Developing Highly Differentiated Antibody Therapeutics

August 2018
Forward Looking Statements

Safe Harbor: 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. Examples of such forward-looking statements include those regarding our investigational product candidates and preclinical and clinical trials and the status and related results thereto, future results of operations and financial positions, business strategy, plans and our objectives for future operations. When used in this presentation, the words “anticipate,” “believe,” “can,” “could,” “estimate,” “expect,” “intend,” “is designed to,” “may,” “might,” “will,” “plan,” “potential,” “predict,” “objective,” “should,” or the negative of these and similar expressions identify forward-looking statements. Such statements, based as they are on the current analysis and expectations of management, inherently involve numerous risks and uncertainties, known and unknown, many of which are beyond the Company’s control. Such risks include, but are not limited to: the impact of general economic conditions, general conditions in the biopharmaceutical industries, changes in the global and regional regulatory environments in the jurisdictions in which the Company does or plans to do business, market volatility, fluctuations in costs and changes to the competitive environment. Consequently, actual future results may differ materially from the anticipated results expressed in the forward-looking statements. In the case of forward-looking statements regarding investigational product candidates and continuing further development efforts, specific risks which could cause actual results to differ materially from the Company’s current analysis and expectations include:

failure to demonstrate the safety, tolerability and efficacy of our product candidates; final and quality controlled verification of data and the related analyses; the expense and uncertainty of obtaining regulatory approval, including from the U.S. Food and Drug Administration and European Medicines Agency; the possibility of having to conduct additional clinical trials and our reliance on third parties such as our licensors and collaboration partners regarding our suite of technologies and product candidates. Further, even if regulatory approval is obtained, biopharmaceutical products are generally subject to stringent on-going governmental regulation, challenges in gaining market acceptance and competition. These statements are also subject to a number of material risks and uncertainties that are described in the Company’s filings with the U.S. Securities and Exchange Commission (“SEC”), 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. The reader should not place undue reliance on any forward-looking statements included in this presentation. These statements speak only as of the date made and the Company is under no obligation and disavows any obligation to update or revise such statements as a result of any event, circumstances or otherwise, unless required by applicable legislation or
Rapidly Emerging Leadership in Immunology
Pioneering differentiated therapeutic antibodies in severe autoimmune diseases and cancer

Winning Strategy
via pipeline in a product approach targeting commercially-viable orphan indications

Demonstrated Proof-of-Concept
across multiple programs – first- and best-in-class potential

Novel Target Biology

Sustainable Value Creation

De-Risked Platform
advanced technology suite – new pipeline candidate each year

Innovative Access Program
enable collaborations and efficient pipeline expansion

Highly strategic portfolio of products and disciplined business model generating differentiated and innovative candidates
Strong Momentum with Important Near-Term Catalysts

Four additional data milestones before YE 2018

### 2018 Accomplishments To-Date

**ARGX-113 efgartigimod**
- MG: Presented full Phase 2 data at AAN
- MG: End-of-Phase 2 meeting with FDA
- PV: Interim data from first cohort in Phase 2
- SC: Phase 1 data using subcutaneous formulation

**ARGX-110**
- AML: Ongoing enrollment in Phase 2 trial

### Upcoming Milestones

**ARGX-113 efgartigimod**
- MG: Phase 3 launch before year end 2018
- ITP: Phase 2 topline data in 3Q18
- ITP: Phase 2 full data at ASH
- PV: Phase 2 full data 1H19

**ARGX-110**
- AML: Phase 1 dose-escalation full data at ASH
- CTCL: Phase 2 full data at ASH

**ARGX-117**
- Novel complement target; potential synergies with ARGX-113

**Other Programs**
- Potential collaborations and partnerships through Innovative Access Program
Augmenting Intrinsic Therapeutic Properties Of Antibodies

<table>
<thead>
<tr>
<th>Antibody</th>
<th>Technology Role</th>
<th>Suite of Technologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>V-region</td>
<td>Unlock novel and complex targets</td>
<td>SIMPLE Antibody™ Platform</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Llama immune system delivers V-regions with high human homology</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Highly diverse antibody output covers a multitude of target epitopes</td>
</tr>
<tr>
<td>Fc region</td>
<td>Modulate immune response</td>
<td>NHance®</td>
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<tr>
<td></td>
<td></td>
<td>• Extends half-life / PD effect</td>
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<td></td>
<td></td>
<td>• Enhances tissue penetration</td>
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<td></td>
<td></td>
<td>ABDEG™</td>
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<tr>
<td></td>
<td></td>
<td>• Clears disease target</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Clears autoantibodies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>POTELLIGENT®</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Boosts cell killing</td>
</tr>
</tbody>
</table>

We apply our unique suite of technologies to create differentiated product candidates against novel targets

Klarenbeek et al. 2015, mAbs
Basilico et al. 2014, J Clin Inv.
<table>
<thead>
<tr>
<th>Product Candidate</th>
<th>Target</th>
<th>Indication</th>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Next Milestone / Commentary</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Wholly-Owned Product Candidates</strong></td>
<td></td>
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<tr>
<td>ARGX-113 (eفارگرگینومود)</td>
<td>FcRn</td>
<td>Myasthenia Gravis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2H18: Start Phase 3</td>
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<td></td>
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<td>Immune Thrombocytopenia</td>
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<td></td>
<td></td>
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<td>3Q18: Phase 2 topline results</td>
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<td></td>
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<td>Pemphigus Vulgaris</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Phase 2 interim data</td>
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<tr>
<td></td>
<td></td>
<td>Subcutaneous Formulation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Phase 1 data</td>
</tr>
<tr>
<td>ARGX-110</td>
<td>CD70</td>
<td>T-Cell Lymphoma</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ASH18: Phase 2 topline results CTCL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acute Myeloid Leukemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ASH18: Phase 1 full data in AML/MDS</td>
</tr>
<tr>
<td>ARGX-117</td>
<td>Novel complement target</td>
<td>Severe Autoimmune Diseases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Antibody-mediated autoimmune diseases Complementary to ARGX-113</td>
</tr>
<tr>
<td>ARGX-111</td>
<td>c-MET</td>
<td>Solid Tumors / Blood Cancer</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Available for partnering</td>
</tr>
<tr>
<td><strong>Partnered Product Candidates</strong></td>
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</tr>
<tr>
<td>ARGX-109 (gerিম্মুজ্মাব)</td>
<td>IL-6</td>
<td>Rheumatoid Arthritis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Development for Chinese market</td>
</tr>
<tr>
<td>ARGX-112</td>
<td>IL-22R</td>
<td>Skin Inflammation</td>
<td></td>
<td></td>
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<td></td>
<td>Eligible for up to ~€100mm in milestones and tiered royalties</td>
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<tr>
<td>ARGX-115</td>
<td>GARP</td>
<td>Cancer Immunotherapy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Received $60mm so far; eligible for up to $625mm milestones &amp; tiered royalties</td>
</tr>
<tr>
<td>ARGX-116</td>
<td>ApoC3</td>
<td>Dyslipidemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Eligible for double-digit royalties and exclusive option to license the program</td>
</tr>
</tbody>
</table>

• **Innovative Access Program**: 7 live programs
• Antibody discovery alliance with Shire focused on multiple rare disease targets – 2 options exercised
Efgartigimod: A Pipeline-in-a-Product Opportunity
Efgartigimod is a human IgG1 Fc-fragment that utilizes ABDEG™ Fc engineering technology

Efgartigimod cannot engage Fcγ receptors when bound to its target FcRn

Efgartigimod targets and binds to FcRn blocking the recycling of IgG leading to an elimination of IgG antibodies

Pathogenic IgG antibodies mediate multiple autoimmune diseases

(3) argenx data
Efgartigimod: Pipeline-In-Product Opportunity
Prioritizing IgG autoantibody mediated diseases

Landscape of IgG severe autoimmune diseases (selection)

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

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

Proof of Concept Value
Spill-over effect into adjacent indications

Myasthenia Gravis
Beachhead neuromuscular diseases

Immune Thrombocytopenia
Beachhead heme disorders

Pemphigus Vulgaris
Beachhead blistering diseases

argenx data
Myasthenia Gravis Overview

What is Myasthenia Gravis (MG)?

- Rare autoimmune disorder;
  - 64,000\(^{(1)}\) patients in U.S.
  - 55,000\(^{(2)}\) with generalized MG (gMG)
- Severe muscle weakness
- Symptoms include: drooping eyelids, double vision, difficulty to speak/swallow, generalized muscle weakness, life-threatening choking

Limited current treatment options with severe side effects

- Cholinesterase inhibitors
- Corticosteroids
- Immunosuppressants
- IVIg, Plasmapheresis (exacerbations or rescue)
- Soliris\(^{®}\)
- Thymectomy (minority of patients)

IVIg, Plasmapheresis and Soliris\(^{®}\) place a heavy cost burden on healthcare systems (~$79,000\(^{(3)}\), ~$101,000\(^{(3)}\) and ~$700,000\(^{(4)}\))

Autoantibodies (IgG type) impact neuromuscular junctions:
- Blocking of Acetylcholine Receptors (AChRs)
- Cross-linking + internalization of AChRs
- Complement recruitment

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(1) Philips et al. 2003, Ann N Y Acad Sci
(3) Heatwole et al. 2011, J Clin Neuromuscul Dis.
(4) Source: Reprinted with permission by First Databank Inc. WAC = Wholesale Acquisition Cost 8/21/17
## Autoantibody Levels (IgGs) Correlate With MG Disease Score

### Comparison between 3 cycles of Plasmapheresis/Immunoadsorption every 24h-48h and 5 cycles of IVIg every 24h

- **Clinically effective if disease score has improved by >50% 14 days after treatment**

### Treatment*

<table>
<thead>
<tr>
<th></th>
<th>Plasmapheresis</th>
<th>Immuno-adsorption</th>
<th>IVIg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decrease in autoantibody levels (%) after treatment</td>
<td>62.6 ± 0.9</td>
<td>55.1 ± 3.2</td>
<td>28.9 ± 3.8</td>
</tr>
<tr>
<td>Decrease in disease score (%) after treatment</td>
<td>60.8 ± 3.5</td>
<td>42.4 ± 4.2</td>
<td>23.8 ± 3.7</td>
</tr>
<tr>
<td>Clinical efficacy rate after 14 days**</td>
<td>12/15</td>
<td>7/10</td>
<td>6/15</td>
</tr>
<tr>
<td>Duration of hospital stay (days)</td>
<td>12.80 ± 0.28</td>
<td>13.50 ± 0.50</td>
<td>16.00 ± 0.50</td>
</tr>
</tbody>
</table>

* Comparison between 3 cycles of Plasmapheresis/Immunoadsorption every 24h-48h and 5 cycles of IVIg every 24h

** Clinically effective if disease score has improved by >50% 14 days after treatment

---

>30% autoantibody reduction clinically meaningful

### Degree of autoantibody reduction correlates with clinical improvement and reduced hospital stay

Liu et al. 2010, Ther Apher Dial.
Efgartigimod: Selective and Lasting IgG Reduction

PD data multiple ascending dose (MAD) study in healthy volunteers

- Potent IgG reduction across isotypes (AChR autoantibodies are IgG1/3; MuSK autoantibodies are IgG4)
- Up to 85% total IgG reduction; single dose delivers 50% total IgG reduction
- After last dose, IgG levels remain reduced by 50% or more for ~3 weeks, return to baseline after > 1 month
- Comparable data for 25 mg/kg, every 7 days (data not shown)
Myasthenia Gravis Phase 2 Trial Design

Screening/Randomization

<table>
<thead>
<tr>
<th>Key inclusion criteria</th>
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<tbody>
<tr>
<td>Generalized MG patients</td>
</tr>
<tr>
<td>MGFA Class II, III, or IVa</td>
</tr>
<tr>
<td>Positive for anti-AChR auto-antibodies</td>
</tr>
<tr>
<td>MG ADL score of ≥ 5 at screening(*)</td>
</tr>
<tr>
<td>On a stable dose of their SoC</td>
</tr>
</tbody>
</table>

Treatment Phase

<table>
<thead>
<tr>
<th>SoC + ARGX-113 (10mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N=12</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SoC + Placebo</th>
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</thead>
<tbody>
<tr>
<td>N=12</td>
</tr>
</tbody>
</table>

Follow-up Phase

<table>
<thead>
<tr>
<th>3 weeks</th>
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<tbody>
<tr>
<td>8 weeks</td>
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</tbody>
</table>

Primary endpoint

Safety & Tolerability

Secondary endpoints

Efficacy
(MG-ADL; QMG; MGC; MG-QoL)

PK

PD
total IgG; pathogenic IgG

Immunogenicity

(*) >50% of the score attributed to non-ocular items

Clinicaltrials.gov: NCT029655573, argenx data
# Baseline Population and Disease Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Placebo (N = 12)</th>
<th>Efgartigimod (N = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (mean ± SD)</strong></td>
<td>43.5 ± 19.3</td>
<td>55.3 ± 13.6</td>
</tr>
<tr>
<td><strong>Gender (N (%))</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Male</td>
<td>4 (33.3%)</td>
<td>5 (41.7%)</td>
</tr>
<tr>
<td>• Female</td>
<td>8 (66.7%)</td>
<td>7 (58.3%)</td>
</tr>
<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Asian</td>
<td>-</td>
<td>1 (8.3%)</td>
</tr>
<tr>
<td>• Black / African American</td>
<td>1 (8.3%)</td>
<td>-</td>
</tr>
<tr>
<td>• White</td>
<td>11 (91.7%)</td>
<td>11 (91.7%)</td>
</tr>
<tr>
<td><strong>MGFA Disease Class at Screening</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Class II</td>
<td>7 (58.4%)</td>
<td>6 (50.0%)</td>
</tr>
<tr>
<td>• Class III</td>
<td>4 (33.3%)</td>
<td>6 (50.0%)</td>
</tr>
<tr>
<td>• Class IV</td>
<td>1 (8.3%)</td>
<td>-</td>
</tr>
<tr>
<td><strong>Baseline QMG score (mean ± SD) (min, median, max score)</strong></td>
<td>11.8 ± 5.4 (3, 12.5, 24)</td>
<td>14.5 ± 6.3 (6, 14, 30)</td>
</tr>
<tr>
<td><strong>Baseline MG-ADL score (mean ± SD) (min, median, max score)</strong></td>
<td>8.0 ± 2.2 (5, 8, 13)</td>
<td>8.0 ± 3.0 (5, 7.5, 15)</td>
</tr>
<tr>
<td><strong>Baseline MGC score (mean ± SD)</strong></td>
<td>14.5 ± 4.5</td>
<td>16.7 ± 8.7</td>
</tr>
<tr>
<td><strong>Baseline MGQoL score (mean ± SD)</strong></td>
<td>14.5 ± 6.1</td>
<td>19.7 ± 5.7</td>
</tr>
<tr>
<td><strong>SoC</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Acetylcholinesterase inhibitors N (%)</td>
<td>11 (91.7%)</td>
<td>12 (100.0%)</td>
</tr>
<tr>
<td>• Corticosteroids N (%)</td>
<td>5 (41.7%)</td>
<td>8 (66.7%)</td>
</tr>
<tr>
<td>• Immunosuppressants N (%)</td>
<td>2 (16.7%)</td>
<td>9 (75.0%)</td>
</tr>
</tbody>
</table>
Efgartigimod Safety And Tolerability Profile

2 hour infusion enabling out-patient administration

- Efgartigimod was well-tolerated in patients confirmed findings from Phase 1 healthy volunteer trial
- The TEAEs 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 the trial

### Treatment Emergent Adverse Events (TEAEs)

<table>
<thead>
<tr>
<th></th>
<th>Placebo (N = 12)</th>
<th>Efgartigimod (N = 12)</th>
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</thead>
<tbody>
<tr>
<td><strong>TEAEs (Total)</strong></td>
<td>10 (83.3%)</td>
<td>10 (83.3%)</td>
</tr>
<tr>
<td><strong>Headache</strong></td>
<td>3 (25.0%)</td>
<td>4 (33.3%)</td>
</tr>
<tr>
<td><strong>Nausea</strong></td>
<td>1 (8.3%)</td>
<td>1 (8.3%)</td>
</tr>
<tr>
<td><strong>Diarrhea</strong></td>
<td>1 (8.3%)</td>
<td>1 (8.3%)</td>
</tr>
<tr>
<td><strong>Abdominal pain upper</strong></td>
<td>1 (8.3%)</td>
<td>1 (8.3%)</td>
</tr>
<tr>
<td><strong>Arthralgia</strong></td>
<td>2 (16.7%)</td>
<td>-</td>
</tr>
<tr>
<td><strong>B-lymphocyte decrease</strong></td>
<td>-</td>
<td>2 (16.7%)</td>
</tr>
<tr>
<td><strong>Lymphocyte count decrease</strong></td>
<td>-</td>
<td>2 (16.7%)</td>
</tr>
<tr>
<td><strong>Monocyte count decrease</strong></td>
<td>-</td>
<td>2 (16.7%)</td>
</tr>
<tr>
<td><strong>Neutrophil count increase</strong></td>
<td>-</td>
<td>2 (16.7%)</td>
</tr>
<tr>
<td><strong>Myalgia</strong></td>
<td>-</td>
<td>2 (16.7%)</td>
</tr>
<tr>
<td><strong>Pruritus</strong></td>
<td>2 (16.7%)</td>
<td>1 (8.3%)</td>
</tr>
<tr>
<td><strong>Rhinorrhea</strong></td>
<td>1 (8.3%)</td>
<td>1 (8.3%)</td>
</tr>
<tr>
<td><strong>Tooth abscess</strong></td>
<td>2 (16.7%)</td>
<td>-</td>
</tr>
<tr>
<td><strong>Toothache</strong></td>
<td>2 (16.7%)</td>
<td>-</td>
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</table>

**Efgartigimod deemed related TEAEs**

<table>
<thead>
<tr>
<th></th>
<th>Placebo (N = 12)</th>
<th>Efgartigimod (N = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Headache</strong></td>
<td>1 (8.3%)</td>
<td>3 (25.0%)</td>
</tr>
<tr>
<td><strong>Monocyte count decrease</strong></td>
<td>0 (0.0%)</td>
<td>2 (16.7%)</td>
</tr>
<tr>
<td><strong>Rhinorrhea</strong></td>
<td>1 (8.3%)</td>
<td>1 (8.3%)</td>
</tr>
</tbody>
</table>
Left:

- PD effect of efgartigimod in the Phase 2 clinical trial very similar to the Phase 1 trial in healthy volunteers
- Significant IgG reduction across IgG subtypes (AChR autoantibodies are IgG1/3; MuSK autoantibodies are IgG4)
- IgM, IgA and albumin levels not affected (data not shown)

Right:

Lasting IgG Reduction

Potent IgG reduction across isotypes

Data sources:
- argenx data
Clinically Meaningful and Long-lasting Reduction of Efficacy Scores
QMG and MG-ADL scores

- Clinically meaningful and statistically significant improvement reached in small patient population (N=24)
- Consistency between QMG and MG-ADL scores
Robust Clinical Improvement Over Placebo Group

- Efgartigimod vs. placebo: increasing differentiation observed with increasing MG-ADL/QMG thresholds

* Missing data point of 1 patient
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
75% of treated patients achieved lasting response

- 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
Conclusions Ph2 Study of Efgartigimod in Patients with gMG

- Consistent and compelling safety & tolerability
- Fast, long-lasting and sustained benefit; clinically meaningful and statistically significant
- Strong correlation between IgG level reduction and disease improvement; validating focus on IgG-mediated diseases
- Significant reduction of AChR autoantibodies
- Phase 2 execution accelerates efgartigimod towards Phase 3
FDA End-of-Phase 2 Meeting

• Clear guidance from FDA following EoP2 meeting

• Identification of key elements of Phase 3 trial:
  • One study, one dose (10 mg/kg)
  • Placebo-controlled 26-week trial
  • Approximately 150 patients
  • Inclusion of both AChR positive and negative gMG patients

• Start of Phase 3: before end of the year
Immune Thrombocytopenia (ITP) Overview

What is Immune Thrombocytopenia?

- Rare bleeding disease
  - estimated 65,000 (1) patients in US
  - ~80% diagnosed with primary ITP
- Symptoms range from mild bruising to severe bleeding
- Symptoms include: mild bruising to severe bleeding, fatigue, fear of bleeding, impact on work and social activities, depression

Limited current treatment options with side effects

- Multiple iterations on corticosteroids & IVIg
- Immunomodulatory agents
- TPO mimetics & splenectomy
- Romiplostim and Eltrombopag, last-line therapies for ITP and have generated global revenues of $584 million(2) and $635 million(3) in 2016

Autoantibodies (IgG type):
- Enhance platelet clearance
- Kill platelets
- Reduce platelet production
- Inhibit platelet function

References:
(1) Wall street research
(2) Amgen Inc. 2016, Form 10-K.
(3) Novartis Annual Report 2016
Autoantibody Levels (IgGs) Correlate With ITP Disease Score

Autoantibodies inhibit platelet production and accelerate platelet destruction.

Platelet levels during IVIg treatment in Adult Immune Thrombocytopenia

- Low bleeding risk
- High bleeding risk

Therapy aimed at reducing autoantibodies like IVIg (shown), plasmapheresis and immunoabsorption results in platelet increase.

- ▼ = IVIg treatment
- ● = Autoantibody level
- = Platelet counts

Efgartigimod Targets all Pathogenic AutoAb Actions Simultaneously
Potential to eliminate therapeutic cycling based on trial-and-error

1. Accelerate platelet clearance
   - Platelets → Macrophage
   - FcγR
   - Efgartigimod

2. Inhibit platelet production
   - Platelets → Megakaryocyte
   - Efgartigimod

3. Induce platelet killing
   - Platelets
   - Efgartigimod

4. Interfere with platelet function
   - Platelets
   - Fibrinogen receptor
   - vWF receptor
   - Collagen receptor
   - Efgartigimod

Autoantibodies

IVIg, Tavalisse, Splenectomy

TPO-RA
Immune Thrombocytopenia Phase 2 Amended Trial Design

<table>
<thead>
<tr>
<th>Screening/Randomization</th>
<th>Treatment Phase</th>
<th>Follow-up Phase</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Key inclusion criteria</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ITP patients with platelet levels $&lt; 30 \times 10^9/L$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- On a stable dose of their SoC treatment prior to randomization</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SoC + ARGX-113 (10mg/kg)</th>
<th>N=12</th>
</tr>
</thead>
<tbody>
<tr>
<td>SoC + ARGX-113 (5mg/kg)</td>
<td>N=12</td>
</tr>
<tr>
<td>SoC + Placebo</td>
<td>N=12</td>
</tr>
</tbody>
</table>

- 4 doses; N= up to 36
- Open label (re)treatment arm of 1 year (all patients) @ 10 mg/kg

**Primary endpoint**
- Safety & Tolerability

**Secondary endpoints**
- Efficacy (platelet counts, rescue therapy and bleeding)
- PK
- PD total IgG; pathogenic IgG
- Immuno-genicity

<table>
<thead>
<tr>
<th>≤2 weeks</th>
<th>3 weeks</th>
<th>21 weeks</th>
</tr>
</thead>
</table>

26
Pemphigus Vulgaris: Overview

What is Pemphigus Vulgaris?

- Chronic, severe auto-immune disease
- 30,000 – 40,000 pemphigus patients (US)\(^1\)
- Mucosal and skin blisters
- Disease severity directly correlates to pathogenic IgG levels against desmoglein-1 (skin) and desmoglein-3 (mucosal)\(^2\)
- Remission and relapse for extended periods

Limited current treatment options with side effects

- Corticosteroids and chronic immunosuppression
- Rituximab, IVIg, immunoabsorption and plasma exchange used for severe or refractory patients (10%), but not curative
- Rituximab therapy shows slow onset of action, risk of developing serious adverse events and significant relapse rate \(^2\) \(^3\) \(^4\)

Diagnosis based on presence of pathogenic autoantibodies targeting desmoglein-1 and -3 in the skin

---

\(^1\) IPPF \(^2\) Kasperkiewicz et al. 2017, Nature Reviews \(^3\) Colliou et al. 2013, Autoimmunity \(^4\) Joly et al. 2017, Lancet
Pemphigus Vulgaris Phase 2 Interim 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 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

**Treatment Phase**

**Induction**
3 weeks

- **COHORT 1:** N= 4
  - ARGX-113 (10 mg/kg)
  - 4 infusions (weekly)

- **COHORT 2 & 3:** N= 4 + 4
  - ARGX-113 (10 mg/kg)
  - 4 infusions (weekly)

**Maintenance**
6 weeks → 8 weeks

- **ARGX-113 (10 mg/kg)**
  - 2 infusions (w2, w6)
  - 4 infusions (w2, w4, w6, w8)

**Follow-up Phase**
8 weeks

- **ARGX-113 (10 mg/kg)**

**Points:**
- **Cohort 1:** 10 mg/kg, induction = 4 infusions (3 weeks), maintenance = 2 infusions (6 weeks)
- **Additional cohorts:**
  - Same dose (10 mg/kg)
  - 2 additional administrations during maintenance phase
  - Extend maintenance duration to 8 weeks
Clinicaltrials.gov: NCT03334084, argenx data

Phase 1 Healthy Volunteer Subcutaneous Formulation
Open Label Trial Design

**Screening/Randomization**

**Inclusion criteria**
- Healthy male subjects
- 18-55 years old
- Body weight: 50 – 100 kg

**Treatment Phase & Follow-up Phase**

- **Single IV dose (10 mg/kg)**
  - N= 8
  - D1
  - D57

- **Single SC dose (10 mg/kg)**
  - N= 8
  - D1
  - D57

- **2X IV dose (20 mg/kg) + 8X SC dose (300 mg)**
  - N= 8 (50-70 kg) + 8 (80-100 kg)
  - D1
  - D4
  - D8
  - D57
  - Weekly subQ dosing

- **3 weeks**
- **8 weeks**
- **8 weeks**

**Read out**

- **Safety & Tolerability**
- **PK**
- **PD**
  - Total IgG; IgG subtypes; IgA & IgM
- **Immunogenicity**
Efgartigimod: Feasibility of Subcutaneous Dosing
Exploring SC formulations for larger patient populations (chronic, ex-hospital)

- Viability of SC formulation demonstrated:
  - Comparable half-life to IV
  - Comparable IgG reduction to IV; steady state 50% IgG reduction achieved by weekly dosing (300 mg fixed dose)
  - Favorable bio-availability (~ 50%)
  - Favorable viscosity and stability profile

**Comparable IgG reduction**

![Graph showing comparable IgG reduction](image)

**Steady state IgG reduction (~50%)**

![Graph showing steady state IgG reduction](image)
ARGX-110: Phase 1 / 2
Mono & Combo Therapy
ARGX-110: Lead Cancer Program Based On Novel Target CD70

Three distinct modes of action to target CD70+ tumor cells

1. Block tumor growth signal
2. Restore immune surveillance
3. Kill tumor

- ARGX-110 is a SIMPLE Antibody™, equipped with POTELLIGENT® Fc engineering technology
- ARGX-110 targets CD70 to block CD27 interaction, kill CD70 expressing cells and restore immune surveillance
- Soluble CD27 is a biomarker
- Phase 1: encouraging safety & tolerability profile and promising preliminary signs of efficacy in CTCL
- Focus on two rare & aggressive hematological tumors: CTCL and newly diagnosed AML / high-risk MDS
  - Interim results from dose escalation part of Phase 1/2 AML/MDS trial expected YE:2017
  - Interim POC data from Phase 2 CTCL trial expected YE:2017

Silence et al. 2014, mAbs.
Acute Myeloid Leukemia (AML) Overview

What is Acute Myeloid Leukemia?

- Rare hematologic cancer characterized by excessive proliferation of myeloid stem cells and their failure to properly differentiate into mature white blood cells
- AML progresses very rapidly and is fatal if left untreated
- ~22,000\(^{(1)}\) new cases per year in the U.S.
- Disease of the elderly — 60% of diagnosed patients are older than 60yr

Limited current treatment options

- Elderly, frail patients unfit for high dose chemotherapy — palliative treatment with hypomethylating agents
  - Median survival of 7 – 10 months
  - ~6\(^{(2)}\) five year survival rate for patients over 65
- First-line treatments for patients <45yr: aggressive chemotherapy followed by stem cell transplant
  - 5-year survival is ~57\(^{(2)}\) for patients under 45

CD70 Provides Unifying Rationale Across Risk & Age Classes In AML
Potential to selectively target leukemic stem cells in AML patients

- Elevated sCD27 serum levels in all newly diagnosed AML patients, regardless of risk or age categories
- sCD27 levels are an independent negative prognostic marker in all newly diagnosed AML patients
- CD70 expressed on ~86-100% of AML blasts, majority of malignant cells are CD70/CD27 double-positive
- CD70/CD27 selectively overexpressed on leukemic stem cells (LSCs), not on hematopoietic stem cells (HSCs)

Legends: adv., adverse; CI, confidence interval; fav., favorable; int., intermediate; OS, overall survival. Statistics: left: one-way ANOVA; middle: log-rank test. *, P < 0.05; **, P < 0.01; ***, P < 0.001.

ARGX-110: Inhibits LSC Proliferation In Lasting Fashion

Long-term effects *ex vivo*

- Reduces LSC colony formation across patient risk categories (favorable/intermediate/adverse risk)
- Reduces LSC numbers as determined in serial re-plating experiments
- Blocking CD70 results in: (1) lasting down-regulation of stem cell genes (2) increasing myeloid differentiation
ARGX-110: Curative Potential Of Monotherapy In Mouse Model

Shown to reduce LSCs, increasing survival in AML model

- Increased survival after secondary transplantation of AML BM cells from primary recipients transiently treated with ARGX-110 variant
- Increased survival observed for AML blasts taken from all 3 AML risk categories (fav/int/adv)

HMA + ARGX-110 Synergistically Eradicate AML LSC

HMA-treated patients, *Ex vivo* colony formation assay
*(primary AML LSC)*

In *vivo* PDX model
*(primary AML LSC)*


- HMA upregulate CD70 protein and mRNA on AML LSC *ex vivo* (~4-fold) and *in vivo* (~5-7-fold) – NOT on HSC
- *Ex vivo*: HMA/αCD70 synergistically reduce colony formation; effect maintained upon serial replating, transient treatment
- *In vivo*: Transient treatment by HMA/αCD70 eradicate human LSCs in therapeutic model; efficacy confirmed in CFU assay

Hinterbrander ASH 2017: Blocking CD70/CD27 signaling in combination with hypomethylating agents eradicates human CD34+ AML stem and progenitor cells; manuscript in preparation
ARGX-110 & Azacitidine For AML/MDS: Phase 1 / 2 Combo

Phase 1 – Dose Escalation

Safety and tolerability

- 20 mg/kg
  - N = 3+3

- 10 mg/kg
  - N = 3+3

- 3 mg/kg
  - N = 3+3

- 1 mg/kg
  - N = 3+3

Vidaza = 75 mg/m^2 (standard of care)

N = up to 24

Phase 2 – Proof of Concept

- 10 mg/kg
  - N = 21

Vidaza = 75 mg/m^2 (standard of care)

- We are here

- 20 mg/kg
  - N = 3+3

- 10 mg/kg
  - N = 3+3

- 3 mg/kg
  - N = 3+3

- 1 mg/kg
  - N = 3+3

N = 3+3

Vidaza = 75 mg/m^2 (standard of care)

N = up to 24

- Hypomethylation agents such as Azacitidine increase CD70 expression\(^1\)
- Population: untreated AML & high risk of myelodysplastic syndrome (MDS)*, eligible for AZA
- Design: open-label, non-controlled, non-randomized

*Some Myelodysplastic Syndrome (MDS) patients are at high risk of developing AML; MDS affects bone marrow cells, reducing their ability to produce red & white blood cells

(1) Zhou et al. 2011, Lupus.
Non-Transplantable Patients With Intermediate & Adverse Risk and High Blast Count in Bone Marrow

9 newly diagnosed AML patients

<table>
<thead>
<tr>
<th>Baseline characteristics (N=9)</th>
<th>ARGX-110 + Azacitidine</th>
<th></th>
<th></th>
<th></th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mg/kg</td>
<td>3 mg/kg</td>
<td>10 mg/kg</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>71</td>
<td>75</td>
<td>71</td>
<td>72</td>
<td></td>
</tr>
<tr>
<td>71-80</td>
<td></td>
<td>71-84</td>
<td>64-75</td>
<td>64-84</td>
<td></td>
</tr>
<tr>
<td>Gender: Male/Female</td>
<td>2/1</td>
<td>1/2</td>
<td>2/1</td>
<td>5/4</td>
<td></td>
</tr>
<tr>
<td>Risk (ELN 2017)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intermediate</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Adverse</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>4</td>
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<tr>
<td>Blasts in the bone marrow</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Median %</td>
<td>51.3</td>
<td>40</td>
<td>70</td>
<td>53.6</td>
<td></td>
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<tr>
<td>24-90</td>
<td></td>
<td>20-60</td>
<td>50-80</td>
<td>20-90</td>
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<tr>
<td>AML classification (WHO 2016)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not other specified</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>With Myelodysplasia- related changes</td>
<td>2</td>
<td></td>
<td></td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Therapy-related myeloid neoplasm</td>
<td>1</td>
<td></td>
<td></td>
<td>1</td>
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<tr>
<td>French-American-British subtypes</td>
<td>M4,M1,M2</td>
<td>M4,M5,M2</td>
<td>M1,M2,M5a</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

Source: argenx data – patient anecdotes – uncleaned data

ELN: European Leukemia Net, Dohner et al. 2017, Blood
Response in 6/6 Evaluable Newly Diagnosed AML Patients
ARGX-110/Aza treatment

<table>
<thead>
<tr>
<th>Risk</th>
<th>Best response</th>
<th>Treatment duration (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adv 1 (1 mg/kg)</td>
<td>CR</td>
<td>6</td>
</tr>
<tr>
<td>Int</td>
<td>CR</td>
<td>5</td>
</tr>
<tr>
<td>Adv</td>
<td>CR</td>
<td>Not yet evaluable</td>
</tr>
<tr>
<td>Int</td>
<td>CR</td>
<td>1</td>
</tr>
<tr>
<td>Int</td>
<td>PR</td>
<td>4</td>
</tr>
<tr>
<td>Adv</td>
<td>PR</td>
<td>9</td>
</tr>
</tbody>
</table>

- So far, all patients responded (3 CR, 1 CRi, 2 PR)
- 1 patient reached CR and bridged to allogeneic stem cell transplant after 5 cycles
- 6/9 patients were still on treatment

### Case 1: Patient Cohort 1 – 1 mg/kg – 5 Cycles on Study

- **75 year old male**
- AML with myelodysplasia-related changes, M1/M2; BM ~40% blasts
- Molecular genetics: U2AF1mut; DNMT3Amut; cytogenetics: normal

#### Treatment Duration (months)

<table>
<thead>
<tr>
<th>Risk</th>
<th>Best response</th>
<th>Treatment Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adv</td>
<td>CRi</td>
<td>Cycle 5 Day 17</td>
</tr>
<tr>
<td>Int</td>
<td>CR</td>
<td></td>
</tr>
<tr>
<td>Adv</td>
<td>CR</td>
<td></td>
</tr>
<tr>
<td>Int</td>
<td>CR</td>
<td></td>
</tr>
<tr>
<td>Int</td>
<td>PR</td>
<td></td>
</tr>
<tr>
<td>Adv</td>
<td>PR</td>
<td></td>
</tr>
<tr>
<td>Int</td>
<td></td>
<td>Not yet evaluable</td>
</tr>
<tr>
<td>Adv</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Cut off: 15 Nov 2017

**Source:** argenx data – patient anecdotes – uncleaned data
Case 1: ARGX-110/Aza Induces Complete Remission & Bridges to Transplant

Bone marrow: % Blasts, flow cytometry

Blood analysis: Absolute counts (G/L)

Source: argenx data – patient anecdotes – uncleaned data
Case 1: ARGX-110/Aza combo reduces AML stemness

ARGX-110 monotherapy reduces LSCs outgrowth

White light microscopy (5,000 cells)

ARGX-110: Cycle 1 Day 1

ARGX-110/Aza increases asymmetric LSC division

ARGX-110/Aza reduces experimental LSC gene signature

- Significantly reduced leukemic stem cell colony formation
- Increased myeloid differentiation (asymmetric division) of leukemic stem cells
- Reduction of LSC gene signature

‘It seems ARGX-110 targets mature blasts as well as LSCs – this is very promising’
(AML KOL)

Source: argenx data – patient anecdotes – uncleaned data
ARGX-110 In Newly Diagnosed AML Patients – Summary

Preliminary data from first 6 patients – additional data needed

Preliminary clinical data confirm preclinical observations

Promising preliminary activity obtained in first set of patients
  • 6/6 responders
  • 1 patient bridged to transplantation

Encouraging safety and tolerability profile
  • No exacerbation of azacitidine toxicity

Highly differentiated drug profile
  • CD70 uniformly & selectively expressed
  • Driving LSCs into myeloid differentiation

‘In an ideal world, a LSC targeting drug should show response regardless of risk category, should show a better response in de-novo vs R/R patients and should allow for deep and durable responses. ARGX-110 may meet these criteria’ (AML KOL)

Source: argenx data – patient anecdotes – uncleaned data
Business development & financials
AbbVie Partnership for Novel Target GARP

Strategic Antibody Collaboration Details

- GARP is a protein specifically present on the surface of activated regulatory T-cells (Tregs)
- AbbVie has option to:
  - Obtain exclusive, worldwide license to develop and commercialize ARGX-115
  - Fund further GARP-related research by argenx beyond ARGX-115
- argenx conducts and funds R&D through IND-enabling studies
- argenx can study ARGX-115 in combo with its pipeline programs

Financial Highlights

- $40mm upfront payment
- Received two of two $10mm preclinical milestones
- $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
## Additional Strategic Collaborations

<table>
<thead>
<tr>
<th>Partner</th>
<th>Asset</th>
<th>Key commentary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genor Biopharma</td>
<td>ARGX-109 (Gerilimzumab)</td>
<td>• Development for Chinese market</td>
</tr>
<tr>
<td>LEO</td>
<td>ARGX-112</td>
<td>• Focused on <em>inflammation-based dermatological indications</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• LEO Pharma funds &gt;50% of all development costs up to CTA approval and all development post-approval of first Phase 1 trial in Europe</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• argenx is eligible for ~€100mm in aggregate milestone payments + tiered royalties</td>
</tr>
<tr>
<td>STATEN BIOTECHNOLOGY</td>
<td>ARGX-116</td>
<td>• Focused on developing an anti-ApoC3 antibody for <em>dyslipidemia</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Jointly responsible for conducting dyslipidemia research — Staten responsible for additional clinical development</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• argenx eligible for royalties in the low twenties</td>
</tr>
<tr>
<td>Shire</td>
<td>Discovery Programs</td>
<td>• Focused on <em>novel rare disease targets</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Provides Shire access to SIMPLE Antibody™ platform + Fc engineering technologies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• argenx has received $12.6 mm in aggregate upfront and milestone payments and R&amp;D fees over the course of the collaboration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Shire purchased €12mm of argenx ordinary shares through participation in July 2014 IPO</td>
</tr>
</tbody>
</table>
Financial Strength
NASDAQ IPO & follow-on financing in 2017

<table>
<thead>
<tr>
<th>EVENT</th>
<th>DATE</th>
<th>GROSS PROCEEDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euronext – Initial Public Offering</td>
<td>July 2014</td>
<td>€42mm</td>
</tr>
<tr>
<td>PIPE</td>
<td>June 2016</td>
<td>€30mm</td>
</tr>
<tr>
<td>Nasdaq – Initial Public Offering</td>
<td>May 2017</td>
<td>$115mm (€102mm)</td>
</tr>
<tr>
<td>Follow-on</td>
<td>December 2017</td>
<td>$266mm (€226mm)</td>
</tr>
</tbody>
</table>
Financial Profile and Investor Composition

Shareholder base > 70% US investors

Additional Key Statistics – Six months ended June 30, 2018

- **Cash position**: €339mm
- **Capital raised since inception**: €475mm (ex. grants)
  - 2017: raised $115mm (€102mm) in NASDAQ IPO
  - 2017: raised $266mm (€226mm) in public offering
- **Non-dilutive funding since inception**: €104mm (incl. grants)
  - 2018: $10mm second preclinical milestone AbbVie
- **108 employees & consultants** — 80 R&D, 28 SG&A

Blue-Chip Investor Base – June 30, 2018

- **US shareholding expanded above 70%**

![Pie chart](chart.png)
### Key Upcoming Expected Milestones & Communications

<table>
<thead>
<tr>
<th>2018</th>
<th>Q1</th>
<th>Q2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ARGX-113</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>ARGX-110</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>Partnerships</strong></td>
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<td></td>
</tr>
<tr>
<td><strong>Partnerships</strong></td>
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</tr>
</tbody>
</table>

#### Q1 Milestones
- **MG Ph2 Full data** *(AAN, April 24, LA)*
- **SubQ Ph1 HV Full data**
- **ARGX-117 Novel complement target**
- **ARGX-112 LEO Pharma CTA milestone**
- **ARGX-115: AbbVie second preclinical milestone ($10mm)**

#### Q2 Milestones
- **PV Ph2 Interim data**
- **AML Ph2 Launch**

#### Q3 Milestones
- **ITP Ph2 Topline data**
- **Shire milestone: Exclusive option**
- **Potential milestone(s)**

#### Q4 Milestones
- **ITP Ph 2 Full data (ASH)**
- **MG Ph 3 Launch**
- **AML Ph1/2 Full data (ASH)**
- **CTCL Ph2 Full data (ASH)**
Appendix
Company Leadership

Management

Tim Van Hauwermeiren
Chief Executive Officer

Eric Castaldi
Chief Financial Officer

Hans de Haard, Ph.D.
Chief Scientific Officer

Torsten Dreier, Ph.D.
Chief Development Officer

Keith Woods
Chief Operating Officer

Nicolas Leupin, M.D.
Chief Medical Officer

Debbie Allen, Ph.D.
SVP, Business Development

Dirk Beeusaert
General Counsel

Scientific Advisors

James Howard
ARGX-113 MG

Adrian Newland
Barts Health
ARGX-113 ITP

Jan Verschuuren
ARGX-113 MG

Sally Ward
FcRn Biology

Sophie Lucas
GARP Biology

Scientific Advisors

David Kuter
ARGX-113 ITP

Martine Bagot
ARGX-110 CTCL

Adrian Ochsenbein
CD70 Biology

Don deBethizy
TARGACEPT

Pamela Klein
Genentech

Tony Rosenberg
Novartis

James Daly
AMGEN

Peter Verhaeghe
Chairman

Werner Lanthaler
evotec

Audit Com

R&D Com

Remuneration Com

Board of Directors

Tim Van Hauwermeiren

David Lacey
AMGEN

Don deBethizy
TARGACEPT

Pamela Klein
Genentech

James Daly
AMGEN

David Kuter
HARVARD MEDICAL SCHOOL

James Howard
UNC

Adrian Newland
Barts Health

Jan Verschuuren
ULM

Sally Ward
FcRn Biology

Sophie Lucas
GARP Biology

Scientific Advisors

MT

ARGX-113 MG
Thank you!