UNITED STATES
SECURITIES AND EXCHANGE COMMISSION

WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): March 5, 2024

Apogee Therapeutics, Inc.

(Exact Name of Registrant as Specified in Its Charter)

Delaware	**001-41740**	**88-0588063**
(State of Incorporation or Organization)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

221 Crescent Street, Building 17, Suite 102b,
Waltham, MA, 02453
(Address of Principal Executive Offices, including Zip Code)

(650) 394-5230
(Registrant's telephone number, including area code)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, par value $0.00001 per share	**APGE**	**The Nasdaq Global Market**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On March 5, 2024, Apogee Therapeutics, Inc. (the "Company") made available a presentation regarding its initial Phase 1 data from its first-in-human study of APG777 on the Company's website.

A copy of the data presentation is furnished as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated by reference herein. The exhibit furnished under Item 7.01 of this Current Report on Form 8-K shall not be deemed to be "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Exchange Act or the Securities Act of 1933, as amended, regardless of any general incorporation language in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) *Exhibits*.

EXHIBIT INDEX

Exhibit No.	Description
99.1	Data Presentation, dated March 2024
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

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

Apogee Therapeutics, Inc.

Date: March 5, 2024

By: /s/ Michael Henderson, M.D.
Michael Henderson, M.D.
Chief Executive Officer

APOGEE THERAPEUTICS

APG777 PHASE 1 DATA

March 2024

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 forward-looking statements, including statements about our plans, objectives, goals, strategies and future events, the efficacy, safety, tolerability, PK and PD profile of APG777, the potential dos regimen of APG777, the potential superiority of APG777 compared to current therapies, our expectations regarding plans for our current and future product candidates and programs, our plans for current and future clinical trials, our plans for clinical trial design, the anticipated timing of the initiation of and results from our clinical trials, the potential clinical benefit and half-life of APG777, APG8 APG990, APG222 and any other potential programs, our expected timing for future pipeline updates and estimates of market size. In some cases, you can identify forward-looking statements by te 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 ter 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 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 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 expresse 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 Annual Report on Form 10-K for the year ended December 31, 2023, filed with the SEC on March 5, 2024, and subsequent disclosure documents we may file with the U.S. Securities and Excha 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 fac that cause results not to be as anticipated, estimated or intended.

This presentation concerns drug candidates that are under clinical investigation, and which have not yet been approved by the U.S. Food and Drug Administration. These are currently limited by fed law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

The assumptions used in the preparation of this presentation, although considered reasonable by us at the time of preparation, may prove to be incorrect. You are cautioned that the informatio based on assumptions as to many factors and that actual results may vary from the results projected and such variations may be material. Accordingly, you should not place undue reliance on forward-looking statements contained herein or rely on them as predictions of future events. All forward-looking statements in this presentation apply only as of the date made and are expres qualified by the cautionary statements included in this presentation. We do not undertake to update any forward-looking statements, except in accordance with applicable securities laws.

The trademarks, trade names and service marks appearing in this presentation are the property of their respective owners. Certain information contained in this presentation relate to or are based studies, publications and other data obtained from third-party sources as well as our own internal estimates and research. While we believe these third-party sources to be reliable as of the date of presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources.

Agenda

Introduction & Executive Summary		Michael Henderson, MD Chief Executive Officer
APG777 Phase 1 Interim Results		Carl Dambkowski, MD Chief Medical Officer
APG777 Phase 2 Trial in Atopic Dermatitis		Kristine Nograles, MD SVP, Clinical Development
Building a Leading I&I Company		Michael Henderson, MD Chief Executive Officer
Analyst Q&A	  	Michael Henderson, MD, CEO Carl Dambkowski, MD, CMO Jane Pritchett Henderson, CFO

Apogee plans to reshape the current standard of care for inflammatory and immune diseases

- Focus on developing differentiated biologics with known biologic drivers
- Near term priority on treatments for atopic dermatitis (AD), asthma and chronic obstructive pulmonary disease (COPD)

- People living with these diseases deserve the best possible treatment
- Significant unmet need continues

Refusing to stop at "good enough"

1Q24 Update reflects significant progress

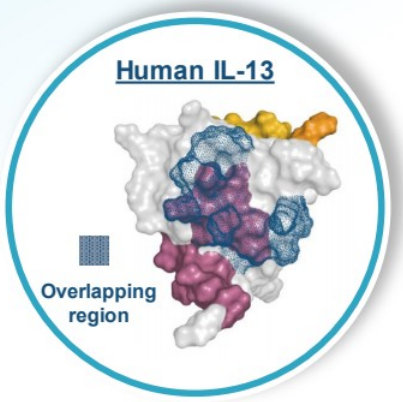
APG777

- **Phase 1 initial data has exceeded all trial objectives**
- Phase 2 in AD anticipated to start in 1H 2024 (ahead of schedule)
- Planned, integrated Phase 2 in AD combines Phase 2a and Phase 2b elements with potential fo significant timeline acceleration (topline data from Part A remains 2H 2025)
- Enhanced 180 mg/mL formulation enables 44% higher dose vs lebrikizumab in the same volum

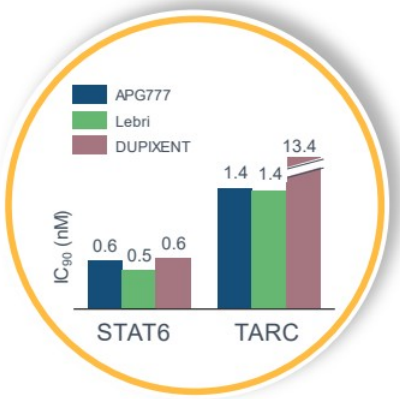
APG808

- Expect to initiate Phase 1 in healthy volunteers in 1H'24 (ahead of schedule)
- Phase 1 interim data accelerated to 2H 2024 (from 2025)

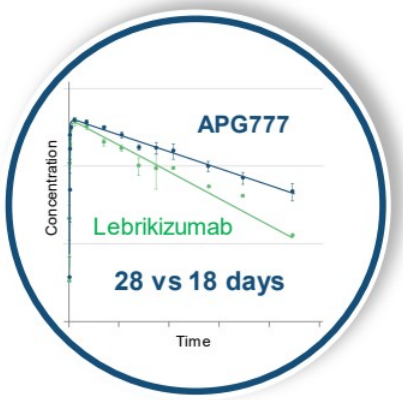
APG777 leverages lebrikizumab's mechanism to deliver a potentially best-in-class, pipeline in a product antibody



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



APG777 NHP half-life is significantly longer than lebrikizumab

NOTE: MoA = Mechanism of Action.

Phase 1

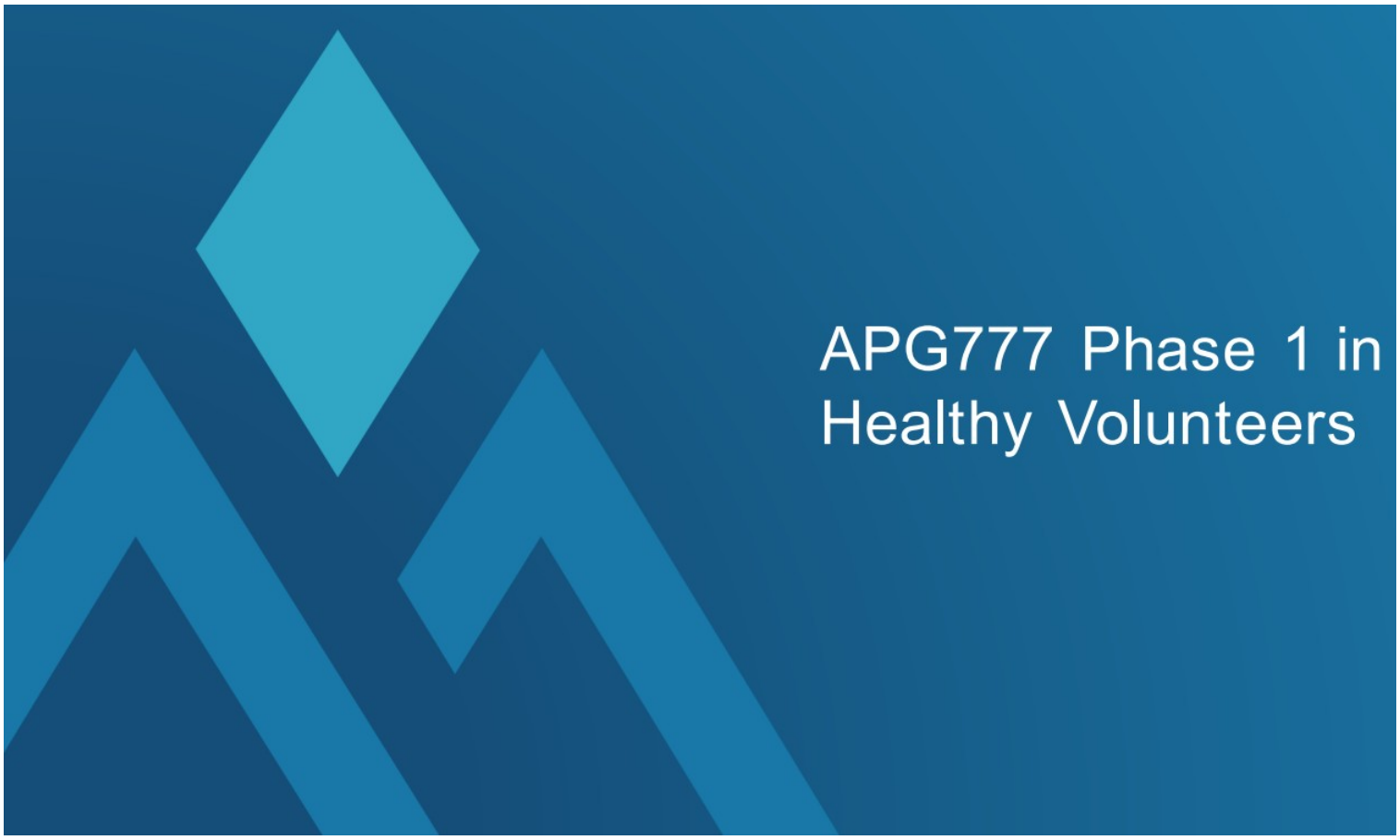
APG777 Phase 1 initial data has exceeded all trial objectives

	Establish **safety** & **PK** profile	Set **Ph2 induction regimen**	Set **Ph2 maintenance regimens**	Supplemental
GOAL	*Well-tolerated with at least 33-day half-life*	*Achieve at least equiv. exposures to lebrikizumab with same or fewer injections*	*Equal lebrikizumab exposure with every 2-month or longer dosing[1]*	*Demonstrate effect on biomarkers pSTAT6 or TARC*
RESULT	• **Half life of ~75 days** • Doses up to 1200mg tested and **well-tolerated** • Initial **multiple-dose** data consistent with PK & safety profile from SAD cohorts	• Regimen modeled to **exceed lebrikizumab exposure by ~30-40% with potential for improved clinical responses (e.g. EASI-75, EASI-90, IGA 0/1)** • **~50% fewer injections than lebrikizumab in induction (6 vs 11)**	• **3- or 6- month maintenance dosing enabled** with modeled exposures similar to or greater than lebrikizumab	• **Extended PD effect** on both pSTAT6 and TARC for ~3 months with follow-up ongoing
	✓ **Exceeded**	✓ **Exceeded**	✓ **Exceeded**	⊕ **Exceeded**

APOGEE THERAPEUTICS

NOTE: PK = Pharmacokinetic

[1] Exposure target based on C_{trough} in maintenance, the minimal concentration of APG777 to have similar exposures to lebrikizumab


APG777 Phase 1 in
Healthy Volunteers

Phase 1

APG777 interim data from ongoing Phase 1 trial in healthy volunteers

Trial design elements

Double-blind, placebo-controlled, first-in-human trial

Single ascending dose component with a nested multiple ascending dose component

N = 40

8 per cohort (6:2 active:placebo)

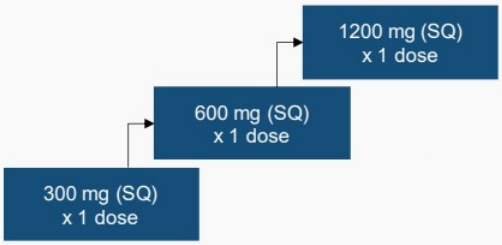
Key inclusion criteria: healthy adult participants

Primary endpoint: safety

Secondary endpoints: PK, ADA

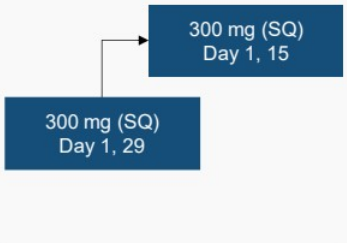
Exploratory biomarkers: pSTAT6, TARC

Single ascending dose



Interim data from all three SAD cohorts with at least 3-6 months follow-up

Multiple ascending dose



Fully enrolled Preliminary data from MAD

Phase 1

Baseline characteristics are in line with our expectations

	Single dose				Multiple dose		
	Placebo N=6	**Cohort 1** 300 mg N=6	**Cohort 2** 600 mg N=6	**Cohort 3** 1,200 mg N=6	**Placebo** N=4	**Cohort 1** 300 mg at Day 1, 300 mg at Day 29 N=6	**Cohort** 300 mg at D 300 mg at Da N=6
Age (yrs), mean (SD)	41.3 (16.2)	30.2 (12.2)	40.2 (18.4)	29.7 (4.6)	42.0 (12.1)	42.7 (13.9)	40.2 (13.
Female	100%	66.7%	83.3%	33.3%	100%	50.0%	50.0%
Caucasian	100%	33.3%	83.3%	100%	75.0%	100%	33.3%
Weight (kg), mean (SD)	72.5 (12.6)	74.3 (14.6)	78.8 (14.0)	77.2 (16.2)	62.3 (9.5)	80.5 (8.9)	66.7 (12.

Demographics were well balanced across cohorts

Phase 1

APG777 was well-tolerated with a favorable safety profile

	Single dose				Multiple dose			Overall trial	
N (%)	**Placebo** N=6	**Cohort 1** 300 mg N=6	**Cohort 2** 600 mg N=6	**Cohort 3** 1,200 mg N=6	**Placebo** N=4	**Cohort 1** 300 mg at Day 1, 300 mg at Day 29 N=6	**Cohort 2** 300 mg at Day 1, 300 mg at Day 15 N=6	**APG777** N=30	**Pla** N
Participants with at least one TEAE	5 (83.3%)	4 (66.7%)	5 (83.3%)	2 (33.3%)	2 (50.0%)	5 (83.3%)	1 (16.7%)	17 (56.7%)	7 (7
Participants with at least one TE-SAE	0	0	0	0	0	0	0	0	
Participants with at least one drug-related AE	3 (50.0%)	0	1 (16.7%)	1 (16.7%)	0	1 (16.7%)	0	3 (10.0%)	3 (3
Participants with at least one ≥Grade 3 TEAE	0	0	0	0	0	0	0	0	
Participants that discontinued study due to TEAE	0	0	0	0	0	0	0	0	
Participants that decreased dose due to TEAE	0	0	0	0	0	0	0	0	

The safety profile is in line with expectations for therapies targeting the IL-13 pathway

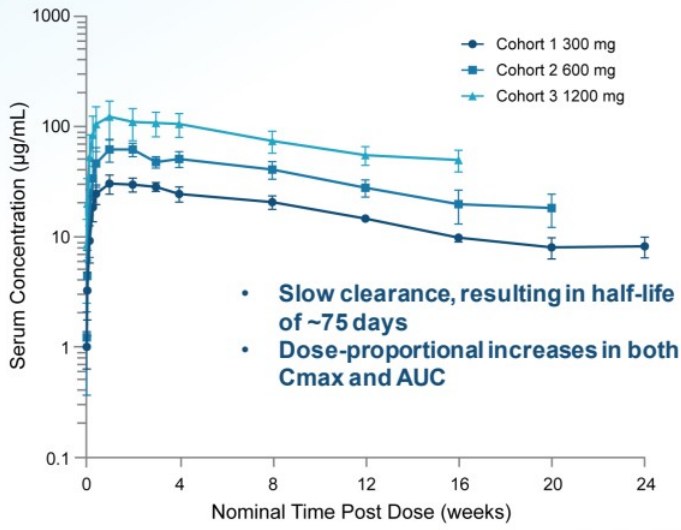
APOGEE THERAPEUTICS

NOTE: TEAE = Treatment-Emergent Adverse Event. TE-SAE = Treatment-Emergent-Serious Adverse Event. Interim data includes AEs reported as of 16 February 2024 data cut. The trial is ongoing.

Phase 1

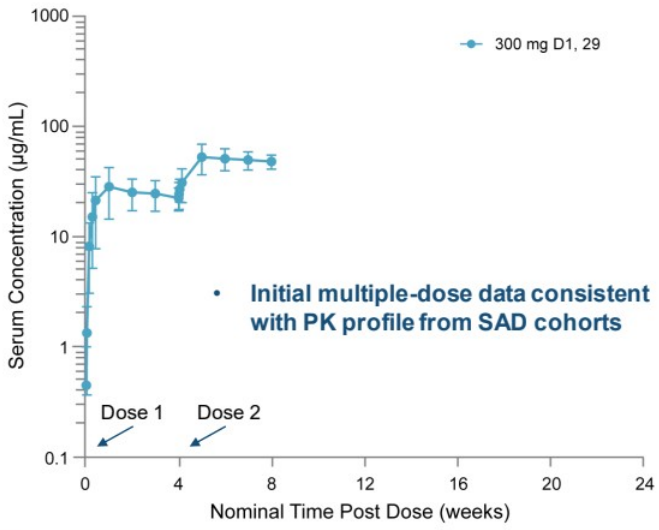
APG777 exhibited a potentially best-in-class PK profile with a half-life of ~75 days

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

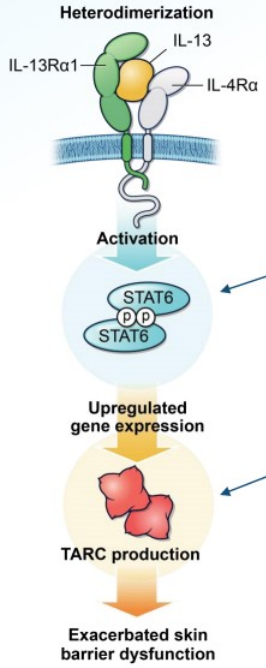


- Initial multiple-dose data consistent with PK profile from SAD cohorts

PK demonstrated dose-proportionality and half-life of ~75 days (approximately 3x lebrikizumab)

Phase 1

pSTAT6 and TARC are biomarkers of IL-13 target engagement and AD severity



APG777 Phase 1 biomarkers

1. **pSTAT6** is one of the **earliest markers of IL-13 receptor activation**

2. **TARC levels are the most strongly correlated to AD severity of any biomarker**

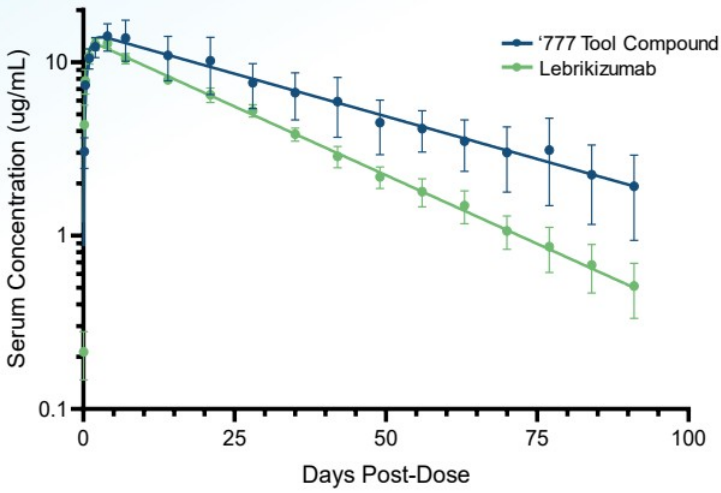
Taken together, **APG777's reduction of these biomarkers confirms inhibition of IL-13 signaling** and allows comparison to other agents

APOGEE THERAPEUTICS

NHP Data

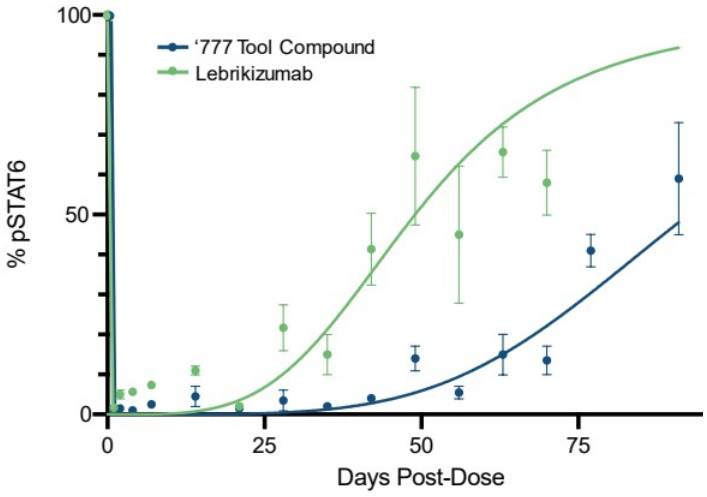
In a head-to-head NHP study, '777 tool compound inhibited pSTAT6 significantly longer than lebrikizumab

Head-to-head PK data in NHP



777 tool compound had **~60% longer half-life** vs. lebrikizumab

Head-to-head PD data in NHP

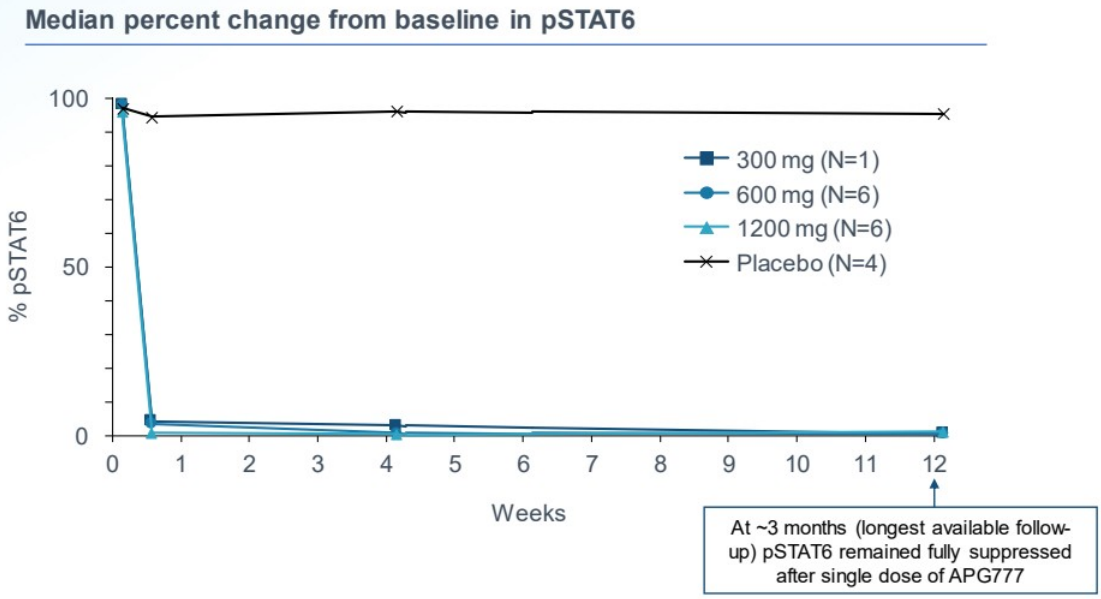


777 tool compound achieved **~2X longer pSTAT6 inhibition** vs. lebrikizumab[1]

*Note: N = 2 for '777 tool compound arm; N = 3 for lebrikizumab arm. Initial pSTAT6 level was normalized to 100% separately for each arm.
1 '777 tool compound sustained at least 50% pSTAT6 inhibition until day 92; lebrikizumab sustained 50% pSTAT6 inhibition until day 48

Phase 1

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



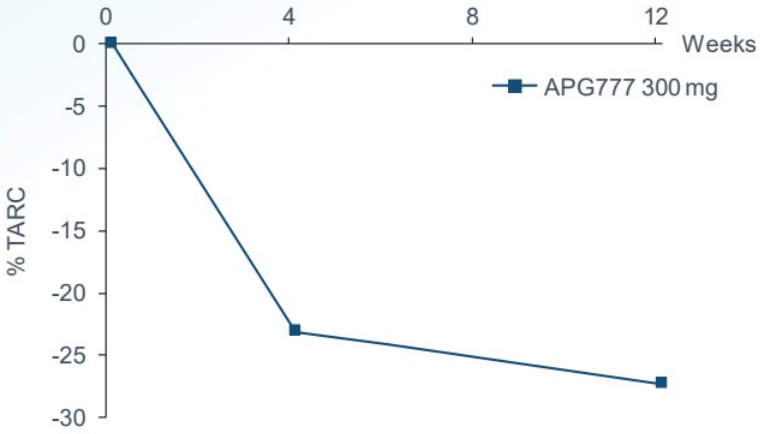
100% pSTAT6 inhibition was demonstrated for approximately 3 months across all doses

NOTE: N = 1 in cohort 1 due to the accelerated timing of study enrollment relative to assay validation. No data has been published showing DUPIXENT or lebrikizumab impact on pSTAT6 in HVs.

Phase 1

Single dose of APG777 led to deep + sustained TARC inhibition for ~3 months (limit of available follow-up)

Median % changes from baseline in TARC inhibition



Median % changes from baseline in TARC inhibition

- **300 mg APG777 showed similar maximum PD marker changes as DUPIXENT**
- **APG777 sustained TARC inhibition demonstrates the potential for better durability**
- **All doses tested of APG777 showed deep TARC inhibition for ~3 months (limit of available follow-up)**

APOGEE THERAPEUTICS

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.

Phase 1

APG777 positive PK readout is a key risk-reducing milestone that validates program and pipeline

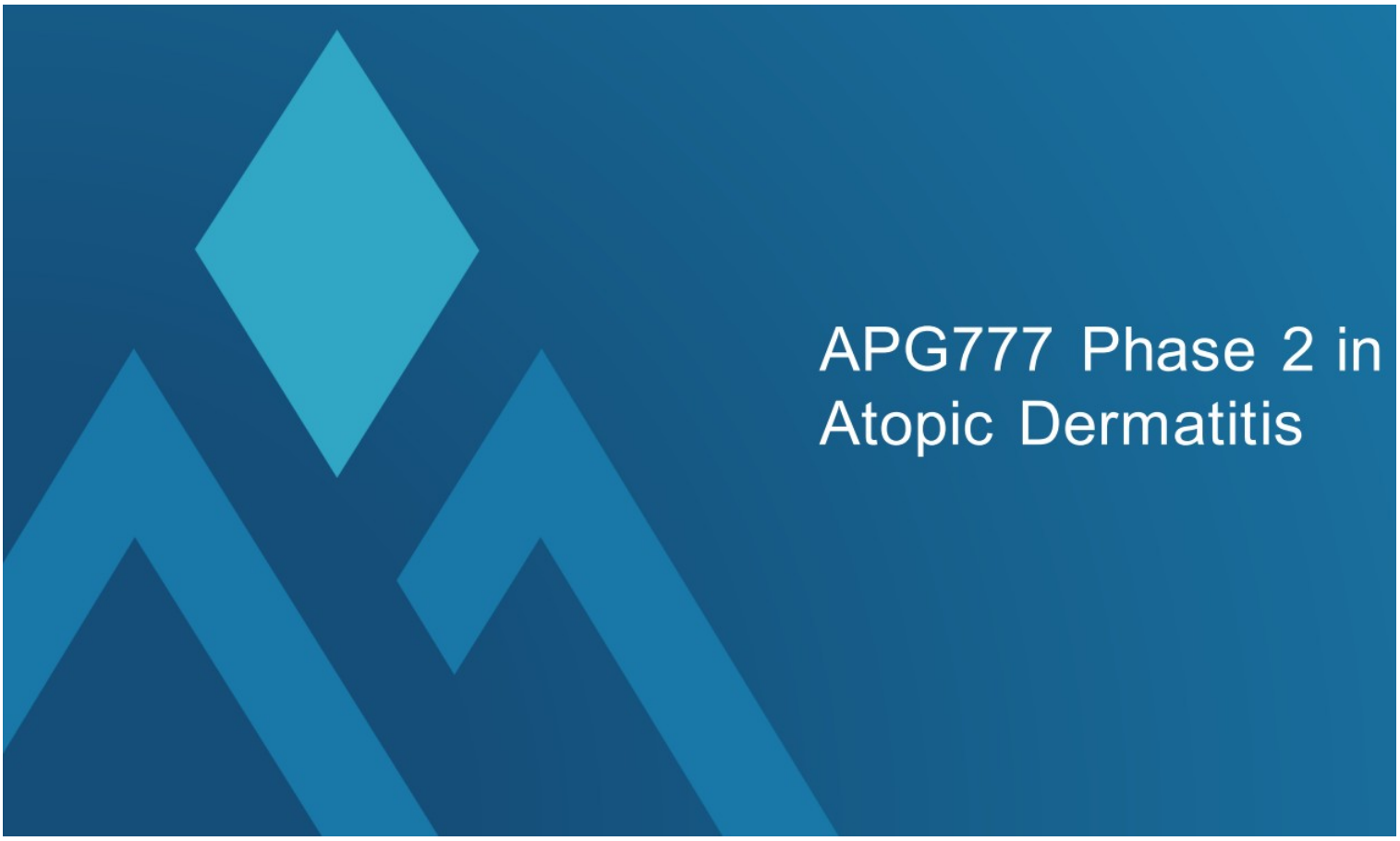
Antibody attributes

- Clinically validated IL-13 target
- Epitope overlaps with lebrikizumab epitope
- Equivalent or better potency vs. 1st generation mAbs across relevant pre-clinical assays

Clinical profile

- Well-tolerated with ability to achieve increased exposures in induction for potential improved clinical responses
- PK data supports every 3- to 6-month maintenance dosing:
  - ~75-day half-life
  - Near maximal pathway suppression for ~3 months (limit of current follow up)

Apogee intends to initiate a Phase 2 in atopic dermatitis in 1H 2024


APG777 Phase 2 in
Atopic Dermatitis

AD Phase 2

APG777 Phase 2 in atopic dermatitis expected to begin 1H 2024 with 16-week efficacy data in 2H 2025

GREATER INDUCTION EXPOSURES

Potential for improved clinical responses (e.g. EASI-75, EASI-90, IGA 0/1) based on ~30-40% greater modeled exposure vs lebrikizumab and ~50% fewer injections

PROLONGED MAINTENANCE DOSING

Every 3- or 6- month maintenance regimens with similar modeled exposure to lebrikizumab Q4W

HIGHER DOSES ENABLED

APG777 180 mg/mL formulation enables 44% greater dose than lebrikizumab in the same volume

INTEGRATED DESIGN

Planned to combine Ph2a and Ph2b elements into a single protocol; significant timeline acceleration over traditional sequenced approach

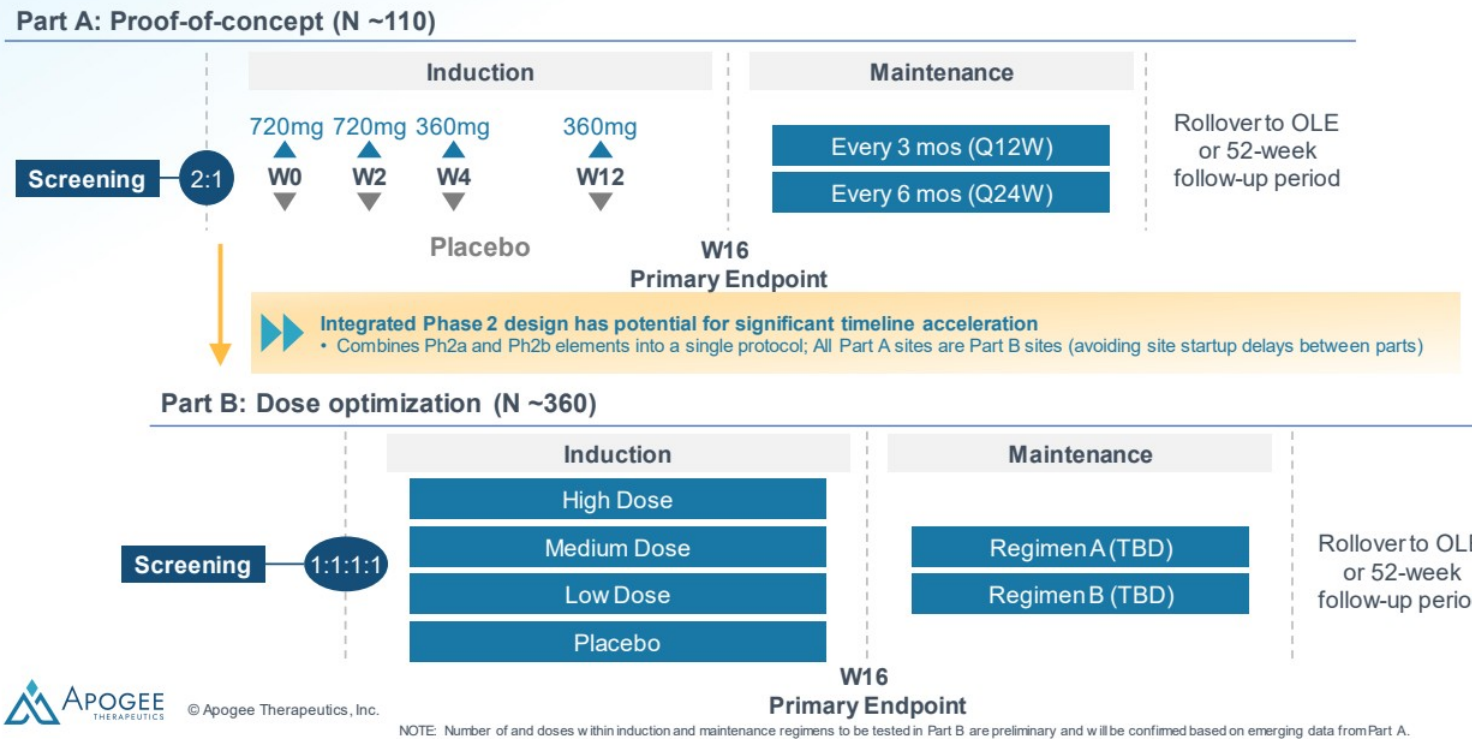
1H 2024 INITIATION

Topline 16 Week data from Part anticipated in 2H 2025

APOGEE THERAPEUTICS

AD Phase 2

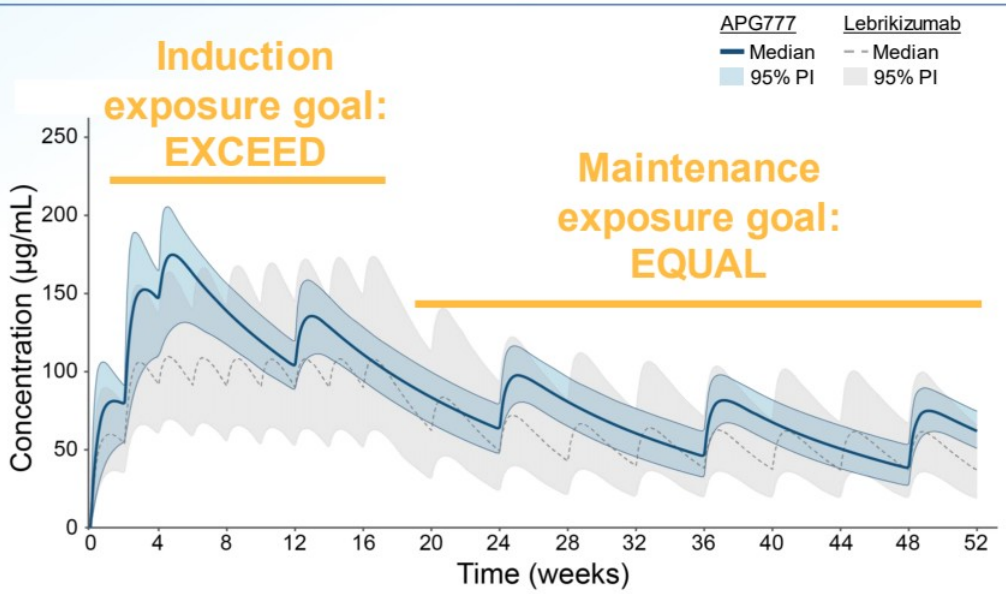
Planned integrated Phase 2 expected to have 16-week topline data in 2H'25



NOTE: 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.

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

Modeled induction and maintenance dosing for APG777[1] and lebrikizumab

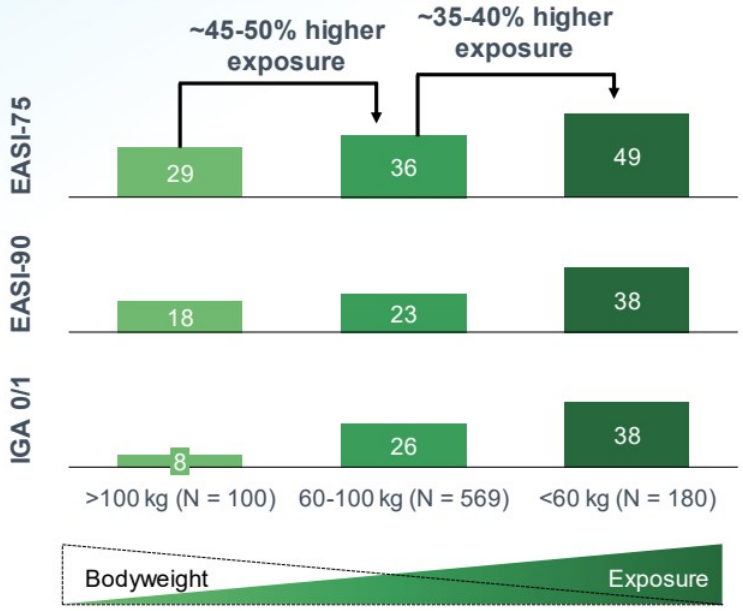


- Lebrikizumab data sugges an **exposure-response (E R) for efficacy in inductio** that underpins our goal **to EXCEED lebrikizumab induction exposures**
- There was **no E-R observ in maintenance for lebrikizumab;** our aim is t **EQUAL its exposure in maintenance**

NOTE: [1]Planned APG777 induction regimen is 720 mg in weeks 0 and 2 and 360 mg in weeks 4 and 12. Maintenance regimen shown is 360 mg every 12 weeks. The above graph is illustrative only with respect to plans for dosing of APG777 and does not present comparative data.

Lebrikizumab Ph3 appears to show an E-R relationship for efficacy in induction that has not been maximized

Lebrikizumab Ph3 response at Week 16 (Placebo-adjusted), %

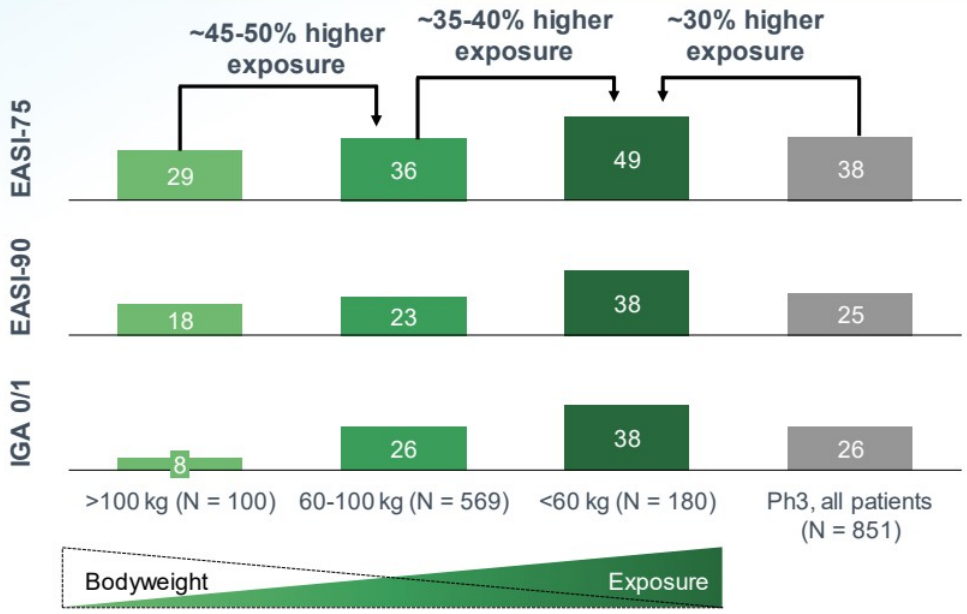


- Lebrikizumab exposure and induction efficacy are both inversely correlated with body weight
- Relationships suggest an **exposure-response for efficac in induction and support testi higher exposures with APG77**
- In lebrikizumab Ph2b and Ph3 there has been **no dose-AE or exposure-AE relationship**
- **APG777 plans to test ~30-40% higher exposures in induction with ~50% fewer injections**

SOURCE: Lebrikizumab European Public Assessment Report
NOTE: 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)

Lebrikizumab Ph3 appears to show an E-R relationship for efficacy in induction that has not been maximized

Lebrikizumab Ph3 response at Week 16 (Placebo-adjusted), %



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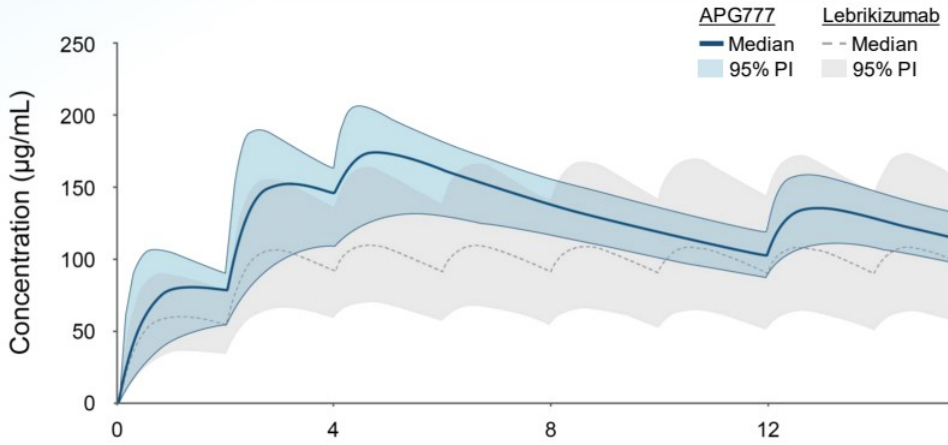
SOURCE: Lebrikizumab European Public Assessment Report
NOTE: Lebrikizumab exposures and efficacy are for the Phase 3 dose (500 mg Loading Dose at Weeks 0 and 2, 250 mg Q2W Weeks 4 to 16)

Modeled Phase 2 induction exposures exceed those of lebrikizumab by ~30-40%

Modeled induction dosing for APG777 and lebrikizumab



~30-40% increase in exposures

Injections

~50% decrease in injections

NOTE: Planned APG777 induction regimen is 720 mg in weeks 0 and 2 and 360 mg in weeks 4 and 12. Exposure increase is based on modeled $C_{average}$ for APG777 vs lebrikizumab. The above graph is illustrative only with respect to plans for dosing of APG777 and does not present comparative data.

AD Phase 2 | Maintenance

Modeled Phase 2 Q3M maintenance exposures equal those of lebrikizumab

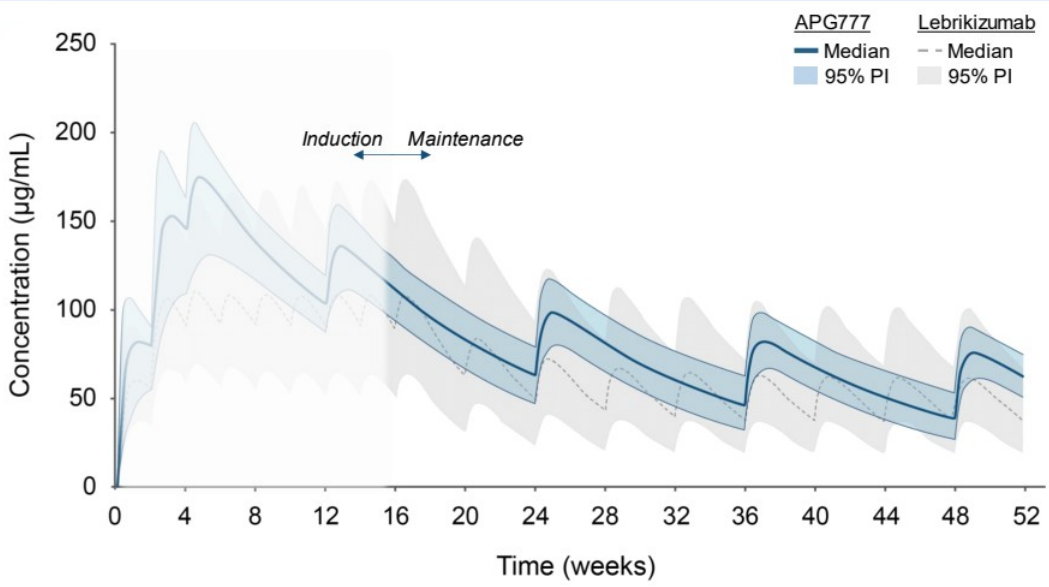
APG777 Q3M

Aiming for annual maintenance injections:

4 *vs* 13-26

for lebrikizumab/ DUPIXENT

Modeled concentration in maintenance



NOTE: The above graph is illustrative only with respect to plans for dosing of APG777 and does not present comparative data.

AD Phase 2 | Maintenance

Modeled Phase 2 Q6M maintenance exposures equal those of lebrikizumab

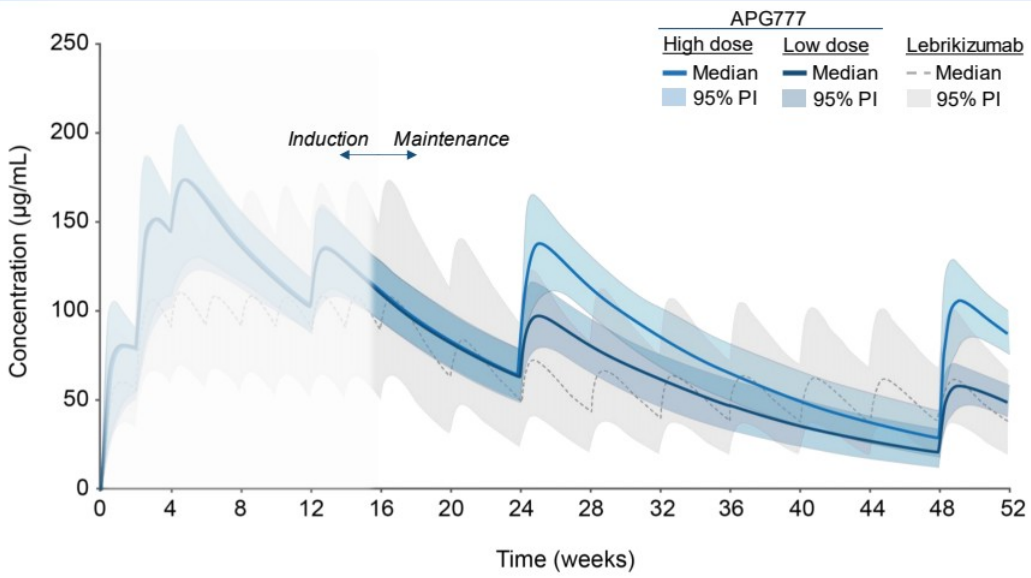
APG777 Q6M

Aiming for annual maintenance injections:

2 *vs* ***13-26***

for lebrikizumab/ DUPIXENT

Modeled concentration in maintenance



NOTE: The above graph is illustrative only with respect to plans for dosing of APG777 and does not present comparative data.


Building a Leading
I&I Company

Corporate

APG777's best-in-class Phase 1 PK profile shows potential to be a leading product in the expected $50B+ AD market[1]

APG777

Potential for improved clinical responses (e.g. EASI-75, EASI-90, IGA 0/1) based on ~30-40% greater modeled induction exposures than lebrikizumab

- Overlapping epitope and equivalent potency as lebrikizumab ($K_D \leq 100$ pM)[2]
- ~30% higher exposure seen in lebrikizumab low bodyweight group resulted in at least 10 PPT better efficacy than overall study population across all key endpoints

Extended dosing interval addresses clear unmet need

- Potential for every 3- or 6-month dosing to improve patient convenience & compliance

Favorable product characteristics and COGS

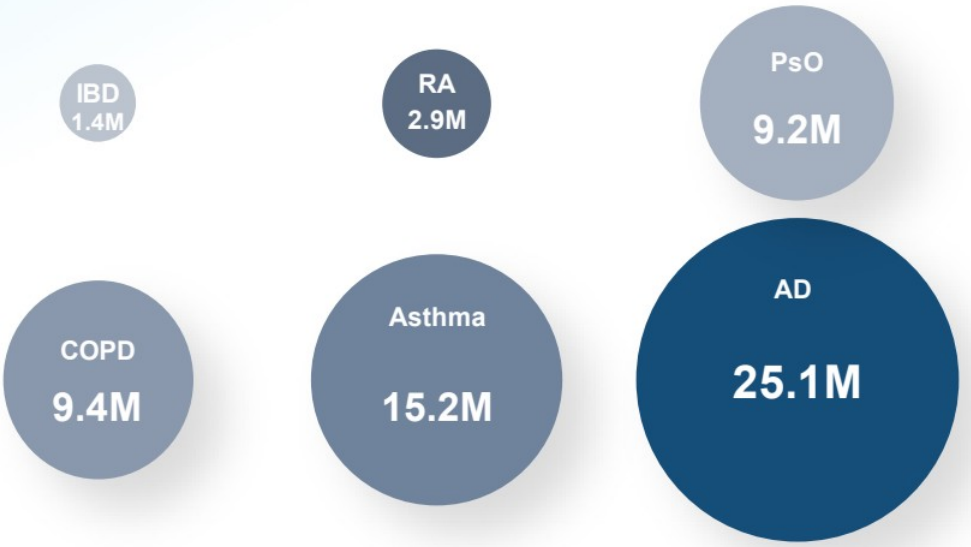
- As few as 2-4 doses per year in maintenance
- Expected improved formulation, manufacturability and viscosity

Novel IP into mid-2040s

NOTE: [1]$50B projected AD market in 2035, based on projected growth of AD market at a similar rate to psoriasis. [2]APG777 has demonstrated equivalent potency to lebrikizumab in our head-to-head preclinical assays. SOURCE: EvaluatePharma.

AD is the largest of the major I&I markets and projected to grow significantly in the next decade

Estimated population size, MM
Moderate or severe in 7 Major Markets[1]

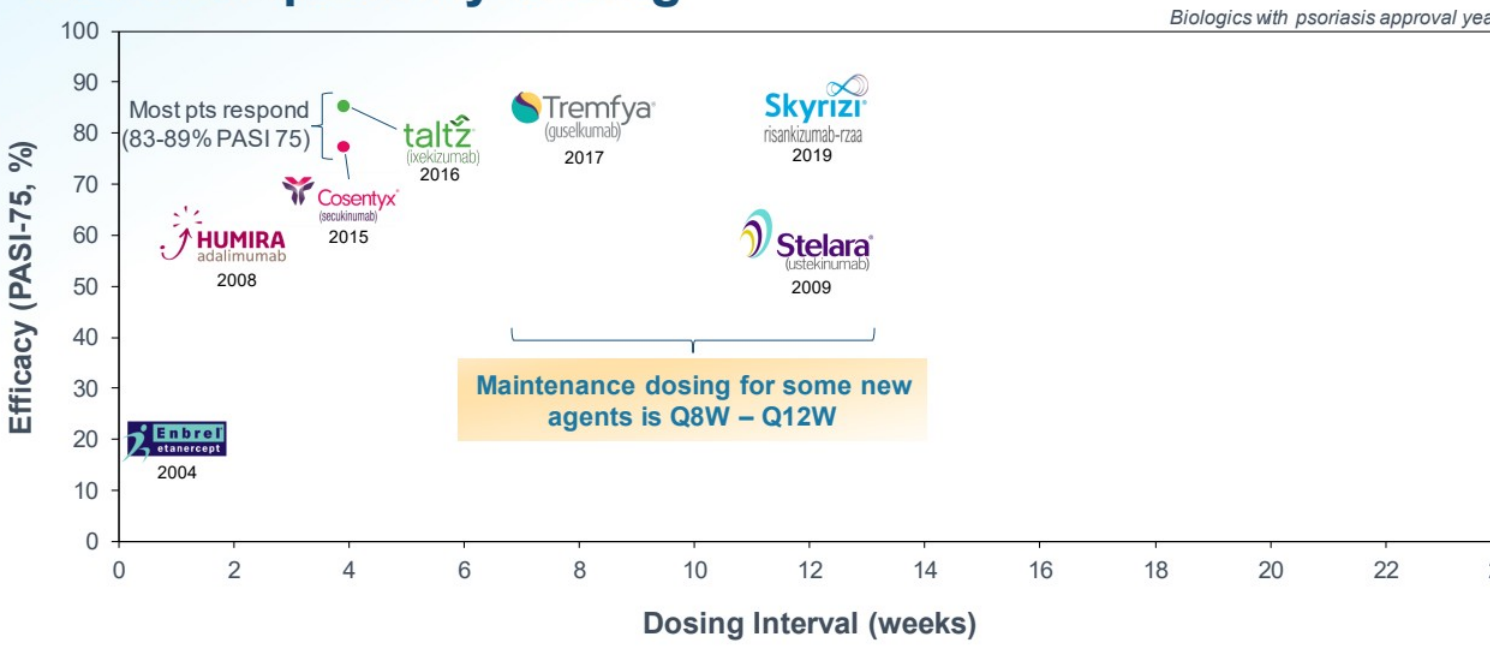


- Psoriasis expected to be a $30B+ market; **atopic dermatitis (AD) represents a larger opportunity** based on **~3 larger patient populatio**
- AD biologics penetration i outpacing early years of psoriasis biologics (8% vs 5% at 5 years)
- **AD market is projected t grow more than any oth I&I market**

[1] The 7 Major Markets are US, Japan, Germany, France, Italy, Spain, and UK. Market size is estimated as of 2022. $30B+ psoriasis projected market size in 2028 and beyond, including both biologics and sma molecules. IBD = Inflammatory bowel disease; RA = Rheumatoid arthritis; PsO = Psoriasis; COPD = Chronic obstructive pulmonary disease; AD = Atopic dermatitis
Source: Company filings, annual reports, press releases, analyst forecasts, academic journals, GlobalData, EvaluatePharma, Clarivate.

Corporate

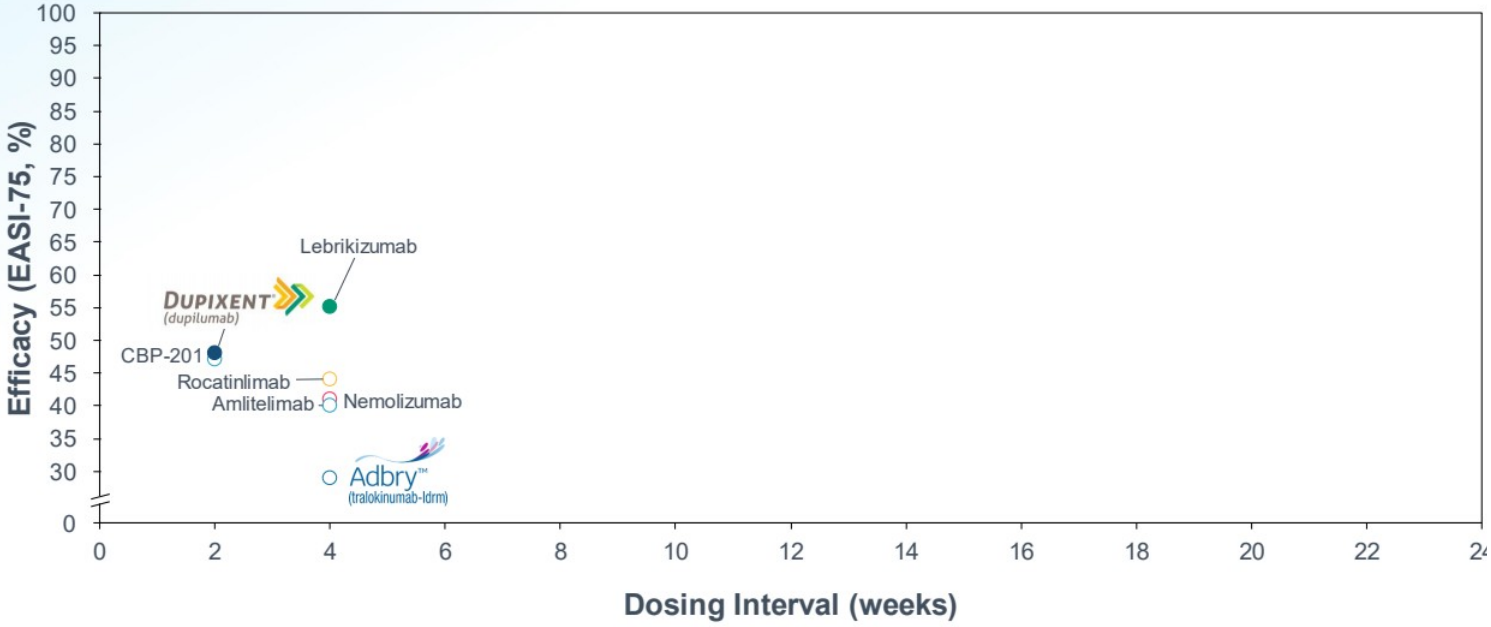
In psoriasis, an analog to AD, Skyrizi has taken the lead with quarterly dosing



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 and patient populations. As a result, cross-trial comparisons cannot be made. No head-to-head trials have been conducted among all biologics shown.
Source: Armstrong AW, et al JAMA Dermatol. 2020. GlobalData. EvaluatePharma. USPIs.

Corporate

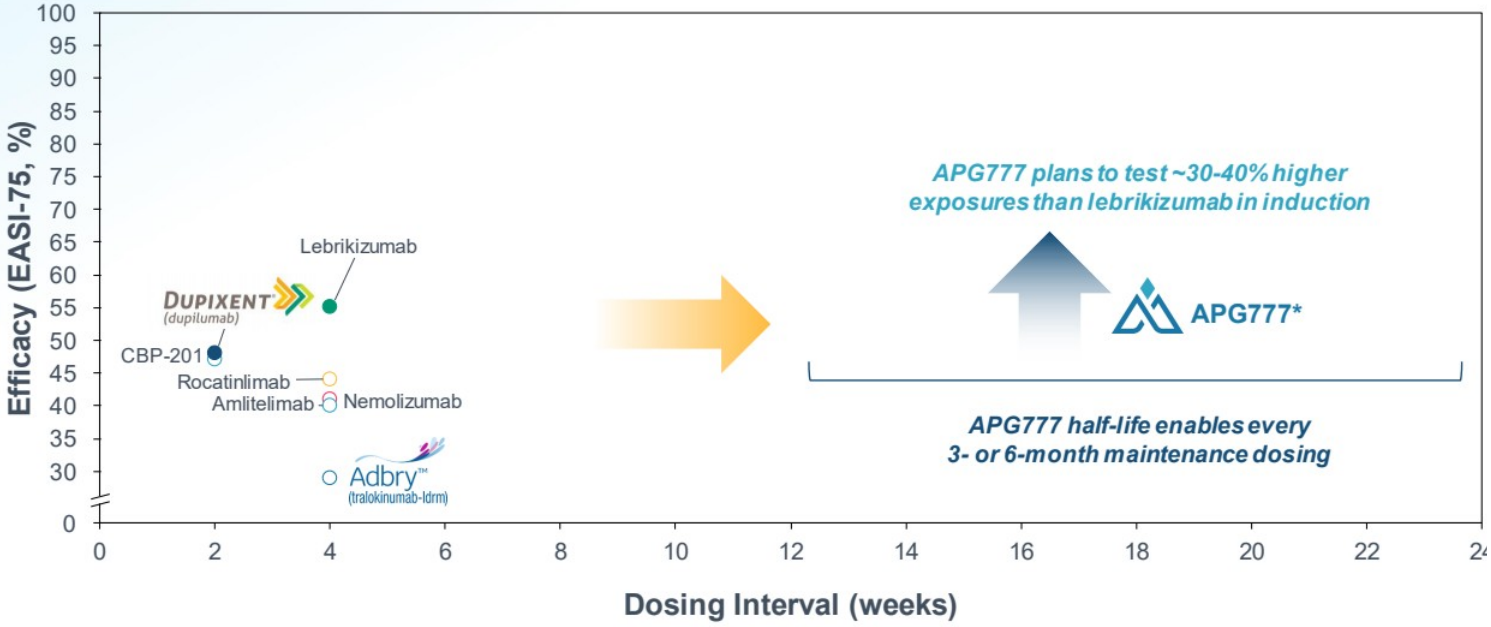
There is significant whitespace in the landscape of approved and in-development biologics for AD



Note: 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. DUPIXENT USPI 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.

Corporate

Apogee plans to advance APG777 into a Phase 2 trial with 3- or 6-month maintenance dosing



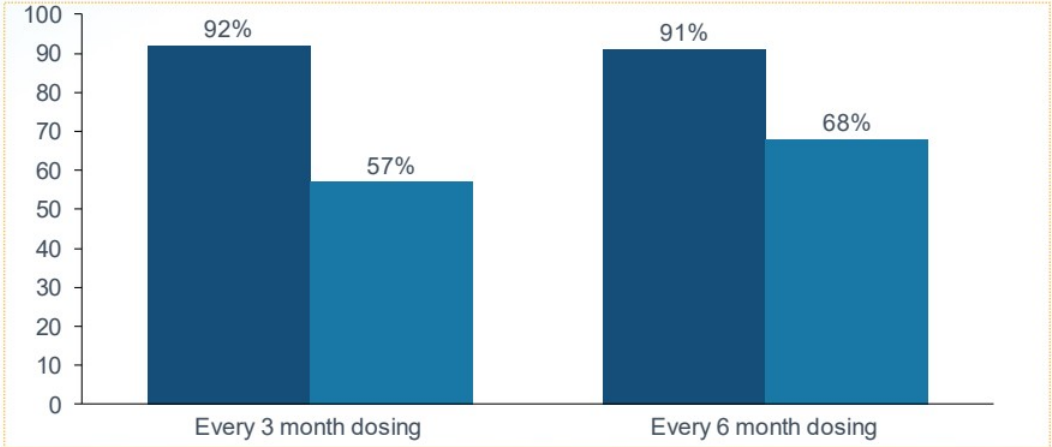
Note: *Positioning of Apogee programs is illustrative and based on interim Phase 1 results only and are illustrative of what we believe we can potentially achieve. Only DUPIXENT and ADBRY are approved. Source: 1. Lebrikizumab 250mg Q2W Ph3 avg. Silverberg JI et al. AAD 2022 2. Dupilumab 300 mg Q2W mono Ph3 avg. DUPIXENT USPI 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 derive 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.

Corporate

Dermatologists view every 3- or 6-month dosing as highly differentiated

Intent to use a product with APG777 Target Product Profile

(Assuming every 3-, or 6-month maintenance dosing and equivalent efficacy and safety to DUPIXENT)



Apogee plans to test every 3- or 6-month maintenance dosing in APG777 Phase 2

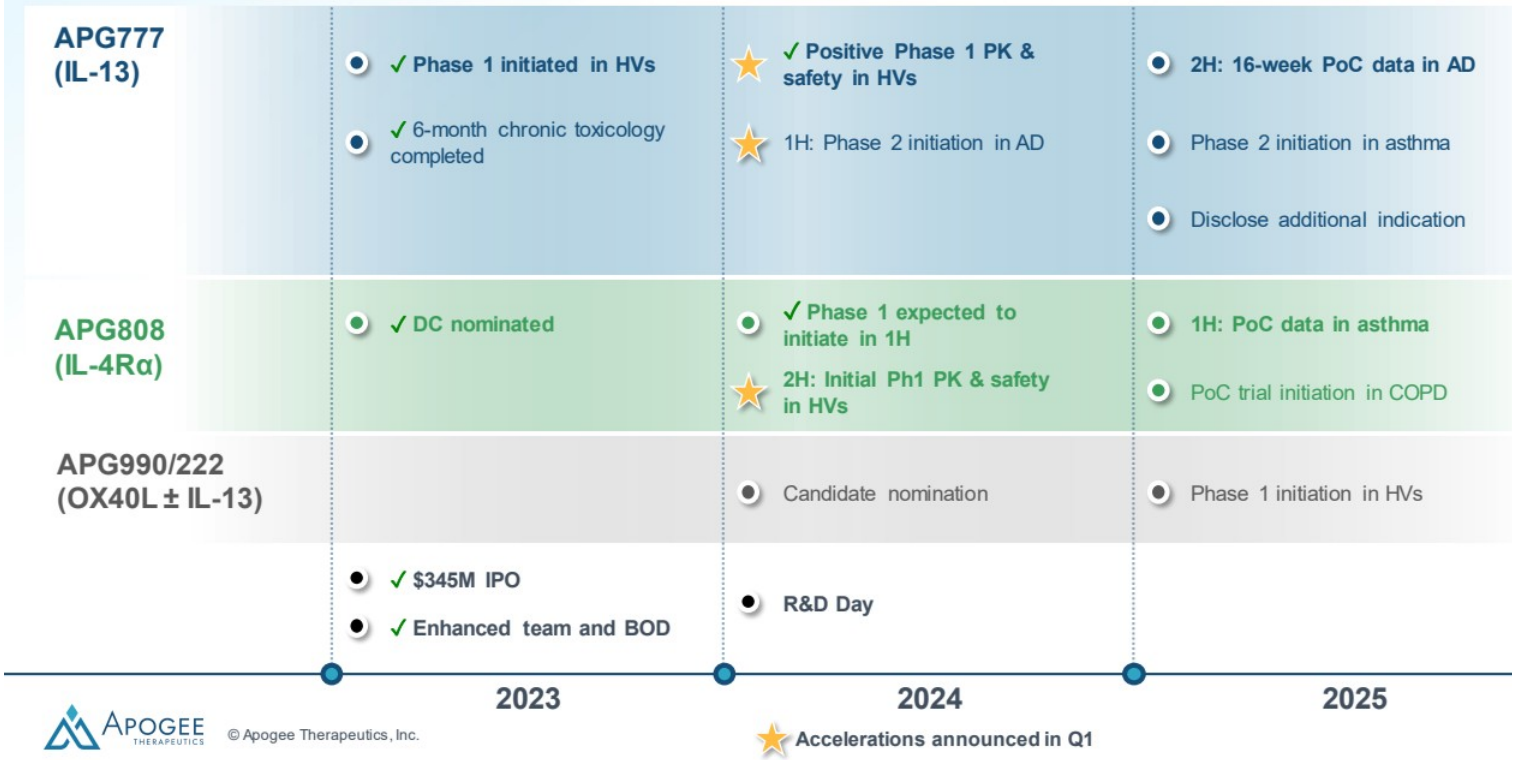
Proportion of new patients (biologic-naïve)

Proportion of switch patients (currently/formerly on a biologic)

Source: In 2023, Apogee conducted a single-blinded market research survey of 25 practicing dermatologists in 14 states in the United States, with the assistance of an expert search network

Note: For providers where likeliness to prescribe Product Y (equivalent efficacy and safety as DUPIXENT™) differs for pediatric and adult patients a blended rate was calculated using the weighted average of the pediatric and adult rates based on the mix of AD patients in that dermatologists' practice

Apogee plans to become a leader in I&I therapeutics



Accelerations announced in Q1


Q&A Backup

Phase 1

APG777 was well-tolerated with a favorable safety profile (TEAEs ≥5% across all cohorts, all grades)

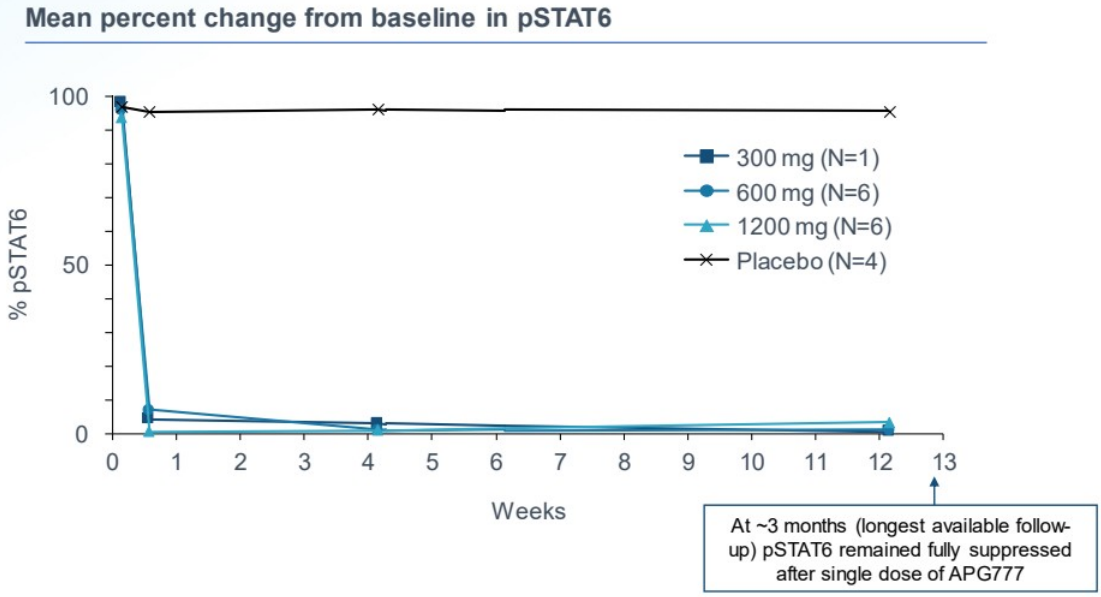
	Single dose				Multiple dose			Overall tria	
N (%)	Placebo N=6	Cohort 1 300 mg N=6	Cohort 2 600 mg N=6	Cohort 3 1,200 mg N=6	Placebo N=4	Cohort 1 300 mg at Day 1, 300 mg at Day 29 N=6	Cohort 2 300 mg at Day 1, 300 mg at Day 15 N=6	APG777 N=30	Pla N
TEAE (≥5% across all cohorts), all grades									
Vascular access site pain*	1 (16.7%)	3 (50.0%)	0	0	0	1 (16.7%)	0	4 (13.3%)	1 (
Vessel puncture site bruise*	2 (33.3%)	0	0	0	1 (25.0%)	2 (33.3%)	0	2 (6.7%)	3 (
Headache	0	0	1 (16.7%)	1 (16.7%)	0	2 (33.3%)	0	4 (13.3%)	0
Vascular access site bruising*	1 (16.7%)	1 (16.7%)	1 (16.7%)	0	0	1 (16.7%)	0	3 (10%)	1 (
Back pain	1 (16.7%)	0	1 (16.7%)	1 (16.7%)	0	0	0	2 (6.7%)	1 (
Injection site bruising*	1 (16.7%)	0	0	2 (33.3%)	0	0	0	2 (6.7%)	1 (
Neutrophil count decrease	3 (50.0%)	0	0	0	0	0	0	0	3 (
Contusion	1 (16.7%)	0	0	0	0	1 (16.7%)	0	1 (3.3%)	1 (
Cough	0	1 (16.7%)	0	0	0	1 (16.7%)	0	2 (6.7%)	0
Dermatitis contact	1 (16.7%)	0	0	0	1 (25.0%)	0	0	0	2 (
Diarrhea	0	1 (16.7%)	0	1 (16.7%)	0	0	0	2 (6.7%)	
Nausea	0	0	1 (16.7%)	1 (16.7%)	0	0	0	2 (6.7%)	
Oropharyngeal pain	0	1 (16.7%)	0	0	0	0	1 (16.7%)	2 (6.7%)	
Pain in extremity	1 (16.7%)	0	1 (16.7%)	0	0	0	0	1 (3.3%)	1 (
Upper respiratory tract infection	1 (16.7%)	0	0	0	0	1 (16.7%)	0	1 (3.3%)	1 (

APOGEE THERAPEUTICS

NOTE: TEAE = Treatment-Emergent Adverse Event. TE-SAE = Treatment-Emergent-Serious Adverse Event. Interim data includes AEs reported as of 16 February 2024 data cut. The trial is ongoing.
*Related to blood draws/needle insertion; not an Injection Site Reaction.

Phase 1

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



100% pSTAT6 inhibition was demonstrated for approximately 3 months across all doses

NOTE: N = 1 in cohort 1 due to the accelerated timing of study enrollment relative to assay validation. No data has been published showing DUPIXENT or lebrikizumab impact on pSTAT6 in HVs.


Company Overview

Apogee plans to transform the I&I space

FOCUS

Engineering antibodies with potential **best-in-class** profiles in largest I&I indications with **highly differentiated dosing**

APPROACH

Technology approach **proven** to create antibodies with significantly **extended half-life** and other optimized properties

EXPANSION

Pipeline-in-a-product potential via **indication expansion** and **combination** approaches

PIPELINE

Four programs leveraging **well-established mechanisms** and addressing I&I indications with **multi-billion-dollar potential**

Program / Target	Discovery	Preclinical	Phase 1	Phase 2	Phase 3
APG777 IL-13 *Same MOA as lebrikizumab*	Atopic Dermatitis			1H 2024: Phase 2 trial initiation[1] 2H 2025: 16-week proof-of-concept data in AD patients	
	Asthma			2025: Phase 2 trial initiation[1]	
APG808 IL-4Rα *Same MOA as DUPIXENT*	COPD		1H 2024: Phase 1 initiation in HV 2H 2024: Initial Phase 1 PK and safety in HV 2025: Proof-of-concept trial initiation in COPD		
APG990 OX40L *Same MOA as amlitelimab*	Atopic Dermatitis	2024: Candidate nomination 2025: Phase 1 initiation in HV			
APG222 Combination IL-13 and OX40L	Atopic Dermatitis				

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 trial of APG777 in healthy participants, we may initiate a Phase 2 trial in asthma and expect to further evaluate opportunities to develop APG777 for other I&I indications, including alopecia 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

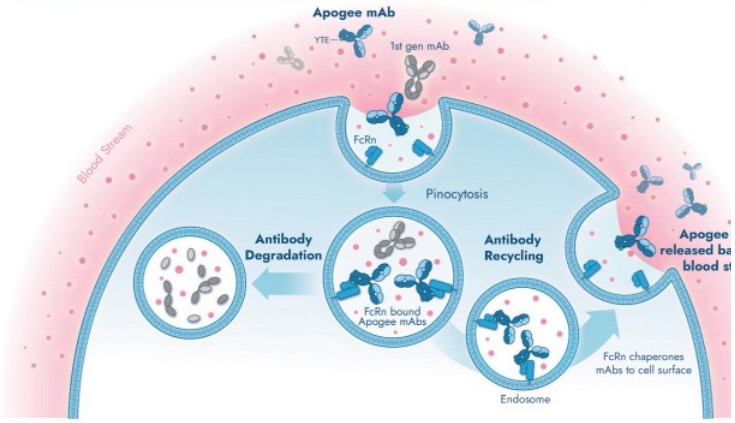
Based on clinically-validated epitopes with performance across five properties:

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

- Designed to maximize antibody recycling
- Drug exists at higher levels for longer effect

Potential for PK that:

- *Optimizes exposures*
- *Decreases variability*
- *Increases half-life*

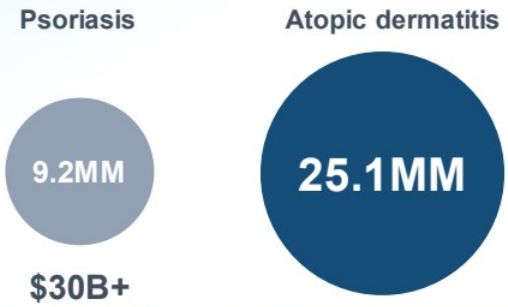


APOGEE THERAPEUTICS


APG777

AD represents a larger opportunity than psoriasis; AD biologics penetration mirrors early years of psoriasis

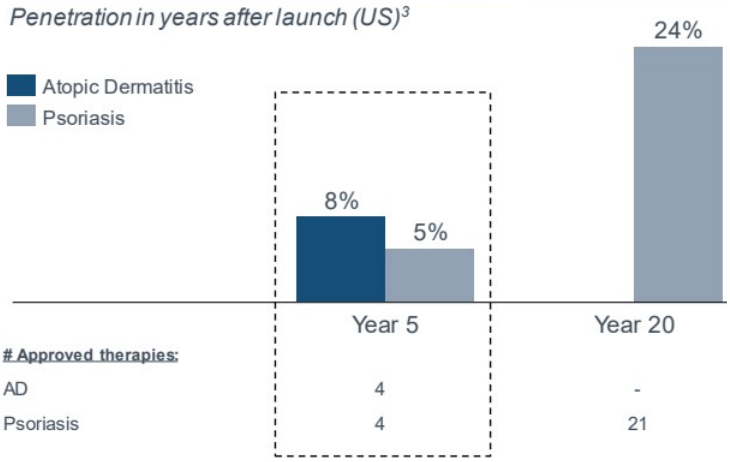
Population size, MM
Moderate or severe in 7 Major Markets[1], 2020



$30B+ market[2]

~3x larger patient population

Penetration of approved systemic therapy in AD expecte ramp 8% → 25%+ by 2032



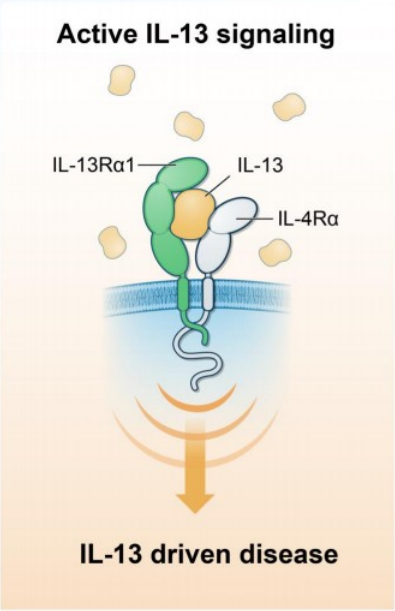
# Approved therapies:	Year 5	Year 20
AD	4	-
Psoriasis	4	21

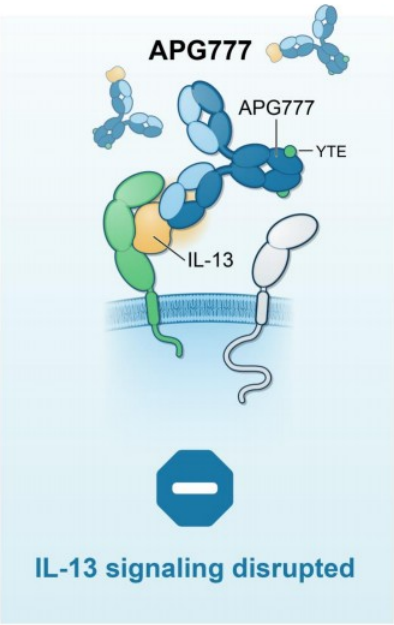
Psoriasis expected to be a $30B+ market; atopic dermatitis (AD) represents a larger opportunity

More convenient dosing could potentially expand AD biologics' penetration beyond projected 25%+

[1] The 7 Major Markets are US, Japan, Germany, France, Italy, Spain, and UK. [2] $30B+ projected market size in 2028 and beyond, including both biologics and small molecules. [3] EvaluatePharma and Clarivate. Source: Company 10-K filings, annual reports, press releases, analyst forecasts, GlobalData,

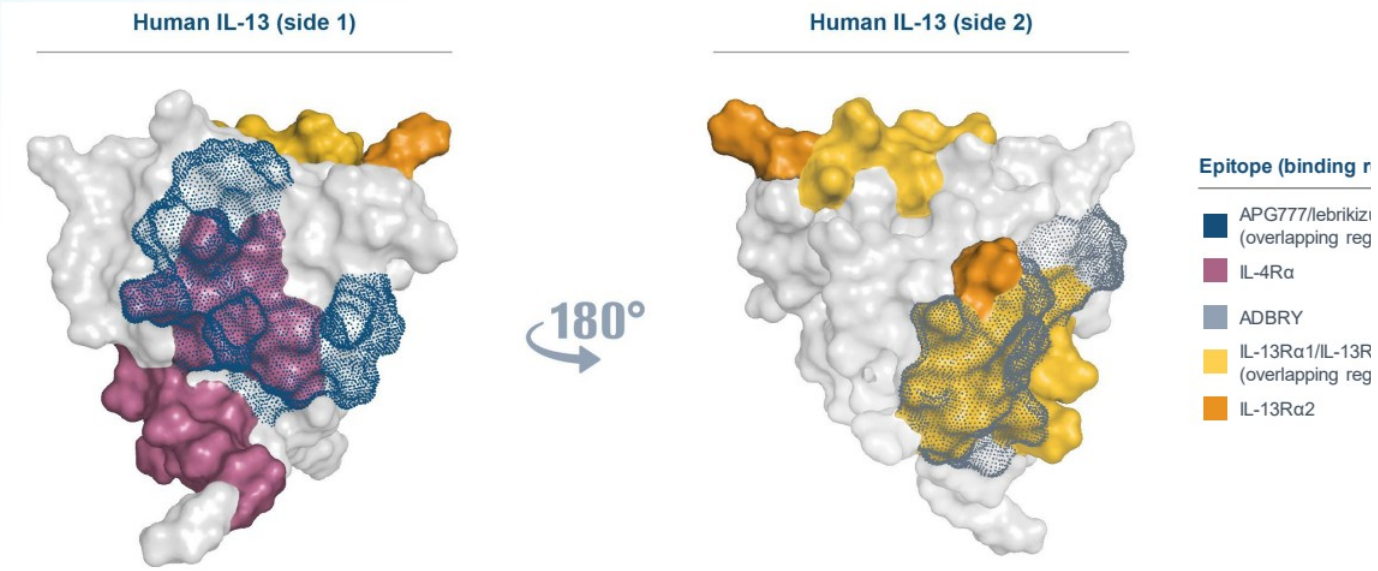
APG777 is designed to disrupt Th2 signaling by preventing formation of IL-13Rα1 / IL-4Rα heterodimer





- IL-13 signaling **begins with binding of IL-13 to IL-13Rα1**
- This forms an inactive complex that then **binds to IL-4Rα to create a complete, active heterodimer**
- **Active IL-13Rα1 / IL-4Rα heterodimer sets off a signaling cascade** that leads to:
  - Skin barrier defects
  - Immune cell recruitment
  - Tissue inflammation
  - Lichenification (skin thickening)
  - Pruritis (skin itching)

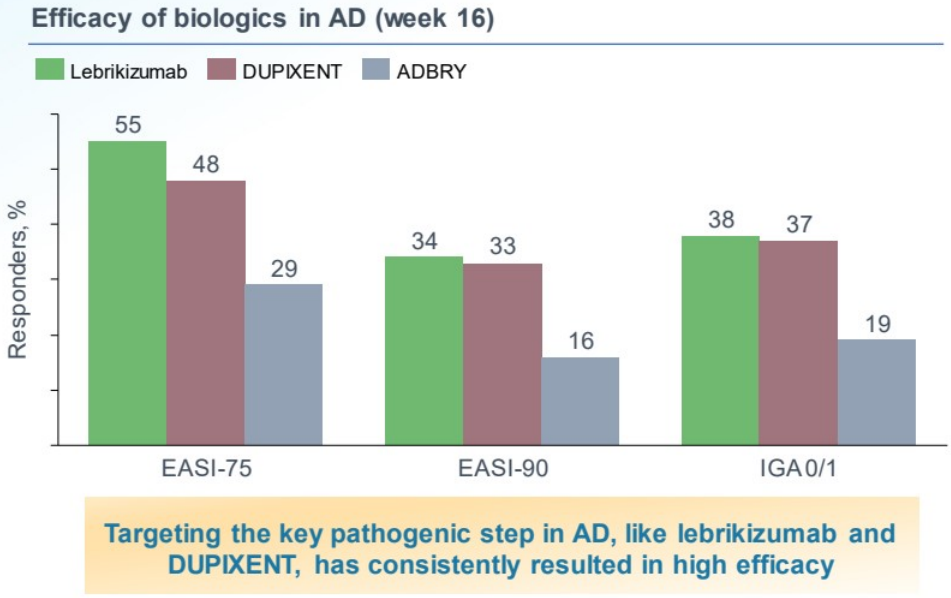
APG777's epitope overlaps with lebrikizumab, differentiating from other approaches to target IL-13



APG777's mechanism of action disrupts Th2 signaling by blocking IL-4Rα binding and subsequent formation of the IL-13Rα / IL-4Rα heterodimer

Note: IL-4Rα binding site derived from PDB 3BPO. Lebrikizumab epitope derived from PDB 4I77. ADBRY epitope derived from PDB 5L6Y.

Lebrikizumab and DUPIXENT have similar efficacy across key AD endpoints



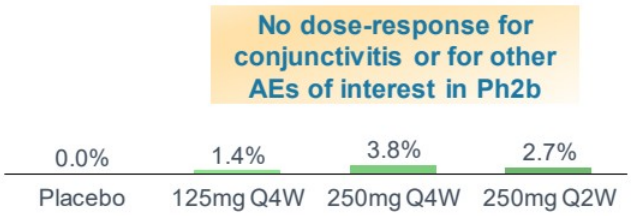
Targeting the key pathogenic step in AD, like lebrikizumab and DUPIXENT, has consistently resulted in high efficacy

- Lebrikizumab and DUPIXENT show **consistently high results across a important efficacy parameters**
  - Mechanistically, **both target the key pathogenic step in AD**, the heterodimerization of IL-4Rα and I 13R1, which may explain the simila efficacy observed
- However, both are **dosed every other week[4],** a burden for patients
- Lebrikizumab showed, at minimum, equivalent maintenance efficacy for both Q2W and Q4W dosing, a main differentiator from DUPIXENT

Source: [1] Lebrikizumab 250mg Q2W Ph3 Avg. Silverberg JI et al AAD 2022. [2] DUPIXENT 300 mg Q2W mono Ph3 Avg. DUPIXENT USPI. [3] ADBRY 300 mg Q2W mono Ph3 Avg. ADBRY USPI [4] In the 16-week induction phase
Note: 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. Only DUPIXENT and ADBRY are FDA approved.

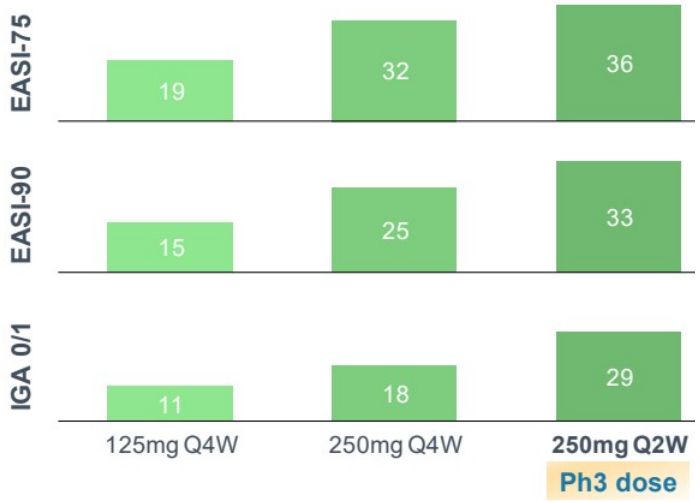
Lebrikizumab showed greater efficacy with higher doses i Ph2b with no dose-dependent increases in AE rates

Conjunctivitis rates by dose level in lebrikizumab Ph2b



No dose-response for conjunctivitis or for other AEs of interest in Ph2b

Response at 16 weeks (placebo-adjusted), % by dose lev in lebrikizumab Ph2b

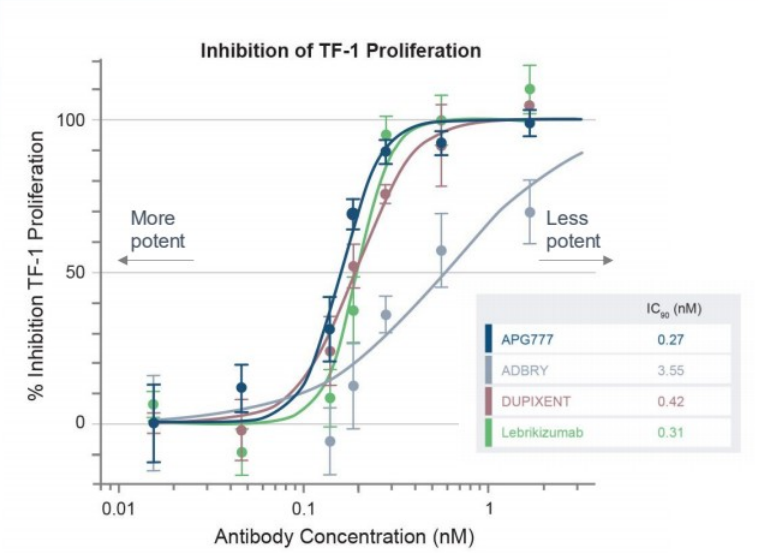


With no plateau in efficacy across doses, a higher dose and/or greater exposures could lead to better efficacy

Source: Guttman-Yassky , E, et al. JAMA Derm, 2020.
NOTE: Each regimen included one or more loading doses (LD): 125 mg every 4 weeks (250-mg LD), 250 mg every 4 weeks (500-mg LD), 250 mg every 2 weeks (500-mg LD at baseline and week 2).

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

APG777 vs DUPIXENT, ADBRY, and lebrikizumab on key potency assay



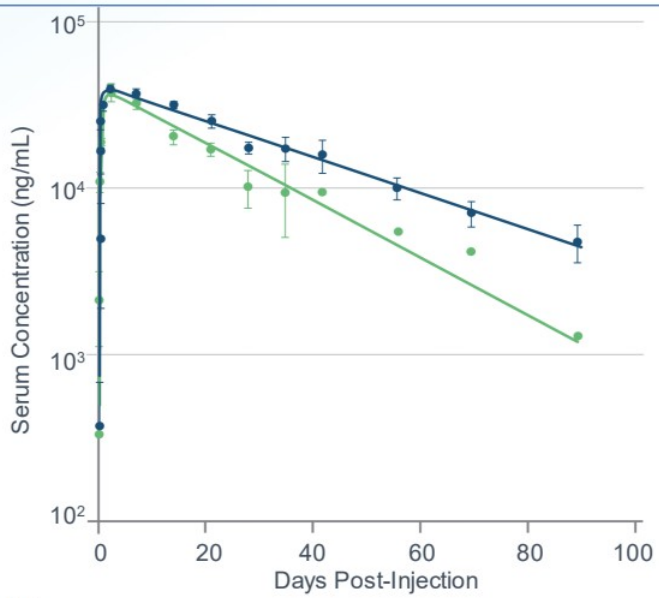
Additional *in vitro* assays support APG777 potenc

Assay	Affinity to human IL-13 by SPR	Inhibition of STAT-6 phosphorylation	Inhibi of TAI secre
Measurement	K_D (pM)	IC_{90} (nM)	IC_{90} (n
APG777	78	0.56	1.4
ADBRY	116	1.34	27.
DUPIXENT		0.58	13.
Lebrikizumab	131	0.46	1.3

Note: TF-1 is a human erythroblast cell line that proliferates in response to IL-13 and is a widely used functional immune assay

APG777 NHP half-life is significantly longer than lebrikizumab

NHP PK, SQ administration



APG777 has advantages over lebrikizumab in our NHP head-to-head studies

NHP average half-life[1]

APG777: 28 days

Lebrikizumab: 18 days

- APG777 shows extended half-life in NHPs
- APG777 had decreased PK variability with potential for greater consistency in response

APG777 can potentially achieve every 2- or 3-month maintenance dosing vs Q4W for lebrikizumab and Q2W for DUPIXENT

*Note: N = 3 per group. 2 of 3 animals in the lebrikizumab. SQ arm developed ADAs by day 40 and those timepoints associated with ADAs are excluded. [1]APG777 and lebrikizumab. half-lives were 27.6 days and 18.0 days, respectively, based on cumulative fit models across SQ and IV groups for each compound. For APG777, the average half-life based on individual fits for each animal was 28.2 days IV group and 27.0 days SQ group. For lebrikizumab, the average half-life based on individual fits for each animal was 18.1 days IV group and 13.5 days SQ group. SOURCE: Zhu E et al EADV 2023.

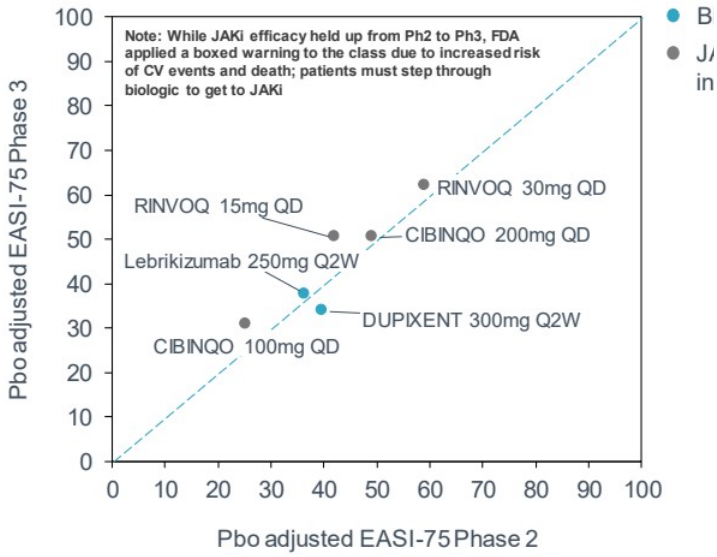
Strong historical correlation between Phase 2 and 3 data makes APG777 16-week AD data a key catalyst

Phase 2 16-week data in atopic dermatitis planned to readout in 2H 2025

Phase 2 objectives

- **2H 2025 POC readout:** % change from baseline in EASI at Week 16 powered >90% to detect effect
  - Induction regimen that exceeds lebrikizumab exposures by ~30%
- **Maintenance POC:** Study every 3- or every 6-month dosing in initial POC study to demonstrate the full potential of APG777 to reduce injection burden of patients
- **Phase 2b dose optimization:** examine range of regimens with exposures at, below, and above lebrikizumab

Strong correlation between Phase 2 and 3 results AD for validated endpoints EASI-75 and IGA 0/1



Source: Ph3 data for DUPIXENT, Rinvoq, Cibinqo, Adbry is from USPI. Thaci et al Lancet 2016 (DUPIXENT Ph2). Guttman-Yassky E et al JAMA Dermatol. 2020 (Lebrikizumab Ph2). Guttman-Yassky E et al J All Clin Immunol. 2020 (Rinvoq Ph2). Gooderham MJ et al JAMA Dermatol. 2019 (Cibinqo Ph2). [1]Exposure target based on C_{trough} in maintenance, the minimal concentration of APG777 to have similar exposures to lebrikizumab

Corporate

PRELIMINARY DATA

APG777 could substantially decrease annual maintenance injections for patients

APG777*	Lebrikizumab	DUPIXENT
2-4	13	26
INJECTIONS	**INJECTIONS**	**INJECTIONS**
ONE INJECTION EVERY 3- or 6- MONTHS	ONE INJECTION EVERY 4 WEEKS	ONE INJECTION EVERY OTHER WEEK

Additional injection relative to Q6M APG777

APOGEE THERAPEUTICS

NOTE: *Positioning of Apogee programs is illustrative and is based only on pre-clinical study results and Phase 1 interim clinical data. APG777 injections per year based on PK simulations. Maintenance dosin reflects injections per year after 12 or 16 week induction period. SOURCE: APG777 preliminary PK data on file. Lebrikizumab: EBGLYSS EMA SmPC. DUPIXENT USPI.


APG808

APG808 targets the same mechanism as DUPIXENT, which has been validated in COPD

COPD represents area of high unmet and a promising opportunity given recent positive DUPIXENT data

10%
of the global population >40 yrs

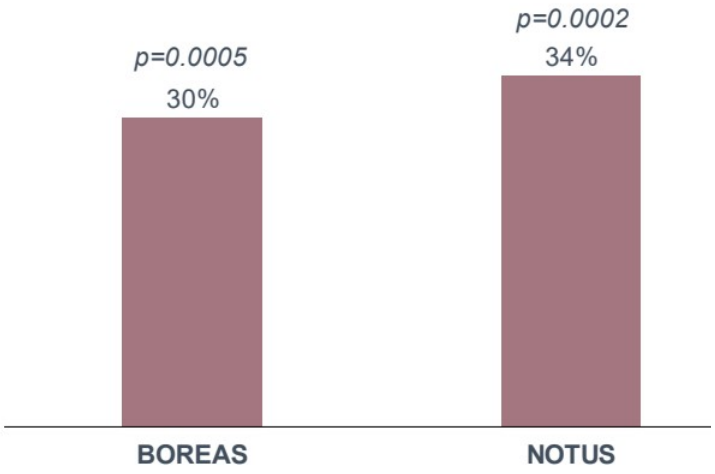
3rd
Leading cause of death in the US in 2019

150K+
People die each year in the US

No biologic therapies are approved for COPD, but **DUPIXENT demonstrated promise in two Phase 3s:**

- Significant, clinically meaningful **reduction in moderate or severe acute COPD exacerbations**
- **Improved lung function from baseline at 12 weeks** compared to placebo with separation from placebo as early as 2 weeks

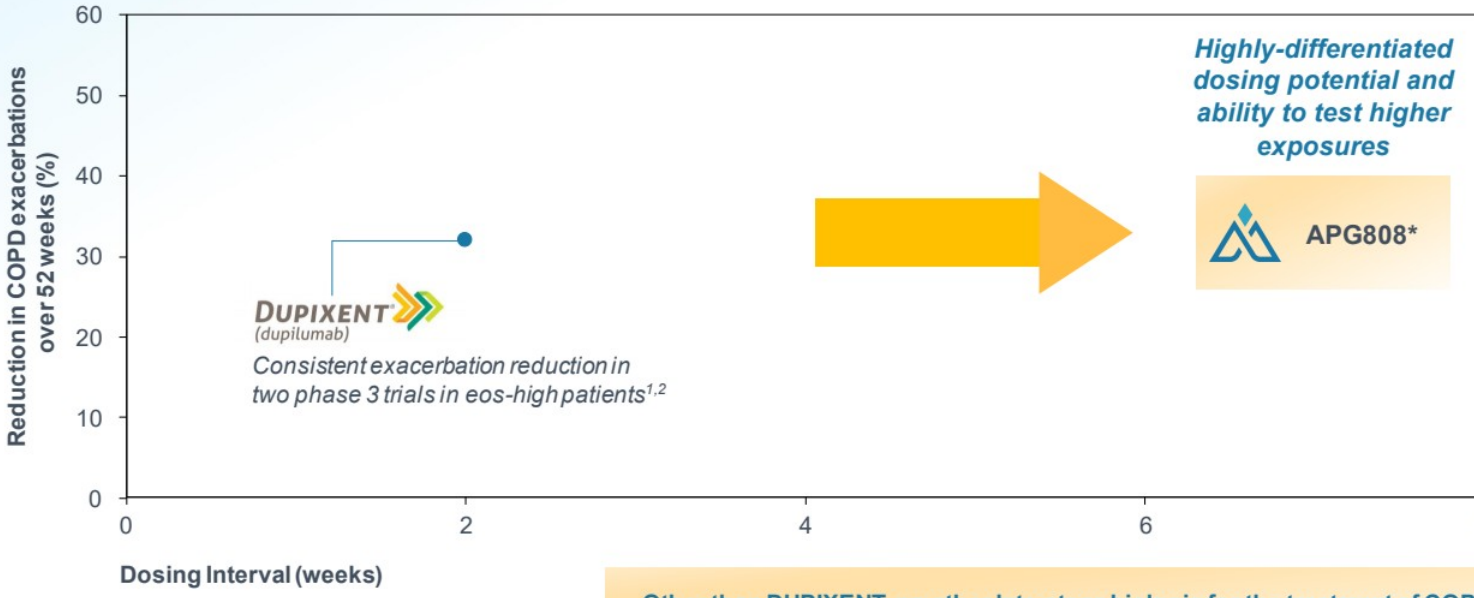
DUPIXENT produced a significant and clinically meanin reduction in exacerbations in two Phase 3 studies



 Source: Bhatt SP et al NEJM 2023; Sanofi press release November 26, 2023, interim analysis, full results not yet disclosed

APG808

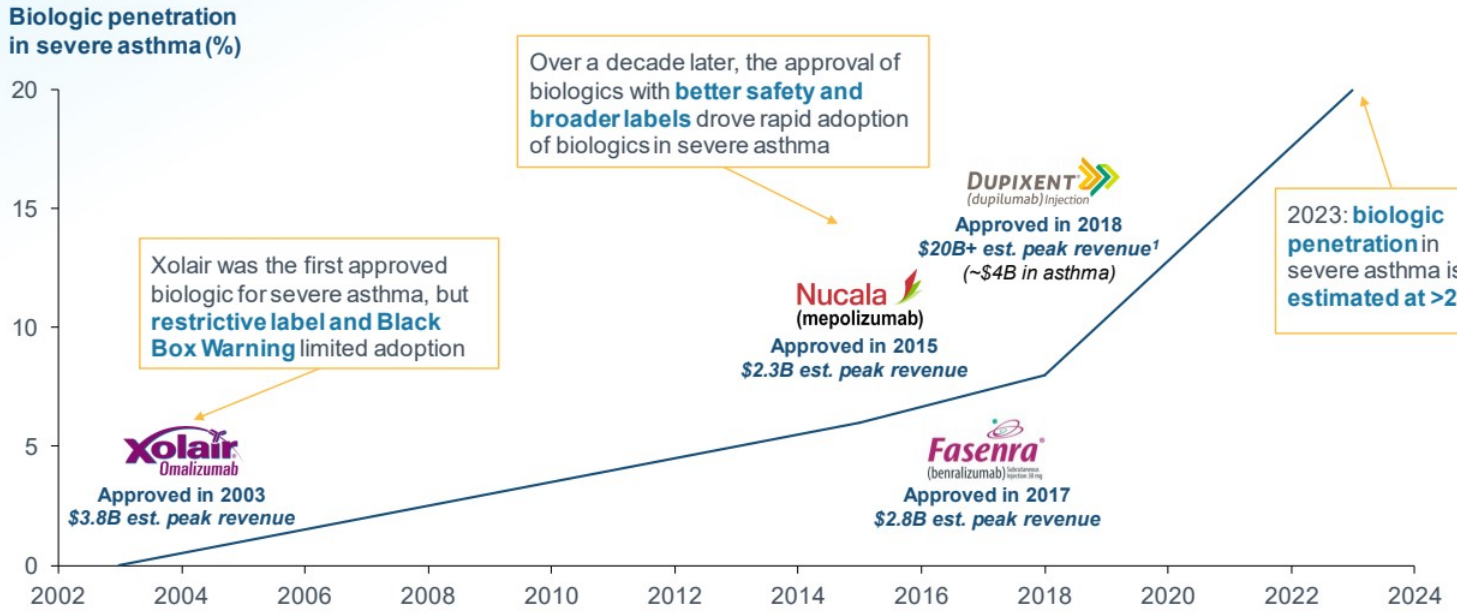
Treatments for moderate-severe COPD are limited



Other than DUPIXENT, no other late-stage biologic for the treatment of COP achieved its primary endpoint, leaving a vast unmet need for dosing beyon

NOTE: *Positioning of Apogee program is illustrative and not based on clinical trial data. Dupixent is not approved for the treatment of COPD
SOURCE: [1]Bhatt SP et al NEJM 2023; [2]*Sanofi press release November 26, 2023, interim analysis, full results not yet disclosed*

Asthma, an analog for COPD, shows how biologics can be rapidly adopted when they address unmet needs



1 Analysts estimate ~20% of DUPIXENT revenue is from asthma
Source: Company 10-K filings, annual reports, analyst forecasts, Citeline; EvaluatePharma

APG808

APG808 Phase 1 expected to initiate in 1H 2024 (ahead of schedule) with planned readout in 2H 2024

Trial design elements

Double-blind, placebo-controlled, first-in-human trial

Single ascending dose in healthy participants

N ~ 32

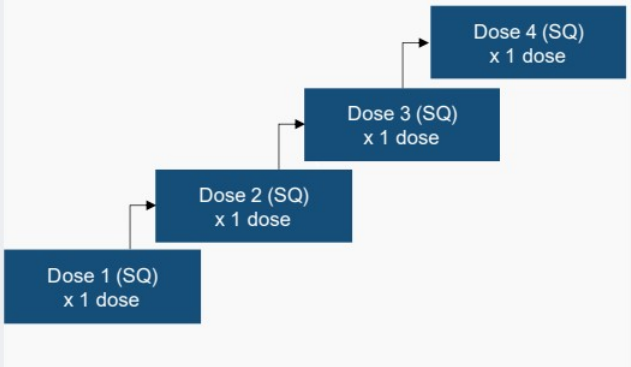
8 per cohort (6:2 active:placebo)

Key inclusion criteria: healthy adult participants

Primary endpoint: safety

Secondary endpoints: PK, ADA

Single ascending dose[1]



Asthma cohort[2]

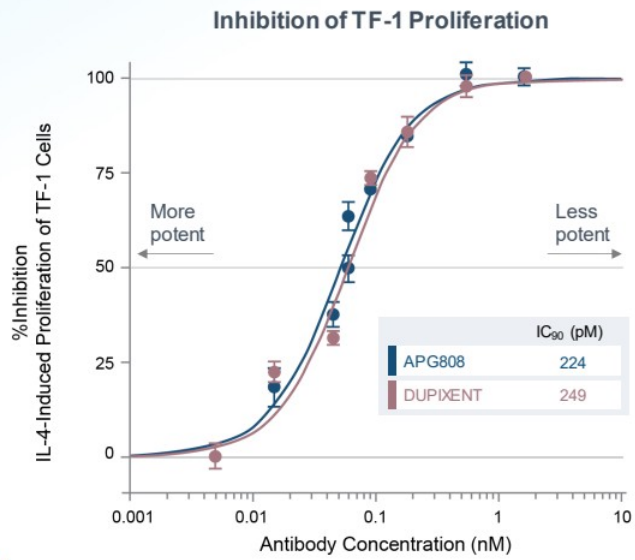
Dose or regimen TBD

2H 2024: Present APG808 safety and PK, including potentially extended half-life, optimized exposures, and low variability

[1] SAD dose ascension will ultimately be determined by the SRC (Scientific Review Committee) based on available data. [2] Design for the expansion portion of the study in asthma patients is to be finalized.

APG808 is as potent as DUPIXENT in key preclinical assays

APG808 vs DUPIXENT on key potency assay



Additional *in vitro* assays support APG808 potenc

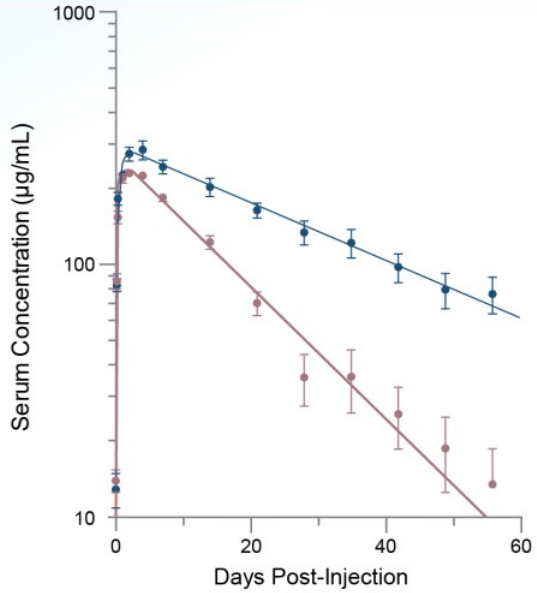
Assay	Affinity to human IL-4Rα[1,2]	Inhibition of STAT-6 phosphorylation	Inhibi of TA secre
Measurement	K_D (pM)	IC_{90} (nM)	IC_{90} (r
APG808	0.4	1.11	1.2
DUPIXENT	12	1.93	1.6

Additional preclinical assays demonstrate APG808 a DUPIXENT have an overlapping binding site on IL-4

NOTE: TF-1 is a human erythroblast cell line that proliferates in response to IL-4 induced stimulation and is widely used in functional immune assays. [1]Affinity to human IL-4Rα by determined by KinExA. [2]FDA MDR reports DUPIXENT affinity for human IL-4Rα for both monomeric (30 pM) and dimeric (12 pM) forms.

APG808 NHP half-life is significantly longer than DUPIXENT

NHP PK, SQ administration



APG808 has advantages over DUPIXENT in our NHP head-to-head studies

NHP average half-life[1]

APG808: ~26 days

DUPIXENT: ~12 days

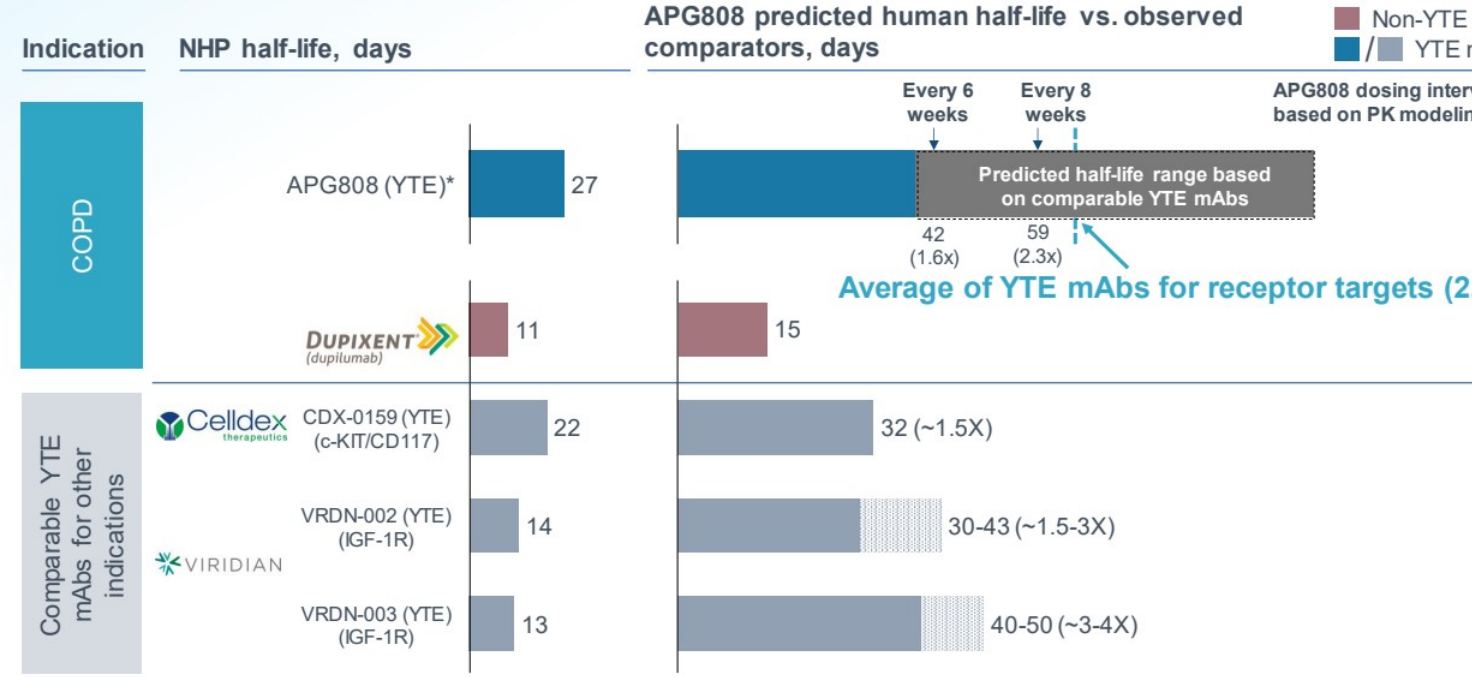
APG808 showed extended half-life in NHPs

- APG808 also showed decreased variability on PK and potential for greater consistency in response

APG808 can potentially achieve 6- or 8-week dosing vs Q2W for DUPIXENT

*NOTE: N = 5 animals per group. 3 of 5 animals in the dupilumab SQ arm developed ADAs by day 35 and those timepoints associated with ADAs are excluded from the half-life estimate based on data through day 56. [1]APG808 and dupilumab preliminary SQ half-lives for 25 mg/kg were 26.4 days and 11.5 days, respectively, based on cumulative fit models across SQ groups for each compound using interim data through day 56. The NHP PK study is ongoing with final data expected out to day 91. Final NHP half-life estimates and PK parameters will be calculated based on full dataset out to day 91.

APG808 NHP half-life suggests potential for significant improvement over DUPIXENT in humans

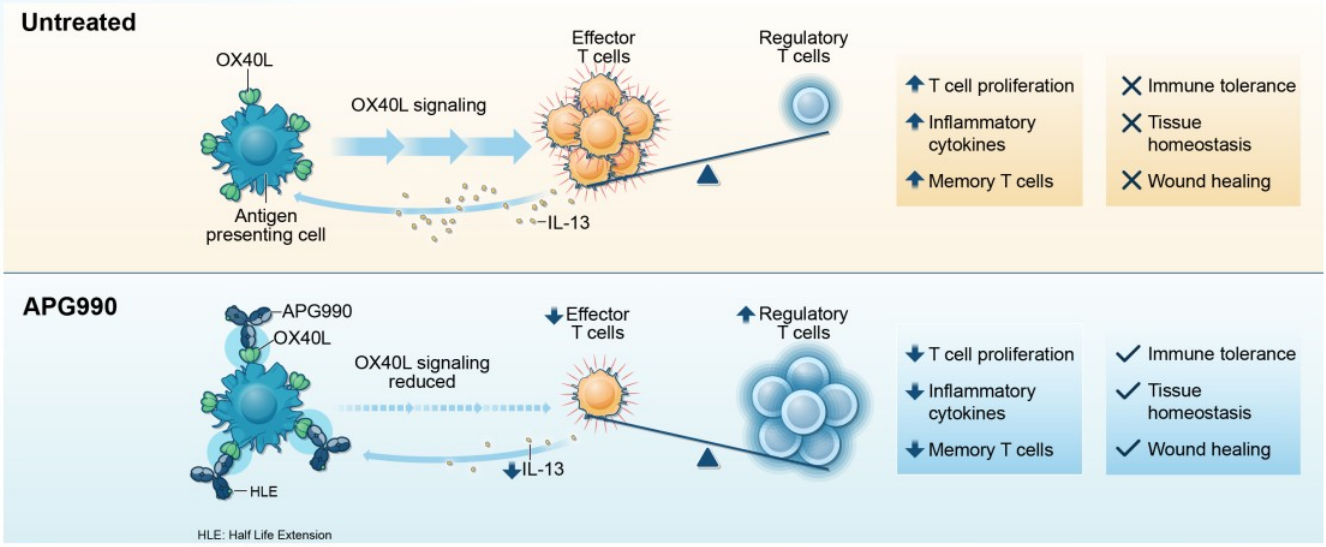


APOGEE THERAPEUTICS

NOTE: Half-lives as reported in studies conducted by the sponsor of each of these product candidates or in the label of approved products. Half-lives are not based on head-to-head studies and are derived from different studies at different points in time, with differences in study design. As a result, cross-study comparisons cannot be made. [1]Based on steady state PK simulations made with parameters for APG808 identical to Dupixent except changes in $k_{elimination}$. *Positioning of Apogee program is illustrative and not based on clinical trial data and is based only on pre-clinical study results


APG990/APG222

APG990 blocks OX40L and potentially rebalances the immune system



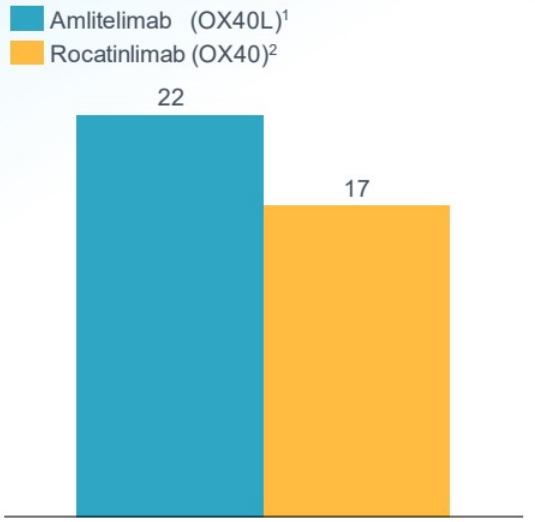
OX40L blockade targets Th2, Th17, and Th22 pathways, which have been implicated in numerous I&I conditions

Upcoming clinical trial readouts could provide PoC for OX40L beyond AD including asthma, hidradenitis suppurati alopecia areata, celiac disease, and systemic sclerosis

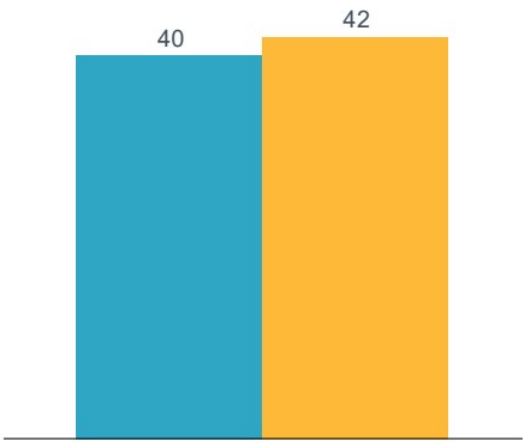
APG990

OX40L and OX40 inhibition have shown similar efficacy, but OX40L has a clear advantage on safety

IGA 0/1 response at Week 16



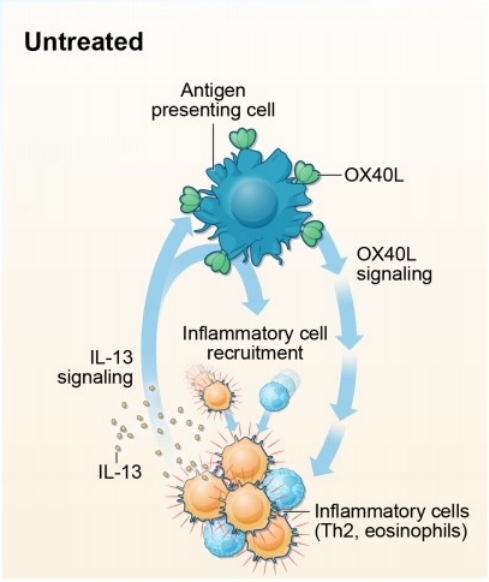
EASI-75 at Week 16

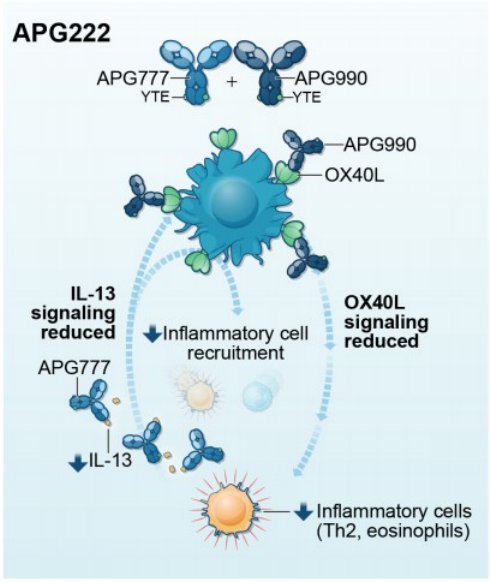


- In Phase 2b, **rocatinlimab (OX40)** **associated with pyre** (17% of patients) **and chills** (11% of patients
- In contrast, **no pyrexia or chills for amlitelim (OX40L)** in Phase 2b

Source: [1]Amelitelimab 250mg Q4W Weidinger et al. EADV oral presentation (2023). [2]Rocatinlimab avg. of 150mg Q4W and 600mg Q4W *Guttman-Yassky E et al. Lancet (2023).* [3]EADV presentation stated no pyrexia *"within 72 hours of injection"*.
Note: 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.

APG222 combines two validated mechanisms and may enhance benefit in AD and other I&I indications





- **OX40L treatment reduces circulating IL-13 levels** supporting the potential for synergy with IL-13 blocker
- Combination potentially enables wider subset of patients to achieve **deeper clinical responses** and **durable remission in AD other I&I indications**

Given strong mechanistic rationale, APG222 program explore combination poten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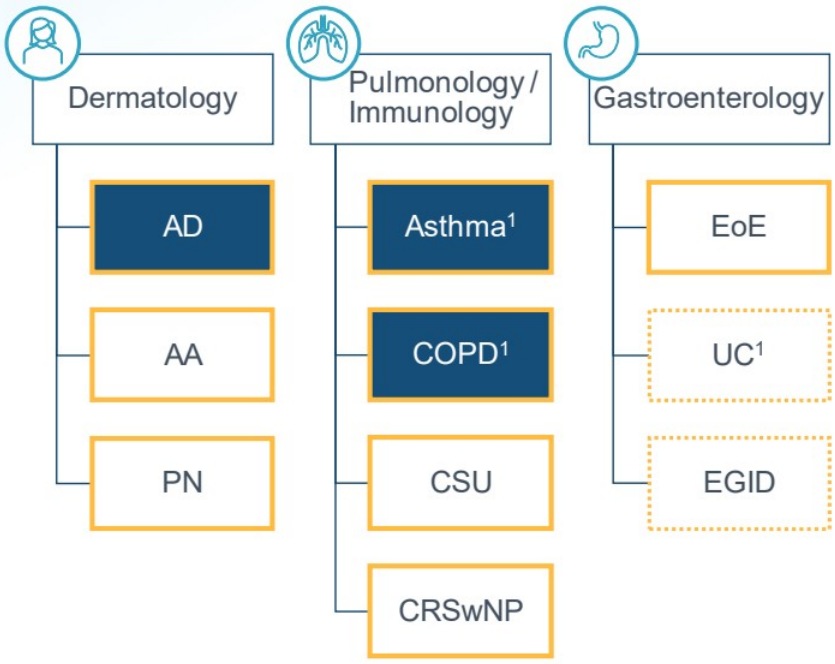
Our programs have broad potential to disrupt the I&I space



Planned Apogee Ph2 based o established mechanism

3rd party clinical data supporti or more Apogee targets

Ongoing 3rd party clinical trials could validate one or more Ap targets

Note: 1 Eosinophilic subtypes
AA = Alopecia Areata. PN = Prurigo Nodularis. CSU = Chronic Spontaneous Urticaria. CRSwNP = Chronic Rhinosinusitis with Nasal Polyps. EoE = Eosinophilic esophagitis. UC = Ulcerative Colitis. EGID = Eosinophilic Gastrointestinal Disorders (non-EoE)


Apogee /ˈapəjē/ *noun*
The highest point in the development of something; a climax or culmination