

CORPORATE OVERVIEW

March 2024



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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, objectives, goals, strategies and future events, the efficacy, safety, tolerability, PK and PD profile of APG777, the potential dosing 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 our 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, APG808, 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 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 f

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Apogee plans to reshape the current standard of care for I&I diseases





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

APPROACH

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

从 EXPANSION

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

PIPELINE

Portfolio 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 Iebrikizumab		Atopic Dermatitis		1H 2024: Phase 2 t 2H 2025: 16-week	rial initiation ¹ proof-of-concept data
		Asthma		2025: Phase 2 trial	initiation ¹
APG808 IL-4Rα Same MOA as DUPIXENT		COPD		24: Initial Phase 1 PK a	-
		Asthma	1H 202	5: Proof-of-concept da	ata
APG990 OX40L Same MOA as amlitelimab	Atopic Dermatitis	2024: Candidate no 2025: Phase 1 initia			
APG222 Combination IL-13 and OX40L	Atopic Dermatitis				

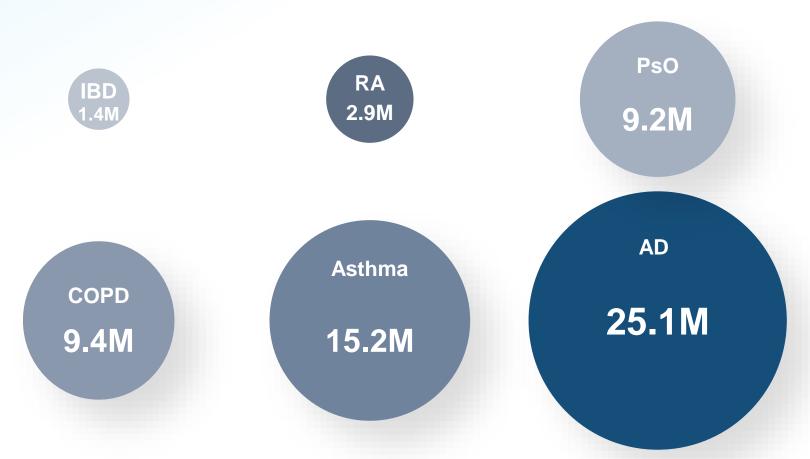


Apogee is pursuing the largest I&I markets with a highly de-risked approach and AD is the largest



Estimated population size, MM

Moderate or severe in 7 Major Markets¹



- Psoriasis expected to be a \$30B+ market; atopic dermatitis (AD) represents a larger opportunity expected to grow to \$50B+ based on ~3x larger patient population
- AD biologics penetration is outpacing early years of psoriasis biologics (8% vs 5% at 5 years)
- AD market is projected to grow more than any other I&I market



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

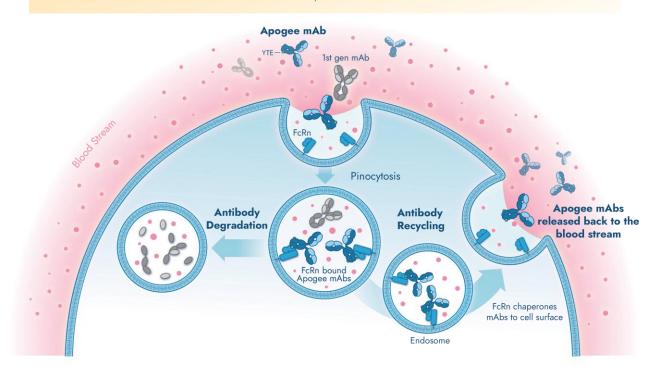


Viscosity

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

Potential for PK that:

- Optimizes exposures
- Decreases variability
- Increases half-life





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APG777

APG777's best-in-class Phase 1 PK profile shows potential to be a leading product in the expected \$50B+ AD market¹





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 $(K_D \le 100 \text{ pM})^2$
- ~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
- 75-day half-life (3x lebrikizumab)

Favorable product characteristics and COGS

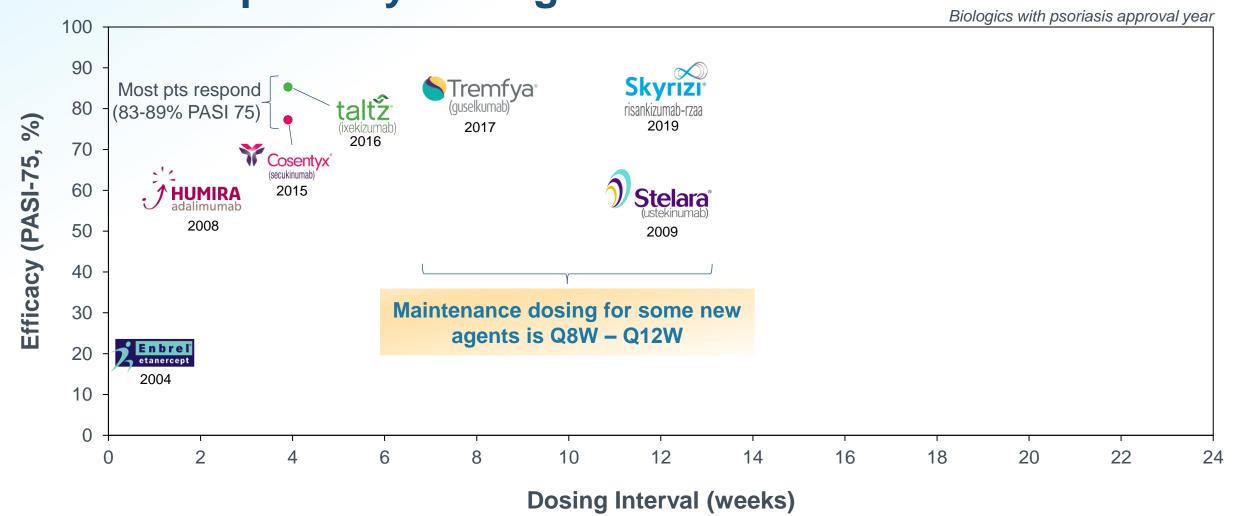
- As few as 2-4 doses per year in maintenance
- Expected improved formulation, manufacturability and viscosity

Novel IP into mid-2040s



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

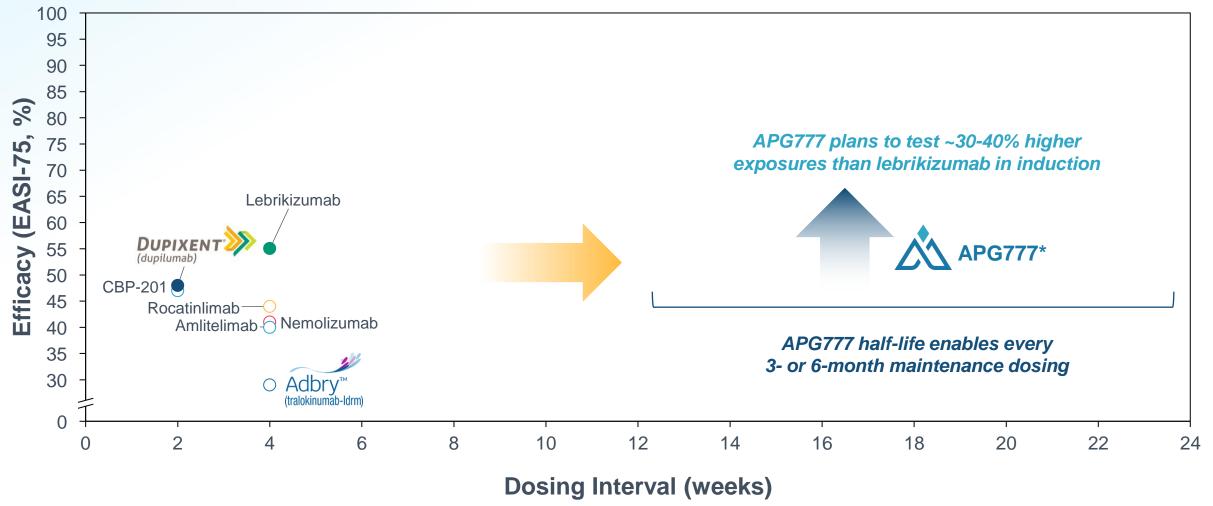






Apogee plans to advance APG777 into a Phase 2 trial with 3- or 6-month maintenance dosing





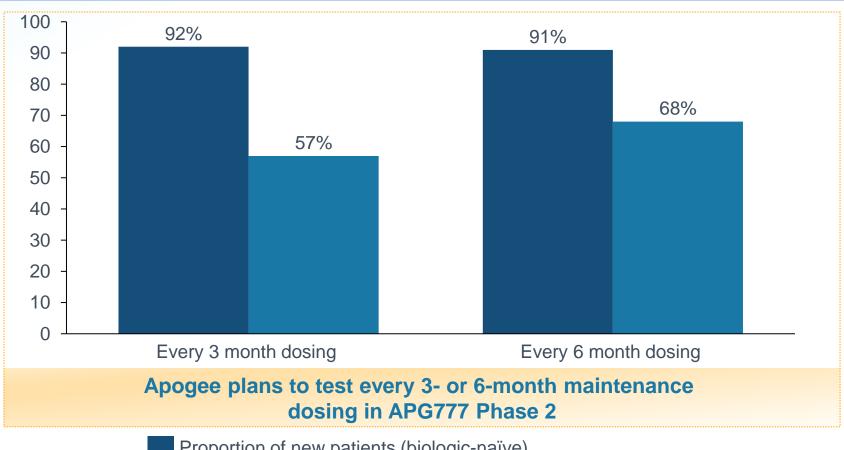


Dermatologists view every 3- or 6-month dosing as highly differentiated



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)



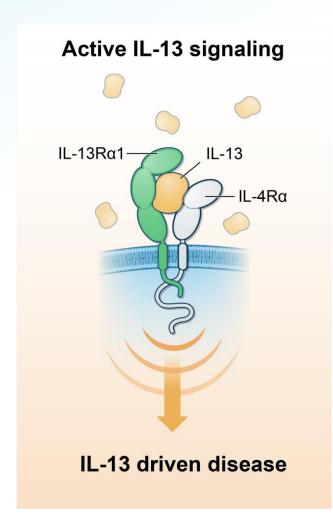


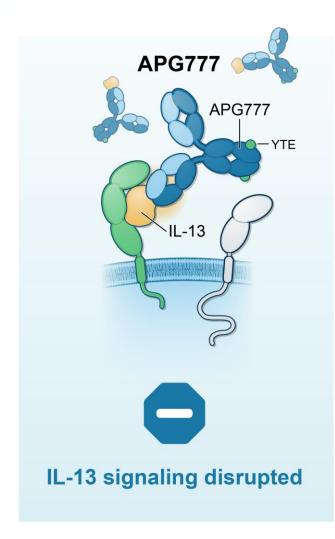
Proportion of switch patients (currently/formerly on a biologic)



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







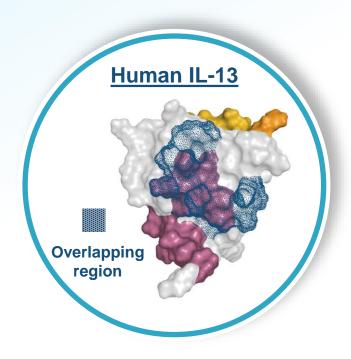
- IL-13 signaling begins with binding of IL-13 to IL-13Rα1
- This forms an inactive complex that then binds to IL-4Rα to create a complete, active heterodimer
- Active IL-13Rα1 / IL-4Rα
 heterodimer sets off a signaling
 cascade that leads to:
 - Skin barrier defects
 - Immune cell recruitment
 - Tissue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

APOGEE

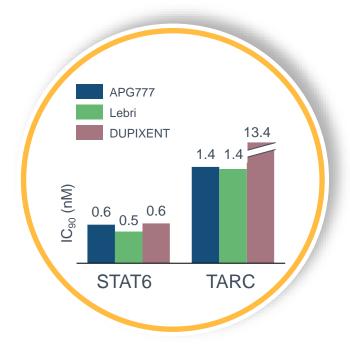
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APG777 leverages lebrikizumab's mechanism to deliver a potentially best-in-class, pipeline in a product antibody

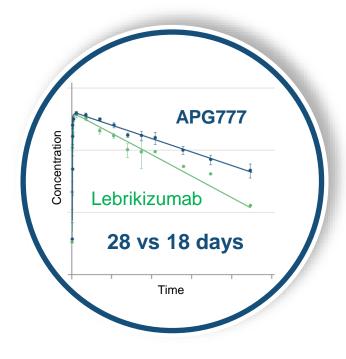




APG777's epitope on IL-13 overlaps with lebrikizumab's and leverages proven MoA and biology



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



APG777 NHP half-life is significantly longer than lebrikizumab



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APG777 Phase 1 in Healthy Volunteers

APG777 Phase 1 initial data exceeded all trial objectives

Establish safety & PK profile

Well-tolerated with at least 33-day half-life

- Half life of ~75 days
- Doses up to 1200mg tested and welltolerated
- Initial multiple-dose data consistent with PK & safety profile from SAD cohorts

Set Ph2 induction regimen

Achieve at least equiv. exposures to lebrikizumab with same or fewer injections

- 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)

Set Ph2 maintenance regimens

Equal lebrikizumab exposure with every 2month or longer dosing¹

• 3- or 6- month maintenance dosing enabled with modeled exposures similar to or greater than lebrikizumab Supplemental

Demonstrate effect on biomarkers pSTAT6 or TARC

Extended PD effect on both pSTAT6 and TARC for ~3 months with follow-up ongoing



Exceeded



Exceeded



Exceeded



Exceeded



APG777 interim data from ongoing Phase 1 trial in healthy volunteers



Trial design elements

Double-blind, placebo-controlled, first-inhuman trial

Single ascending dose component with a nested multiple ascending dose component

N = 40

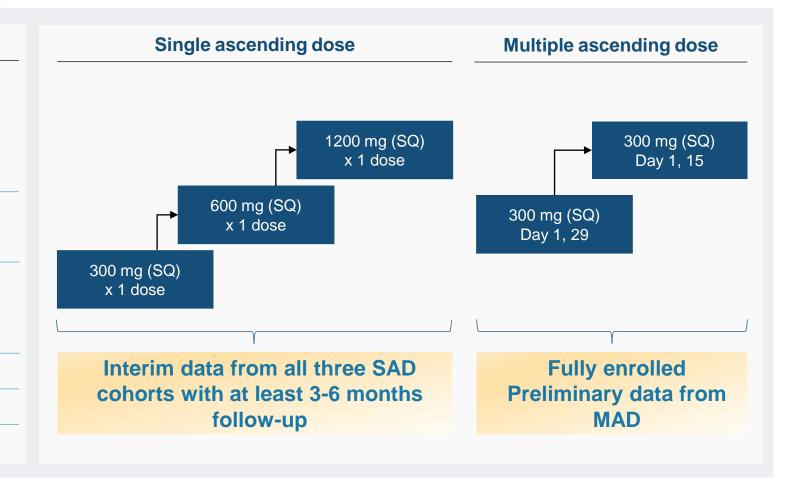
8 per cohort (6:2 active:placebo)

Key inclusion criteria: healthy adult participants

Primary endpoint: safety

Secondary endpoints: PK, ADA

Exploratory biomarkers: pSTAT6, TARC





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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	Placebo N=10
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 (70.0%)
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 (30.0%)
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



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

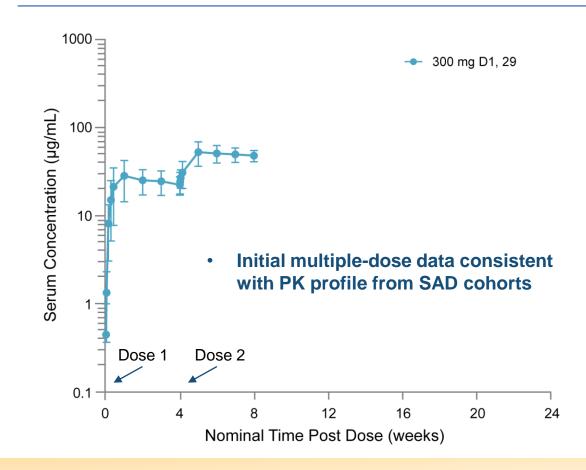


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Single-dose concentration-time profile

1000 -Cohort 1 300 mg Cohort 2 600 mg Cohort 3 1200 mg Serum Concentration (µg/mL) Slow clearance, resulting in half-life of ~75 days **Dose-proportional increases in both Cmax and AUC** 12 16 20 24 Nominal Time Post Dose (weeks)

Multi-dose concentration-time profile



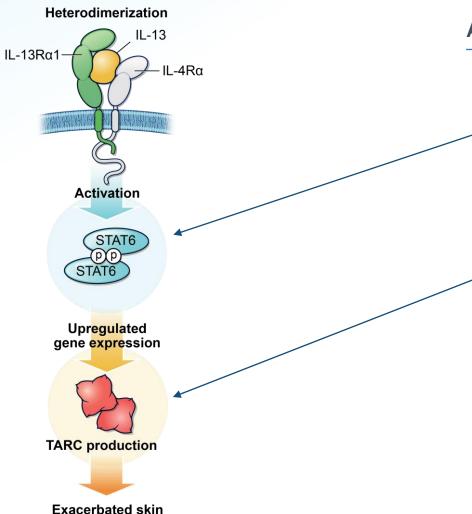
PK demonstrated dose-proportionality and half-life of ~75 days (approximately 3x lebrikizumab)



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





barrier dysfunction

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

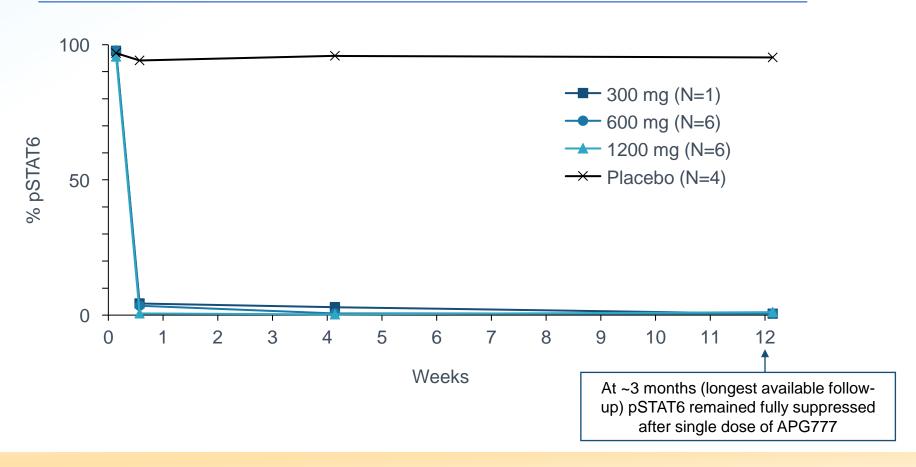
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Single dose APG777 showed near complete pSTAT6 inhibition for ~3 months (limit of available follow-up)



Median percent change from baseline in pSTAT6



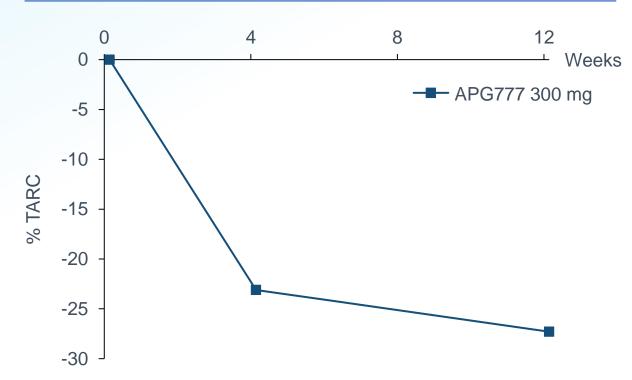
100% pSTAT6 inhibition was demonstrated for approximately 3 months across all doses



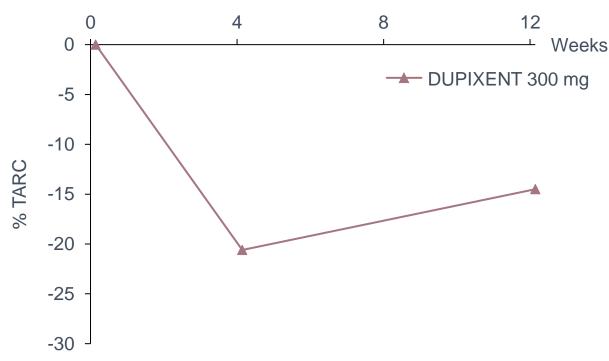
Single dose of APG777 led to deep + sustained TARC inhibition for ~3 months (limit of available follow-up)



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)



APG777 Phase 2 in Atopic Dermatitis

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 A anticipated in 2H 2025

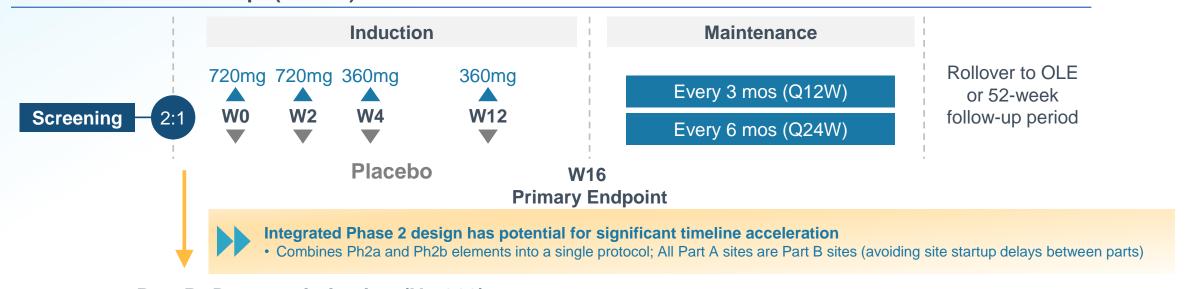


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Planned integrated Phase 2 expected to have 16week topline data in 2H'25



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



Part B: Dose optimization (N ~360)

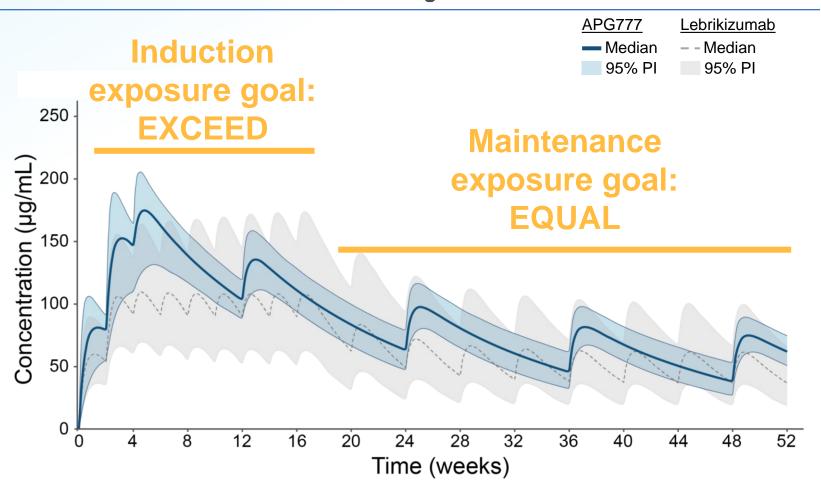


Primary Endpoint

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



Modeled induction and maintenance dosing for APG777¹ and lebrikizumab



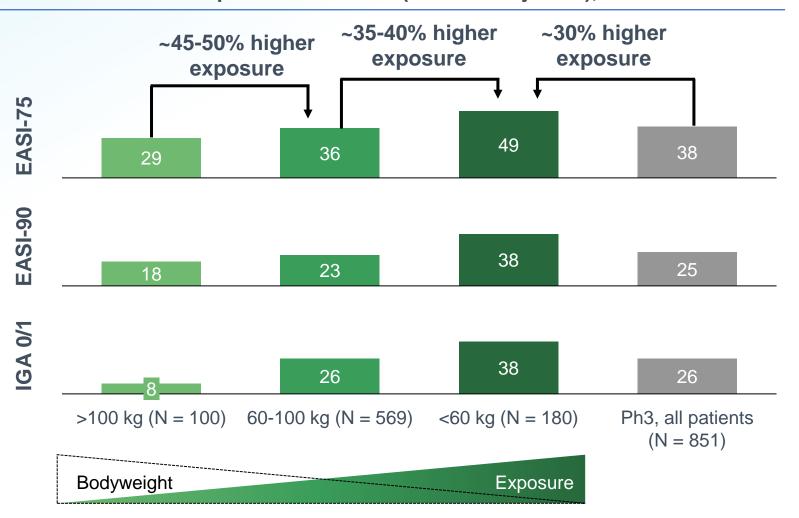
- Lebrikizumab data suggests an exposure-response (E-R) for efficacy in induction that underpins our goal to EXCEED lebrikizumab induction exposures
- There was no E-R observed in maintenance for lebrikizumab; our aim is to EQUAL its exposure in maintenance



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), %



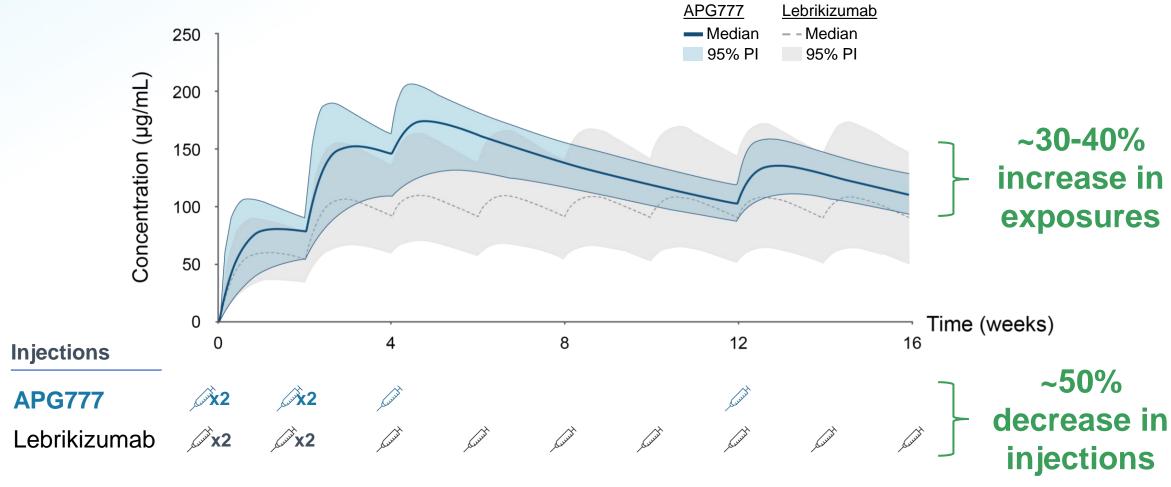
- Lebrikizumab exposure and induction efficacy are both inversely correlated with body weight
- Relationships suggest an exposure-response for efficacy in induction and support testing higher exposures with APG777
- In lebrikizumab Ph2b and Ph3 there has been no dose-AE or exposure-AE relationship
-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



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



Modeled induction dosing for APG777 and lebrikizumab





Modeled Phase 2 Q3M maintenance exposures <u>equal</u> those of lebrikizumab

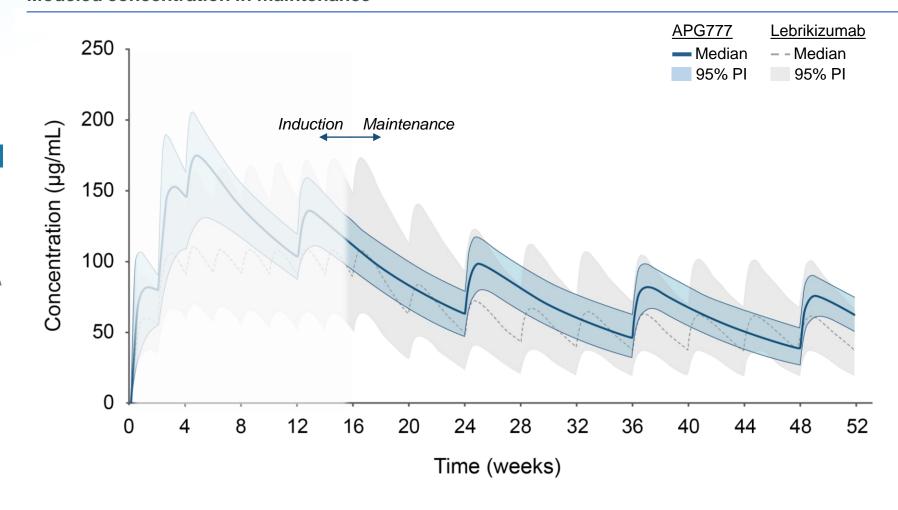


Modeled concentration in maintenance

APG777 Q3M

Aiming for annual maintenance injections:

4 vs 13-26 for lebrikizumab/





Modeled Phase 2 Q6M maintenance exposures <u>equal</u> those of lebrikizumab

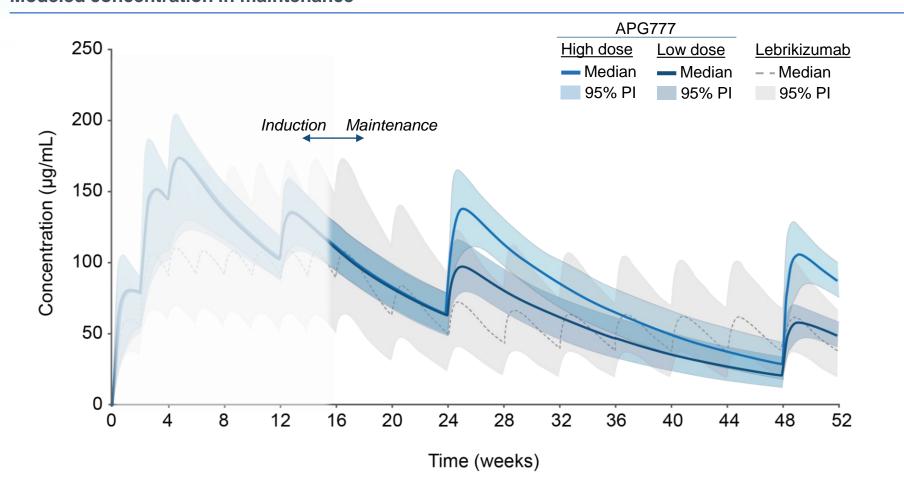


Modeled concentration in maintenance

APG777 Q6M

Aiming for annual maintenance injections:

2 vs 13-26 for lebrikizumab/





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

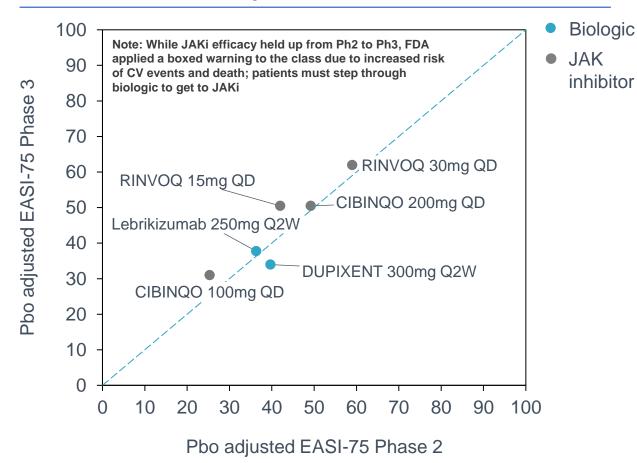


Phase 2 16-week data in atopic dermatitis planned to readout in 2H 2025

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 6month 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 in AD for validated endpoints EASI-75 and IGA 0/1





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

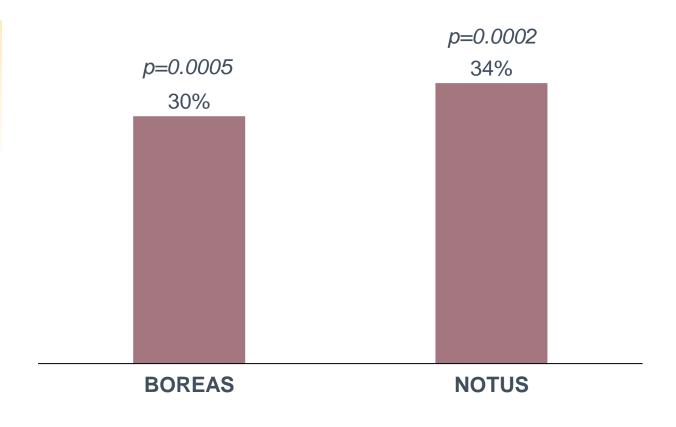
150K+

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

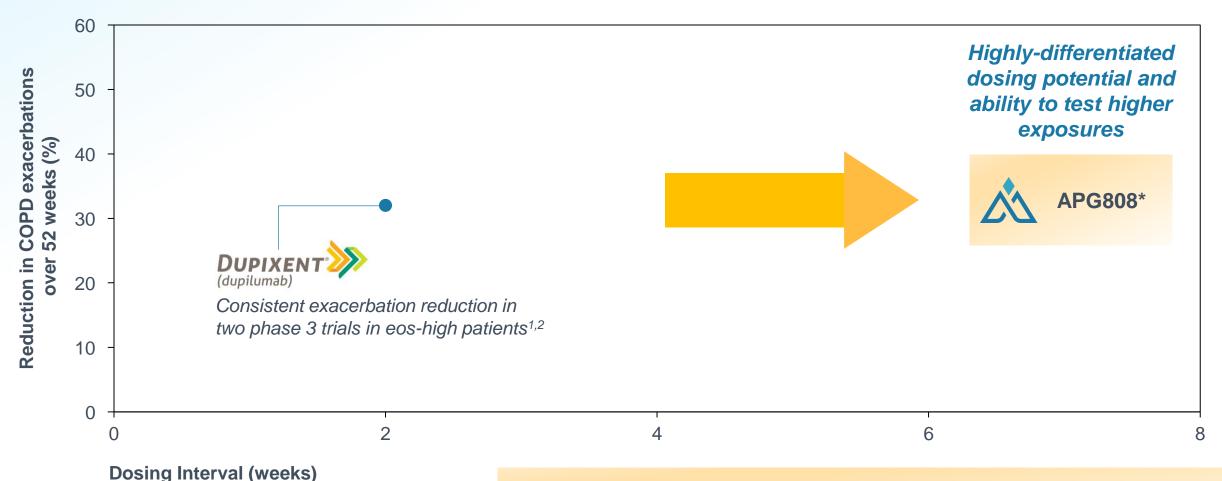
DUPIXENT produced a significant and clinically meaningful reduction in exacerbations in two Phase 3 studies





Treatments for moderate-severe COPD are limited

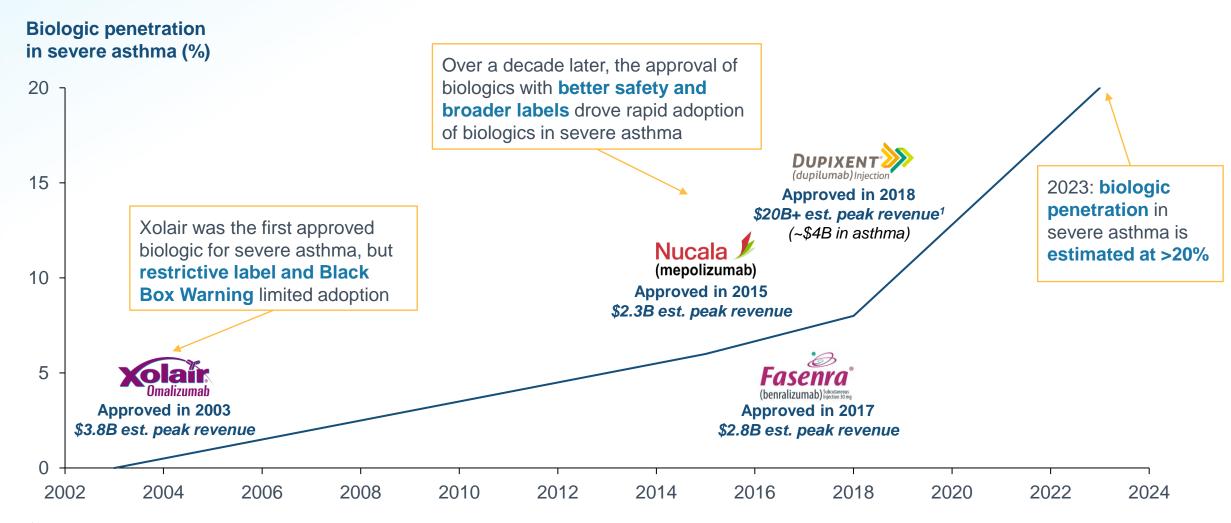




Other than DUPIXENT, no other late-stage biologic for the treatment of COPD has achieved its primary endpoint, leaving a vast unmet need for dosing beyond Q2W



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





APG808 Phase 1 initiated in 1H 2024 (ahead of schedule) with planned readout in 2H 2024



Trial design elements

Double-blind, placebo-controlled, first-inhuman trial

Single ascending dose in healthy participants

 $N \sim 32$

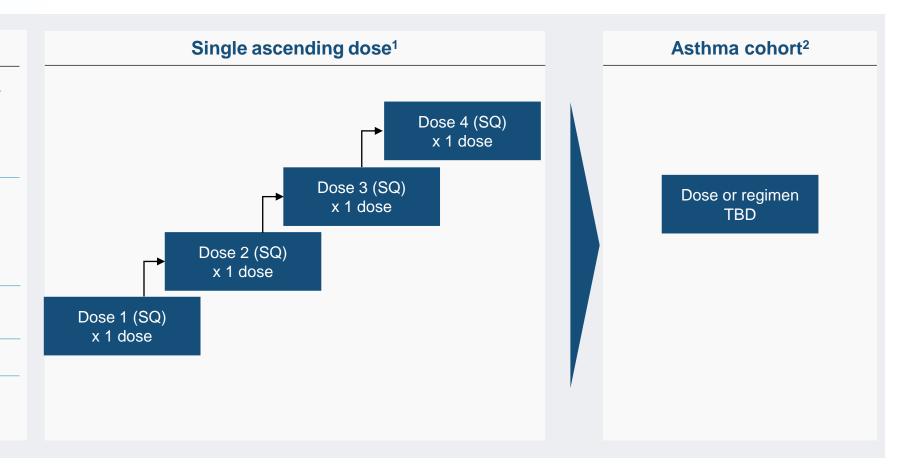
8 per cohort (6:2 active:placebo)

Key inclusion criteria: healthy adult

participants

Primary endpoint: safety

Secondary endpoints: PK, ADA





APG808 Phase 1 clinical trial objectives



OBJECTIVES

Confirm tolerable safety profile

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

Determine dosing regimens to sustain exposures similar to DUPIXENT

2H 2024: confirm potential for best-in-class dosing intervals



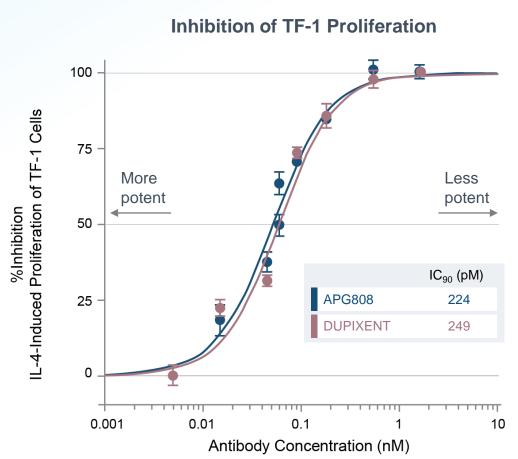
Dosing Goal: every 6- or 8-weeks (vs. every 2 weeks for DUPIXENT¹)



APG808 is as potent as DUPIXENT in key preclinical assays



APG808 vs DUPIXENT on key potency assay



Additional in vitro assays support APG808 potency

Assay	Affinity to human IL- $4R\alpha^{1,2}$	Inhibition of STAT-6 phosphorylation	Inhibition of TARC secretion	
Measurement	K _D (pM)	IC ₉₀ (nM)	IC ₉₀ (nM)	
APG808	0.4	1.11	1.25	
DUPIXENT	12	1.93	1.67	

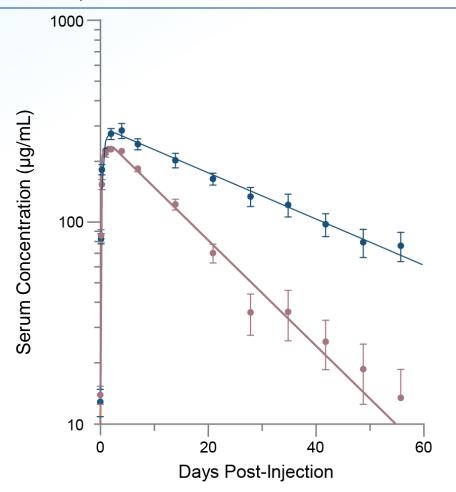
Additional preclinical assays demonstrate APG808 and DUPIXENT have an overlapping binding site on IL-4R α



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-life¹

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

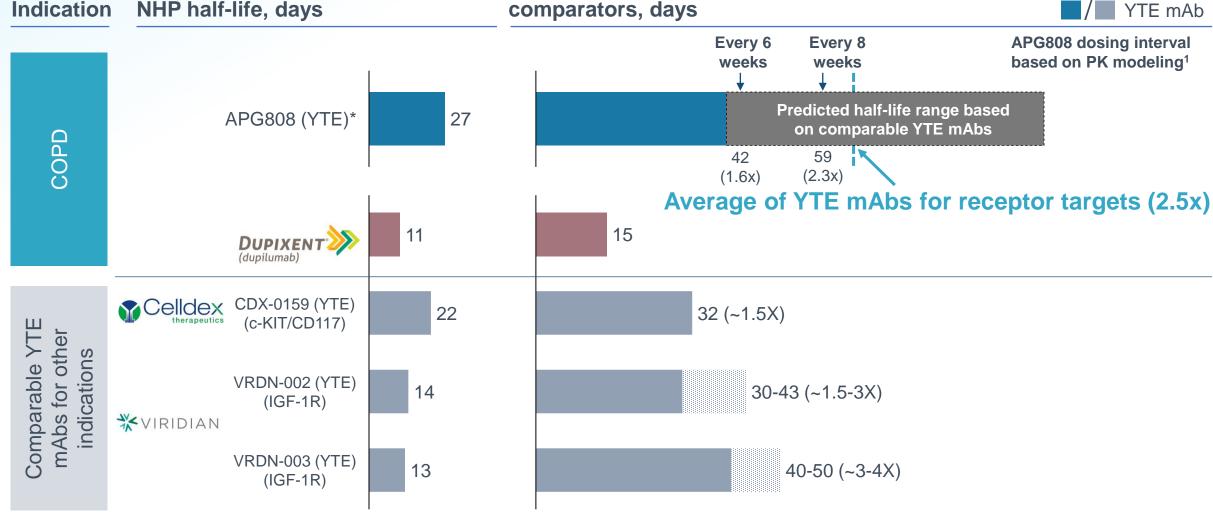


APG808 NHP half-life suggests potential for significant improvement over DUPIXENT in humans



APG808 predicted human half-life vs. observed comparators, days



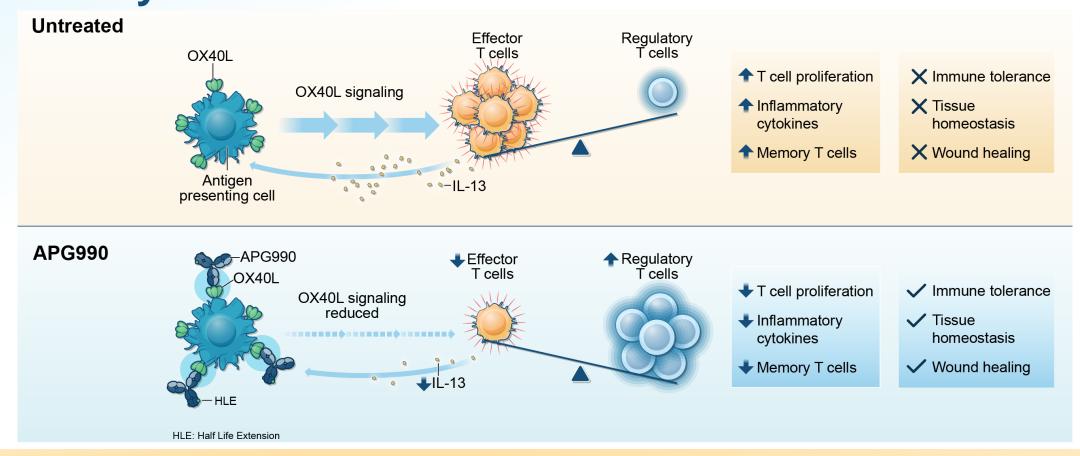




APG990/APG222

APG990 blocks OX40L and potentially rebalances the immune system





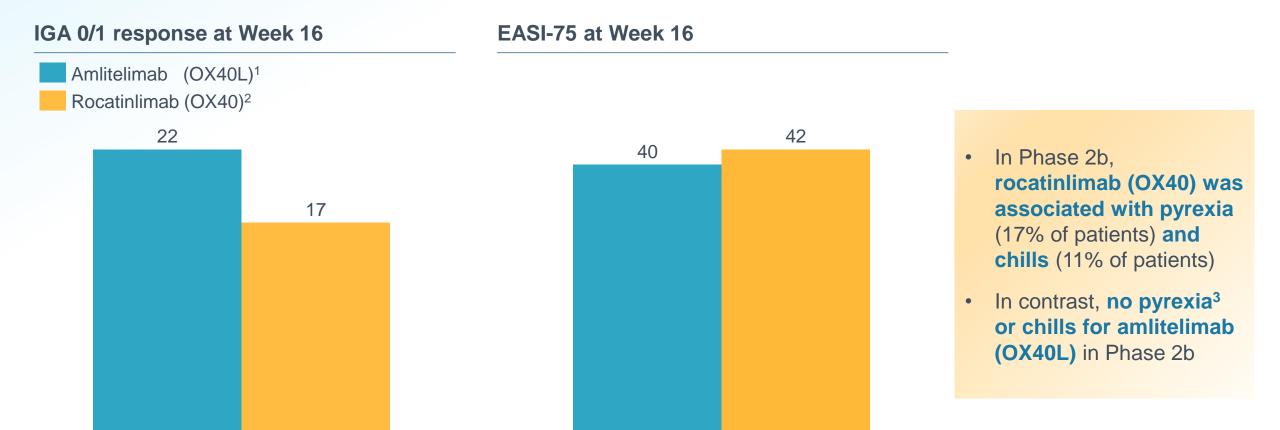
OX40L blockade targets Th2, Th17, and Th22 pathways, which have been implicated in numerous I&I conditions

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



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

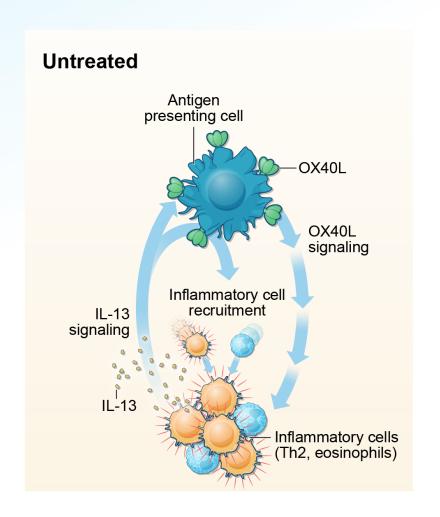


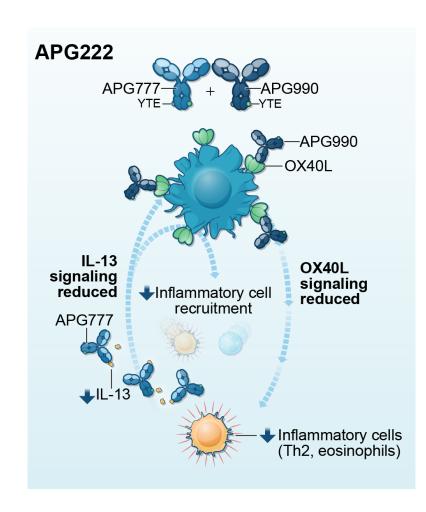




APG222 combines two validated mechanisms and may enhance benefit in AD and other I&I indications







- 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 and other I&I indications

Given strong mechanistic rationale, APG222 program will explore combination potential





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 Technical Officer







Matt Batters, JD General Counsel





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, commercial and management expertise





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Michael Henderson, MD CEO, Apogee Therapeutics









Jennifer Fox CFO & CBO, Zenas **BioPharma**







Andrew Gottesdiener, MD Venrock





Peter Harwin Managing Member, Fairmount

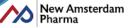
BAUSCH-Health Johnson Johnson







BJ Jones CCO. NewAmsterdam Pharma



AstraZeneca 2







Tomas Kiselak Managing Member, Fairmount







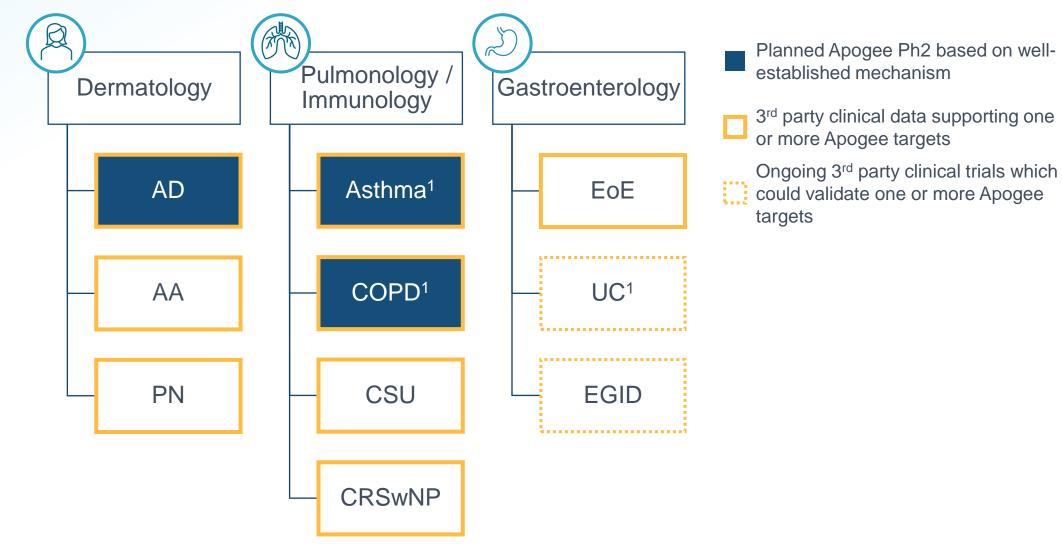
Nimish Shah Venrock





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







Apogee plans to become a leader in I&I therapeutics



APG77	7
(IL-13)	

● ✓ Phase 1 initiated in HVs

√ 6-month chronic toxicology completed



√ Positive Phase 1 PK & safety in HVs



1H: Phase 2 initiation in AD

2H: 16-week PoC data in AD

Phase 2 initiation in asthma

Disclose additional indication

APG808 $(IL-4R\alpha)$ JC nominated

√ Phase 1 initiated in HVs



2H: Initial Ph1 PK & safety in HVs

• 1H: PoC data in asthma

PoC trial initiation in COPD

APG990/222 $(OX40L \pm IL-13)$

Candidate nomination

Phase 1 initiation in HVs

- \$345M IPO
- √ Enhanced team and BOD

2023

- ✓ \$483M Follow-on
- R&D Day

