Developing Highly Differentiated Antibody Therapeutics

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Corporate presentation
April 2019

Forward-Looking Statements

Company Leadership

Management

Tim Van Hauwermeiren
Chief Executive Officer

Keith Woods
Chief Operating Officer

Hans de Haard, Ph.D.
Chief Scientific Officer

Torsten Dreier, Ph.D.
Chief Development Officer

Eric Castaldi
Chief Financial Officer

Nicolas Leupin, M.D.
Chief Medical Officer

Debbie Allen, Ph.D.
SVP, Business Development

Dirk Beeusaert
General Counsel

Board of Directors

Peter Verhaeghe
Chairman

Tim Van Hauwermeiren

Werner Lanthaler — evotec
Audit Com

David Lacey — AMGEN

Don deBethizy — TARGACEPT santaris pharma a/s
Remuneration Com

R&D Com

Pamela Klein — Genentech

Tony Rosenberg — NOVARTIS

James Daly — AMGEN Incyte

Scientific Advisors

James Howard — UNC SCHOOL OF MEDICINE
ARGX-113 MG

David Kuter — HARVARD MEDICAL SCHOOL
ARGX-113 ITP

Adrian Newland — Barts Health NHS Trust
ARGX-113 ITP

Jan Verschuuren — LUMC
ARGX-113 MG

Martine Bagot — Hôpitaux Universitaires SAINT-LOUIS LARIBOISIÈRE FERNAND-WIDAL
ARGX-110 CTCL

Sophie Lucas — de Duve INSTITUTE
GARP Biology

Adrian Ochsenbein — CD70 Biology

Sally Ward — A&M
FcRn Biology

Rapidly Emerging Leadership in Immunology

Pioneering differentiated therapeutic antibodies in severe autoimmune diseases and cancer

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1 Novel Target Biology

- Integrated via advanced technology suite
- First- and best-in-class potential

2 Innovative Access Program

- Robust science
- Collaborative
- Efficient pipeline expansion

5 Multi-Asset Late-Stage Platform

- Phase 3 in MG and ITP
- Pre-commercial activities in MG

3 Highly Productive Development Engine

- Rapid development timeline
- New candidate each year

4 Maximum Value per Asset

- Pipeline-in-a-product strategy
- Strong biological rationale

Translate immunology breakthroughs into novel medicines which truly impact patients' lives

Recent news

Efgartigimod (ARGX-113):
- MG Ph3: First patient treated – recruiting up to 150 patients (Sept '18)
- ITP Ph2: Favorable tolerability profile and robust efficicay signal – second PoC (ASH - Dec '18)
- ITP: Orphan Drug Designation (FDA – Jan '19)
- Exclusive collaboration with Halozyme for ENHANZE® subcutaneous delivery technology (Feb '19)

Cusatuzumab (ARGX-110):
- AML Ph1/2: Favorable tolerability profile and 92% ORR mainly CR/CRi (ASH – Dec '18)
- Strategic partnership with Janssen: accelerating and broadening clinical development plan (Dec '18)
- AML: Orphan Drug Designation (FDA – Jan '19)

ARGX-115 (ABBV-151): Initiation of first-in-human clinical trial - $30mm milestone payment

ARGX-114: SIMPLE Antibody™ agonizing MET, spun out into AgomAb (€21mm series A round - April '19)

Augmenting Intrinsic Therapeutic Properties of Antibodies

Exclusive collaboration with Halozyme for subcutaneous delivery technology

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Antibody	Technology Role	Suite of Technologies	Subcutaneous Delivery Technology

V-region

Fc region

Unlock novel and complex targets

SIMPLE Antibody™ Platform
- Llama immune system delivers V-regions with high human homology
- Highly diverse antibody output covers a multitude of target epitopes

Modulate immune response

NHance®
- Extends half-life / PD effect
- Enhances tissue penetration

ABDEG™
- Clears disease target
- Clears autoantibodies

POTELLIGENT®
- Boosts cell killing

Halozyme

ENHANZE® technology for efgartigimod
- Exclusive access for targeting FcRn
- Exclusive access for two additional targets
- Industry-validated approach
- Facilitates de-risked standalone subcutaneous product with well-established development path
- Leverages favorable bioavailability and intrinsic physicochemical properties of efgartigimod
- 3 formulations available for efgartigimod: IV, SC and SC ENHANZE®

Unique suite of technologies enables development of differentiated product candidates against novel targets

Exclusive access to ENHANZE ® solidified leadership position in FcRn space

Klarenbeek et al. 2015, mAbs
Basilico et al. 2014, J Clin Inv.

Deep Pipeline of Wholly-Owned Candidates for Orphan Indications

Product Candidate	Target	Indication	Preclinical	Phase 1	Phase 2	Phase 3	BLA	Next Milestone / Commentary
Wholly-Owned & Co-Development Product Candidates								
ARGX-113 Efgartigimod	FcRn	Myasthenia Gravis						3Q18: Phase 3 initiated
		Immune Thrombocytopenia (ITP)						2H19: Phase 3 initiation
		ITP Subcutaneous Formulation						1H19: Phase 2 initiation
		Pemphigus Vulgaris						1H19: Cohort 3 initiation
		Chronic Inflammatory Demyelinating Polyneuropathy						2H19: Phase 2 initiation
ARGX-117	Novel complement target	Severe Autoimmune Diseases						Antibody-mediated autoimmune diseases / Complementary to ARGX-113
ARGX-110 Cusatuzumab	CD70	Acute Myeloid Leukemia						$500 million upfront / Eligible for up to $1.3 billion in milestones; tiered royalties

Innovative Access Program Allows Strategic Partnering

Partner activity focused in therapeutic areas outside severe autoimmune and cancer

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Product Candidate	Target	Indication	Preclinical	Phase 1	Phase 2	Phase 3	BLA	Next Milestone / Commentary
Partnered Product Candidates								
ARGX-112 (LEO)	IL-22R	Skin Inflammation	██					Eligible for up to ~€100mm in milestones; tiered royalties
ARGX-115 (ABBV-151) (abbvie)	GARP	Cancer Immunotherapy	██	First-in-human clinical trial $30mm milestone payment				Received $90mm in upfront and preclinical milestone payments. Eligible for up to $625mm milestones; tiered royalties
ARGX-116 (STATEN BIOTECHNOLOGY)	ApoC3	Dyslipidemia	█					Eligible for double-digit royalties and exclusive option to license the program; collaboration with Novo Nordisk
ARGX-114 (AGOMAB THERAPEUTICS)	Met	Fibrosis	█					Golden share

- **Innovative Access Program**: 7 live programs
- Antibody discovery alliance with **Shire** focused on multiple rare disease targets – 2 options exercised
- Additional programs include ARGX-111 targeting c-MET in solid tumors and blood cancers (P1 concluded, wholly-owned, available for partnering) and ARGX-109 (gerilimzumab) targeting IL-6 for rheumatoid arthritis (P1 concluded, partnered with Genor Biopharma)

Efgartigimod:
A Pipeline-in-a-Product Opportunity

Efgartigimod: Human IgG1 Fc Fragment with ABDEG™ Mutations

Exploits Natural Fc/FcRn Interaction and retains pH dependent binding

IgG antibodies recycle through FcRn[1]...
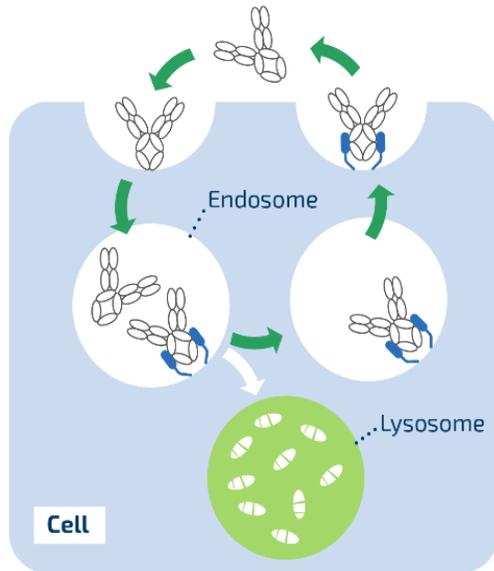
efgartigimod potently blocks FcRn...
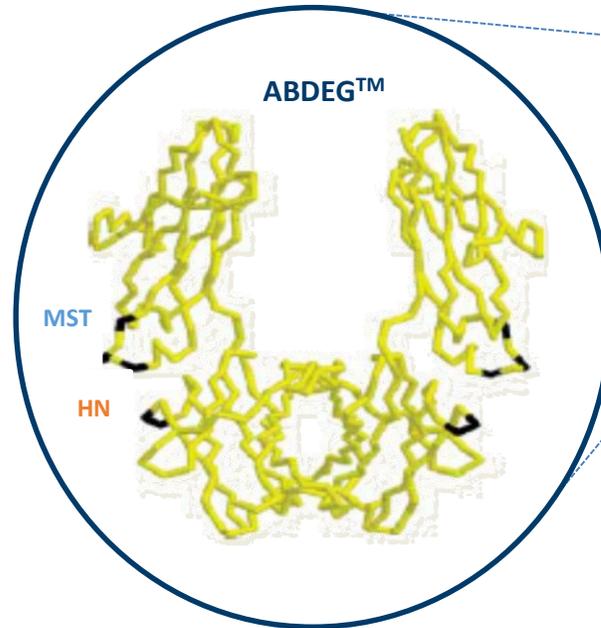
leading to IgG elimination[2]

Endosome

Lysosome

Cell

ABDEG™

MST

HN

Endosome

Lysosome

Cell

Antibody   efgartigimod   FcRn
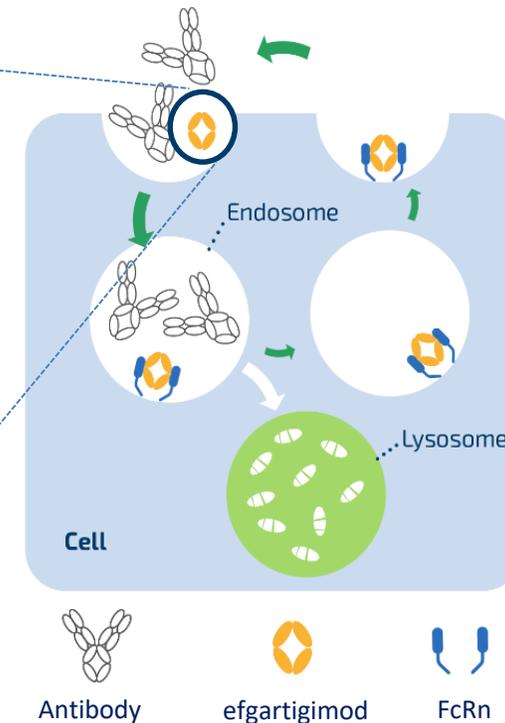
(1) Roopenian et al. 2007, Nat Rev Immunol.
(2) Vaccaro et al. 2005, Nat Biotech.

(3) Ulrichts et al. 2018, J Clin Invest.
(4) argenx data

Efgartigimod Emerges as First-In-Class and Best-In-Class

- **Human IgG1 Fc fragment**
- **With ABDEG™ mutations**
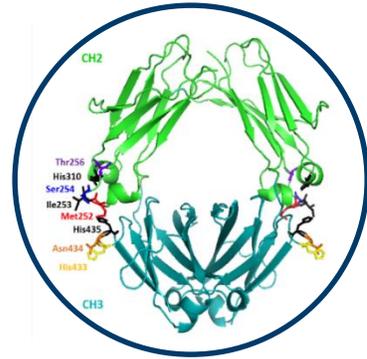
- **Natural ligand of FcRn**
- **Enhanced, pH dependent binding**

First-in-class features

Best-in-class clinical attributes

- **Reduced FcγR, C1q binding**

- **Clean tolerability profile**
  No headache or GI AE profile (~120 subjects)

- **Endosomal recycling FcRn-efgart complex; no lysosomal degradation**

- **No decrease in albumin**
  (Ulrichts et al., JCI, 2018)

- **Long half-life**
  Unparalleled tissue penetration & distribution

- **Can rebind FcRn**

- **Long-lasting, potent PD effect**
  Fast onset of clinical benefit

- **1/3 size of IgG; excellent physicochemical stability**

- **Lower dose enables convenient subQ administration, high concentration formulations and lower COGS**

Novel Treatment Modality in Severe Autoimmune Diseases

Efgartigimod Beachhead Indications

Myasthenia Gravis

- Block acetylcholine receptors
- Cross-link + internalize AChRs
- Complement recruitment
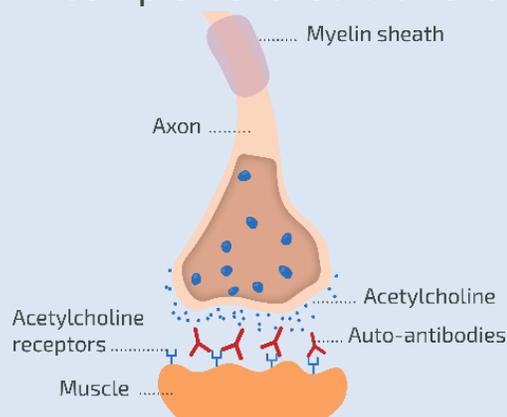
Immune Thrombocytopenia

- Enhance platelet clearance
- Platelet killing
- Inhibit platelet production
- Reduced platelet function
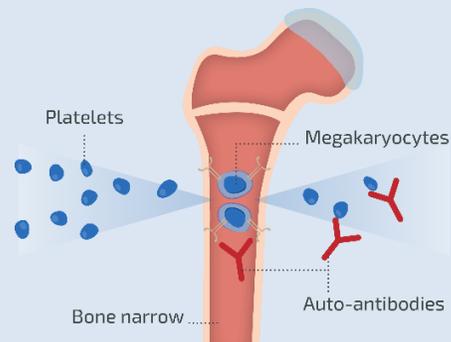
Pemphigus Vulgaris

- Sterically hinder epithelial adhesion affecting skin and mucosal integrity
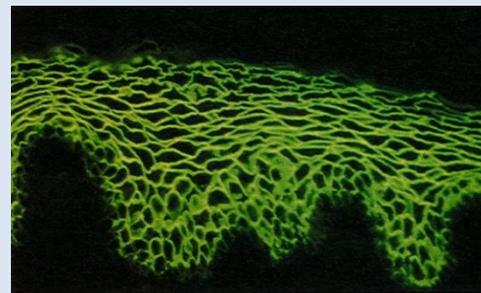
Common Characteristics of Beachhead Indications

- Pathogenic auto-antibodies causal to disease biology
- Typical treatment options: corticosteroids, broad immunosuppressants, IVIG, plasmapheresis, Rituxan – with mixed response rates and serious side effects
- Orphan potential in U.S. (MG: 50-60K[1]; ITP: 50K[2]; PV: 30-40K[3])
- Potential pharmacoeconomic benefit to healthcare system given price of targeted therapies (e.g., Soliris for refractory MG ~$700K / year)[4]

(1) Philips et al. 2003, Ann N Y Acad Sci; Drachman et al. 1993, New Eng J Med
(2) Wall street research; Estimated 65K ITP patients in US with ~80% diagnosed with primary ITP
(3) IPPF (www.pemphigus.org)
(4) Source: Reprinted with permission by First Databank Inc.; WAC = Wholesale Aquisition Cost 8/21/17

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- Consistent and favorable tolerability profile

- Fast, long-lasting and sustained benefit; clinically meaningful and statistically significant

- Strong correlation between IgG level reduction and disease improvement; supporting focus on IgG-mediated diseases

- Significant reduction of AChR autoantibodies

- Phase 2 execution advances efgartigimod into Phase 3 (initiated)

Efgartigimod Safety And Tolerability Profile

2 hour infusion enabling outpatient administration

- Efgartigimod was well-tolerated in patients; confirmed findings from Phase 1 healthy volunteer trial
- TEAE profile was balanced between efgartigimod and placebo
- TEAEs were mostly mild (grade 1) in severity; no severe AEs were reported
- No deaths, serious AEs or TEAEs leading to discontinuation of treatment were reported during trial

Treatment Emergent Adverse Events (TEAEs) Reported in ≥ 2 patients	Placebo (N = 12)	Efgartigimod (N = 12)
TEAEs (Total)	10 (83.3%)	10 (83.3%)
• Headache	3 (25.0%)	4 (33.3%)
• Nausea	1 (8.3%)	1 (8.3%)
• Diarrhea	1 (8.3%)	1 (8.3%)
• Abdominal pain upper	1 (8.3%)	1 (8.3%)
• Arthralgia	2 (16.7%)	-
• B-lymphocyte decrease	-	2 (16.7%)
• Lymphocyte count decrease	-	2 (16.7%)
• Monocyte count decrease	-	2 (16.7%)
• Neutrophil count increase	-	2 (16.7%)
• Myalgia	-	2 (16.7%)
• Pruritus	2 (16.7%)	1 (8.3%)
• Rhinorrhea	1 (8.3%)	1 (8.3%)
• Tooth abscess	2 (16.7%)	-
• Toothache	2 (16.7%)	-
Efgartigimod deemed related TEAEs	3 (25.0%)	8 (66.7%)
• Headache	1 (8.3%)	3 (25.0%)
• Monocyte count decrease	0 (0.0%)	2 (16.7%)
• Rhinorrhea	1 (8.3%)	1 (8.3%)

Assessment for all efficacy scales
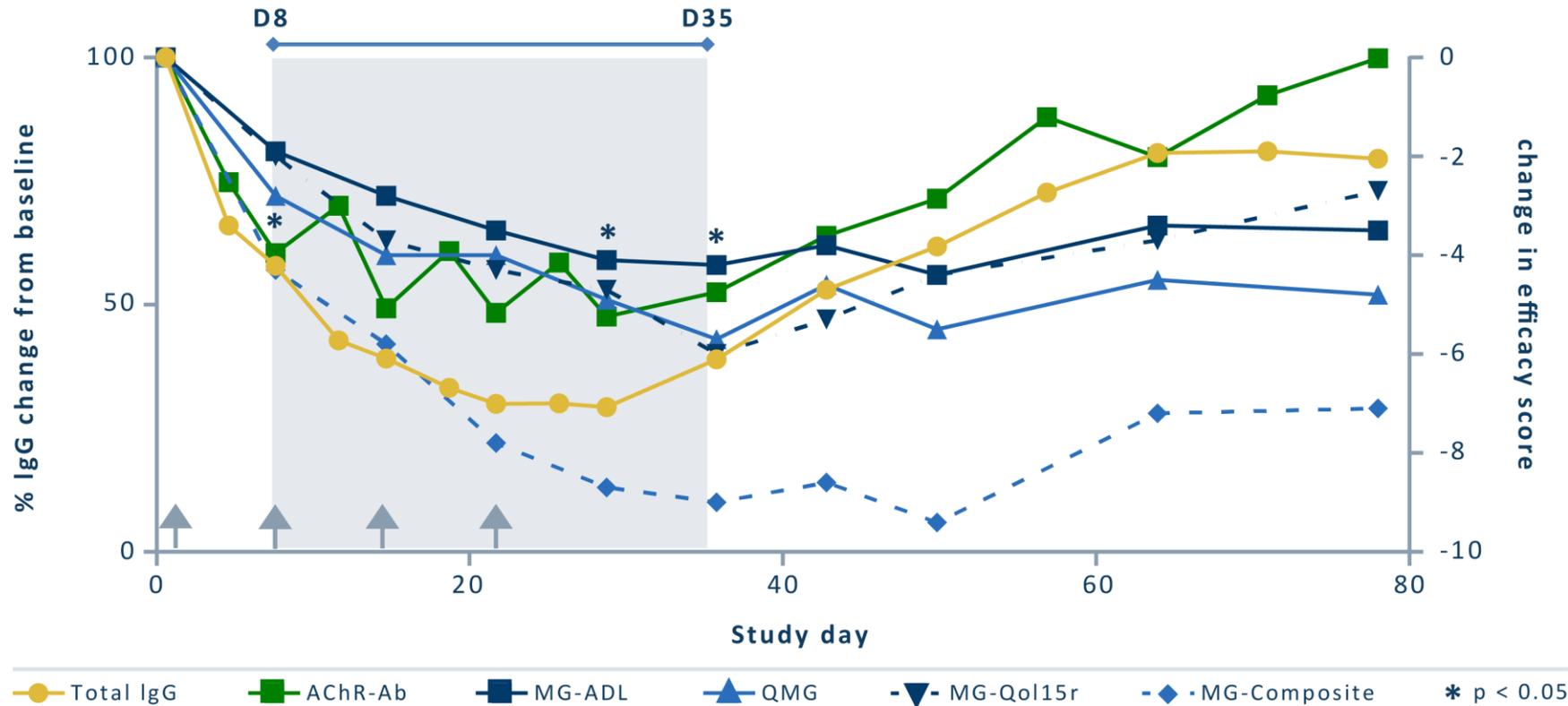
- Clinical improvement persists despite return of IgG levels

- Potential differentiation from PLEX where clinical benefit was reported to be lost 2-4 weeks after end of treatment [1]

(1) Kuks and Skallebaek, 1998, Transfus Sci

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75% of Treated Patients Achieved Lasting Response

Patients with MG-ADL ≥ 2 for a period of <u>at least</u> 6 weeks

* (p = 0.0391)

75%

25%

Placebo
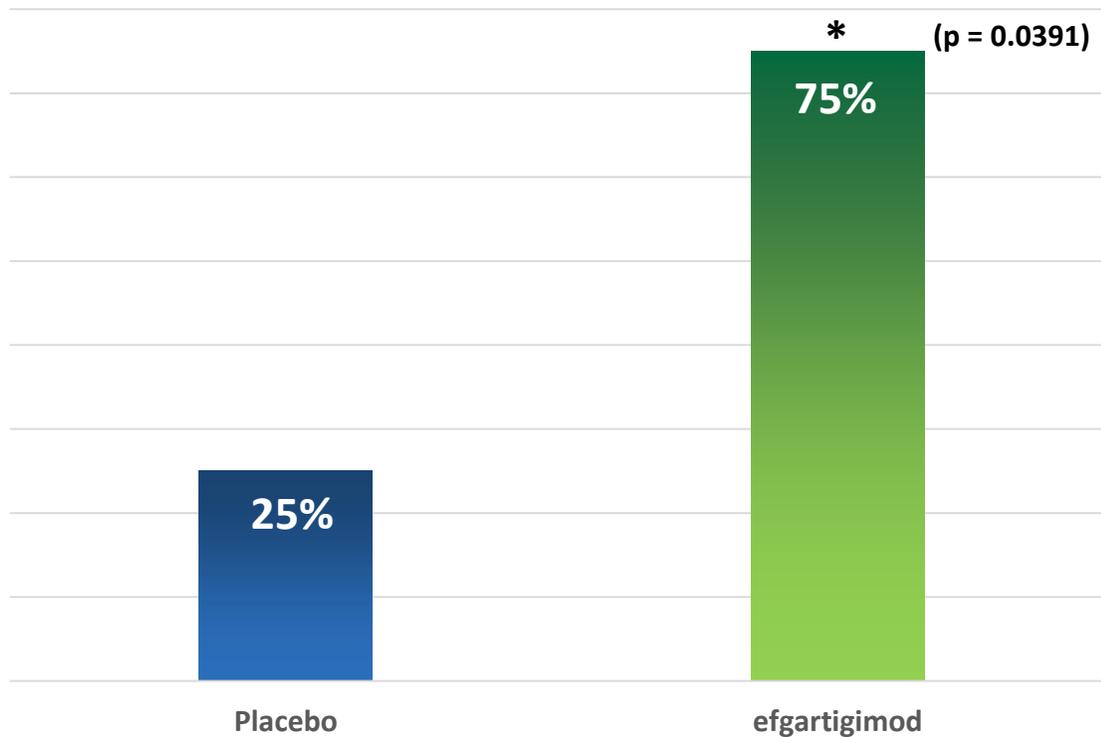
efgartigimod

- 83% of patients treated with efgartigimod achieved a clinically meaningful response (MG-ADL ≥2)

- 75% of patients treated with efgartigimod had a clinically meaningful and statistically significant improvement in MG-ADL score for a period of at least 6 consecutive weeks versus 25% of patients on placebo

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Robust Clinical Improvement Over Placebo Group

Day 29, 1 week after last dosing

Day 36, 2 weeks after last dosing

MG-ADL change from baseline

QMG change from baseline
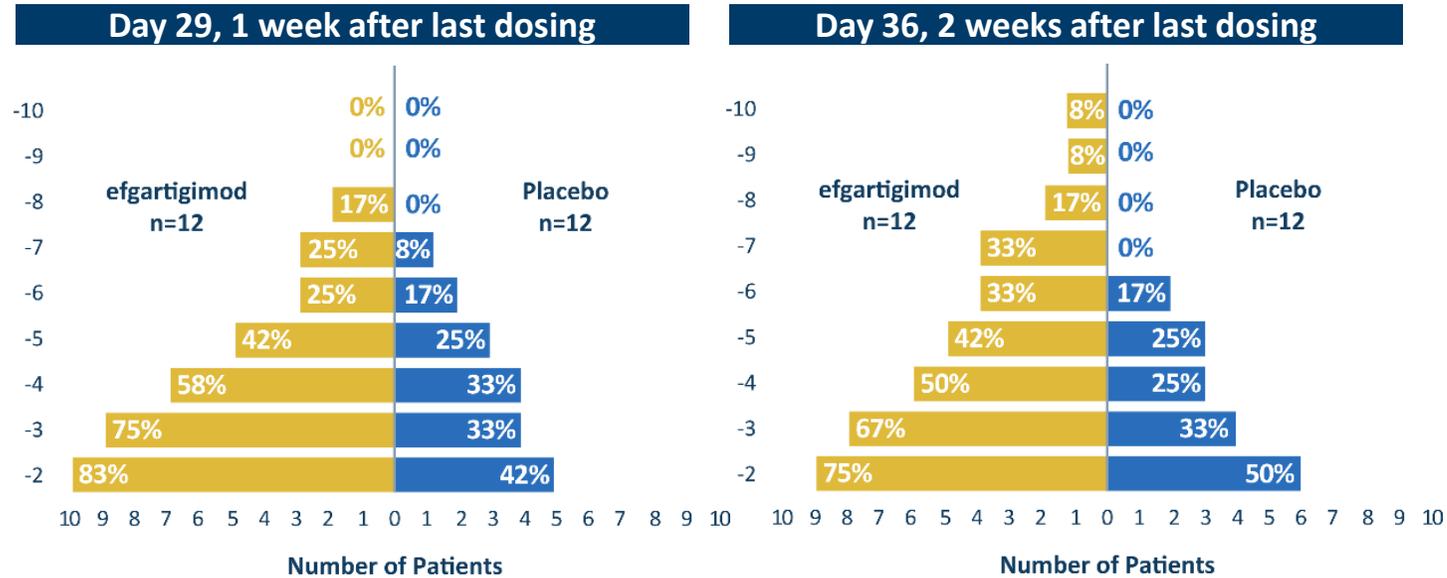
* Efgartigimod *vs.* placebo: increasing differentiation observed with increasing MG-ADL/QMG thresholds
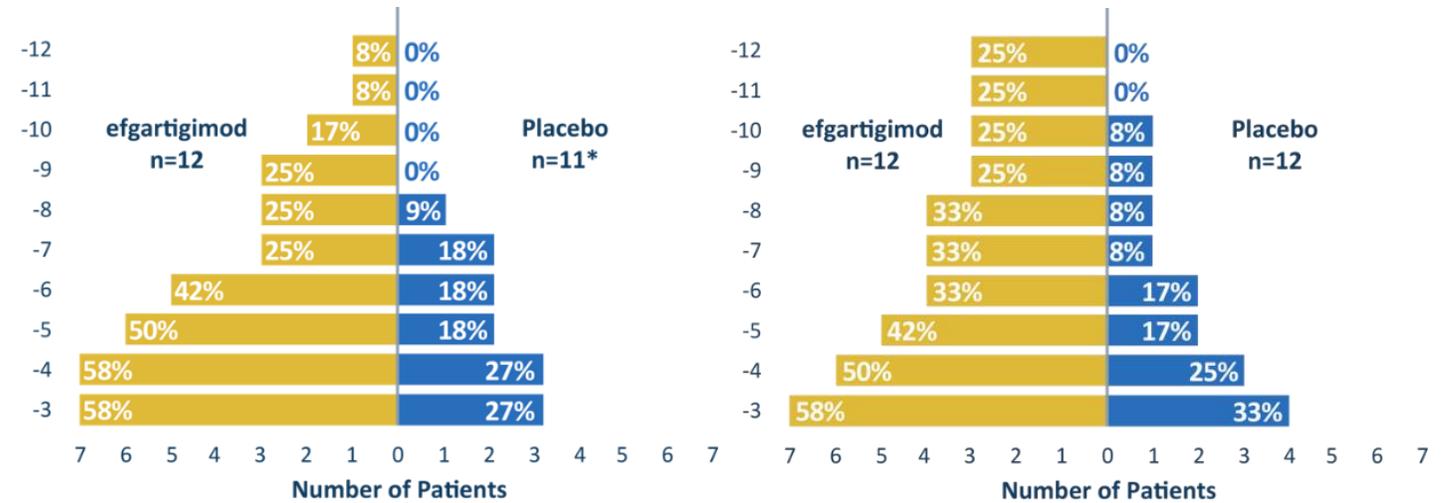
* Missing data point of 1 patient

Myasthenia Gravis Phase 3 ADAPT Trial Design
Same Primary Endpoint as Successful Phase 2 Trial

▶ Randomized, double-blind, placebo-controlled, multicenter trial enrolling 150 patients in North America, Europe and Japan

▶ 10 mg/kg intravenous (IV) dose of efgartigimod over 26-week period

▶ Enrolling AChR positive and AChR negative patients with disease driven primarily by MuSK and LRP4 autoantibodies

▶ Patients in the ADAPT trial will be able to roll over into an open-label extension trial for a period of one year

▶ First patient dosed in September 2018

▶ Based on PMDA feedback, this Phase 3 trial, if data is positive, to also serve as a basis for Japan registrational submission

adapt
myasthenia
gravis study
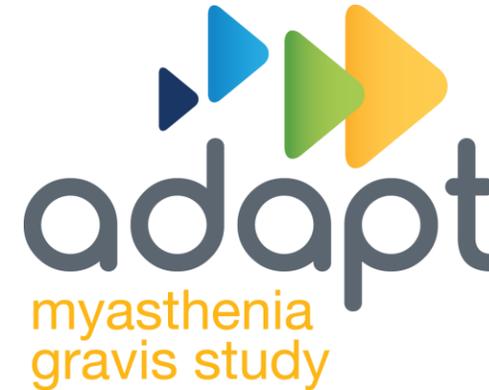
Primary endpoint

> **Myasthenia Gravis Activities of Daily Living (MG-ADL) Score**

Secondary endpoints

> **Efficacy, Safety, Tolerability, Quality of Life and Impact on Normal Daily Activities Measures**

ITP Phase 2 Results Establish Hematologic Beachhead

Novel approach beyond boosting platelet production or broad immuno-suppression

Favorable and consistent safety and tolerability profile
- No trends seen for infections or headaches across all studies
- No decreases in IgM, IgE, IgA or albumin

Robust efficacy signal in relapsed/refractory population after short drug exposure
- Clinically meaningful increase in platelet counts over placebo
- 50% of patients came on study with platelets $<15 \times 10^9$

Strong correlation between IgG reduction, platelet count improvement and reduction of bleeding events

Data enable Phase 3 in ITP (IV) and launch of Phase 2 in ITP (SC)

Efgartigimod Targets All Pathogenic AutoAb Actions Simultaneously

Potential to eliminate therapeutic cycling based on trial-and-error

1. Accelerate platelet clearance

FcγR
Platelets
Macrophage

IVIg Tavalisse Splenectomy Efgartigimod

2. Inhibit platelet production

Platelets
Megakaryocyte

TPO-RA Efgartigimod

4. Interfere with platelet function

Platelets
Fibrinogen receptor
vWF receptor Collagen receptor
Efgartigimod

3. Induce platelet killing

Efgartigimod

Autoantibodies
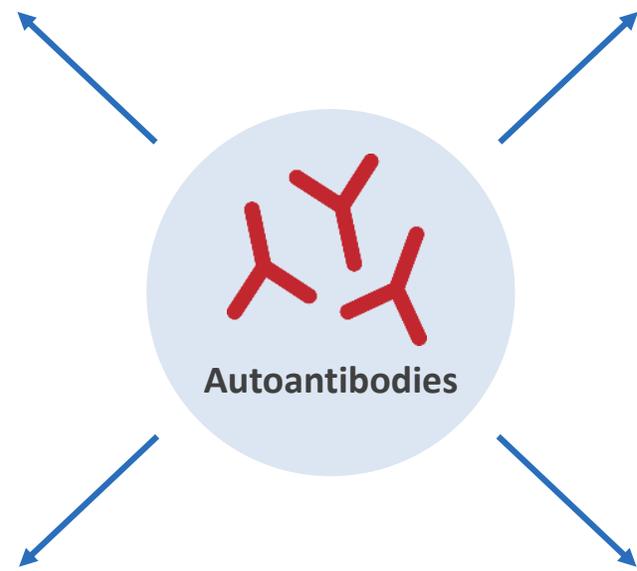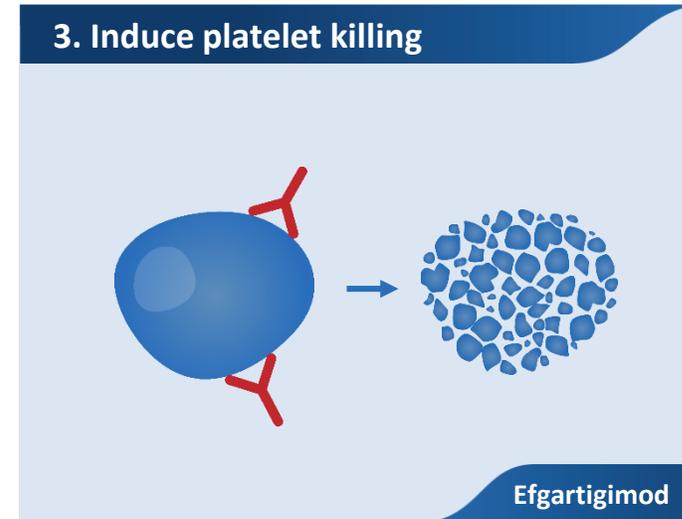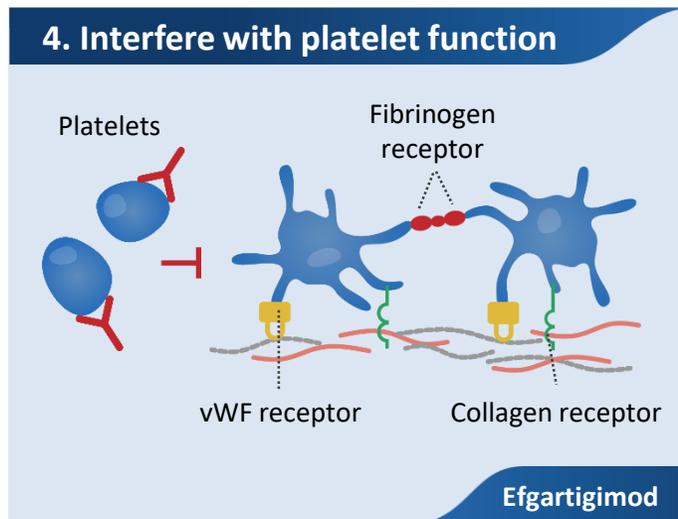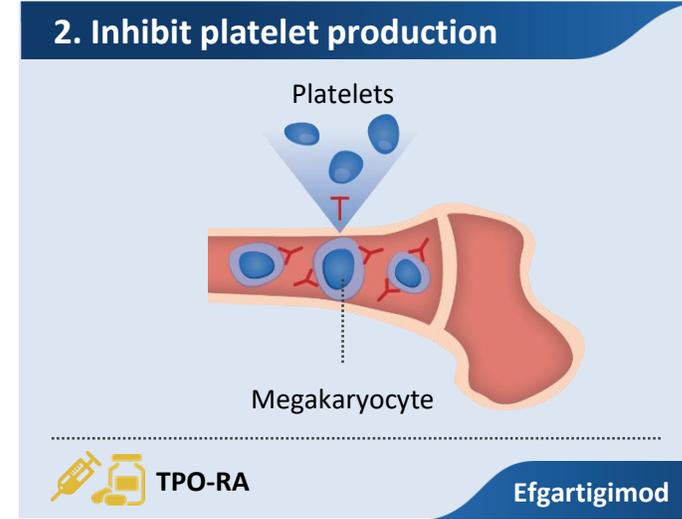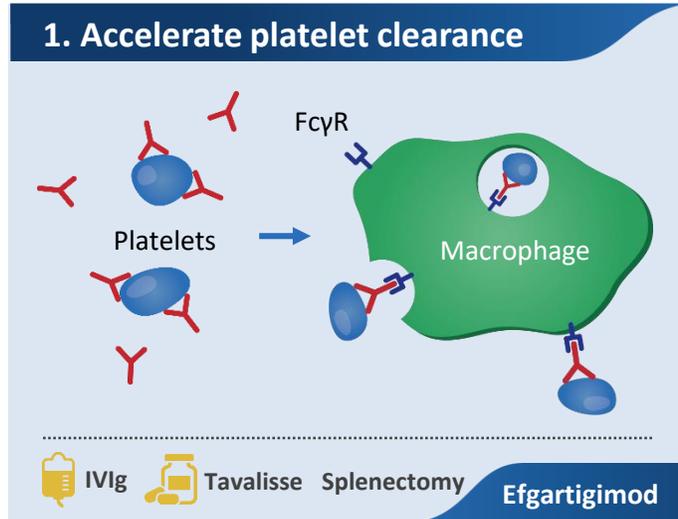
Favorable Tolerability Profile Consistent with Previous Studies

Treatment-emergent adverse events balanced between active and placebo arms

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- Tolerability profile consistent with Phase 2 myasthenia gravis (MG) and Phase 1 healthy volunteer (HV) trials
- TEAEs mostly mild in severity (grade 1)
- No deaths or TEAEs leading to discontinuation of treatment reported*

Bleeding TEAEs not included

Treatment Emergent Adverse Events (TEAEs) Reported in ≥ 2 subjects	Placebo (N = 12)	Efgartigimod 5 mg/kg (N = 13)	Efgartigimod 10 mg/kg (N = 13)
Most common TEAEs N (%)			
Headache	2 (16.7)	1 (7.7)	-
Hypertension	1 (8.3)	-	2 (15.4)
Vomiting	-	-	2 (15.4)
Cystitis	-	1 (7.7)	1 (7.7)
Rash	-	1 (7.7)	1 (7.7)
Productive cough	1 (8.3)	1 (7.7)	-
TEAEs *deemed related* to study intervention N (%)			
Headache	1 (8.3)	-	-
Vomiting	-	-	1 (7.7)
Pubic pain	1 (8.3)	-	-
Vaginal discharge	1 (8.3)	-	-
Amenorrhoea	1 (8.3)	-	-

* One thrombocytopenia downgraded per protocol after database lock
argenx data: Table 14.3.1.2a & 14.3.1.5a - Treatment-Emergent Adverse Events by System Organ Class, Preferred Term and Relationship to Study Drug - Main Study

Strong Improvement of Platelet Counts Across Doses

46-67% of patients exceeded platelet counts ≥ 50×10^9/L during at least two visits

Patients achieving platelet counts of ≥ 50×10^9/L at least two times

Main Study | OLE (1st treatment cycle)

% of patients with an improvement of platelet counts ≥ 50×10^9/L for at least two visits

- 25% N=3 — Placebo + SOC, N=12
- 46% N=6 — efgartigimod 5 mg/kg + SOC, N=13
- 46% N=6 — efgartigimod 10 mg/kg + SOC, N=13
- 67% N=8 — efgartigimod 10 mg/kg + SOC, N=12
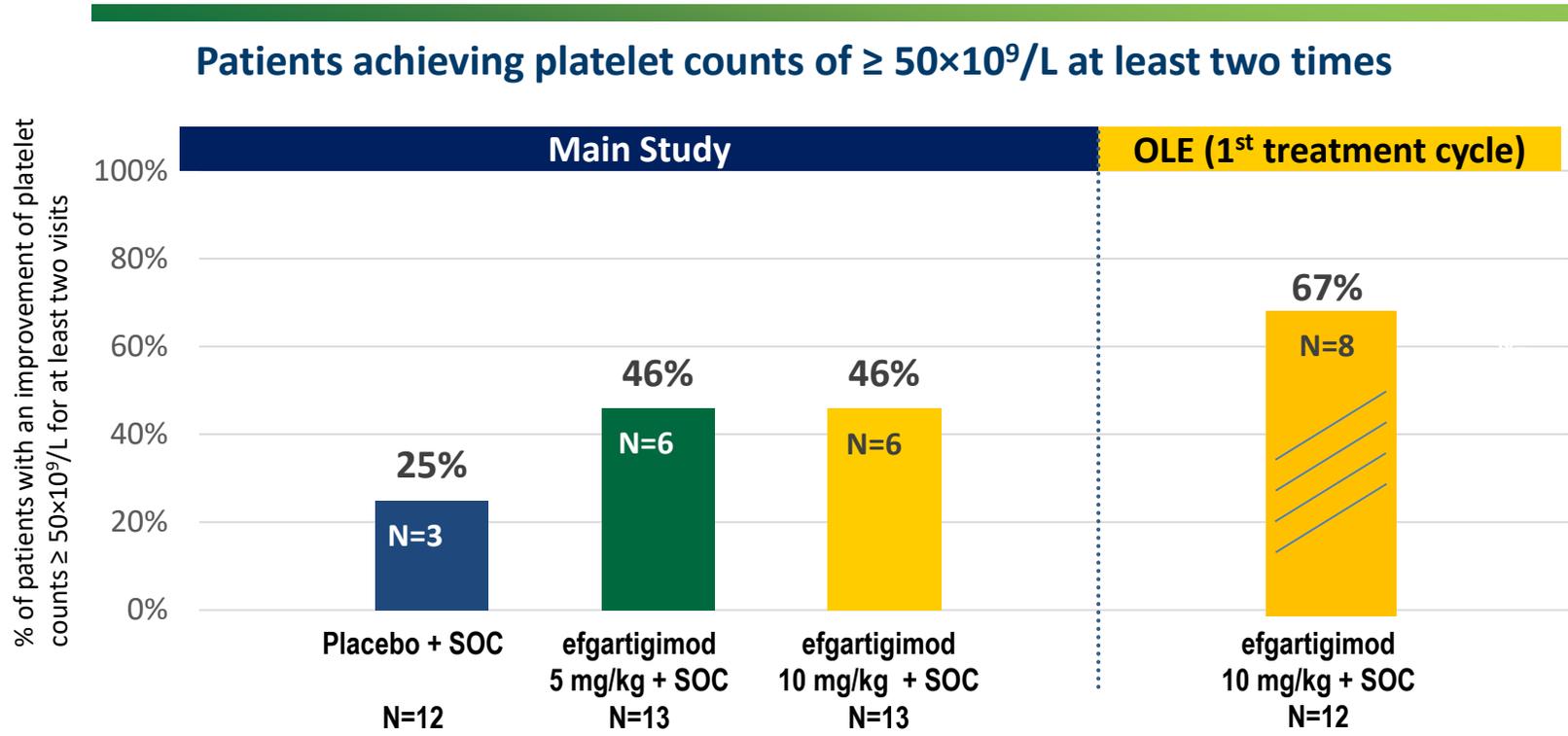
*After cut-off date not QC-ed

- OLE acts as true fourth cohort since patients' platelets had to fall below 30×10^9/L to be eligible for a treatment cycle; patients still in response from primary study were not eligible

- Responses seen across newly diagnosed (in 5mg/kg arm), persistent and chronic ITP patients

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Robust Improvement of Platelet Count

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Post-hoc analysis of increasing thresholds of efficacy

| placebo + SOC (N=12) | efgartigimod + SOC (pooled N=26) |

Response Rate (%)

≥ 50×10^9/L (>10 cumulative days)
: 0% (N=0) — (N=10) 38% *P*= 0.03

≥ 100×10^9/L
: 8% (N=1) — (N=11) 42%

≥ 50×10^9/L (at least two visits)
: 25% (N=3) — (N=12) 46%
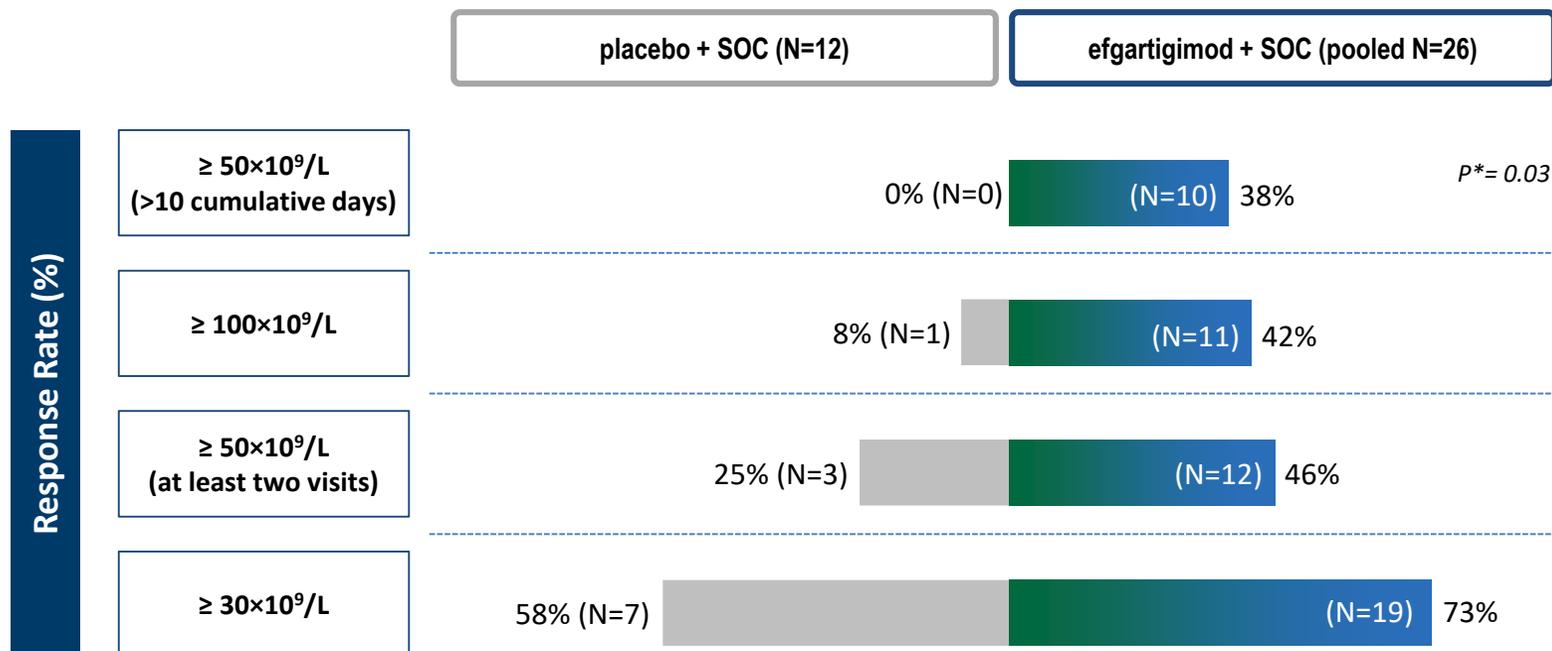
≥ 30×10^9/L
: 58% (N=7) — (N=19) 73%

- Efgartigimod generated therapeutic effect at multiple relevant thresholds of efficacy

- Duration of platelets remaining ≥50x10^9/L ranged from 1 - 20 weeks with five patients above that platelet threshold for more than a month

Note: Increasing threshold analysis based exact logistic regression model with the baseline result as a factor

Reduction of Total IgGs Correlates with Increased Platelet Counts and Reduced Bleeding Events

Mean platelet counts versus total WHO scale versus total IgGs

Placebo

10 mg/kg efgartigimod

- % total IgGs
- Mean platelet counts (x10⁹/L)
- % patients with total WHO scale >0

Pemphigus Vulgaris Phase 2 Cohort 1 Data
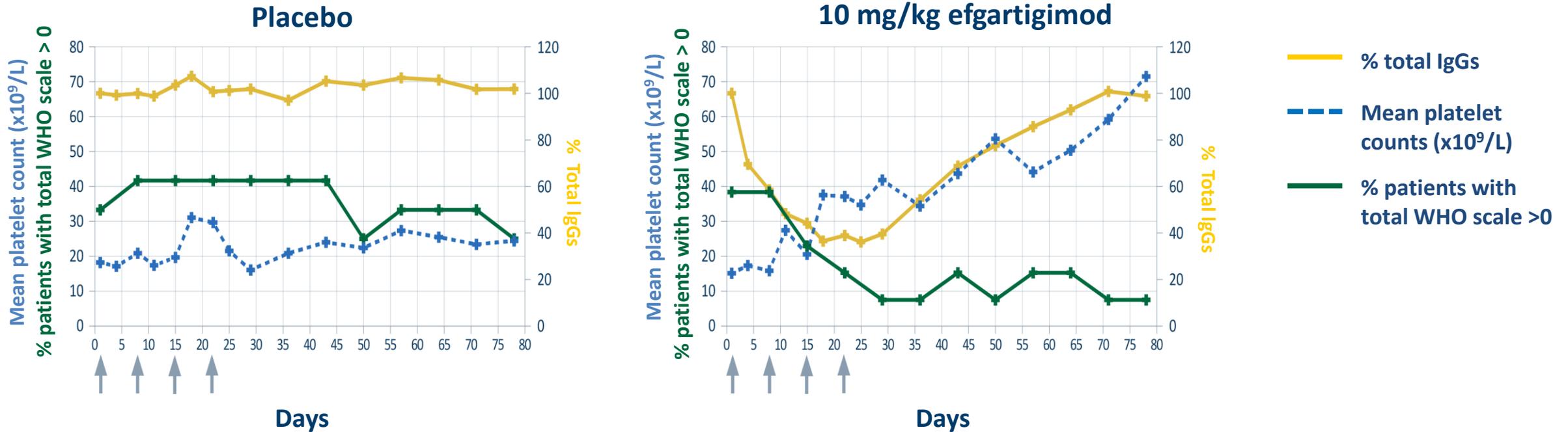
Rapid disease control in 4 out of 6 PV patients:
- 3 within 1 week
- 1 within 4 weeks

Patients with disease control:
- Mean max reduction in Pemphigus Disease Area Index (PDAI) score: 55%
- Mean max decrease in pathogenic IgGs: 57%

Favorable tolerability profile

No meaningful anti-drug antibody signals (ADA) reported

Pemphigus Vulgaris Phase 2 Adaptive Design

Third cohort to start in 1H 2019

Treatment Phase

Induction

3 weeks

COHORT 1: N= 4

efgartigimod (10 mg/kg)

4 infusions (weekly)

COHORT 2: N= 4 + 4

efgartigimod (10 mg/kg)

4 infusions (weekly)

Maintenance

6 weeks → 8 weeks

efgartigimod (10 mg/kg)

2 infusions (w2, w6)

efgartigimod (10 mg/kg)

4 infusions (w2, w4, w6, w8)

Follow-up Phase

8 weeks

IDMC recommendation for Cohort 3 to reach clinical remission (with/without minimal therapy):

- Weekly infusions 25 mg/kg (induction phase) until disease control (DC) with minimum of 5

- Biweekly dosing after DC

- Start maintenance based on DC

- Treatment duration limited to 34 weeks (induction + maintenance)

Efgartigimod: a Pipeline-in-a-Product Opportunity

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Landscape of IgG-mediated severe autoimmune diseases (sampling)

Immune Thrombocytopenia

Scleroderma

Lupus

Epidermolysis Bullosa Acquisita

Myasthenia Gravis

Rheumatoid Arthritis

Pemphigus

Multiple Sclerosis

Anca Vasculitis

Bullous Pemphigoid

Solid Biology Rationale
Disease proven to be predominantly mediated by pathogenic IgGs

Feasible for Biotech
Orphan potential, economically viable, efficient clinical & regulatory pathway

Proof-of Concept:

Myasthenia Gravis ✓

Immune Thrombocytopenia ✓

Pemphigus Vulgaris

Therapeutic Area Beachheads with Expansion Possibilities into Adjacent Indications

Neuromuscular Diseases

Hematology Disorders

Blistering Diseases

Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)

Phase 2 CIDP study to start in 2H 2019

ARGX-110 (Cusatuzumab): Phase 1 / 2
Mono & Combo Therapy

Cusatuzumab Mode-of-Action Targets both Leukemic Stem Cells and Blasts

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Cusatuzumab induces LSC differentiation

- **2** Kill LSCs
- LSC
- LSC
- CD27 CD70
- **1** Induce myeloid differentiation
- Differentiated cell

Cusatuzumab kills blasts

- **3** Kill Blasts
- Blast
- Blast
- CD27 CD70
- **4** Block proliferation & survival signal

Activation of the pathway leads to release of sCD27, which is a biomarker
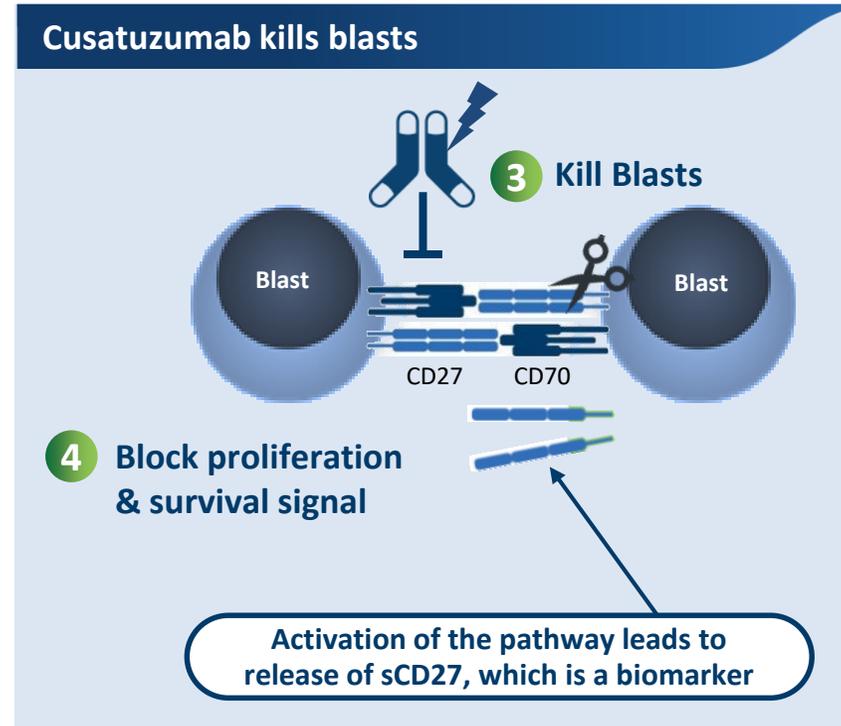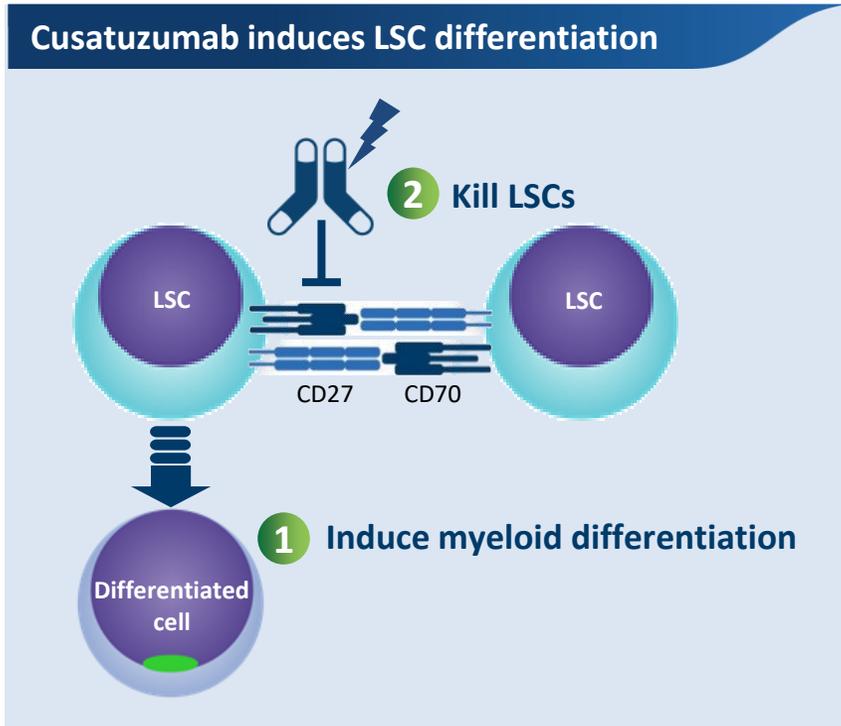
- Cusatuzumab is a potentially first-in-class anti-CD70 ADCC enhanced SIMPLE Antibody™ which selectively targets LSCs and blasts in AML and other heme indications

Silence et al. 2014, mAbs., Riether et al. 2017, J Exp Med.

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Ongoing Phase 1/2 Combination Trial

Newly diagnosed AML patients unfit for intensive chemotherapy

Open label, non-controlled, non-randomized

Phase 1 – Dose Escalation

Phase 2 – Proof of Concept at 10 mg/kg

20 mg/kg
N = 3+3

10 mg/kg
N = 3+3

Currently enrolling Phase 2

3 mg/kg
N = 3+3

Endpoints
- Safety, tolerability
- Clinical outcome
- Translational data

1 mg/kg
N = 3+3
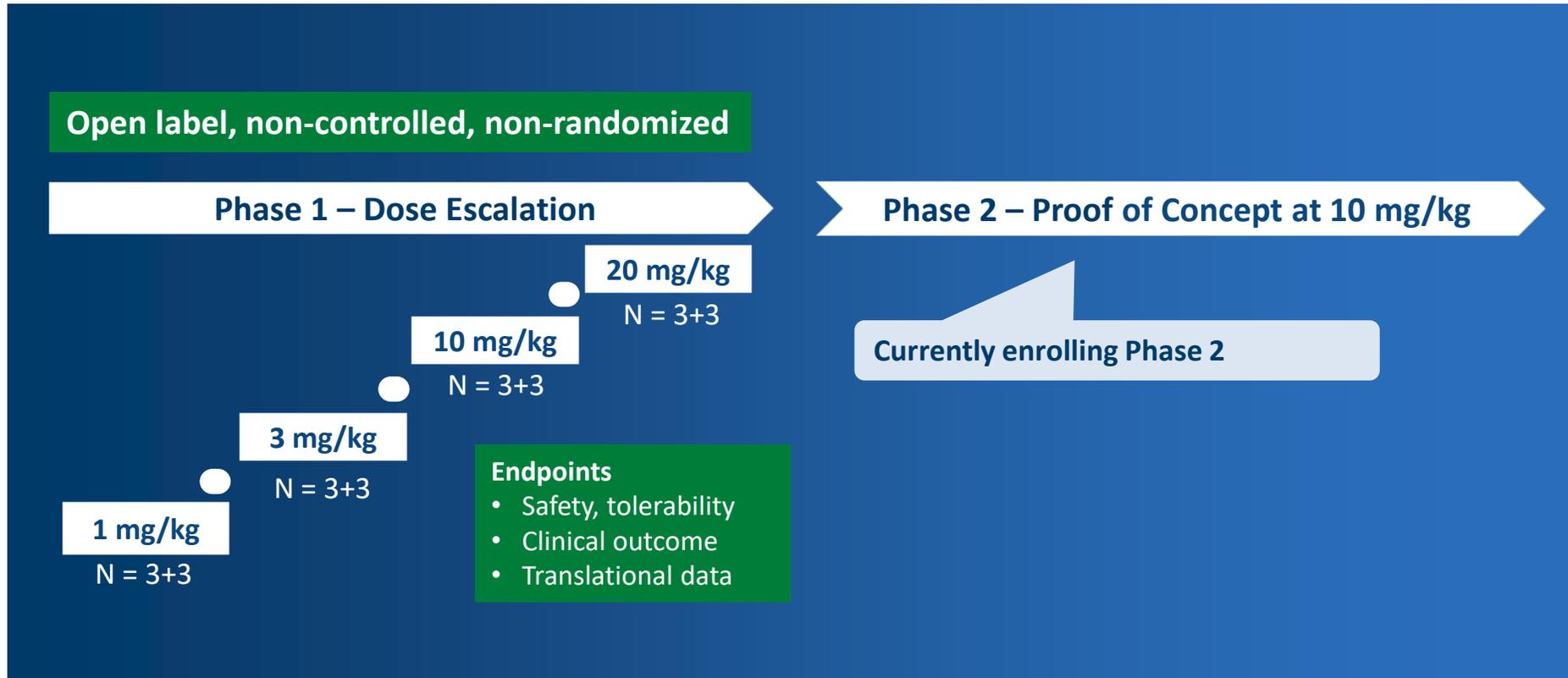
- Efficacy seen across doses in Phase 1 dose escalation
- Up to 21 patients to enroll in initial Phase 2 study with potential to expand enrollment to 40
- 10 mg/kg selected for Phase 2 to saturate bone marrow and maintain clean tolerability profile

Overall Conclusions: Phase 1 Dose Escalation

Favorable tolerability profile
- No obvious toxicity on top of Vidaza toxicity
- No dose-limiting toxicity observed

Encouraging proof-of-biology data in 12 patients (4 dose cohorts; 3 pts each)
- 92% response rate (11/12) mainly CR/CRi
- 3 patients responded after cusatuzumab monotherapy
- Significant blast reduction in bone marrow after cusatuzumab monotherapy
- MRD negativity in 42% (5/12) treated patients

Supported by **translational dataset**
- Decreased sCD27 levels
- Reduced LSC colony formation
- Increased myeloid differentiation – asymmetric division

Recommended Phase 2 dose: 10 mg/kg

92% (11/12) Response Rate – CR/CRi/PR

Three patients on study for more than 12 months
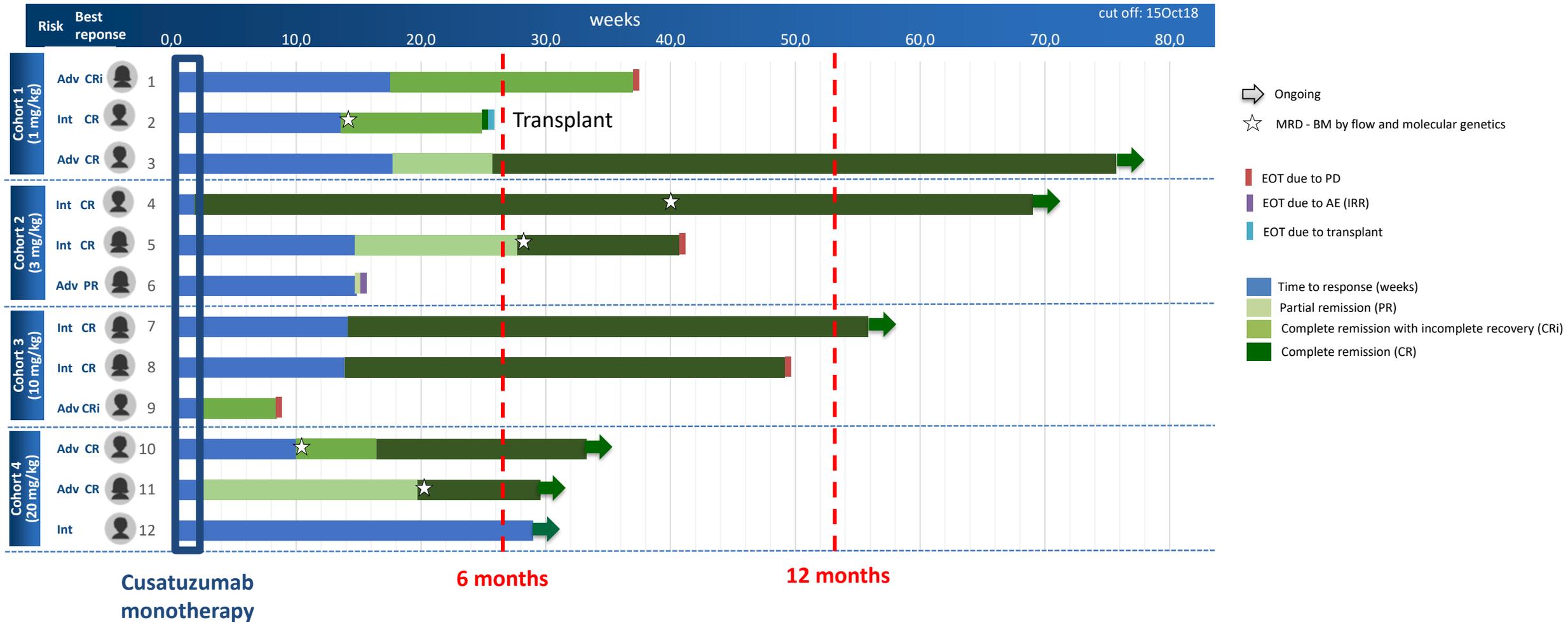
Business Development & Financials

Cusatuzumab Strategic Alliance with Janssen Pharmaceuticals

argenx objectives | Janssen alliance

argenx objectives	Janssen alliance
Accelerate & broaden development plan	Joint development plan focused on AML, MDS and other heme malignancies
Secure strong financials	Upfront $ 300m + $ 200m equity @ 20% premium, 1.3Bn in milestones, double digit royalties OUS
Retain commercial upside	50 % of US economics on a royalty basis, up to 50% commercial efforts

"We believe that cusatuzumab can become a foundational therapy
for all lines of AML and high-risk MDS." Brian Kenney, J&J spokesperson

AbbVie Partnership for Novel Target GARP

argenx

Initiation
first-in-human
clinical trial
$30mm milestone
payment

Strategic Antibody Collaboration Details

- **GARP** is a protein specifically present on the surface of activated regulatory T-cells (Tregs)

- **AbbVie** exercised option in August 2018 to:
  - Obtain exclusive, worldwide license to develop and commercialize ARGX-115
  - Fund further GARP-related research by argenx beyond ARGX-115

- **argenx** can study ARGX-115 in combo with its pipeline programs
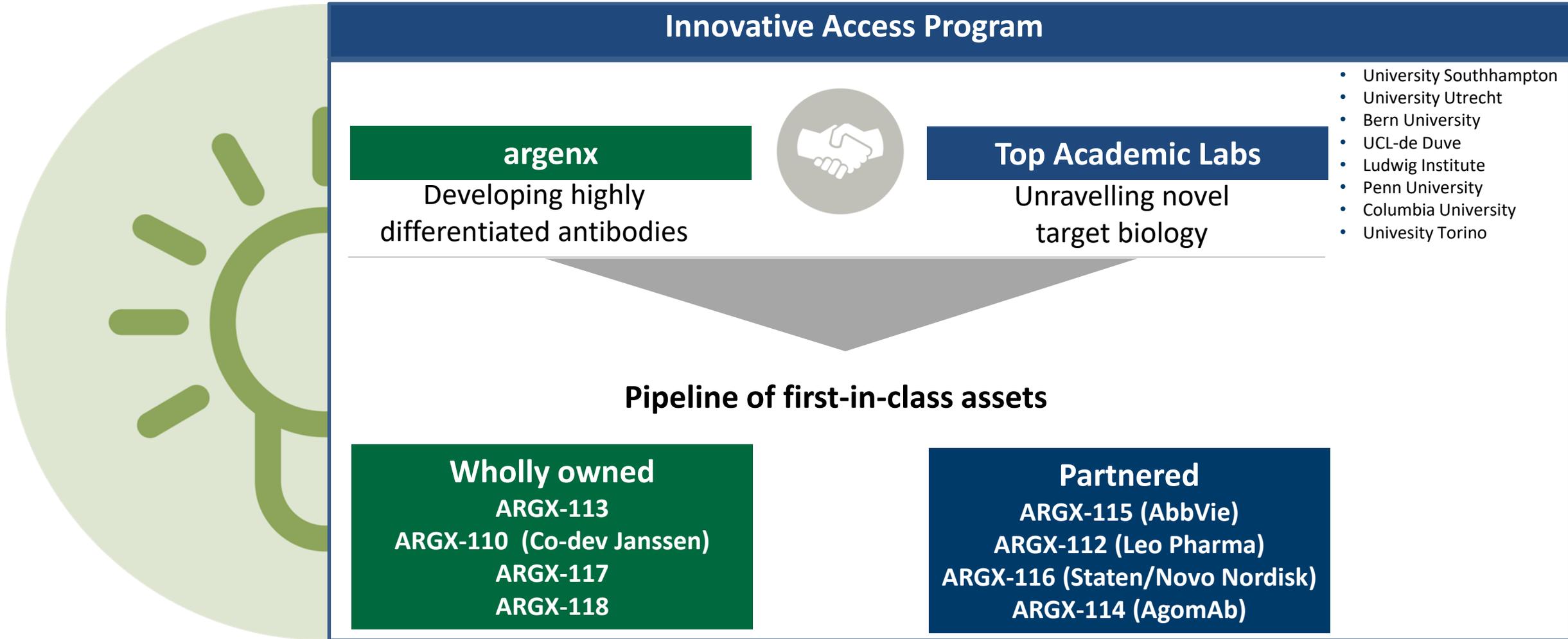
Financial Highlights

- **$90mm** received to date

- **$625mm in potential** development, regulatory and commercial **milestones**

- **Tiered royalties** on sales at percentages ranging from **mid-single digits to low teens**

- **Co-promotional** rights for ARGX-115-based products in the **European Economic Area and Switzerland**

Cuende et al., 2015, Science Trans Med.

35

Success formula with proven track record

Innovative Access Program

argenx	🤝	**Top Academic Labs**
Developing highly differentiated antibodies		Unravelling novel target biology

- University Southhampton
- University Utrecht
- Bern University
- UCL-de Duve
- Ludwig Institute
- Penn University
- Columbia University
- Univesity Torino

Pipeline of first-in-class assets

Wholly owned	**Partnered**
ARGX-113	**ARGX-115 (AbbVie)**
ARGX-110 (Co-dev Janssen)	**ARGX-112 (Leo Pharma)**
ARGX-117	**ARGX-116 (Staten/Novo Nordisk)**
ARGX-118	**ARGX-114 (AgomAb)**

argenx

Additional Key Statistics – Dec 31, 2018

- **Cash position: €564.6 mm** (+ $500 mm Janssen deal)

- **Capital raised since inception: €717 mm (ex. grants)**
  - 2017: raised $115 mm (€102 mm) in Nasdaq IPO
  - 2017: raised $266 mm (€226 mm) in public offering
  - 2018: raised $300 mm (€256 mm) in public offering

- **Non-dilutive funding since inception: €137mm (incl. grants)**
  - 2018: $10mm second preclinical milestone AbbVie
  - 2019: $30mm first clinical milestone AbbVie

- **132 employees & consultants —97 R&D, 35 SG&A**

Blue-Chip Investor Base

8% 2%

- Historical shareholders
- Free float
- Stock options

90%
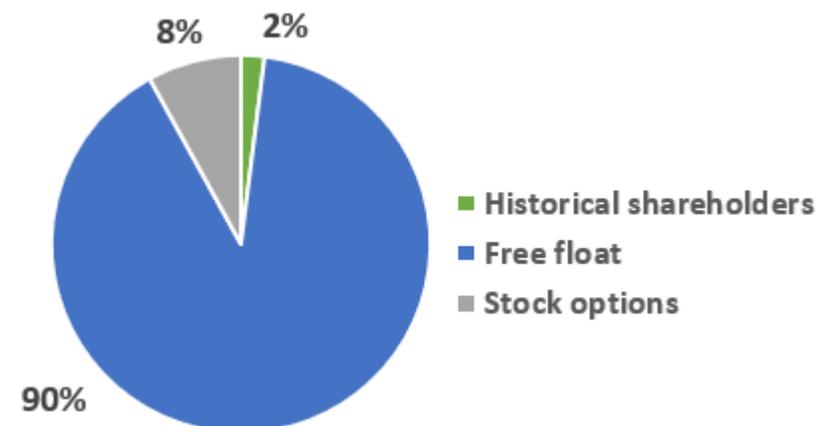
- Outstanding shares (Feb 22, 2019): **37,907,551**
- Outstanding stock options (Feb 22, 2019): **3,371,311**
- U.S. shareholding: **above 70%**

Key Upcoming Expected Milestones & Communications

argenx

2019	Q1	Q2

**R&D day
May 22 (NY)**

Efgartigimod
- ITP: Outcome FDA/PMDA/EMA EoPh2 meeting
- PV: Start Cohort 3 Ph2 1H
- ITP: Launch Ph2 SC 1H

Cusatuzumab
- Potential Milestones in Strategic Partnership with Janssen

New assets
- ARGX-117 First Indication
- ARGX-118 New Asset

Partnerships
- ✓ Exclusive collaboration Halozyme for ENHANZE®
- ✓ AbbVie: First-in-human clinical trial $30mm milestone payment
- ✓ ARGX-114: Spun out into AgomAb
- Potential Milestones

2019	Q3	Q4

**Breakfast event
CIDP (NY)**

Efgartigimod
- ITP: Launch Ph3 IV 2H
- CIDP: Launch Ph2 2H

Cusatuzumab
- Potential Milestones in Strategic Partnership with Janssen

New assets
- ARGX-117 CTA Filling

Partnerships
- Potential Milestone(s)

Thank you!