Disclaimers and Forward-looking statements

This presentation contains certain "forward-looking statements" within the meaning of applicable securities laws. Other than statements of historical facts, all statements included in this presentation are forward-looking statements, including statements about our plans, our plans for our current and future product candidates and programs, our plans for current and future clinical trials, including a Phase 2 trial for APG777 in asthma, a Phase 1b trial of APG808 in asthma, a Phase 1 trial for APG990, and a Phase 1 trial for APG333; our plans for clinical trial design; the anticipated timing of the initiation of and results from our clinical trials, including data from our Phase 2 trial of APG777 and our Phase 1 trial of APG808; the potential clinical benefit and half-life of APG777, APG808, APG990, APG333 and any other potential programs, including combination therapies; our expected timing for future pipeline updates; our expectations regarding the time period over which our capital resources will be sufficient to fund our anticipated operations, and estimates of market size. In some cases, you can identify forward-looking statements by terms such as "anticipate," "believe," "can," "could," "design," "estimate," "expect," "intend," "likely," "may," "might," "plan," "potential," "predict," "suggest," "target," "will," "would," or the negative of these terms, and similar expressions intended to identify forward-looking statements. The forward-looking statements are based on our beliefs, assumptions and expectations of future performance, taking into account the information currently available to us. These statements are only predictions based upon our current expectations and projections about future events. Forward-looking statements are subject to known and unknown risks, uncertainties and other factors that may cause our actual results, level of activity, performance or achievements to be materially different from those expressed or implied by such forward-looking statements, including those risks described in "Risk Factors" and "Management's Discussion and Analysis of Financial Condition and Results of Operations" in our Annual Report on Form 10-K for the year ended December 31, 2023, filed with the SEC on March 5, 2024, our Quarterly Report on Form 10-Q for the quarter ended March 31, 2024, filed with the SEC on May 13, 2024, and subsequent disclosure documents we may file with the U.S. Securities and Exchange Commission. Although we have attempted to identify important factors that could cause actual results to differ materially from those contained in forward-looking statements, there may be other factors that cause results not to be as anticipated, estimated or intended.

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Apogee plans to reshape the current standard of care for I&I diseases with its potential therapies

<table>
<thead>
<tr>
<th>Best-in-class monotherapy in atopic dermatitis</th>
<th>First-in-class combination therapy in atopic dermatitis</th>
<th>Best-in-class combination therapies in asthma and COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Less frequent dosing with potential for <em>increased efficacy</em> through higher exposures</td>
<td>• <em>Rational combination</em> targeting orthogonal mechanisms with potential for <em>best-in-class efficacy and dosing</em></td>
<td>• <em>Strategic optionality</em> to combine orthogonal validated mechanisms across pipeline</td>
</tr>
</tbody>
</table>

Broad potential in I&I with 10+ possible expansion indications
Apogee’s approach is to achieve differentiated efficacy and dosing in the markets it is pursuing

<table>
<thead>
<tr>
<th>Strategy</th>
<th>Program</th>
<th>Discovery</th>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potential best-in-class monotherapy in AD</td>
<td>APG777 IL-13</td>
<td>Atopic Dermatitis</td>
<td>2H 2025: Phase 2 16-week induction PoC data</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Potential best-in-class mAbs for combinations</td>
<td>APG808 IL-4Rα</td>
<td>Healthy Volunteers</td>
<td>Q4 2024: Initial Phase 1 PK and safety in HVs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>APG990 OX40L</td>
<td>Healthy Volunteers</td>
<td>2025: Initial Phase 1 PK &amp; safety in HVs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>APG333 TSLP</td>
<td></td>
<td>2024: DC nomination</td>
<td>2025: Initiate Phase 1 PK &amp; safety in HVs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Potential first- or best-in-class combination approaches</td>
<td>APG777 ± APG990 IL-13 ± OX40L</td>
<td>Atopic Dermatitis</td>
<td>2025: Clinical trial initiation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>APG777 ± APG333 IL-13 ± TSLP</td>
<td>Asthma</td>
<td>TBD: Clinical trial initiation¹</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Additional combination(s) IL-13/IL-4Rα + OX40L/TSLP</td>
<td>COPD</td>
<td>Q4 2024: Additional combination(s) to be announced at R&amp;D Day</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Apogee agents mentioned above are currently under investigation. Their safety and effectiveness for the listed target indications have not yet been established. (1) Pending final data from our Phase 1 trials of APG777 and APG333 in healthy participants, we may initiate a Phase 2 trial in asthma and expect to further evaluate opportunities to develop APG777 and the APG777+APG333 combination for other I&I indications, including atopicus areata, chronic rhinosinusitis with nasal polyps, chronic spontaneous urticaria, eosinophilic esophagitis and prurigo nodularis.
Apogee mAbs are engineered for best-in-class properties, including half-life extension

Based on clinically-validated epitopes with performance across five properties:

- **Backbone**
- **Potency**
- **PK**
- **Stability**
- **Viscosity**

**Potential for PK that:**
- Optimizes exposures
- Decreases variability
- Increases half-life

- Designed to maximize antibody recycling
- Drug exists at higher levels for longer effect
Apogee is pursuing the largest I&I markets with a de-risked development approach; AD is the largest

Estimated population size (in millions)
Moderate or severe, WW

US biologics penetration:

- AD, asthma and COPD are the largest and least penetrated markets
- Mature I&I markets have consistently achieved high biologics penetration (~30-70% after 15-20 years), suggesting potential for significant growth in AD (8%), asthma (~20%) and COPD (0%)

### Apogee’s current indications

- **COPD**: 59.6M
- **Asthma**: 65.5M
- **AD**: 81.6M

NOTE: IBD = Inflammatory bowel disease; RA = Rheumatoid arthritis; PsO = Psoriasis; COPD = Chronic obstructive pulmonary disease; AD = Atopic dermatitis

SOURCE: Academic journals, disease foundations, WHO, CDC, census data, analyst research
Potential best-in-class monotherapy in atopic dermatitis
In psoriasis, an analog to AD, Skyrizi has taken the lead with quarterly dosing

**Biologics with estimated WW peak sales in psoriasis and approval year**

- In 2024, 6 biologics each projected to exceed $2B in PsO alone
- With quarterly dosing, Skyrizi leads a crowded market with 35% share
- Patients stay on Skyrizi longer and are less likely to switch

**Efficacy (PASI-75, %)**

- Most pts respond (83-89% PASI-75)

**Dosing Interval (weeks)**

**NOTE:** Year denotes US launch year for adults with moderate to severe plaque psoriasis. Efficacy data are derived from different clinical trials at different points in time, with differences in trial design, patient populations, and statistical analysis. As a result, cross-trial comparisons cannot be made. No head-to-head trials have been conducted among all biologics shown. Assumes 1 EUR = 1.07 USD.

1Real-world evidence shows Skyrizi patients experienced fewer drug changes and a higher probability of drug survival compared with those treated with other biologic therapies for PsO and PsA.


**Erik L et al ACR Convergence 2023. PsO = Psoriasis. PsA = Psoriatic Arthritis.**
There is significant whitespace in the landscape of approved and in-development biologics for AD.

NOTE: Only DUPIXENT and ADBRY are approved in the US. SOURCE: 1. Lebrikizumab 250mg Q2W Ph3 avg. Silverberg JI et al. AAD 2022 2. Dupilumab 300 mg Q2W mono Ph3 avg. DUPIXENT USP I 3. Tralokinumab 300 mg Q2W mono Ph3 avg. Adbry USPI 4. CBP-201 300 mg Q2W Ph2. Connect Biopharma Press Release Jan. 5, 2022 5. Nemolizumab 30 mg Q4W Ph3 avg. Silverberg J et al EADV 2023 6. Rocatinlimab 150mg Q4W Ph2b Guttman-Yassky E et al Lancet 2023 7. Amlitelimab 250mg Q4W Ph2b Weidinger S et al EADV oral presentation 2023. Efficacy data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.
Apogee is pursuing potentially best-in-class monotherapy and first-in-class combination in AD

NOTE: *Positioning of Apogee programs is illustrative and based on interim Phase 1 results for APG777 only and illustrates what we believe we can potentially achieve. Only DUPIXENT and ADBRY are approved.

SOURCE: 1. Lebrikizumab 250mg Q2W Ph3 avg. Silverberg JI et al. AAD 2022 2. Dupilumab 300 mg Q2W mono Ph3 avg. DUPLEXUS USPI 3. Tralokinumab 300 mg Q2W Ph3 mono. Adbry USPI 4. CBP-201 300 mg Q2W Ph2. Connect Biopharma Press Release Jan. 5, 2022 5. Nemolizumab 30 mg Q4W Ph3 avg. Silverberg J et al EADV 2023 6. Rocatinlimab 150mg Q4W Ph2b Gutman-Yaissy E et al Lancet 2023 7. Amlitelimab 250mg Q4W Ph2b Weidinger S et al EADV oral presentation 2023. Efficacy data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.
APG777 leverages lebrikizumab's mechanism to deliver a potentially best-in-class, pipeline-in-a-product antibody.

APG777 disrupts Type 2 inflammation by preventing formation of IL-13Rα1 / IL-4Rα heterodimer.

APG777's epitope on IL-13 overlaps with lebrikizumab’s and leverages proven MoA and biology.

APG777 is as potent as lebrikizumab and DUPIXENT in key preclinical assays.

**NOTE:** MoA = Mechanism of Action.
APG777 exhibited a potentially best-in-class PK profile with a half-life of ~75 days

**Single-dose concentration-time profile**

- Slow clearance, resulting in half-life of ~75 days
- Dose-proportional increases in both Cmax and AUC

**Multi-dose concentration-time profile**

- Initial multiple-dose data consistent with PK profile from SAD cohorts

PK demonstrated dose-proportionality and half-life of ~75 days (approximately 3x lebrikizumab)
pSTAT6 and TARC are biomarkers of IL-13 target engagement and AD severity

1. pSTAT6 is a proximal and sensitive marker of IL-13 receptor activation

2. TARC is historically correlated with AD severity and initial treatment response

Taken together, APG777’s reduction of these biomarkers confirms inhibition of IL-13 signaling and allows comparison to other agents

APG777 Phase 1 biomarkers
Single dose APG777 showed near complete pSTAT6 inhibition for ~3 months (limit of available follow-up)

Median percent pSTAT6

- 300 mg (N=1)
- 600 mg (N=6)
- 1200 mg (N=6)
- Placebo (N=4)

Sustained APG777 pSTAT6 inhibition supports ability to suppress IL-13 signaling at potential maintenance doses of Q3M+
Single dose of APG777 led to deep and sustained TARC inhibition for ~3 months (limit of available follow-up)

Median % changes from baseline in TARC inhibition

APG777 showed similar maximal inhibition of TARC compared to DUPIXENT but improved durability

NOTE: These data are derived from different clinical trials conducted at different points in time, with differences in trial design, dosing regimen and patient populations. DUPIXENT data derived from a Phase 1 trial with 6 healthy volunteers receiving a single SC injection of 300 mg DUPIXENT. APG777 data derived from our Phase 1 trial in 6 healthy volunteers receiving a single SC injection of 300 mg of APG777. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted. APG777 is an investigational drug and has not been approved by the FDA as safe and effective. SOURCE: Li, Z, et al. ACCP, 2020. Data for time points on nominal day post dose 1, 29, 85. No data has been published showing lebrikizumab impact on TARC in HVs.
APG777 Phase 2 regimen is designed to achieve two goals

**Induction**
- Lebrikizumab data suggests an exposure-response for efficacy in induction

**Goal:** *Exceed* lebrikizumab exposures

**Maintenance**
- There was no exposure-response observed in maintenance for lebrikizumab

**Goal:** *Equal* lebrikizumab exposures
### Efficacy of biologics in AD (Week 16, placebo-adjusted)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Lebrikizumab Ph3, all patients (N = 851)</th>
<th>Lebrikizumab Ph3, &lt;60 kg subgroup (N = 180)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DUPIXENT Ph3</td>
<td>34% EASI-75</td>
<td>26% EASI-75</td>
</tr>
<tr>
<td></td>
<td>38% EASI-90</td>
<td>28% EASI-90</td>
</tr>
<tr>
<td></td>
<td>38% IGA 0/1</td>
<td>26% IGA 0/1</td>
</tr>
</tbody>
</table>

- **Exposure-response** in induction demonstrated by lebrikizumab
- **~30% higher exposures** in lebrikizumab low bodyweight subgroup led to **improved efficacy across endpoints**

**NOTE:** In lebrikizumab Ph2b and Ph3 there has been no dose-AE or exposure-AE relationship. Lebrikizumab exposures and efficacy are for the Phase 3 dose (500 mg Loading Dose at Weeks 0 and 2, 250 mg Q2W Weeks 4 to 16). Efficacy data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

**SOURCE:** Lebrikizumab European Public Assessment Report. DUPIXENT® USPI.
Lebrikizumab has demonstrated superior maintenance compared to DUPIXENT

Maintenance of response in AD (Week 52)

<table>
<thead>
<tr>
<th></th>
<th>DUPIXENT(^1)</th>
<th>Lebrikizumab(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EASI-75 Responders, %</td>
<td>72</td>
<td>82</td>
</tr>
<tr>
<td>IGA 0/1 Responders, %</td>
<td>54</td>
<td>77</td>
</tr>
</tbody>
</table>

- No dose-response or exposure-response in maintenance was observed for lebrikizumab
- Lebrikizumab has shown superior maintenance responses compared to DUPIXENT
- Real-world data for DUPIXENT shows poor compliance; ~50% of patients discontinue before two years\(^3\)

APG777 maintenance regimens are designed to equal lebrikizumab exposures with only 2-4 injections per year (vs. 13-26 injections per year)

NOTE: The labeled dose of lebrikizumab is Q4W in maintenance. Efficacy data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

APG777 Phase 2 exposures are designed to exceed lebrikizumab in induction and equal in maintenance

Modeled concentration of APG777 in induction and maintenance (Q3M) vs lebrikizumab

**Induction goal:**
*Exceed* lebri exposures by ~30-40%

6 vs 10-11 injections for lebri/DUPIXENT

**Maintenance goal:**
*Equal* lebri exposures

2-4 vs 13-26 injections for lebrikizumab/DUPIXENT

NOTE: Every 3-month maintenance dosing regimen shown. The above graph is illustrative only with respect to plans for dosing of APG777 and does not present comparative data.
Ongoing integrated Phase 2 trial expected to have 16-week Part A topline data in 2H’25

Part A: Proof-of-concept (N ~110)

- **Induction**
  - Induction regimen
  - Placebo

- **Maintenance**
  - Every 3 mos (Q12W)
  - Every 6 mos (Q24W)

W16 Primary Endpoint

Phase 2 design is >90% powered in both Part A / B and has potential for significant acceleration

Part B: Dose optimization (N ~360)

- **Induction**
  - High Dose
  - Medium Dose
  - Low Dose
  - Placebo

- **Maintenance**
  - Regimen A (TBD)
  - Regimen B (TBD)

W16 Primary Endpoint

**NOTE:** Induction regimen in Part A is two injections (720mg) week 0 and week 2 followed by a single injection (360mg) at week 4 and 12. Number of and doses within induction and maintenance regimens to be tested in Part B are preliminary and will be confirmed based on emerging data from Part A.
Phase 2 16-week Part A induction data in atopic dermatitis is planned to readout in 2H 2025

**OBJECTIVES**

**Safety**
Confirm well tolerated safety profile as seen in Phase 1 HV study and in line with other agents in class (e.g., DUPIXENT, lebrikizumab)

**Efficacy**
- **primary endpoint**
  Primary endpoint of percent change from baseline in EASI at Week 16 in line with standard of care (approx. 65-70% topline)
- **key secondary endpoints**
  Proportion of patients achieving key secondary endpoints at Week 16 (future approvable endpoints) in line with standard of care:
  - EASI-75: approx. 45-50% (topline)
  - IGA 0/1: approx. 35-40% (topline)

**NOTE:**
1 Based on FDA label for DUPIXENT
Strong historical correlation between Ph2 and Ph3 data makes APG777 16-week induction data a key catalyst

Strong correlation between Phase 2 and 3 results in AD for validated endpoint EASI-75

Note: While JAKi efficacy held up from Ph2 to Ph3, FDA applied a boxed warning to the class due to increased risk of CV events and death; patients must step through biologic to get to JAKi

Phase 3 failure in AD is rare

A 2020 review examining failed trials for AD did not find any completed, placebo-controlled Phase 3s that did not meet the primary endpoint6
Potential first-in-class combination therapy in atopic dermatitis
AD is heterogenous – Type 2 is the core pathway with varying involvement of Type 1 and Type 3

OX40L / OX40 interaction drives Type 1, 2, and 3 inflammation in atopic dermatitis

- OX40L is expressed on antigen-presenting cells (APCs)
- OX40L / OX40 interaction promotes inflammatory T cell responses in AD
- T cells produce Type 1, 2, and 3 cytokines that drive inflammation and AD symptoms
- Type 2 (IL-13) is the core pathway in AD; Type 1 and 3 play a secondary role in specific subpopulations

Blocking OX40L / OX40 interaction has the potential to broadly inhibit Type 1, 2, and 3 inflammation
OX40L inhibition is clinically validated in AD and has demonstrated broad cytokine suppression

<table>
<thead>
<tr>
<th>Indication</th>
<th>PoC achieved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atopic dermatitis</td>
<td>PoC achieved</td>
</tr>
<tr>
<td>Asthma</td>
<td>Ph2b data in H2 2024</td>
</tr>
<tr>
<td>Hidradenitis suppurativa</td>
<td>Ph2 data in 2025</td>
</tr>
<tr>
<td>Alopecia areata</td>
<td>Ph2 data in 2025</td>
</tr>
<tr>
<td>Celiac disease</td>
<td>Ph2 start in 2024</td>
</tr>
<tr>
<td>Systemic sclerosis</td>
<td>Ph2 start in 2024</td>
</tr>
</tbody>
</table>

**Amlitelimab (OX40L) Phase 2b AD biomarker data**

<table>
<thead>
<tr>
<th>Type 2</th>
<th>Type 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>IL-13</td>
<td>IL-17A</td>
</tr>
<tr>
<td>-62%</td>
<td>-40%</td>
</tr>
<tr>
<td>IL-22</td>
<td>-63%</td>
</tr>
</tbody>
</table>

Potential additional benefit from deeper inhibition of Type 2 inflammation

**Upcoming amlitelimab (OX40L) POC readouts**

- **Indication where IL-13 / IL-4Rα inhibition also achieved PoC**

**NOTE:** Eligible patient population for each indication is based on Sanofi estimates specific to amlitelimab

**SOURCE:** Sanofi 2023 R&D day presentation
APG990 leverages amlitelimab’s mechanism to deliver a potentially best-in-class, pipeline-in-a-product antibody

APG990 epitope overlaps with amlitelimab to leverage proven MoA

APG990 is as potent as amlitelimab across preclinical assays

APG990 NHP half-life is extended relative to amlitelimab

APG990 Phase 1 initiation planned for Q3 2024
Potential first-in-class APG777+APG990 combo targets all inflammatory types, including full Type 2 inhibition

- APG990 targets upstream OX40L/OX40 interaction
- Potential for Type 1, 2 and 3 inhibition without safety/tolerability issues associated with JAK inhibitors

- APG777 targets downstream IL-13
- APG777 Phase 1 demonstrated near complete inhibition of Type 2 inflammatory biomarker pSTAT6

APG777+APG990 combination enables potentially best-in-class efficacy and dosing (Q3M+).
IL-13 and OX40L are the two orthogonal mechanisms with greatest efficacy in AD

Selected targets for 777+990 combination

<table>
<thead>
<tr>
<th>Target</th>
<th>IL-13&lt;sup&gt;1,2&lt;/sup&gt;</th>
<th>IL-4R&lt;sub&gt;α&lt;/sub&gt; &lt;sup&gt;3&lt;/sup&gt;</th>
<th>OX40&lt;sup&gt;4&lt;/sup&gt;</th>
<th>IL-22&lt;sup&gt;5&lt;/sup&gt;</th>
<th>IL-31&lt;sup&gt;6&lt;/sup&gt;</th>
<th>TSLP&lt;sup&gt;7&lt;/sup&gt;</th>
<th>IL-22&lt;sup&gt;8&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agent</td>
<td>Lebrikizumab (high-exposure)</td>
<td>Lebrikizumab</td>
<td>Amlitelimab</td>
<td>LEO138559</td>
<td>Nemolizumab</td>
<td>Tezspire (tezspire)</td>
<td>Fezakinumab</td>
</tr>
<tr>
<td>Inflammation targeted</td>
<td>Type 2</td>
<td>Type 2</td>
<td>Type 1, 2, 3</td>
<td>Type 3</td>
<td>Type 2</td>
<td>Type 2, 3</td>
<td>Type 3</td>
</tr>
<tr>
<td>EASI-75 % responders pbo adjusted</td>
<td>49</td>
<td>38</td>
<td>29</td>
<td>28</td>
<td>13</td>
<td>15</td>
<td>Not available</td>
</tr>
<tr>
<td>IGA(0,1) % responders pbo adjusted</td>
<td>38</td>
<td>26</td>
<td>17</td>
<td>20</td>
<td>11</td>
<td>17</td>
<td>10</td>
</tr>
<tr>
<td>Status in AD</td>
<td>Approved</td>
<td>In development</td>
<td>Terminated</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

We are combining two of the most active and orthogonal MOAs with potential to exceed monotherapy efficacy

Targeting all inflammatory types may provide greater efficacy

Efficacy of advanced systemics in AD (Week 16, placebo-adjusted)

- JAKs inhibit Type 1, 2 and 3 inflammation but carry a black box warning limiting uptake
- DUPIXENT and lebrikizumab block Type 2 inflammation
- Amlitelimab partially inhibits Type 1, 2, and 3 inflammation with an acceptable safety profile

APG777 shows near complete inhibition of Type 2 inflammation – the core driver of AD

APG990 provides potential for broader inhibition to also address heterogenous Type 1 and Type 3 inflammation in AD

NOTE: In lebrikizumab Ph2b and Ph3 there has been no dose-AE or exposure-AE relationship. Lebrikizumab exposures and efficacy are for the Phase 3 dose (500 mg Loading Dose at Weeks 0 and 2, 250 mg Q2W Weeks 4 to 16). Efficacy data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

Potential best-in-class combination therapies in asthma and COPD
Apogee is pursuing potentially best-in-class combinations in respiratory diseases

Estimated WW peak sales in asthma and approval year

Asthma labeled population

$4.6B (Approved 2018)

DUPIXENT (dupilumab)

$3.1B (Approved 2021)

TEZSPIRE (tezepelumab)

$2.3B (Approved 2015)

Nucala (mepolizumab)

$2.0B (Approved 2017)

Fasenra (benralizumab)

Potential for best-in-class efficacy

APG777 / APG808

APG990 / APG333

Potential for every 2-to-3-month maintenance dosing

DUPIXENT (Ph3) and TEZSPIRE (Ph2) have shown lower AER reductions in COPD patients

NOTE: AER = Annualized Exacerbation Rate. These data are derived from different clinical trials conducted at different points in time, with differences in trial design, dosing regimen and patient populations. DUPIXENT label indicates reductions in exacerbations were significant in those with eos ≥150. TEZSPIRE data from population without a biomarker requirement. NUCALA data from population with eos ≥150 at screening or ≥300 in prior year. FASENRA data from two Phase 3 trials in patients with eos ≥300. DUPIXENT COPD data reflective of two Ph3 trials in patients with eos ≥300. TEZSPIRE COPD data shown for patients with eos ≥150.

SOURCE: EvaluatePharma, FDA labels
Recent data has suggested combo inhibition can lead to additive efficacy in respiratory indications

Recent data has suggested combo inhibition can lead to additive efficacy in respiratory indications.

Combined blockade of Type 2 inflammation through IL-13 inhibition and disrupted alarmin signaling by TSLP neutralization demonstrates a potential increase in effect not previously seen by monotherapies alone.

PLACEBO-ADJUSTED FeNO CHANGE (PPB)

**Anti-IL-4Rα trials**

**Anti-TSLP trials**

**Anti-IL-13 trials**

**Anti-IL-13 + Anti-TSLP trial**

**Phase 2a**

**QUEST**

**VENTURE**

**PATHWAY**

**NAVIGATOR**

**SOURCE**

**MILLY**

**LUTE/VERSE**

**MESOS**

**Phase 1b**

NOTE: FeNO level reflects data from marketed dose, where available. Data shown is placebo-adjusted reduction at 29d, with the exception of QUEST, where level was reported at 12 weeks. LUTE/VERSE data from was periostin-high enrollees.

Apogee’s portfolio uniquely enables multiple combos with best-in-class potential in respiratory indications.

- Only known portfolio with IL-13, IL-4R\(\alpha\), OX40L, and TSLP inhibitors to enable optimal respiratory combination approaches.
- Potential to bring Q3M+ dosing to an IL-13 / TSLP combo approach that has been validated by 3rd party data.
- Potential for deeper and broader responses by targeting orthogonal mechanisms.
- Potential for best-in-class dosing via coformulation approach.

APG777 / APG808
APG990 / APG333
Apogee’s TSLP combinations have potential to exceed monotherapy efficacy in a broader population

<table>
<thead>
<tr>
<th>Differentiators</th>
<th>Potential monotherapies without TSLP</th>
<th>Potential best-in-class combos with TSLP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>APG777 in asthma</td>
<td>APG777+APG333 in asthma</td>
</tr>
<tr>
<td></td>
<td>APG808 in COPD</td>
<td>Best-in-class combo in COPD</td>
</tr>
<tr>
<td>Potential patient population (%)</td>
<td>High EOS</td>
<td>Potential for broader population</td>
</tr>
<tr>
<td>eligible</td>
<td></td>
<td><em>Based on TSLP MoA + data in all-comers asthma</em></td>
</tr>
<tr>
<td>Potential efficacy advantage</td>
<td>TBD, based on optimizing exposure</td>
<td>IL-13+TSLP has demonstrated additive FeNO benefit</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Potential to exceed monotherapy ceiling</td>
</tr>
</tbody>
</table>
APG808 leverages DUPIXENT's mechanism to deliver a potentially best-in-class, pipeline-in-a-product antibody

APG808's epitope on IL-4R\(\alpha\) overlaps with DUPIXENT's and leverages proven MoA and biology

APG808 is as potent as DUPIXENT across preclinical assays

APG808 NHP half-life is more than 2x longer than DUPIXENT
APG808 Phase 1a clinical trial objectives

OBJECTIVES

Confirm tolerable safety profile

Establish optimized PK profile with a half-life of at least 42 days

Determine dosing regimens to sustain exposures similar to DUPIXENT

Q4 2024: confirm potential for best-in-class dosing intervals

Dosing Goal: every 6- or 8-weeks (vs. every 2 weeks for DUPIXENT1)

NOTE: PK = Pharmacokinetic. 1 Based on FDA label for DUPIXENT™
Expansion indications
Our programs have broad potential to disrupt the I&I space

Expansion

Our programs have broad potential to disrupt the I&I space

**Dermatology**
- AD
- AA
- PN
- BP
- HS

**Pulmonology / Immunology**
- Asthma
- COPD
- CRSwNP
- CSU
- AR
- ColdU
- CPUO
- Food allergy
- SS

**Gastroenterology**
- EoE
- Celiac Disease
- EGID
- UC

- Planned Apogee Ph2 based on well-established mechanism
- 3rd party clinical data supporting one or more Apogee targets
- Ongoing 3rd party clinical trials which could validate one or more Apogee targets
- Potential blockbuster expansion

NOTE: 1 Eosinophilic subtypes 2 Perennial
We are pursuing the largest markets in I&I with a total addressable population over 300M

WW addressable patient population across indications (in millions)

<table>
<thead>
<tr>
<th>Indication</th>
<th>Market 2030E</th>
<th>Addressable Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>AD¹</td>
<td>$29.0B</td>
<td>81.6</td>
</tr>
<tr>
<td>Asthma¹</td>
<td>$20.0B</td>
<td>65.5</td>
</tr>
<tr>
<td>COPD¹</td>
<td>$13.5B</td>
<td>59.6</td>
</tr>
<tr>
<td>CSU²</td>
<td>$4.5B</td>
<td>64.4</td>
</tr>
<tr>
<td>CRSwNP²</td>
<td>$2.0B</td>
<td>60.3</td>
</tr>
<tr>
<td>AA²</td>
<td>$1.0B</td>
<td>6.1</td>
</tr>
<tr>
<td>PN²</td>
<td>$0.5B</td>
<td>3.8</td>
</tr>
<tr>
<td>EoE²</td>
<td>$0.5B</td>
<td>2.8</td>
</tr>
<tr>
<td>EGID &amp; AR</td>
<td>TBD</td>
<td>300M+</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Total WW Addressable Population</th>
<th>TBD</th>
</tr>
</thead>
<tbody>
<tr>
<td>2030E Market Size</td>
<td>$70B++</td>
</tr>
</tbody>
</table>

NOTE: AD = Atopic Dermatitis, COPD = Chronic Obstructive Pulmonary Disease, CSU = Chronic Spontaneous Urticaria, CRSwNP = Chronic Rhinosinusitis with Nasal Polyps, EoE = Eosinophilic Esophagitis, PN = Prurigo Nodularis, AA = Alopecia Areata, EGID = Eosinophilic Gastrointestinal Disorders (non-EoE), AR = Allergic Rhinitis.

¹ Encompasses moderate-to-severe population. ² Encompasses prevalent population.

SOURCE: Academic journals, disease foundations, WHO, CDC, census data, analyst research, EvaluatePharma. 2030E market size rounded to nearest $0.5B.
Corporate & Commercial
Experienced team with proven history of clinical development and commercial execution

Michael Henderson, MD
Chief Executive Officer, Director

Carl Dambkowski, MD
Chief Medical Officer

Jane Pritchett Henderson
Chief Financial Officer

Rebecca Dabora, PhD
Chief Development Officer

Matt Batters, JD
Chief Legal Officer

Wendy Aspden-Curran
SVP of Clinical Operations

Drew Badger, PhD
SVP of Regulatory Affairs & Toxicology

Dan Mulreany
SVP of Business Development & Strategy

Kristine Nograles, MD, MSc
SVP of Clinical Development
Board of Directors with industry-leading development, regulatory, commercial and management expertise

Mark McKenna  
Chairman & CEO, Mirador Therapeutics

Michael Henderson, MD  
CEO, Apogee Therapeutics

Lisa Bollinger, MD  
CEO & President of Bollinger Regulatory Consulting, LLC

Jennifer Fox  
CFO & CBO, Zenas BioPharma

Andrew Gottesdiener, MD  
Venrock

Boston, MA  

Peter Harwin  
Managing Member, Fairmount

BJ Jones  
CCO, NewAmsterdam Pharma

Tomas Kiselak  
Managing Member, Fairmount

Nimish Shah  
Venrock

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### APG777 could command #1 market share in the potentially $50B+ AD market

<table>
<thead>
<tr>
<th>Analog</th>
<th>Differentiation</th>
<th>Peak Market Share</th>
<th>Corresponding Peak Sales in the potentially $50B AD Market</th>
<th>Apogee’s Path</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potential for APG777 to be the first</td>
<td>No known analogs have best-in-class dosing and efficacy</td>
<td>Potential for 33%+</td>
<td>~$17B+</td>
<td>APG777 has the potential to “win the day” with anticipated best-in-class dosing and efficacy</td>
</tr>
<tr>
<td>Skyrizi (risankizumab-rzaa)</td>
<td>Skyrizi is primarily differentiated by its quarterly dosing profile</td>
<td>~33%</td>
<td>~$17B</td>
<td>Apogee’s path to dosing differentiation is de-risked by its potential best-in-class PK</td>
</tr>
<tr>
<td>Relatively undifferentiated</td>
<td></td>
<td>~8 to 10%</td>
<td>~$5B</td>
<td>Apogee’s existing data suggest an already differentiated profile making this case unlikely</td>
</tr>
</tbody>
</table>

“Skyrizi of AD” base case suggests ~$17B revenue potential for APG777; significant upside to revenue potential if efficacy advantage is demonstrated
## Multiple anticipated milestones through 2025 with $790M in cash providing expected runway into 2028

### Key readout

<table>
<thead>
<tr>
<th>Potential best-in-class monotherapy in AD</th>
<th>2024</th>
<th>2025</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>APG777</strong> IL-13</td>
<td>✓ Positive Phase 1 PK &amp; safety in HVs</td>
<td>✓ 1H: Phase 2 initiated in AD</td>
</tr>
<tr>
<td><strong>APG808</strong> IL-4Rα</td>
<td>✓ Phase 1 initiated in HVs</td>
<td></td>
</tr>
<tr>
<td><strong>APG990</strong> OX40L</td>
<td>✓ Candidate nomination</td>
<td></td>
</tr>
<tr>
<td><strong>APG333</strong> TSLP</td>
<td></td>
<td>• Candidate nomination</td>
</tr>
</tbody>
</table>

### Potential best-in-class mAbs for combinations

| **APG777 ± APG990** IL-13 ± OX40L | | • Clinical trial initiation in AD |
| **APG777 ± APG333** IL-13 ± TSLP | | |

### Potential first- or best-in-class combination approaches

| Additional combination(s) IL-13/IL-4Rα ± OX40L/TSLP | | • 4Q: Additional respiratory combination(s) to be announced at R&D Day in December |

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NOTE: As of June 30, 2024, Apogee had cash, cash equivalents and marketable securities of $790M.
Apogee /ˈapəjē/ noun

The highest point in the development of something; a climax or culmination