APOGEE THERAPEUTICS

CORPORATE OVERVIEW

April 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 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 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 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.

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 federal 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 information is 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 any 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 expressly 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 on 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 this 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.

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

FOCUS

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

EXPANSION

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

PIPELINE

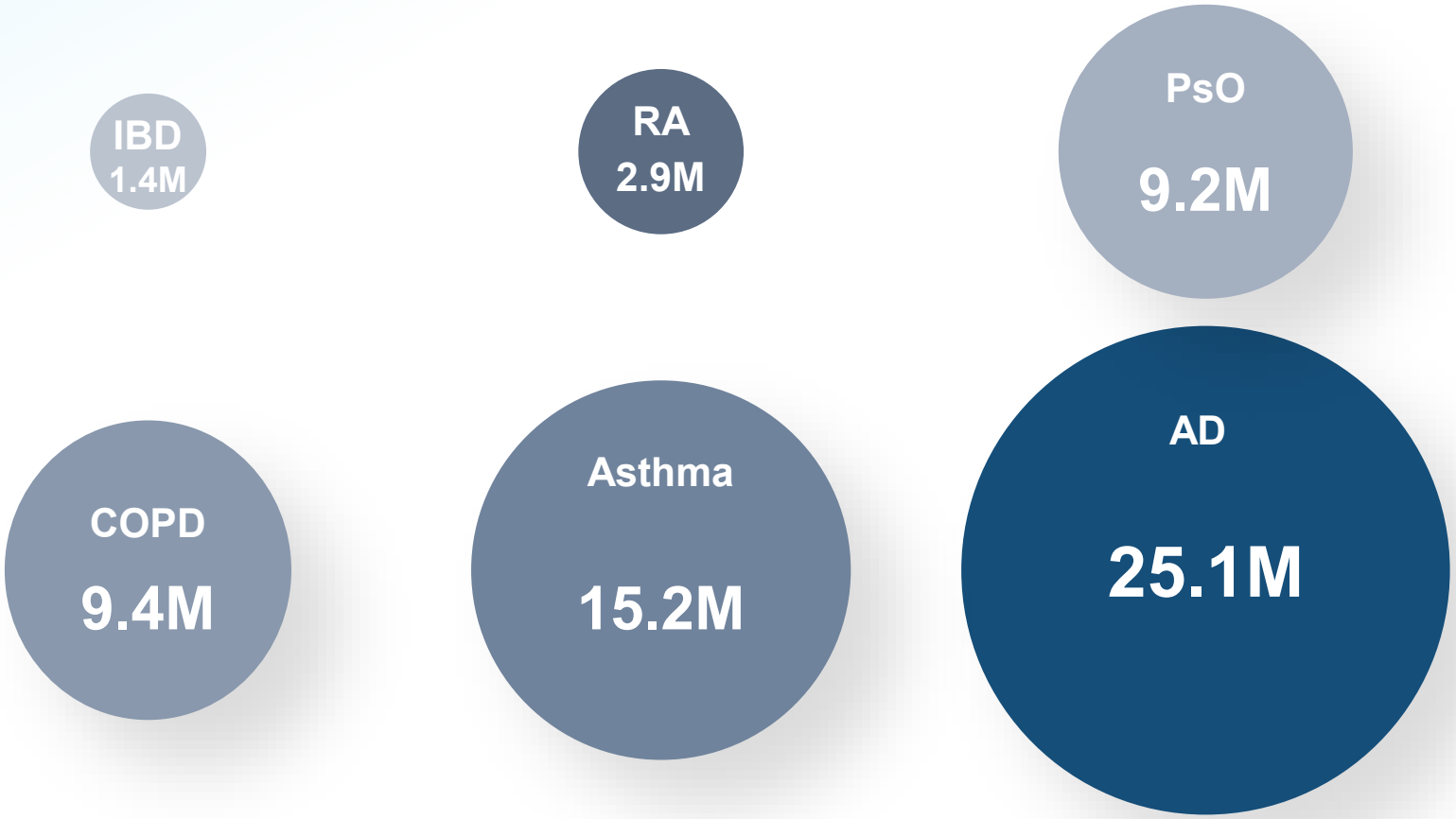
Portfolio leveraging **well-established mechanisms** and addressing I&I indications with **multi-billion-dollar potential**

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



- 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**

[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 small 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.

Apogee mAbs are engineered for best-in-class properties, including half-life extension

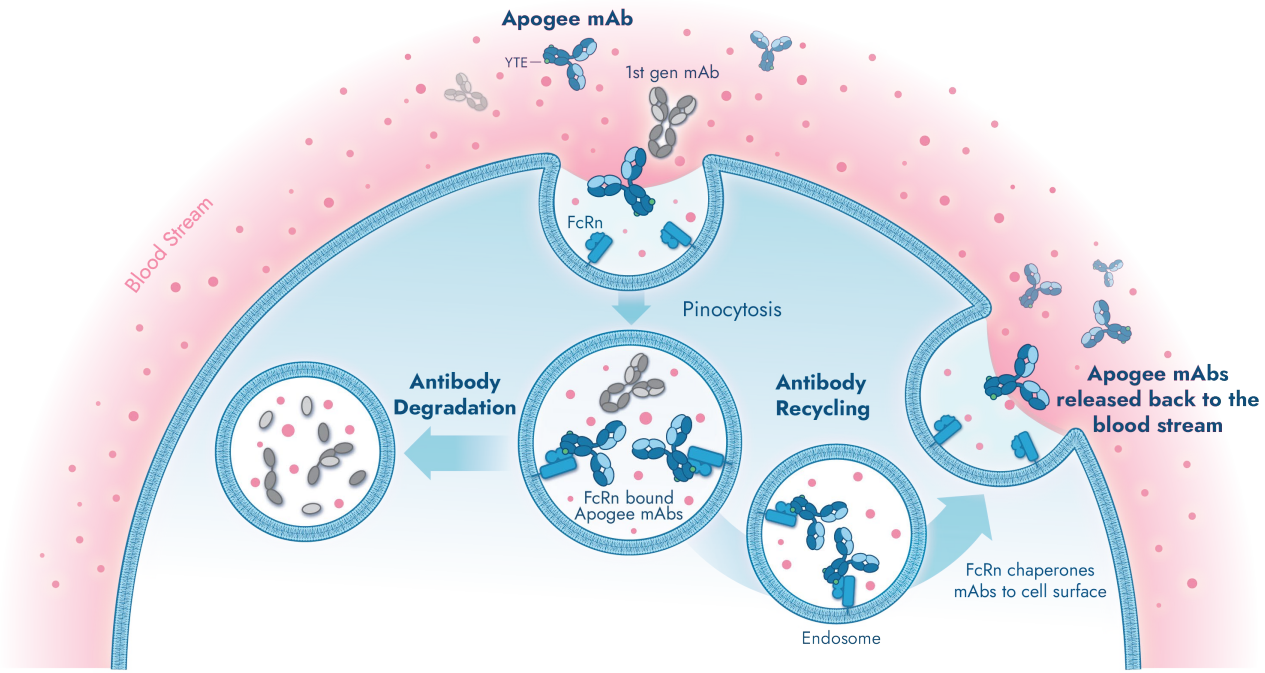
Based on clinically-validated epitopes with performance across five properties:

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

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

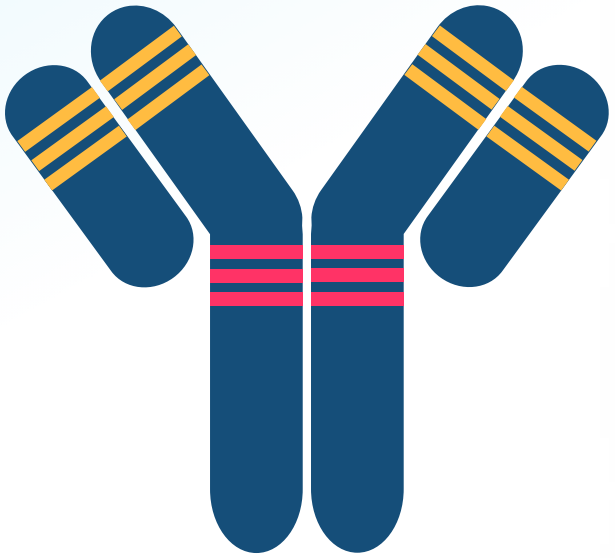
Potential for PK that:

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



APG777

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
- 75-day half-life (3x lebrikizumab)

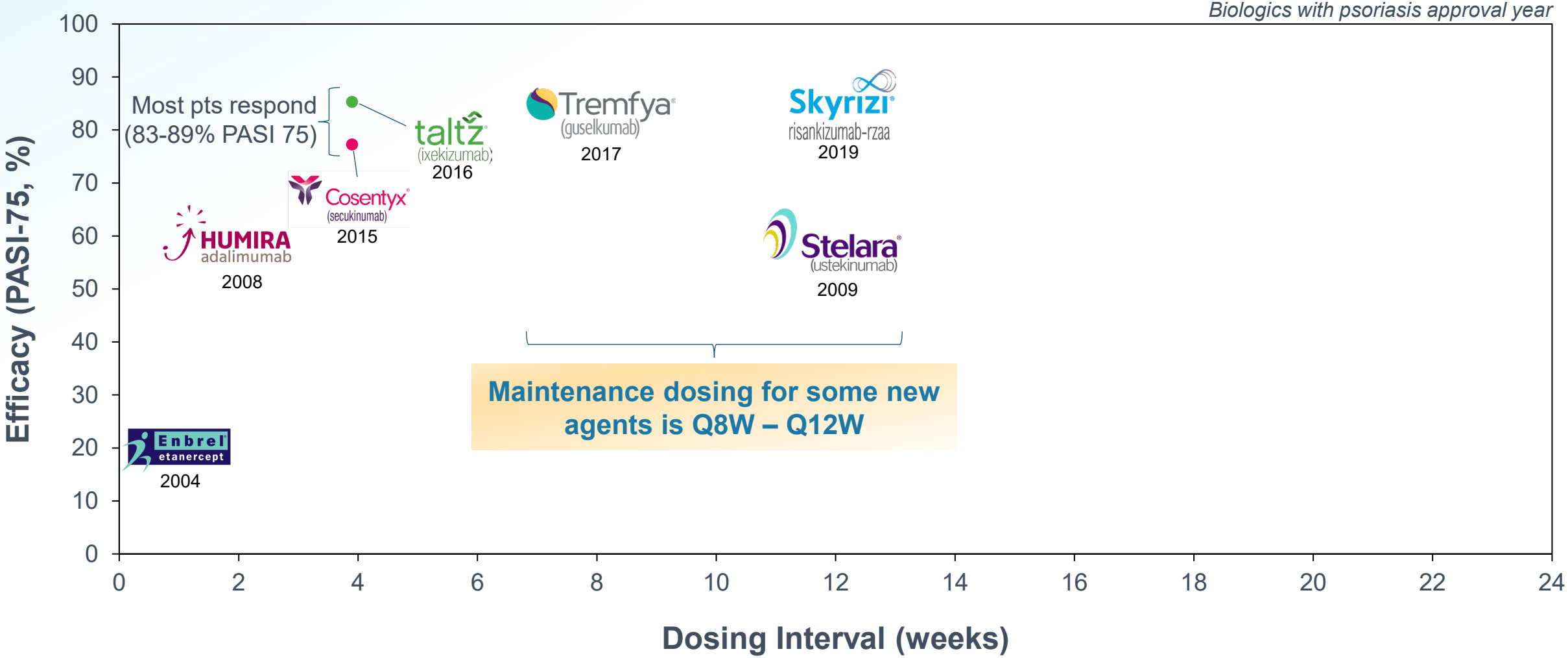
Favorable product characteristics and COGS

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

Novel IP into mid-2040s

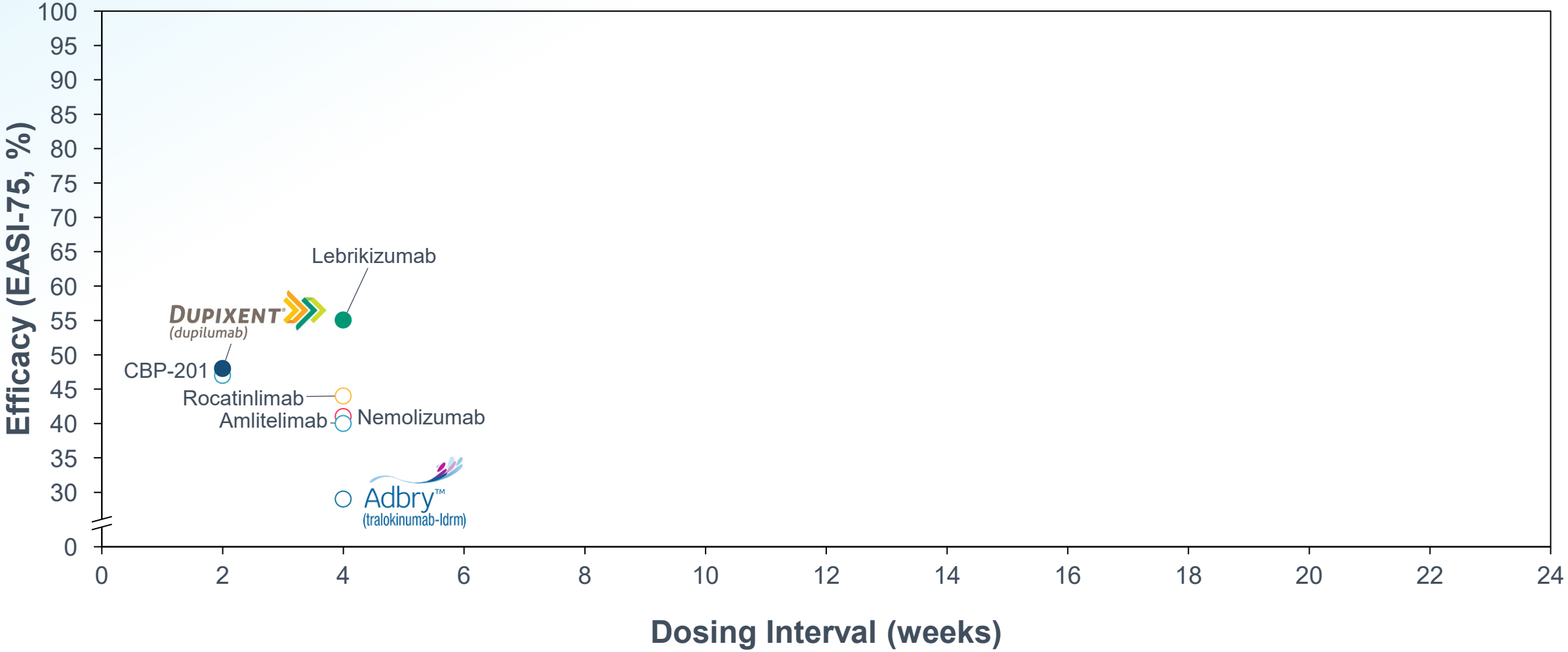
NOTE: [1]$50B projected AD market in 2035, based on projected growth of AD market at a similar rate to psoriasis. [2]APG777 has demonstrated equivalent potency to lebrikizumab in our head-to-head preclinical assays. SOURCE: EvaluatePharma.

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



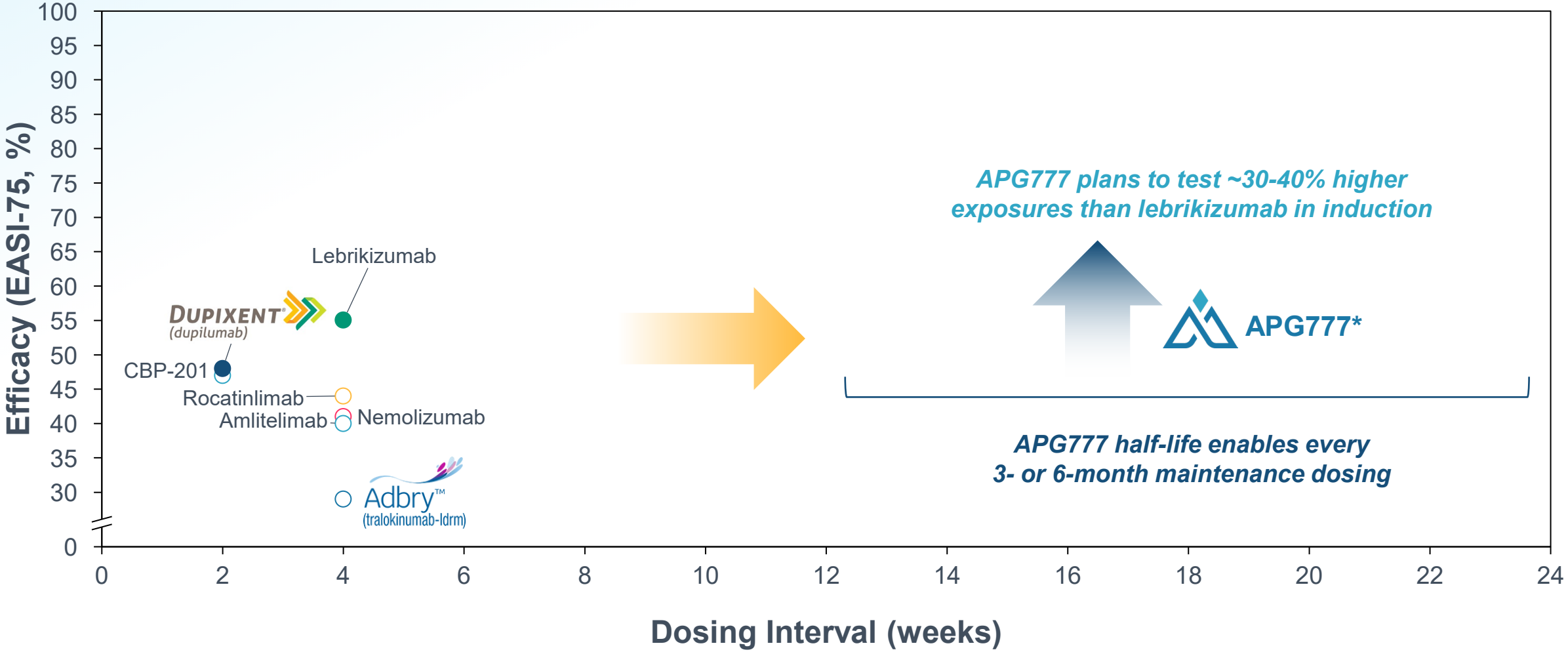
NOTE: Year denotes US launch year for adults with moderate to severe plaque psoriasis. Efficacy data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made. No head-to-head trials have been conducted among all biologics shown.
SOURCE: Armstrong AW, et al JAMA Dermatol. 2020. GlobalData. EvaluatePharma. USPIs.

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.

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

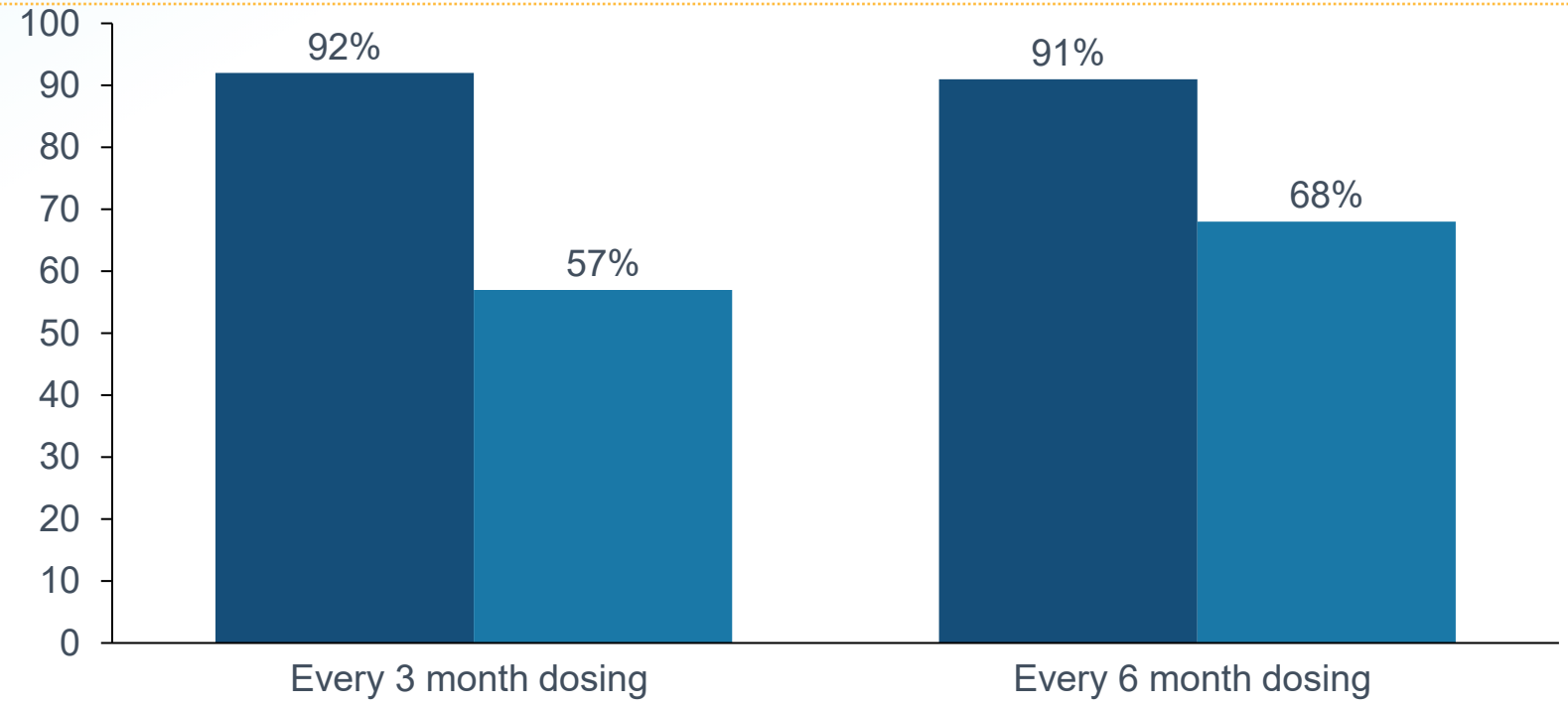


NOTE: *Positioning of Apogee programs is illustrative and based on interim Phase 1 results only and are illustrative of what we believe we can potentially achieve. Only DUPIXENT and ADBRY are approved.
SOURCE: 1. Lebrikizumab 250mg Q2W Ph3 avg. Silverberg JI et al. AAD 2022 2. Dupilumab 300 mg Q2W mono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

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)

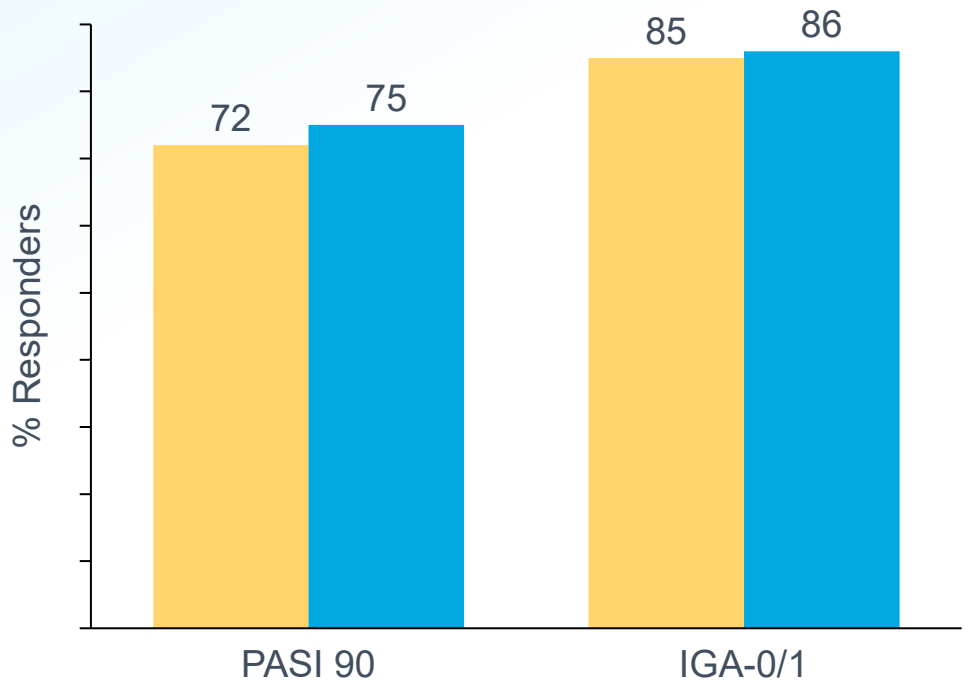
Proportion of switch patients (currently/formerly on a biologic)

NOTE: For providers where likeliness to prescribe Product Y (equivalent efficacy and safety as DUPIXENTTM) 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

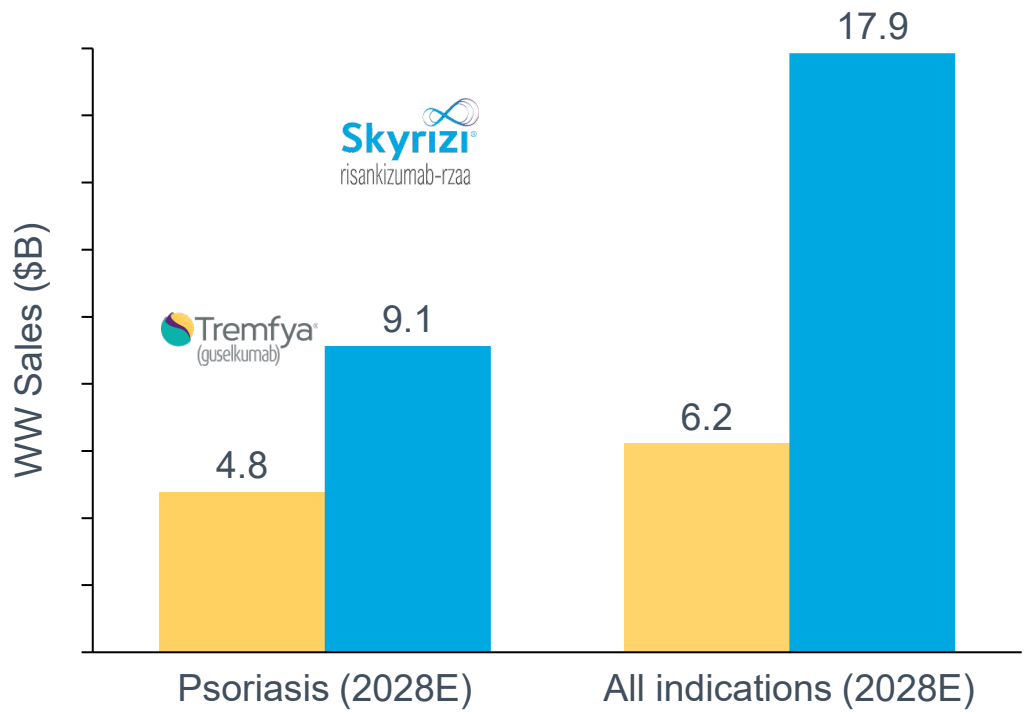
SOURCE: In 2023, Apogee conducted a single-blinded market research survey of 25 practicing dermatologists in 14 states in the United States, with the assistance of an expert search network

Psoriasis analogs demonstrate the value of reaching quarterly dosing

Efficacy of anti-IL23 biologics in psoriasis



Sales of anti-IL23 biologics ($B)

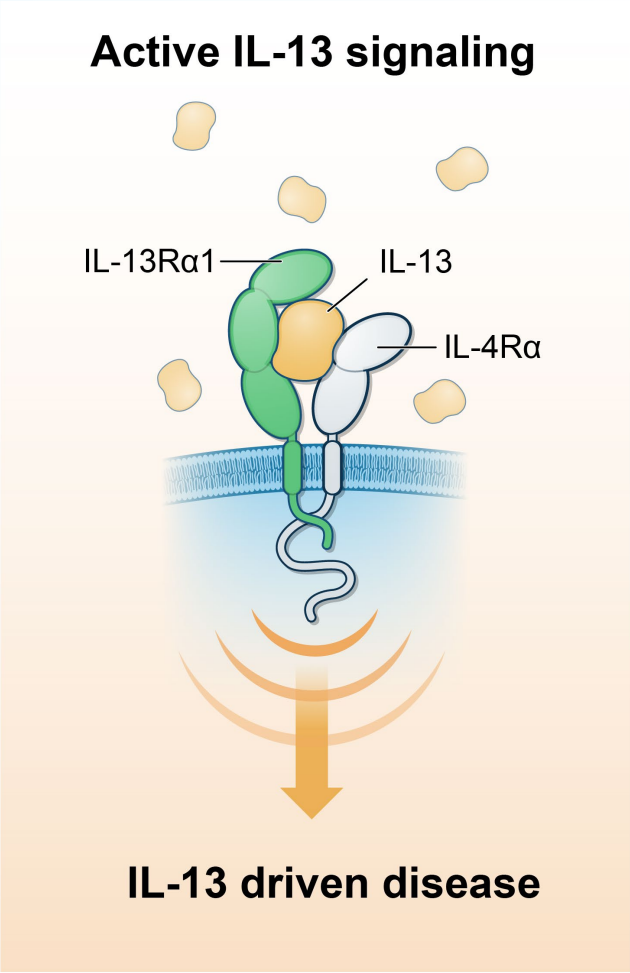


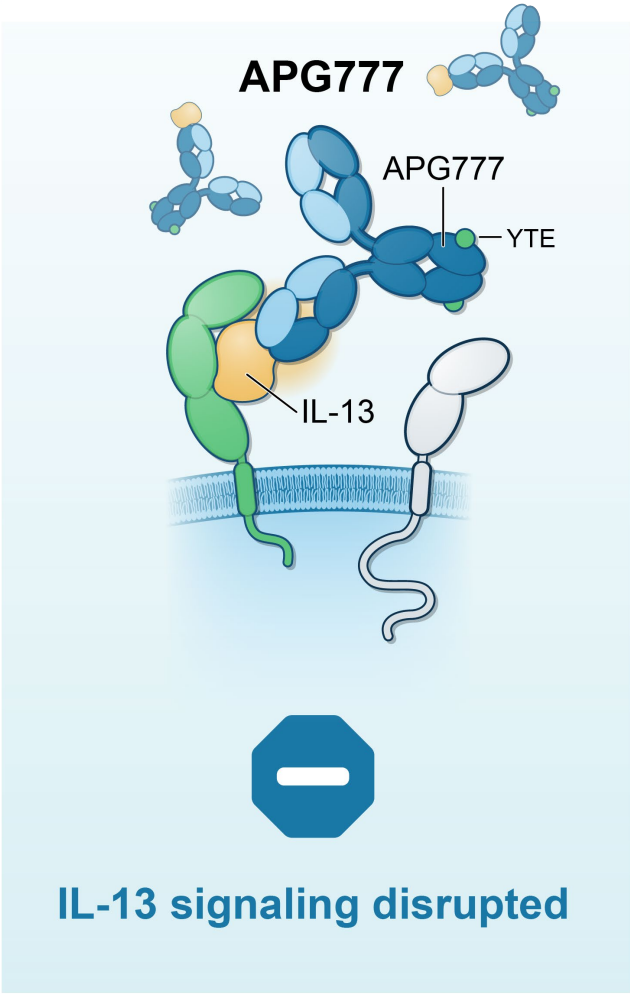
Tremfya (Q8W) - Approved 2017
Skyrizi (Q12W) - Approved 2019

Skyrizi's best-in-class dosing translated to a market-leading position

SOURCE: Skyrizi and Tremfya USPIs. EvaluatePharma WW consensus sales estimates

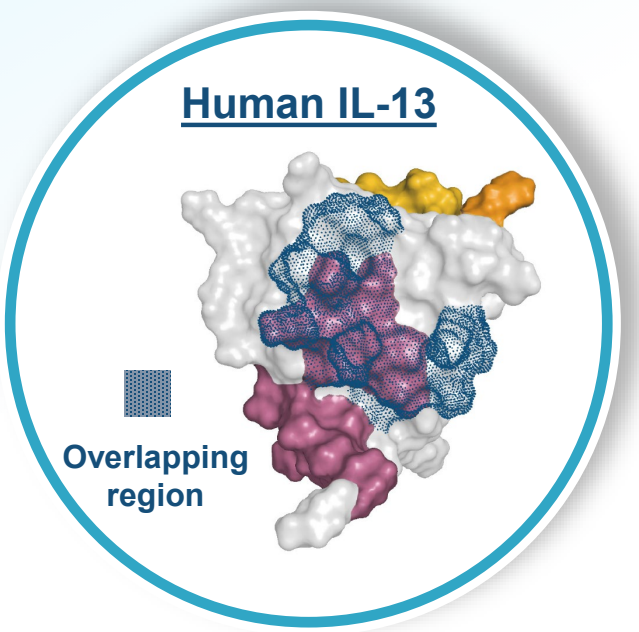
APG777 is designed to disrupt Th2 signaling by preventing formation of IL-13Rα1 / IL-4Rα heterodimer



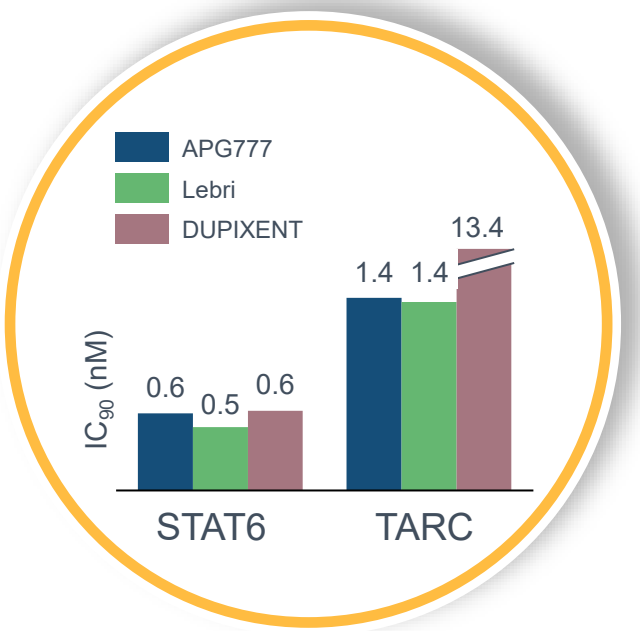


- IL-13 signaling **begins with binding of IL-13 to IL-13Rα1**
- This forms an inactive complex that then **binds to IL-4Rα to create a complete, active heterodimer**
- **Active IL-13Rα1 / IL-4Rα heterodimer sets off a signaling cascade** that leads to:
  - Skin barrier defects
  - Immune cell recruitment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

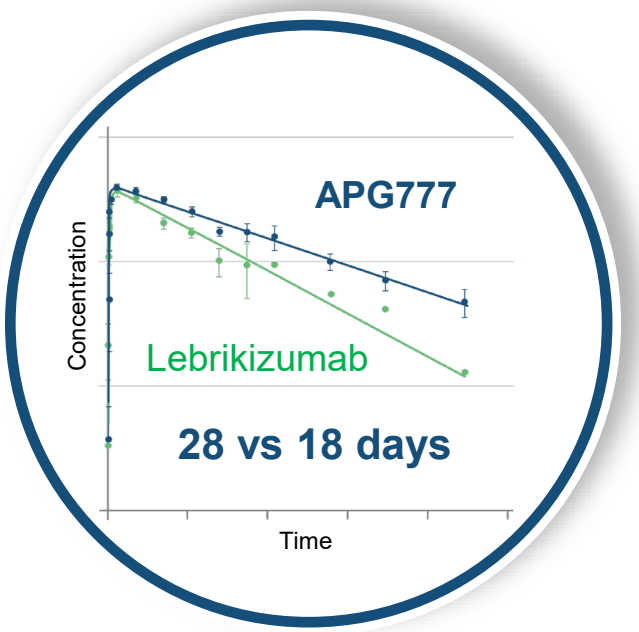
APG777 leverages lebrikizumab's mechanism to deliver a potentially best-in-class, pipeline-in-a-product antibody



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.

APG777 Phase 1 in Healthy Volunteers

APG777 Phase 1 initial data exceeded all trial objectives

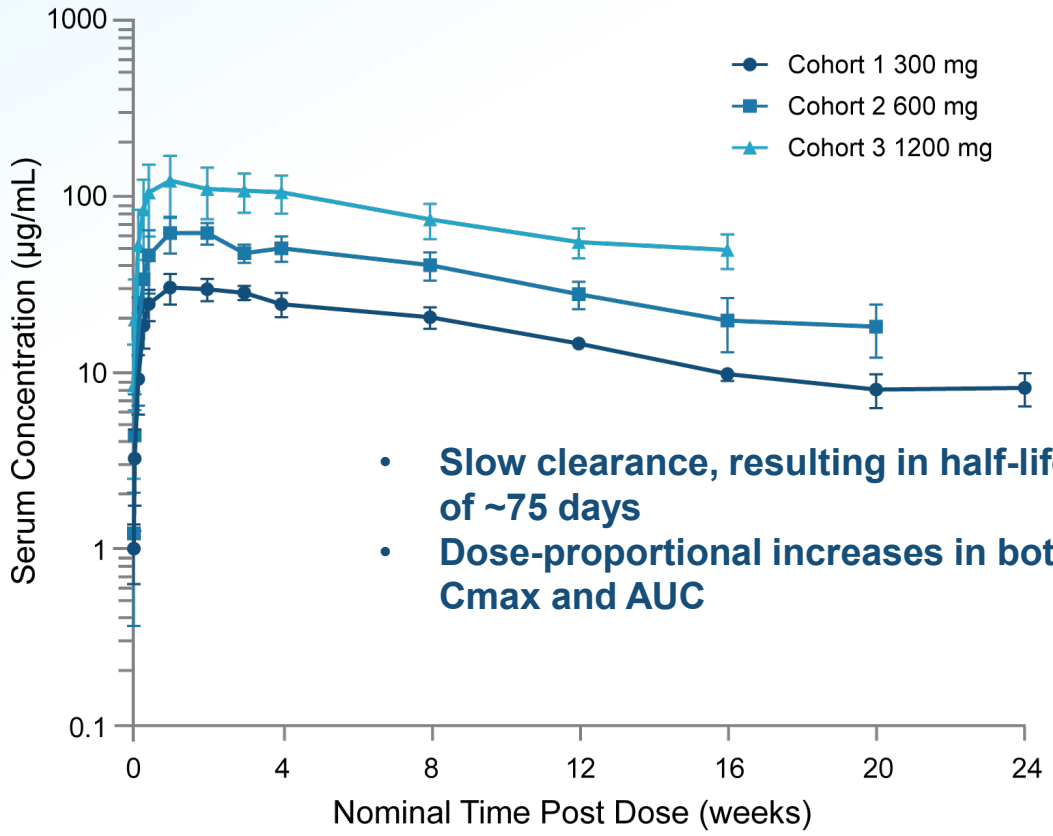
	Establish **safety** & **PK** profile	Set **Ph2 induction regimen**	Set **Ph2 maintenance regimens**	Supplemental
GOAL	*Well-tolerated with at least 33-day half-life*	*Achieve at least equiv. exposures to lebrikizumab with same or fewer injections*	*Equal lebrikizumab exposure with every 2-month or longer 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**

NOTE: PK = Pharmacokinetic

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

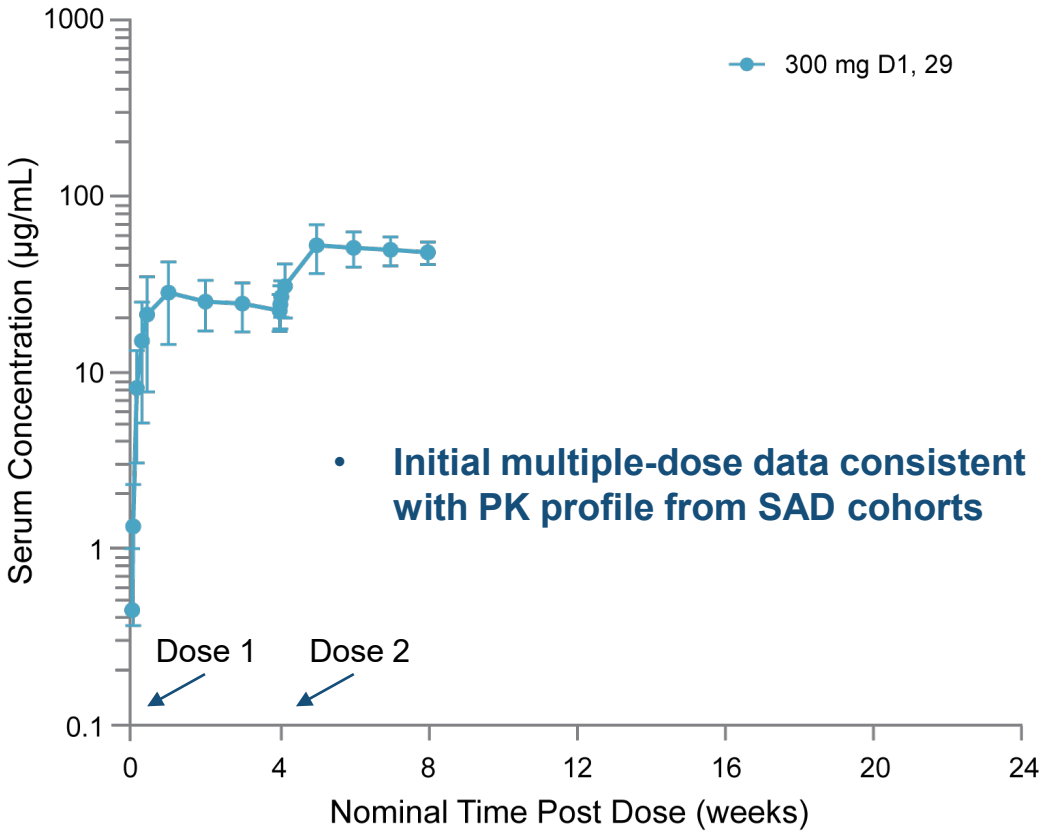
APG777 exhibited a potentially best-in-class PK profile with a half-life of ~75 days

Single-dose concentration-time profile



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

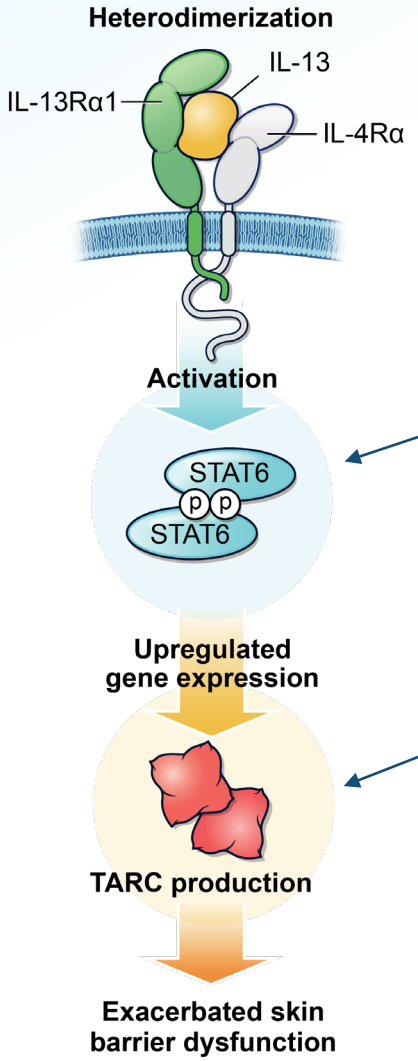
Multi-dose concentration-time profile



- **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



APG777 Phase 1 biomarkers

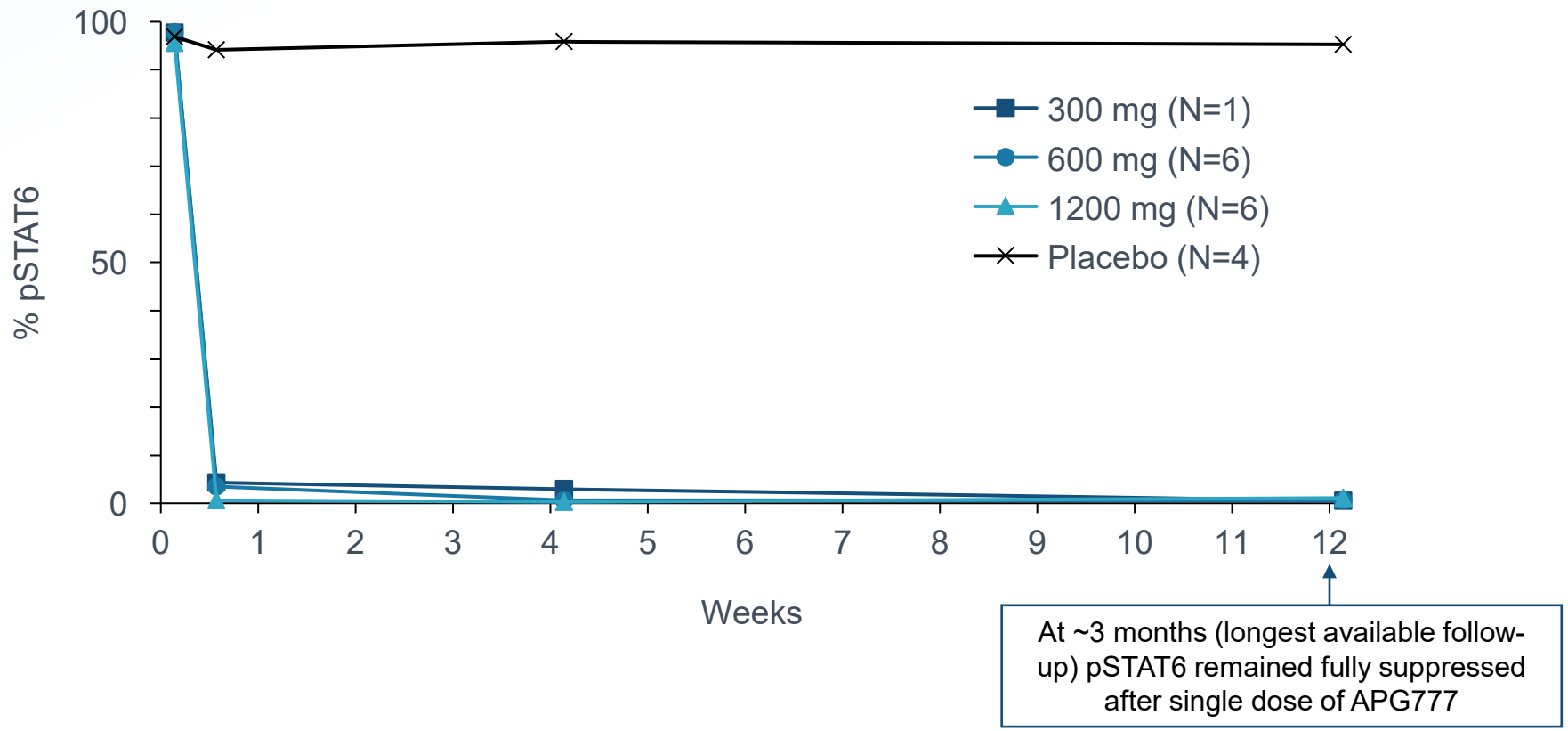
1. **pSTAT6** is one of the **earliest markers of IL-13 receptor activation**

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

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

Median percent change from baseline in pSTAT6

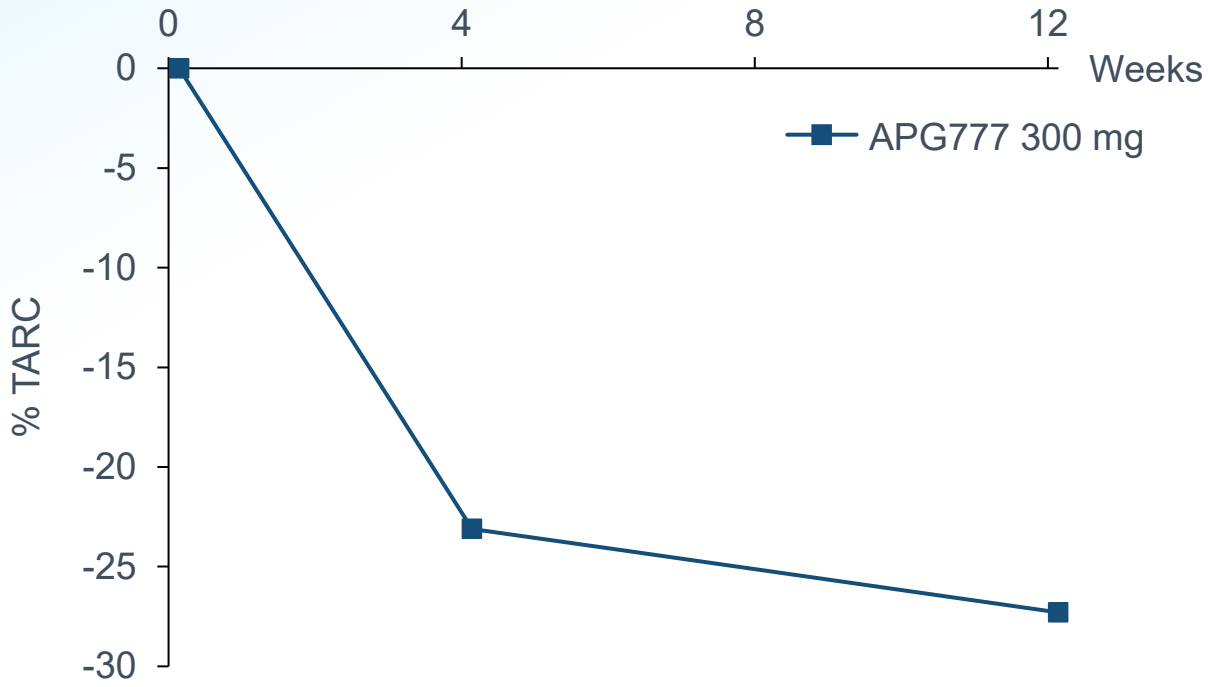


100% pSTAT6 inhibition was demonstrated for approximately 3 months across all doses

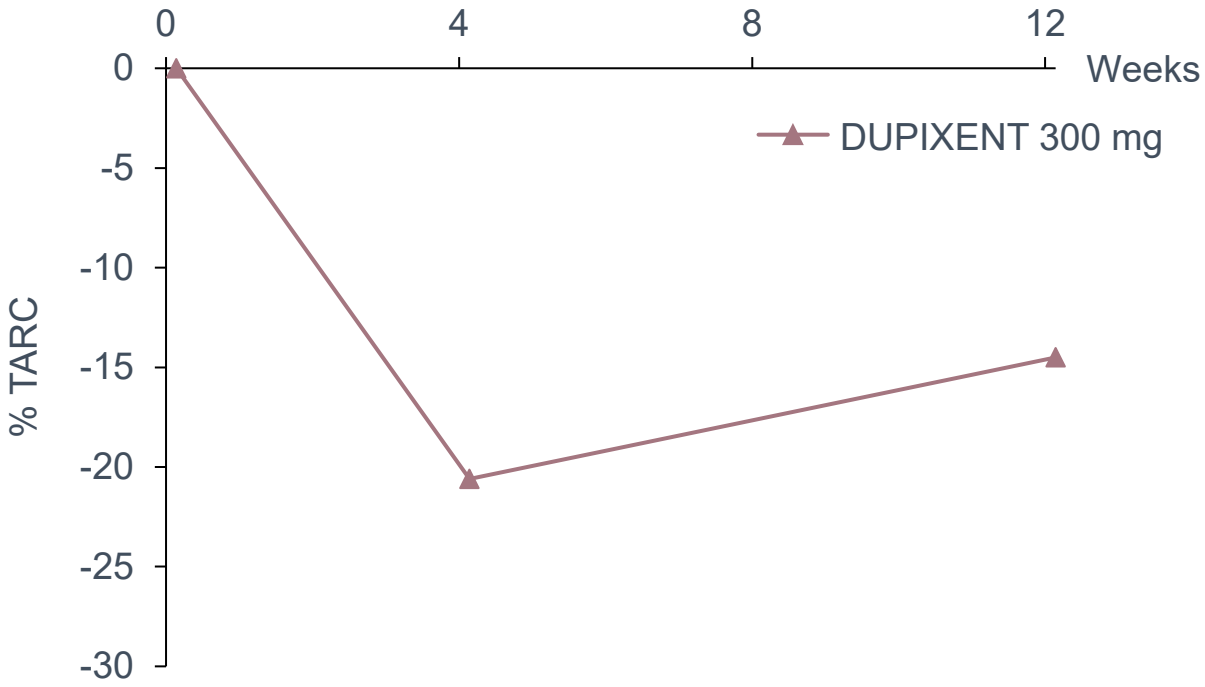
NOTE: N = 1 in cohort 1 due to the accelerated timing of study enrollment relative to assay validation. No data has been published showing DUPIXENT or lebrikizumab impact on pSTAT6 in HVs.

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.

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

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

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

Screening — 2:1

Induction

720mg W0 | 720mg W2 | 360mg W4 | 360mg W12

Placebo

Maintenance

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

Rollover to OLE or 52-week follow-up period

W16 Primary Endpoint

Integrated Phase 2 design has potential for significant timeline acceleration

- Combines Ph2a and Ph2b elements into a single protocol; All Part A sites are Part B sites (avoiding site startup delays between parts)

Part B: Dose optimization (N ~360)

Screening — 1:1:1:1

Induction

- High Dose
- Medium Dose
- Low Dose
- Placebo

Maintenance

- Regimen A (TBD)
- Regimen B (TBD)

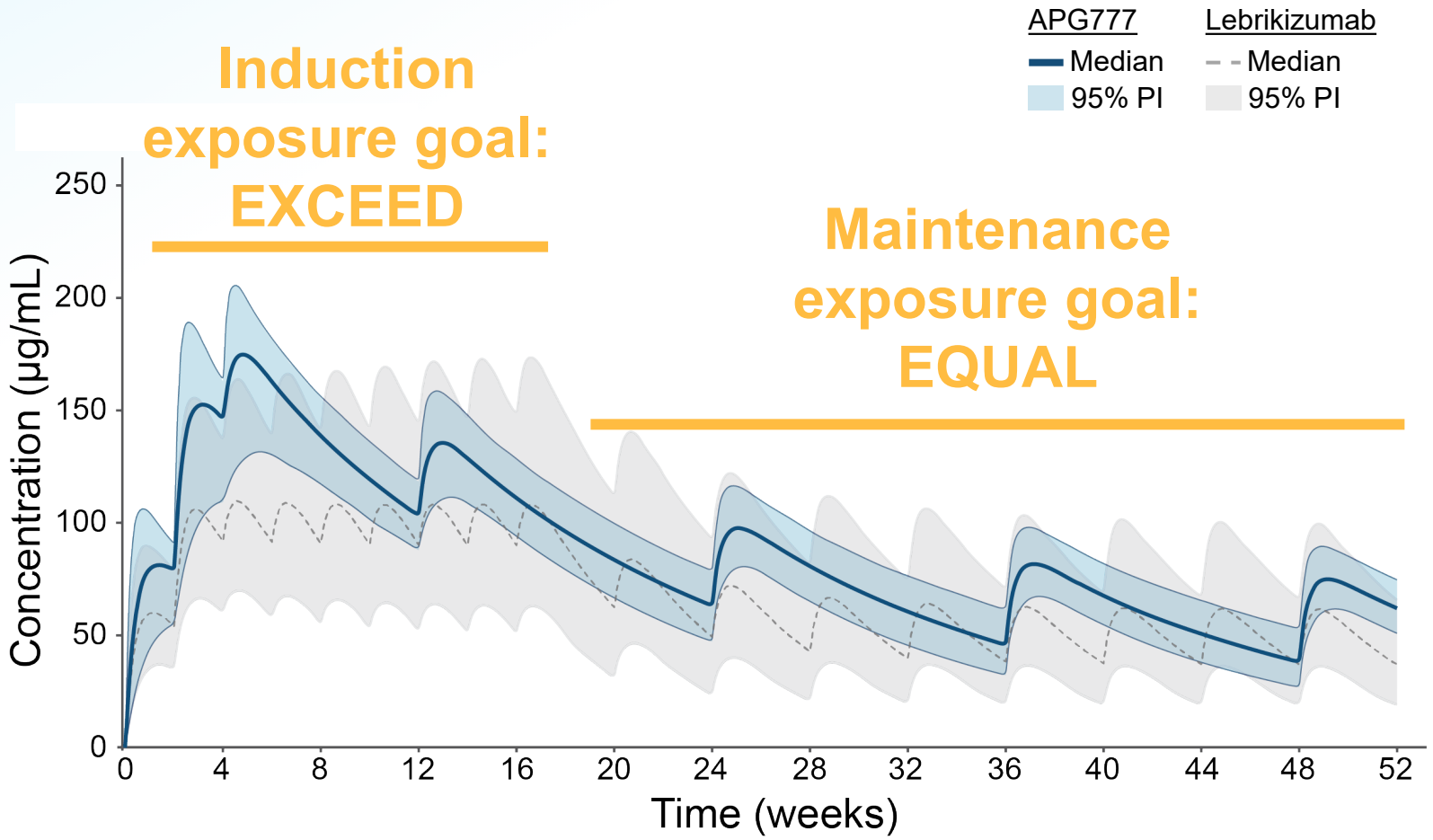
Rollover to OLE or 52-week follow-up period

W16 Primary Endpoint

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

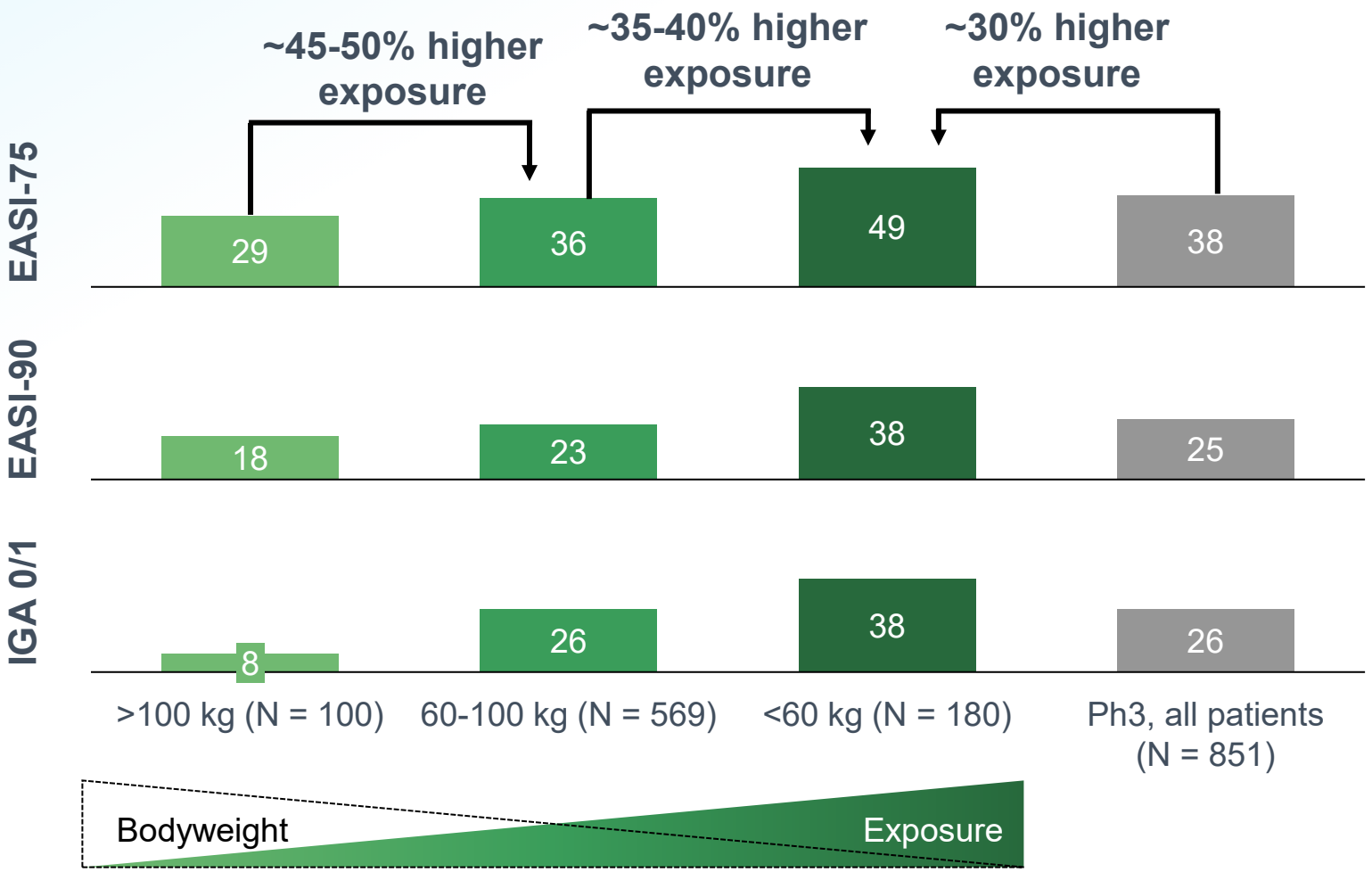


- Lebrikizumab data suggests an **exposure-response (E-R) for efficacy in induction** that underpins our goal **to EXCEED lebrikizumab induction exposures**
- There was **no E-R observed in maintenance for lebrikizumab;** our aim is to **EQUAL its exposure in maintenance**

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), %

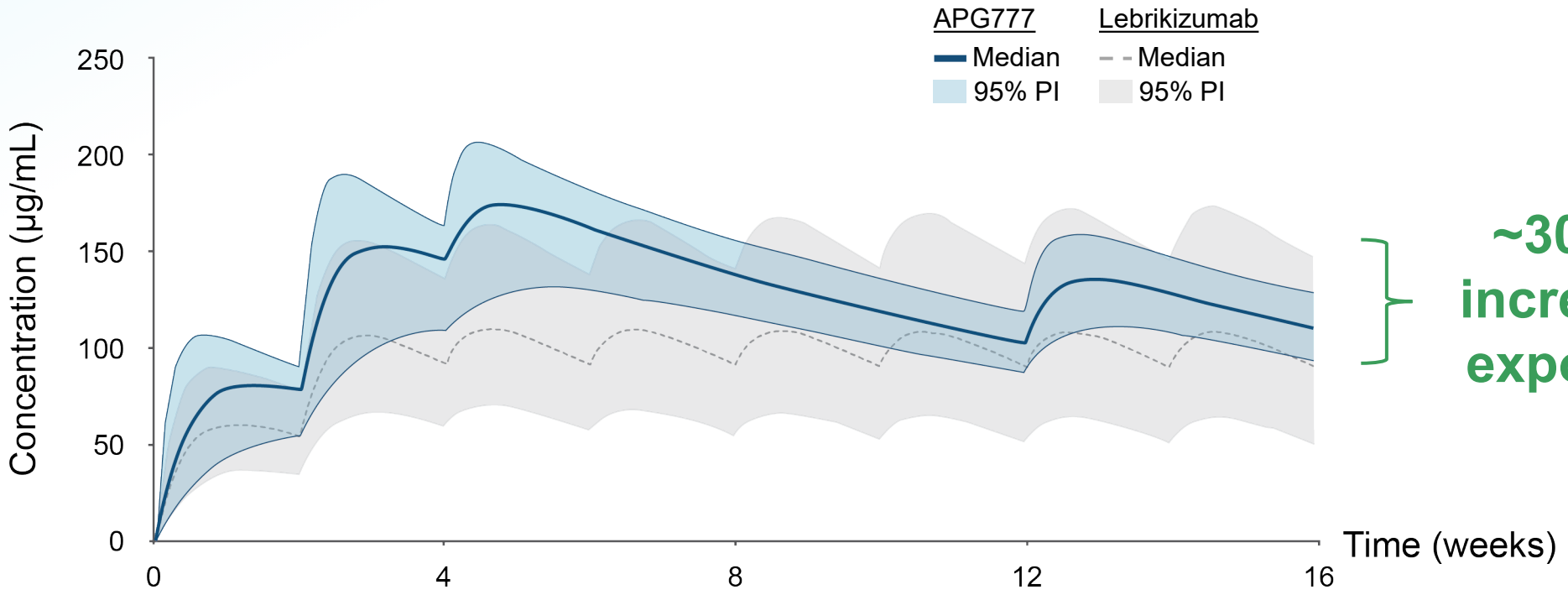


- Lebrikizumab exposure and induction efficacy are both inversely correlated with body weight
- Relationships suggest an **exposure-response for efficacy in induction and support testing higher exposures with APG777**
- In lebrikizumab Ph2b and Ph3 there has been **no dose-AE or exposure-AE relationship**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

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)
SOURCE: Lebrikizumab European Public Assessment Report

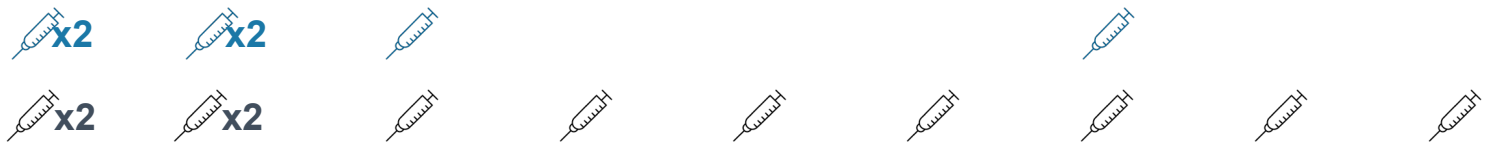
Modeled Phase 2 induction exposures exceed those of lebrikizumab by ~30-40%

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

Modeled Phase 2 Q3M maintenance exposures equal those of lebrikizumab

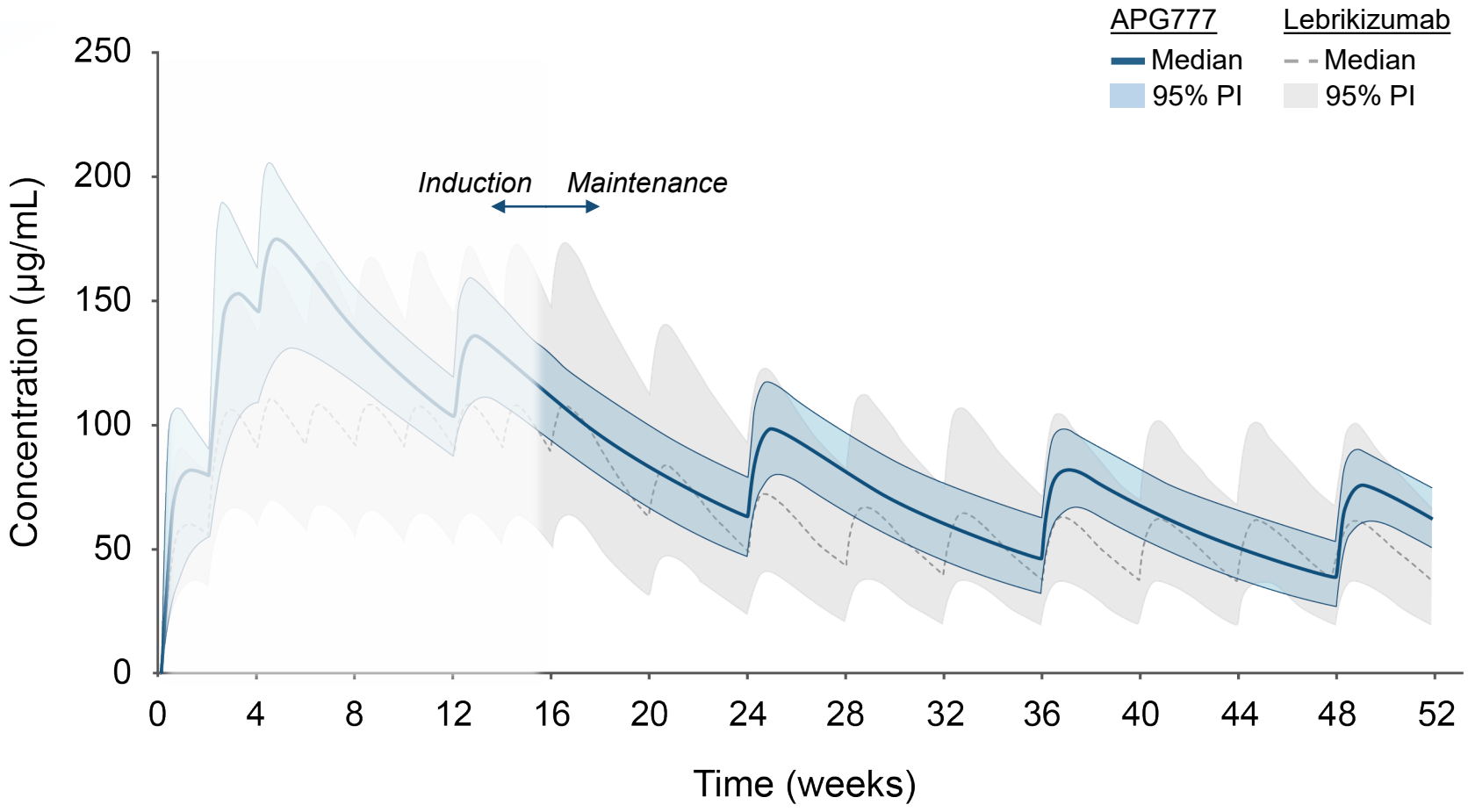
APG777 Q3M

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.

Modeled Phase 2 Q6M maintenance exposures equal those of lebrikizumab

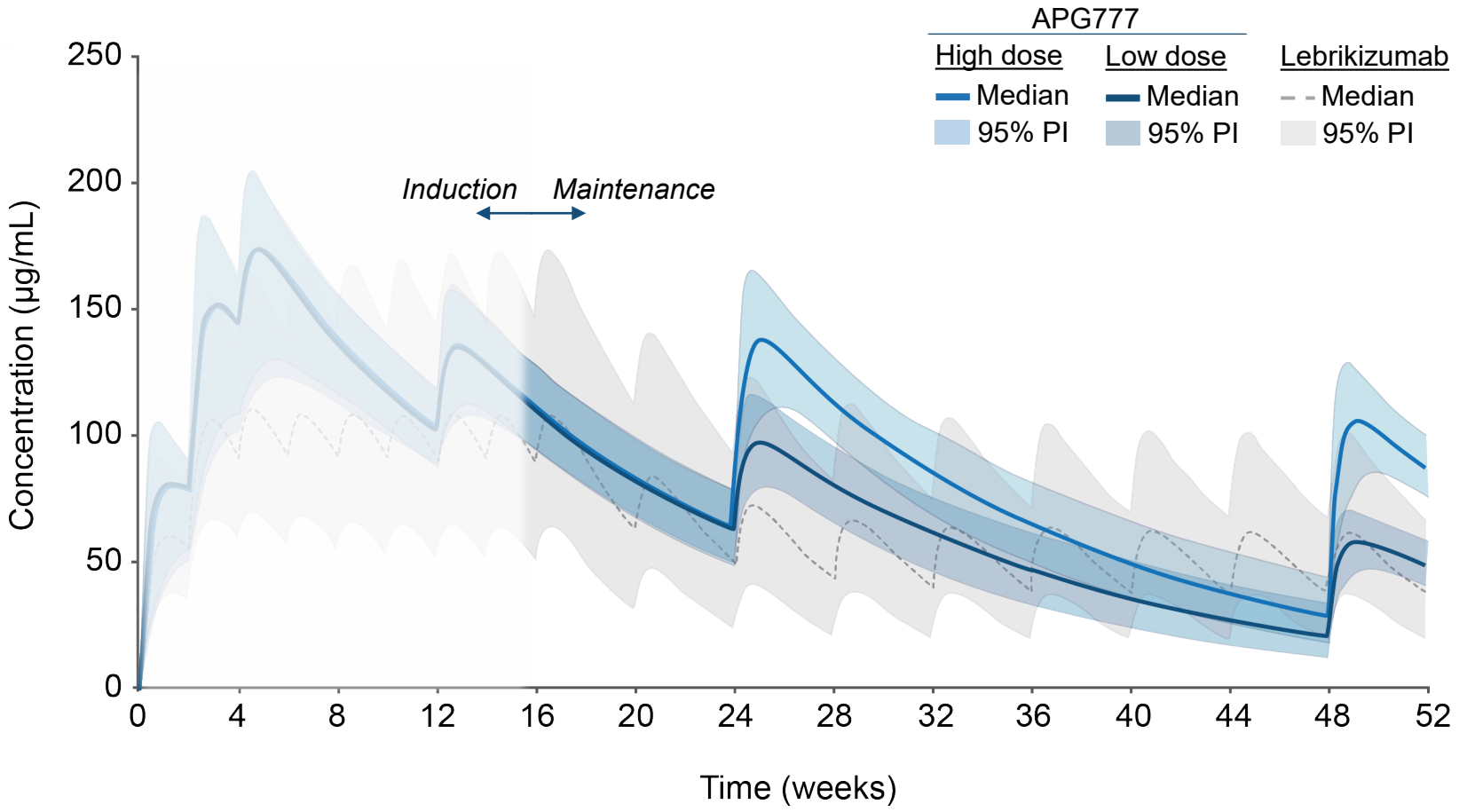
APG777 Q6M

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.

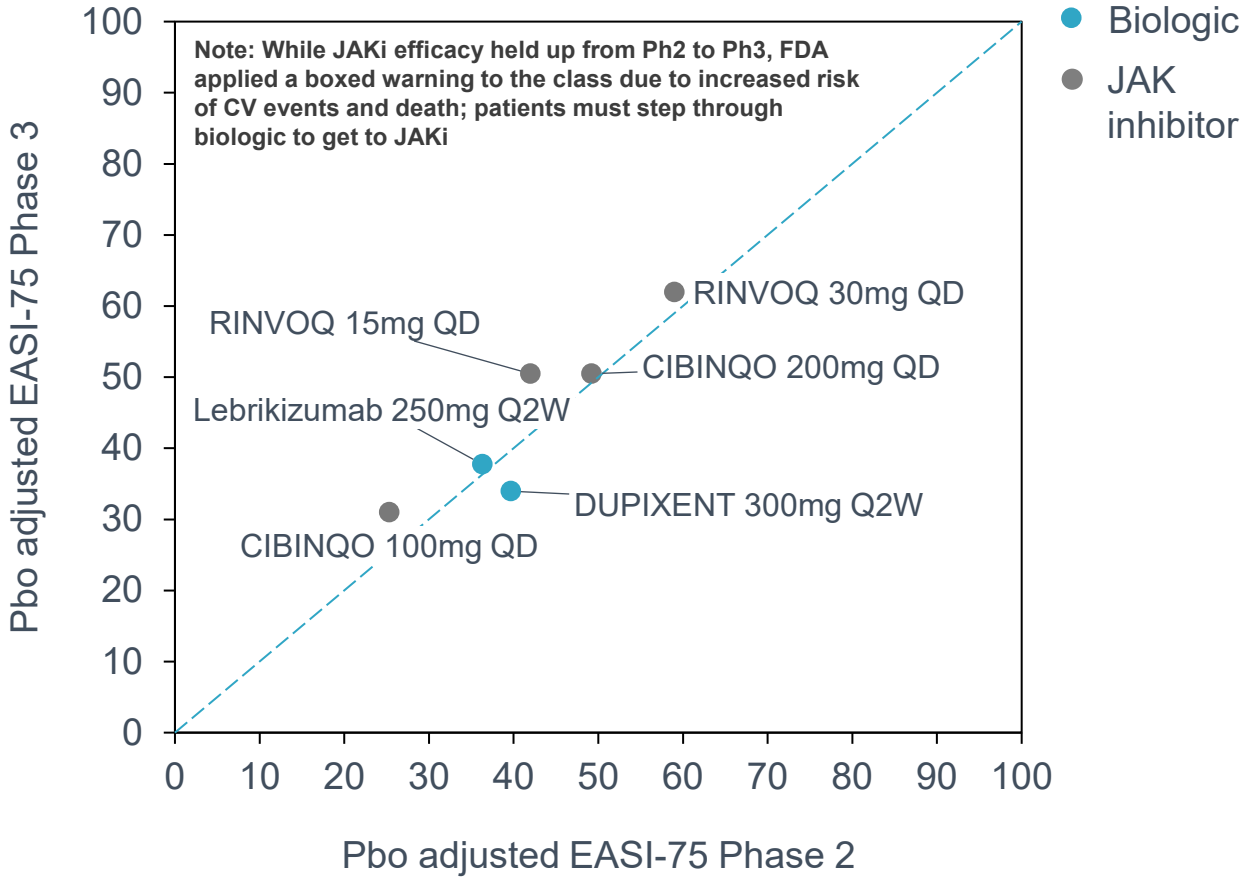
Strong historical correlation between Phase 2 and 3 data makes APG777 16-week AD data a key catalyst

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 in 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

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

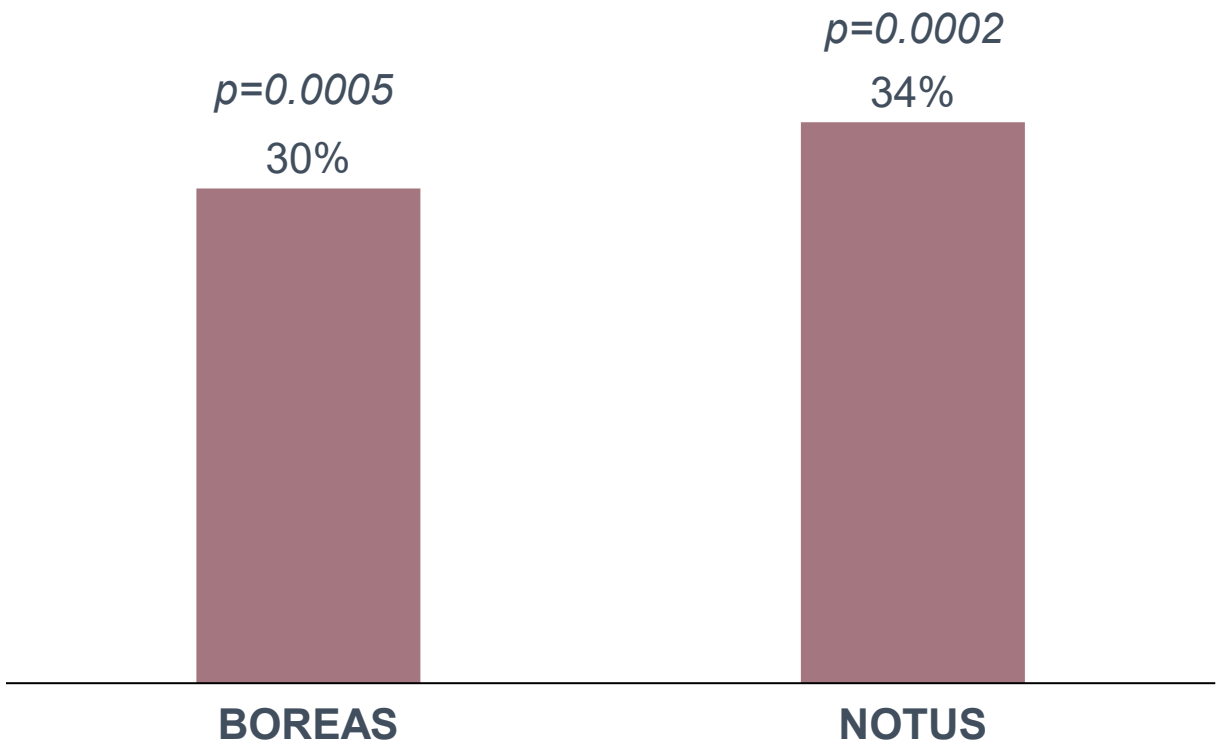
3rd
Leading cause of death in the US in 2019

150K+
People die each year in the US

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

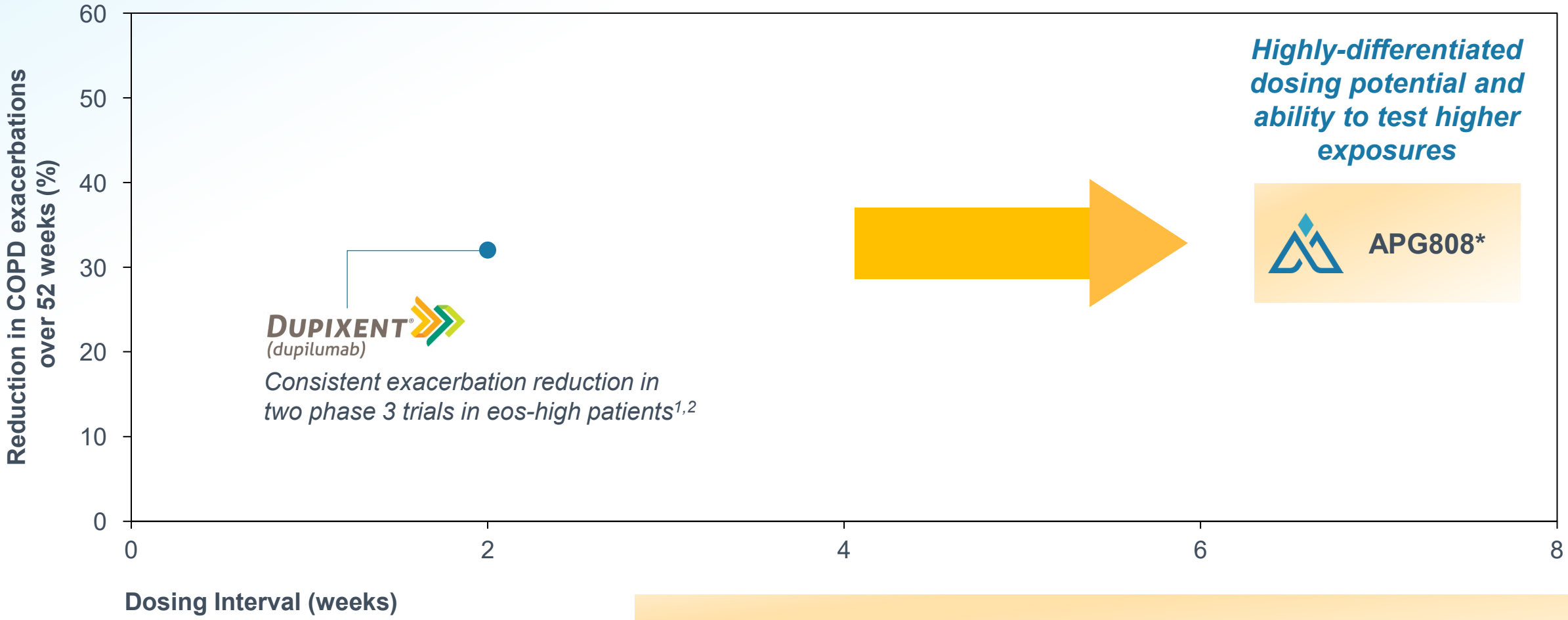
- Significant, clinically meaningful **reduction in moderate or severe acute COPD exacerbations**
- **Improved lung function from baseline at 12 weeks** compared to placebo with separation from placebo as early as 2 weeks

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



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

Treatments for moderate-severe COPD are limited

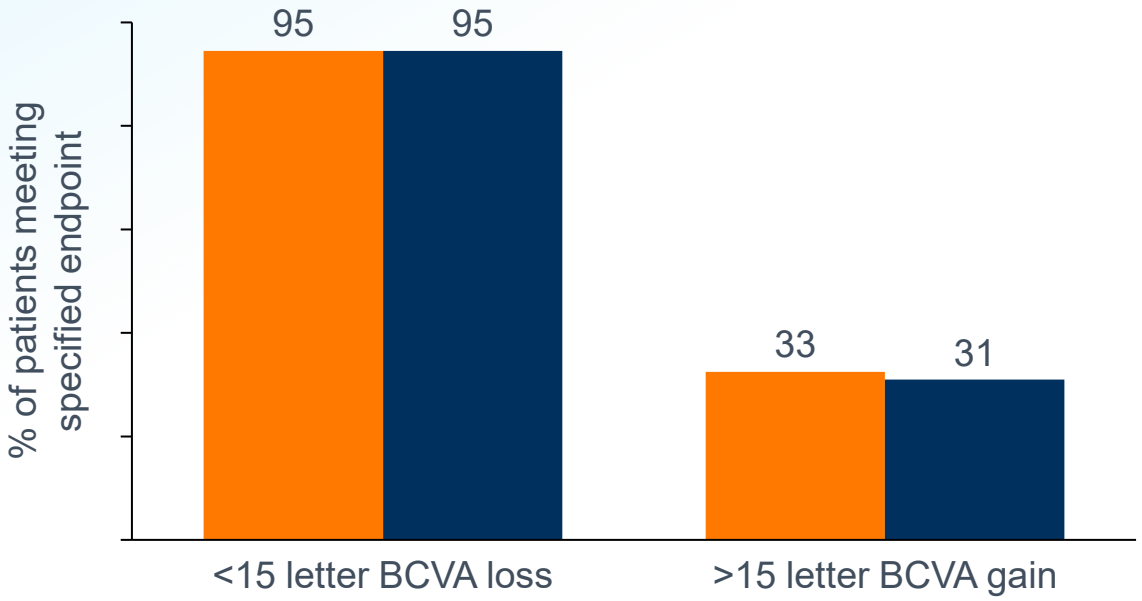


Other than DUPIXENT, no other late-stage biologic for the treatment of COPD has achieved its primary endpoint, leaving a vast unmet need for dosing beyond Q2W

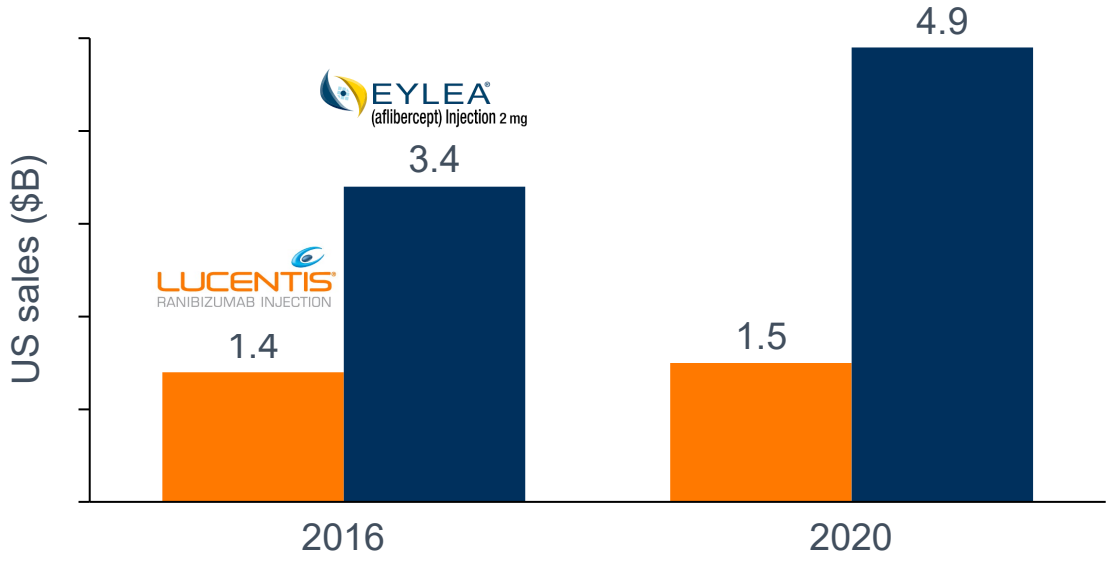
NOTE: *Positioning of Apogee program is illustrative and not based on clinical trial data. Dupixent is not approved for the treatment of COPD
SOURCE: [1]Bhatt SP et al NEJM 2023; [2]*Sanofi press release November 26, 2023, interim analysis, full results not yet disclosed*

With similar efficacy, dosing drives market share: Eylea (Q8W) significantly outsold Lucentis (Q4W) despite later launch

Efficacy in wAMD registrational trials



US sales ($B)



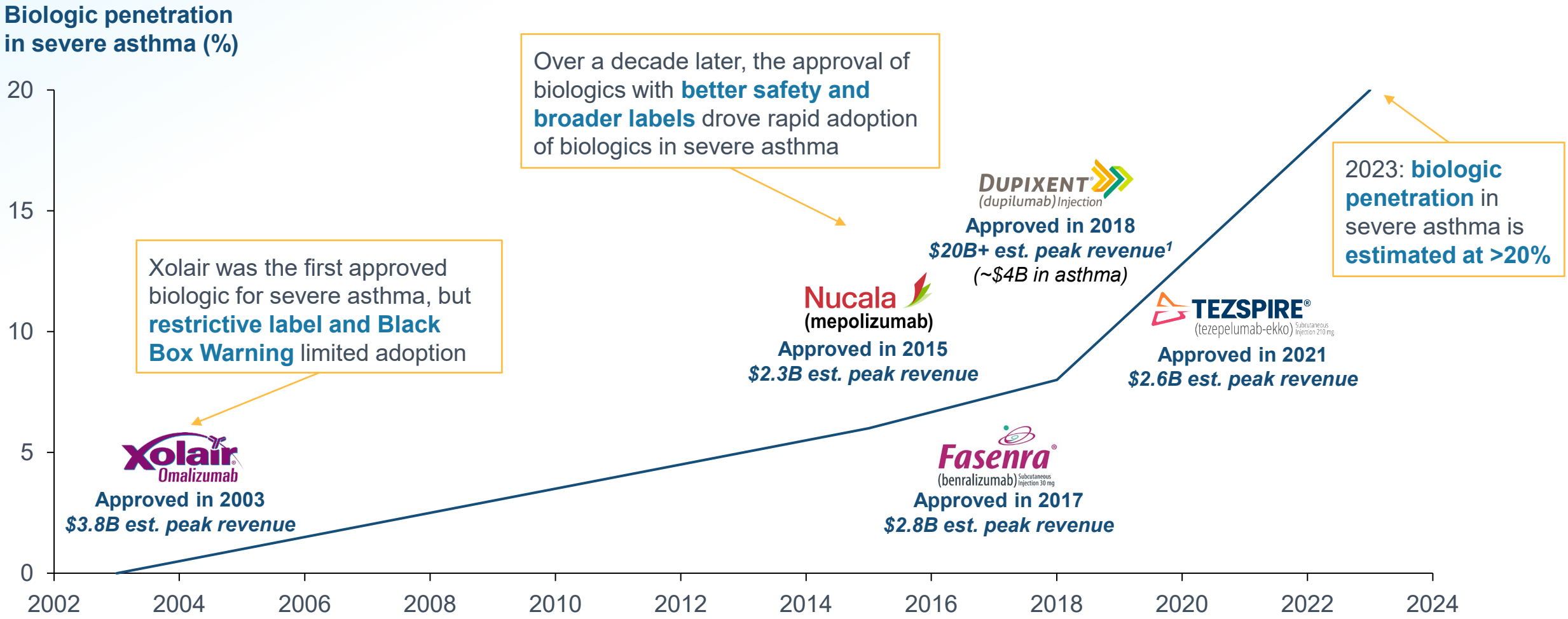
Lucentis (Q4W) – Approved in 2006 (LOE in 2020)

Eylea (Q8W) – Approved in 2011

Eylea's success demonstrates how dosing differentiation can drive commercial success, even for a smaller biotech competing against an established incumbent (Eylea was Regeneron's first major launch)

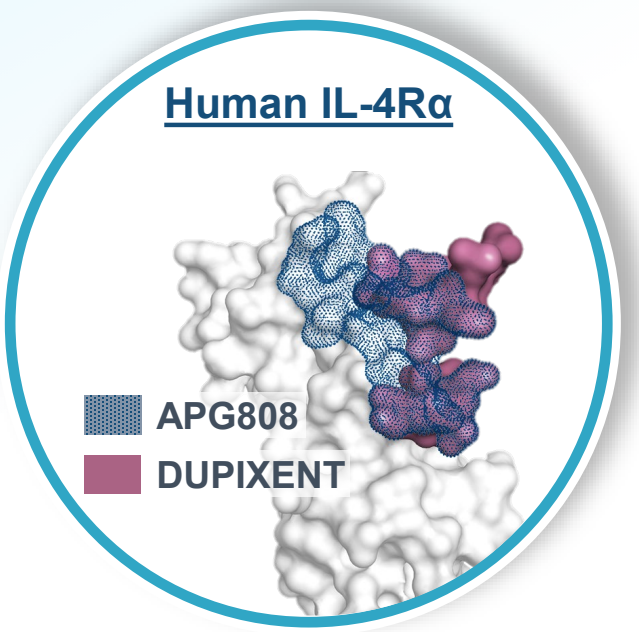
SOURCE: Eylea FDA Label (average of VIEW1 and VIEW2 trials that compared Eylea and Lucentis H2H); equity analyst reports

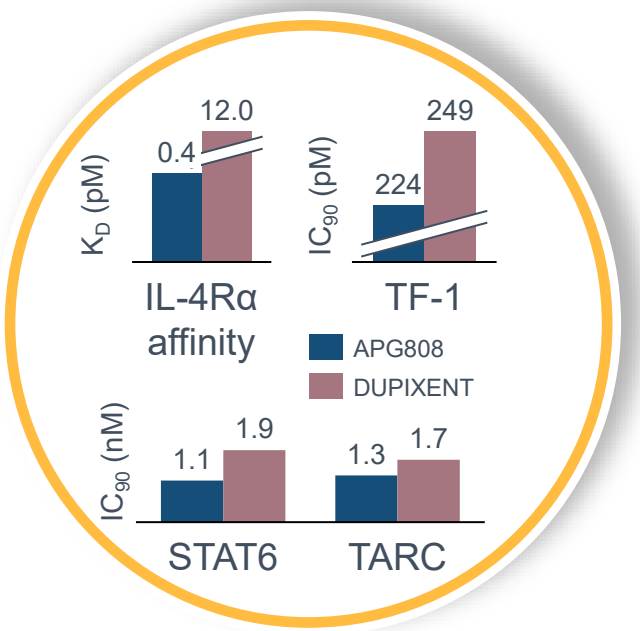
Asthma, an analog for COPD, shows how biologics can be rapidly adopted when they address unmet needs

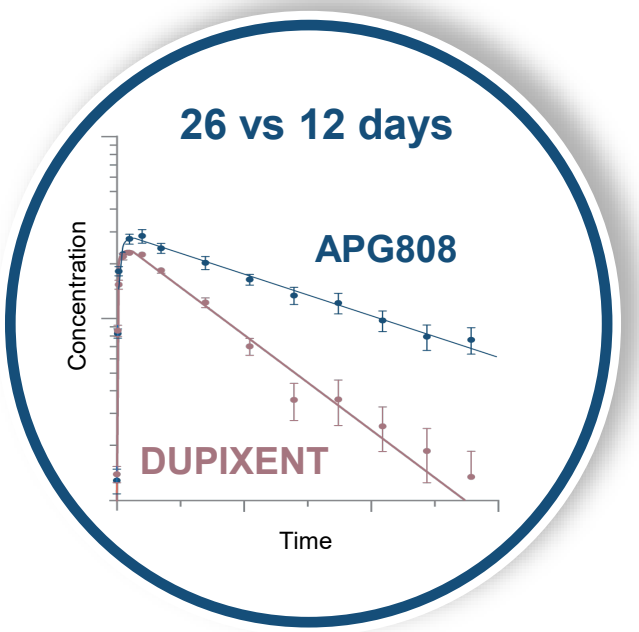


NOTE: Analysts estimate ~20% of DUPIXENT revenue is from asthma
SOURCE: Company 10-K filings, annual reports, analyst forecasts, Citeline; EvaluatePharma

APG808 leverages DUPIXENT's mechanism to deliver a potentially best-in-class, pipeline-in-a-product antibody







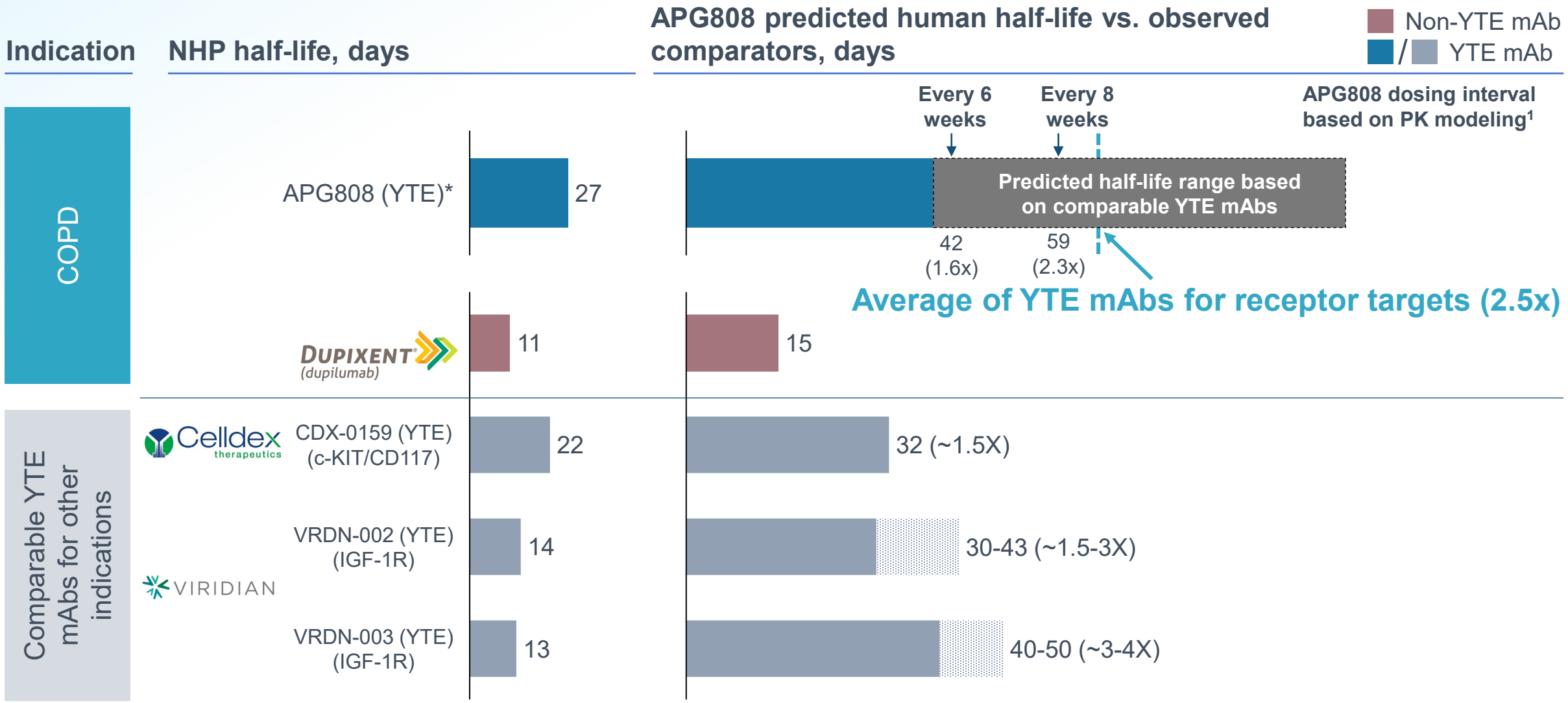
APG808's epitope on IL-4Rα overlaps with DUPIXENT's and leverages proven MoA and biology

APG808 is as potent as DUPIXENT across preclinical assays

APG808 NHP half-life is more than 2x longer than DUPIXENT

NOTE: MoA = Mechanism of Action.

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



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

APG808 Phase 1 is underway with planned interim readout in 2H 2024

Trial design elements

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

Single ascending dose in healthy participants

N ~ 32

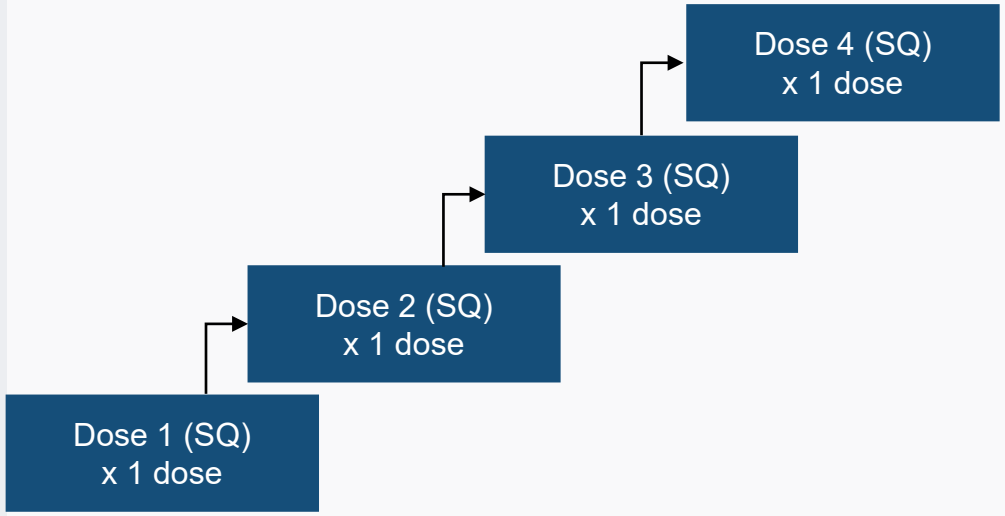
8 per cohort (6:2 active:placebo)

Key inclusion criteria: healthy adult participants

Primary endpoint: safety

Secondary endpoints: PK, ADA

Single ascending dose[1]



Asthma cohort[2]

Dose or regimen TBD

Ph1 readout in 2H 2024 will confirm potential for best-in-class dosing

Ph1b readout in 1H 2025 will demonstrate APG808 effect on FeNO in mild asthmatics

NOTE: [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 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

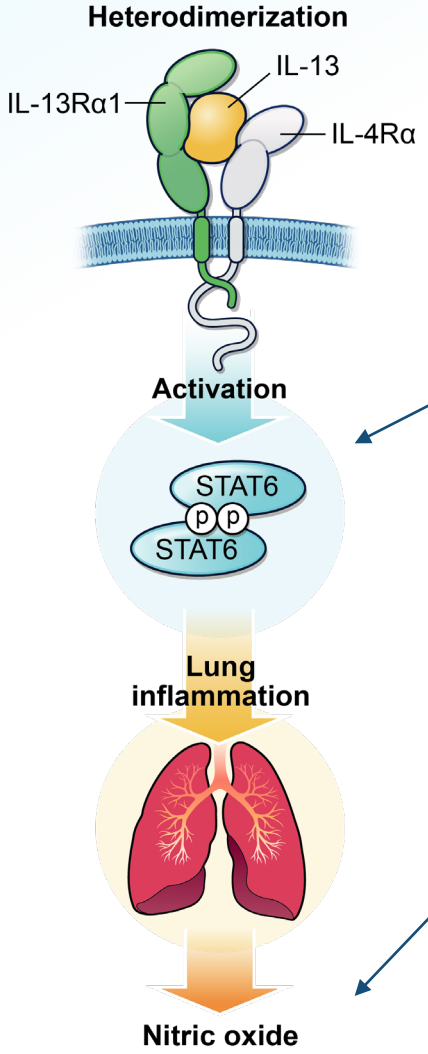
2H 2024: confirm potential for best-in-class dosing intervals

Dosing Goal: every 6- or 8-weeks

(vs. every 2 weeks for DUPIXENT[1])

NOTE: PK = Pharmacokinetic. [1] Based on FDA label for DUPIXENT™

FeNO is a biomarker of IL-4Rα engagement and correlates with lung dysfunction and exacerbations



APG808 Phase 1b biomarkers

1. **pSTAT6** is one of the **earliest markers of IL-4Rα receptor activation**

2. Fractional exhaled nitric oxide **(FeNO) levels are correlated to severity in both asthma and COPD**

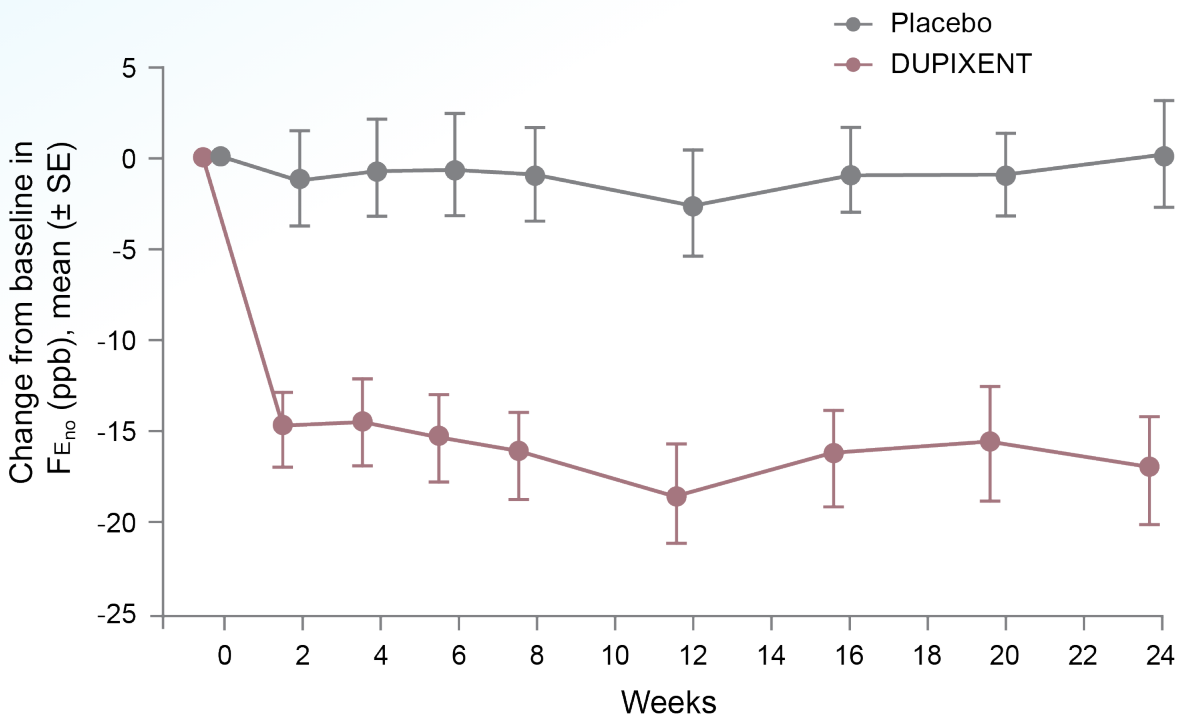
   – **After Dupixent treatment in asthma in COPD, FeNO decreases,** with corresponding **reduction in exacerbations** and **improvement in FEV1**

Phase 1b will test APG808's ability to reduce this biomarker in mild asthmatics, **confirming inhibition of IL-4Rα signaling** and allowing comparison to other agents

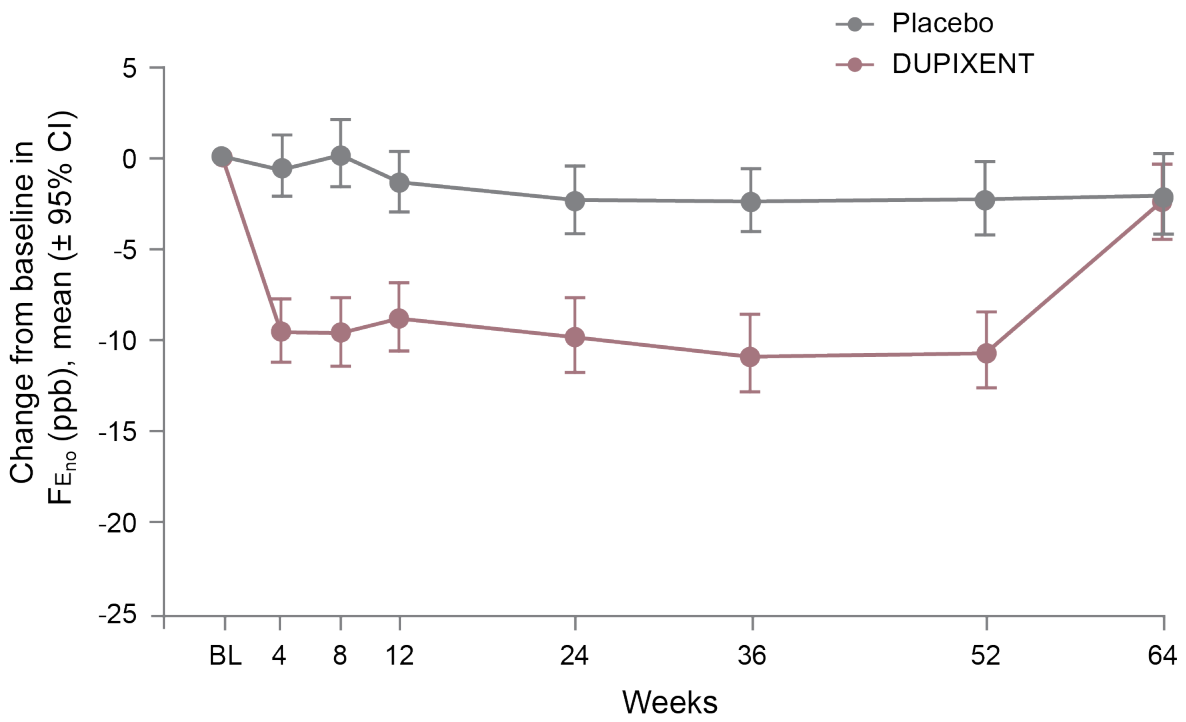
SOURCE: Bhatt SP et al NEJM 2023.

FeNO quickly responds to IL-4Rα inhibition in both asthma and COPD patients

IL-4Rα inhibition rapidly reduces FeNO in asthma patients



IL-4Rα inhibition rapidly reduces FeNO in COPD patients



FeNO is a robust biomarker of Type 2 inflammation in the lungs and quickly responds to therapy in both eosinophilic asthma and COPD

SOURCE: Rabe KF et al. NEJM 2018, Bhatt SP et al. NEJM 2023.

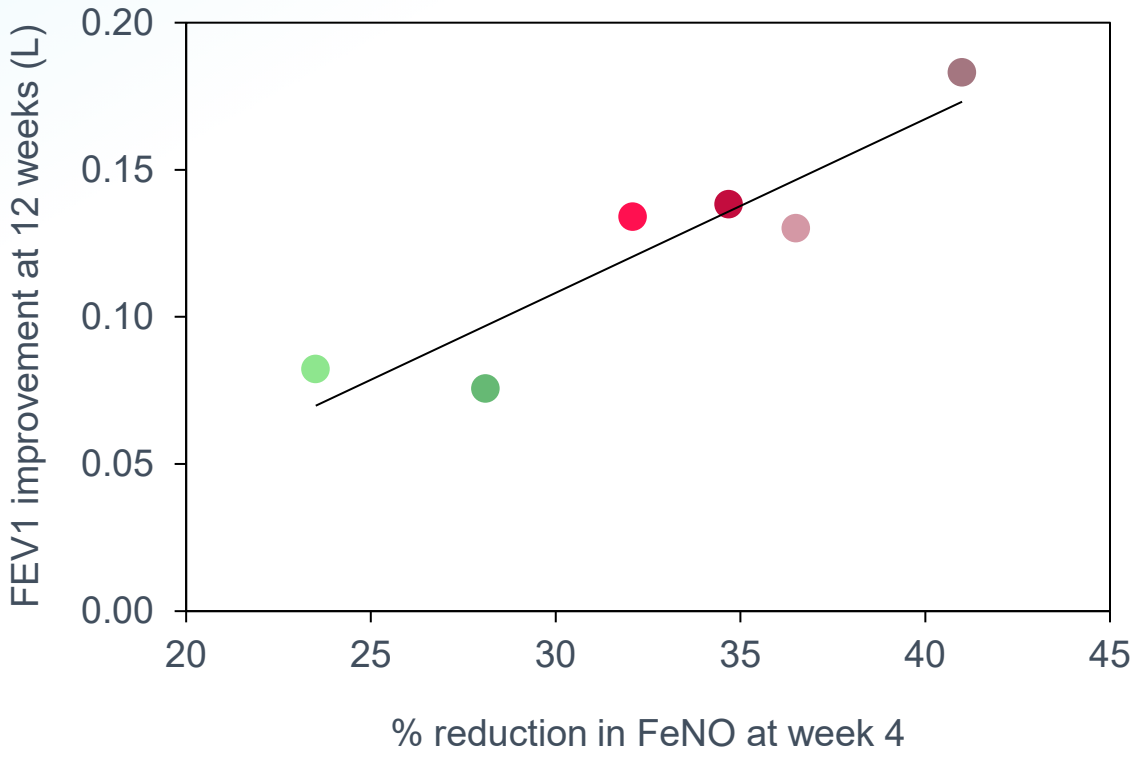
FeNO correlates well to FEV1 and exacerbations in asthma; DUPIXENT showed effect on all 3 in COPD

Lebrikizumab — LAVOLTA I[1], LAVOLTA II[1]

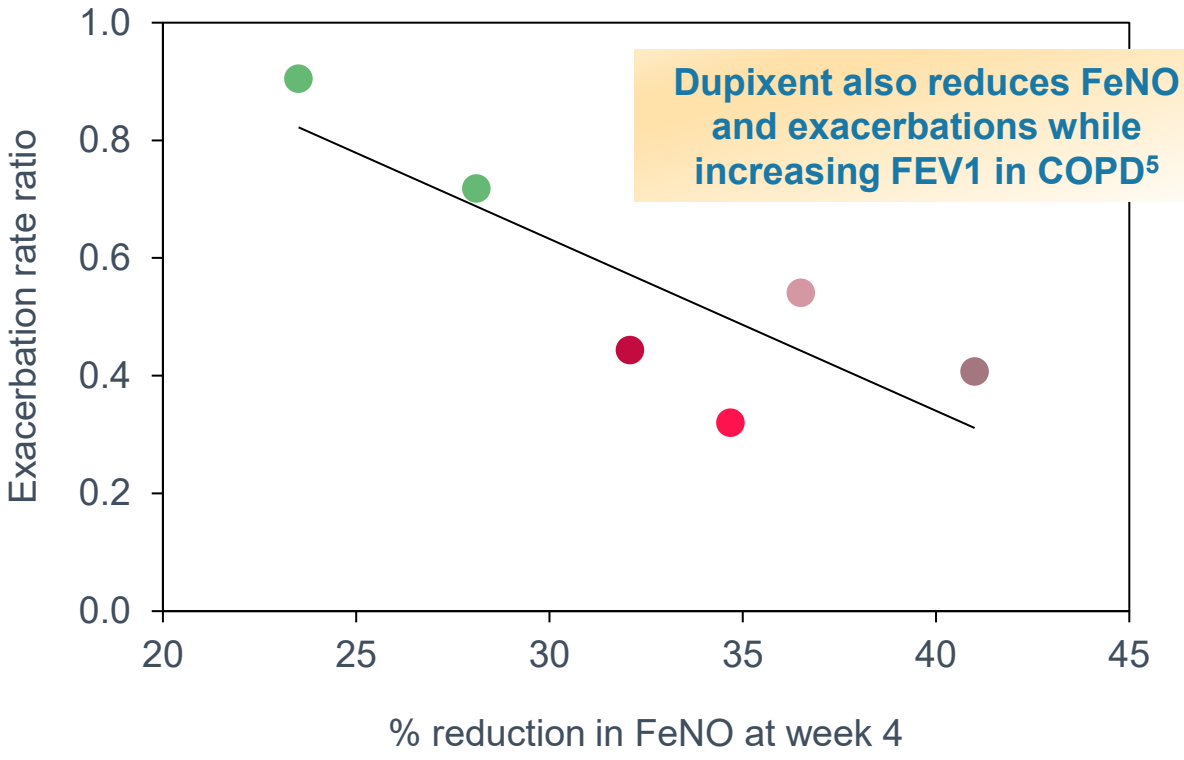
Tezspire — PATHWAY[2], NAVIGATOR[3]

Dupixent — VENTURE[4], QUEST[4]

FeNO correlates with lung function as measured by FEV1



FeNO correlates with exacerbations in asthma

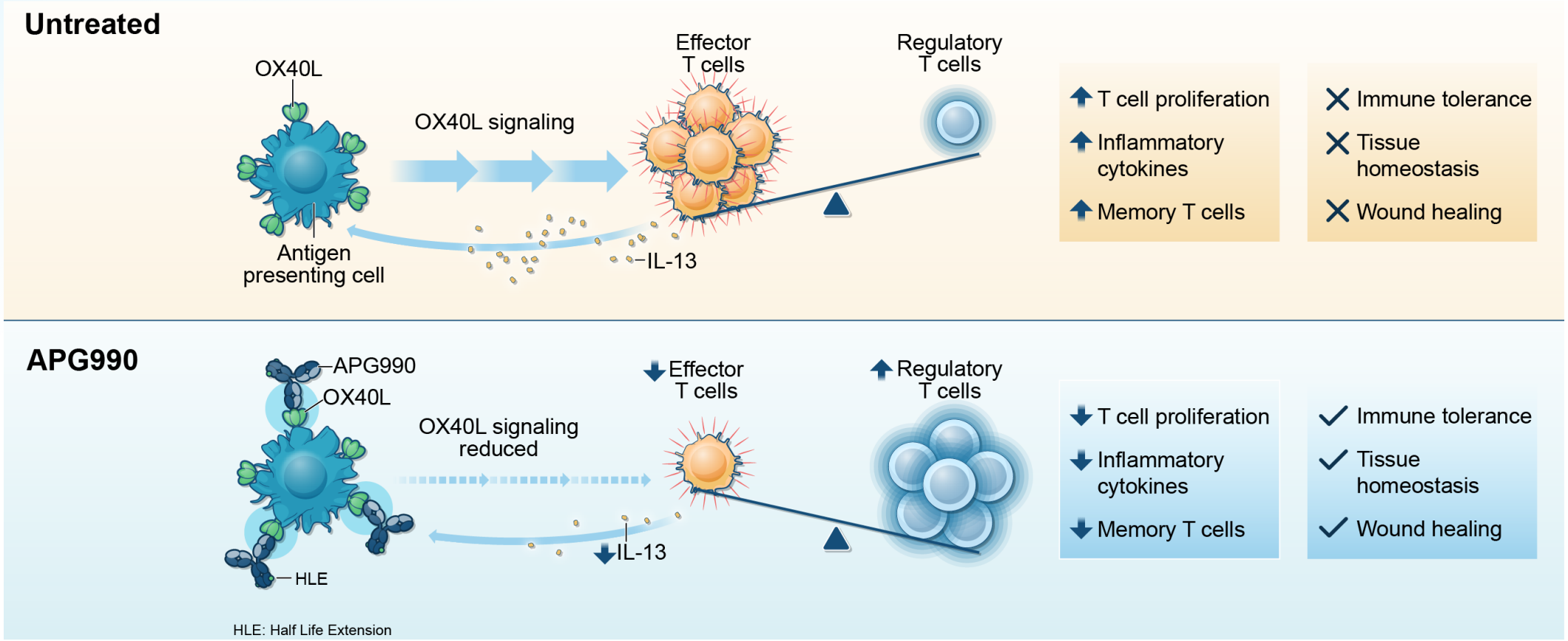


Dupixent also reduces FeNO and exacerbations while increasing FEV1 in COPD[5]

NOTE: Data shown for each trial corresponds to highest dose regimen tested, FeNO reductions are absolute change from baseline at 4 weeks except for QUEST, which reported 12-week data as earliest timepoint
SOURCE: 1) Hanania, N, et al. Lancet Respir Med, 2016. (average of high and low biomarker groups) 2) Menzies-Gow, A, et al. NEJM, 2021. 3) Corren ,J, et al. NEJM, 2019. 4) Castro, M, et al. NEJM, 2018. 5) Bhatt SP et al. NEJM 2023

APG990/APG222

APG990 blocks OX40L and potentially rebalances the immune system

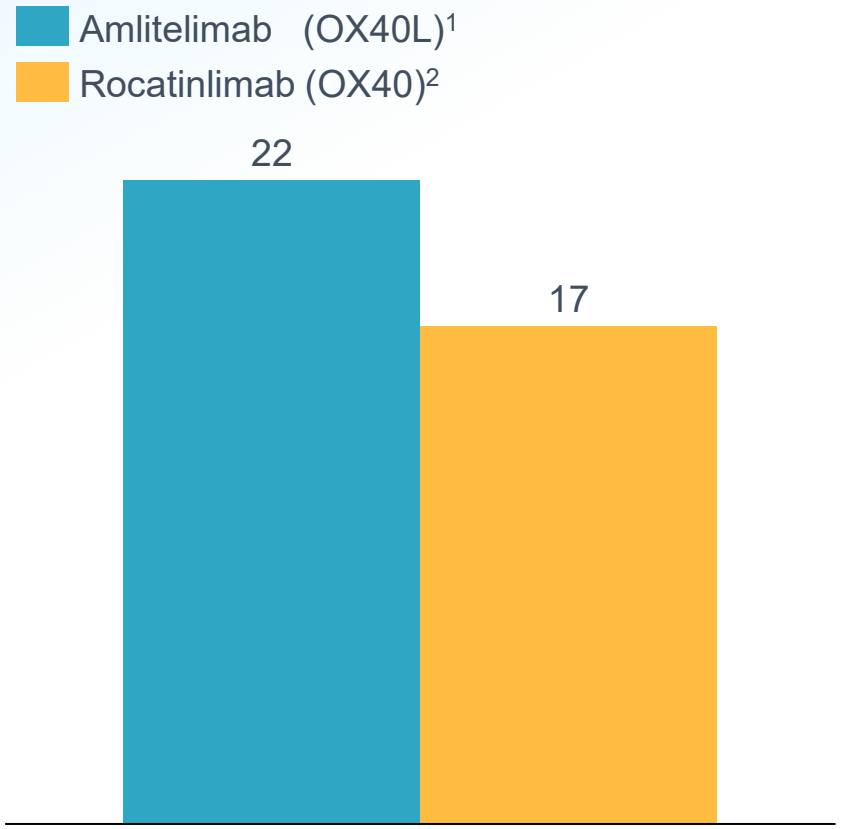


OX40L blockade targets Th2, Th17, and Th22 pathways, which have been implicated in numerous I&I conditions

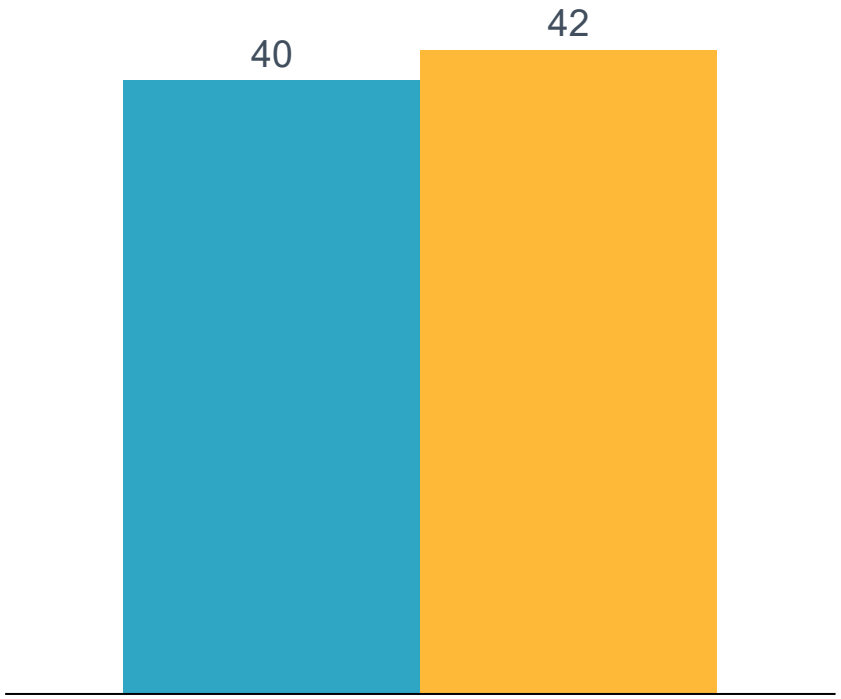
Upcoming clinical trial readouts could provide PoC for OX40L beyond AD including asthma, hidradenitis suppurativa, alopecia areata, celiac disease, and systemic sclerosis

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

IGA 0/1 response at Week 16



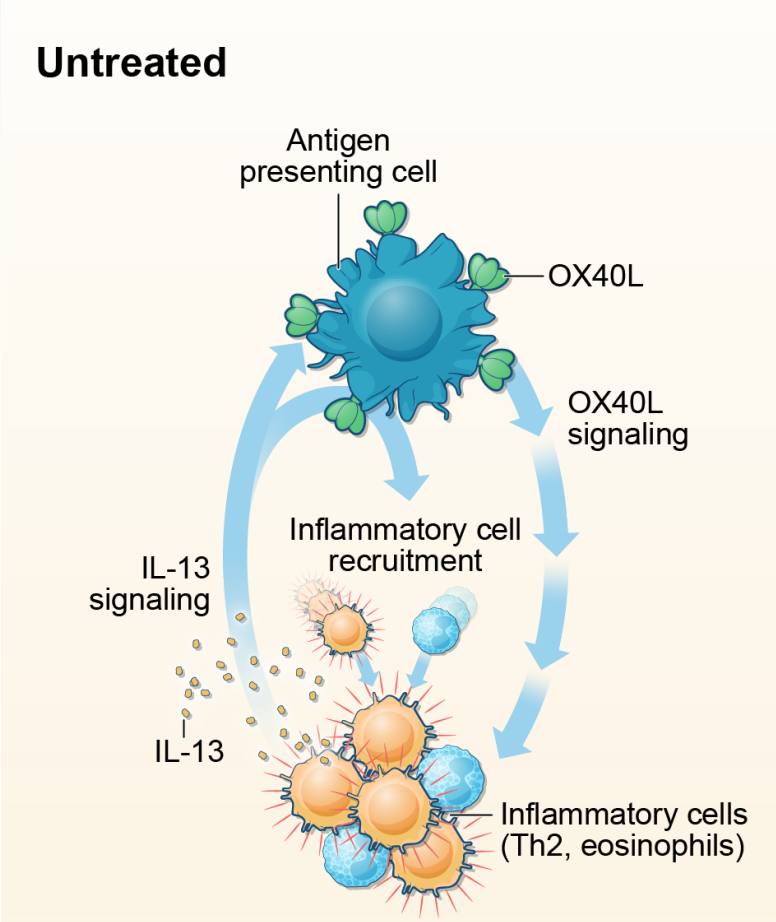
EASI-75 at Week 16

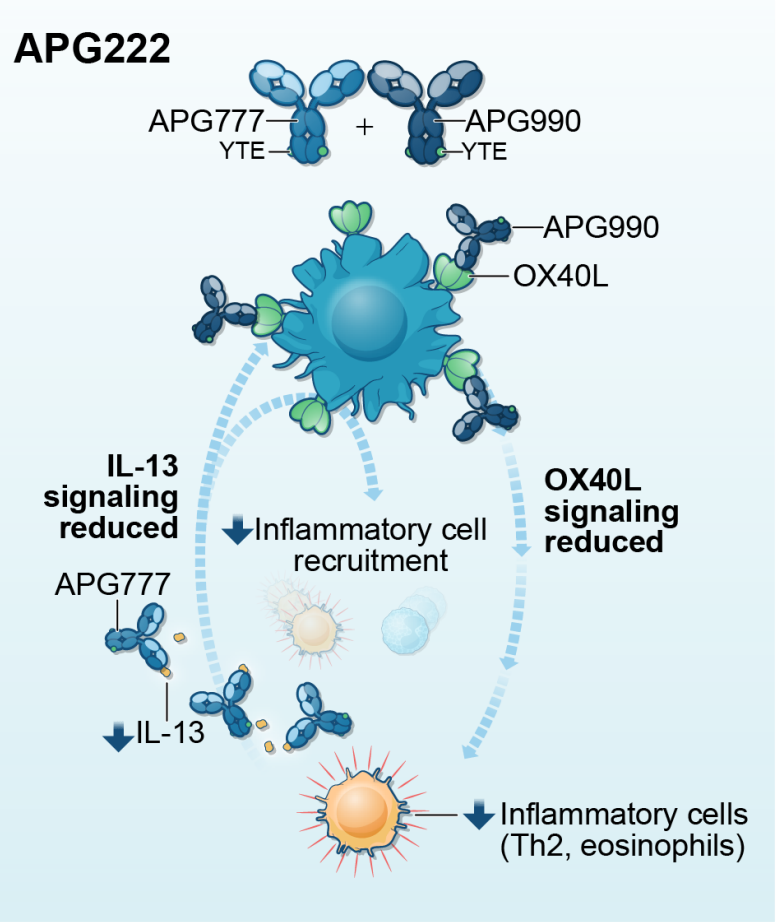


- In Phase 2b, **rocatinlimab (OX40) was associated with pyrexia** (17% of patients) **and chills** (11% of patients)
- In contrast, **no pyrexia[3] or chills for amlitelimab (OX40L)** in Phase 2b

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.
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".*

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

Corporate

Experienced team with proven history of clinical development and commercial execution

Michael Henderson, MD
Chief Executive Officer, Director

bridgebio, QED therapeutics (a bridgebio company), pellepharm (a bridgebio company), McKinsey & Company

Carl Dambkowski, MD
Chief Medical Officer

bridgebio, navire (a bridgebio company), QED therapeutics (a bridgebio company), origin (a bridgebio company), McKinsey & Company

Jane Pritchett Henderson
Chief Financial Officer

adagio, TURNSTONE BIOLOGICS, voyager THERAPEUTICS, kolltan Pharmaceuticals, Inc., LEHMAN BROTHERS, Salomon Brothers

Rebecca Dabora, PhD
Chief Development Officer

INVIVYD, SwanBio THERAPEUTICS, aspyrian Therapeutics, Inc., Biogen

Matt Batters, JD
Chief Legal Officer

ARVINAS, ALEXION, Skadden, DLA PIPER

Wendy Aspden-Curran
SVP of Clinical Operations

Pfizer, ARENA PHARMACEUTICALS, insmed

Drew Badger, PhD
SVP of Regulatory Affairs & Toxicology

CAPSIDA BIOTHERAPEUTICS, Dermira, Lilly, AMGEN

Dan Mulreany
SVP of Business Development & Strategy

QED therapeutics (a bridgebio company), bridgebio, BAIN & COMPANY, navire (a bridgebio company)

Kristine Nograles, MD, MSc
SVP of Clinical Development

CARA THERAPEUTICS, sparc, Celgene, MERCK, Bristol Myers Squibb

Board of Directors with industry-leading development, commercial and management expertise

Mark McKenna
Chairman

Prometheus Biosciences · Salix Pharmaceuticals · Bausch Health · Johnson & Johnson

Michael Henderson, MD
CEO, Apogee Therapeutics

bridgebio · QED therapeutics (a bridgebio company) · pellepharm (a bridgebio company) · McKinsey & Company

Jennifer Fox
CFO & CBO, Zenas BioPharma

Zenas BioPharma · citigroup · Nuvation Bio

Andrew Gottesdiener, MD
Venrock

venrock

Peter Harwin
Managing Member, Fairmount

Fairmount · Paragon Therapeutics

BJ Jones
CCO, NewAmsterdam Pharma

New Amsterdam Pharma · biohaven · AstraZeneca · Takeda

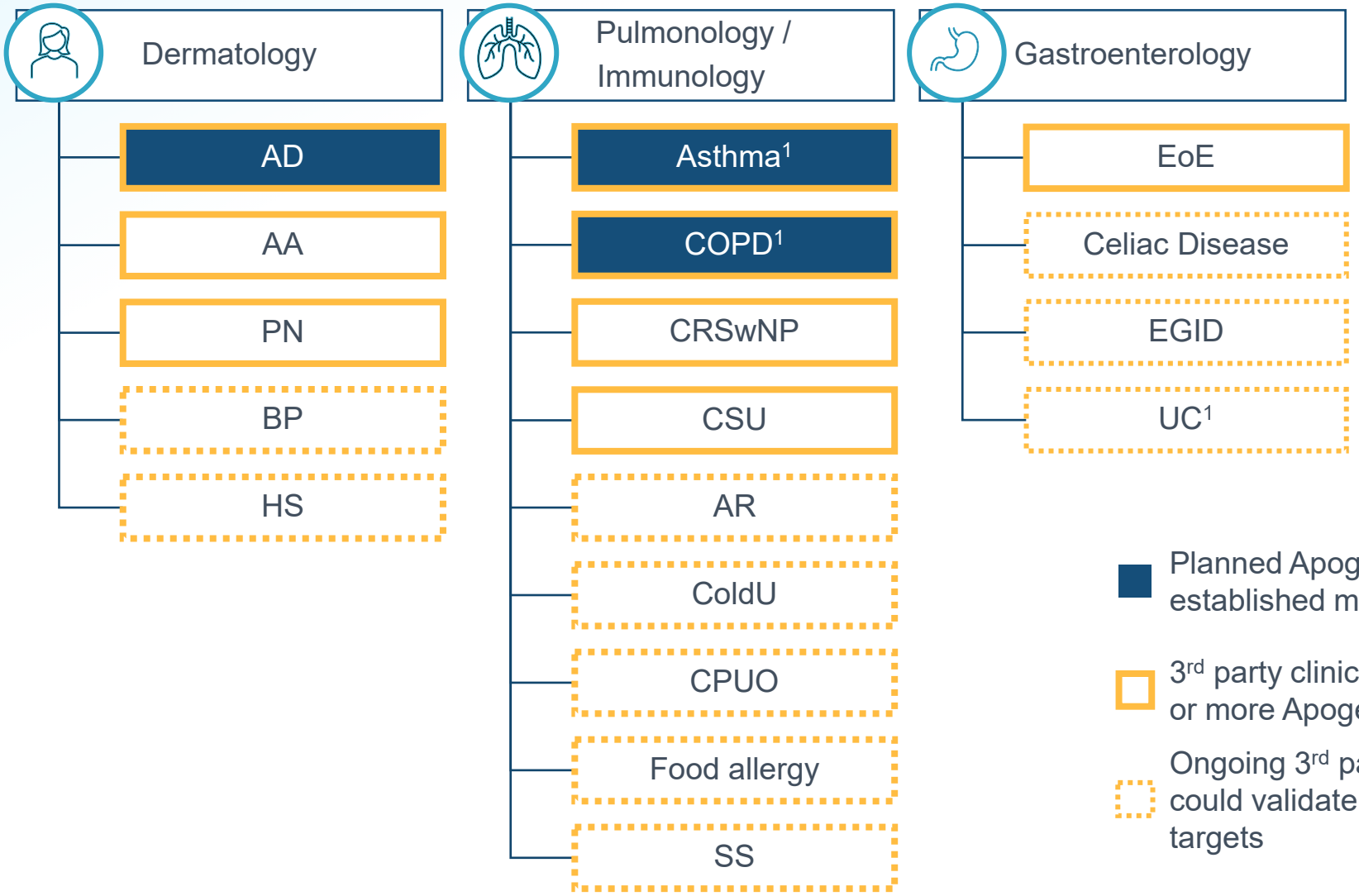
Tomas Kiselak
Managing Member, Fairmount

Fairmount · Paragon Therapeutics

Nimish Shah
Venrock

venrock

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



- 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

NOTE:[1] Eosinophilic subtypes
AA = Alopecia Areata. PN = Prurigo Nodularis. BP= Bullous Pemphigoid. HS = hidradenitis suppurativa. CSU = Chronic Spontaneous Urticaria. CRSwNP = Chronic Rhinosinusitis with Nasal Polyps ColdU = Cold Inducible Urticaria. CPUO = Chronic Pruritis of Unknown Origin.. AR = Allergic Rhinitis. SS= Systemic Sclerosis. EoE = Eosinophilic esophagitis. UC = Ulcerative Colitis. EGID = Eosinophilic Gastrointestinal Disorders (non-EoE)

Apogee plans to become a leader in I&I therapeutics

	2024	2025
APG777 (IL-13)	✓ **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-4Rα)	✓ **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 ± IL-13)	Candidate nomination	Phase 1 initiation in HVs
	✓ **$483M Follow-on** **2H: R&D Day**	

★ **Key readouts**

Apogee /ˈapəjē/ *noun*

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