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

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**FORM 6-K**

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**REPORT OF FOREIGN PRIVATE ISSUER  
PURSUANT TO RULE 13a-16 OR 15d-16  
UNDER THE SECURITIES EXCHANGE ACT OF 1934**

**For the Month of July 2024**

**Commission File Number: 001-38097**

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**ARGENX SE**

(Translation of registrant's name into English)

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**Laarderhoogtweg 25  
1101 EB Amsterdam, the Netherlands**  
(Address of principal executive offices)

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Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.

Form 20-F  Form 40-F

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1):

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7):

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**EXPLANATORY NOTE**

On July 16, 2024, argenx SE (the "Company") issued an investor presentation, a copy of which is attached hereto as Exhibit 99.1, and is incorporated by reference herein.

*The information contained in this Current Report on Form 6-K, including Exhibit 99.1, shall be deemed to be incorporated by reference into the Company's Registration Statements on Forms F-3 (File No. [333-258251](#)) and S-8 (File Nos. [333-225375](#), [333-258253](#), and [333-274721](#)), and to be part thereof from the date on which this Current Report on Form 6-K is filed, to the extent not superseded by documents or reports subsequently filed or furnished.*

<b>Exhibit</b>	<b>Description</b>
<a href="#">99.1</a>	<a href="#">Investor Presentation dated July 16, 2024</a>

**SIGNATURES**

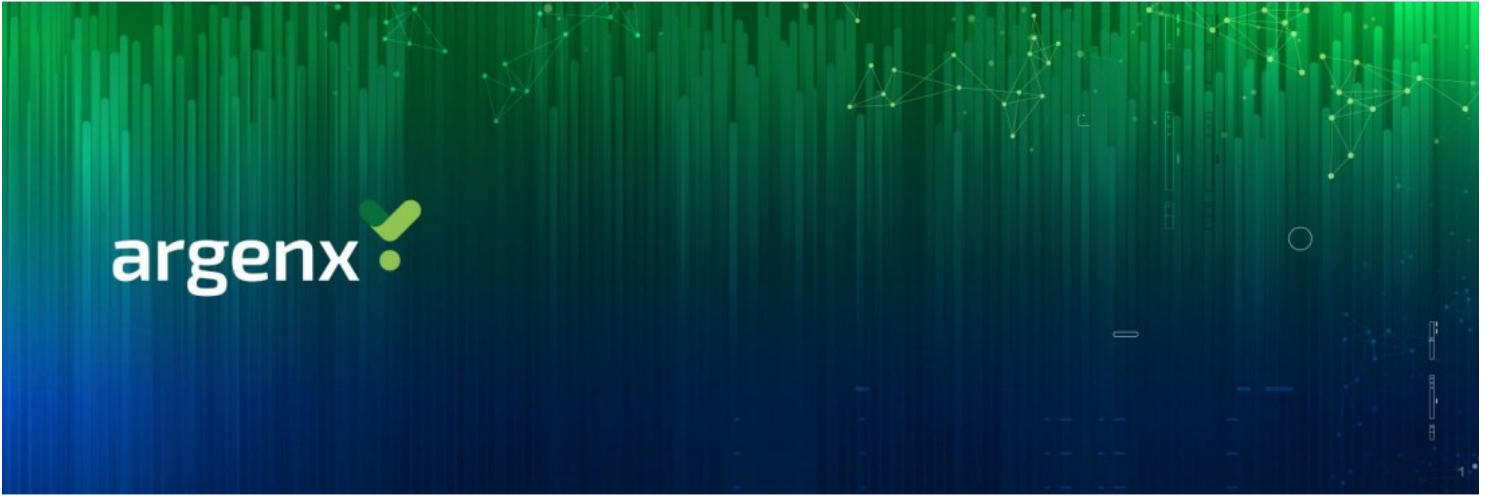
Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

**ARGENX SE**

Date: July 16, 2024

By: /s/ Hemamalini (Malini) Moorthy  
Name: Hemamalini (Malini) Moorthy  
Title: General Counsel

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# Welcome & Opening Remarks

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

## Forward Looking Statements

This presentation has been prepared by argenx se ("argenx" or the "company") for informational purposes only and not for any other purpose. Nothing contained in this presentation is, or should be construed as, a recommendation, promise or representation by the presenter or the company or any director, employee, agent, or adviser of the company. This presentation does not purport to be all-inclusive or to contain all of the information you may desire. Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the company's own internal estimates and research. While argenx believes these third-party studies, publications, surveys and other data to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, no independent source has evaluated the reasonableness or accuracy of argenx's internal estimates or research, and no reliance should be made on any information or statements made in this presentation relating to or based on such internal estimates and research.

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 emapiciclib in MMN; the anticipated timing of the Phase 2 EMPACIFIC clinical trial, the anticipated timing of the analysis for the Phase 2 ALKNIVA 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 providers 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.

This presentation contains trademarks, trade names and service marks of other companies, which are the property of their respective owners.

The logo for argenx, featuring the word "argenx" in a white, lowercase, sans-serif font. To the right of the text is a stylized graphic consisting of two overlapping shapes: a larger, light green shape resembling a checkmark or a stylized 'A', and a smaller, solid green circle below it.

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# Agenda

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

argenx 

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

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

Novel Disease  
Biology Insights

Foundational  
Immune  
Targets

Best-in-Field  
Antibody  
Engineering

First-in-Class  
Antibodies

Pipeline-in-  
a-Product  
Development

Differentiated  
Patient  
Outcomes



## Co-Creation is Our Innovation Formula

### WORLD CLASS ANTIBODY ENGINEERING CAPABILITY

#### V-REGION CAPABILITIES

SIMPLE  
Antibody™  
Platform



argenx

Collaborators

### DISEASE BIOLOGY INSIGHTS



## Our Innovation Model Has A Strong Track Record

IL6

CD70

CMET

IL22R

FcRn

GARP

ApoC3

C2

Galectin-10

MuSK

IgA


'ARGX-220'

**8/12** demonstrated human POC

**9** first-in-class targets

**5** partnered

Broad applicability **across 35+ indications**

argenx 

# 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

Committed to our Patients and their Communities

Rooted in Science through our IIP

Enviably Immunology Pipeline

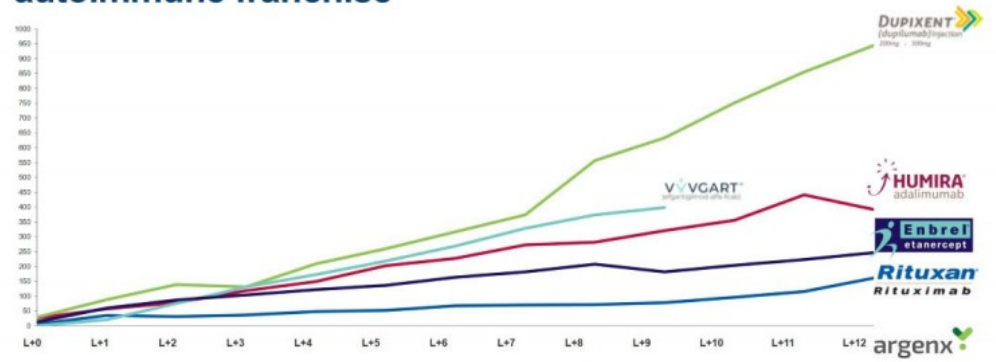
## VYVGART is a Global Blockbuster

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



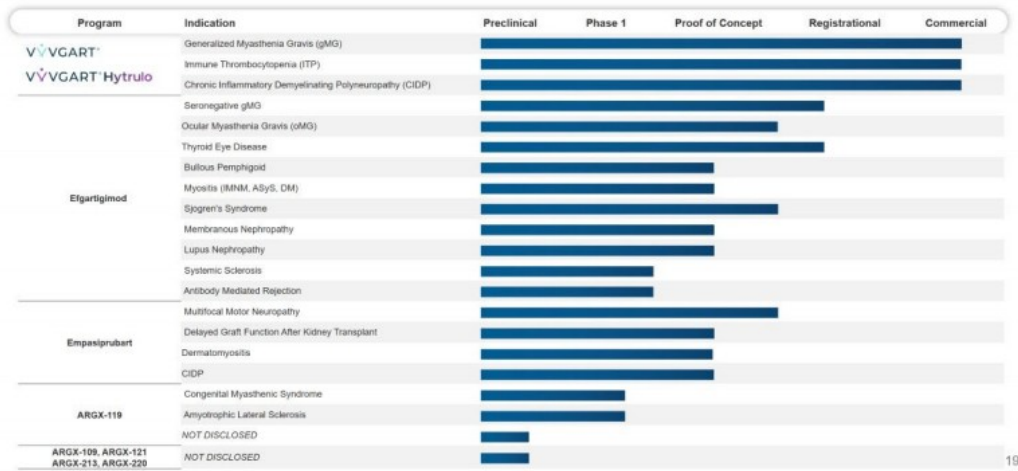
# Robust Pipeline of Multi-Indication Assets

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<sup>1</sup>

VYVGART and VYVGART Hytrulo<sup>2</sup> approved across 3 continents within one calendar year

## Reaching Patients Across the Globe

 VYVGART<sup>®</sup>  
 VYVGART<sup>®</sup> Hytrulo



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

## Staying True to our Scientific Roots

From IIP to marketplace, science is our common language

Robust patent portfolio

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

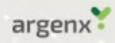




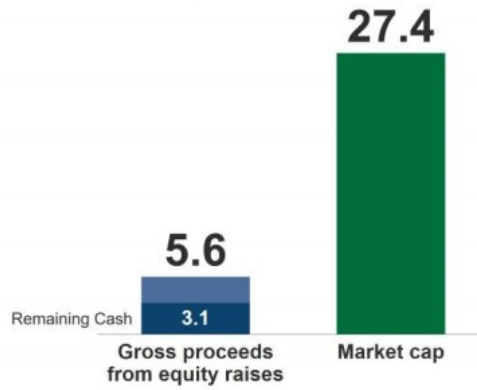
## Creating Superior Shareholder Value on our Path to Self-Sustainability

Rapid transition to sustainable company

Disciplined scaling



Total Shareholder Return since IPO in 2014  
\$B



## Vision 2030

5

New Molecules  
in Phase 3

10

Labeled  
Indications

50k

Patients on  
Treatment

### COMMITMENT TO OUR TRANSFORMATION MISSION

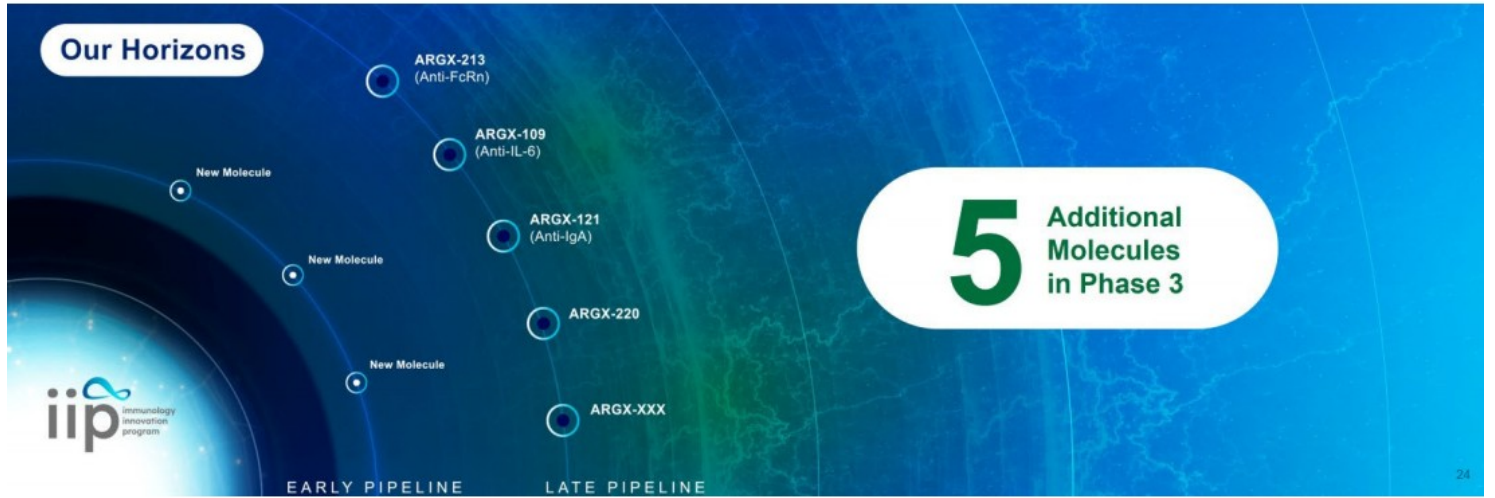
Continuous Pipeline of Innovation

Leadership in FcRn

Disciplined Scaling

argenx 

## Our Horizons



# 10

Labeled Indications

## In-Market Expansion



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

## Next Wave of Potential Launches



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

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

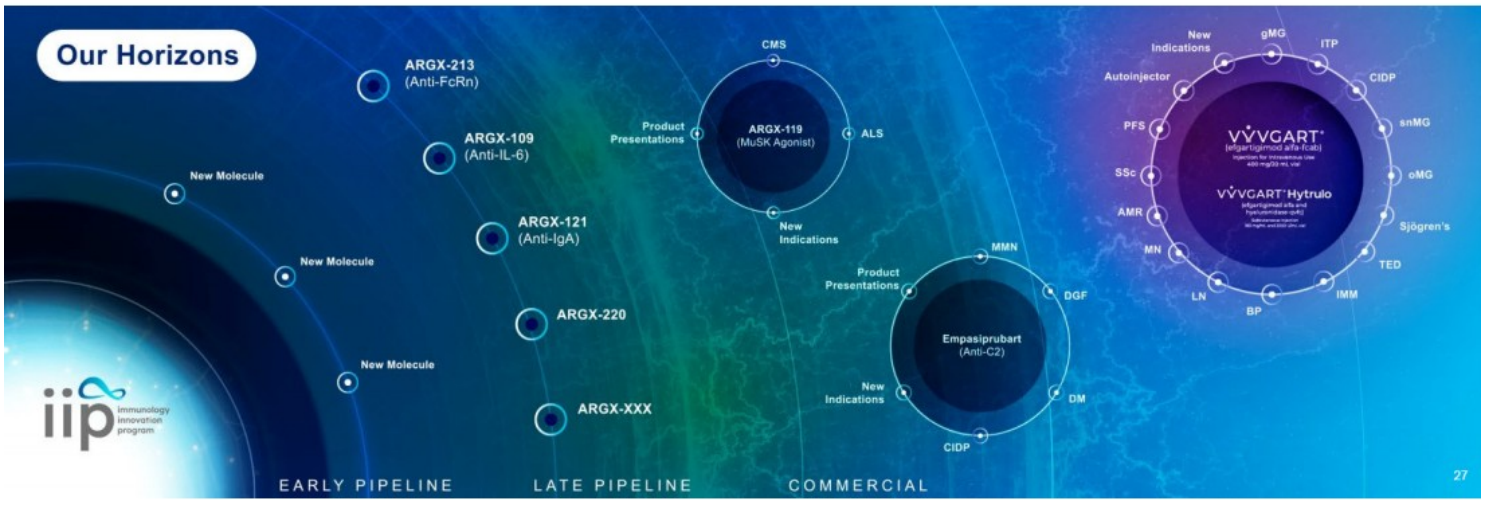


Innovation has no meaning unless it reaches patients and provides real benefit

argenx <sup>26</sup>

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# Our Horizons



argenx



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

Peter Ulrichs /// Chief Scientific Officer



## Immunology Innovation Program: Model of Co-Creation

**ARGX-113**

Efgartigimod

Foundational  
Immune Targets

**ARGX-117**

Empasiprubart

First-in-Class  
Antibodies

**ARGX-119**

Differentiated Patient  
Outcomes

# ARGX-117

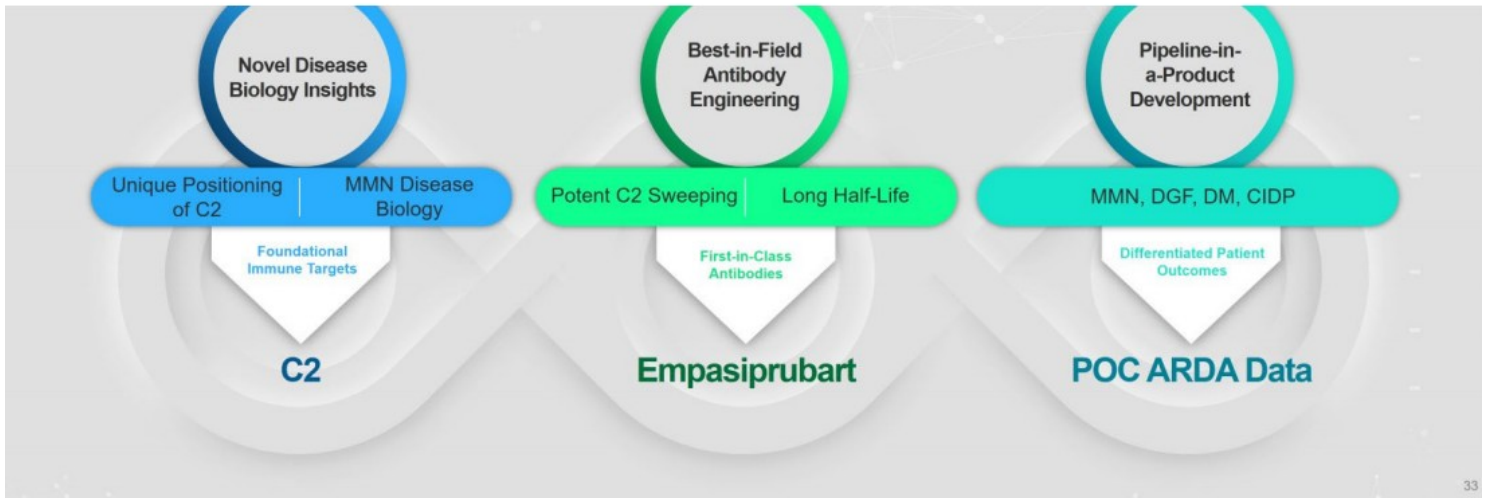
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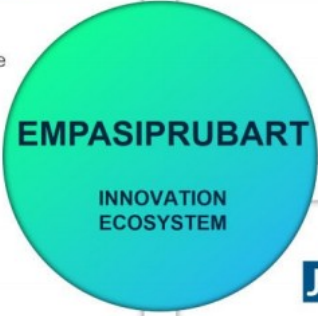
## Unraveling Central Role of C2 in Complement Cascade

Novel Disease  
Biology Insights

Best-in-Field  
Antibody  
Engineering

Pipeline-in-  
a-Product  
Development





**Unique complement toolkit**

C2-KO and human C2-transgenic mice

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>30 complement assays in house across different species

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

**Extensive network of experts**

Logos of partner institutions: University of Glasgow, Cedars Sinai, Aarhus University, UMC Utrecht, KU Leuven, Leibniz Universität Medizinisch-Gesundheit, University of Leicester, VIB, and others.

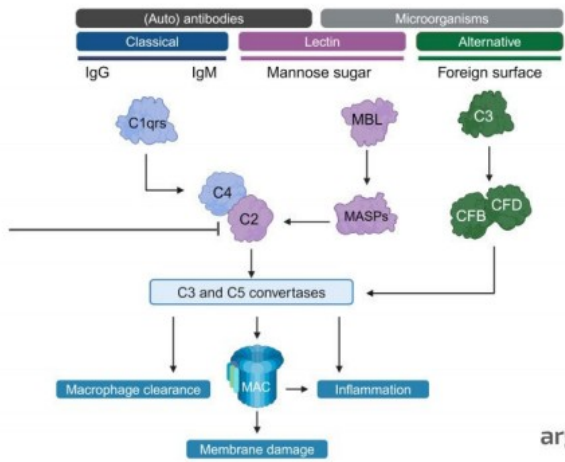
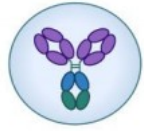
**Advancing science**

Logos of journals: JACI (The Journal of Allergy and Clinical Immunology), BRAIN COMMUNICATIONS, and NEUROSCIENCE.

# Empasiprubart in Action

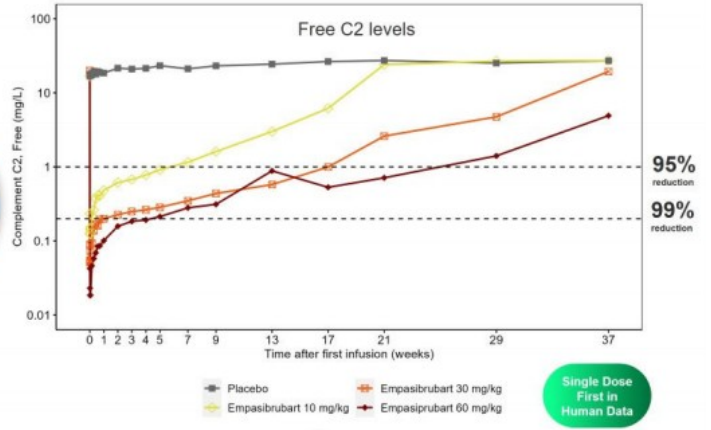
# C2 is Uniquely Positioned in Complement Cascade

Empasiprubart



# Empasiprubarb 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





## Immunology Innovation Program: Model of Co-Creation

Novel Disease  
Biology Insights

Foundational  
Immune Targets

Best-in-Field  
Antibody  
Engineering

First-in-Class  
Antibodies

Pipeline-in-  
a-Product  
Development

Differentiated Patient  
Outcomes

## Immunology Innovation Program: Model of Co-Creation

**ARGX-113**

Efgartigimod

Foundational  
Immune Targets

**ARGX-117**

Empasiprubart

First-in-Class  
Antibodies

**ARGX-119**

Differentiated Patient  
Outcomes

# ARGX-119

## Strengthening the Neuromuscular Junction through MuSK Activation

Novel Disease  
Biology Insights

Best-in-Field  
Antibody  
Engineering

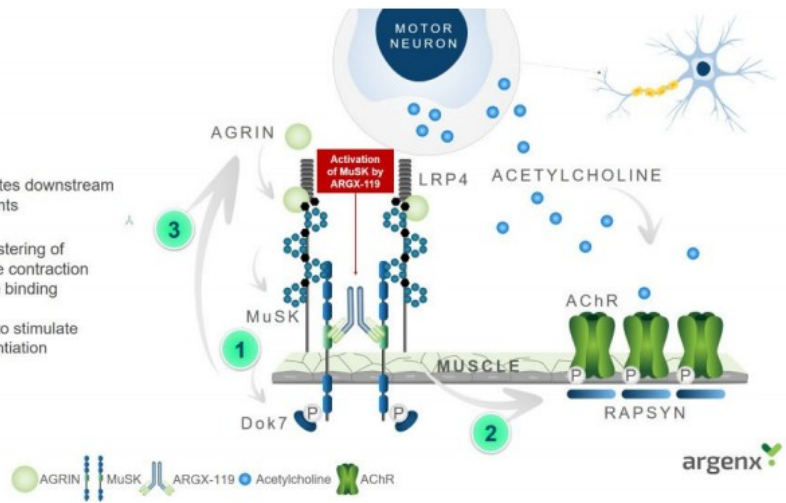
Pipeline-in-  
a-Product  
Development





# ARGX-119 Boosts Functioning of NMJs by Improving AChR Clustering

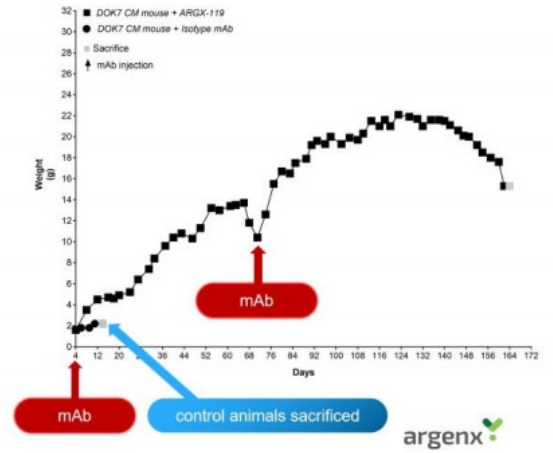
- 1 MuSK phosphorylates downstream signaling components
- 2 MuSK-induced clustering of AChRs and muscle contraction upon Acetylcholine binding
- 3 Retrograde/signal to stimulate presynaptic differentiation



## 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

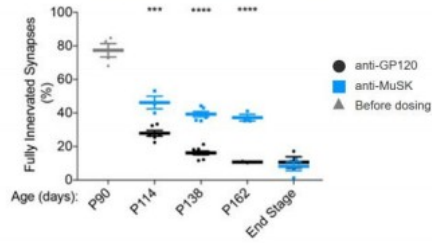




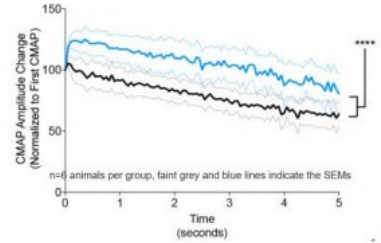
# ALS Rationale

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. 2016; Pérez-Gea et al. 2012; argenx internal data.

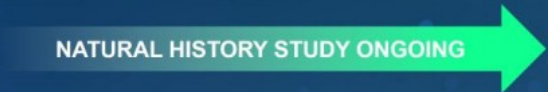
# Path Forward for ARGX-119



Proof of Biology  
Intra-Patient Dosing



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



## Immunology Innovation Program: Model of Co-Creation

Novel Disease  
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## Immunology Innovation Program: Model of Co-Creation

**ARGX-113**

Efgartigimod

Foundational  
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**ARGX-117**

Empasiprubart

First-in-Class  
Antibodies

**ARGX-119**

Differentiated Patient  
Outcomes

**ARGX-113**

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## Leadership in FcRn

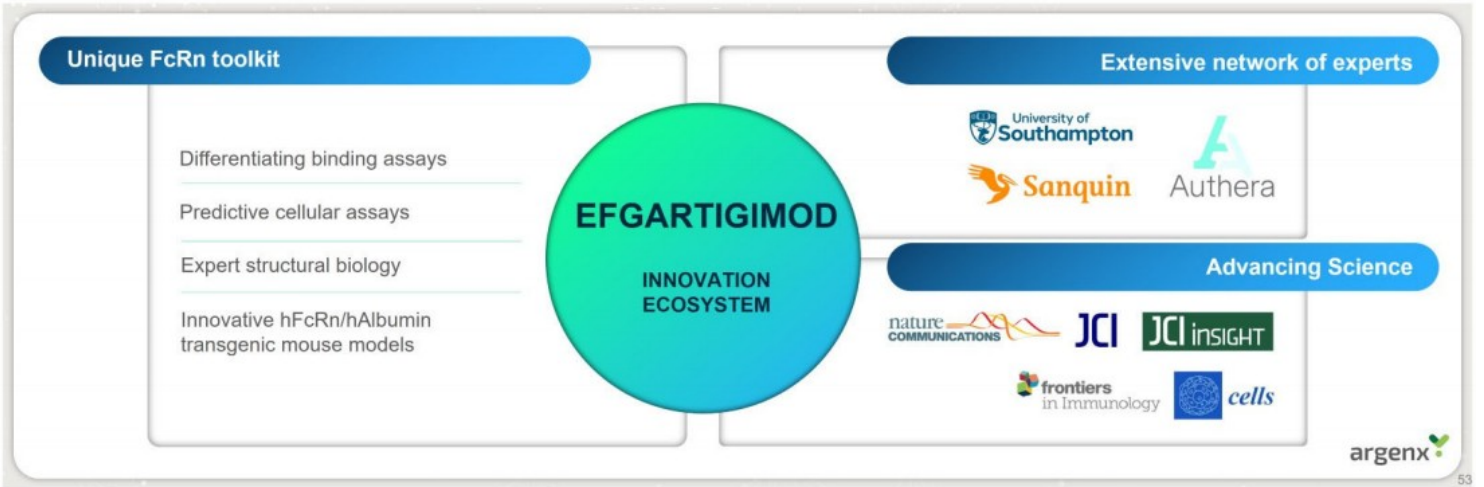
Novel Disease  
Biology Insights

Best-in-Field  
Antibody  
Engineering

Pipeline-in-  
a-Product  
Development

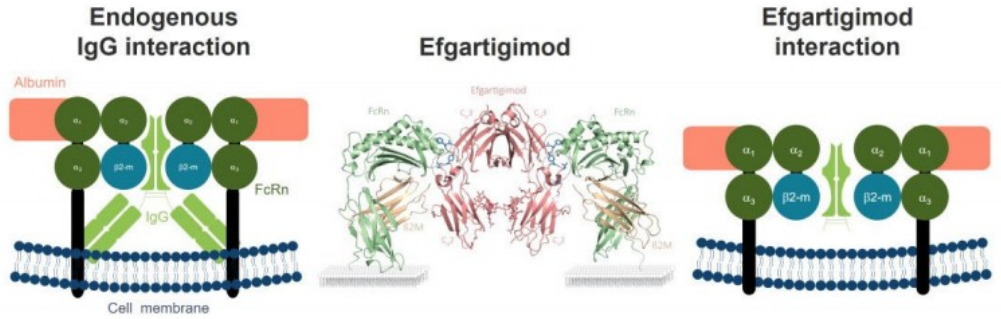
\* as of Q1 2024





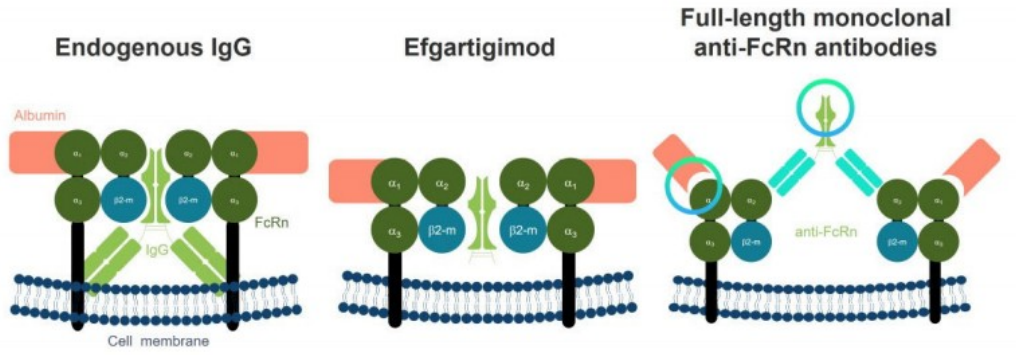


# Efgartigimod Binds to FcRn in Same Formation As Endogenous IgG



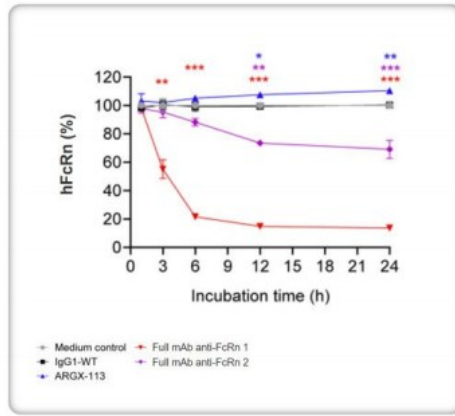
FcRn, neonatal Fc receptor for IgG, Immunoglobulin G.  
 1. Ulichis P, et al. *J Clin Invest*. 2016;126:4372-4380. 2. Howard JF, et al. *Lancet Neurol*. 2021;20:528-538. 3. VYVGART GmPC. Available at [https://www.ema.europa.eu/en/documents/product-information/vyvgart-gm-pc-product-information\\_en.pdf](https://www.ema.europa.eu/en/documents/product-information/vyvgart-gm-pc-product-information_en.pdf).  
 4. Knutson Sand KM, et al. *Front Immunol*. 2015;6:1-21. 5. Ward GS, et al. *Front Immunol*. 2022;13:802534.

# Efgartigimod is Unique Among FcRn Antagonists in How it Binds

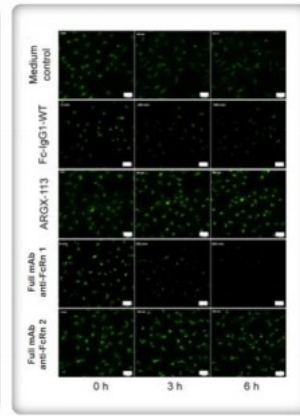


FcRn: neonatal Fc receptor; IgG: immunoglobulin G.  
 1. Ulichis P, et al. *J Clin Invest*. 2018;128:4372-4380. 2. Howard JF, et al. *Lancet Neurol*. 2021;20:528-538. 3. VYVGART GmPC. Available at [https://www.ema.europa.eu/en/documents/product-information/vyvgart-gm-pc-product-information\\_en.pdf](https://www.ema.europa.eu/en/documents/product-information/vyvgart-gm-pc-product-information_en.pdf).  
 4. Knudsen Sand KM, et al. *Front Immunol*. 2015;6:1-21. 5. Ward GS, et al. *Front Immunol*. 2022;13:802534.

# Unique Binding of Efgartigimod Leads to Differentiated Intracellular FcRn Trafficking



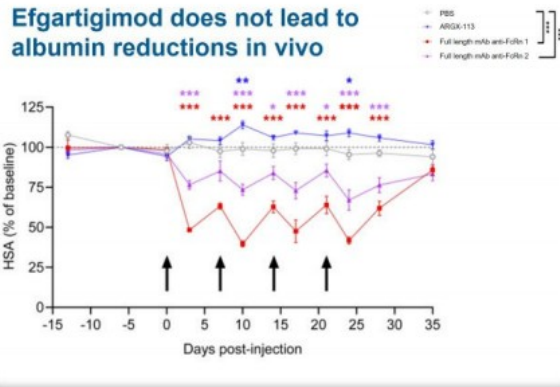
Ma et al., 2024 (10.1172/jci.insight.179186)



No interference of efgartigimod with albumin binding and recycling

No degradation of FcRn induced by efgartigimod

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



Ma et al. 2024 (10.1172/jci.insight.179166)

- 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



# Evolution of a Novel Target to a Novel Platform

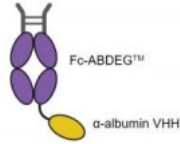


# Next Wave of First-in-class Immunology Targets

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**

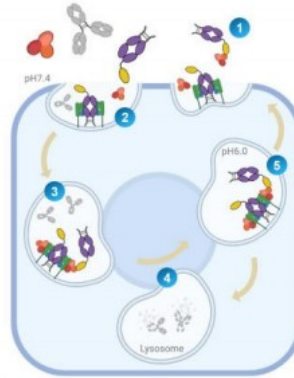
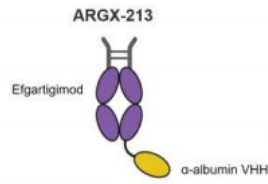
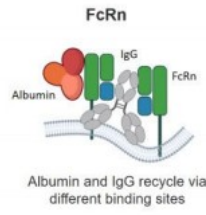


**ARGX-121**

**ARGX-XXX**

**ARGX-XXX**

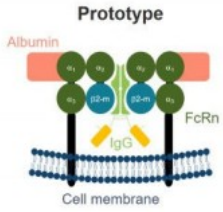
# Improving Pharmacokinetics of Efgartigimod Through Binding to Serum Albumin



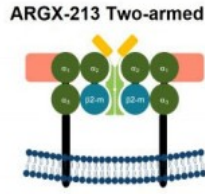
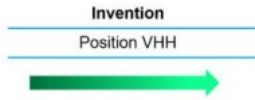
- 1 ARGX-213 adopts long albumin half-life
- 2 Pinocytosis
- 3 In endosomes, ARGX-213 prevents IgG binding to FcRn
- 4 IgG degraded in lysosomes
- 5 ARGX-213 recycles via FcRn or albumin



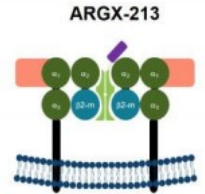
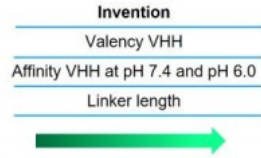
# ARGX-213 is Designed For Optimal FcRn Binding and Equipped with Unique Features



- Enhanced PK
- Sustained PD
- Albumin sparing

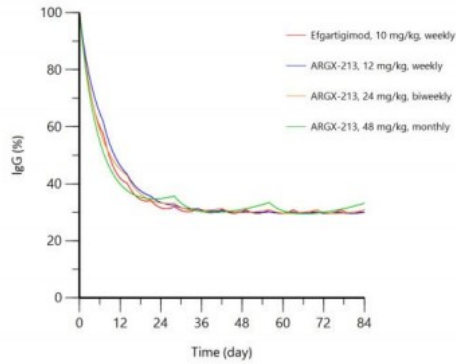


- Enhanced PK
- Sustained PD
- Albumin sparing



- Enhanced PK
- Sustained PD
- Albumin sparing

## 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

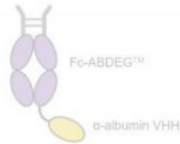
Finalize GLP  
Tox Study

Submit Clinical  
Trial Application  
1H25

Phase 1 to Start  
in 2H25

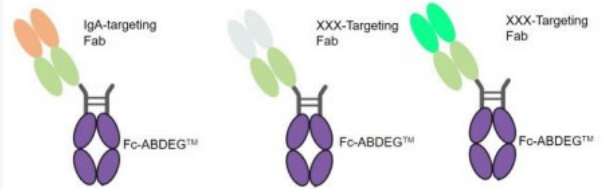
**Deep Knowledge  
of FcRn Biology  
Builds New  
Pipeline  
Candidates**

**Leverage Albumin to Broaden  
FcRn Targeting Portfolio**



ARGX-213

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



ARGX-121

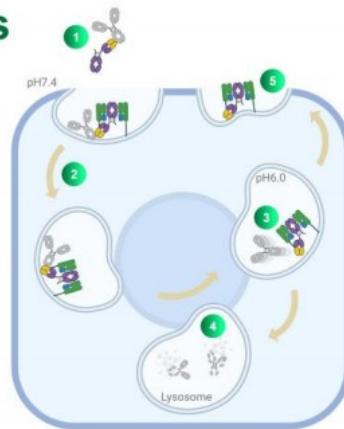
ARGX-XXX

ARGX-XXX

# ARGX-121 Mode of Actions

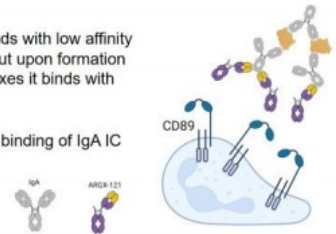
## I. FcRn-mediated IgA degradation

- 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-ABDEG™

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

**Invention**  
Affinity at pH 7.4 and pH 6.0  
FcRn degradation

→

**ARGX-121 Two-armed**

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

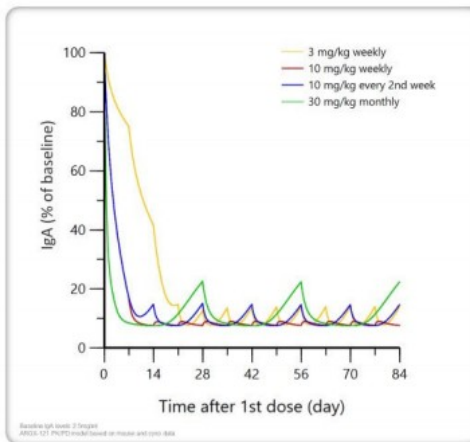
**Invention**  
Immune complex formation  
FcRn occupancy

→

**ARGX-121 One-armed**

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

## ARGX-121 Rapidly and Drastically Impacts Circulating IgA Levels

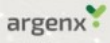


>90% IgA reduction within 1 week

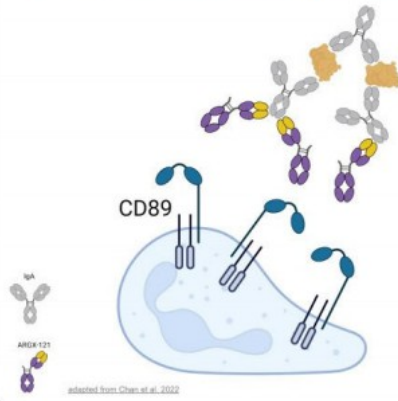
Enables flexible dosing







Broad therapeutic potential

# ARGX-121 Pipeline-in-a- Product Potential



## IgA is Fundamental in Many Diseases



-  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

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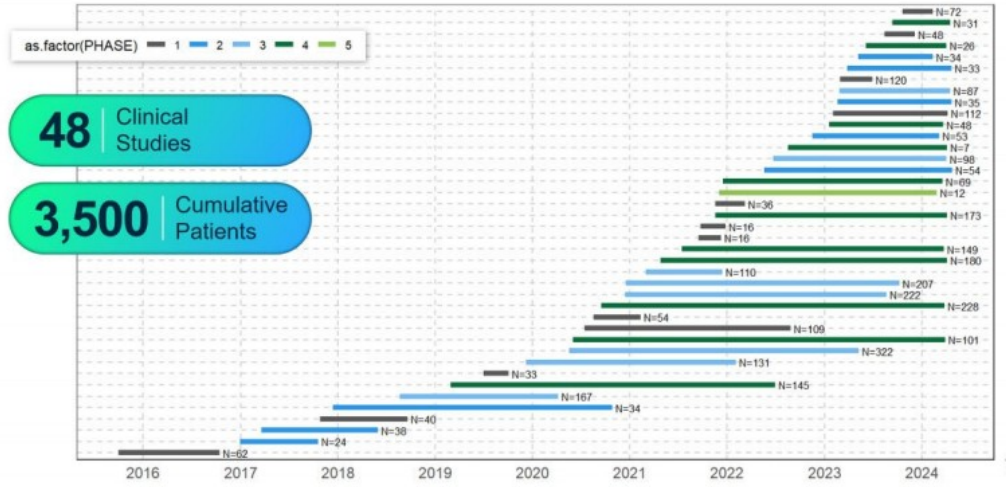
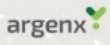
# Clinical Development

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

# Clinical Development: Bridging Innovation and Unmet Patient Need



# Rapidly Scaling our Clinical Footprint



# Pioneering in MG to Set New Standard for Treatment

IgG lowering effect of efgartigimod

MSE >50% across studies

Equivalence of SC to IV

Value broadly recognized by payor bodies

Reduction in steroid use

Earlier treatment lines Seronegative, Ocular

Central role of IgG in MG

Extended clinical effect of cyclical dosing

Highest MG-ADL and QMG response

Positive benefit risk profile

Sustained responses across dosing schedules

Broadest safety database

Market leader among advanced biologics

2010 - 2017

2018

2019

2020

2021

2022

2023

2024

FUTURE



Liu et al, 2010



Ulrichs, 2017



Howard et al, 2019



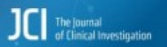
Howard et al, 2021



Howard et al, 2024



Howard et al, accepted



Ward, 2024

# Applying Our Innovation Approach to Clinical Development

## Innovation



### Evidence Generation

Build broadest data to guide treatment decisions for patients

## Co-Creation



### Patient Insights

Patient engagement in trial design and execution

## Execution



### Speed

Bring medicines to patients as quickly as possible



# Ocular and Seronegative MG



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## 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

**p rho** STUDY

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## 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

# Immune Mediated Myopathies (IMM)



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## 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

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## 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  
—

## CIDP is 4<sup>th</sup> Indication for Empasiprubart

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### 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

14 per 100,000 diagnosed

Mid-adult onset, more common in females

Increased mortality

No FDA-approved therapies across myositis subtypes

## Disease Burden

Muscle weakness and Pain

Fatigue

Large impact on quality of life

Corticosteroid side effects

### Myositis subtypes mediated by autoantibodies:

immune-mediated necrotizing myopathy (IMNM), Antisynthetase syndrome (ASyS) and dermatomyositis (DM)



**Melissa**  
Living with Myositis

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

## MSA target different autoantigens

### IMNM



SRP: protein translation  
HMGR: cholesterol synthesis

### ASyS



tRNA synthetases

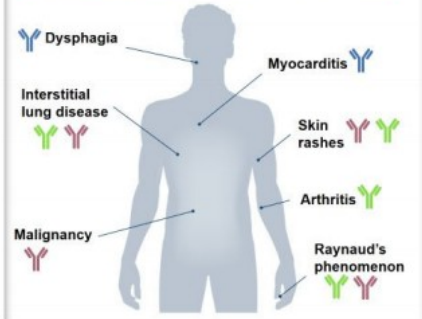
### DM



Mi2: transcriptional repressor  
Others: IFN regulators

\* 25 - 30% Myositis associated antibodies (MAA) or antibodies against unidentified targets

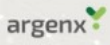
## Clinical symptoms



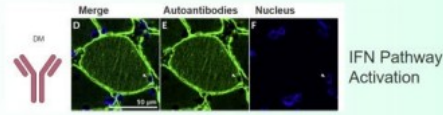
## Common hallmark: proximal muscle weakness

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

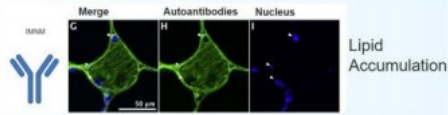
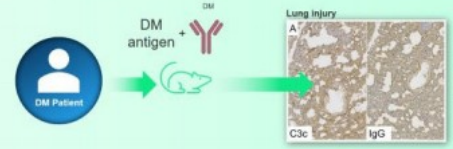
# Myositis Auto-antibodies are Pathogenic



## Pathogenic auto-antibodies enter muscle fibers



## Auto-antibodies induce IIM symptoms



Clinical (human data)

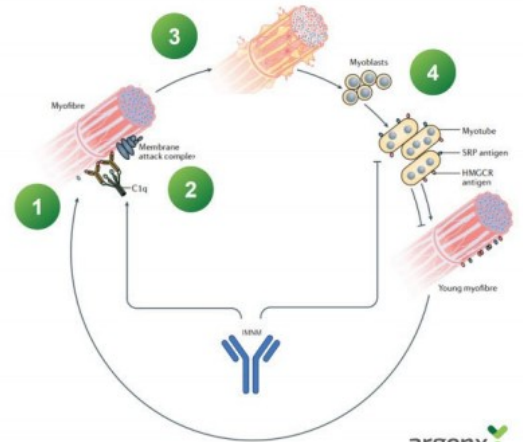
Preclinical (mouse data)

1. Pinal-Fernandez I, et al. Ann Rheum Dis. 2024 [preprint]. 2. Pinal-Fernandez I, et al. Ann Rheum Dis. 2023; 32(10). 3. Bergsøe 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:

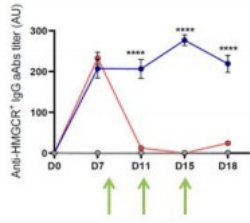
- 1 Bind muscle fiber
- 2 Activate complement
- 3 Cause necrosis
- 4 Impair muscle regeneration



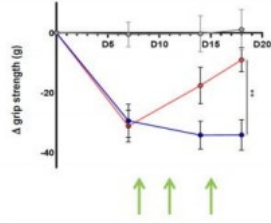
1. Figure adapted from: Allenbach Y, et al. Nat Rev Rheum. 2020; 16(12) 2. Bergua C, et al. Ann Rheum Dis. 2019; 78(1) 3. Aroache-Deleguerre L, et al. Ann Neurol. 2017; 81(4)

# Efgartigimod Reduces IMNM Antibodies and Restores Mouse Muscle Function

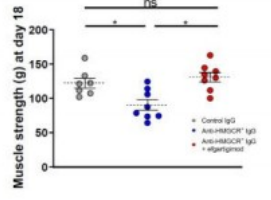
## Antibodies



## Grip Strength

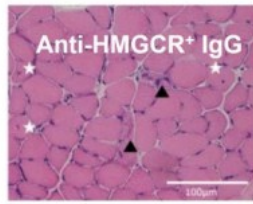


## Muscle Strength

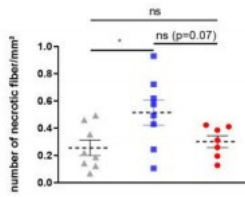


↑ Efgartigimod treatment

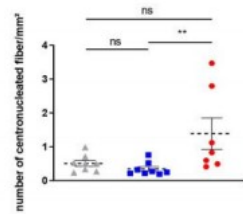
# Efgartigimod Prevents Necrosis & Allows Regeneration of Muscle Fibers



Necrosis



Regeneration

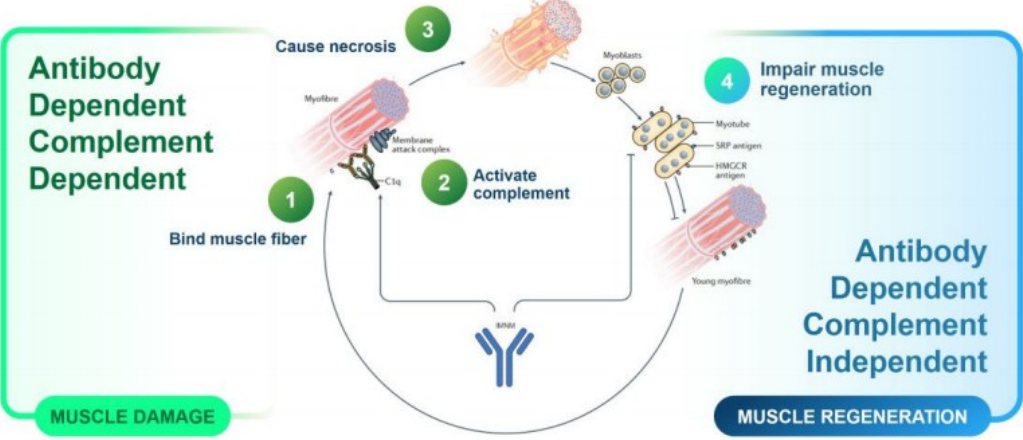
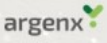


- ▲ Control IgG
- Anti-HMGCR+ IgG
- Anti-HMGCR+ IgG + efgartigimod



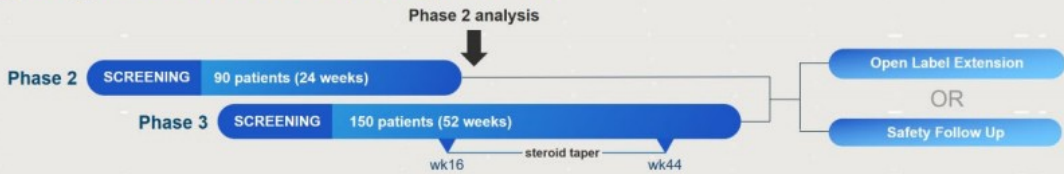
Julien S, et al. Rheumatology 2023; 62 (12)

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



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

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



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



## 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

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# Efgartigimod in Sjögren's Disease

Julie Jacobs Ph.D /// Principal Scientist

Lisa  
Living with Sjögren's Disease

# 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**

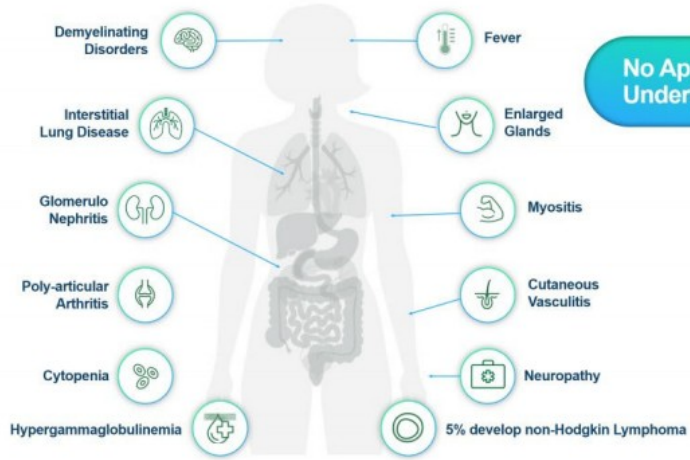


**Lisa**  
Living with Sjögren's Disease

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

# Systemic Manifestations of Sjögren's Disease

Brito-Zaragoza P, et al. *Ann Rev Dis Primaries*. 2018; Both T, et al. *Int J Med Sci*. 2017; Nagini S, et al. *Chn Exp Med*. 2022



No Approved Treatments to Target Underlying Disease

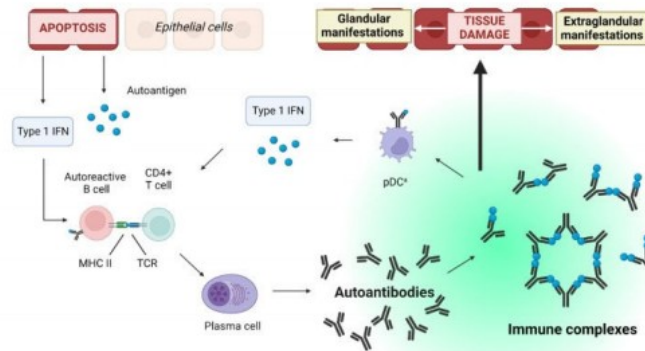
## Primary Symptoms

### Sicca Symptoms

Dry eye, mouth and vagina

### Fatigue and Pain

## Auto-antibodies are Key Players in Sjögren's Disease



Mariette X et al. 2018; Nocturne G et al. 2016; Pringle S, et al. 2019

## 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

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



Screening Period  
≤4 weeks

**Key inclusion criteria**

ACR/EULAR 2016 SjD diagnosed

ESSDAI ≥5

Ani-Ro+

Residual (un)stimulated salivary flow

Treatment Period  
Weekly - 24 weeks

Placebo (n=9)

week 0 1 2 3 4 — 23 24

Efgartigimod IV 10mg/kg (n=22)

Open-label Extension  
48 weeks

Efgartigimod IV 10mg/kg

Weekly or biweekly dosing depending on response

Treatment-free Follow-up Phase

**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 (GRESS)

**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

## Limitations



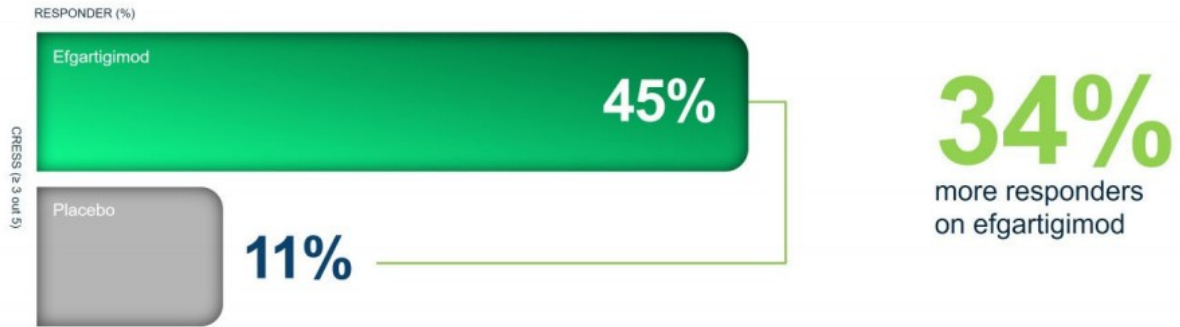
NOVEL ENDPOINT  
(IN VALIDATION)

## Strengths



ACCOUNTS FOR  
HETEROGENEOUS  
DISEASE

## Efgartigimod Demonstrated Effect on Primary Endpoint CRESS



## Observed Treatment Effect in 4 Items of CRESS



## Secondary Endpoint: STAR



### OBJECTIVE:

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

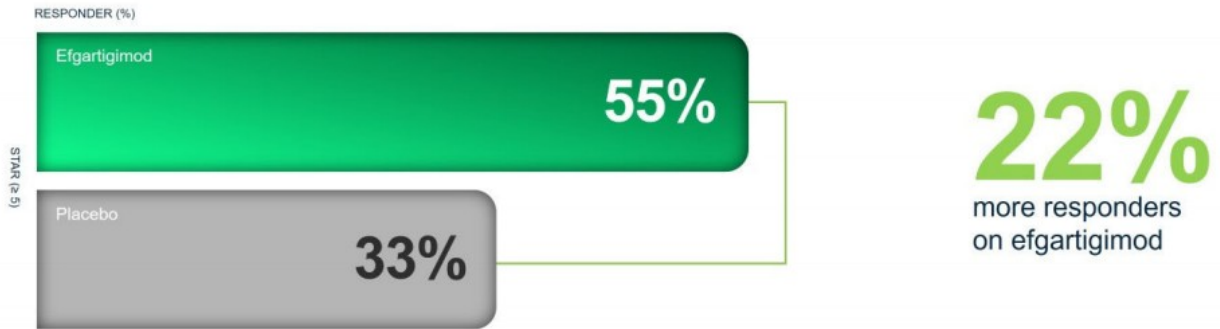
**CRESS  $\geq 3$  out of 5**

Requires response in 3 out of 5 items  
Responder systemic disease: ClinESSDAI  $< 5$

**STAR  $\geq 5$**

Requires response on PRO and/or systemic disease  
Responder systemic disease: ClinESSDAI decrease  $\geq 3$

## Efgartigimod Demonstrated Effect on STAR



## Secondary Endpoint: ESSDAI



### OBJECTIVE:

To demonstrate increased response rates on ESSDAI

### Limitations

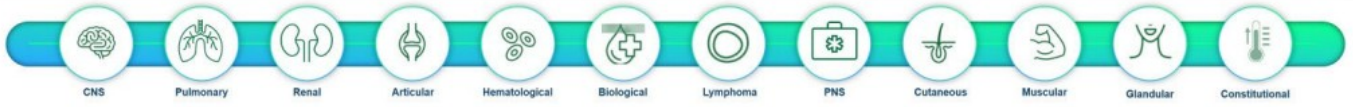
HIGH PLACEBO RESPONSES

DOES NOT CAPTURE ALL DISEASE FEATURES

### Strengths

ESTABLISHED ENDPOINT WITH FOCUS ON SYSTEMIC DISEASE SEVERITY

# Efgartigimod Demonstrated Effect on ESSDAI

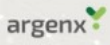


Efgartigimod  
Placebo

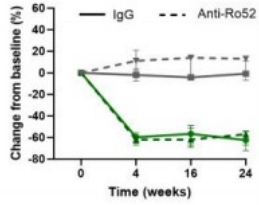
ESSDAI

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



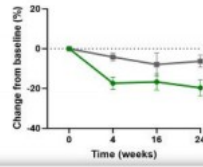
Reduction IgG and Disease-Specific Auto-Antibody



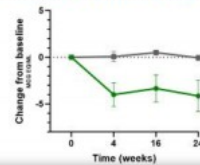
\*Median ±IQR

■ Efgartigimod    ■ Placebo

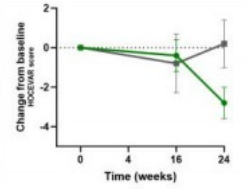
Reduction Rheumatoid Factor



Reduction C1Q Immune Complexes

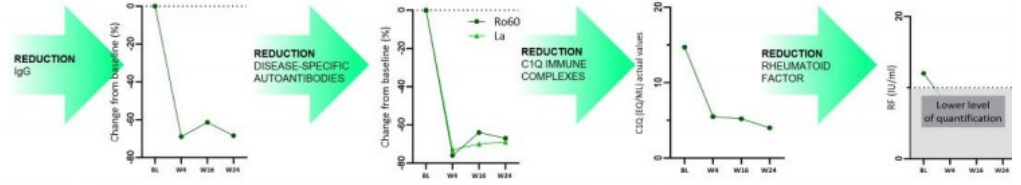
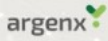


Reduction HOCEVAR Score on Salivary Gland Ultrasound





# Patient Narrative Confirms Effect of FcRn Inhibition with Efgartigimod



## RESPONDER ON STAR AND CRESS



## 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**

## Consistency of Data Demonstrates Path Forward

**p rho** STUDY

Phase 2 Nipocalimab Data  
(DAHLIA Study)



**Justifies  
Advancement To a  
Phase 3 Study**

# Path Forward for Sjögren's Disease

End of Phase 2  
Meeting



Phase 3 to Start by  
End of 2024

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# Sjögren's Disease

**Moderated by:** Luc Truyen Ph.D., M.D. // Chief Medical Officer

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# Sjögren's Disease

# Q&A

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The logo for argenx, featuring the word "argenx" in a white, lowercase, sans-serif font. To the right of the text is a stylized graphic consisting of two overlapping shapes: a larger, light green shape resembling a checkmark or a stylized 'A', and a smaller, solid green circle below it.

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# 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

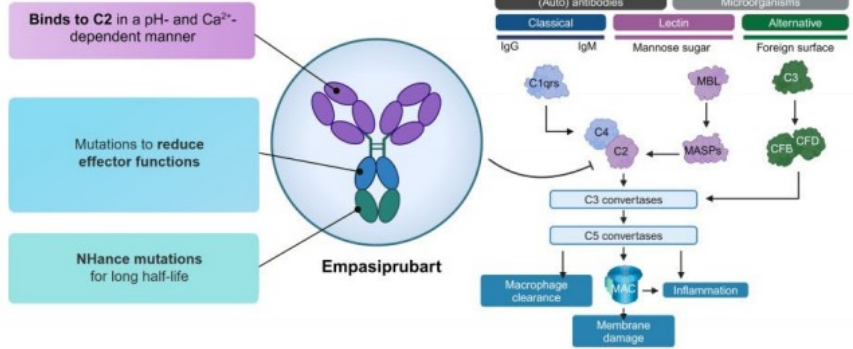
Impact on social life, activities and work

Exhaustion and fatigue



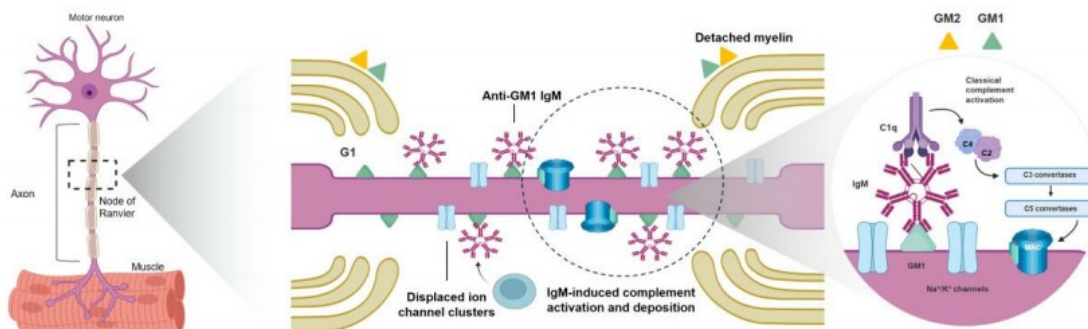
# Empasiprubart

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



1. Goh, J. *et al.* *Antibodies* 2012, 2(1), 1-12.  
 2. Shalita, A. *et al.* *Journal of Immunology* 2012, 188(12), 6183-6191.  
 3. Shalita, A. *et al.* *Journal of Immunology* 2012, 188(12), 6183-6191.  
 4. Hachimi, M. *et al.* *J. Biol. Chem.* 2007, 282(12), 8248-8254.

# Complement Activation Drives Axonal Damage in MMN

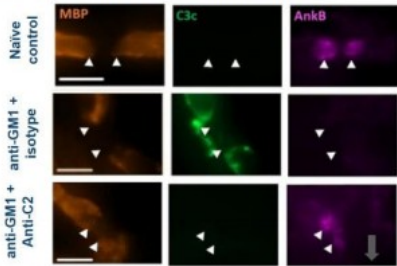


New Learning  
GM2 also plays role in  
subset of patients

Figure created with BioRender.com, adapted from Vainu L, et al. *Acta Neurol Scand* 2011; 123: 40-50 and Salzer A, Cosack JC, in: *StatPearls: Treasure Island (FL): StatPearls Publishing; 2021. <https://www.ncbi.nlm.nih.gov/books/MB/000000000/>*

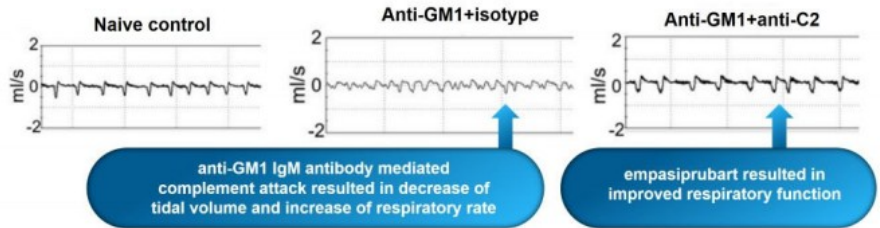
# C2 Inhibition Improves Respiratory Function in vivo

C2 inhibition reduced structural injury to Schwann cell nodal membranes



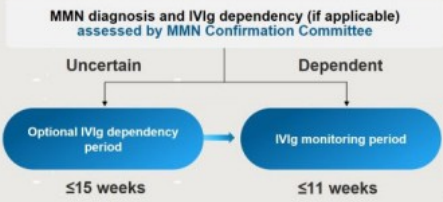
anti-GM1 antibody mediated complement attack on the Schwann cell membrane → Significant disruption at node of Ranvier (hall mark of MMN) w/o empasprubart

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



# Phase 2 Trial Design

Screening (≤28 days)



Double-blinded Treatment Period



**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

argenx EFNS, European Federation of the Neurology Societies; IV, intravenous; IVIg, intravenous immunoglobulin; MCC, MMN Confirmation Committee; MSIS, modified motor neuropathy; PND, Pharyngeal Nerve Society

IVIg dependency parameters are summarized in the key inclusion criteria, but details provided at <https://www.clinicaltrials.gov/ct2/show/study/NCT02227815>. The length of the monitoring period will depend on an individual's IVIg dose frequency: blood every 2 weeks - up to 30 days monitoring; blood every 3 weeks - 40 days monitoring; blood every 4 weeks - 60 days monitoring; blood every 5 weeks - 77 days monitoring. \*Blood drawn frequency: blood every 2 days after first IVIg administration. Participants will be randomized to IVIg therapy in a one-to-one manner. †Patients through study entry into the trial.

1. ClinicalTrials.gov Identifier: NCT02227815 | Updated July 20, 2020 | Accessed April 2019 | <https://www.clinicaltrials.gov/ct2/show/study/NCT02227815> 2. See the POC, et al. Poster presented at NMSO Annual Scientific Meeting, September 10-24, 2019, Orlando, FL.



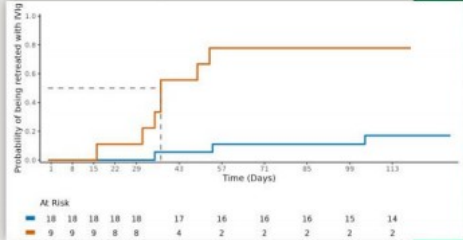
# ARDA Study Results

Jeff Gupthill /// Neuromuscular Franchise Lead Clinical Development

# Empasiprubart Reduced Risk of IVIg Retreatment

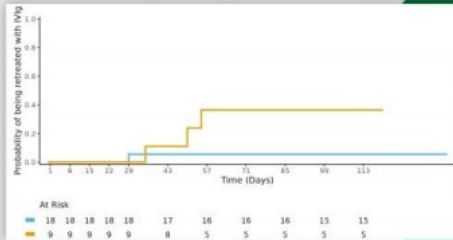


## Cohort 1

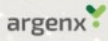


Reduced risk of IVIG retreatment by **91%**

## Cohort 2



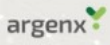
Reduced risk of IVIG retreatment by **84%**



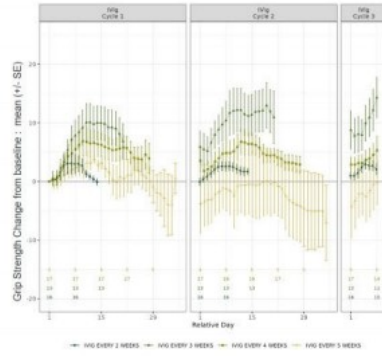
CI, confidence interval; DTP, double-blind treatment period; IVIg, intravenous immunoglobulin.  
 \*P < 0.05 for treatment with IVIg is defined as the first time you fully administered before retreatment (including scheduled visits) up to the first IVIg retreatment during the DTP.

■ Empasiprubart ■ Placebo

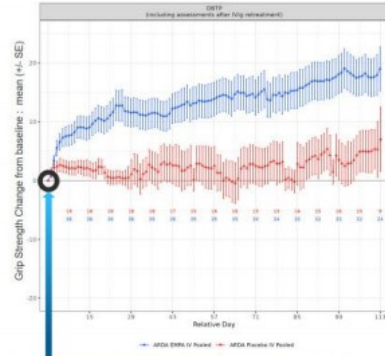
# Empasiprubart Improved Grip Strength in Both Hands



## IVIg Treatment → Clear Fluctuating Effect



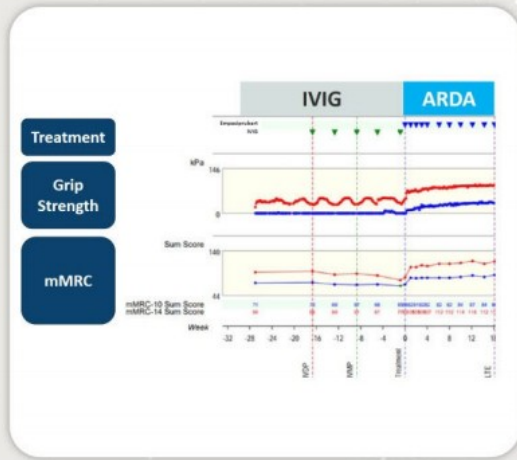
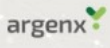
## Grip Strength



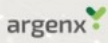
**Patients at IVIG best**



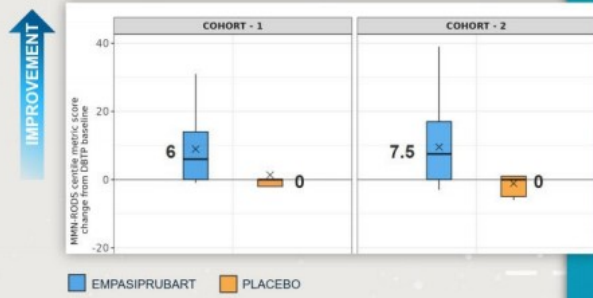
# ARDA Participant Journey



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



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

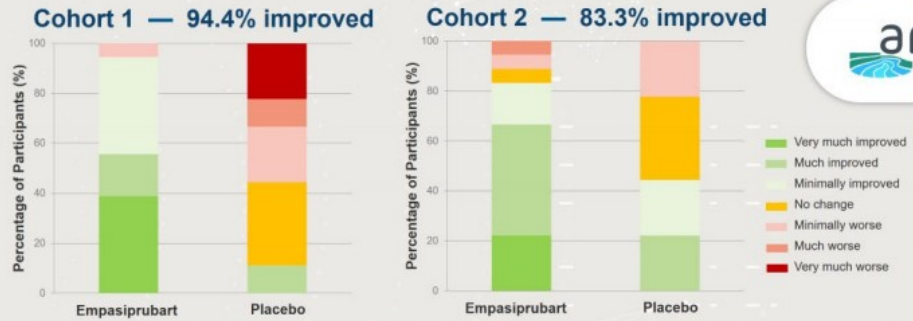


## Are you able to:

- 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?

## Empasiprubart Treated Patients Feel Better than their Best on MIG

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



**Consistent improvement observed for each dose of empasiprubart**

## Path Forward for MMN

End of Phase 2  
Meeting 3Q 2024

Phase 3 to Start  
in 4Q 2024

IMMERSION STUDY



## Trials Ongoing with Empasiprubart

varvara  
Delayed Graft Function Study

# DGF

Delayed Graft Function

empacific  
Dermatomyositis Study

# DM

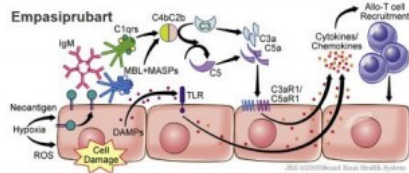
Dermatomyositis

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133



# Empasiprubart in Delayed Graft Function After Kidney Transplant

## Biological Rationale



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

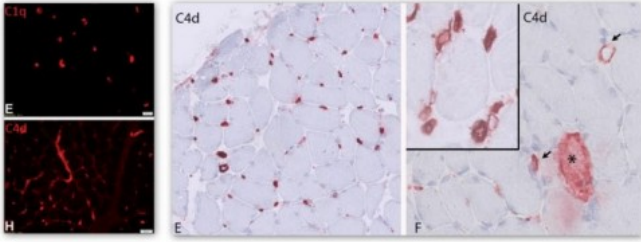
## Disease Characteristics

- 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

# Empasiprubart in Dermatomyositis

## 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

© Pohlman et al. 1994. *Pathol. Annot. Immunopathol. Clin. Med.*  
Revised 2004. *Cell Tissue Res*. © Ciba-Geigy AG, 2007.  
Lafrenie et al. *Brain*. 2002. Jul 1;125(7):1503-1511. doi:10.1093/brain/awf049. © Ciba-Geigy, 2002.

© Orskov et al. *Lancet*. 2003. Sep 29;362(9319):971-92. © Ciba-Geigy AG. All rights reserved. 2004. *Pathol. Annot. Clin. Med.*  
Appel et al. *Diabetologia*. 2004. Jun 25;47(12):2129-32.  
Bendtsen et al. *Lancet*. 2003. Sep 29;362(9319):971-92. © Ciba-Geigy, 2003.

## Our Next Pipeline-in-a-Product Asset



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## MMN Disease

**Moderated by:** Luc Truyen Ph.D., M.D. /// Chief Medical Officer

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

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

# MMN Disease

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# Sustainable Commercial Engine

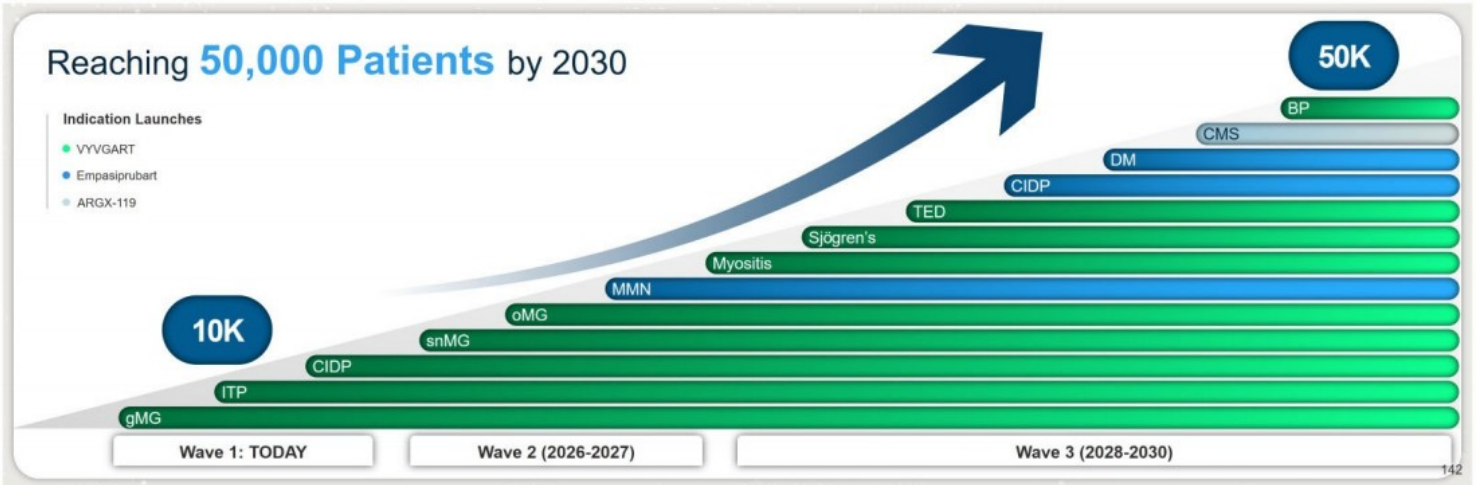
Karen Massey /// Chief Operating Officer



# Reaching 50,000 Patients by 2030

## Indication Launches

- VYVGART
- Empasiprubar
- ARGX-119



# MG Launch Set Standard on Commercial Excellence

## Innovation



### Evidence Generation

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

Meaningful **steroid reduction**



## Co-Creation



### Empowering Patients

Direct to Consumer engagement

92% Brand awareness



## 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

# Future Drivers of Growth in MG

## Innovation



**Evidence Generation**



## Co-Creation



**Empowering Patients**

**Product Presentations**



PFS



Autoinjector

## Execution

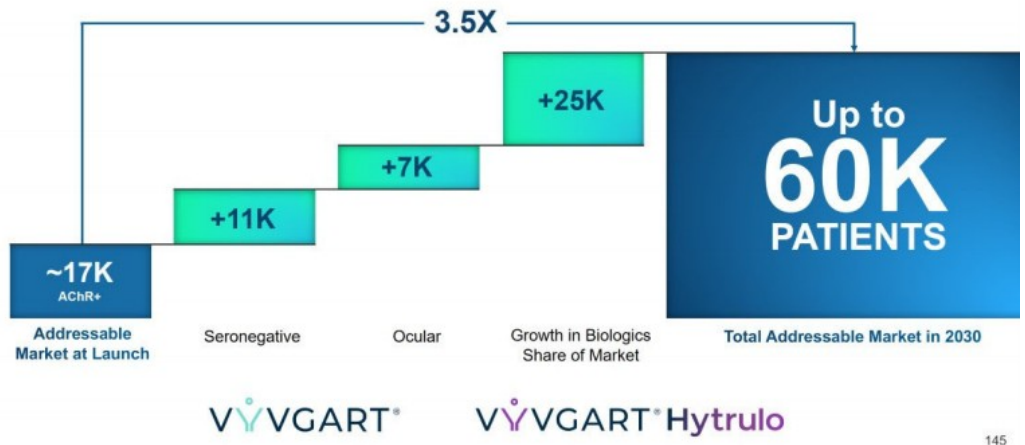
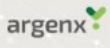


**Speed**

**Global Expansion**

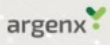


# Expanding MG Opportunity

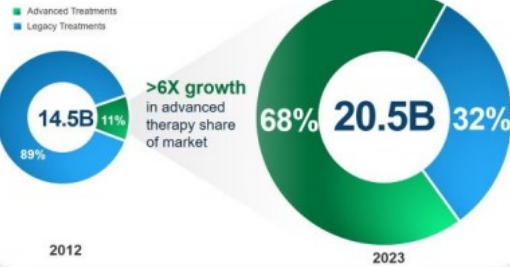


# Innovation Builds Markets

## 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

## 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

My VYVGART<sup>®</sup> Path

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

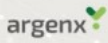
**GBS|CIDP**  
Foundation International

Execution



Deep existing neurology relationships

# 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

\*% prevalence numbers, argenx market research



## 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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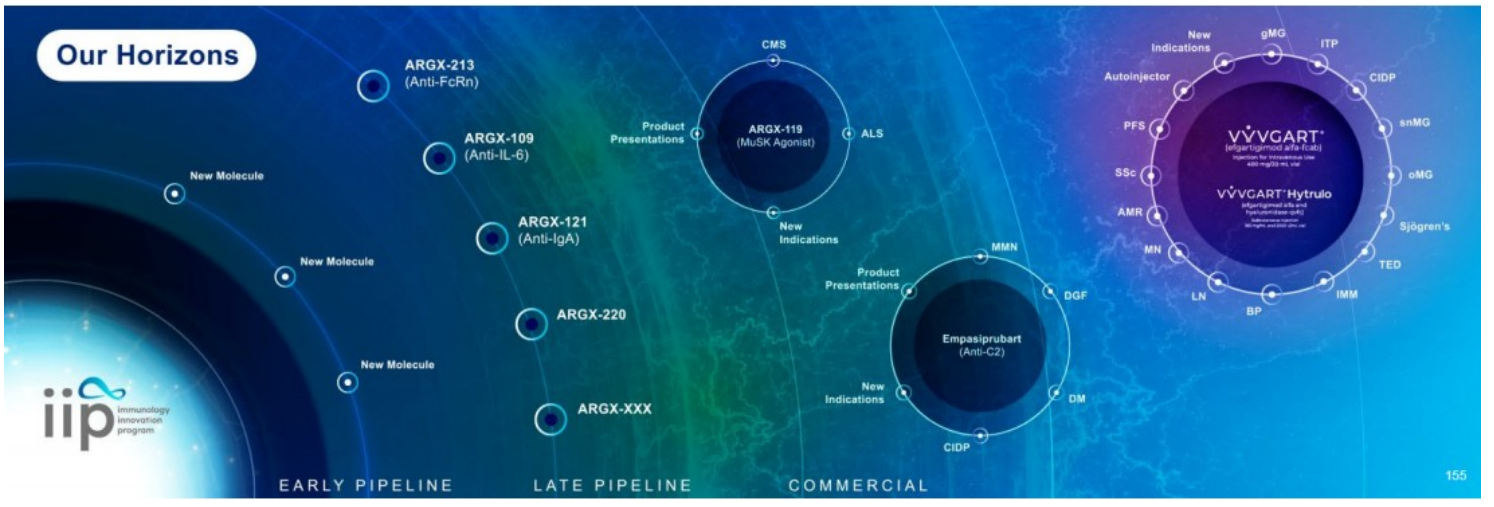
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# Q&A

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# Our Horizons



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