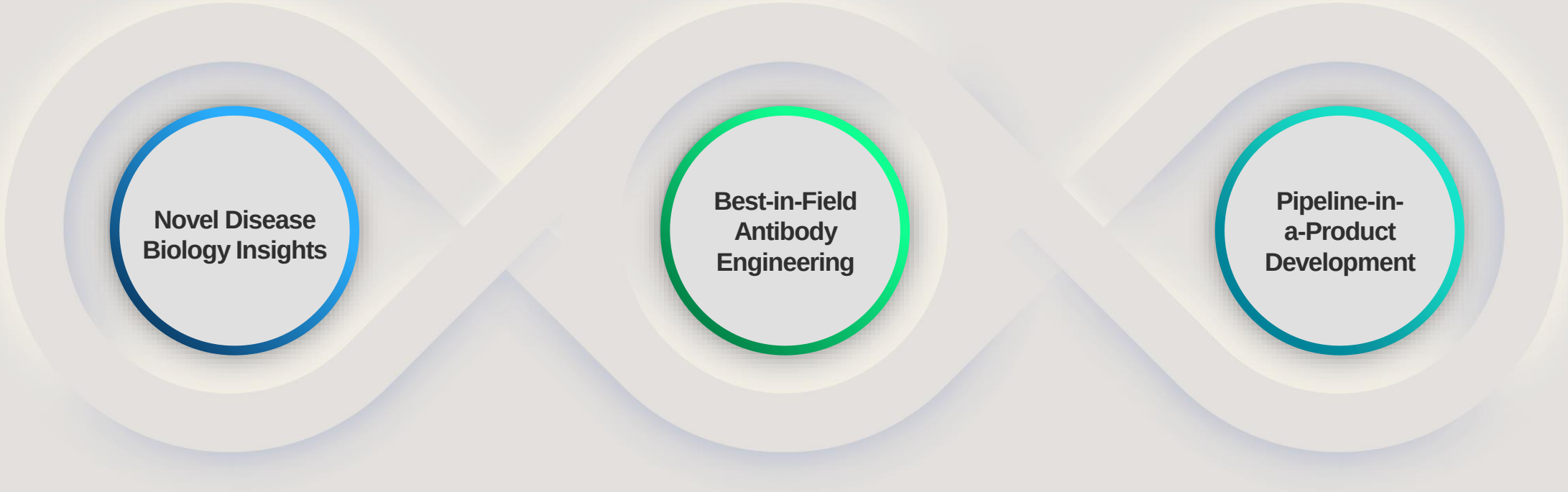
Phase 2a reALiSe
to start in 4Q24

Innovation Within Discovery
MScan
(MScan-derived Motor Unit Number)

NATURAL HISTORY STUDY ONGOING

ARGX-113

Leadership in FcRn



Leadership in FcRn

Unique FcRn toolkit

- Differentiating binding assays
- Predictive cellular assays
- Expert structural biology
- Innovative hFcRn/hAlbumin transgenic mouse models

EFGARTIGIMOD

INNOVATION ECOSYSTEM

Extensive network of experts

University of Southampton

Sanquin

Authera

Advancing Science

nature COMMUNICATIONS

JCI

JCI insight

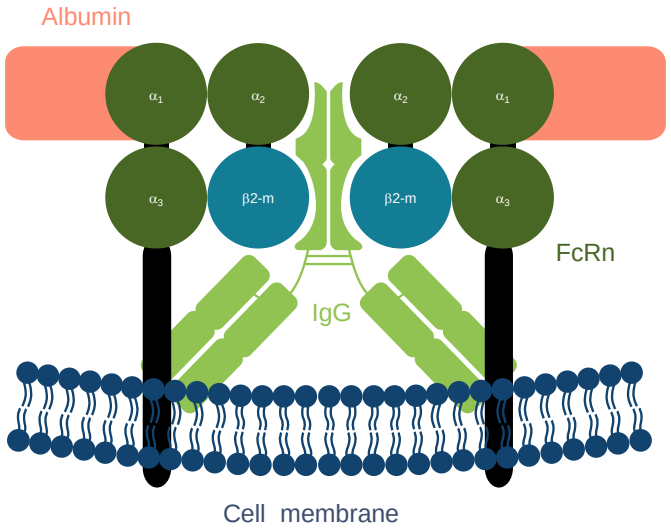
frontiers in Immunology

cells

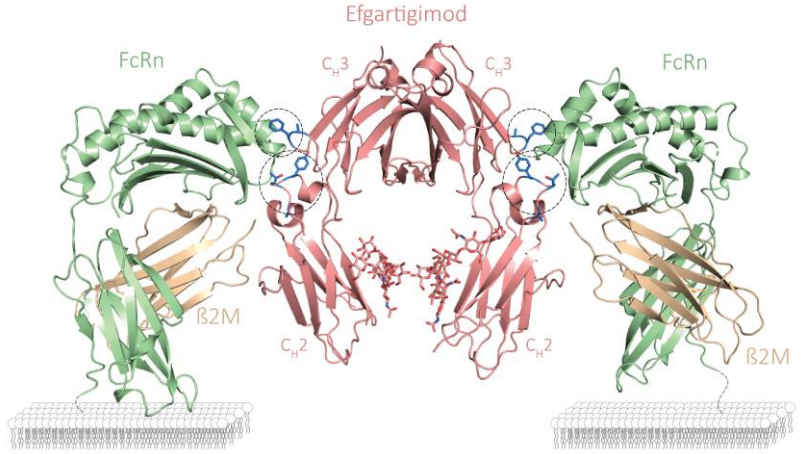
argenx

Efgartigimod Binds to FcRn in Same Formation as Endogenous IgG

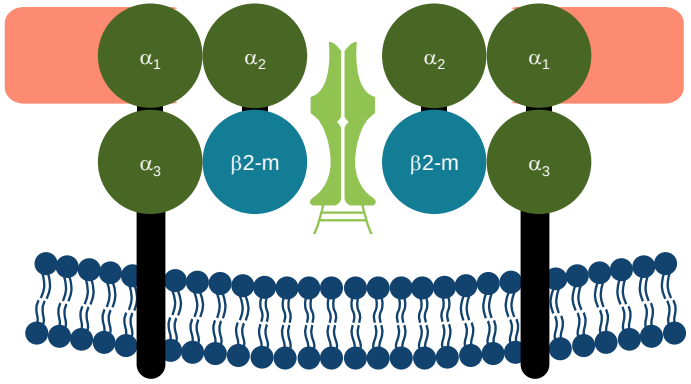
Endogenous IgG interaction



Efgartigimod



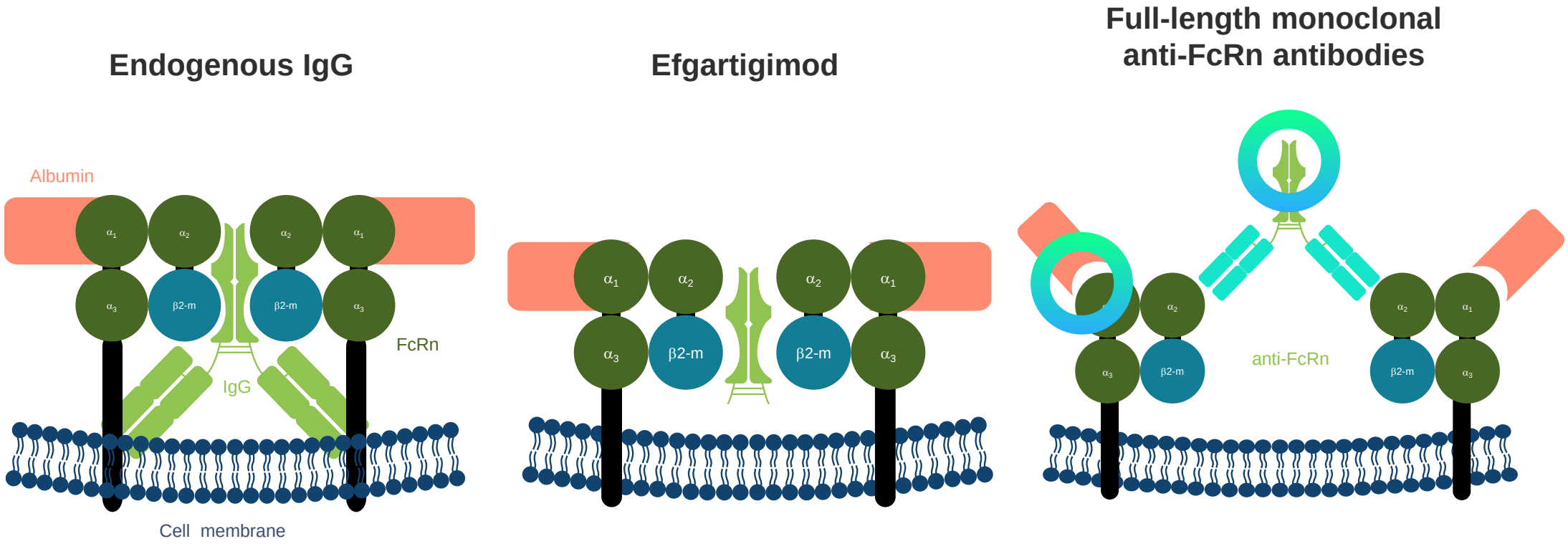
Efgartigimod interaction



FcRn, neonatal Fc receptor; IgG, immunoglobulin G.

1. Ulrichts P, et al. J Clin Invest. 2018;128:4372–4386; 2. Howard JF Jr, et al. Lancet Neurol. 2021;20:526–536; 3. VYVGART SmPC. Available at https://www.ema.europa.eu/en/documents/product-information/vyvgart-epar-product-information_en.pdf; 4. Knudsen Sand KM, et al. Front Immunol. 2015;5:1–21; 5. Ward ES, et al. Front Immunol. 2022;13:892534;

Efgartigimod is Unique Among FcRn Antagonists in How it Binds



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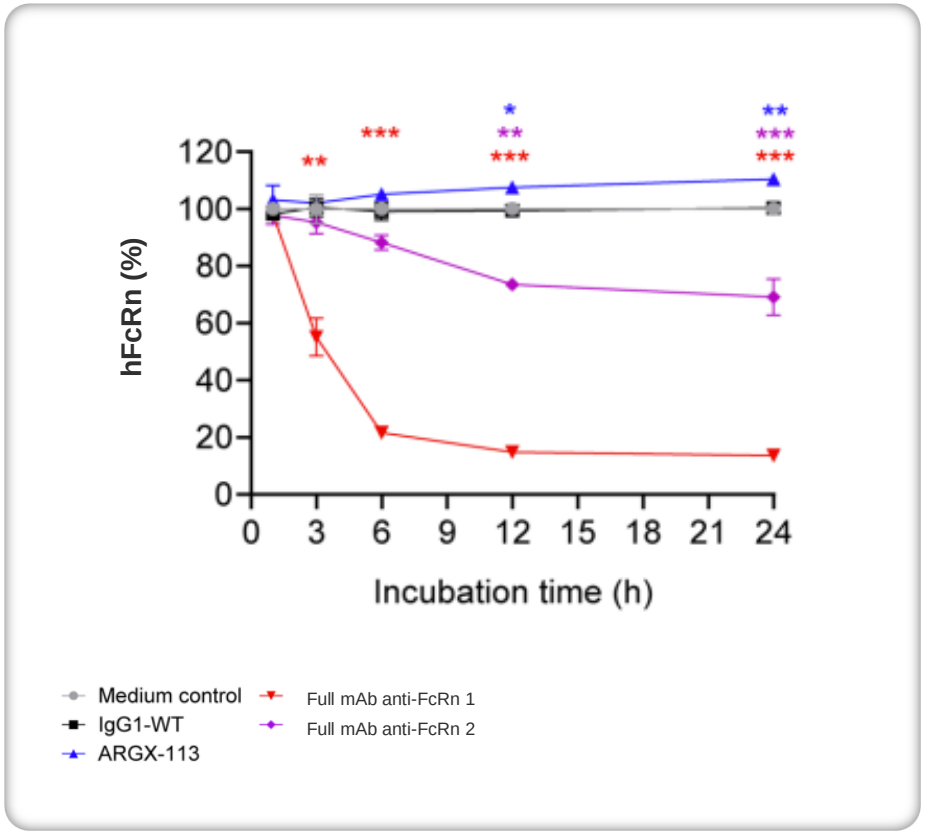
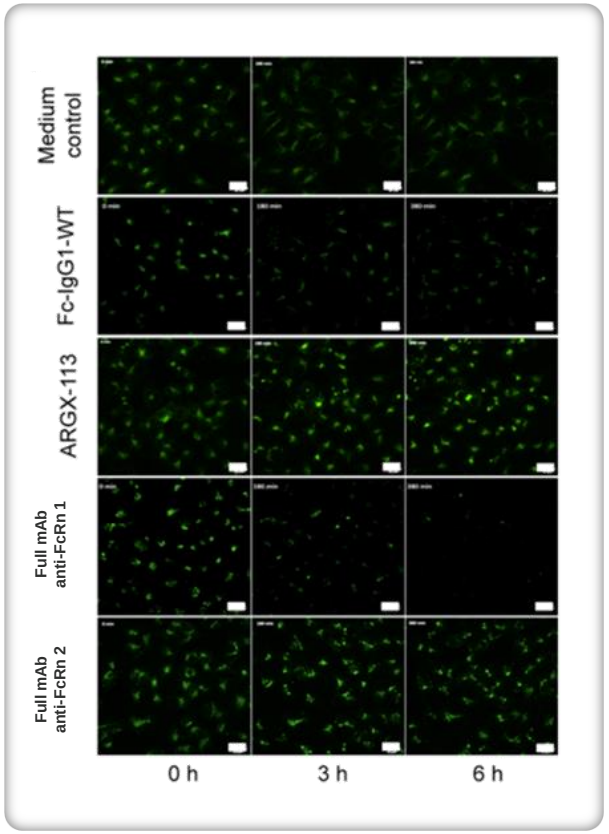
1. Ulrichts P, et al. J Clin Invest. 2018;128:4372–4386; 2. Howard JF Jr, et al. Lancet Neurol. 2021;20:526–536; 3. VYVGART SmPC. Available at https://www.ema.europa.eu/en/documents/product-information/vyvgart-epar-product-information_en.pdf; 4. Knudsen Sand KM, et al. Front Immunol. 2015;5:1–21; 5. Ward ES, et al. Front Immunol. 2022;13:892534;

Unique Binding of Efgartigimod Leads to Differentiated Intracellular FcRn Trafficking





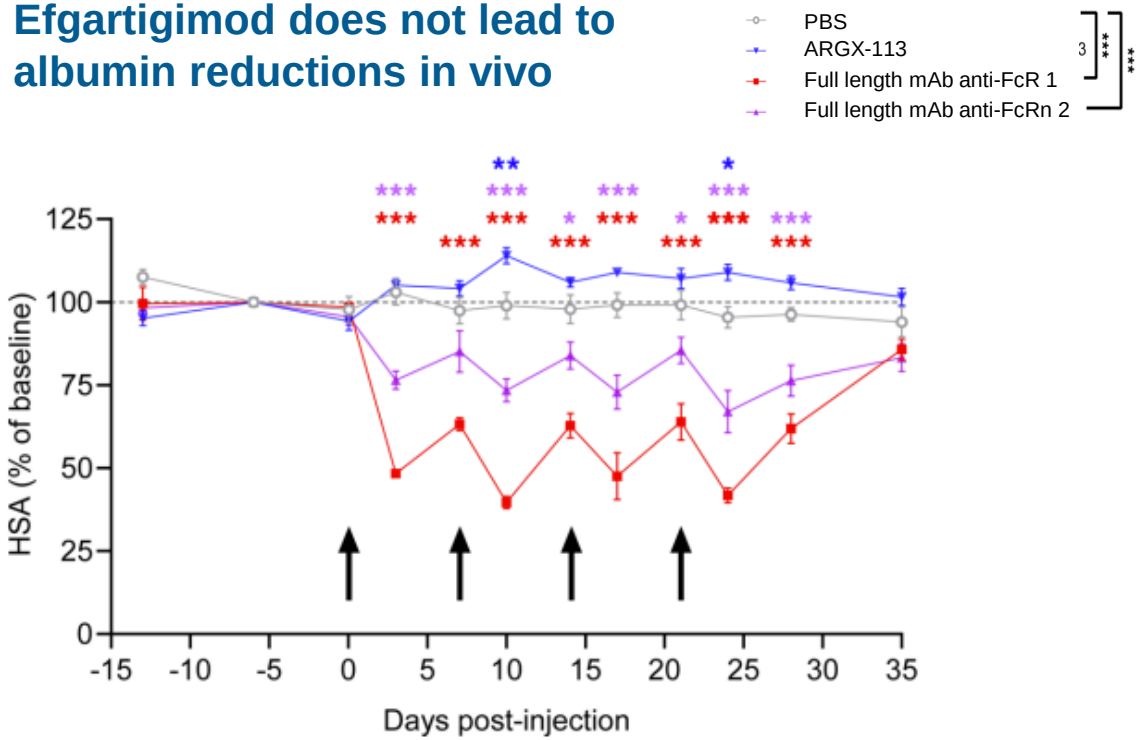
No interference of efgartigimod with albumin binding and recycling

No degradation of FcRn induced by efgartigimod

Ma et al, 2024 (10.1172/jci.insight.176166)

Unique Binding of Efgartigimod Positively Impacts in vivo Albumin Levels and Safety Profile

Efgartigimod does not lead to albumin reductions in vivo



Efgartigimod treatment results in a favorable safety profile in the clinic

No albumin reduction

No edema, hyperlipidemia or muscle cramps

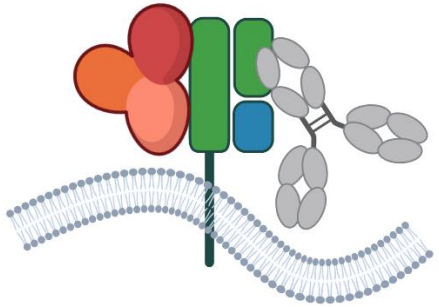
No aseptic meningitis

No clearance by anti-drug antibodies

Ma et al, 2024 (10.1172/jci.insight.176166)

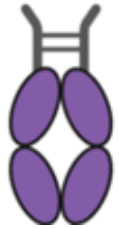
Evolution of a Novel Target to a Novel Platform

FcRn



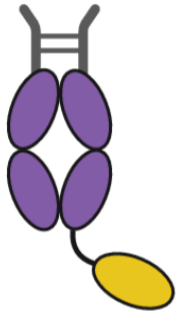
Novel Target

Efgartigimod



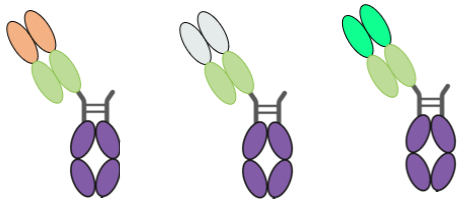
Pipeline in-a-Product

ARGX-213



Next Antagonist in FcRn Portfolio

ARGX-121 | ARGX-XXX



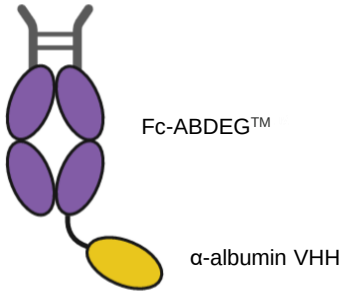
Sweeping Antibody Platform

Next Wave of First-in-class Immunology Targets

Karen Silence /// Preclinical Product Development

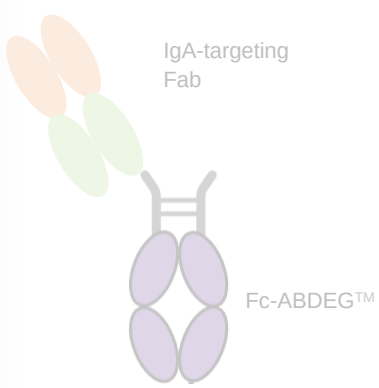
Deep Knowledge of FcRn Biology Builds New Pipeline Candidates

Leverage Albumin to Broaden FcRn Targeting Portfolio



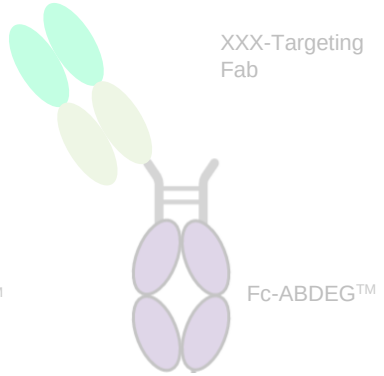
ARGX-213

Leveraging Efgartigimod Backbone to Build New Class of Highly Potent Sweeping Molecules



ARGX-121

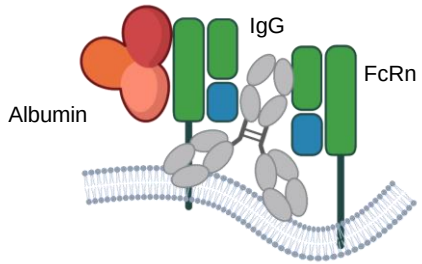
ARGX-XXX



ARGX-XXX

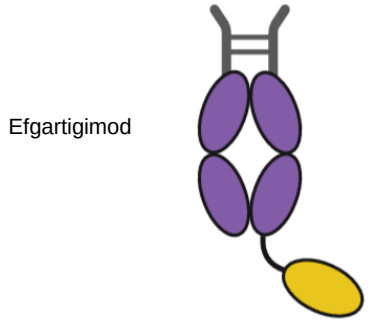
Improving Pharmacokinetics of Efgartigimod Through Binding to Serum Albumin

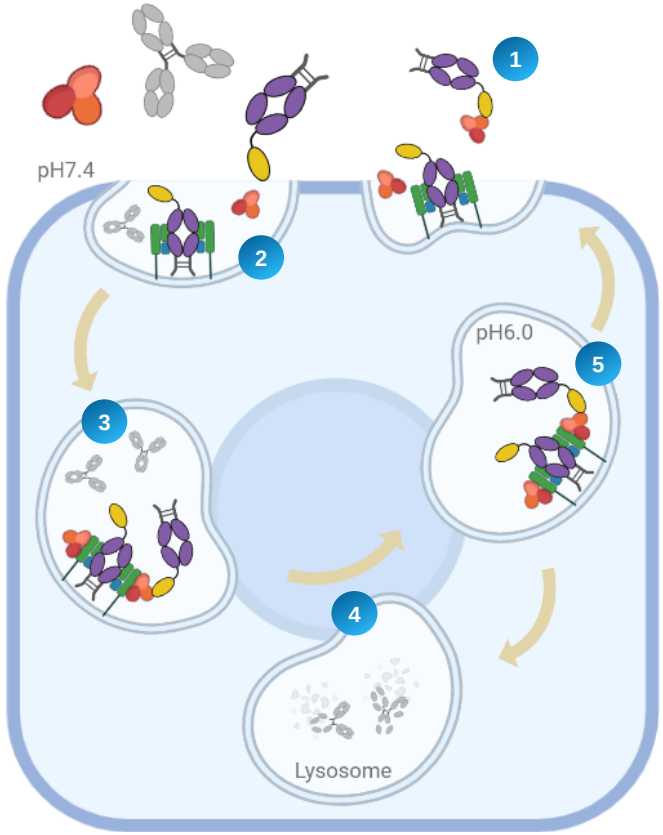
FcRn



Albumin and IgG recycle via different binding sites

ARGX-213



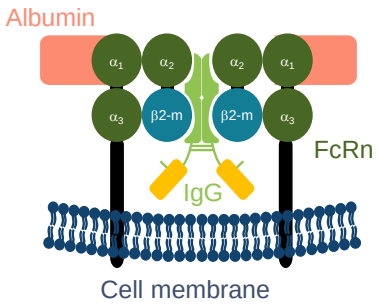


1. ARGX-213 adopts long albumin half-life
2. Pinocytosis
3. In endosomes, ARGX-213 prevents IgG binding to FcRn
4. IgG degraded in lysosomes
5. ARGX-213 recycles via FcRn or albumin

argenx

ARGX-213 is Designed For Optimal FcRn Binding and Equipped with Unique Features

Prototype

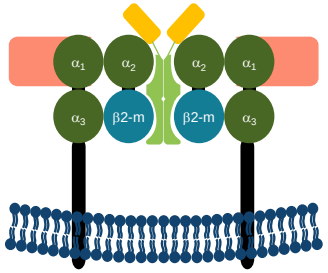


- Enhanced PK
- Sustained PD
- Albumin sparing

Invention

Position VHH

ARGX-213 Two-armed



- Enhanced PK
- Sustained PD
- Albumin sparing

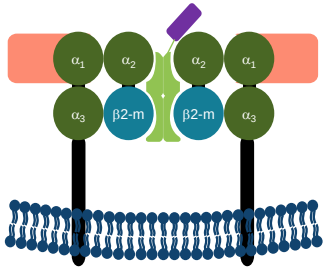
Invention

Valency VHH

Affinity VHH at pH 7.4 and pH 6.0

Linker length

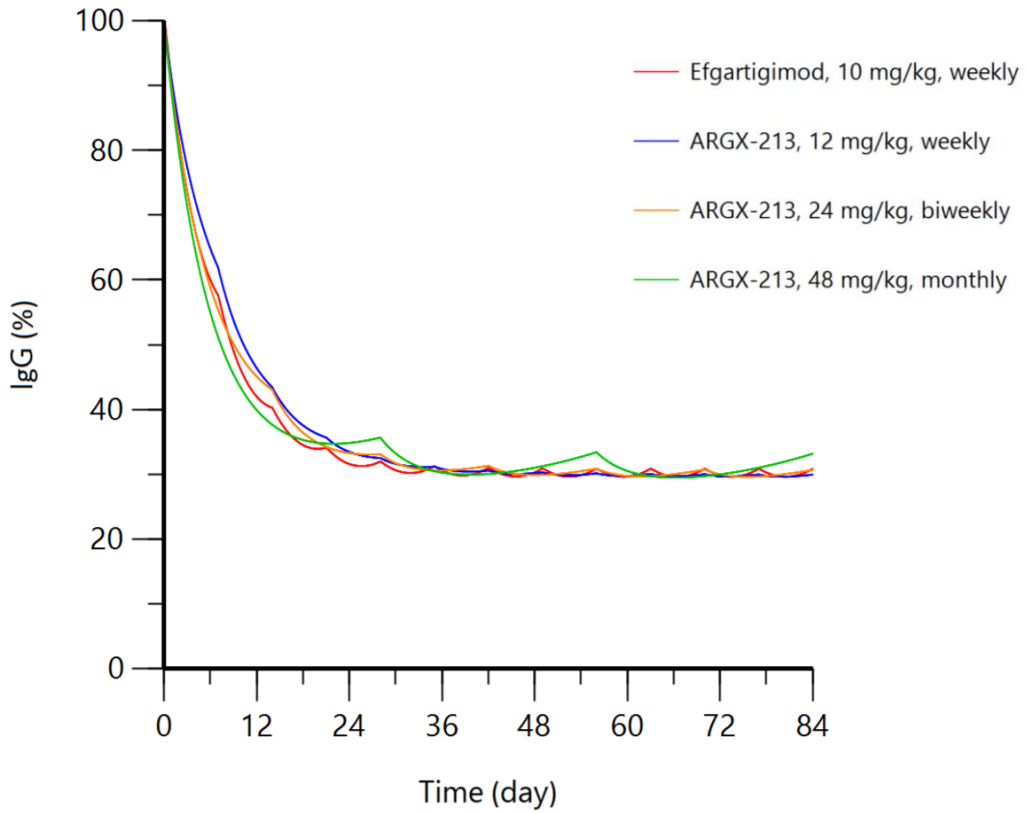
ARGX-213



- Enhanced PK
- Sustained PD
- Albumin sparing

argenx

ARGX-213 Can Achieve Extended Dosing



ARGX-213 has increased half-life compared to efgartigimod resulting in prolonged PD effect

Simulations predict potential for monthly dosing

10 mg/kg efgartigimod and 12 mg/kg ARGX-213 are equimolar doses ARGX-213 PK/PD model based on mouse and cyno data

Path Forward for ARGX-213

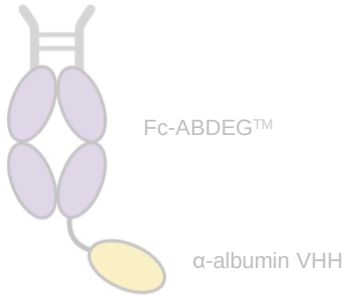
Finalize GLP Tox Study

Submit Clinical Trial Application 1H25

Phase 1 to Start in 2H25

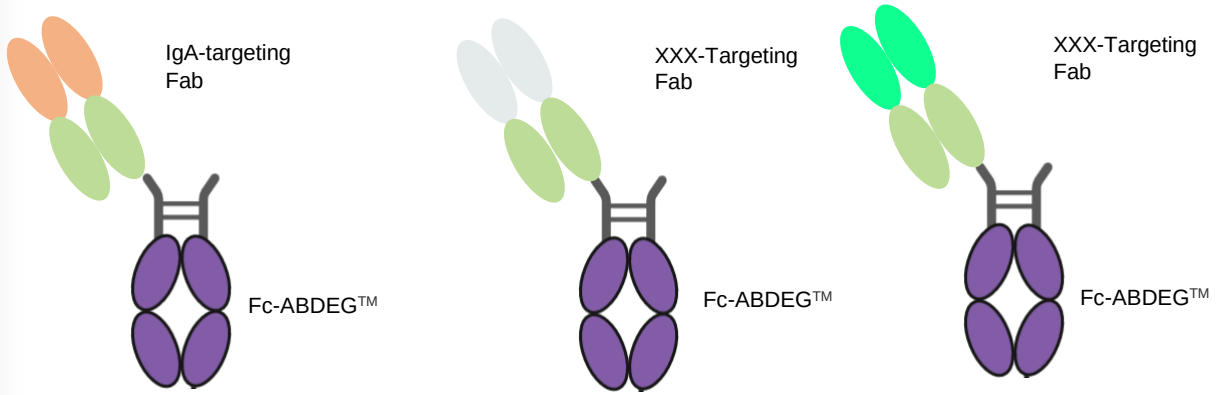
Deep Knowledge of FcRn Biology Builds New Pipeline Candidates

Leverage Albumin to Broaden FcRn Targeting Portfolio



ARGX-213

Leveraging Efgartigimod Backbone to Build New Class of Highly Potent Sweeping Molecules

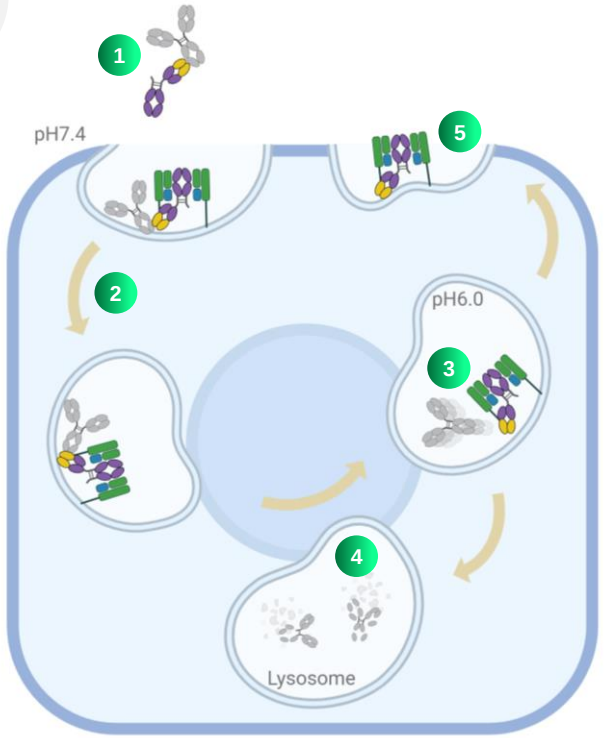


ARGX-121 **ARGX-XXX** **ARGX-XXX**

ARGX-121 Mode of Actions

I. FcRn-mediated IgA degradation

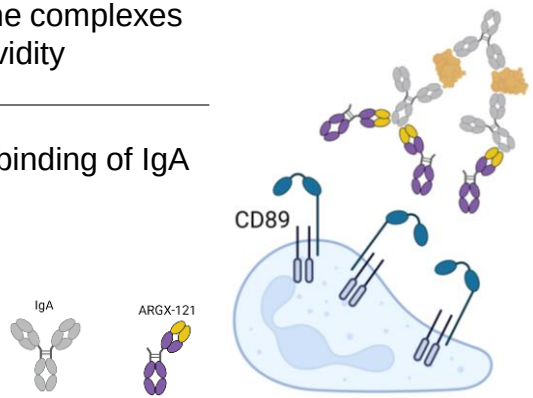
1. ARGX-121 binds to IgA (1-3 mg/ml)
2. Enhanced endocytosis of ARGX-121 IgA complex
3. Complex dissociates at pH 6.0 in endosomes
4. IgA is degraded in lysosomes
5. ARGX-121 recycles through enhanced FcRn binding at pH 6.0



II. Blocking of IgA:CD89 mediated signalling

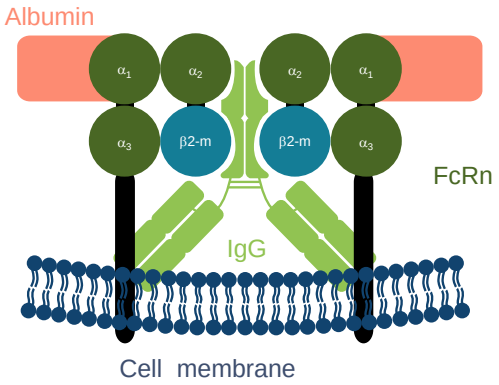
Monomeric IgA binds with low affinity to CD89 (FcαRI) but upon formation of immune complexes it binds with high avidity

ARGX-121 blocks binding of IgA IC to CD89



ARGX-121 Innovative Design Breakthrough

Prototype
IgG1-LALAPG-ABDEGᵀᴹ

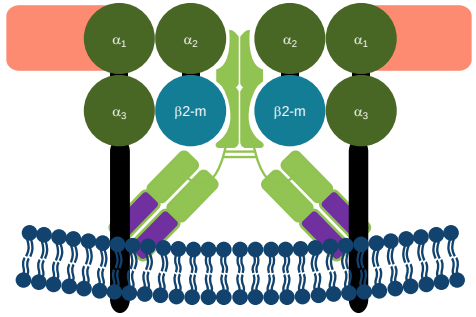


Invention

Affinity at pH 7.4 and pH 6.0

FcRn degradation

ARGX-121 Two-armed

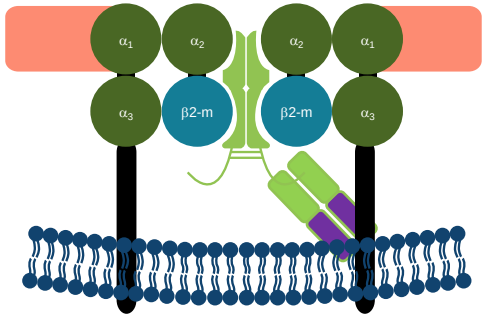


Invention

Immune complex formation

FcRn occupancy

ARGX-121 One-armed



pH-dependent target binding	++
Risk for making immune complexes	+++
FcRn degradation	++
FcRn occupancy	-
IgA depletion in cyno	+

pH-dependent target binding	+++
Risk for making immune complexes	++
FcRn degradation	+
FcRn occupancy	+
IgA depletion in cyno	++

pH-dependent target binding	+++
Risk for making immune complexes	-
FcRn degradation	-
FcRn occupancy	++
IgA depletion in cyno	+++

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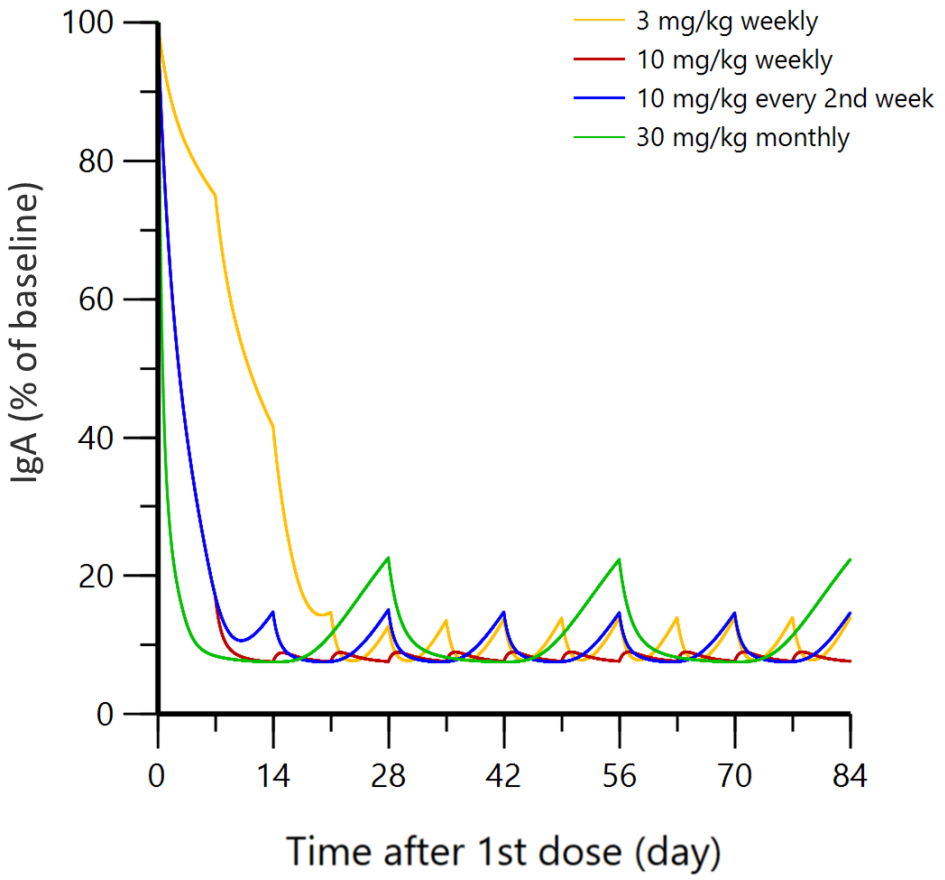
ARGX-121 Rapidly and Drastically Impacts Circulating IgA Levels



Baseline IgA levels 2.5mg/ml
ARGX-121 PK/PD model based on mouse and cyno data

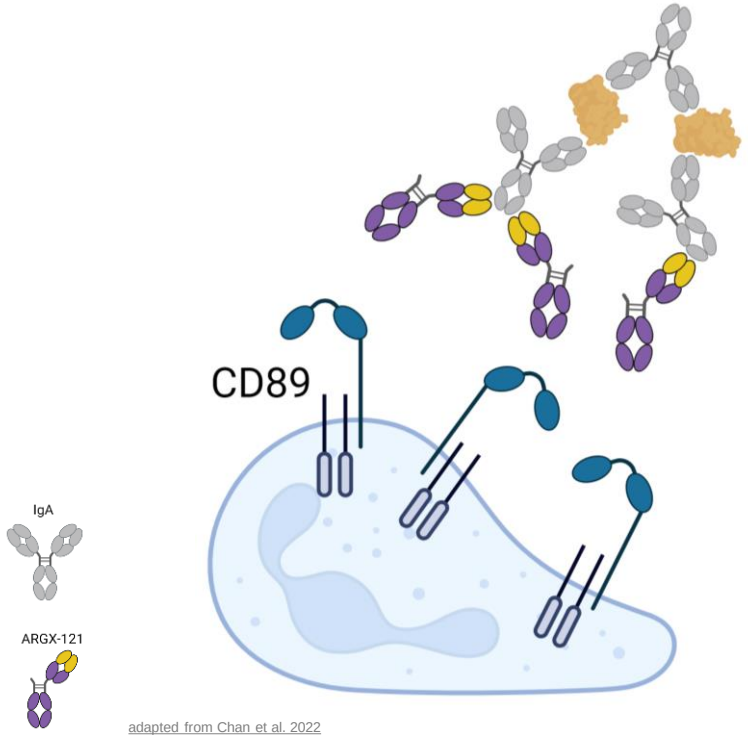
>90% IgA reduction within 1 week

Enables **flexible dosing**

Broad therapeutic potential

ARGX-121 Pipeline-in-a-Product Potential

IgA is Fundamental in Many Diseases



adapted from Chan et al. 2022

- IgA nephropathy
- IgA vasculitis
- Celiac disease
- Sjogren's disease
- Rheumatoid arthritis
- Systemic lupus erythematosus
- Linear IgA bullous disease
- Dermatitis herpetiformis
- Epidermolysis bullosa acquisita
- Mucous membrane pemphigoid
- Bullous pemphigoid

Path Forward for ARGX-121

Finalize GLP Tox Study

Submit Clinical Trial Application 1H25

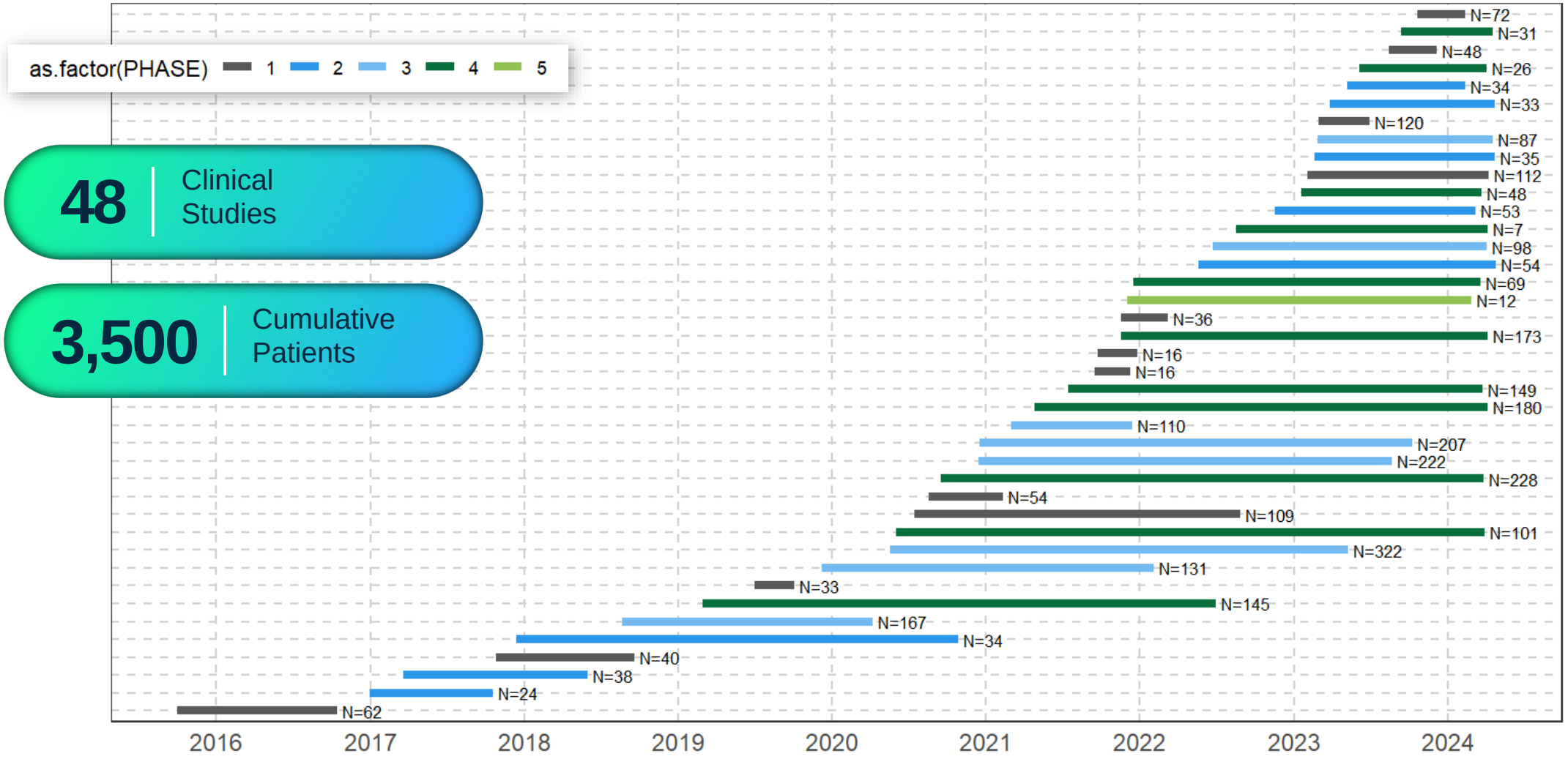
Phase 1 to Start in 2H25

Clinical Development

Luc Truyen, M.D., PhD /// Chief Medical Officer

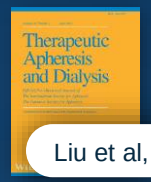
Clinical Development: Bridging Innovation & Unmet Patient Need

Rapidly Scaling our Clinical Footprint



Pioneering in MG to Set New Standard for Treatment

2010 - 2017
- IgG lowering effect of efgartigimod
- Central role of IgG in MG



Liu et al, 2010

2018
- Extended clinical effect of cyclical dosing

JCI The Journal of Clinical Investigation

Ulrichts, 2017

2019
- MSE >50% across studies
- Highest MG-ADL and QMG response

Neurology®

Howard et al, 2019

2020
- Equivalence of SC to IV
- Positive benefit risk profile

THE LANCET Neurology

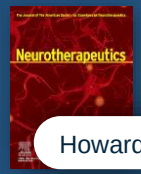
Howard et al, 2021

2021
- Value broadly recognized by payor bodies
- Sustained responses across dosing schedules

frontiers in Neurology

Howard et al, 2024

2022
- Reduction in steroid use
- Broadest safety database



Howard et al, accepted

2023
- *Market leader among advanced biologics*

JCI The Journal of Clinical Investigation

Ward, 2024

2024
- *Earlier treatment lines*
- *Seronegative, Ocular*

FUTURE

argenx

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Applying Our Innovation Approach to Clinical Development

Innovation

Evidence Generation

Build broadest data to guide treatment decisions for patients

Co-Creation

Empowering Patients

Patient engagement in trial design and execution

Execution

Speed

Bring medicines to patients as quickly as possible

adapt SERON myasthenia gravis study

adapt Oculus myasthenia gravis study

rho STUDY

alkivia Idiopathic Inflammatory Myopathy Study

arda Multifocal Motor Neuropathy Study

iMMersioN Multifocal Motor Neuropathy

argenx

Ocular and Seronegative MG

adapt SERON myasthenia gravis study

adapt Oculus myasthenia gravis study

Expanding MG Leadership Across Treatment Paradigm

Evidence Generation

ADAPT/ADAPT+

Real-world data

Patient Insights

Significant need

Lack of innovation

Speed

Efficient studies

Significant underserved population

Sjögren's Disease

rho STUDY

Working to Reach Patients Faster

Evidence Generation

Depth of data from RHO study

Leveraging all FcRn data

Patient Insights

Endpoint selection

PRO measures

Speed

Phase 3 to start by end of 2024

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Immune Mediated Myopathies (IMM)

alkivia
Idiopathic Inflammatory Myopathy Study

One Study Across Multiple Myositis Subtypes

Evidence Generation

Subtype selection based on pathogenic IgG rationale

Patient Insights

Common TIS endpoint

Speed

Seamless Phase 2/3 Study with interim analysis

Multifocal Motor Neuropathy (MMN

arda Multifocal Motor Neuropathy Study

iMMersioN Multifocal Motor Neuropathy

Pioneering First-in-Class Novel MoA

Evidence Generation

Robust PoC from ARDA

EoP2: endpoint alignment

Patient Insights

Natural history study exceeds 100 patients to date

Speed

Leveraging Ph2 and iMMersioN to accelerate recruitment

argenx

CIDP is 4th Indication for Empasiprubart

Developing a Winning Strategy in CIDP

Evidence Generation

Building on MMN data

Broadening knowledge in complement biology

Patient Insights

High medical need

Opportunity for multiple innovations

Speed

Registrational trial with interim analysis

Bringing Innovation to Patients

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Efgartigimod in Myositis

Leentje De Ceuninck, Ph.D. /// Senior Clinical Scientist

Melissa
Living with Myositis

Idiopathic Inflammatory Myopathies (IIM) or Myositis

Characteristics	Disease Burden
14 per 100,000 diagnosed	**Muscle weakness** and **Pain**
Mid-adult onset, more common in **females**	**Fatigue**
Increased mortality	**Large impact** on quality of life
No FDA-approved therapies across myositis subtypes	Corticosteroid **side effects**

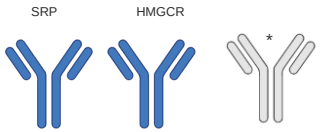
Myositis subtypes mediated by autoantibodies:
immune-mediated necrotizing myopathy (IMNM),
Antisynthetase syndrome (ASyS) and dermatomyositis (DM)

Reference - 1. Meyer A, et al. Rheumatology. 2015; 54(1) 2. Lundberg I, et al. Nat Rev Dis Primers. 2021; 7(1)

Myositis Specific Autoantibodies (MSA) are Associated with Different Clinical Symptoms

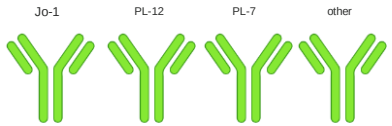
MSA target different autoantigens

IMNM



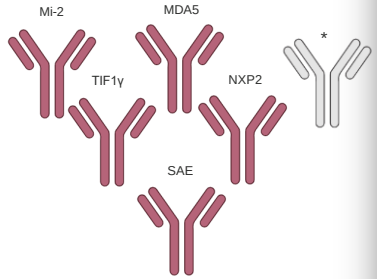
SRP: protein translation
HMGCR: cholesterol synthesis

ASyS



tRNA synthetases

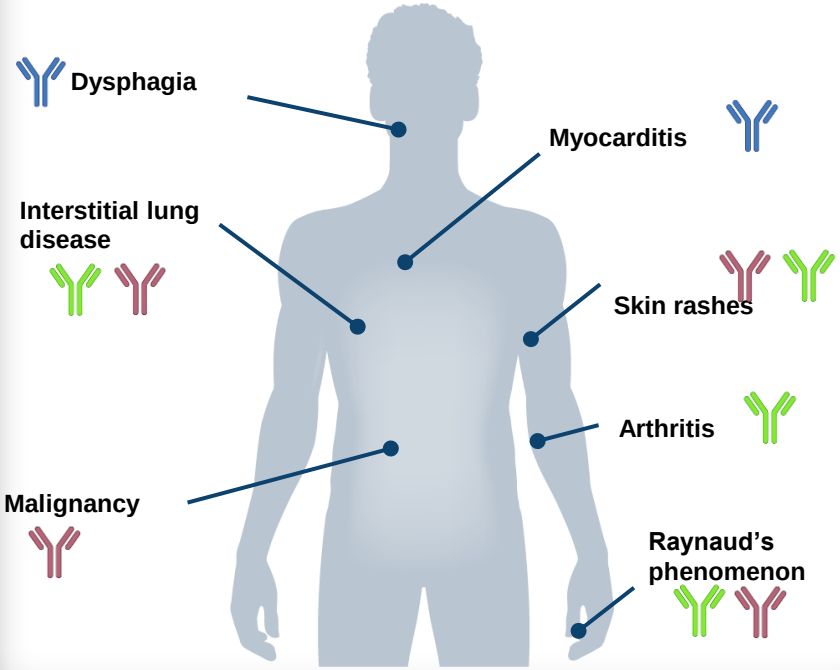
DM



Mi2: transcriptional repressor
Others: IFN regulators

* 25 – 30%: Myositis associated antibodies (MAA) or antibodies against unidentified targets

Clinical symptoms

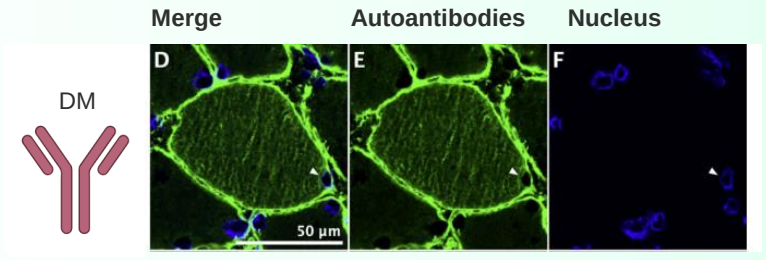


Common hallmark: proximal muscle weakness

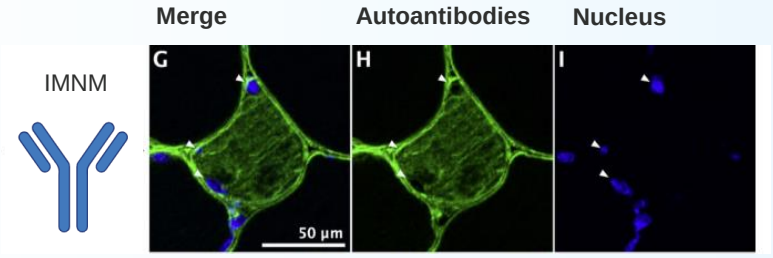
1. McHugh J, et al. *Nat Rev Rheum.* 2018; 14(5) **2.** Lundberg I, et al. *Nat Rev Dis Primers.* 2021; 7(1)

Myositis Auto-antibodies are Pathogenic

Pathogenic auto-antibodies enter muscle fibers

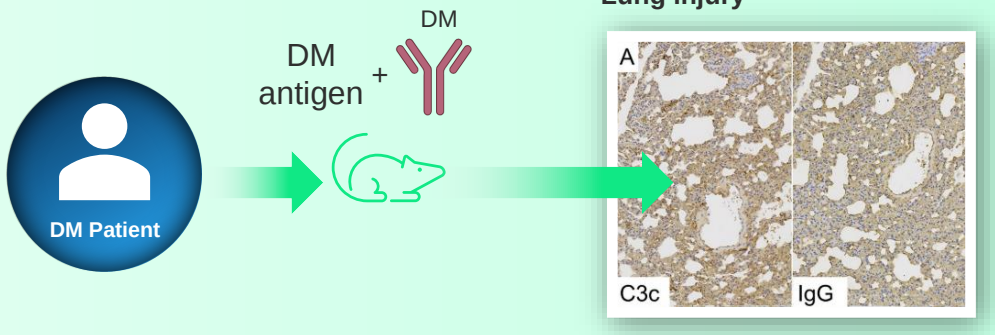


IFN Pathway Activation



Lipid Accumulation

Auto-antibodies induce IIM symptoms



Clinical (human data)

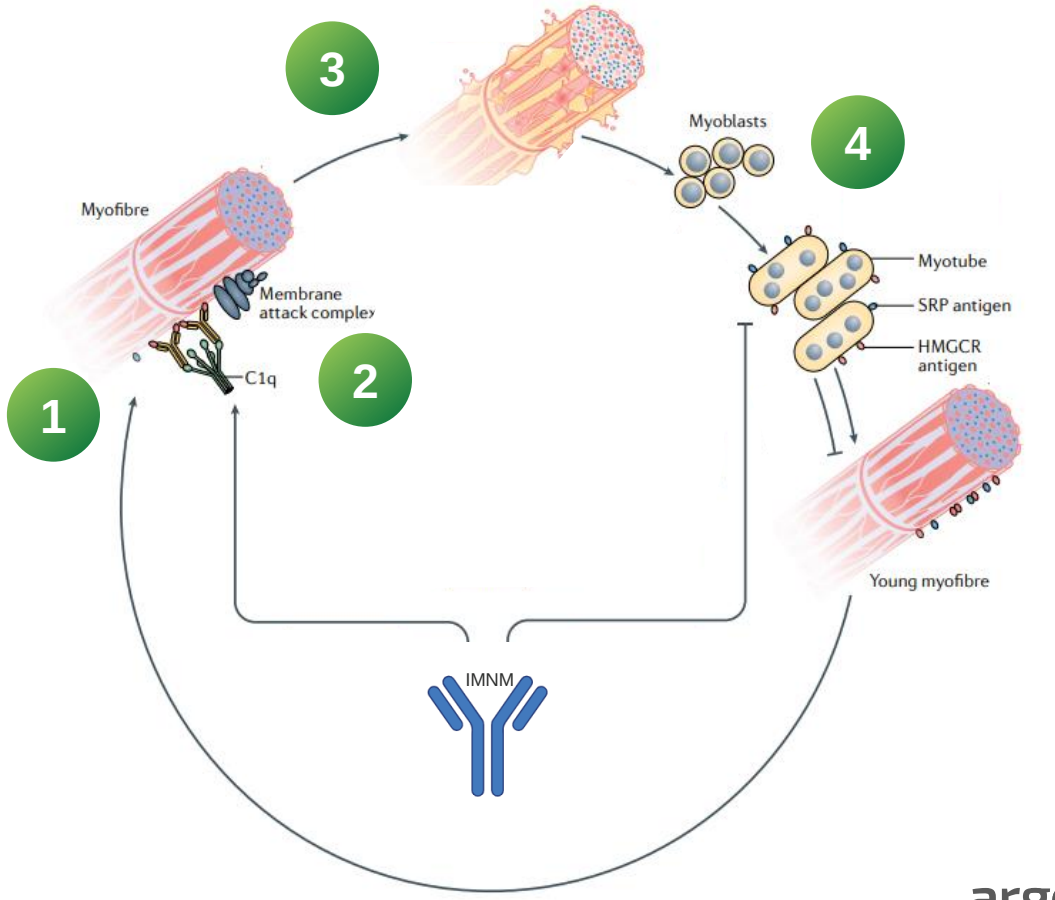
Preclinical (mouse data)

1. Pinal-Fernandez I, et al. Ann Rheum Dis. 2024 (preprint) 2. Pinal-Fernandez I, et al. Ann Rheum Dis. 2023; 82(8) 3. Bergua C, et al. Ann Rheum Dis. 2019; 78(1) 4. Zaizen Y, et al. Respir Res. 2023; 24(1)

IMNM Antibodies Trigger Muscle Damage and Impair Muscle Regeneration

Auto-antibodies:

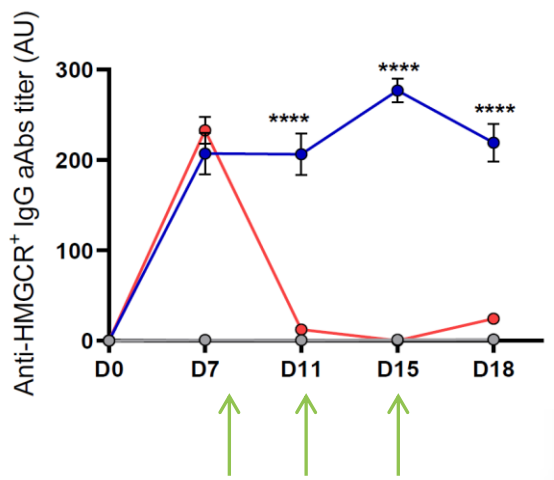
1. Bind muscle fiber
2. Activate complement
3. Cause necrosis
4. Impair muscle regeneration



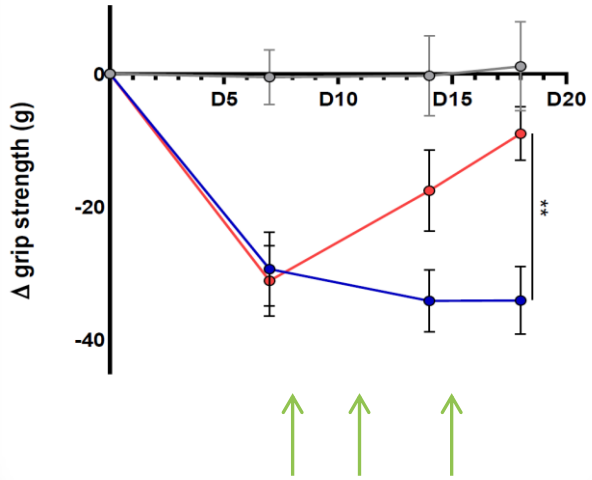
1. Figure adapted from: Allenbach Y, et al Nat Rev Rheum. 2020; 16(12) 2. Bergua C, et al. Ann Rheum Dis. 2019; 78(1) 3. Arouche-Delaperche L, et al Ann Neur. 2017; 81(4)

Efgartigimod Reduces IMNM Antibodies and Restores Mouse Muscle Function

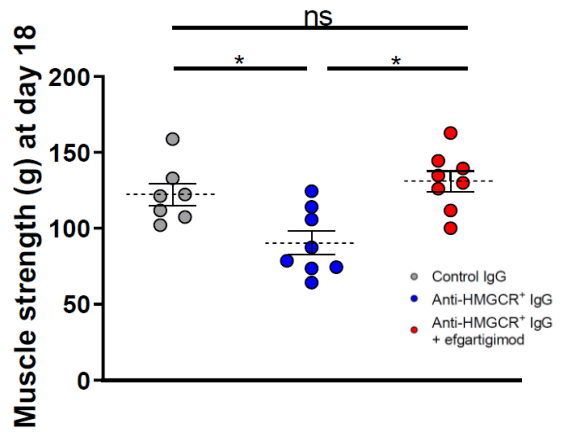
Antibodies



Grip Strength



Muscle Strength



↑ Efgartigimod treatment

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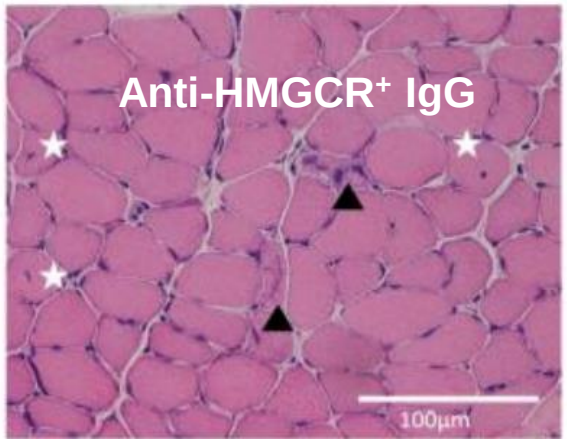
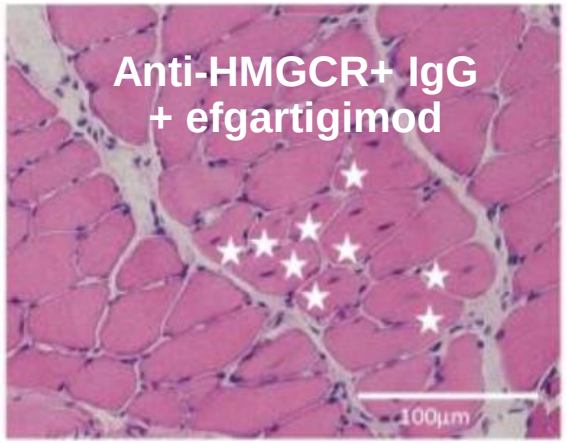
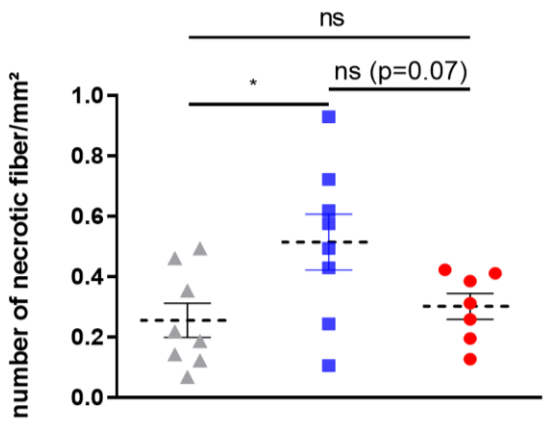
Efgartigimod Prevents Necrosis & Allows Regeneration of Muscle Fibers

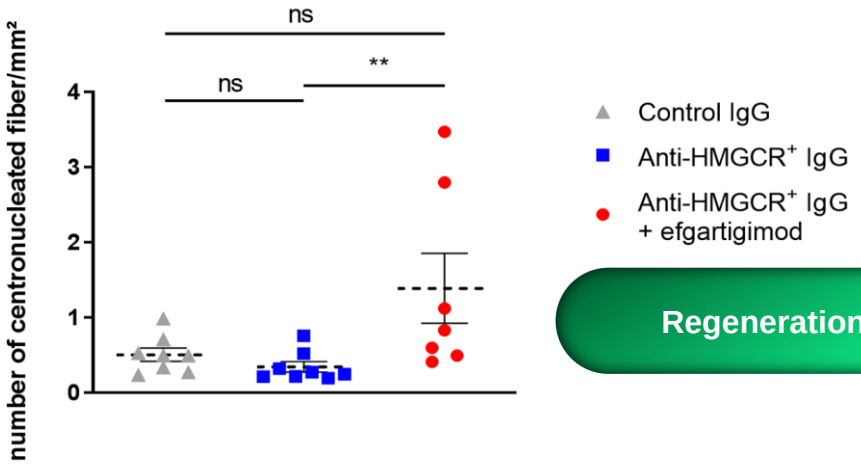




Regenerating muscle fiber

Necrotic fiber





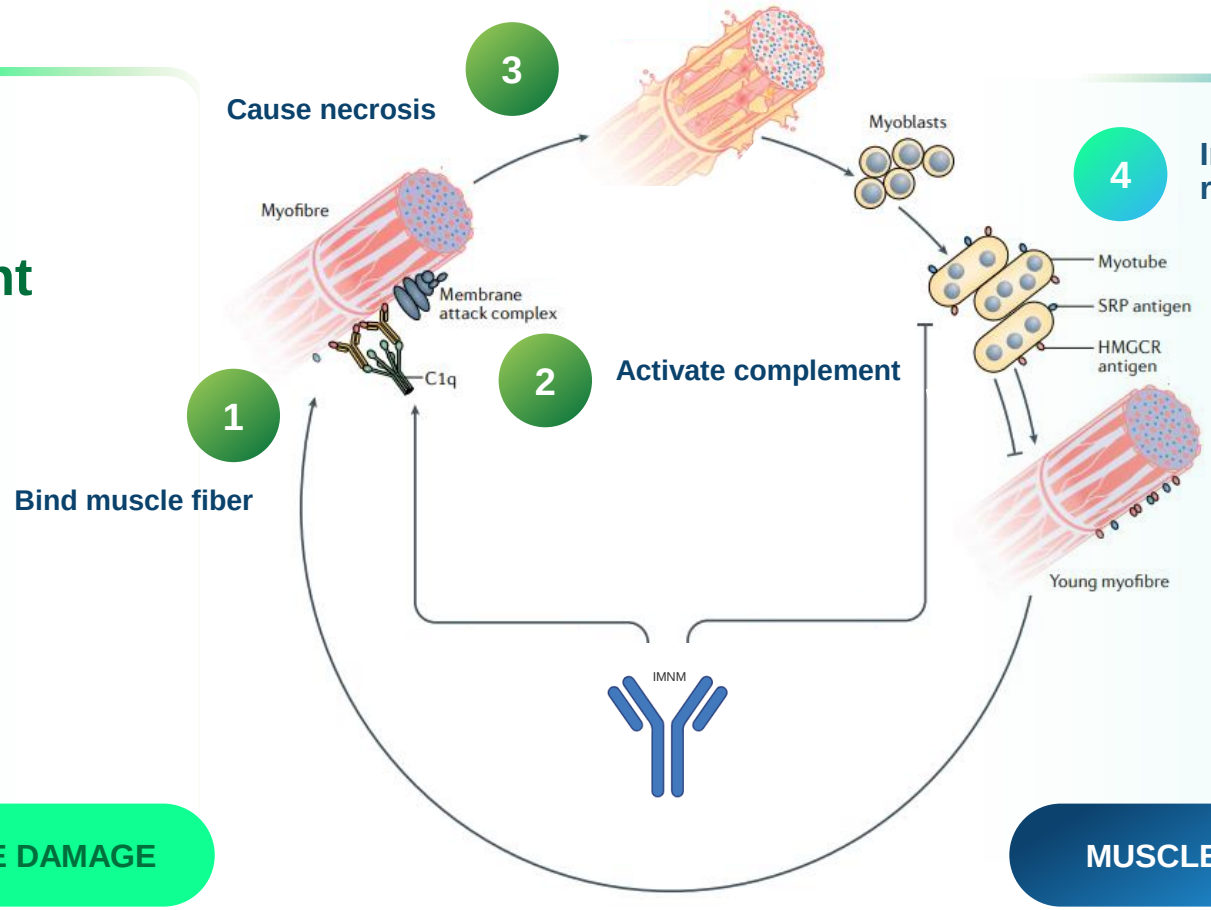
Necrosis

Regeneration

Julien S, et al. Rheumatology 2023; 62 (12)

Efgartigimod Leads to Full Regain of Muscle Function in the IMNM Mouse Model

Antibody Dependent Complement Dependent



Antibody Dependent Complement Independent

MUSCLE DAMAGE

MUSCLE REGENERATION

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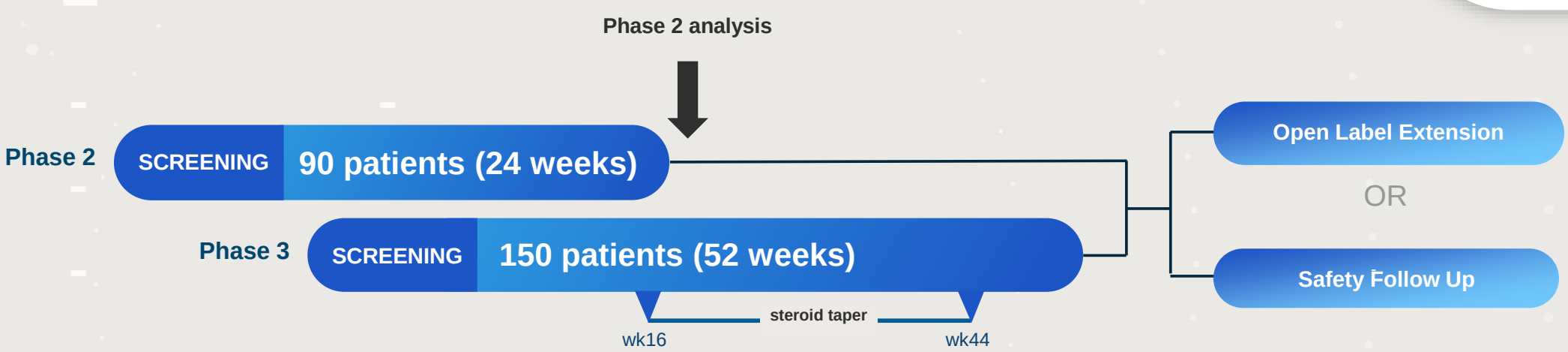
1. Figure adapted from: Allenbach Y, et al Nat Rev Rheum. 2020; 16(12) 2. Bergua C, et al. Ann Rheum Dis. 2019; 78(1) 3. Arouche-Delaperche L, et al Ann Neur. 2017; 81(4)

Phase 2 / Phase 3 Adaptive Basket Trials with Efgartigimod in IMNM, ASyS, DM

alkivia
Idiopathic Inflammatory Myopathy Study



Adults

Active disease and muscle weakness despite stable dose of SoC

Weekly efgartigimod or PBO

+

background treatment

Phase 2 analysis

Go/NoGo per Myositis subtype

Primary endpoint: TIS

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Path Forward for Myositis

Seamless Phase 2 / Phase 3

Ongoing in IMNM, ASyS, DM

Phase 2 analysis By Year End 2024

Go / Go No decision on each subtype

Efgartigimod in Sjögren's Disease

Julie Jacobs Ph.D /// Principal Scientist

Lisa
Living with Sjögren's Disease

89

Sjögren's Disease

Characteristics

- **3 years** time to diagnosis
- **103 per 100,000** diagnosed
- **55 years** average age
- **14:1** female:male ratio
- **29-53%** extra-glandular manifestations

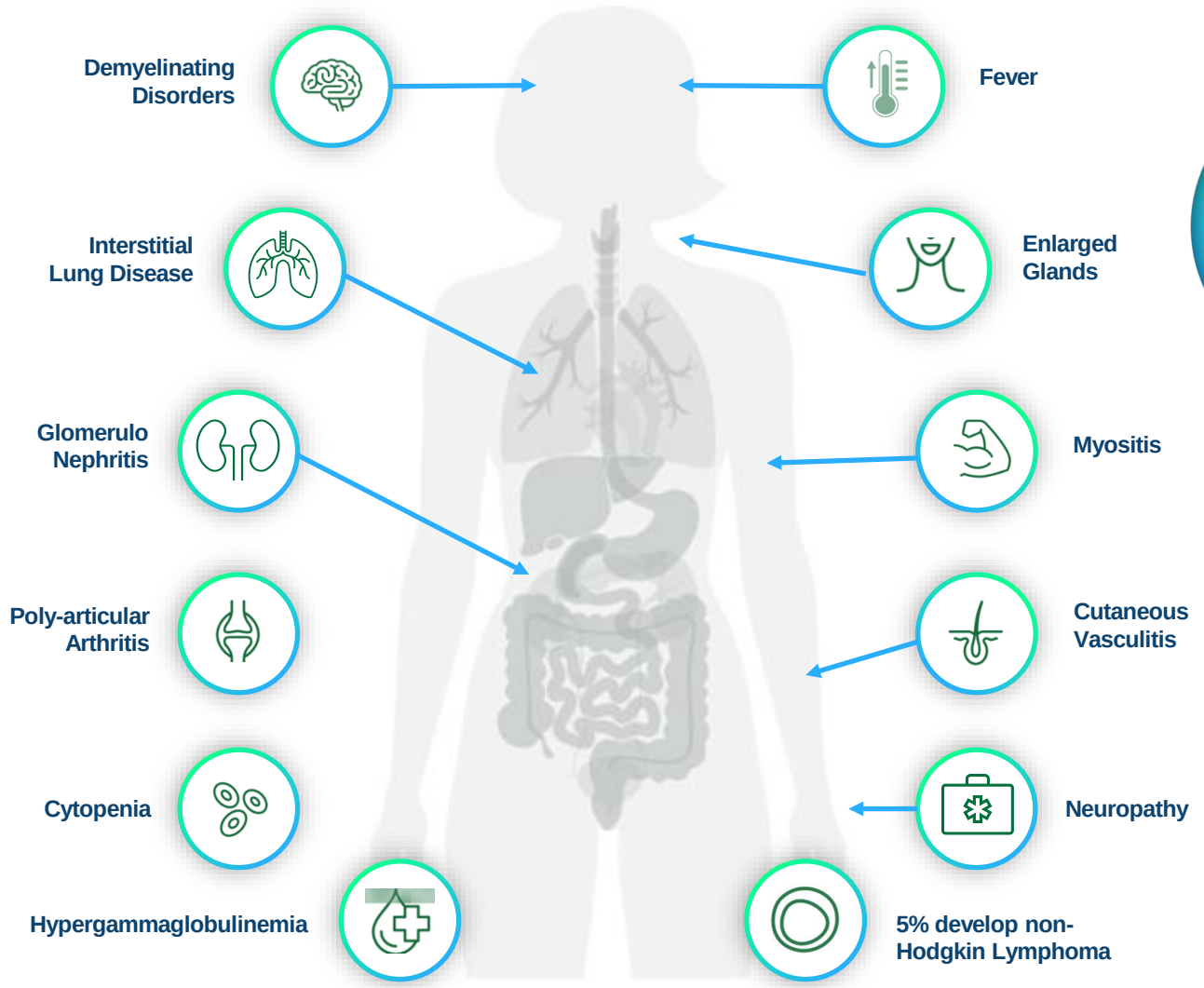
Disease Burden

- **5-10%** develop lymphoma
- **Decreased** physical performance
- **Depression** and **Fatigue**
- **Anxiety** and **Pain**
- Negatively impacting **daily activities**

Lisa
Living with Sjögren's Disease

Aguiñaga MDLG, et al. *Int J Appl Dent*. 2022; Brito-Zerón P, et al. *Nat Rev Dis Primers*. 2016; Negrini S, et al. *Clin Exp Med*. 2022; Ture HY et al. Life ; Omma A et al. Arch Med Sci 2018; Maciel G et al. Arthiris Care Res 2017

Systemic Manifestations of Sjögren's Disease



No Approved Treatments to Target Underlying Disease

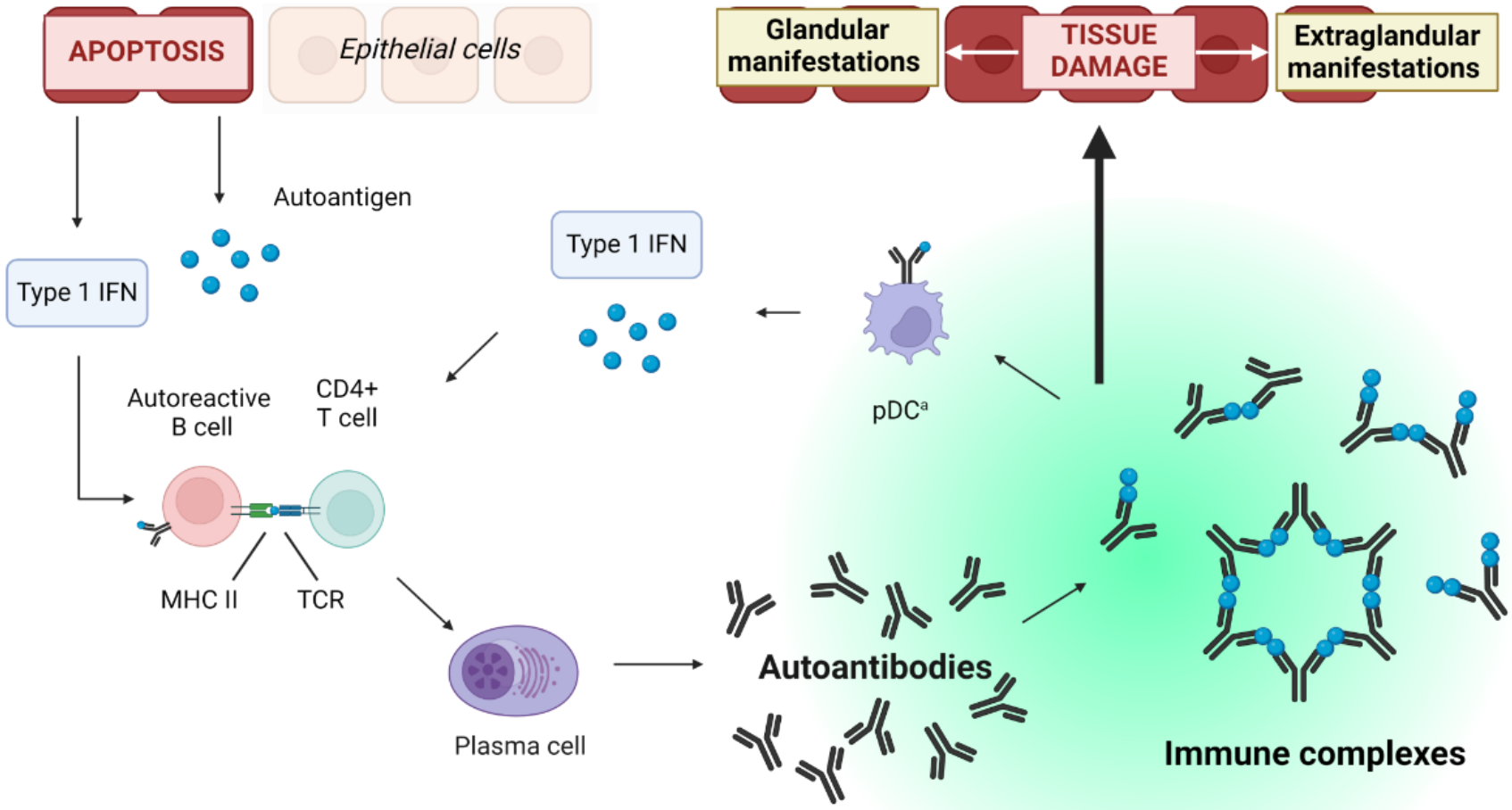
Primary Symptoms

Sicca Symptoms
Dry eye, mouth and vagina

Fatigue and Pain

Brito-Zerón P, et al. *Nat Rev Dis Primers*. 2016; Both T, et al. *Int J Med Sci*. 2017; Negrini S, et al. *Clin Exp Med*. 2022

Auto-antibodies are Key Players in Sjögren's Disease



Pathogenicity of Autoantibodies

Abnormally elevated IgG levels and presence of IgG auto-antibodies (anti-Ro/anti-La)

Auto-antibody immune complexes induce and maintain type 1 IFN signature resulting in immune-activation and tissue damage

Mariette X et al. 2018; Nocturne G et al. 2018; Pringle S, et al. 2019

RHO Trial: Proof-of-Concept in Sjögren's Disease

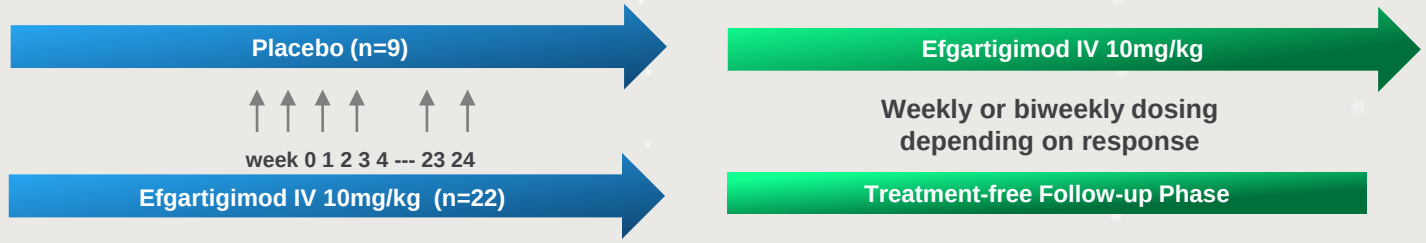
Screening Period
≤4 weeks

Key inclusion criteria

- ACR/EULAR 2016 SjD diagnosed
- ESSDAI ≥5
- Anti-Ro+
- Residual (un)stimulated salivary flow

Treatment Period
Weekly - 24 weeks

Open-label Extension
48 weeks



Demographics and baseline characteristics

- Median age 49yo (29-70)
- ~ 5 years since diagnosis
- 68% of participants with ESSDAI ≥ 10
- Majority of patients on stable dose of hydroxychloroquine and/or low dose steroids
- 50% of patients with hypergammaglobulinemia (IgG>16 g/L)

Objectives to see consistency across measures

Primary endpoint

Proportion of responders to composite of relevant endpoints for Sjögren's disease (CRESS)

Secondary endpoints

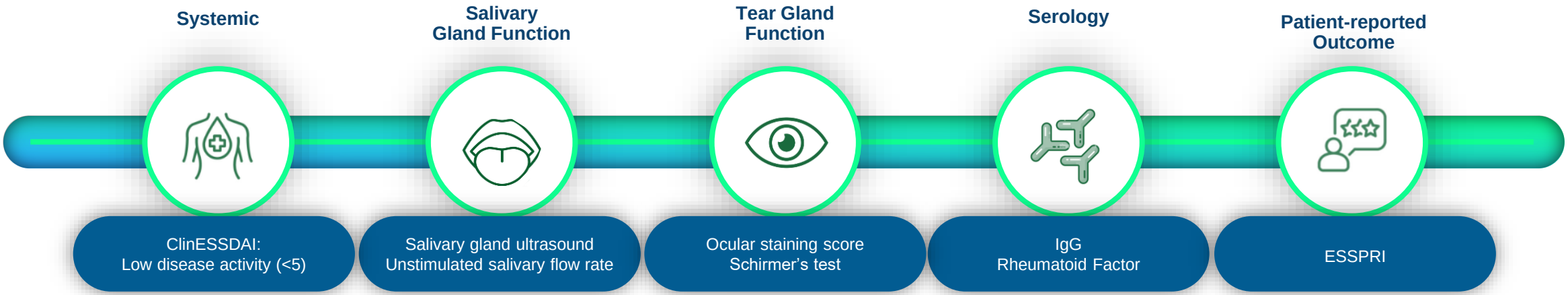
Treatment effect on

- Systemic disease (ClinESSDAI, ESSDAI)
- Patient-reported outcome (ESSPRI)
- Composite endpoint (STAR)

Biomarkers

IgG, RF, auto-antibodies, Immune complexes, IFN, histology and complement

Primary Endpoint: CRESS



OBJECTIVE:
To demonstrate more CRESS responders (at least 3 out of 5 items) at week 24 in the active arm

Arends et al. The Lancet Rheumatology, 2021

Limitations

NOVEL ENDPOINT (IN VALIDATION)

Strengths

ACCOUNTS FOR HETEROGENEOUS DISEASE

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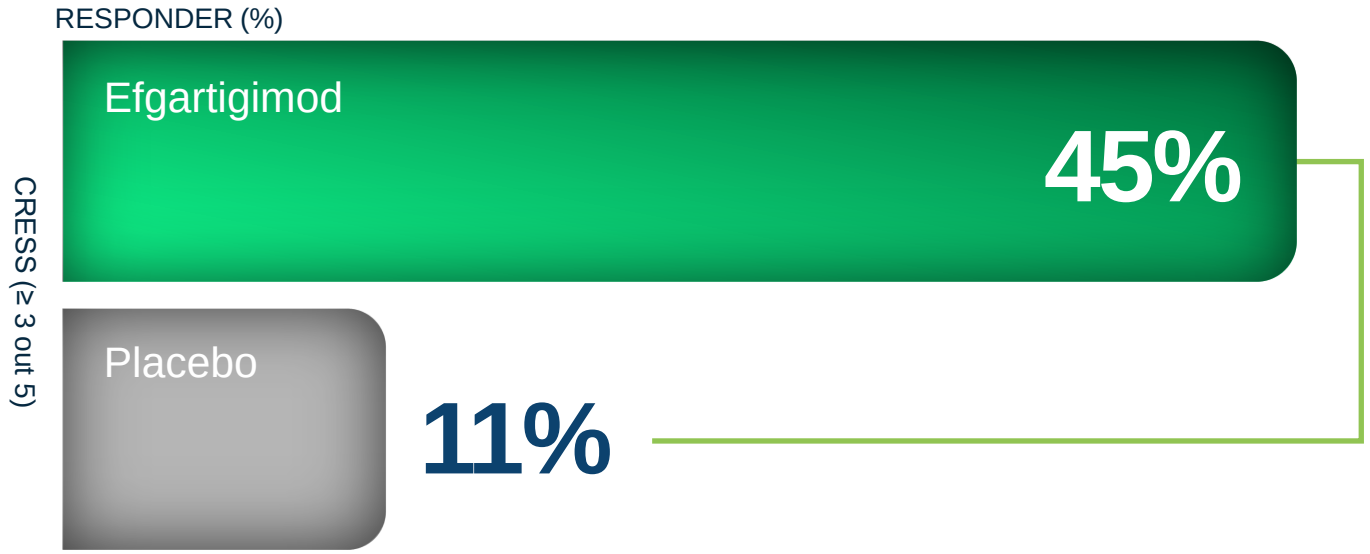
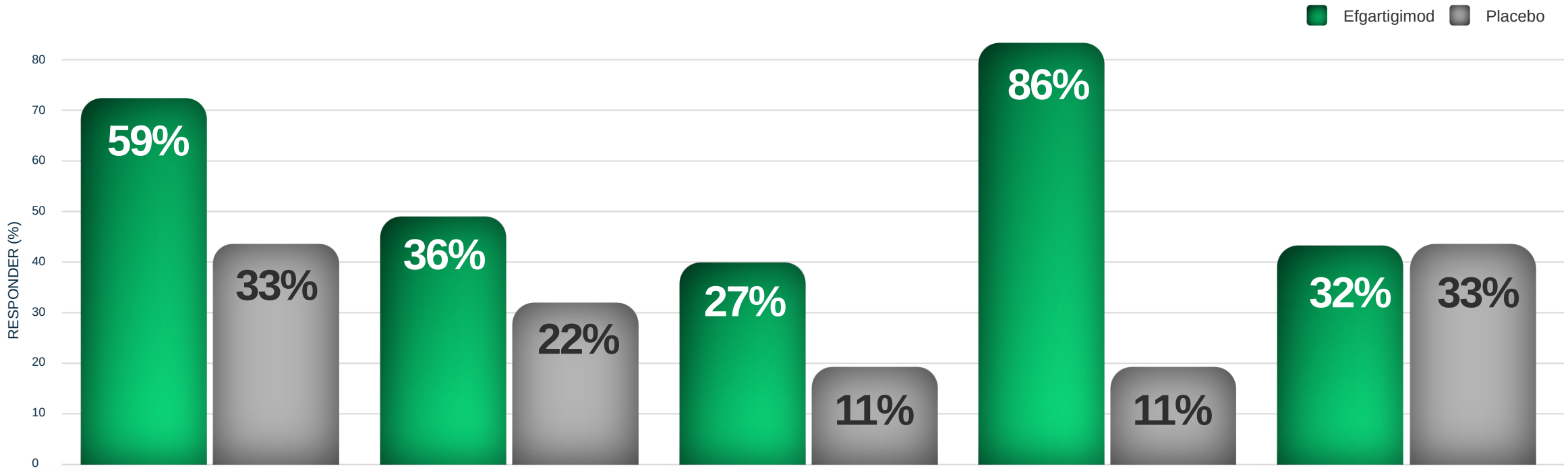
Efgartigimod Demonstrated Effect on Primary Endpoint CRESS



34%

more responders
on efgartigimod

Observed Treatment Effect in 4 Items of CRESS



Systemic

ClinESSDAI:
Low disease activity (<5)

Salivary Gland Function

Salivary gland ultrasound
Unstimulated salivary flow rate

Tear Gland Function

Ocular staining score
Schirmer's test

Serology

IgG
Rheumatoid Factor

Patient-reported Outcome

ESSPRI

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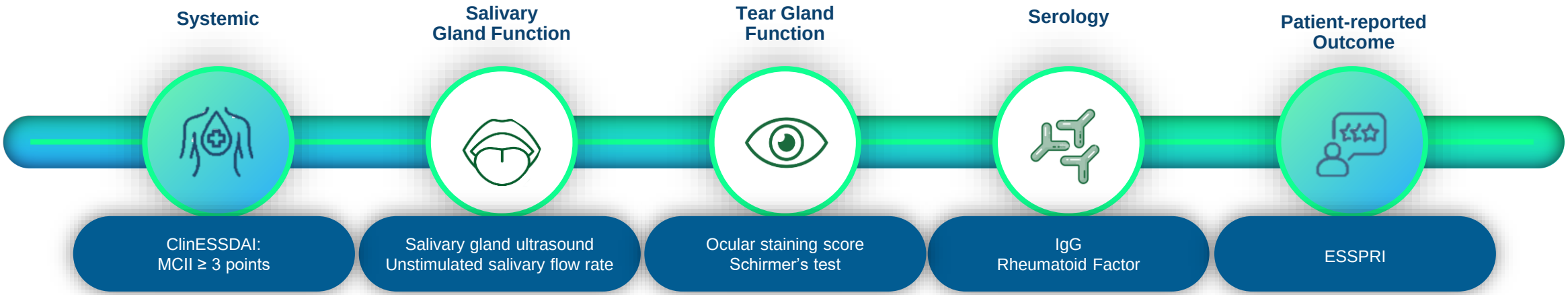
Secondary Endpoint: STAR



OBJECTIVE:
To demonstrate more STAR responders (at least 5 points) at week 24 in the active arm

CRESS ≥3 out 5	Requires response in 3 out of 5 items Responder systemic disease: ClinESSDAI < 5
STAR ≥ 5	Requires response on PRO and/or systemic disease Responder systemic disease: ClinESSDAI decrease ≥ 3

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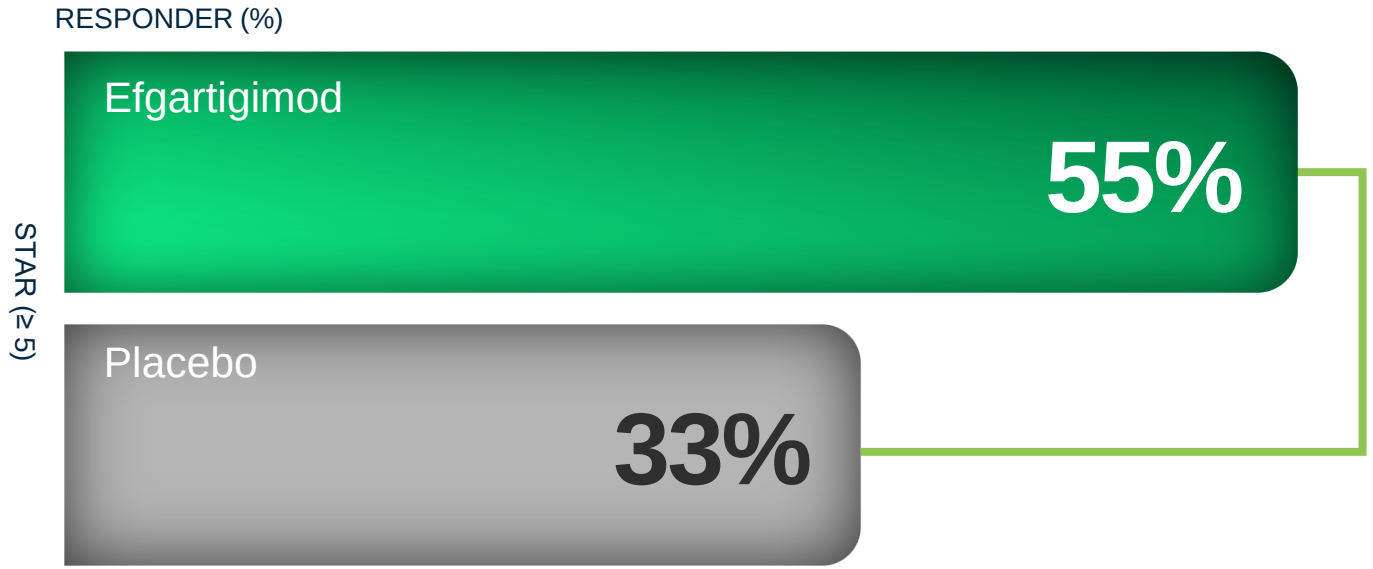
Seror R. et al. Ann Rheum Dis 2022

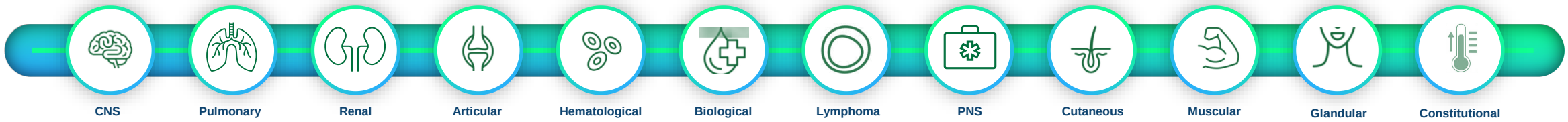
Efgartigimod Demonstrated Effect on STAR



22%

more responders
on efgartigimod

Secondary Endpoint: ESSDAI



OBJECTIVE:
To demonstrate increased response rates on ESSDAI

Limitations

- HIGH PLACEBO RESONSES
- DOES NOT CAPTURE ALL DISEASE FEATURES

Strengths

- ESTABLISHED ENDPOINT WITH FOCUS ON SYSTEMIC DISEASE SEVERITY

Seror R. et al. RMD Open 2015

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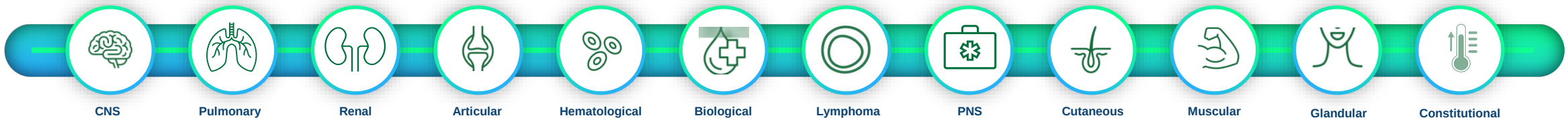
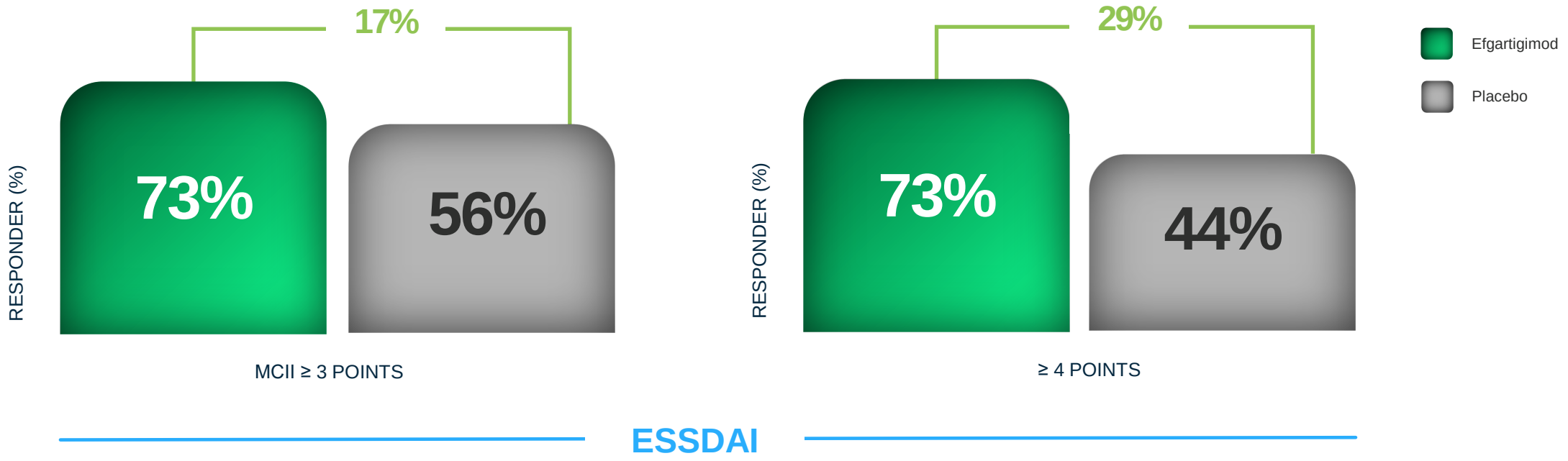
Efgartigimod Demonstrated Effect on ESSDAI





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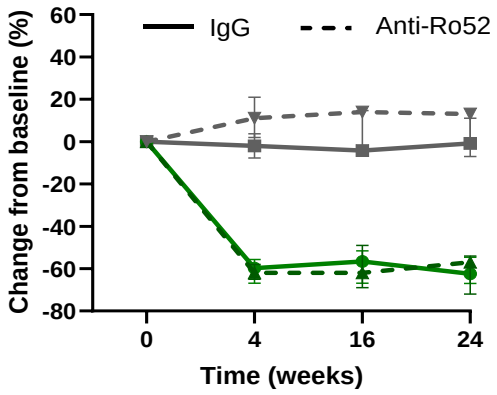
Efgartigimod Shows Potential to Break Loop of Immune Activation and Tissue Damage

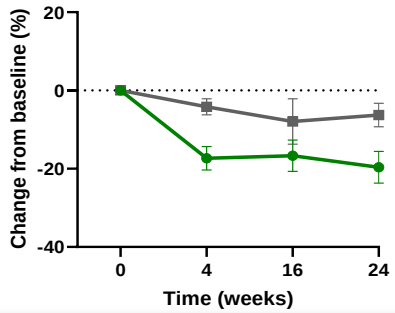
Reduction IgG and Disease-Specific Auto-Antibody



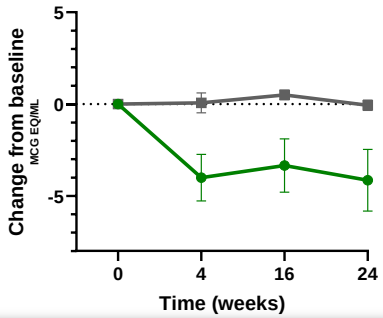
*Median ±IQR

Efgartigimod Placebo

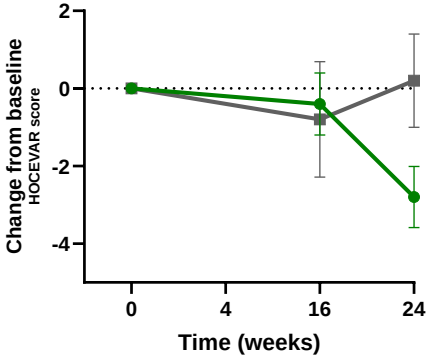
Reduction Rheumatoid Factor



Reduction C1Q Immune Complexes

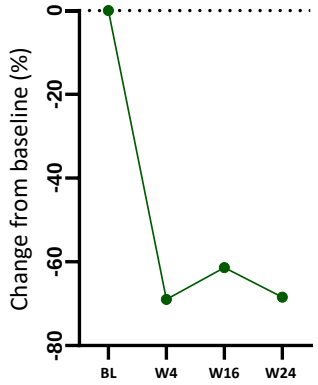


Reduction HOCEVAR Score on Salivary Gland Ultrasound

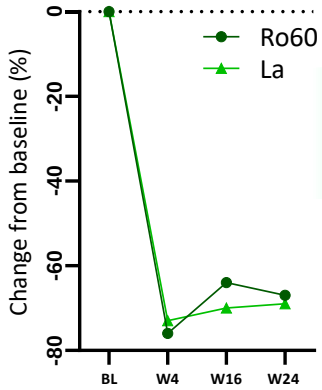


Patient Narrative Confirms Effect of FcRn Inhibition with Efgartigimod

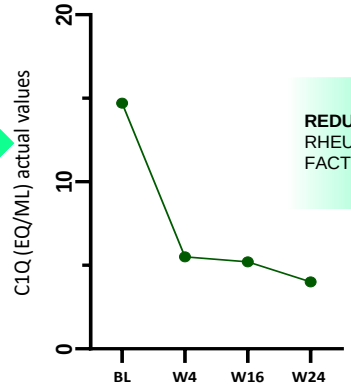
REDUCTION
IgG



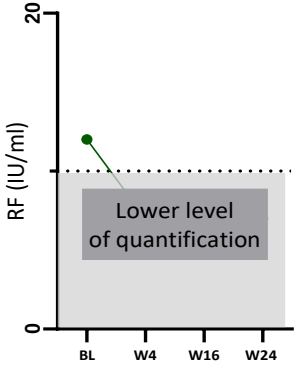
REDUCTION
DISEASE-SPECIFIC
AUTOANTIBODIES



REDUCTION
C1Q IMMUNE
COMPLEXES



REDUCTION
RHEUMATOID
FACTOR



RESPONDER ON STAR AND CRESS

REDUCED
SYSTEMIC
DISEASE

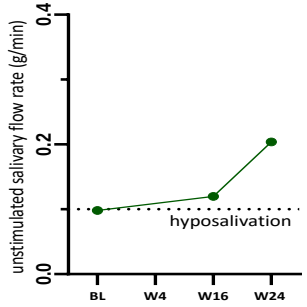
ClinESSDAI-10

GLANDULAR SWELLING NOW ABSENT

LYMPHADENOPATHY RESOLUTION

STEPWISE ARTICULAR IMPROVEMENT

IMPROVED
SALIVARY
FUNCTION



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Proof-of-Concept Established in Sjögren's Disease

60% IgG reductions consistent with other clinical trials

Reduction of auto-antibodies, immune complexes and rheumatoid factor

Increased response on composite endpoints (22-34%)

Response observed in 4 out of 5 items of CRESS

Improvement over time

Safe & well tolerated

IgG Reduction and Biomarker Data Correlate to Clinical Benefit

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Consistency of Data Demonstrates Path Forward

rho STUDY

Phase 2 Nipocalimab Data (DAHLIA Study)

Justifies Advancement To a Phase 3 Study

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Path Forward for Sjögren's Disease

End of Phase 2 Meeting

Phase 3 to Start by End of 2024

Empasiprubart

Inge Van de Walle /// Research Fellow

Brenda
Living with MMN

Multifocal Motor Neuropathy (MMN)

Characteristics

- **~1.5 years** to diagnosis
- **Progressive** and often **misdiagnosed as ALS**
- **Severe disability** in **20%** of patients
- **IVIG only** approved therapy

Disease Burden

- **Muscle weakness** and **cramping**
- **Difficulty** walking
- **Impact** on social life, activities and work
- **Exhaustion** and **fatigue**

Refs: Guimarães-Costa R et al. Presse Med. 2013. Beadon K et al. Curr Opin Neurol. 2018; Bozovic et al. J Neurol Sci.2019. Allen J et al, 2024 Mayo Clin Proc Innov Qual Outcomes

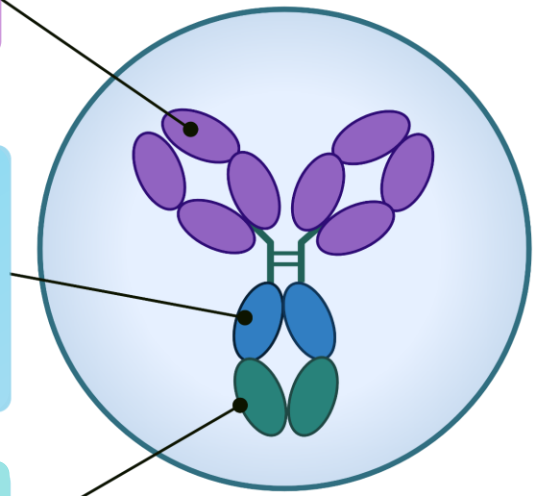
Empasiprubart

Novel C2-Specific Humanized Monoclonal Antibody With Mutations That Facilitate a Long Half-Life

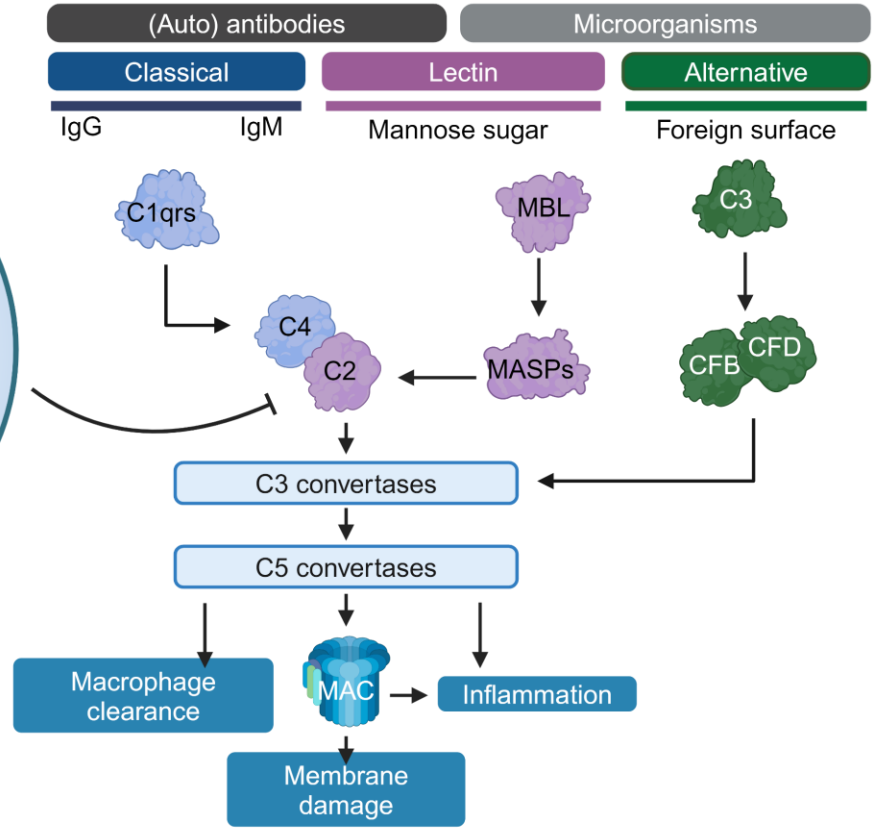
Binds to C2 in a pH- and Ca^{2+}-dependent manner

Mutations to **reduce effector functions**

NHance mutations for long half-life



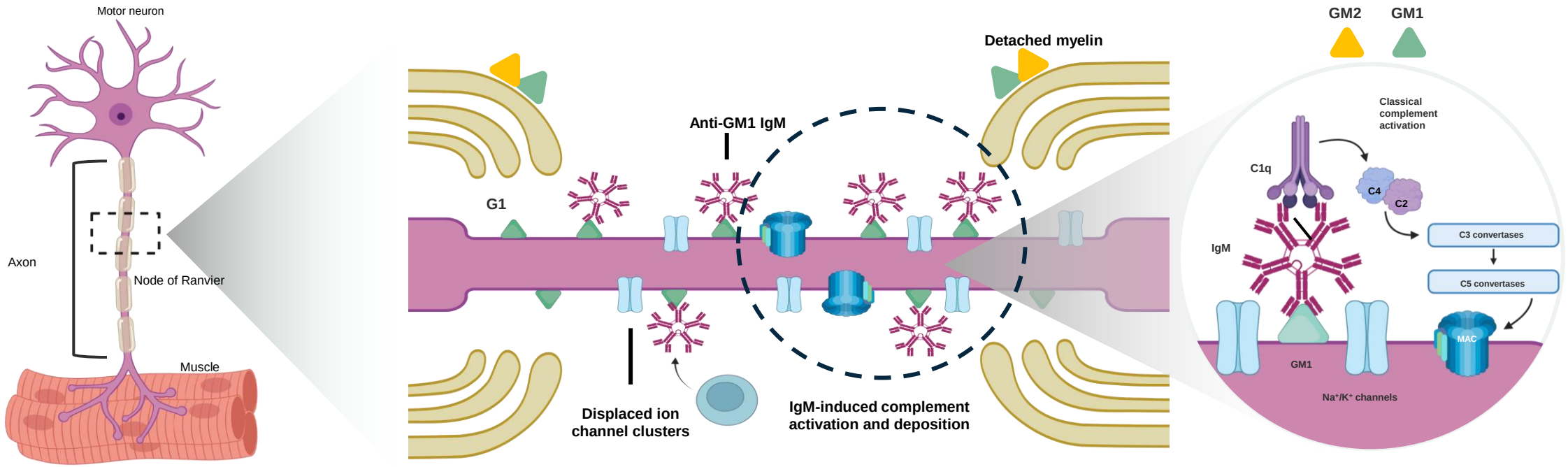
Empasiprubart



FcRn, neonatal Fc receptor.

1. Murphy K. *Janeway's Immunobiology*. 8th ed. Garland Science; 2012. **2.** Sarma JV, Ward PA. *Cell Tissue Res.* 2011;343(1):227-235. **3.** Van de Walle I, et al. *Clin Immunol.* 2021;147(4):1420-1429. **4.** Hezareh M, et al. *J Virol.* 2001;75(24):-12161-12168. **5.** Vaccaro C, et al. *Proc Natl Acad Sci.* 2006;103(49):18709-18714.

Complement Activation Drives Axonal Damage in MMN



New Learning
GM2 also plays role in subset of patients

Figure created with BioRender.com, adapted from Vlam L, et al. Nat Rev Neurol. 2011;8(1):48–58 and Sathe A, Cusick JK. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023. https://www.ncbi.nlm.nih.gov/books/NBK555995/.

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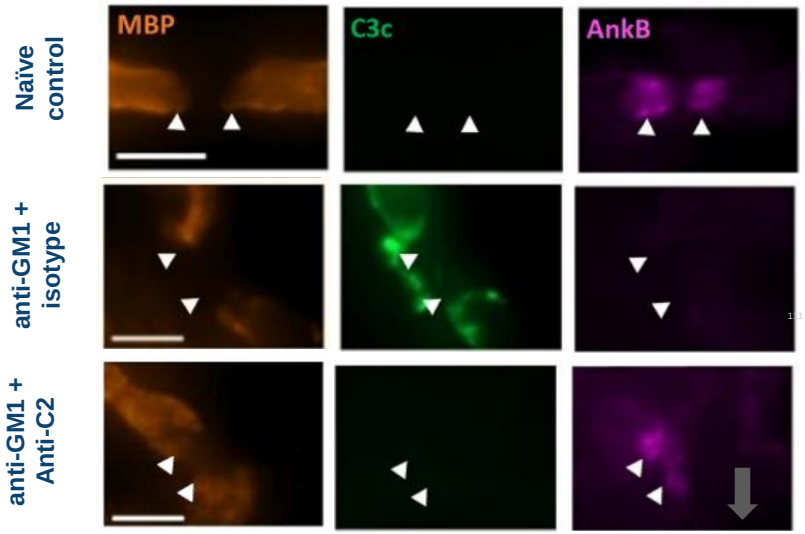
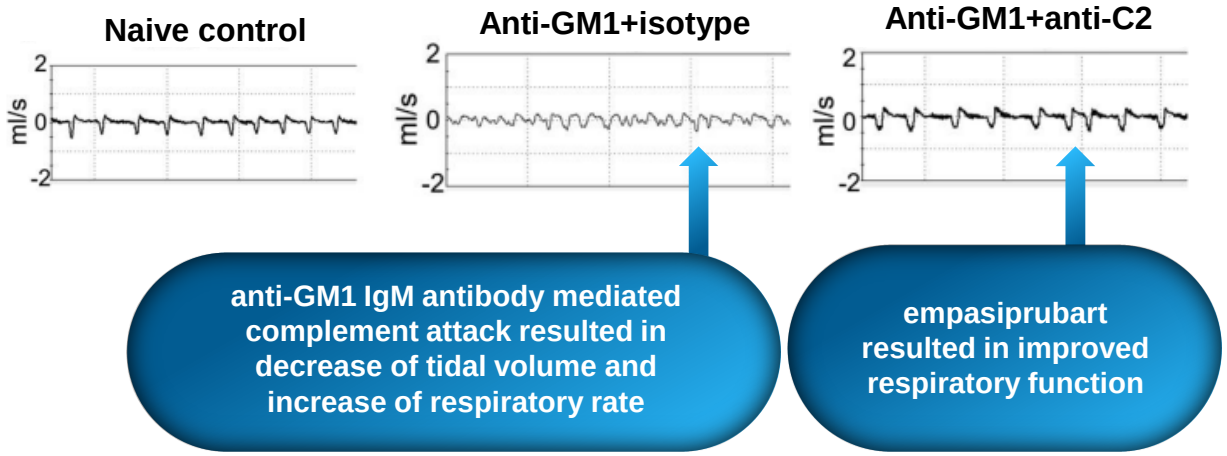
C2 Inhibition Improves Respiratory Function in vivo

C2 inhibition reduced structural injury to Schwann cell nodal membranes



anti-GM1 antibody mediated complement attack on the Schwann cell membrane → Significant disruption at node of Ranvier (hall mark of MMN) w/o empaspribrubart

Empasiprubart significantly reduced injury to paranodal proteins and improves respiratory function in vivo



Campbell CI et al. 2022 Nov 23; 4(6) fcac306

Phase 2 Trial Design

arda
Multifocal Motor Neuropathy Study

Screening (≤28 days)

MMN diagnosis and IVIg dependency (if applicable)
assessed by MMN Confirmation Committee

Uncertain | **Dependent**

Optional IVIg dependency period → **IVIg monitoring period**

≤15 weeks | **≤11 weeks**

Double-blinded Treatment Period

Cohort 1, n=16
(dosing regimen 1)
R 2:1
- **Empasiprubart IV**
- **Placebo IV**

DSMB

Cohort 2, n=16
(dosing regimen 2)
lower dose empasiprubart
R 2:1
- **Empasiprubart IV**
- **Placebo IV**

16 weeks

Enter the 15-month treatment-free safety follow-up period

OR

Rollover to the long-term extension study (ARDA+)

Primary endpoint
Safety outcomes based on AE monitoring and other safety assessments (clinical laboratory tests)

Secondary and additional endpoints
- Time to first retreatment with IVIg
- Evaluation of efficacy measures
- Evaluation of productivity, treatment satisfaction and QoL measures
- Evaluation of PK, PD, and immunogenicity

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EFNS, European Federation of the Neurology Societies; IV, intravenous; IVIg, intravenous immunoglobulin; MCC, MMN Confirmation Committee; MMN, multifocal motor neuropathy; PNS, Peripheral Nerve Society.
[a]IVIg dependency parameters are summarized in the key inclusion criteria, full details provided at https://www.clinicaltrials.gov/study/NCT05225675. [b]The length of the monitoring period will depend on an individual's IVIg dose frequency: dosed every 2 weeks–up to 35 days monitoring, dosed every 3 weeks–49 days monitoring, dosed every 4 weeks–63 days monitoring, dosed every 5 weeks–77 days monitoring. [c]Double-blinded treatment period will begin 7 days after final IVIg administration. Participants will be retreated with IVIg if there is a clinically meaningful deterioration in muscle strength and/or motor function.

1. ClinicalTrials.gov identifier: NCT05225675. Updated July 20, 2023. Accessed April,2024. https://www.clinicaltrials.gov/study/NCT05225675. **2**. van der Pol, WL, et al. Poster presented at: NMSG Annual Scientific Meeting; September 22–24, 2023; Orlando, FL.

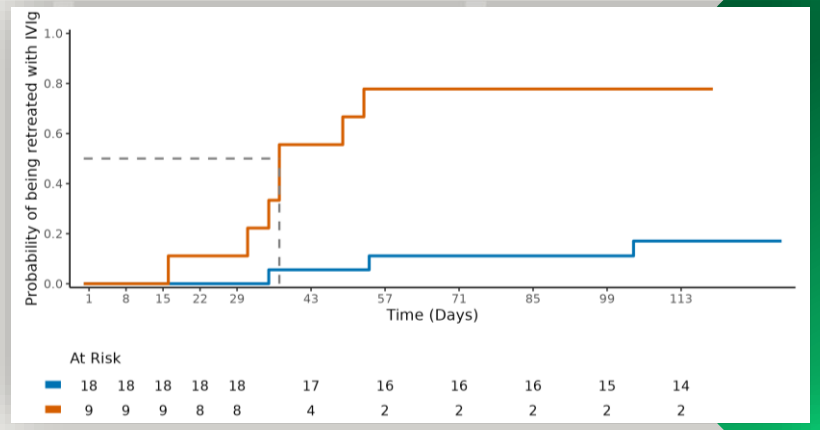
ARDA Study Results

Jeff Guptill /// Neuromuscular Franchise Lead Clinical Development

Empasiprubart Reduced Risk of IVIg Retreatment

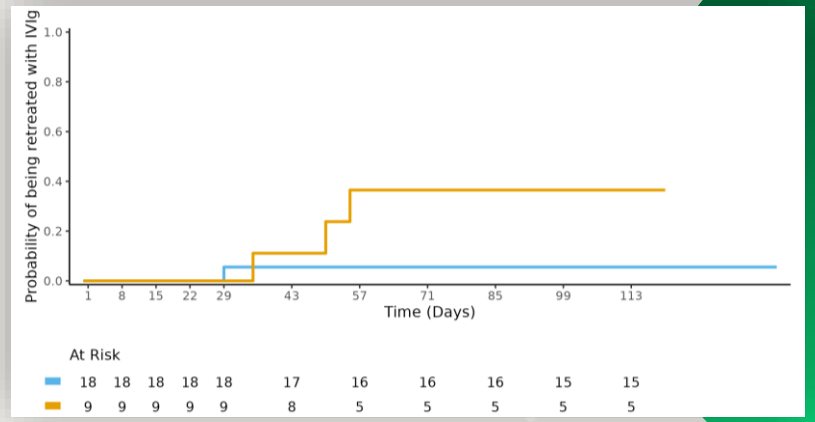
arda Multifocal Motor Neuropathy Study

Cohort 1



Reduced risk of IVIG retreatment by **91%**

Cohort 2



Reduced risk of IVIG retreatment by **84%**

Empasiprubart Placebo

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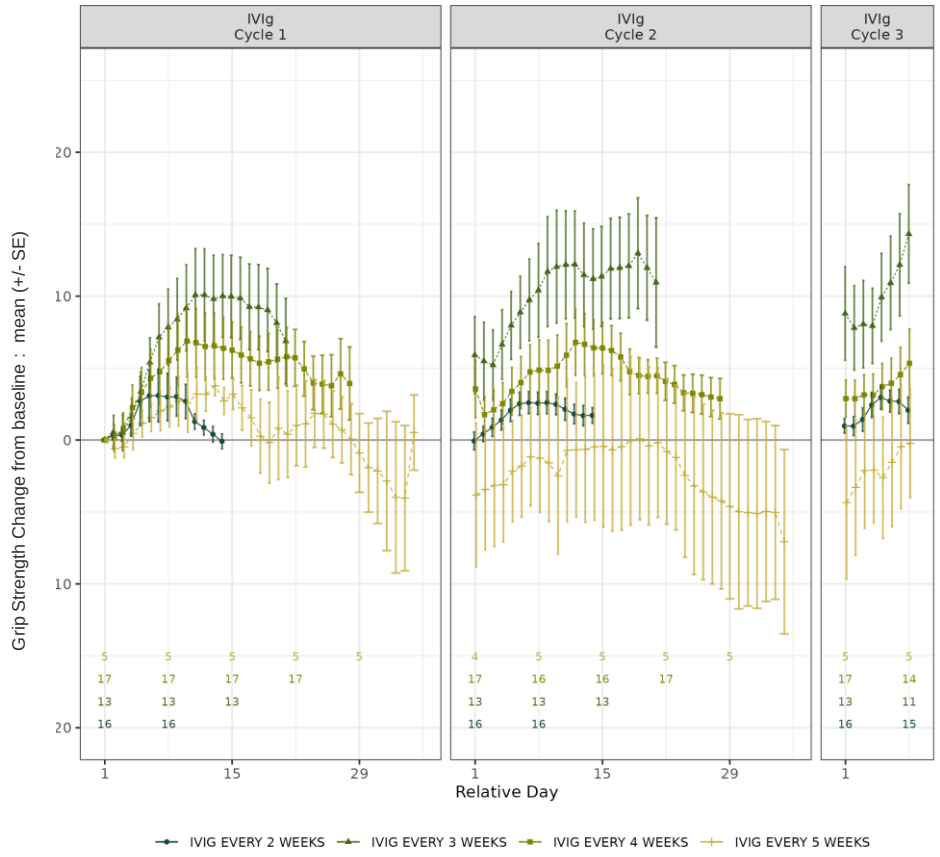
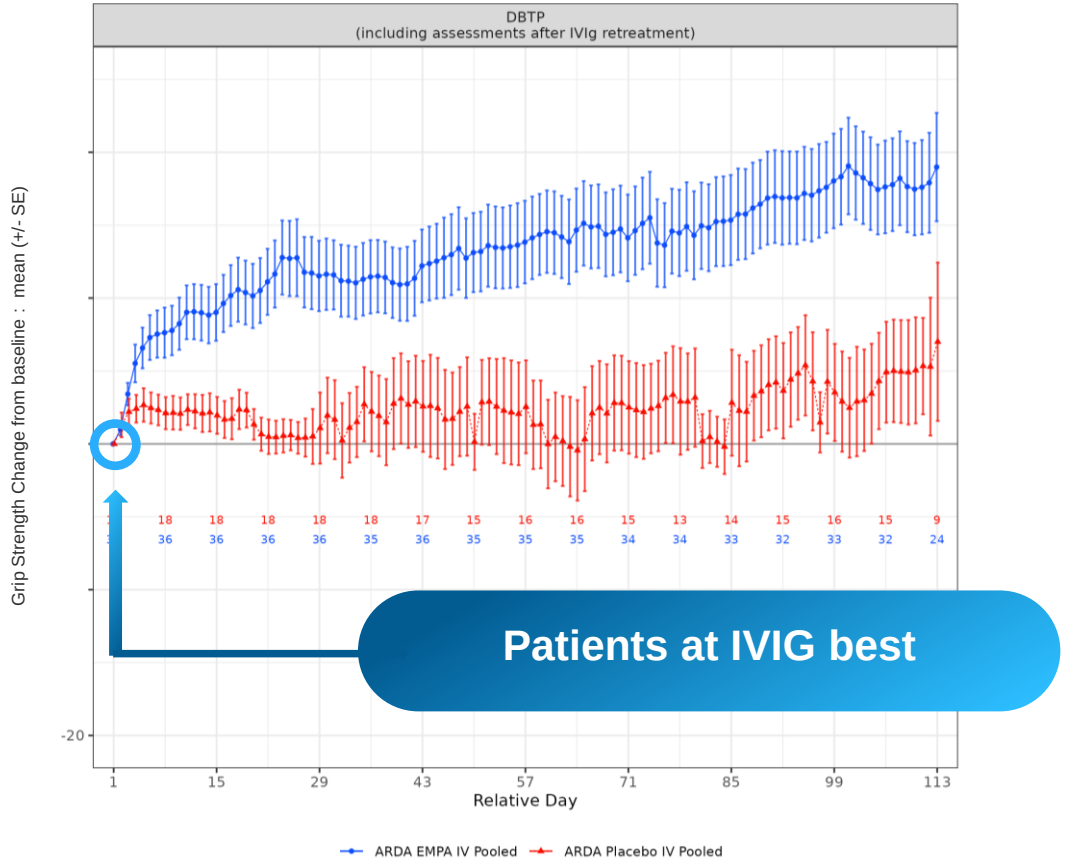
CI, confidence interval; DBTP, double-blinded treatment period; IVIg, intravenous immunoglobulin.
[a]Time to first treatment with IVIg is defined as the time from last IVIg administration before randomization (including unscheduled visits) up to the first IVIg retreatment during the DBTP.

Empasiprubart Improved Grip Strength in Both Hands

IVIg Treatment → Clear Fluctuating Effect



Grip Strength

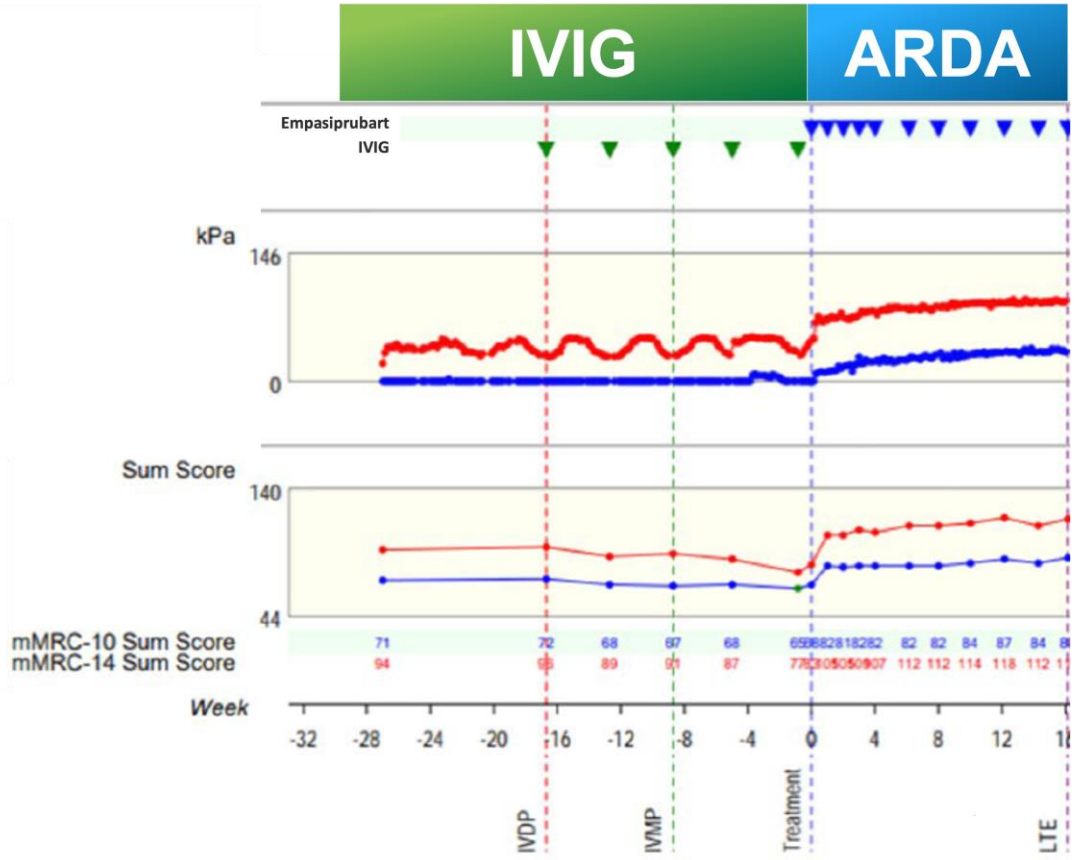


ARDA Participant Journey

Treatment

Grip Strength

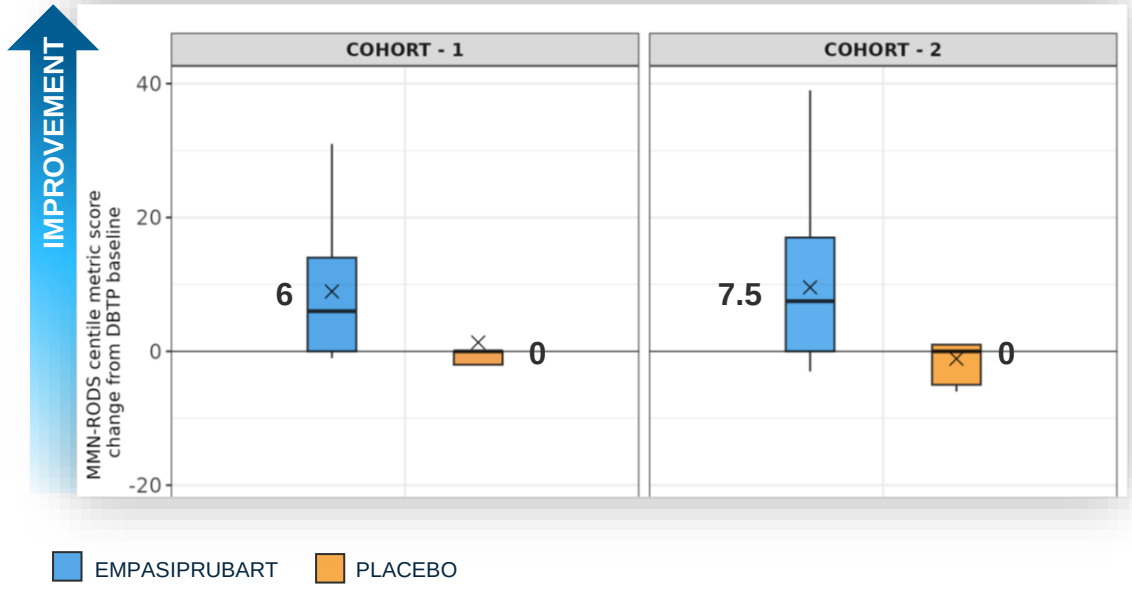
mMRC



Empasiprubart Improved Disease-Specific Activity Limitations Indicating Improvement in Functionality Levels

arda
Multifocal Motor Neuropathy Study

Change From Baseline of MMN-RODS Score by Treatment Group at Last Assessment During Treatment Period



Are you able to:

- Read a book?
- Make a telephone call?
- Eat?
- Open and close a door?
- Dress your upper body?
- Brush your teeth?
- Drink out of mug/glass?
- Turn a key in a lock?
- Use knife/fork (spoon)?
- Clean after toilet?
- Fill in a form/write?
- Zip your trousers?
- Get money from cash machine?
- Do your own cooking?
- Pick up small object?
- Work on a computer?
- Do the bed?
- Fold laundry?
- Throw an object (e.g., ball)?
- Slice vegetables?
- Peel an apple/orange?
- Handle small objects (e.g., coin)?
- Tie your laces?
- Clip your finger nails?
- Button your shirt/blouse?

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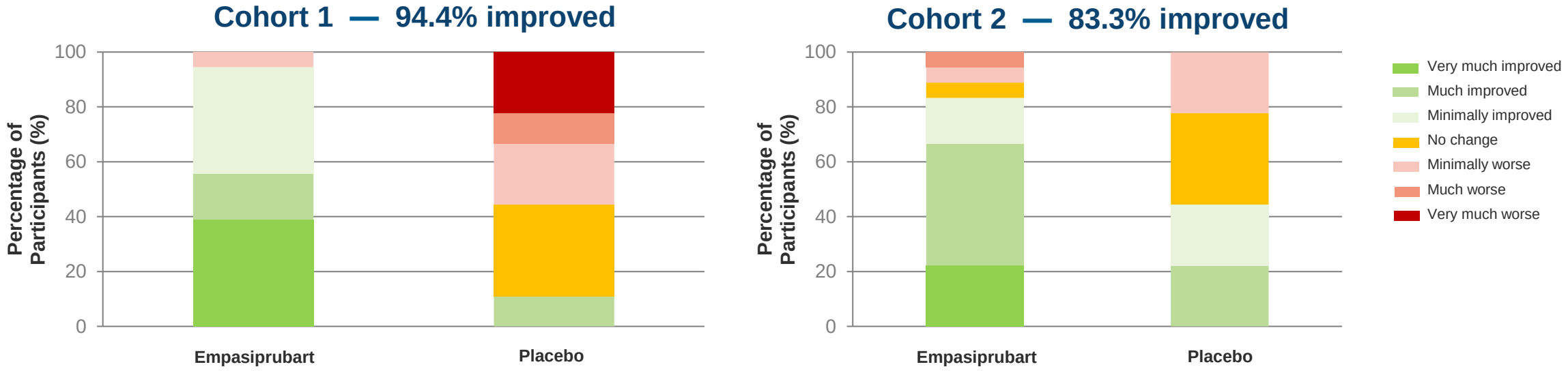
Empasiprubart Treated Patients Feel Better than their Best on IVIG

How much has your condition (MMN) changed as compared to the time you received the first treatment in this trial?

arda Multifocal Motor Neuropathy Study



PGIC, Patient Global Impression of Change

Consistent improvement observed for each dose of empasiprubart

Patient global impression of change

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Path Forward for MMN

End of Phase 2 Meeting 3Q 2024

Phase 3 to Start in 4Q 2024

IMMERSION STUDY

Trials Ongoing with Empasiprubart

Delayed Graft Function

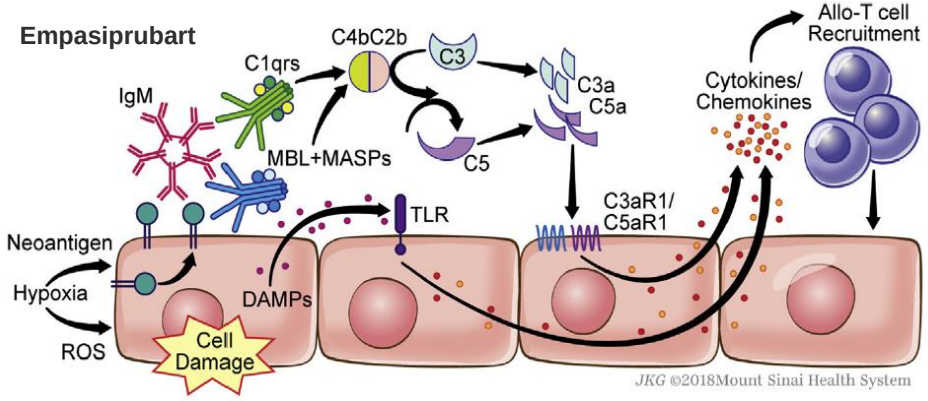
Dermatomyositis

120

Empasiprubart in Delayed Graft Function After Kidney Transplant

varvara
Delayed Graft Function Study

Biological Rationale



- Complement activation due to damaged endothelial
- Clear involvement of Classical and Lectin Pathways
- Blocking C2 improved kidney function

Disease Characteristics

- **40%** occurrence among cold kidney transplants
- Ischemia reperfusion injury (IRI) contributor to DGF
- Short and long-term graft negative effects
- **No current FDA-approved therapies**

Timeline Phase 2 ongoing

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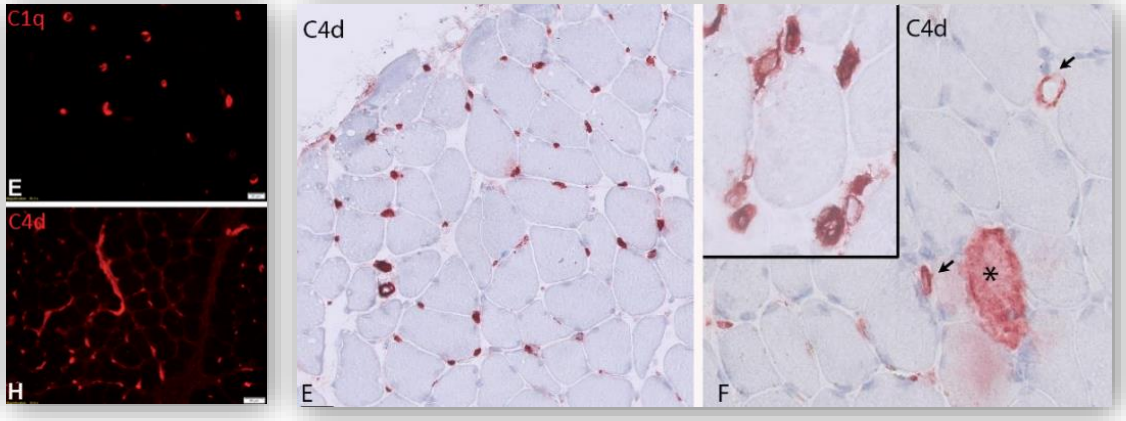
1. Pascual J, et al. *Am J Kidney Dis.* 2008;52(3):553-586. **2.** Biglarnia AR, et al. *Nat Rev Nephrol.* 2018;14(12):767-781. **3.** Yarlagadda SG, et al. *Nephrol Dial Transplant.* 2009;24(3):1039-1047. **4.** Castellano G, et al. *Am J Pathol.* 2010;176(4):1648-1659. **5**. Horwitz JK, et al. *Clin Lab Med.* 2019;39(1):31-43.

Empasiprubart in Dermatomyositis

empacific
Dermatomyositis Study

Complement Deposition in Biopsies



Disease Characteristics

- **Multifactorial, idiopathic inflammatory myopathy**
- **Progressive** and **symmetric** proximal muscle weakness
- **IVIg is only approved treatment**

Timeline Phase 2 study planned to start this year

a. Basta et al. 1994; Pytel, Appl Immunohistochem Mol Morphol. 2014 Oct;22(9):696-704; b. Campo et al. 2007; c Lahoria et al,. Brain. 2016 Jul;139(Pt 7):1891-903 d. Emslie-Smith and Engel. 1990; e. Dalakas. 2015.

2. Dalakas et al., Lancet. 2003 Sep 20;362(9388):971-82 ; **3.** Dalakas, Nat Clin Pract Rheumatol. 2006 Apr;2(4):219-27; Aggarwal R, Charles-Schoeman C, Schessl J, Dimachkie MM, Beckmann I, Levine T.Medicine (Baltimore). 2021 Jan 8;100(1):e23677

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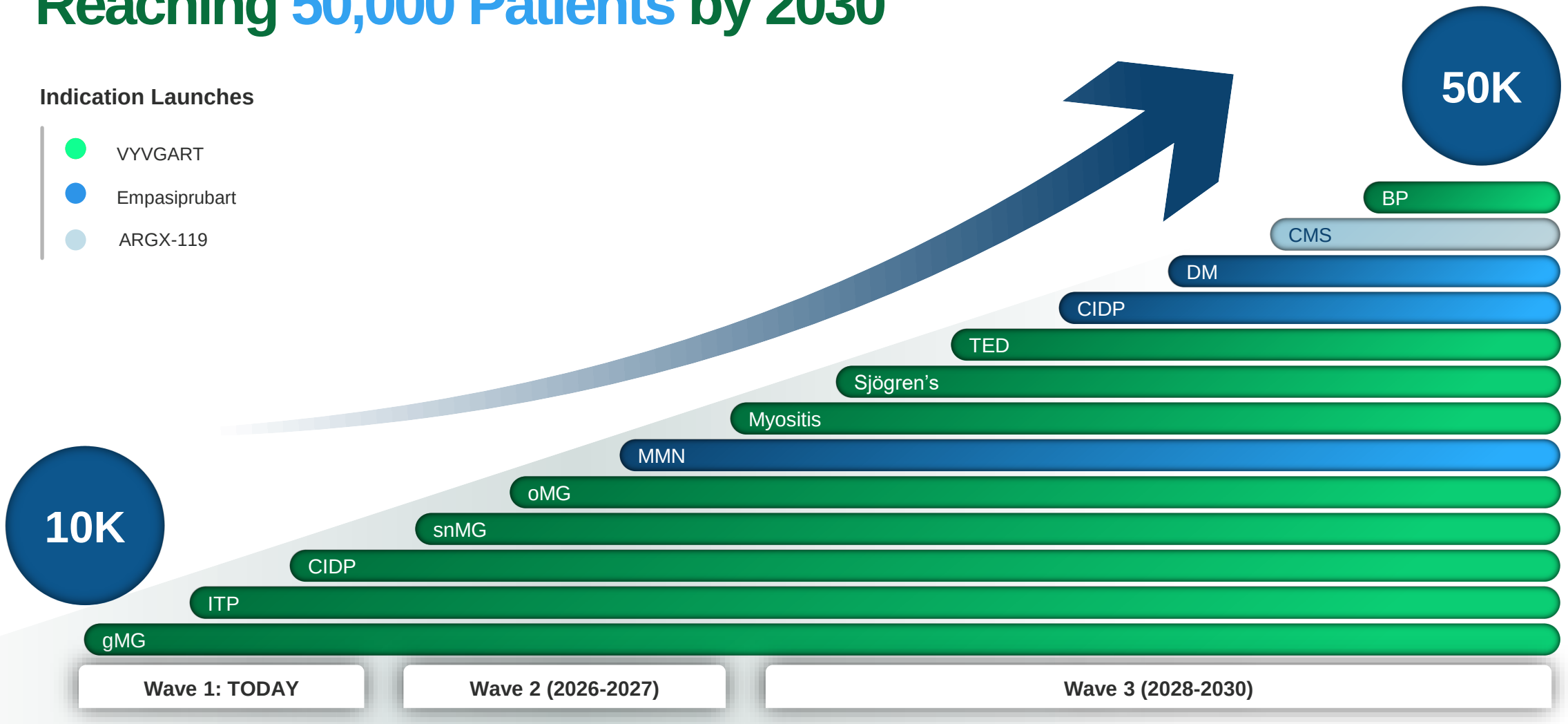
Our Next Pipeline-in-a-Product Asset

Sustainable Commercial Engine

Karen Massey /// Chief Operating Officer

124

Reaching 50,000 Patients by 2030



MG Launch Set Standard on Commercial Excellence

Innovation

Evidence Generation

~50% MSE demonstrated across trials,sustained response across 9 cycles

Meaningful **steroid reduction**

adapt NXT myasthenia gravis study

Co-Creation

Empowering Patients

Direct to Consumer engagement

92% Brand awareness

My VYVGART® Path

Execution

Speed

>2,700 US prescribers

#1 among advanced biologics

Approved across **3 continents** within one calendar year

>$1BN in year 2 of launch | **9 quarters of growth** | **>10,000 patients globally**

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Future Drivers of Growth in MG

Innovation

Evidence Generation

adapt myasthenia gravis study Oculus

adapt myasthenia gravis study SERON

Co-Creation

Empowering Patients

Product Presentations

PFS

Autoinjector

Execution

Speed

Global Expansion

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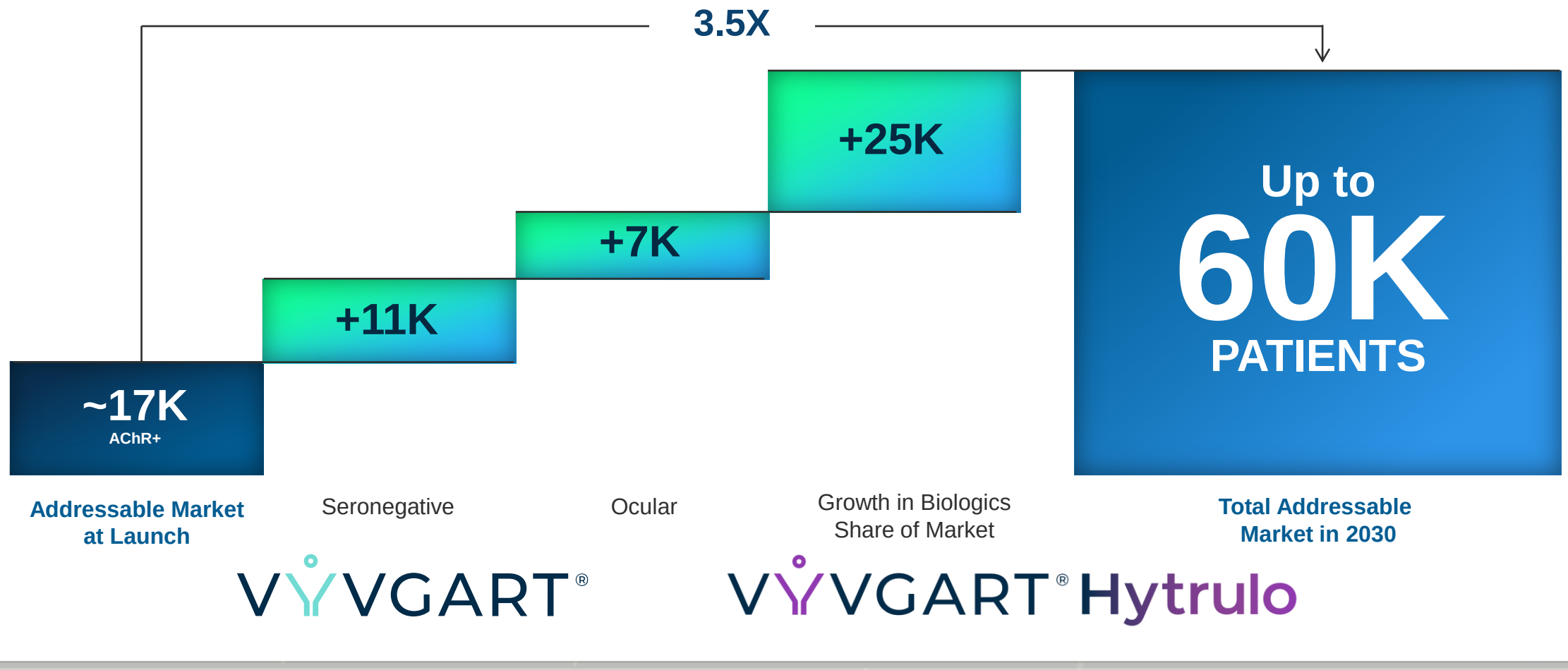
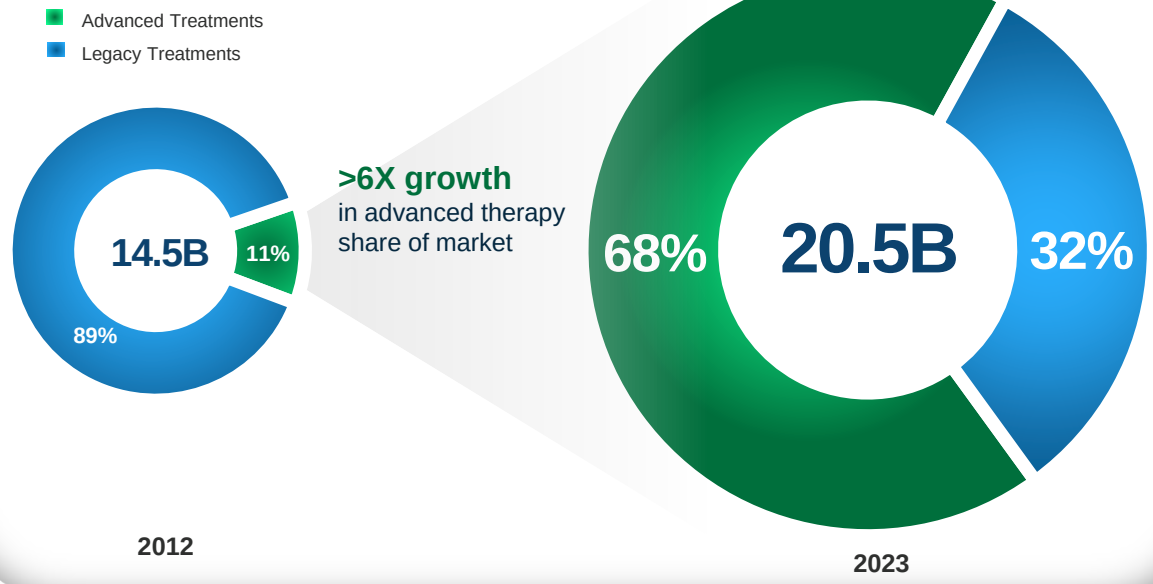
Expanding MG Opportunity



Innovation Builds Markets: MG Market Dynamics are Similar to MS

Multiple Sclerosis Market



Over 10+ Year Period… Market Growth was Driven by

Novel mechanisms of action

Multiple launched assets

~15% prevalence increase

More Innovation = More Prescribers, Better Outcomes for More Patients

argenx market research

Early Excitement in CIDP

Rapid Execution

25% of key target physicians
reached in 14 days

First payor policies in principle

Early Adoption

Prescriber breadth and depth
~20% are new to VYVGART

First patients on treatment

MMN: Opportunity to Build a Market

MMN Today

10K
PATIENTS

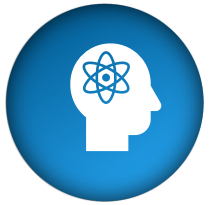
More Innovation =
More Prescribers,
Better Outcomes
For Patients

The argenx advantage

Innovation

Natural History Study to understand real-world experience

Co-creation



Engagement with patients

GBS|CIDP Foundation International

Execution

Deep **existing neurology relationships**

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TED and Sjögren's Disease Represent MG Sized Opportunities

Path to Transforming Outcomes with Differentiated Treatments

Thyroid Eye Disease

100K Prevalence → **~80K** Chronic Patients

- Limitations of existing therapies, considering safety and efficacy

Sjögren's Disease

330K Prevalence → **~100K** Moderate to Severe

- No currently approved treatments to target underlying disease

*U.S. prevalence numbers; argenx market research

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Vision 2030

5 New Molecules in Phase 3

10 Labeled Indications

50k Patients on Treatment

COMMITMENT TO OUR TRANSFORMATION MISSION

- Continuous Pipeline of Innovation
- Leadership in FcRn
- Disciplined Scaling

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Innovation has no meaning unless it reaches patients and provides real benefit

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