APOGEE THERAPEUTICS

R&D Day 2024

December 2nd, 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 for our current and future product candidates and programs, our plans for current and future clinical trials, including a Phase 2 trial of APG 777 in atopic dermatitis, Phase1b and 2b trials of APG777 in asthma and a trial of APG777 in eosinophilic esophagitis, a Phase 1b trial of APG808 in asthma, a Phase 1 trial for APG990, a Phase 1 trial for APG333, and a clinical trial of the combination of APG777 and APG990; our plans for clinical trial design; the anticipated timing of the initiation of and results from our clinical trials, including data from our Phase 2 trial of APG777and our Phase 1 trial of APG990; the potential clinical benefit, half-life and dosing regimen of APG777, APG808, APG990, APG333 and any other potential programs, including the combinations of APG777 and APG990, and APG777 and APG333; our expected timing for future pipeline updates; our expectations regarding the time period over which our capital resources will be sufficient to fund our anticipated operations, and estimates of market size. In some cases, you can identify forward-looking statements by terms such as “anticipate,” “believe,” “can,” “could,” “design,” “estimate,” “expect,” “intend,” “likely,” “may,” “might,” “plan,” “potential,” “predict,” “suggest,” “target,” “will,” “would,” or the negative of these terms, and similar expressions intended to identify forward-looking statements. The forward-looking statements are based on our beliefs, assumptions and expectations of future performance, taking into account the information currently available to us. These statements are only predictions based upon our current expectations and projections about future events. Forward-looking statements are subject to known and unknown risks, uncertainties and other factors that may cause our actual results, level of activity, performance or achievements to be materially different from those expressed or implied by such forward-looking statements, including those risks described in “Risk Factors” and “Management’s Discussion and Analysis of Financial Condition and Results of Operations” in our Annual Report on Form 10-K for the year ended December 31, 2023, filed with the SEC on March 5, 2024, our Quarterly Report on Form 10-Q for the quarter ended September 30, 2024, filed with the SEC on November 12, 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.

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Agenda

Our vision for building a next-gen biotech

APG808 Phase 1 Interim Results

Building a leading franchise in AD

- APG777 demonstrates best-in-class PK and path to annual dosing
- Broader cytokine targeting can better address heterogeneity in AD *(Emma Guttman-Yassky, MD, PhD)*
- IL-13+OX40L combination has the potential to raise the bar in AD

Breaking through the efficacy ceiling in asthma and COPD

- Alarmins and Type 2 cytokines in obstructive airway disease *(Dave Singh, MD, FERS, FBPhS)*
- IL-13+TSLP combination targets both central and local drivers of obstructive airway disease

Commercial opportunity and strategy

Closing remarks and Q&A

Today's invited speakers

Emma Guttman-Yassky MD, PhD
Mount Sinai

Professor Dave Singh MD, FERS, FBPhS
University of Manchester

Michael Henderson, MD
Chief Executive Officer

Carl Dambkowski, MD
Chief Medical Officer

Rebecca Dabora, PhD
Chief Development Officer

Jeff Hartness
Chief Commercial Officer

Kristine Nograles, MD, MSc
SVP, Clinical Development

Lukas Dillinger, PhD
VP, Research and Translational Medicine

Amol Kamboj, MD
VP, Clinical Development

Noël Kurdi
VP, Investor Relations

Our vision for building a next-gen biotech

Michael Henderson, MD
Chief Executive Officer

Apogee plans to transform the standard-of-care for I&I diseases

Novel antibodies engineered against validated targets

- Potential higher exposures and longer half-lives could improve efficacy and transform dosing
- Expected novel IP into the mid-2040s[1]

First biotech to pursue combination approaches in the largest I&I markets

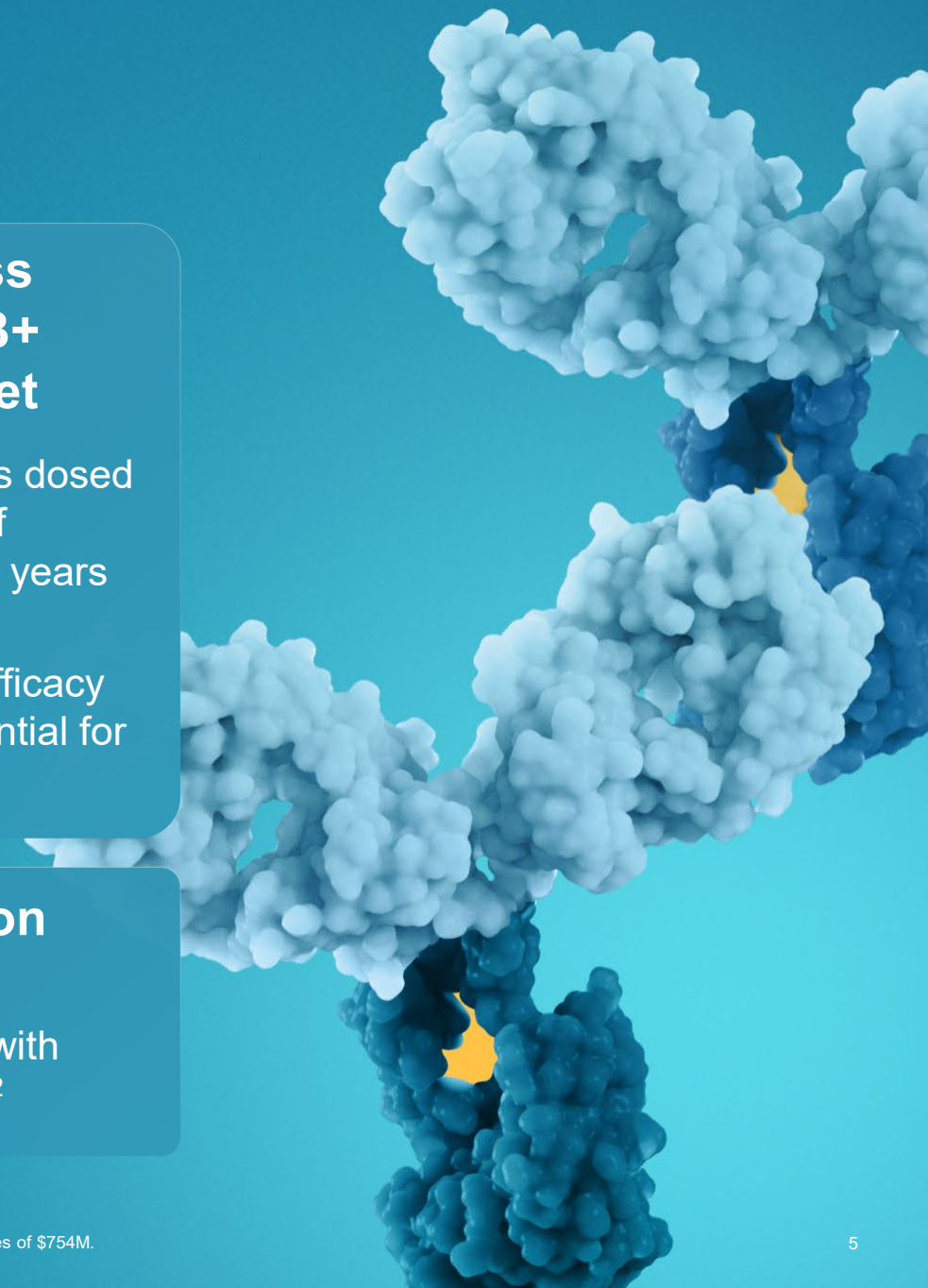
- AD combo trial expected to start 2025, asthma + COPD to follow

Potentially best-in-class therapy for future $50B+ atopic dermatitis market

- Market leader, DUPIXENT, is dosed every 2 weeks; nearly half of patients discontinue within 2 years
- APG777 Phase 2 could demonstrate best-in-class efficacy signal in mid-2025 with potential for annual dosing

Strong financial position

- $754m total cash providing expected runway into 2028 with multiple near-term catalysts[2]



NOTE: [1] Based on current applications issuing as patents. [2] As of September 30, 2024, Apogee had cash, cash equivalents and marketable securities of $754M.

Apogee's approach is to achieve differentiated efficacy and dosing for validated targets

STRATEGY	PROGRAM	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3
Potential best-in-class monotherapy Higher exposures for better efficacy with less frequent dosing	**APG777** IL-13	Atopic Dermatitis				**Mid-2025:** Phase 2 16-week induction PoC readout
		Asthma				**2025: 1H** Phase 1b trial initiation **2H** Phase 2b trial initiation
		Eosinophilic Esophagitis				**2026:** Phase 2 trial initiation
Potential first- or best-in-class combination approaches	**APG777+APG990** IL-13 OX40L	Atopic Dermatitis				**2025:** Phase 1b PoC trial initiation (against DUPIXENT)
	APG777+APG333 IL-13 TSLP	Asthma				**2025:** Additional clinical plan announced
		COPD				**2025:** Additional clinical plan announced

APG808 IL-4Rα ⟶ **Ph1b in asthma** readout expected in 1H 2025

APG990 OX40L ⟶ **Phase 1** healthy volunteer trial readout expected in 1H 2025

APG333 TSLP ⟶ **Phase 1** healthy volunteer trial initiation expected by EOY 2024

The Apogee agents mentioned above are currently under investigation. Their safety and effectiveness for the listed target indications have not yet been established.

Announced at R&D Day (Dec 2)

APG808
IL-4Rα

Phase 1 initial data exceeded objectives

Well-tolerated at all doses tested

Optimized PK profile enables up to **every 2-month dosing**

Extended PD effect: near complete pSTAT6 inhibition at ~3 months supports path to **every 3-month dosing**

APG777
IL-13

Program continues to show best-in-class potential

Updated Phase 1 data supports path to **annual dosing**

Ph2 Part A readout accelerated to **mid-2025**

Initiation of trials in asthma and EoE expected over next 12-18 months

APG777+APG990
IL-13 OX40L

Preclinical POC achieved; moving into clinic in 2025

Broader impact on inflammation vs. monotherapy in preclinical assays

Coformulation POC achieved

Planned Ph1b against DUPIXENT in AD: expected readout in 2026

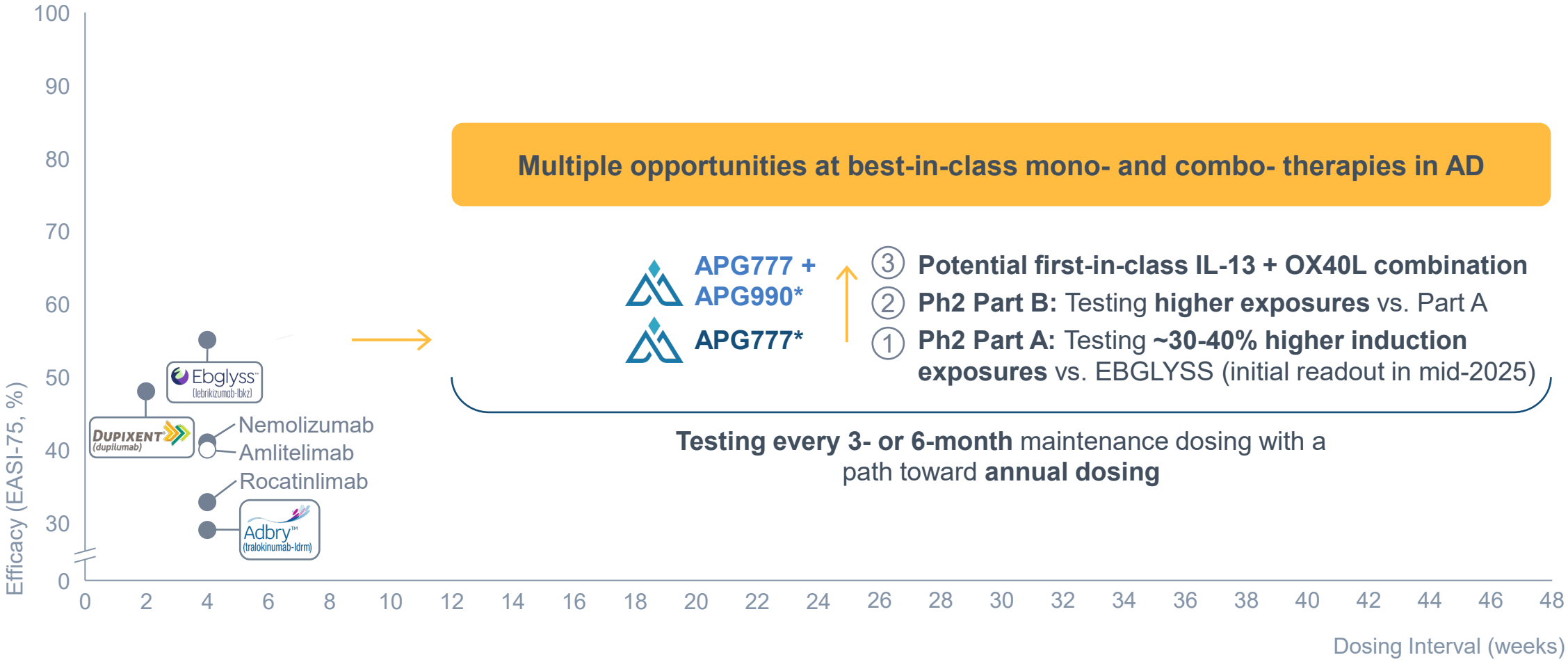
APG777+APG333
IL-13 TSLP

Preclinical POC achieved; clinical planning underway

Broader impact on drivers of respiratory disease vs. monotherapy in preclinical assays

Combo-enabling monotherapy asthma studies expected to initiate in 2025

Apogee is potentially the first in atopic dermatitis to provide transformational dosing and efficacy



NOTE: *Positioning of Apogee programs is illustrative and based on interim Phase 1 results for APG777 only and illustrates what we believe we can potentially achieve. Only DUPIXENT, ADBRY, and EBGLYSS are approved in the US. 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] EBGLYSS 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] Nemolizumab 30 mg Q4W Ph3 avg. Silverberg J et al EADV 2023. [5] Rocatinlimab 150mg Q4W Ph2b Guttman-Yassky E et al Lancet 2023. [6] Amlitelimab 250mg Q4W Ph2b Weidinger S et al EADV oral presentation 2023.

Our vision for building a next-gen biotech

APG777 in AD: Best-in-class monotherapy

- **Potential megablockbuster** in the future **$50B+ AD market**
- **Accelerated mid-2025 Ph2 POC readout** testing higher induction exposures for potentially better efficacy and every 3- or 6-month dosing
- **Path to annual dosing**

APG777: Pipeline-in-a-product

- Path to leadership in **10+ potential expansion indications** starting with:
  - **Asthma** Ph2b initiation expected **in 2025**
  - **EoE** Ph2 initiation expected **in 2026**

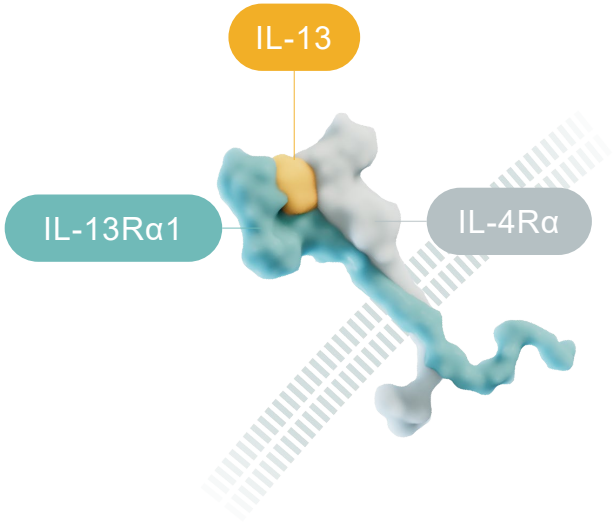
Best-in-class combinations

- **Potential to break through the monotherapy efficacy ceiling** via rational combos
- **Combos rapidly advancing** behind 777 mono with even greater **pipeline-in-a-product potential**:
  - **777+990: Ph1b against DUPIXENT** initiation expected in 2025; readout expected in 2H 2026
  - **777+333: asthma and COPD** clinical planning underway

APG808 Phase 1 Interim Results

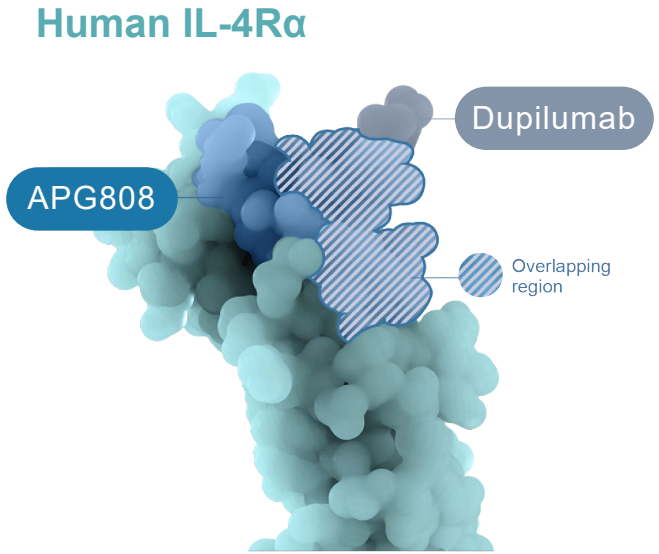
Carl Dambkowski, MD
Chief Medical Officer

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

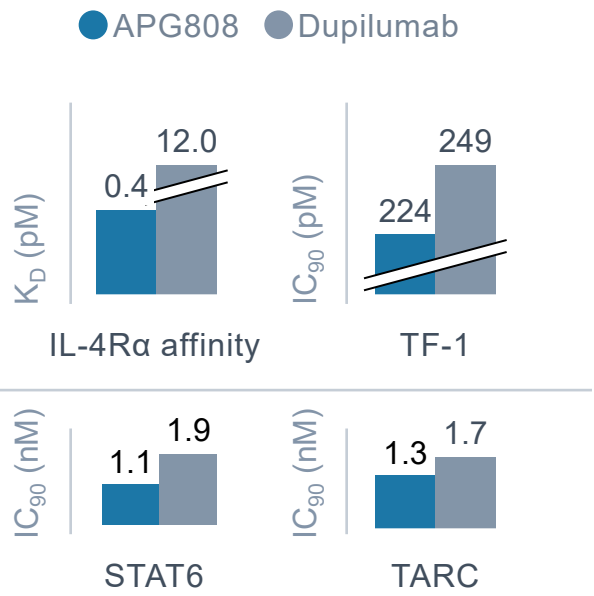


Heterodimerization

APG808 disrupts Type 2 inflammation by **preventing formation** of IL-13Rα1 / IL-4Rα heterodimer



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



APG808 has **30x higher affinity** for IL-4Rα vs. dupilumab

NOTE: MoA = Mechanism of Action. Dupilumab is the active ingredient in DUPIXENT. Affinity data shown for APG808 and dupilumab are from different preclinical experiments conducted at different points in time. Based on DUPIXENT FDA Pharmacology Review, dupilumab binds to recombinant soluble monomeric human IL4Rα with a KD of ~ 30 pM and to dimeric human IL4Rα with a KD of ~ 12 pM.

APG808 Phase 1 initial data exceeded trial objectives

GOAL

Confirm tolerable **safety profile**

RESULT

Doses up to 1200mg tested and **well-tolerated**

✓ ACHIEVED

GOAL

Establish optimized **PK profile**

At least a 42-day half-life

RESULT

~55-day half-life[1]

>5x longer half-life than DUPIXENT[2]

✓ EXCEEDED

GOAL

Determine **dosing regimens**

Equal DUPIXENT exposure with every 6-week or longer dosing[3]

RESULT

Up to every 2-month dosing enabled with modeled exposures comparable to DUPIXENT[3]

✓ EXCEEDED

GOAL

Supplemental

Demonstrate effect on biomarkers pSTAT6 and TARC

RESULT

Extended PD effect provides path to **every 3-month dosing**

Near complete pSTAT6 inhibition for 3 months and **deeper TARC reduction** vs DUPIXENT

✓ EXCEEDED

NOTE: PK = Pharmacokinetic

[1] Half-life dependent on dose and dosing frequency; 55-day half-life based on model simulated 4mL Q6W high concentration formulation at steady-state, calculated from linear portion of the model, where nonlinear elimination is fully saturated. [2] DUPIXENT PDMA Review reported 8.77-day half-life for highest single SQ dose cohort (600mg) and may represent nonlinear and linear elimination. [3] Exposure target based on C_{trough} in maintenance, the minimal concentration of APG808 to have similar exposures to DUPIXENT, based on mean observed Ctrough,ss for dupilumab in adults with asthma (69.0 µg/mL) and COPD (61.8 µg/mL) in two clinical trials reported in DUPIXENT European Public Assessment Report (EMEA/H/C/004390/II/0079).

APG808 Phase 1 in Healthy Volunteers

APG808 interim data from ongoing Phase 1 trial in healthy volunteers

Trial design elements

Double-blind, placebo-controlled, first-in-human trial
Single ascending dose in healthy participants

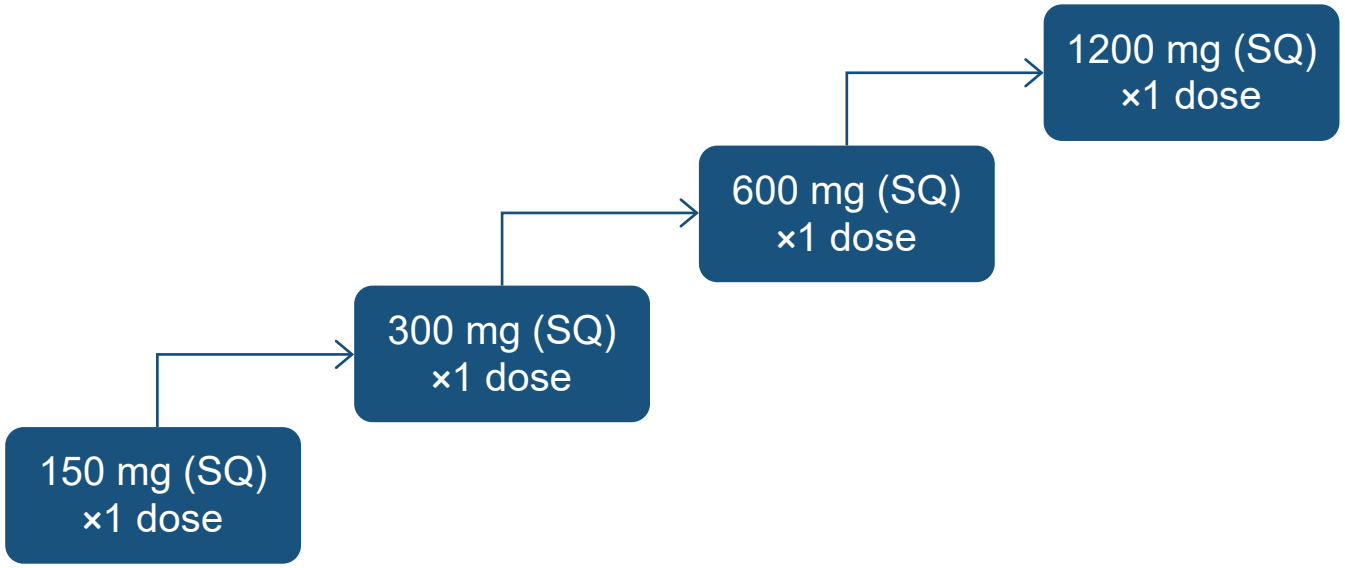
N ~ 32
8 per cohort (6:2 active:placebo)

Key inclusion criteria: healthy adult participants

Primary endpoint: safety

Secondary endpoints: PK, ADA

Single ascending dose



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

NOTE: SQ = subcutaneous.

Baseline characteristics are in line with expectations

Phase 1 Single Ascending Dose: By Cohort

	Placebo N=8	Cohort 1 150 mg N=6	Cohort 2 300 mg N=6	Cohort 3 600 mg N=6	Cohort 4 1,200 mg N=6
Age (yrs), mean (SD)	46.6 (15.4)	48.5 (12.9)	32.0 (10.6)	41.3 (14.8)	41.7 (14.1)
Female	62.5%	66.7%	83.3%	33.3%	66.7%
Caucasian	75.0%	83.3%	50.0%	66.7%	100%
Weight (kg), mean (SD)	74.5 (15.0)	73.3 (17.1)	75.8 (19.7)	82.1 (15.2)	81.0 (18.9)

Demographics were well balanced across cohorts

APG808 was well-tolerated with a favorable safety profile, as expected for the mechanism

	Phase 1 Single Ascending Dose: By Cohort					Overall	
N (%)	Placebo N=8	Cohort 1 150 mg N=6	Cohort 2 300 mg N=6	Cohort 3 600 mg N=6	Cohort 4 1,200 mg N=6	Placebo N=8	APG808 N=24
≥1 TEAE	5 (62.5%)	4 (66.7%)	5 (83.3%)	4 (66.7%)	4 (66.7%)	5 (62.5%)	17 (70.8%)
≥1 serious TEAE	0	0	0	1 (16.7%)*	0	0	1 (4.2%)*
≥1 Grade 3 TEAE	0	0	0	1 (16.7%)*	0	0	1 (4.2%)*
≥1 drug-related TEAE	1 (12.5%)	1 (16.7%)	0	2 (33.3%)	2 (33.3%)	1 (12.5%)	5 (20.8%)
≥1 drug-related serious TEAE	0	0	0	0	0	0	0
≥1 drug-related Grade 3 TEAE	0	0	0	0	0	0	0

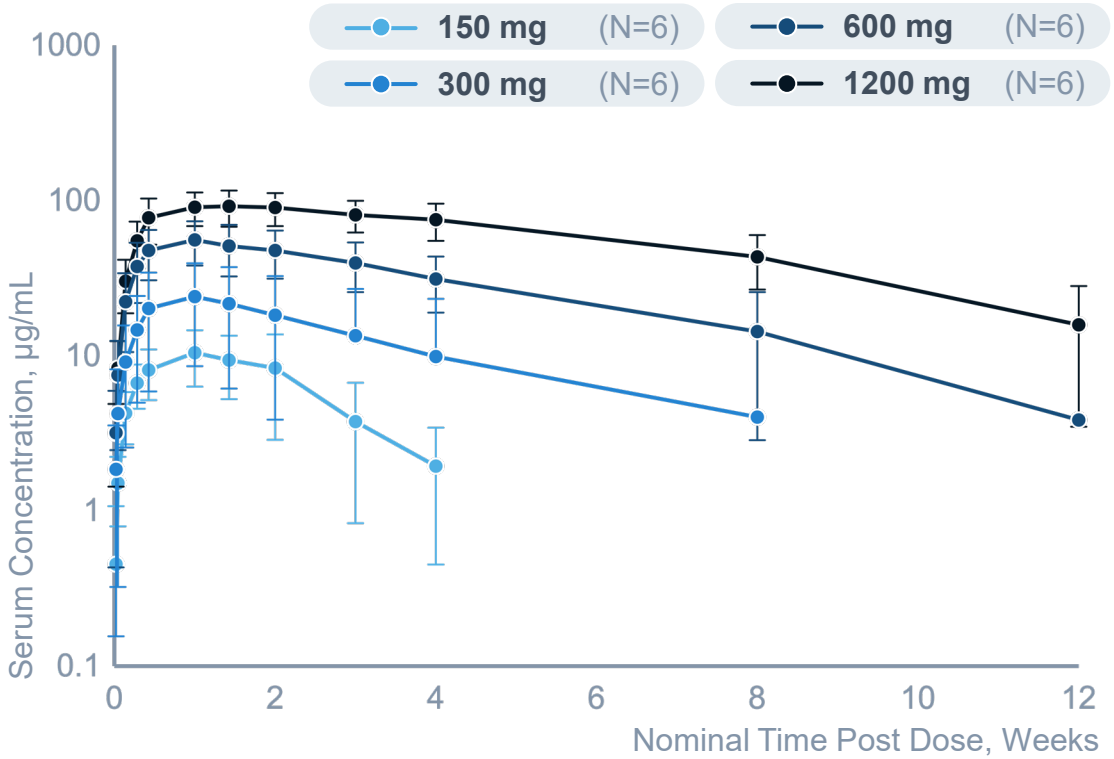
The safety profile is in line with expectations for therapies targeting IL-4Rα

NOTE: TEAE = Treatment-Emergent Adverse Event. Interim data includes AEs reported as of 5 November 2024 data cut. The trial is ongoing.

*SAE for Grade 3 non-cardiac chest pain onset D55 that resolved in 1 day without acute intervention. Non-cardiac chest pain was deemed as not related to study drug and likely related to dyspepsia or musculoskeletal causes.

APG808 exhibited a potentially best-in-class PK profile with a ~55-day half-life at or above target exposure

Single-dose concentration-time profile[1]



APG808 half-life was >5x longer than DUPIXENT

APG808

~55 days[2]

DUPIXENT

~9 days[3]

>5x longer half-life

PK profile enables up to every 2-month dosing, 4-8x less frequent than DUPIXENT's every 1- to 2-week dosing

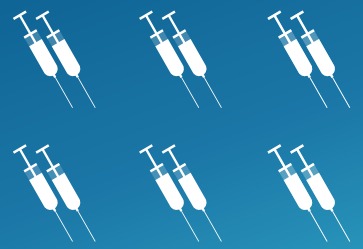
NOTE: PK = Pharmacokinetic.
[1] Mean (SD) profiles. [2] Half-life dependent on dose and dosing frequency; 55-day half-life based on model simulated 4mL Q6W high concentration formulation at steady-state, calculated from linear portion of the model, where nonlinear elimination is fully saturated. [3] DUPIXENT PDMA Review reported 8.77-day half-life for highest single SQ dose cohort (600mg) and may represent nonlinear and linear elimination. No DUPIXENT PK data has been published for single SQ doses higher than 600mg.

Modeled APG808 every 2-month exposures are comparable to DUPIXENT

APG808 every 2-months

6

Annual injection days



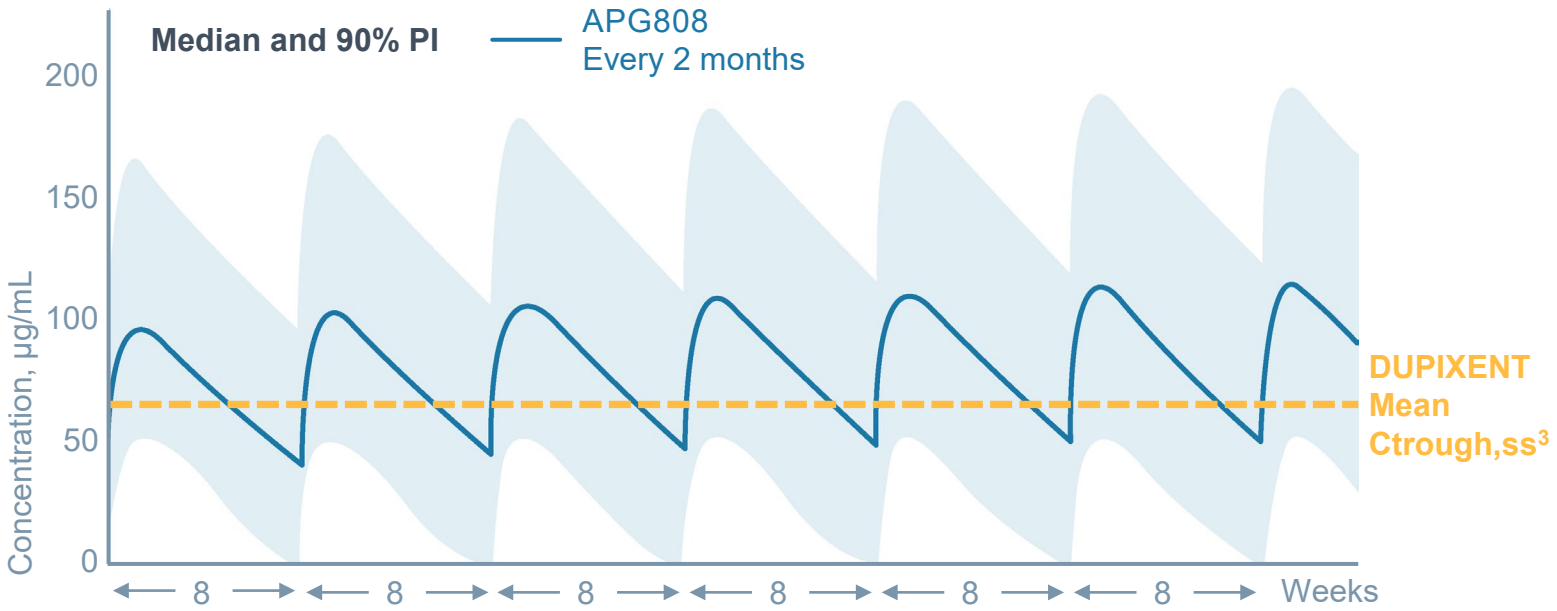
VS.

DUPIXENT every 2-weeks

26

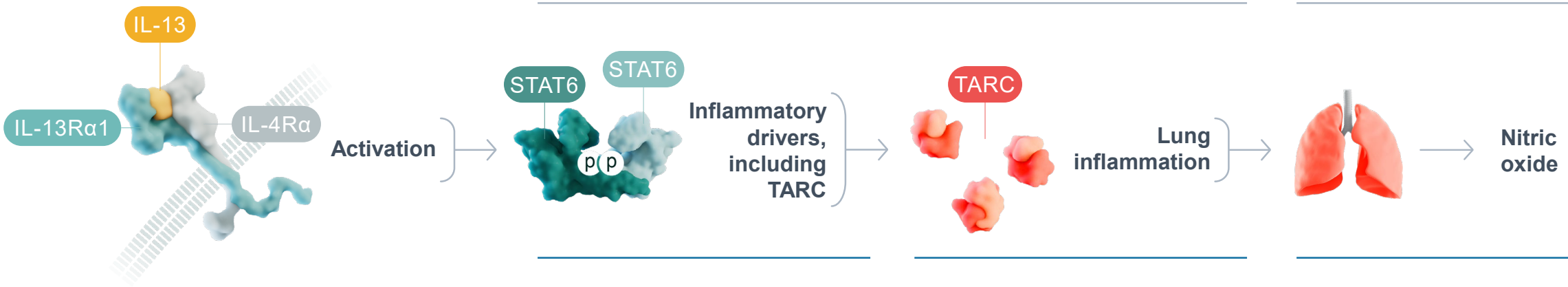
Annual injection days

Modeled concentration based on APG808 Phase 1 PK[1,2]



[1] Modeled dose based on 4mL high concentration formulation. [2] Solid blue line represents population PK (PPK) model predicted median concentrations of APG808 for Q2M dosing regimen, shaded blue area represents 90% prediction interval (PI). [3] Based on mean observed Ctrough,ss for dupilumab in adults with asthma (69.0 µg/mL) and COPD (61.8 µg/mL) in two clinical trials reported in DUPIXENT European Public Assessment Report (EMEA/H/C/004390/II/0079).
NOTE: The above graph is illustrative only with respect to plans for dosing of APG808 and does not present comparative data.

pSTAT6 and TARC are biomarkers of IL-4Rα target engagement and pathway activation



APG808 Phase 1 HV biomarkers

pSTAT6 is a proximal and sensitive marker of IL-4Rα pathway activation[1]

Suppressed TARC release validates **target engagement** in healthy volunteers

APG808 Phase 1b asthma biomarker

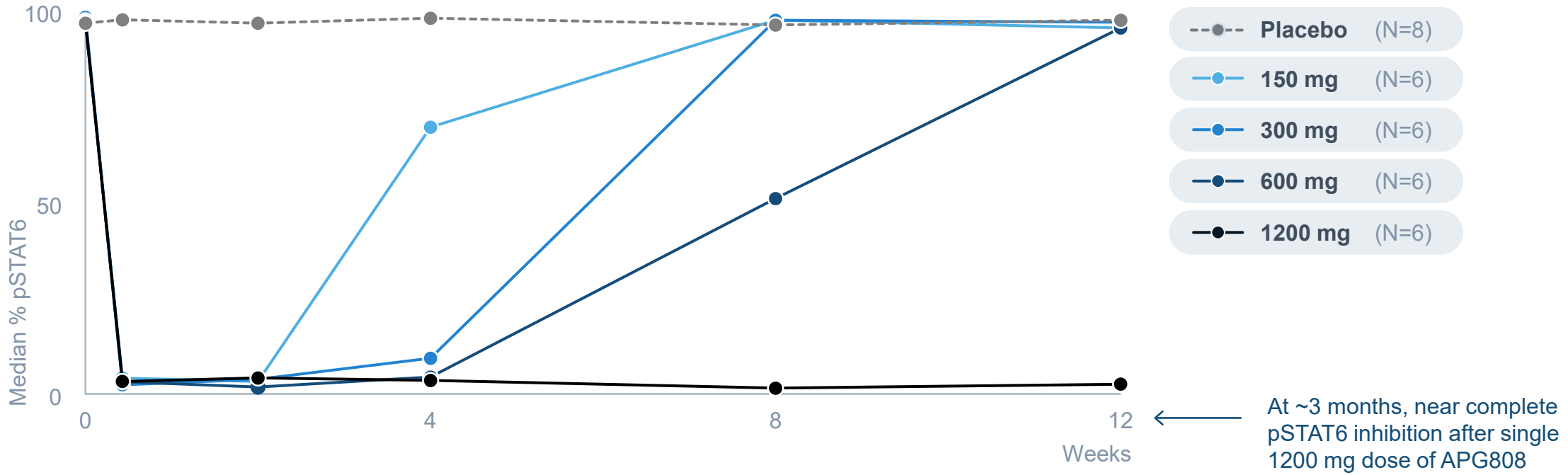
FeNO (fractional exhaled nitric oxide) is a **biomarker of Th2 inflammation** and will be assessed in the Phase 1b in patients with asthma

APG808's reduction of these biomarkers confirm inhibition of IL-4Rα signaling and allows comparison to other agents

SOURCE: [1] McCormick SM and Heller NM. Cytokine 2015;75.

APG808 showed near complete pSTAT6 inhibition for ~3 months at the top dose

Median % pSTAT6

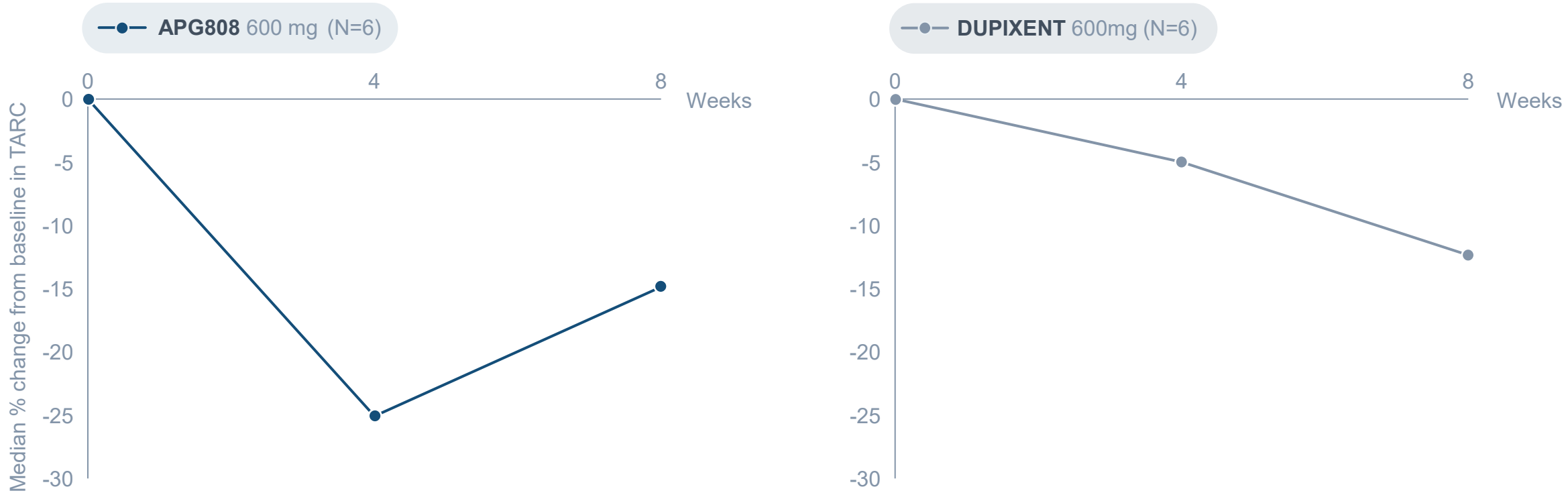


Near complete pSTAT6 inhibition for ~3 months provides path to every 3-month dosing based on extended PD effect resulting from APG808's >5x greater half-life[1] and 30x higher affinity[2] for IL-4Rα vs. DUPIXENT

NOTE: No data has been published showing DUPIXENT impact on pSTAT6 in HVs. pSTAT6 measured using flow cytometry of whole blood samples stimulated with 10 ng/mL IL-13 (approximately 100 times the level of IL-13 present in the sputum of severe asthma patients). Similar results were obtained for pSTAT6 measured following stimulation with IL-4.
[1] Half-life dependent on dose and dosing frequency; 55-day half-life based on model simulated 4mL Q6W high concentration formulation at steady-state, calculated from linear portion of the model, where nonlinear elimination is fully saturated. DUPIXENT PDMA Review reported 8.77-day half-life for highest single SQ dose cohort (600mg) and may represent nonlinear and linear elimination. [2] 30X higher affinity of APG808 versus DUPIXENT (dupilumab) based on different preclinical experiments conducted at different points in time; DUPIXENT affinity based on FDA Pharmacology Review.
SOURCE: Saha S et. al. J Allergy Clin Immunol 2008.

Single dose of APG808 led to deeper TARC reduction compared to DUPIXENT

Median % change from baseline in TARC



APG808 shows greater depth of TARC reduction compared to DUPIXENT at most relevant APG808 dose

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 one Phase 1 trial with 6 healthy volunteers receiving a single SQ injection of 600 mg DUPIXENT. APG808 data derived from our Phase 1 trial in 6 healthy volunteers receiving a single SQ injection of 600 mg of APG808. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted. APG808 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, 57 (TDU12265).

APG808 positive interim Phase 1 readout is a de-risking milestone for program and pipeline

Antibody attributes

- **Clinically validated** IL-4Rα target
- **Overlapping epitope** with DUPIXENT
- **30x greater affinity for IL-4Rα vs. DUPIXENT**[4] and at least equivalent potency across relevant pre-clinical assays

Clinical profile

- **PK data enables up to every 2-month dosing:**
  - ~55-day half-life[1], allowing comparable exposure to DUPIXENT with 4-8x fewer injection days[2,3]
- **Extended PD effect** provides path to **every 3-month dosing**
  - Near complete pSTAT6 inhibition for ~3 months after a single dose
- **Well tolerated**

NOTE: [1] Half-life dependent on dose and dosing frequency; 55-day half-life based on model simulated 4mL Q6W high concentration formulation at steady-state, calculated from linear portion of the model, where nonlinear elimination is fully saturated. [2] DUPIXENT PDMA Review reported 8.77-day half-life for highest single SQ dose cohort (600mg) and may represent nonlinear and linear elimination. [3] Exposure target based on C_{trough} in maintenance, the minimal concentration of APG808 to have similar exposures to DUPIXENT, based on mean observed Ctrough,ss for DUPIXENT in adults with asthma (69.0 µg/mL) and COPD (61.8 µg/mL) in two clinical trials reported in DUPIXENT European Public Assessment Report (EMEA/H/C/004390/II/0079). [4] 30X higher affinity of APG808 versus DUPIXENT (dupilumab) based on different preclinical experiments conducted at different points in time. DUPIXENT affinity based on FDA Pharmacology Review.

APG808 Phase 1b in asthma patients

APG808 Phase 1b in asthma patients on track for readout in 1H 2025

Design elements for Phase 1b trial

Double-blind, placebo-controlled
Single dose regimen in patients with asthma

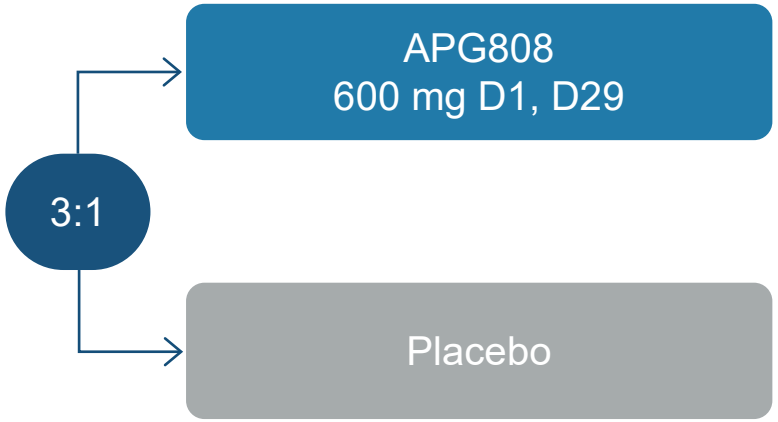
N ~ 20

Key inclusion criteria:
- Mild-to-moderate asthma
- FeNO >25 ppb

Primary endpoint: safety

Additional endpoints: PK, ADA, fractional exhaled nitric oxide (FeNO)

Schematic for Phase 1b trial



Study objectives

Confirm safety of APG808 as monotherapy in **asthmatic patient population**

Demonstrate activity of APG808 via **maximal suppression of FeNO in line with standard of care** (~10-15 parts per billion change from baseline)

Show **durable suppression of FeNO supporting every 2-month dosing or less frequent**

APG777 continues to show best-in-class potential

Carl Dambkowski, MD
Chief Medical Officer

APG777 Phase 1 continues to exceed all trial objectives; Phase 2 readout accelerated to mid-2025

UPDATE

Final SAD data confirms **safety & PK** profile from interim readout

OUTCOME

- APG777 continues to be **well-tolerated**
- **Half-life of 77 days**

UPDATE

Extended follow-up demonstrates durability of inhibition

OUTCOME

- **Extended PD effect:** Near complete pSTAT6 inhibition for **12 months**

UPDATE

Part A, with induction regimen designed to **exceed EBGLYSS exposures,** is **enrolling strongly**

OUTCOME

- **16-week PoC readout accelerated to mid-2025**

UPDATE

Part A tests every 3- or 6-month maintenance dosing with exposures matched to EBGLYSS[1] and is ongoing

OUTCOME

- **52-week topline readout anticipated in 1H 2026**

NOTE: PK = Pharmacokinetic.
[1] Exposure target based on C_{trough} in maintenance, the minimal concentration of APG777 to have similar exposures to EBGLYSS.

APG777 Phase 1 SAD complete; MAD fully enrolled with 9-months follow-up

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)

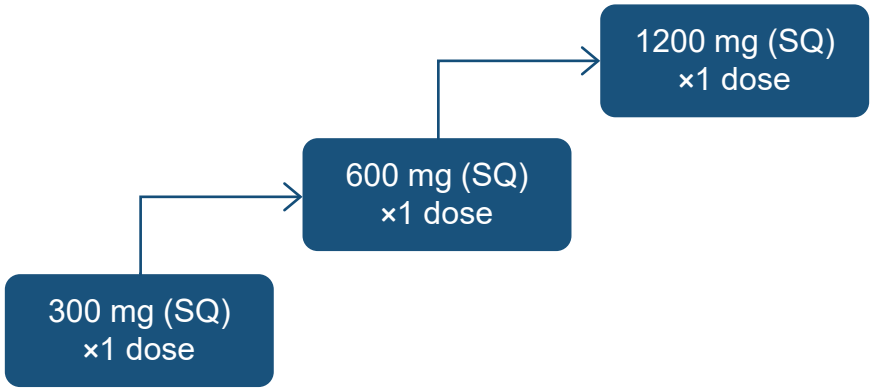
Key inclusion criteria: healthy adult volunteers

Primary endpoint: safety

Secondary endpoints: PK, ADA

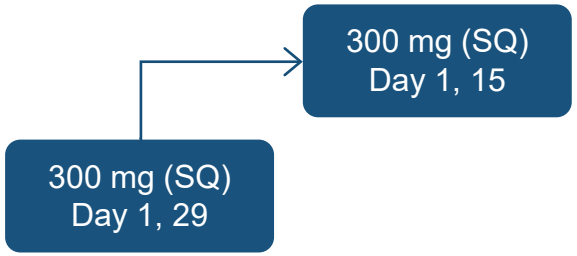
Exploratory biomarkers: pSTAT6, TARC

Single ascending dose



SAD cohorts complete – 12 months of follow-up on all cohorts

Multiple ascending dose



MAD cohorts with 9 months follow-up

APG777 continues to be well-tolerated and as expected for class

	Single dose				Multiple dose			Overall trial	
N (%)	Placebo N=6	Cohort 1 300 mg N=6	Cohort 2 600 mg N=6	Cohort 3 1200 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	Placebo N=10	APG777 N=30
≥1 TEAE	5 (83.3%)	5 (83.3%)	6 (100%)	3 (50.0%)	3 (75.0%)	5 (83.3%)	6 (100%)	8 (80.0%)	25 (83.3%)
≥1 serious TEAE	0	0	0	0	0	0	0	0	0
≥1 drug-related TEAE	3 (50.0%)	1 (16.7%)	1 (16.7%)	1 (16.7%)	1 (25.0%)	1 (16.7%)	2 (33.3%)	4 (40.0%)	6 (20.0%)
≥1 Grade 3 TEAE	0	0	1 (16.7%)*	0	0	0	0	0	0
Discontinued study due to TEAE	0	0	0	0	0	0	0	0	0
Decreased dose due to TEAE	0	0	0	0	0	0	0	0	0

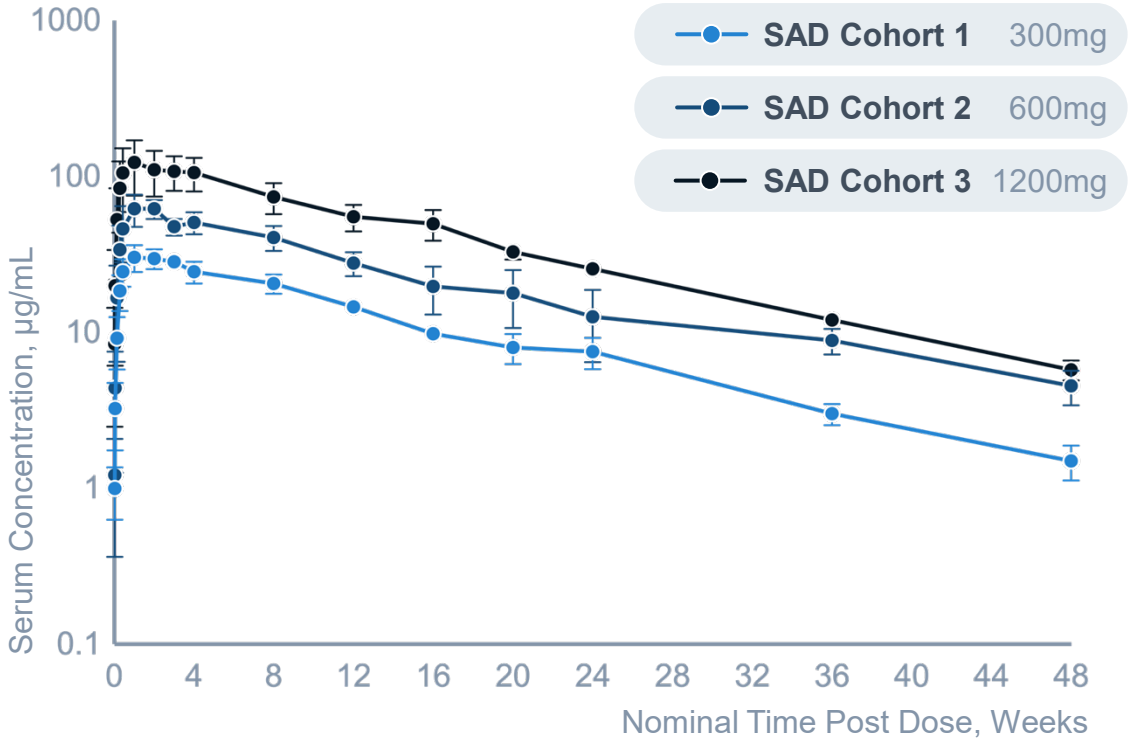
The safety profile is in line with expectations for therapies targeting the IL-13 pathway

NOTE: TEAE = Treatment-Emergent Adverse Event. TE-SAE = Treatment-Emergent-Serious Adverse Event. Interim data includes AEs reported as of 12 September 2024 data cut. The trial is ongoing.

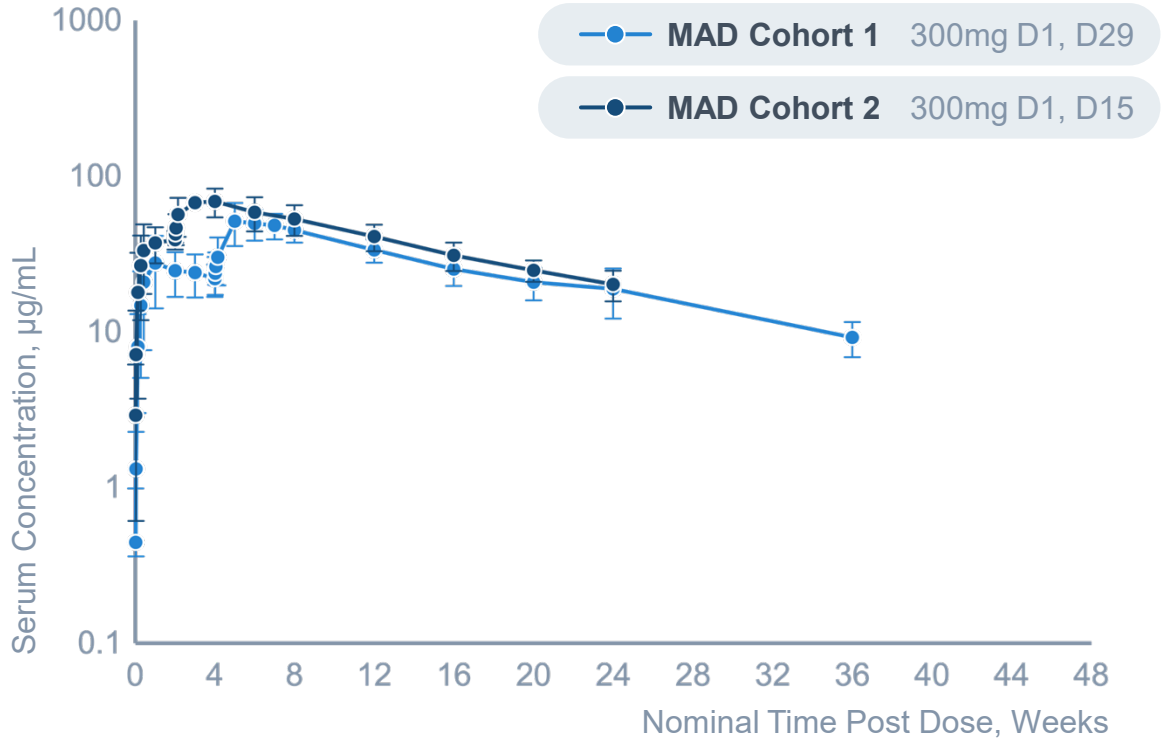
*Transient Grade 3 AST elevation (7.6 – 8.3x ULN) due to rhabdomyolysis from exercise from Days 253-256 of the study. TEAE was deemed not related to study drug.

APG777 Phase 1 SAD demonstrated best-in-class PK profile with a half-life of 77 days

Single-dose concentration-time profile[1]



Multi-dose concentration-time profile[1]

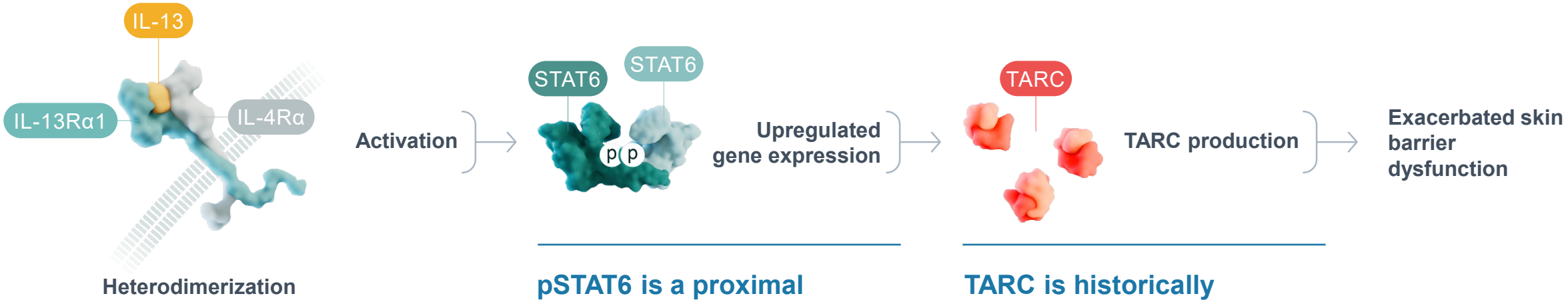


PK demonstrated dose-proportionality and half-life of 77 days (approximately 3-5x approved biologics)

NOTE: PK = Pharmacokinetic
[1] Mean (SD) profiles.

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

APG777 Phase 1 biomarkers



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

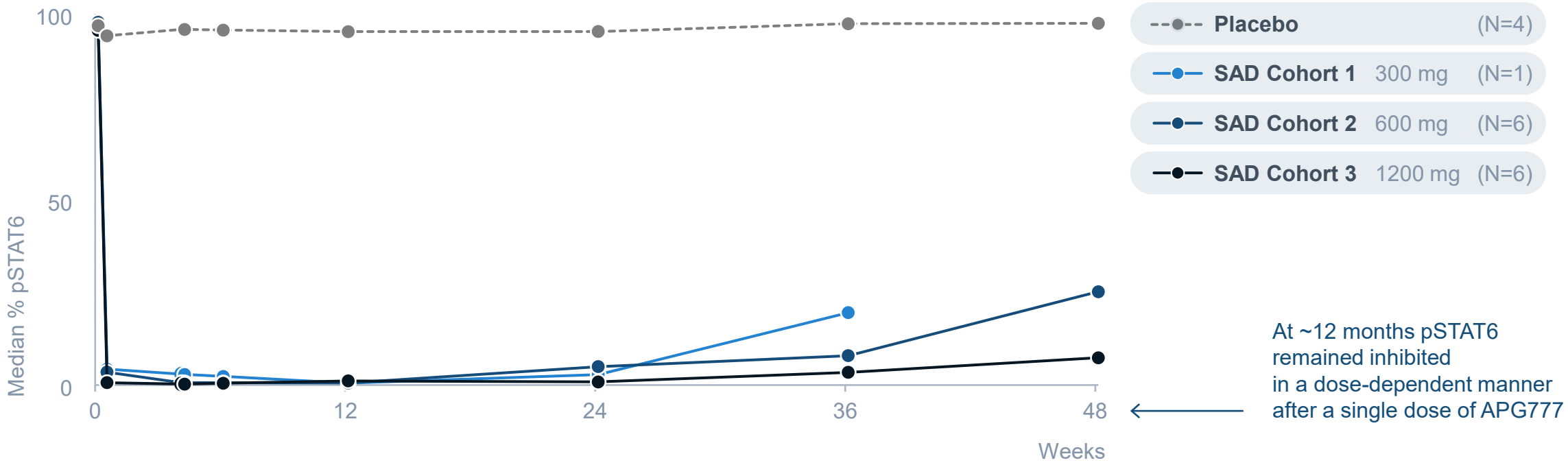
TARC is historically correlated with AD severity and initial treatment response[2]

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

SOURCE: [1] McCormick SM and Heller NM. Cytokine 2015;75. [2] Jahnz-Rozyk K, et al. Allergy 2005;60.

Single dose of APG777 showed extended pSTAT6 inhibition for up to 12 months

Median % pSTAT6



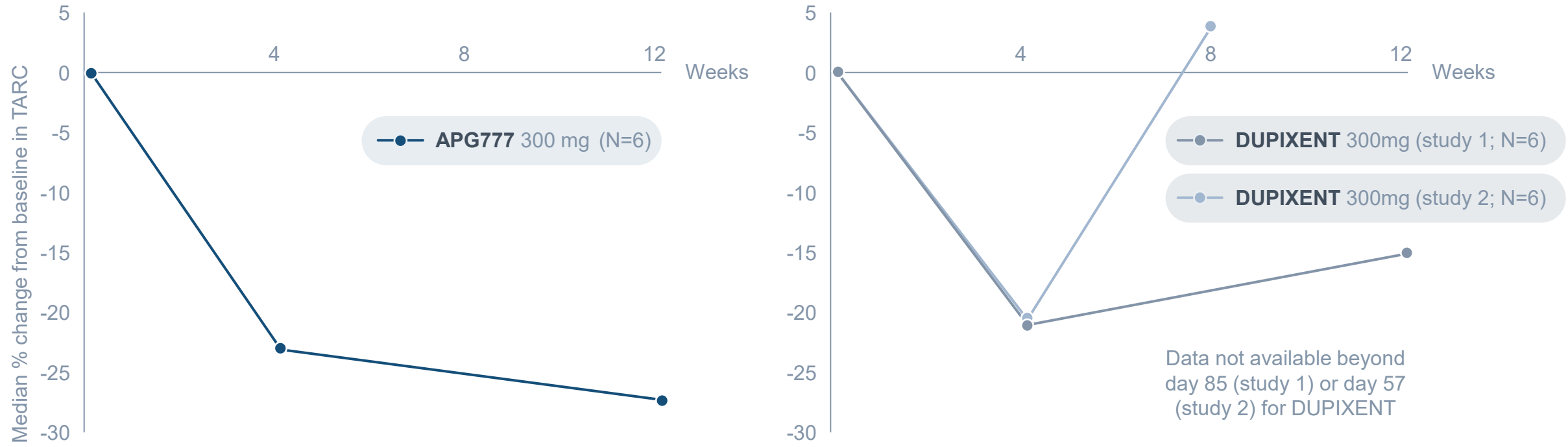
At ~12 months pSTAT6 remained inhibited in a dose-dependent manner after a single dose of APG777

Near complete pSTAT6 inhibition up to ~12 months at the top dose supports the potential for annual dosing

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 EBGLYSS impact on pSTAT6 in HVs. pSTAT6 measured using flow cytometry of whole blood samples stimulated with 10 ng/mL IL-13 (approximately 500 times the level of IL-13 present in lesional skin of moderate-severe AD patients).
SOURCE: Szegedi K et. al. JEADV 2015.

Single dose of APG777 led to deeper and more sustained TARC reduction vs DUPIXENT

Median % change from baseline in TARC

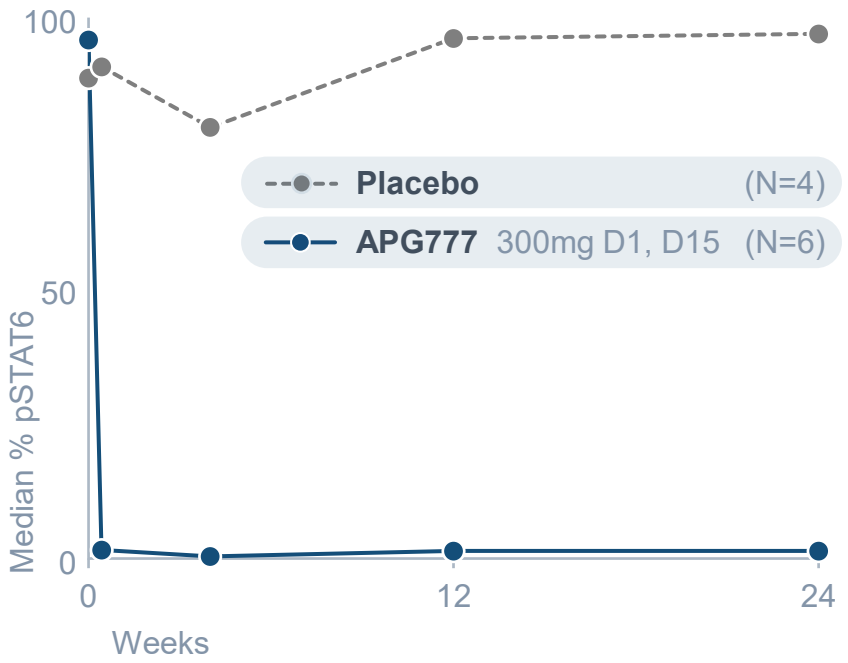


- **300 mg APG777 showed similar maximum PD marker changes as DUPIXENT**
- **APG777's sustained TARC reduction demonstrates the potential for better durability**

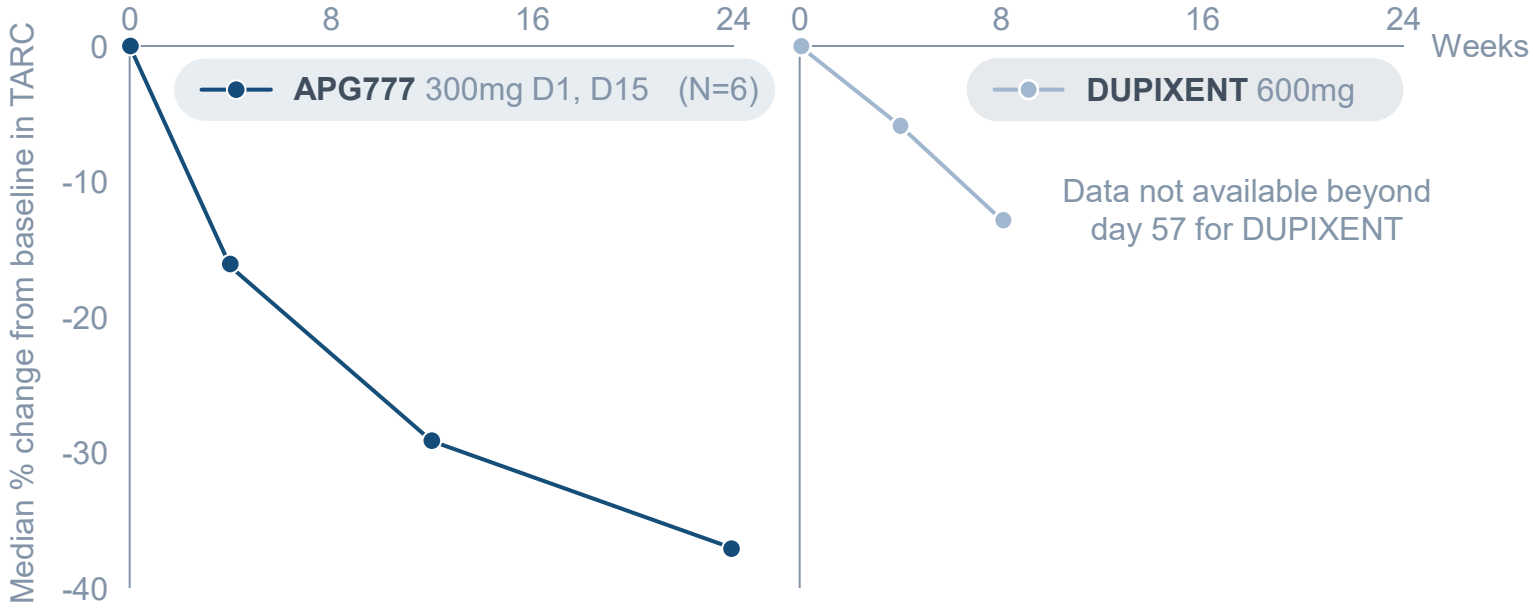
NOTE: These data are derived from different clinical trials conducted at different points in time, with differences in trial design, dosing regimen and patient populations. DUPIXENT data derived from two separate Phase 1 trials, each with 6 healthy volunteers receiving a single SQ injection of 300 mg DUPIXENT. APG777 data derived from our Phase 1 trial in 6 healthy volunteers receiving a single SQ 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 (study 1; R668-AS-0907) and 1, 29, 57 (study 2; TDU12265). No data has been published showing EBGLYSS impact on TARC in HVs.

Multiple doses of APG777 showed near complete pSTAT6 inhibition and deep TARC inhibition up to 6-months (limit of available follow-up)

Median % pSTAT6



Median % changes from baseline in TARC



APG777 shows greater depth of TARC reduction compared to the same total dose of DUPIXENT

NOTE: No data has been published showing DUPIXENT or EBGLYSS impact on pSTAT6 in HVs. pSTAT6 measured using flow cytometry of whole blood samples stimulated with 10 ng/mL IL-13 (approximately 500 times the level of IL-13 present in lesional skin of moderate-to-severe AD patients). TARC 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 one Phase 1 trial with 6 healthy volunteers receiving a single SQ injection of 600 mg DUPIXENT. APG777 data derived from our Phase 1 trial in 6 healthy volunteers receiving two SQ 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. No data has been published showing EBGLYSS impact on TARC in HVs.
SOURCE: Li, Z, et al. ACCP, 2020. Data for time points on nominal day post dose 1, 29, 57 (TDU12265).

APG777 Phase 2 induction exposures designed to exceed EBGLYSS for potentially greater efficacy

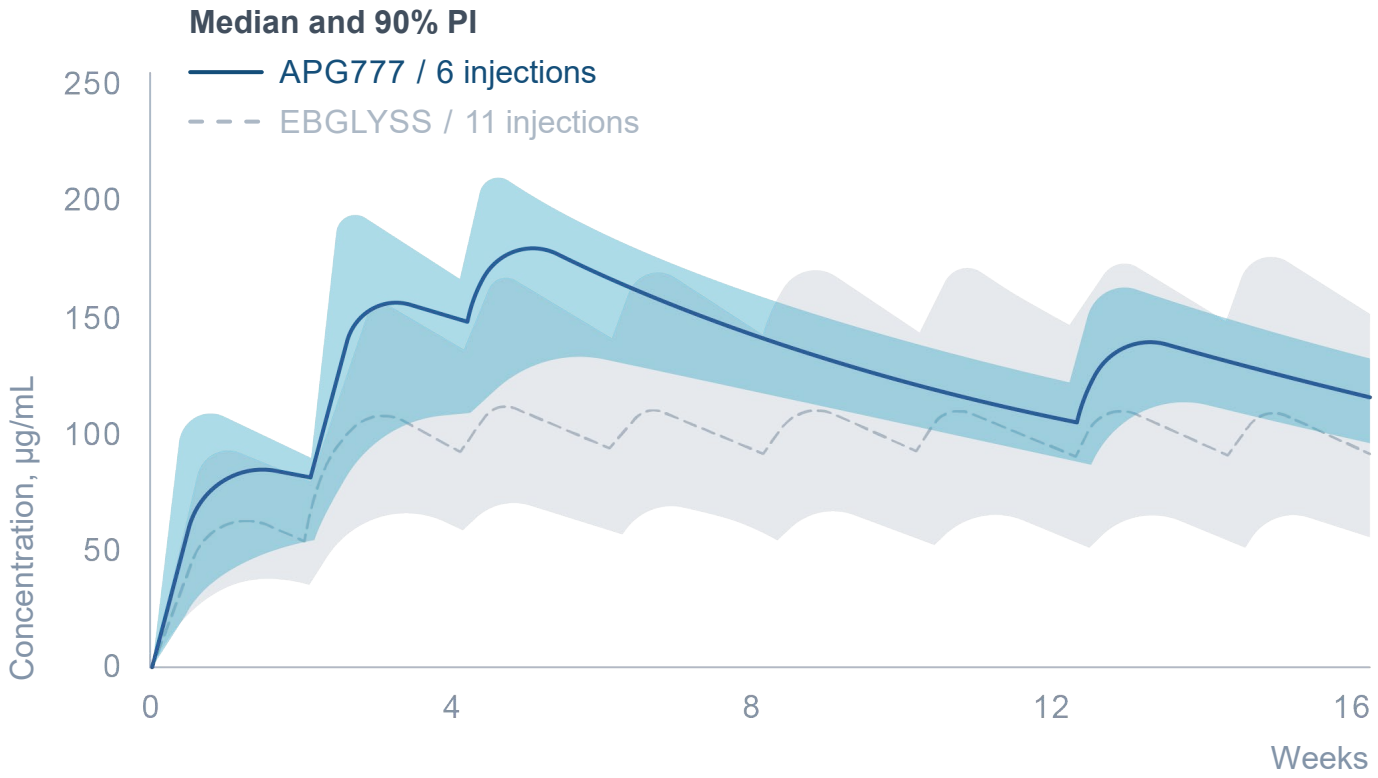
Evidence suggests additional benefit to higher exposures for EBGLYSS in induction

1. EBGLYSS Phase 2b showed dose-response that did not plateau with no dose-AE or exposure-AE relationship[1]

2. ~30% greater exposures in EBGLYSS low-bodyweight patients led to improved efficacy across endpoints[2]

3. EBGLYSS exposure-response model predicts better efficacy possible[2]

APG777 Phase 2 dose targets higher induction exposures than EBGLYSS[3]



~30-40% higher predicted exposure with ~half the number of injections

NOTE: The above graph is illustrative only with respect to plans for dosing of APG777 and does not present comparative data. [1] Guttman-Yassky E et al JAMA Dermatology 2020. [2] EBGLYSS (lebrikizumab) European Public Assessment Report. [3] Solid blue line represents population PK (PPK) model predicted median concentrations of APG777 at Phase 2 induction dose, shaded blue area represents 90% prediction interval (PI) (N=1000); Simulated EBGLYSS exposures (gray) based on PPK model reported in EPAR. Induction regimen in Part A is two injections (720mg) week 0 and week 2 followed by a single injection (360mg) at week 4 and 12.

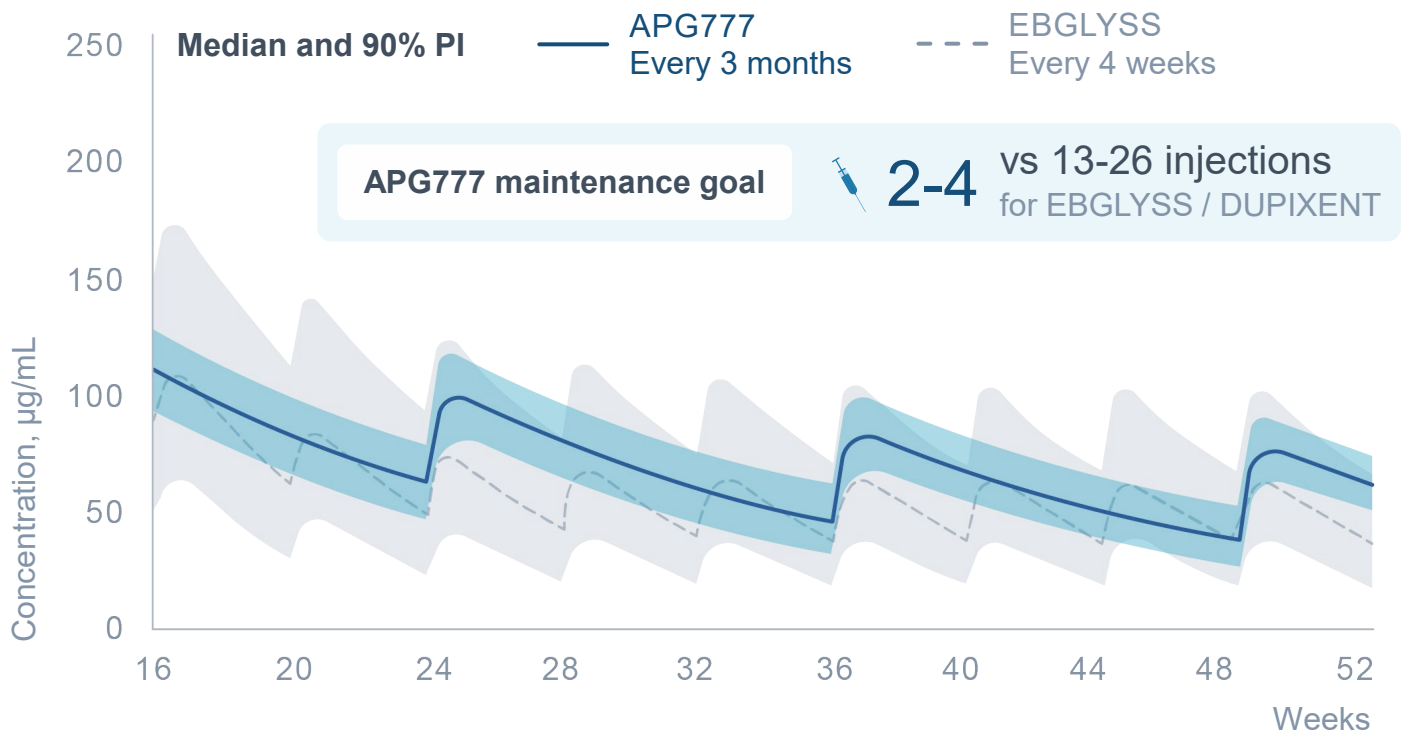
APG777 Phase 2 maintenance exposures designed to equal EBGLYSS

Evidence suggests no additional benefit to higher exposures for EBGLYSS in maintenance

1. EBGLYSS Q4W maintenance data compares favorably to DUPIXENT[1]

2. EBGLYSS Q2W and Q4W regimens had similar maintenance of response[1]

APG777 Phase 2 doses target similar maintenance exposures to EBGLYSS[2]



NOTE: Every 3-month maintenance dosing regimen shown. The above graph is illustrative only with respect to plans for dosing of APG777 and does not present comparative data. [1] EBGLYSS (lebrikizumab) European Public Assessment Report. [2] Solid blue line represents population PK (PPK) model predicted median concentrations of APG777 at Phase 2 maintenance dose, shaded blue area represents 90% prediction interval (PI) (N=1000); Simulated EBGLYSS exposures (Gray) based on PPK model reported in EPAR.

Ongoing integrated Phase 2 trial expected to have 16-week Part A topline data in mid-2025

Part A: Proof-of-concept

N ~110

Screening → 2:1

Induction

- 720mg W0, W2, 360mg W4, W12
- Placebo

Maintenance

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

Rollover to LTE or 52-week follow-up period

W16 Primary Endpoint

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

Part B: Dose optimization

N ~280

Screening → 1:1:1:1

Induction

- High dose
- Mid dose (Part A dose)
- Low dose
- Placebo

Maintenance

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

Rollover to LTE or 52-week follow-up period

W16 Primary Endpoint

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

Phase 2 16-week Part A induction data in atopic dermatitis is planned to readout in mid-2025

Objectives

Safety

Confirm well tolerated safety profile as seen in Phase 1 HV study and **in line with other agents in class**

e.g., DUPIXENT, EBGLYSS

Efficacy (primary)

Primary endpoint of % change from baseline in EASI at Week 16 **in line with standard of care**

~ 65-70% decrease (topline)

Efficacy (key secondary)

Proportion of patients achieving key secondary endpoints at Week 16 (future approvable endpoints) **in line with standard of care:**

EASI-75: ~45-50% (topline)

IGA 0/1: ~35-40% (topline)

NOTE: Based on FDA label for DUPIXENT and EBGLYSS.

Today's updates for APG777 and APG808 demonstrate the potential of Apogee's pipeline to transform the standard-of-care for I&I diseases

APG777

- **Well tolerated with best-in-class PK profile** including 77-day half-life enabling every 3- or 6-month dosing
- **Extended PD effect**: near complete pSTAT6 inhibition for 12 months provides **path to annual dosing**
- **Accelerated Ph2 POC readout to mid-2025** could demonstrate potential for better efficacy and transformative dosing in AD

APG808

- **Well tolerated with potential best-in-class PK profile enabling up to every 2-month dosing:**
  - ~55-day half-life[1], allowing comparable exposure to DUPIXENT with 4-8x fewer injection days[2,3]
- **Extended PD effect:** near complete pSTAT6 inhibition for 3 months provides **path to every 3-month dosing**
- **1H 2025 Ph1b readout** could demonstrate similar activity to DUPIXENT in asthma with potential for extended durability of effect

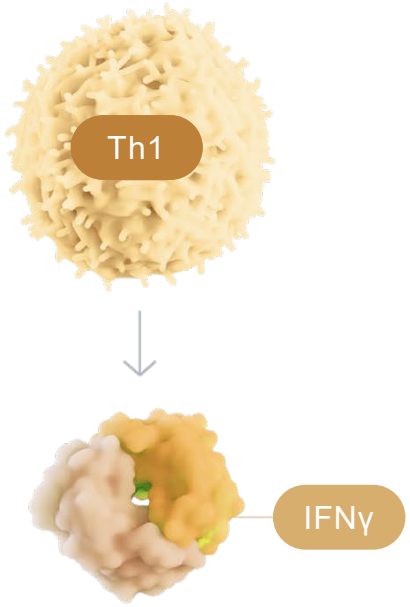
NOTE: [1] Half-life dependent on dose and dosing frequency; 55-day half-life based on model simulated 4mL Q6W high concentration formulation at steady-state, calculated from linear portion of the model, where nonlinear elimination is fully saturated. [2] DUPIXENT PDMA Review reported 8.77-day half-life for highest single SQ dose cohort (600mg) and may represent nonlinear and linear elimination. [3] Exposure target based on C_{trough} in maintenance, the minimal concentration of APG808 to have similar exposures to DUPIXENT, based on mean observed Ctrough,ss for DUPIXENT in adults with asthma (69.0 µg/mL) and COPD (61.8 µg/mL) in two clinical trials reported in DUPIXENT European Public Assessment Report (EMEA/H/C/004390/II/0079).

APG777+APG990: Raising the bar in AD

Rebecca Dabora, PhD
Chief Development Officer

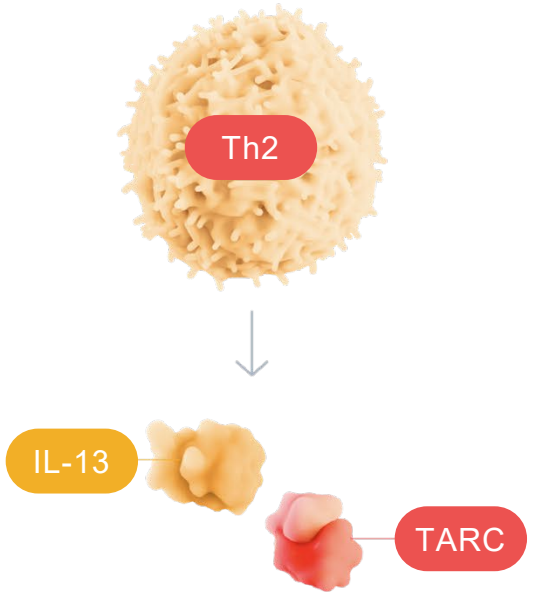
Type 1, 2, and 3 inflammation play distinct roles in AD with Type 2 inflammation being the core driver

Type 1



Chronic lesions

Type 2



Core inflammation, barrier disruption, and itch

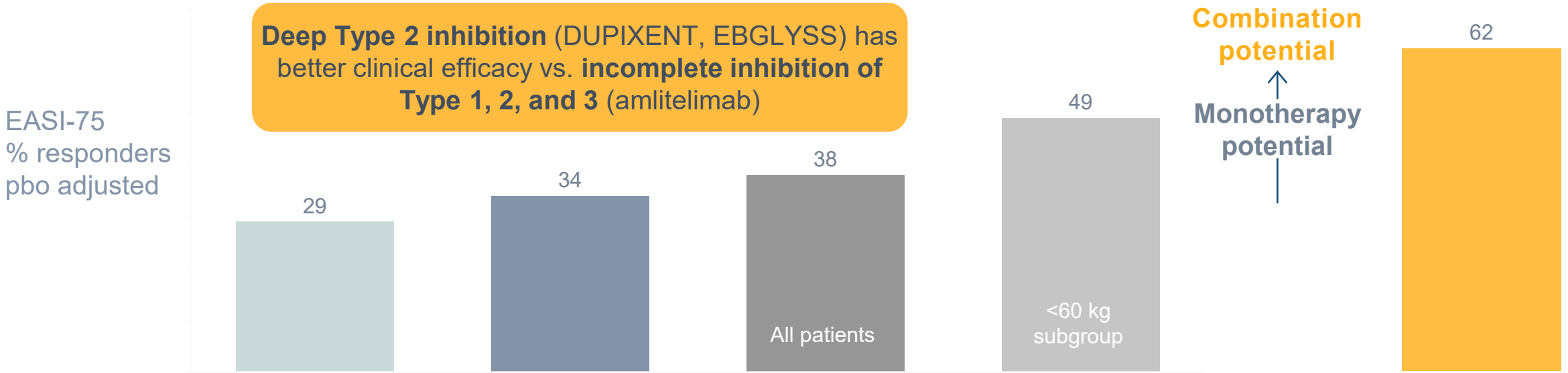
Type 3

Epidermal thickening, barrier disruption

SOURCE: Adapted from Guttman-Yassky et al. Br J Dermatol 2024; 00:1–9.

JAK inhibitors broadly inhibit Type 1, 2 and 3 inflammation with strong clinical efficacy but are limited by safety

Target	OX40L	IL-4Rα	IL-13	JAK
Agent	Amlitelimab	DUPIXENT® (dupilumab) Injection 300mg	Ebglyss™ (lebrikizumab-lbkz)	RINVOQ® upadacitinib
Inflammation targeted	**Type 1, 2, 3** (partial inhibition)	**Type 2**	**Type 2**	**Type 1, 2, 3** (strong inhibition)



NOTE: In lebrikizumab Ph2b and Ph3 there has been no dose-AE or exposure-AE relationship. Lebrikizumab exposures and efficacy are for the Phase 3 dose (500 mg Loading Dose at Weeks 0 and 2, 250 mg Q2W Weeks 4 to 16). Efficacy data are derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.
SOURCE: EBGLYSS European Public Assessment Report. DUPIXENT USPI. RINVOQ USPI. Weindinger, S. Oral Presentation. AAD (2024).

APG777+APG990 targets Type 1, 2, and 3 inflammation similar to JAKs, but with potentially better tolerability

✓ Strong inhibition ✓ Partial inhibition ✗ No inhibition	① Type 1	② Type 2 (core driver)	③ Type 3	Safety / tolerability profile[1]
JAKs	✓	✓	✓	✗ **JAKs carry class black box warning**
APG777 IL-13	✗	✓	✗	APG777 was **well-tolerated** in Phase 1
APG990 OX40L	✓	✓	✓	Amlitelimab (OX40L) has been **well-tolerated** across multiple clinical trials
APG777+APG990 IL-13 OX40L	✓	✓	✓	**Combination of two well-tolerated MoAs** without overlapping tox

NOTE: [1] Safety characterization for: APG777 is based on Phase 1 results, APG990 is based on potential Phase 1 results, APG777 + APG990 is based on historical experience with amlitelimab and EBGLYSS as monotherapy agents, but they have not been tested in combination.

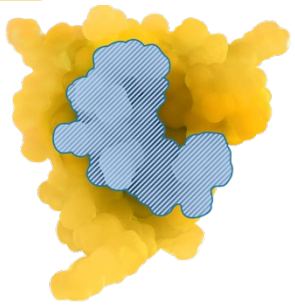
APG777 and APG990 are engineered for best-in-class properties against validated mechanisms

APG777

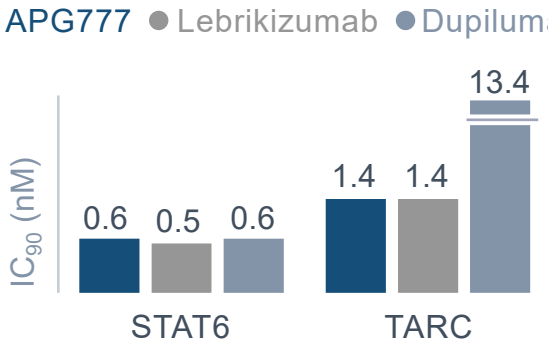
OVERLAPPING EPITOPE

HUMAN IL-13

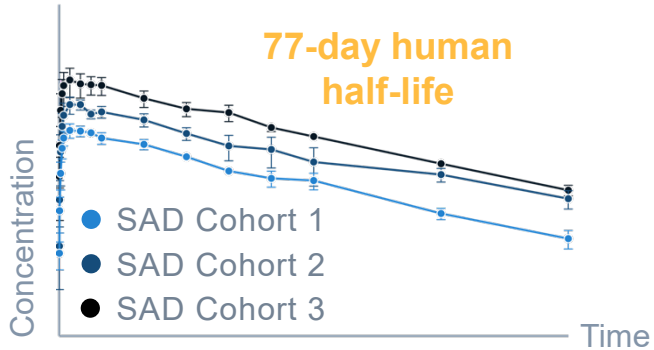
Overlapping region (vs. lebrikizumab)



SIMILAR POTENCY



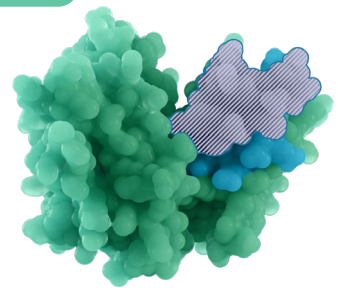
EXTENDED HALF-LIFE

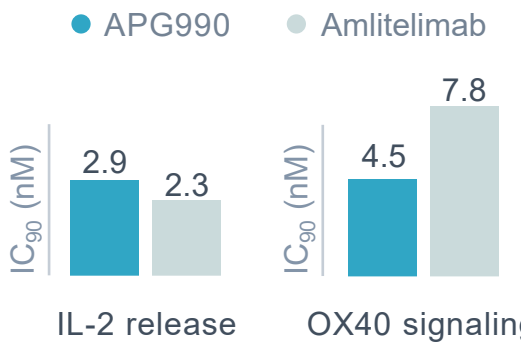


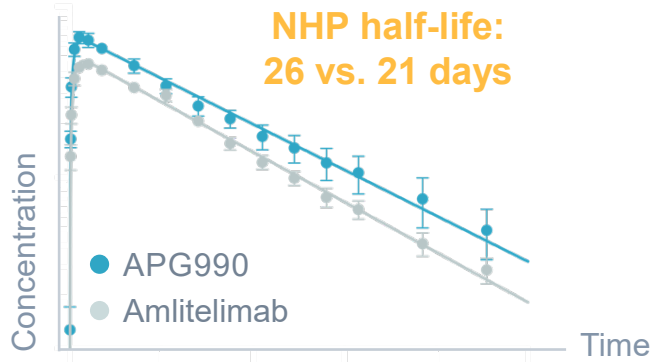
APG990

HUMAN OX40L

Overlapping region (vs. amlitelimab)







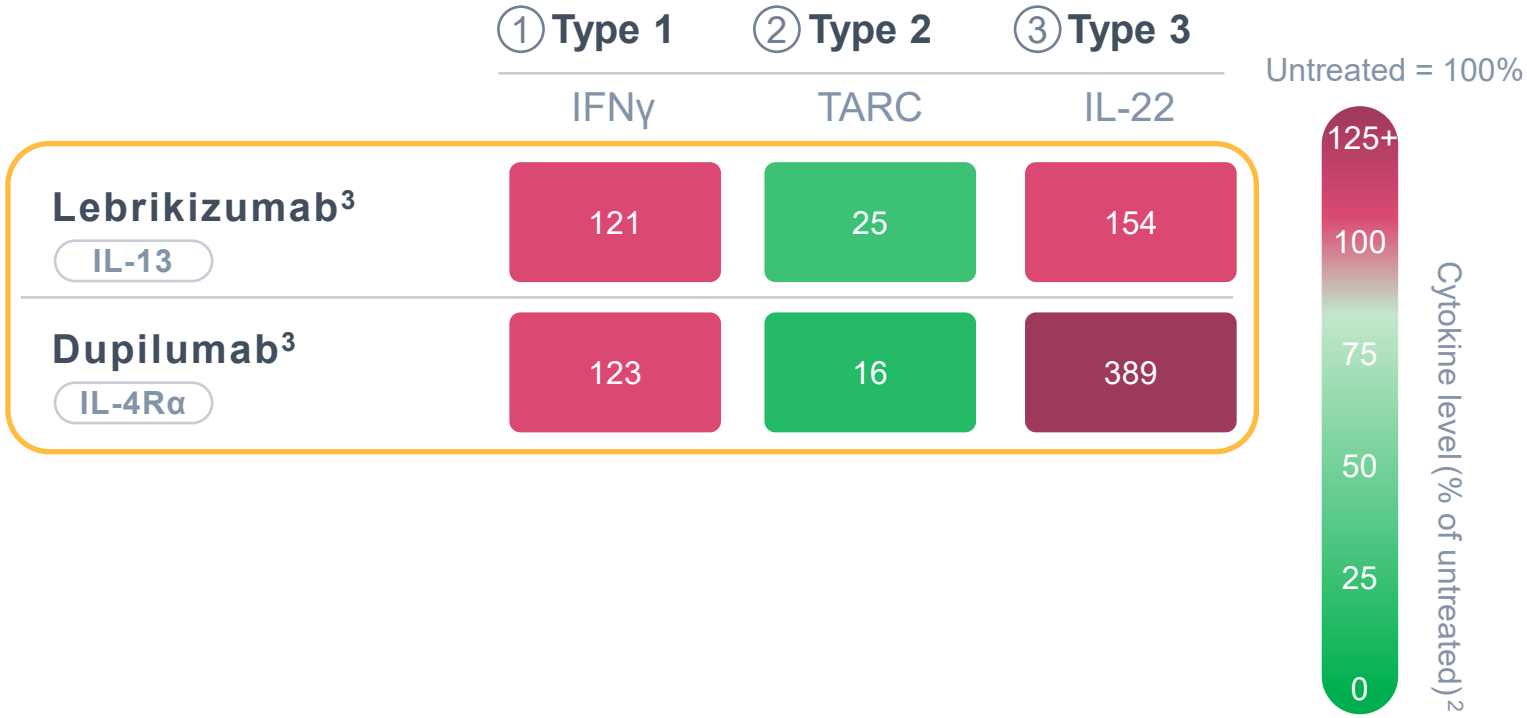
NOTE: NHP = non-human primate.

In our preclinical studies, lebrikizumab and dupilumab deeply inhibit Type 2 inflammation but do not address Type 1 and Type 3

- **Adding Type 1 and Type 3 inhibition** to deep Type 2 inhibition could **provide additional benefit**

- **Dupilumab may increase Type 3 inflammation** (and lebrikizumab to a lesser extent); elevated IL-22 and related side effects have been reported in some DUPIXENT-treated patients (e.g., head and neck dermatitis)[4,5]

Ex vivo human allogeneic lymphocyte reaction (ALR) assay[1]



NOTE: [1] The ALR was performed using TSLP-primed mDCs paired with allogeneic CD4 cells for 5 days. [2] Cytokine levels for lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 are reported as the mean percent of isotype control across four donor pairs. [3] Lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 were tested at 45 µg/mL that is comparable to DUPIXENT steady-state trough concentrations for the approved dose (300mg Q2W) in atopic dermatitis. [4] Varandas et al. (2022) Clin Exp Allergy. DOI: 10.1111/cea.14147. [5] Bangert et al. (2024) Nature Communications. DOI: 10.1038/s41467-024-46540-0.

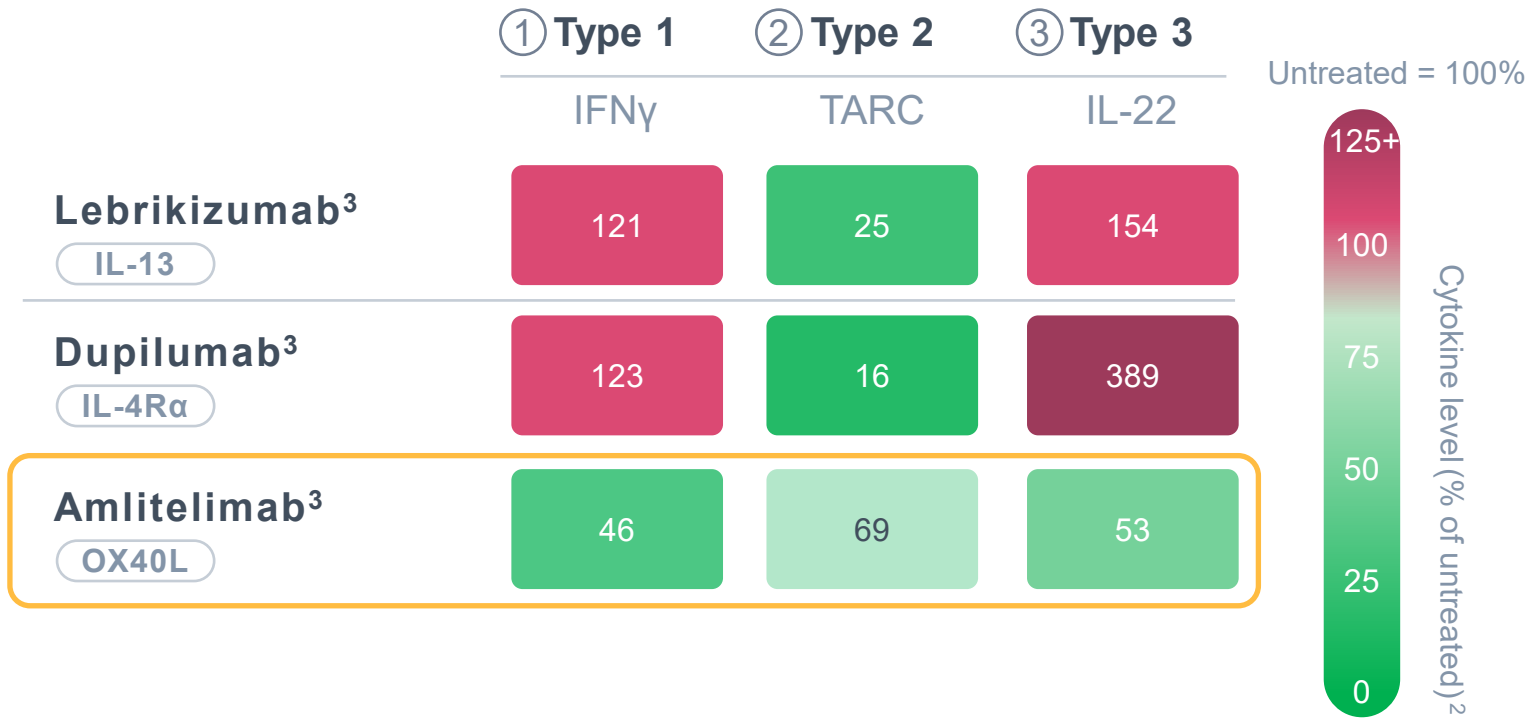
OX40L inhibitor amlitelimab achieves broad but partial inhibition

- **Amlitelimab is the first clinically-validated OX40L inhibitor** and has demonstrated broad inhibition of inflammatory biomarkers

- Amlitelimab **moderately inhibits Type 1 and Type 3** inflammation, but **only partially inhibits Type 2** inflammation

- Partial Type 2 inhibition **may explain amlitelimab's lower clinical efficacy** vs. Type 2 specific inhibitors (e.g., EBGLYSS, DUPIXENT)

Ex vivo human allogeneic lymphocyte reaction (ALR) assay[1]



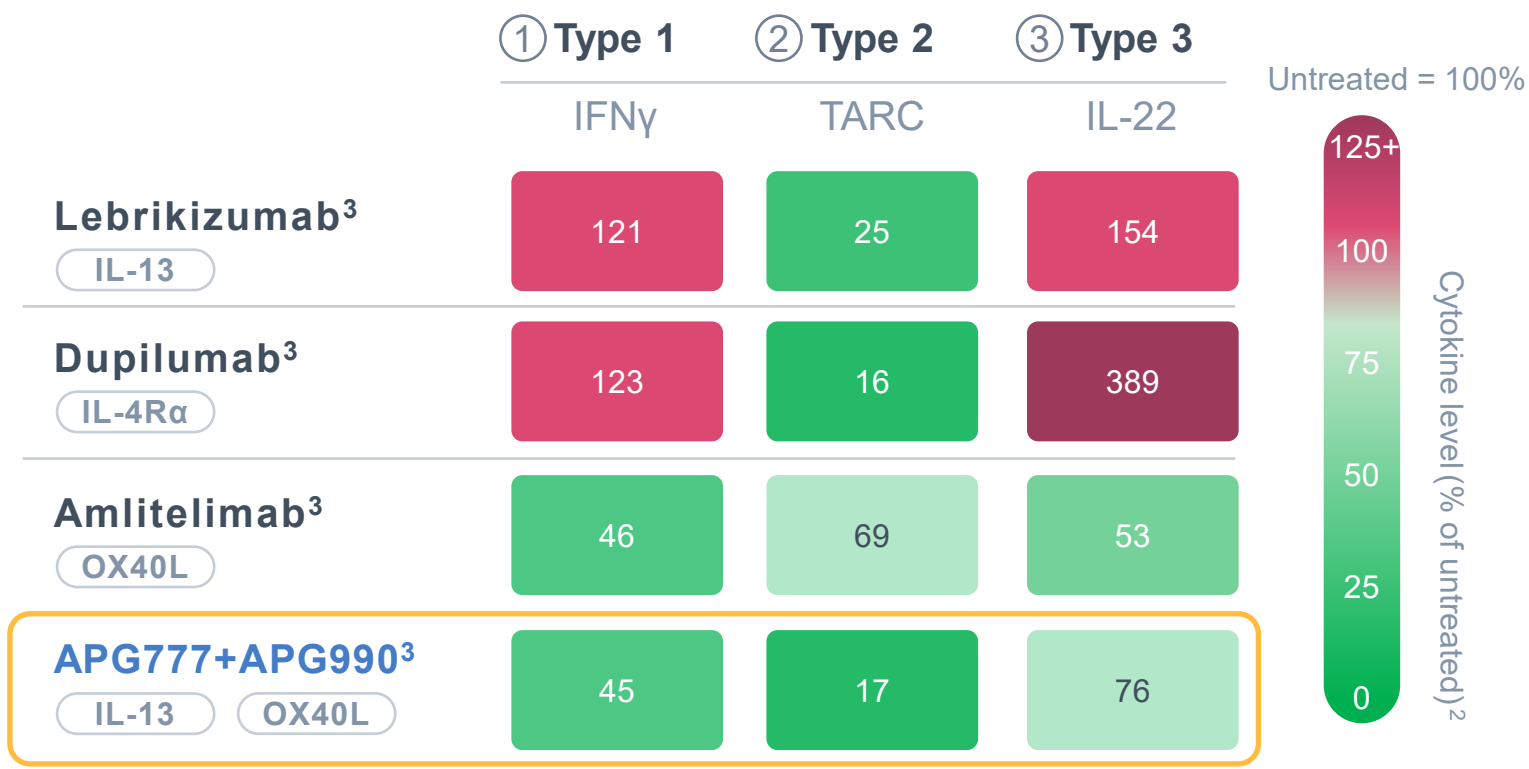
NOTE: [1] The ALR was performed using TSLP-primed mDCs paired with allogeneic CD4 cells for 5 days. [2] Cytokine levels for lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 are reported as the mean percent of isotype control across four donor pairs. [3] Lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 were tested at 45 µg/mL that is comparable to DUPIXENT steady-state trough concentrations for the approved dose (300mg Q2W) in atopic dermatitis.

Potential first-in-class APG777+APG990 targets all inflammatory types, including near complete Type 2 inhibition

- **APG777 targets IL-13 for deep inhibition of Type 2** inflammation, addressing the **core driver** of inflammation in AD

- **APG990 targets OX40L for broader inhibition of Type 1 and 3** inflammation, addressing other heterogenous **secondary drivers** of AD

Ex vivo human allogeneic lymphocyte reaction (ALR) assay[1]



NOTE: [1] The ALR was performed using TSLP-primed mDCs paired with allogeneic CD4 cells for 5 days. [2] Cytokine levels for lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 are reported as the mean percent of isotype control across four donor pairs. [3] Lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 were tested at 45 µg/mL that is comparable to DUPIXENT steady-state trough concentrations for the approved dose (300mg Q2W) in atopic dermatitis.

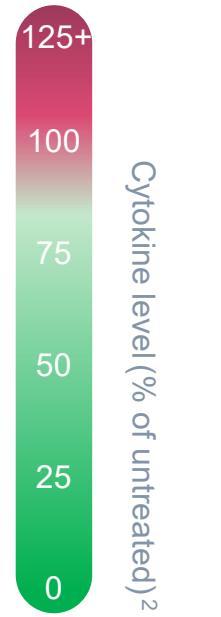
APG777+APG990 enables potentially best-in-class efficacy and dosing

- **APG777+APG990** has the potential to improve clinical outcomes while still **minimizing the injection burden**

- APG777+APG990 has the potential for **every three-month dosing** (or less frequent) vs. every 2- or 4-weeks for approved monotherapies

Ex vivo human allogeneic lymphocyte reaction (ALR) assay[1]

	Dosing	① Type 1 IFNγ	② Type 2 TARC	③ Type 3 IL-22
Lebrikizumab[3] IL-13	Q2W-Q4W	121	25	154
Dupilumab[3] IL-4Rα	Q2W	123	16	389
Amlitelimab[3] OX40L	Q4W	46	69	53
APG777+APG990[3] IL-13 OX40L	**Q12W+**	45	17	76



NOTE: [1] The ALR was performed using TSLP-primed mDCs paired with allogeneic CD4 cells for 5 days. [2] Cytokine levels for lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 are reported as the mean percent of isotype control across four donor pairs. [3] Lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 were tested at 45 µg/mL that is comparable to DUPIXENT steady-state trough concentrations for the approved dose (300mg Q2W) in atopic dermatitis.

APG777+APG990 demonstrates inhibition of Type 1, 2 and 3 inflammation, similar to JAK-inhibitor upadacitinib

- **JAKs broadly inhibit inflammation** including Type 1, 2, and 3 and have best-in-class efficacy in AD

- Targeting all inflammatory types may **provide greater efficacy** vs. "Type 2 only" inhibitors (e.g., DUPIXENT)

Ex vivo human allogeneic lymphocyte reaction (ALR) assay[1]

	Dosing	① Type 1 IFNγ	② Type 2 TARC	③ Type 3 IL-22
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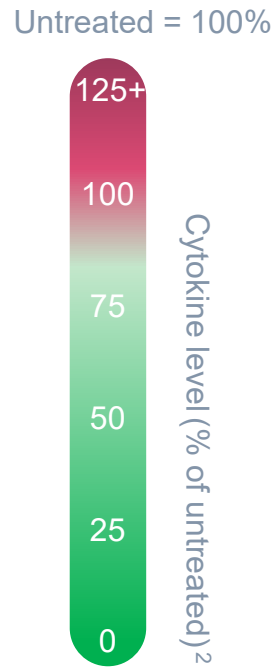
APG777+APG990[3] IL-13 OX40L	**Q12W+**	45	17	76
Upadacitinib[4] JAK	DAILY	8	10	30

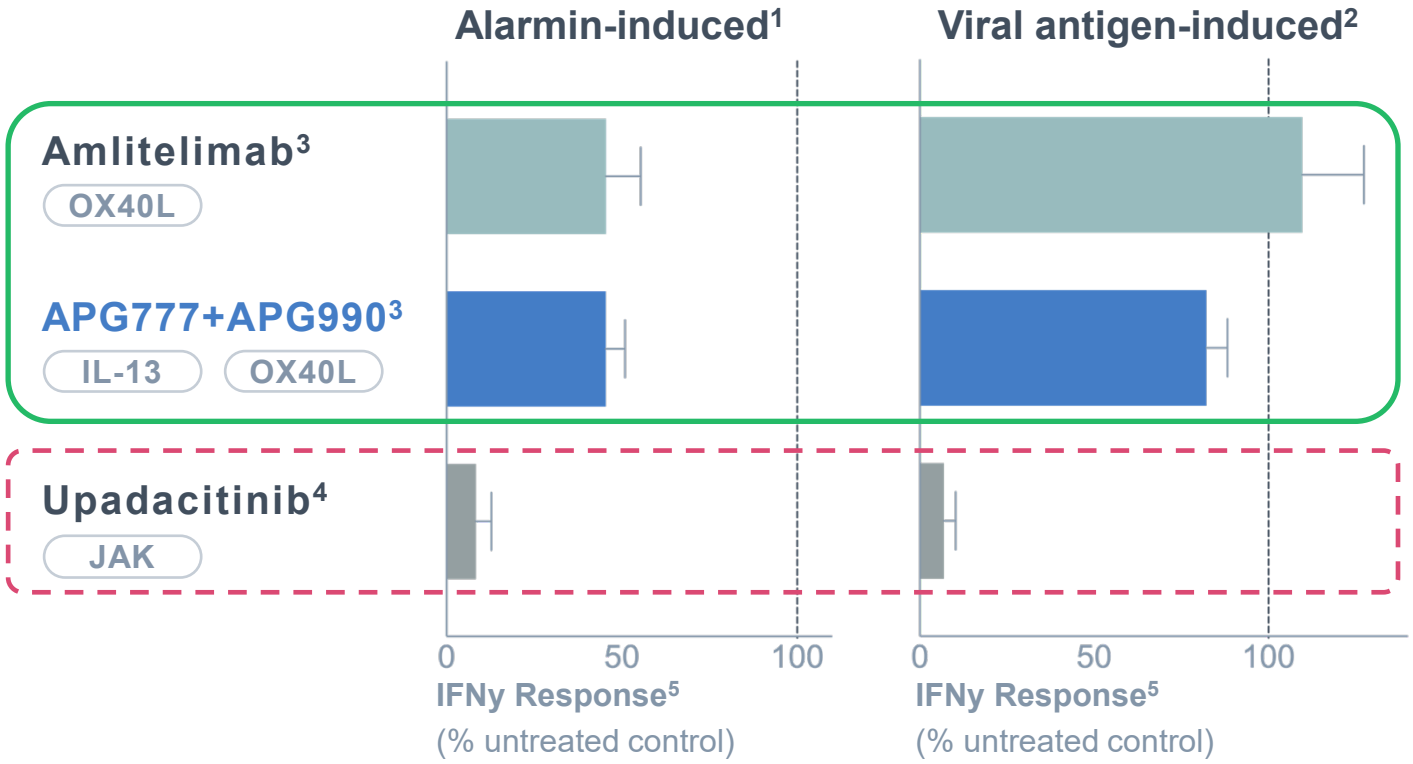


NOTE: [1] The ALR was performed using TSLP-primed mDCs paired with allogeneic CD4 cells for 5 days. [2] Cytokine levels for lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 are reported as the mean percent of isotype control across four donor pairs; upadacitinib reported as mean percent of DMSO control across four donor pairs. [3] Lebrikizumab, dupilumab, amlitelimab, and APG777+APG990 were tested at 45 µg/mL that is comparable to DUPIXENT steady-state trough concentrations for the approved dose (300mg Q2W) in atopic dermatitis. [4] Upadacitinib was tested at the Cmax concentration for RINVOQ 15mg (31 ng/mL), reflecting maximum inhibition achieved briefly after dosing.

APG777+APG990 selectively blocks alarmin-induced Type 1 cytokine responses associated with AD, whereas JAKs are non-selective

- **Alarmin-induced Type 1** inflammation drives chronic **atopic dermatitis**
- **Viral antigen-induced Type 1** inflammation is an important component of **antiviral immunity**
- **JAKs non-selectively inhibit Type 1** inflammation and carry a **black box warning** for infection risk
- **APG777+APG990** and amlitelimab **preserve viral antigen-induced Type 1** inflammation

Type 1 inflammation assessed by two different preclinical models



Selective

- **Inhibits** alarmin-induced Type 1 inflammation
- **Preserves** viral antigen-induced Type 1 inflammation

Non-selective

- **Inhibits both** alarmin-induced and viral antigen-induced Type 1 inflammation

NOTE: [1] ALR performed using four donor pairs of TSLP-primed mDCs plus allogeneic CD4 cells for 5 days. [2] PBMCs from three donors were stimulated with cytomegalovirus antigens for four days. [3] Amlitelimab and APG777+APG990 were tested at 45 µg/mL, which is comparable to DUPIXENT steady-state trough concentrations for the approved dose (300mg Q2W) in atopic dermatitis. [4] Upadacitinib was tested at the Cmax concentration for RINVOQ 15mg (31 ng/mL), reflecting maximum inhibition achieved briefly after dosing. [5] IFNγ levels for amlitelimab and APG777+APG990 are reported as mean percent of isotype control; Upadacitinib reported as mean percent of DMSO control.

APG777+APG990: Coformulation PoC

Coformulations could enable potentially best-in-class efficacy while maintaining best-in-category dosing

Characteristics	Coformulation approach	Bispecific approach
Dosing potential	Every 3-months or less frequent	Every 1-4 weeks
Potential to optimize dose for effective target inhibition	✓	✗
COGS	⇩	⇧
Potential to deliver in simple presentation (e.g., single autoinjector)	✓	✓
Approval precedent (total # of approvals in last 20 years)	134	10

NOTE: Coformulation characteristics are illustrative based on what we believe we can achieve. Bispecific characteristics based on properties of FDA approved bispecifics. Coformulated drugs limited to FDA approved products 2004-2024.
SOURCE: FDA

APG777+APG990 coformulation proof-of-concept achieved

Stability

Stable at high concentrations (i.e., >150 mg/mL)

Injectability

Expected injection time comparable to DUPIXENT

Convenience

Compatible with commercial presentation (e.g., 2 mL PFS)

Potency

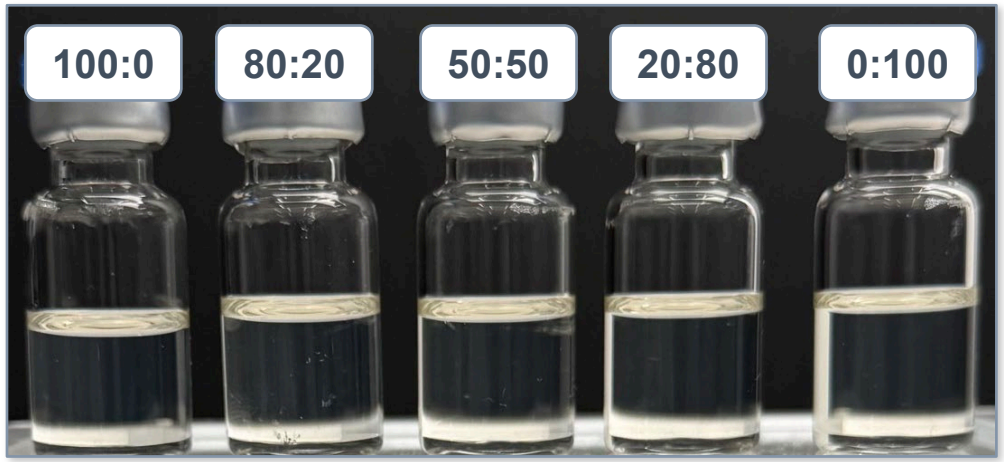
Potency equivalent to each component tested individually

NOTE: PFS = pre-filled syringe.

APG777+APG990 coformulation retains the stability and injectability of individual components; compatible with 2 mL pre-filled syringe

APG777+APG990 coformulation retains stability of individual components at high concentration (>150mg/mL)

100% APG777 ⟷ 100% APG990



APG777+APG990 is stable in 2 mL PFS and expected to have comparable injection time as DUPIXENT

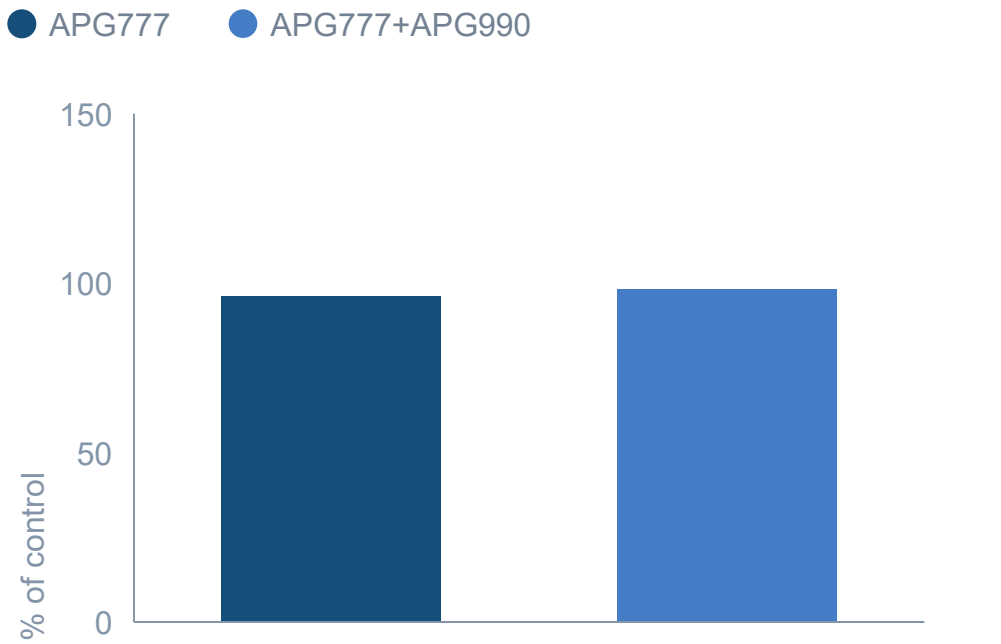
Pre-filled Syringes
APG777+APG990 (50:50)

Injection time expected to be comparable to DUPIXENT in PFS and autoinjector formats[1]

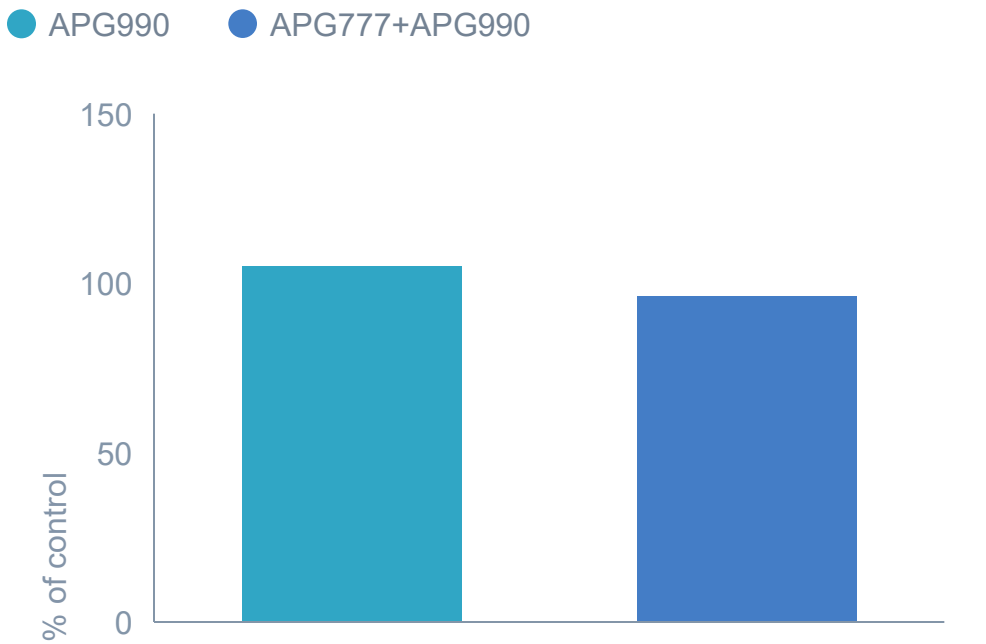
NOTE: [1] Expected injection time for a 2 mL autoinjector (AI) or pre-filled syringe (PFS).

Coformulation maintains potency of individual components in proof-of-concept study

APG777 potency[1,2]



APG990 potency[1,3]



Coformulation maintains high potency against targets under accelerated storage conditions *(as shown)*

NOTE: [1] Potency for APG777, APG990, and APG777+APG990 (50:50 ratio) measured under accelerated storage conditions. [2] Direct binding of IL-13 to APG777 determined by ELISA. [3] Direct binding of OX40L to APG990 determined by ELISA.

APG777+APG990: Moving into the clinic in 2025

Kristine Nograles, MD
SVP, Clinical Development

APG990 Phase 1 is underway with initial data readout expected in 1H 2025

Trial design elements

Double-blind, placebo-controlled, first-in-human trial
Single ascending dose in healthy volunteers

N ~ 40

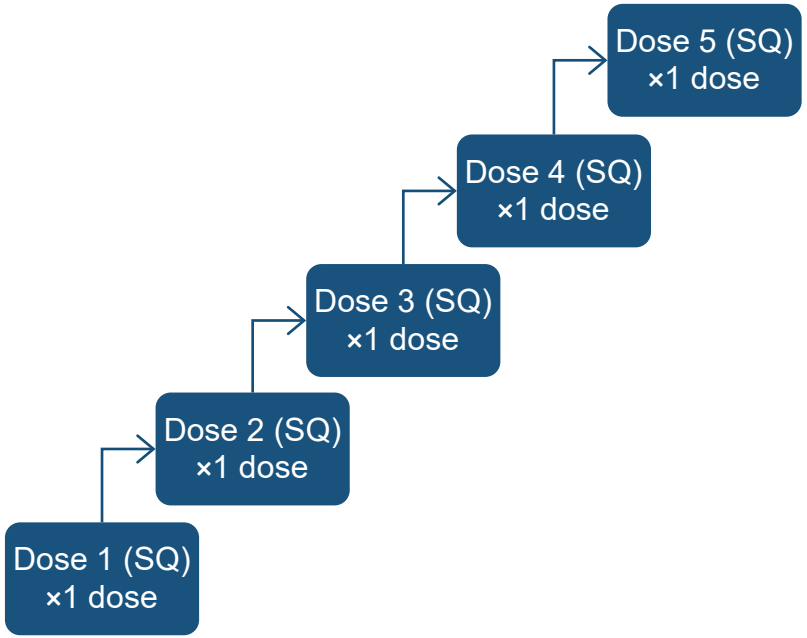
8 per cohort (6:2 active:placebo)

Key inclusion criteria: healthy adult volunteers

Primary endpoint: safety

Secondary endpoints: PK, ADA

Single ascending dose [1]



Study objectives

Confirm tolerable **safety profile to enable future combination trials**

Establish **optimized PK profile** with a half-life of at least 21 days

Determine **dosing regimens** to sustain exposures similar to amlitelimab

Phase 1 readout in 1H 2025 could confirm potential for best-in-class dosing (goal of every 3- or 6-months)

NOTE: [1] SAD dose ascension will ultimately be determined by the SRC (Scientific Review Committee) based on available data.

Planned Phase 1b of APG777+APG990 against DUPIXENT expected to initiate in 2025

Trial design elements

Randomized assessor-blinded, active comparator trial

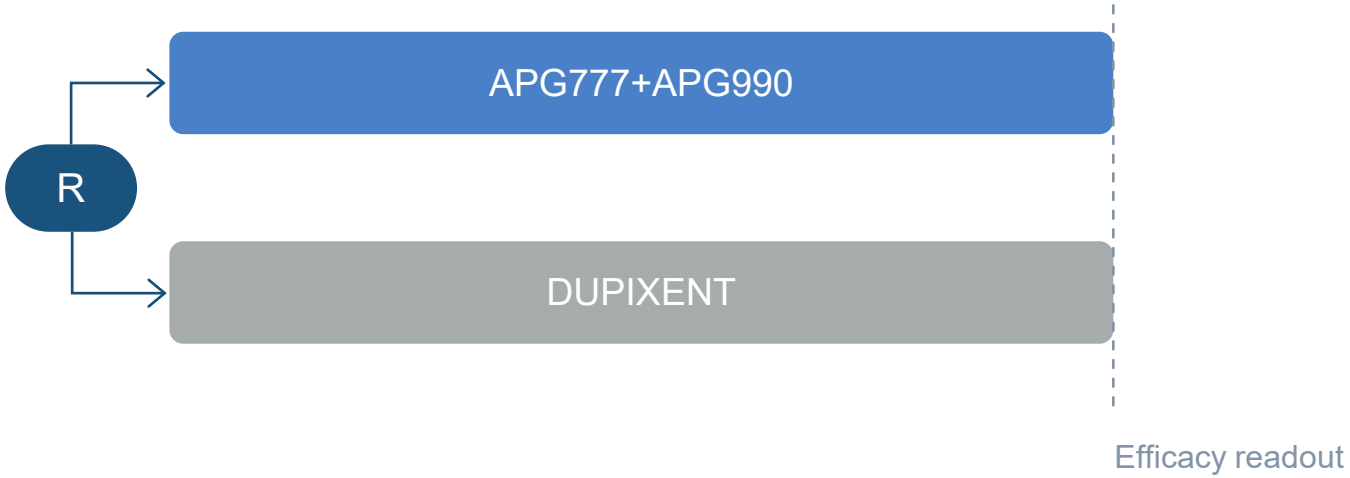
N ~50-75

Key inclusion criteria: biologic naïve, moderate-to-severe AD at screening and baseline (EASI ≥16, IGA ≥3, BSA ≥10)

Primary endpoint: safety/tolerability

Secondary endpoints: efficacy (EASI75, IGA0/1), PK, biomarkers

Phase 1b trial in moderate-to-severe AD



Phase 1b readout against DUPIXENT in 2026 could demonstrate potential for transformational efficacy and dosing

APG777+APG990 Phase 1b clinical trial objectives

Objectives

Safety

Confirm tolerable **safety profile to enable additional combination trials**

PD biomarkers

Demonstrate **broader pharmacodynamic effect** on biomarkers of Type 1, 2, and 3 inflammation compared with standard of care

Efficacy

Proportion of patients achieving key endpoints (e.g., EASI75, IGA0/1) at **higher rates than with standard of care**

APG777+APG333: targeting both central and local drivers of respiratory disease

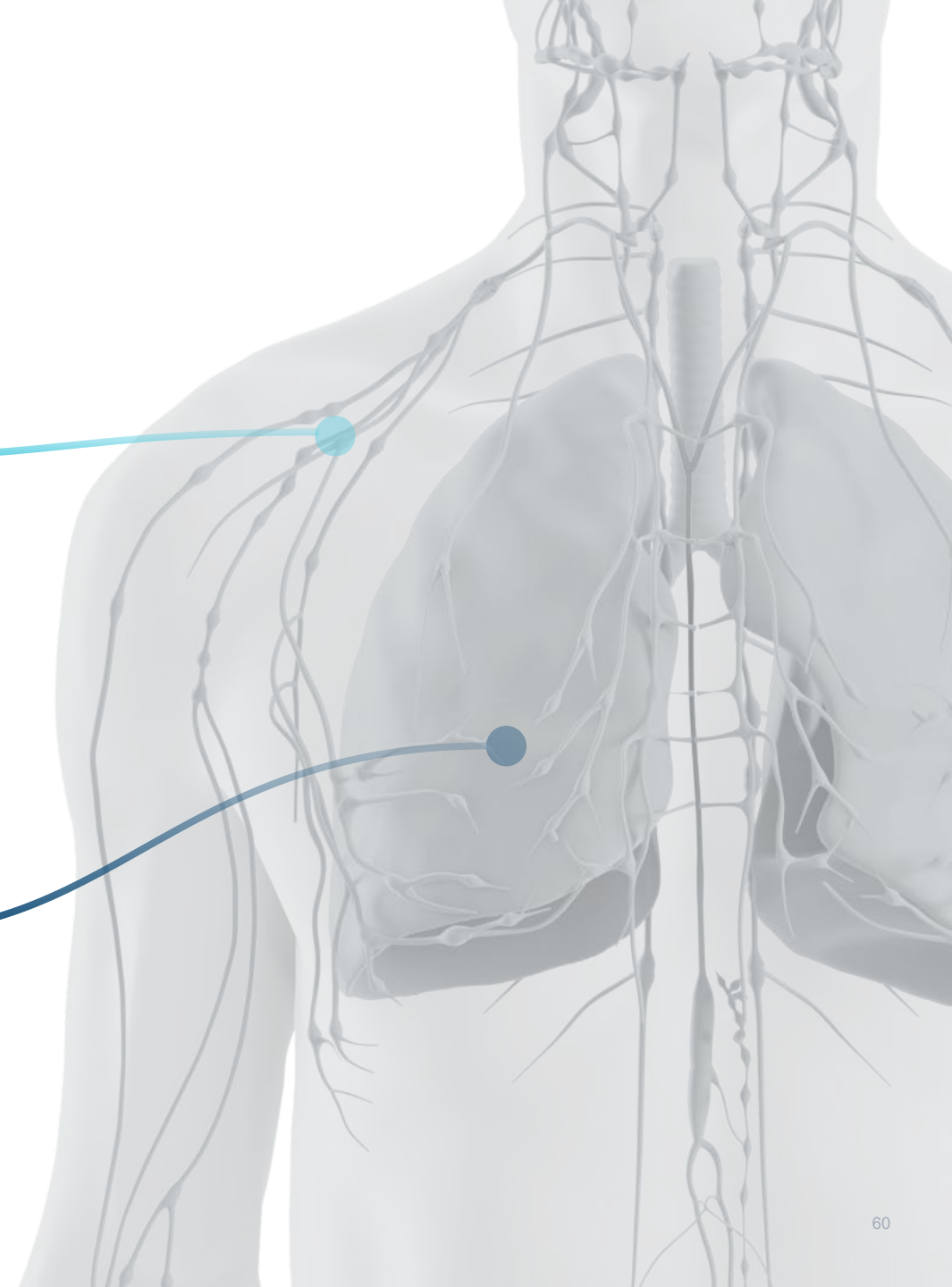
Lukas Dillinger, PhD
VP, Research and Translational Medicine

Alarmins and Type 2 cytokines drive obstructive airway disease

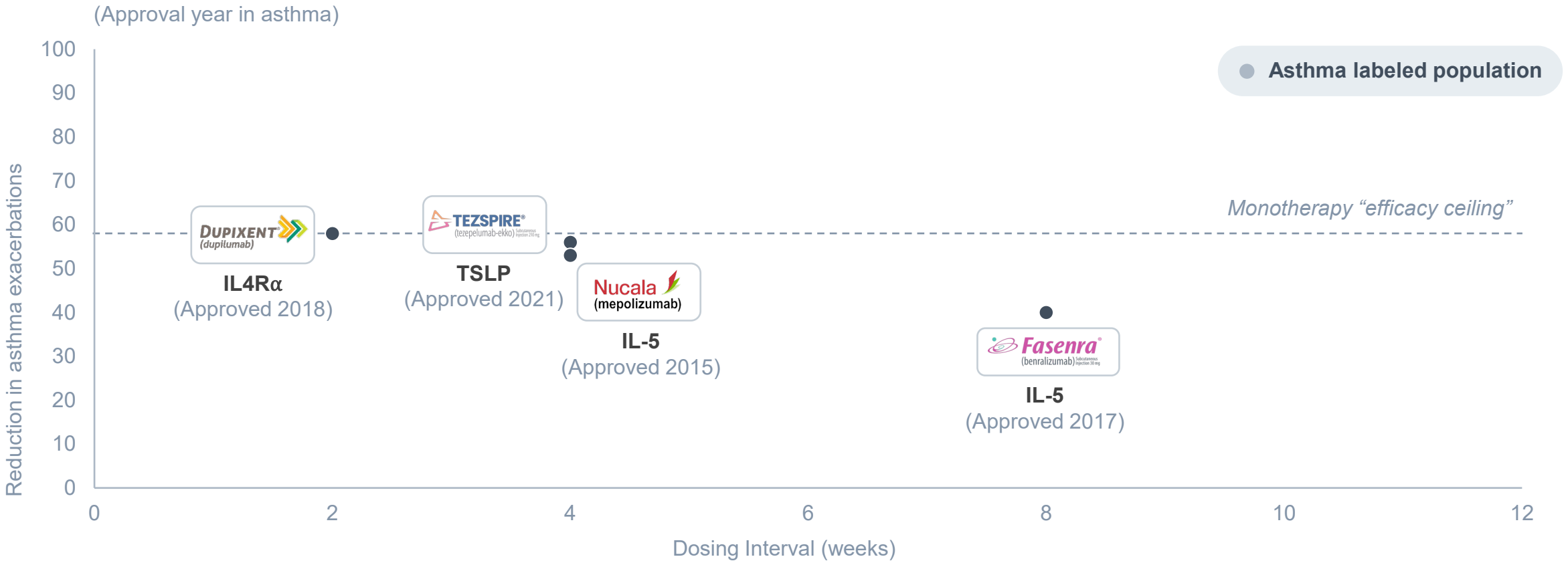
- **Alarmins (including TSLP)** act primarily on the immune system to recruit and activate immune cells
- Alarmins drive **central inflammation** including Type 2 and Non-Type 2 cytokine production

+

- **Type 2 cytokines (including IL-13)** act primarily in the periphery
- Type 2 inflammatory factors drive **local airway responses** including smooth muscle cell activation and epithelial dysfunction



Multiple novel treatments targeting alarmins or Type 2 cytokines have been approved in asthma, but efficacy has hit a ceiling



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

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

APG777+APG333 combines validated mechanisms to address both central and local drivers of obstructive airway disease

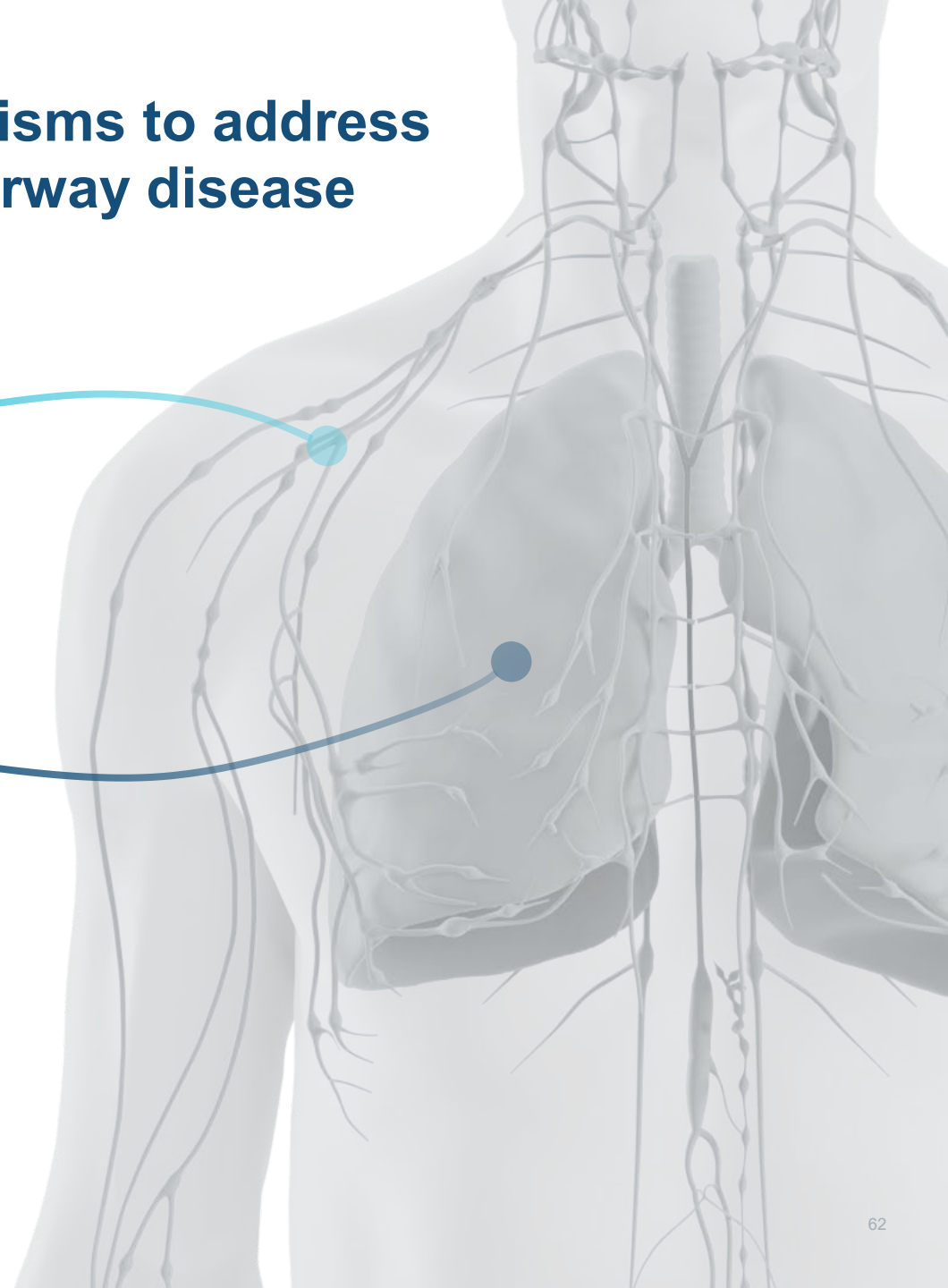
APG333 targets TSLP to block central inflammation

+

APG777 targets IL-13 to address local airway responses

↓

Potential to **break through the monotherapy efficacy ceiling** in obstructive airway disease



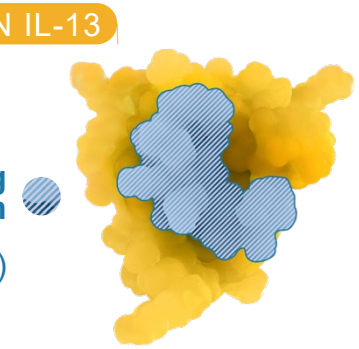
APG777 and APG333 are engineered for best-in-class properties against validated mechanisms

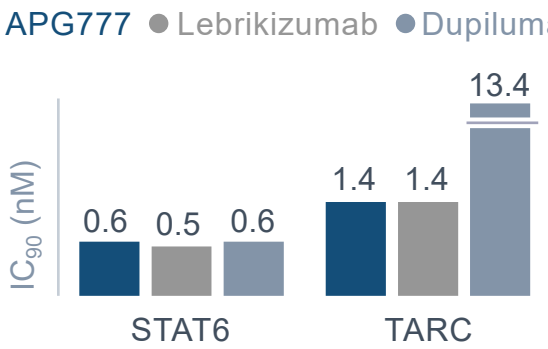
OVERLAPPING EPITOPE

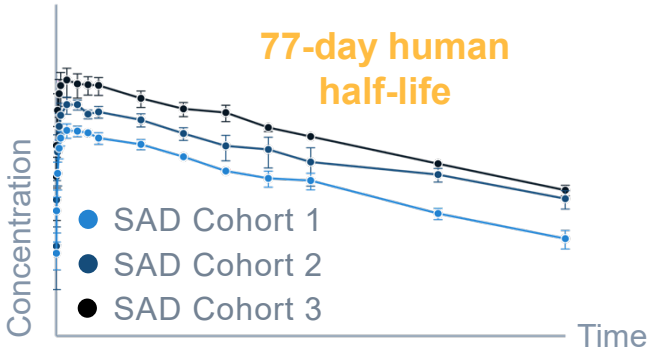
SIMILAR POTENCY

EXTENDED HALF-LIFE

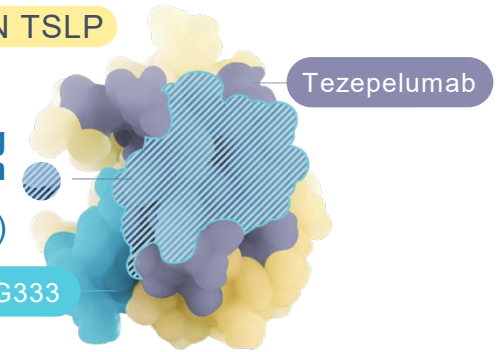
APG777

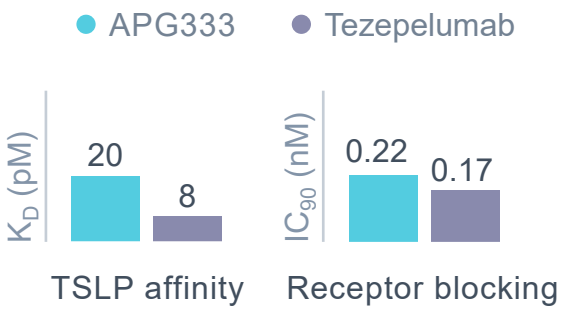


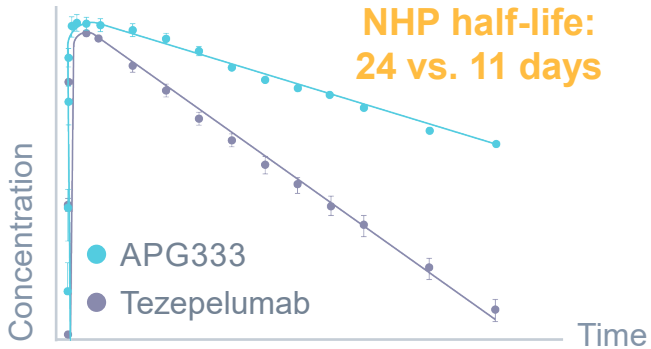




APG333







NOTE: NHP = non-human primate.

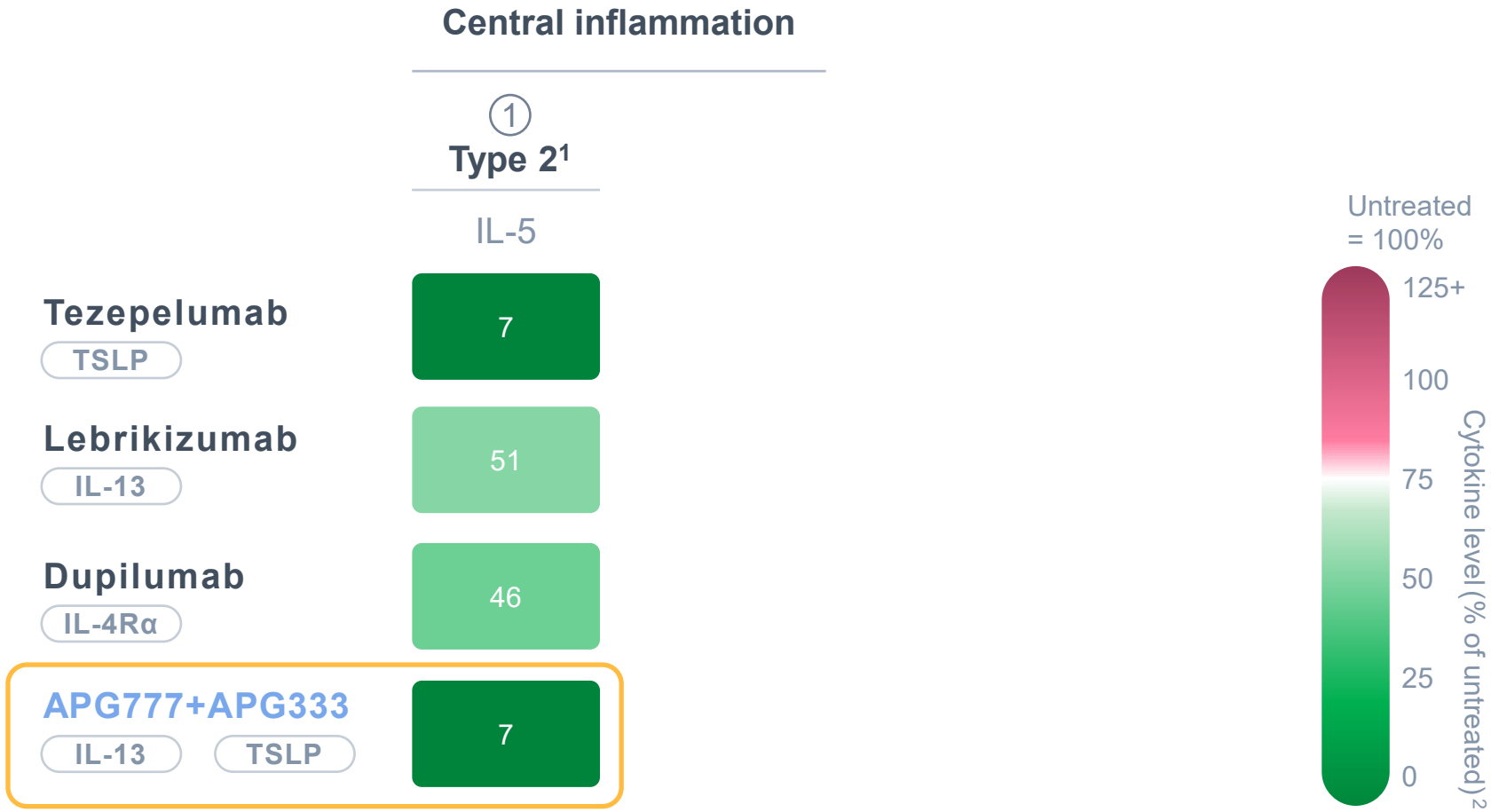
APG777+APG333 can potentially address key drivers of airway disease more broadly vs. monotherapy

	Central inflammation		Local airway responses	
	① Type 2 cytokine release	② Non-Type 2 cytokine release	③ BSMC activation	④ Epithelial dysfunction
APG777 IL-13	✗	✗	✓	✓
APG333 TSLP	✓	✓	✗	✗
APG777+APG333 IL-13 TSLP	✓	✓	✓	✓

APG777+APG333 achieves deeper inhibition of Type 2 inflammation vs. lebrikizumab and dupilumab

- **TSLP drives Type 2 cytokine release** (e.g., IL-5) that contributes to **inflammation centrally**

- For example, **IL-5 recruits eosinophils** into the airway

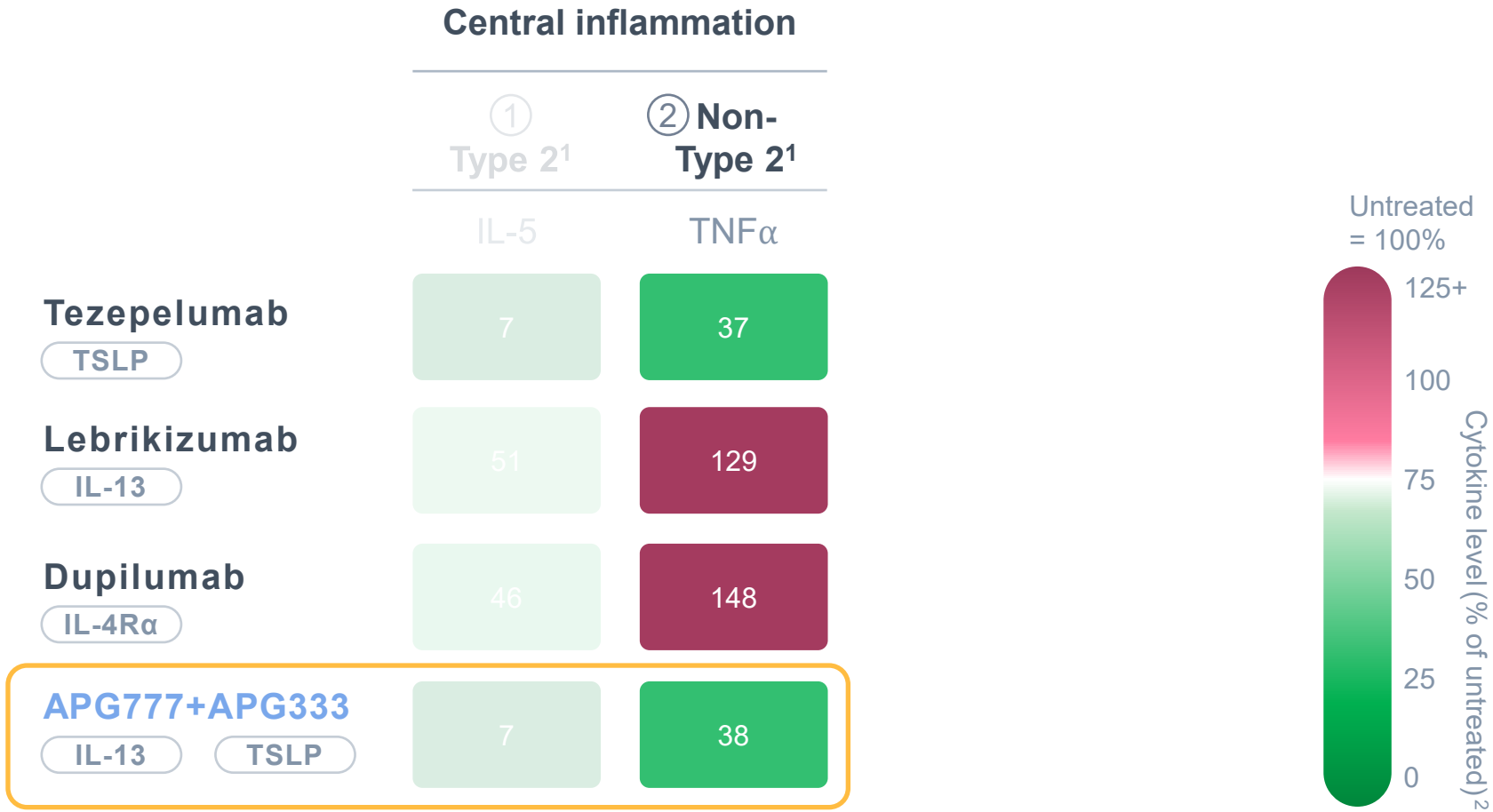


NOTE: [1] ALR performed using four donor pairs of TSLP-primed mDCs plus allogeneic CD4 cells for 5 days. [2] Responses are reported as mean percent of control across all donors.

APG777+APG333 also inhibits non-Type 2 inflammation; in contrast, lebrikizumab and dupilumab may promote non-Type 2

- **TSLP also drives non-Type 2** cytokine release (e.g., TNFα)

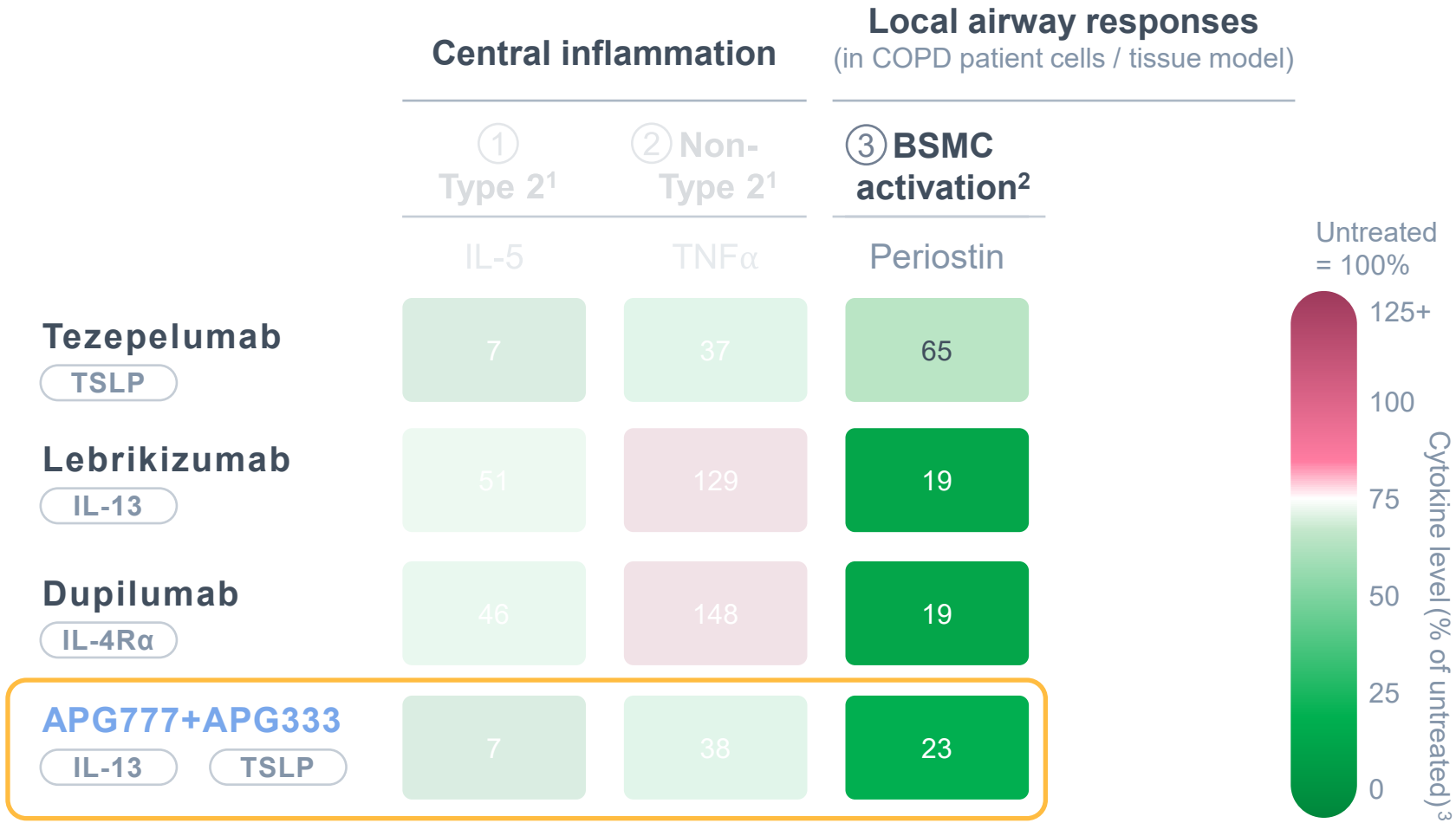
- **TNFα levels are elevated in COPD patients[3]**



NOTE: [1] ALR performed using four donor pairs of TSLP-primed mDCs plus allogeneic CD4 cells for 5 days. [2] Responses are reported as mean percent of control across all donors.
[3] Yao et al. Ther Adv Respir Dis. 2019.

APG777+APG333 inhibits activation of bronchial smooth muscle cells from COPD patients; tezepelumab had a weaker effect

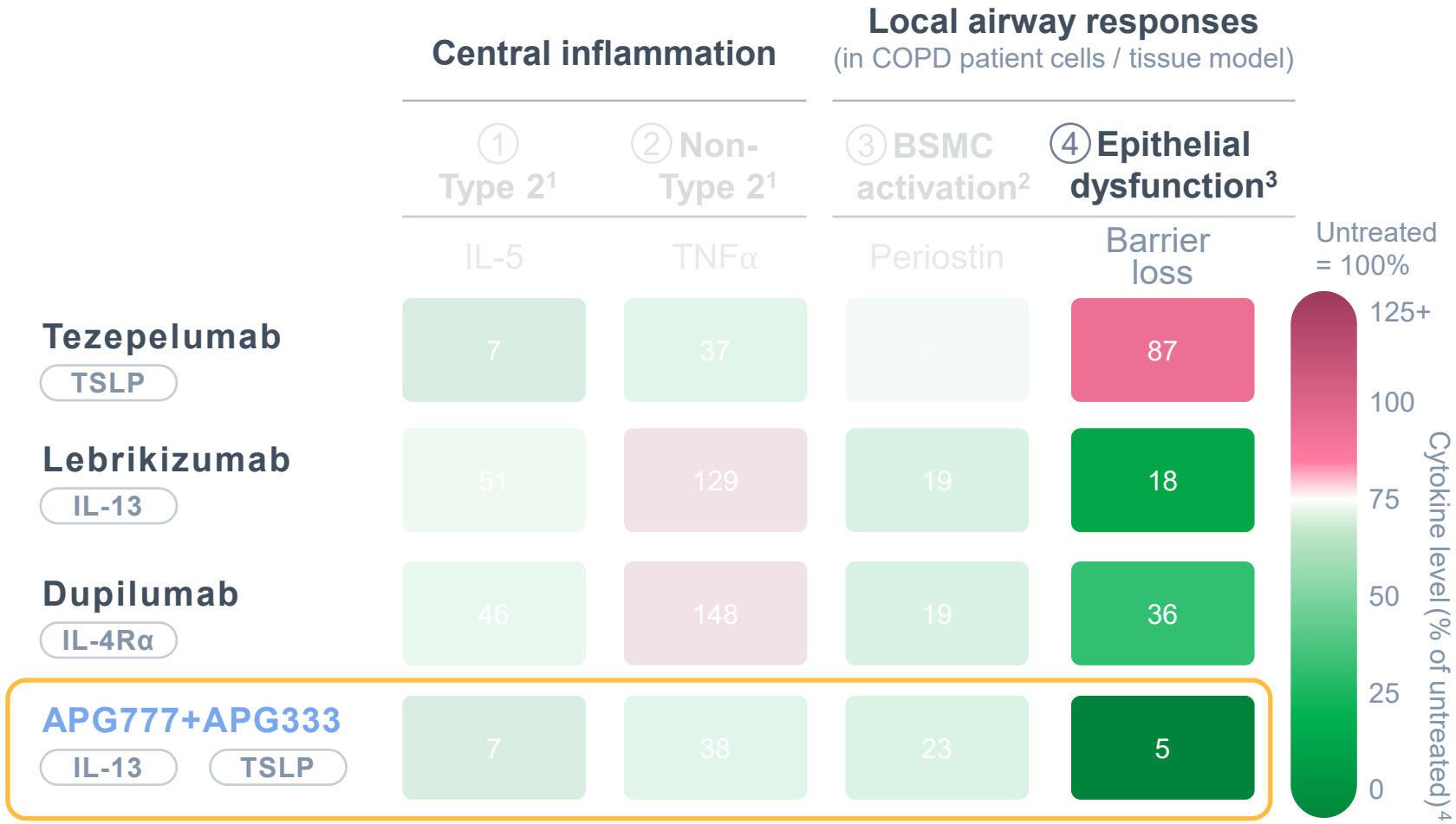
- **IL-13 activates bronchial smooth muscle cells** (BSMCs) that promote local airway responses
- For example, **BSMCs promote airway inflammation** via local release of inflammatory factors (e.g., periostin)



NOTE: [1] ALR performed using four donor pairs of TSLP-primed mDCs plus allogeneic CD4 cells for 5 days. [2] BSMCs from three COPD donors were stimulated with TSLP+IL-13 for 24 hours and cytokines were measured in the supernatant. [3] Responses are reported as mean percent of control across all donors.

APG777+APG333 prevents epithelial dysfunction in a COPD patient lung tissue model; tezepelumab had a weaker effect

- **IL-13 promotes airway epithelial dysfunction** including barrier integrity loss
- **Barrier loss amplifies inflammation** by enabling deeper penetration of allergens, viruses, bacteria, and other harmful particles

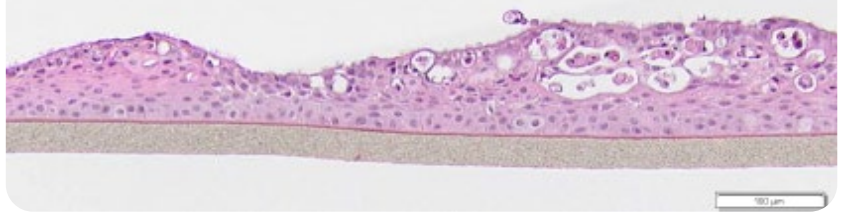


NOTE: [1] ALR performed using four donor pairs of TSLP-primed mDCs plus allogeneic CD4 cells for 5 days. [2] BSMCs from three COPD donors were stimulated with TSLP+IL-13 for 24 hours and cytokines were measured in the supernatant. [3] Human airway epithelial cells from one COPD donor were cultured at the air-liquid interface and treated with TSLP+IL-13. After 7 days, cytokines were measured in the basal supernatants and barrier integrity was measured using transepithelial electrical resistance. [4] Responses are reported as mean percent of control across all donors.

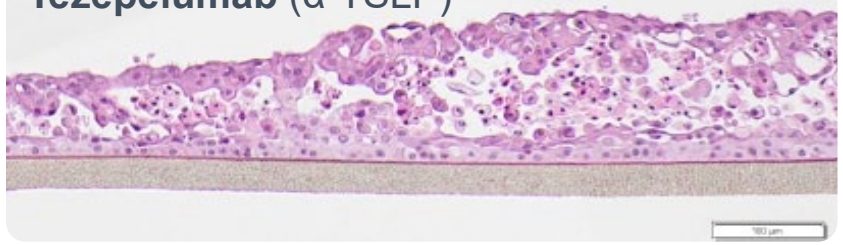
APG777+APG333 protects airway epithelial barrier integrity in a COPD patient lung tissue model

H&E staining of COPD patient lung tissue model (IL-13+TSLP stimulation)

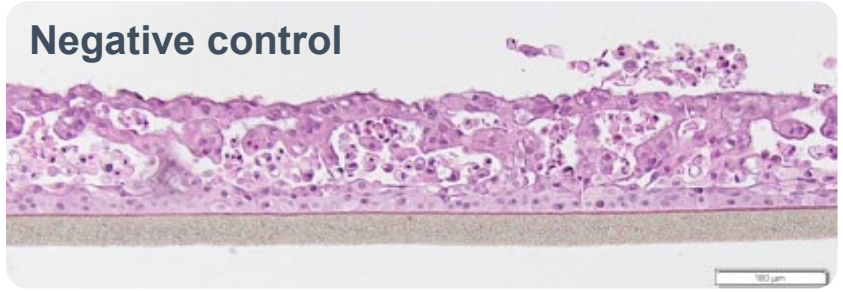
Unstimulated control



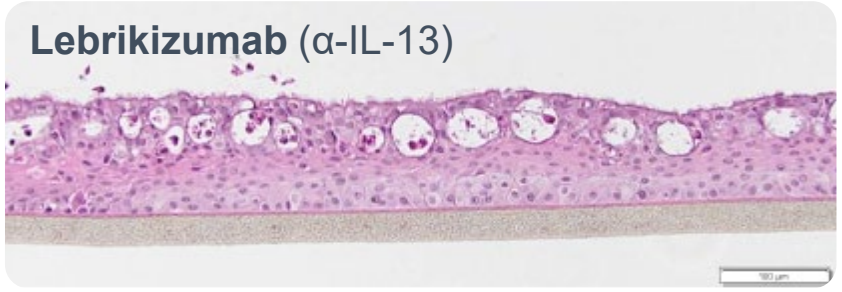
Tezepelumab (α-TSLP)



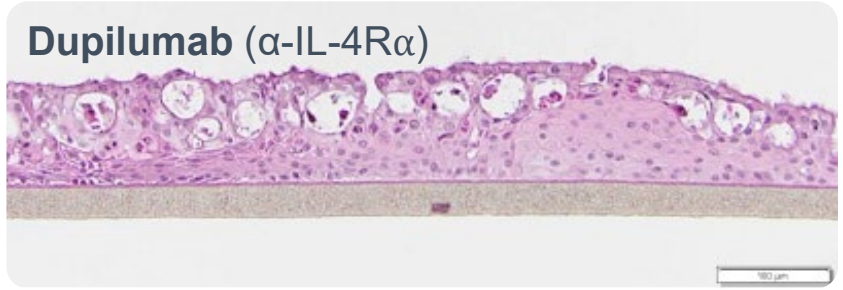
Negative control



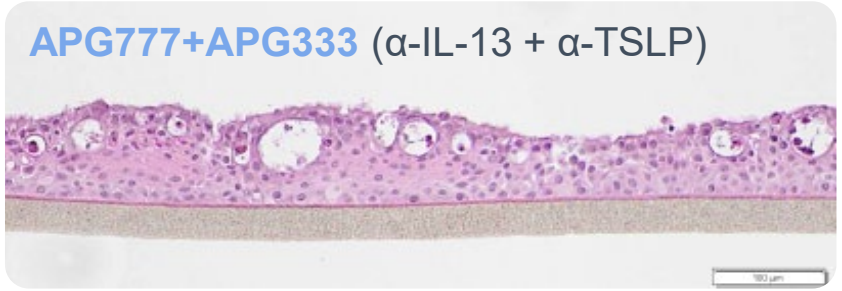
Lebrikizumab (α-IL-13)



Dupilumab (α-IL-4Rα)



APG777+APG333 (α-IL-13 + α-TSLP)



- **Tezepelumab has little effect** on barrier loss
- Epithelial layer clearly disrupted

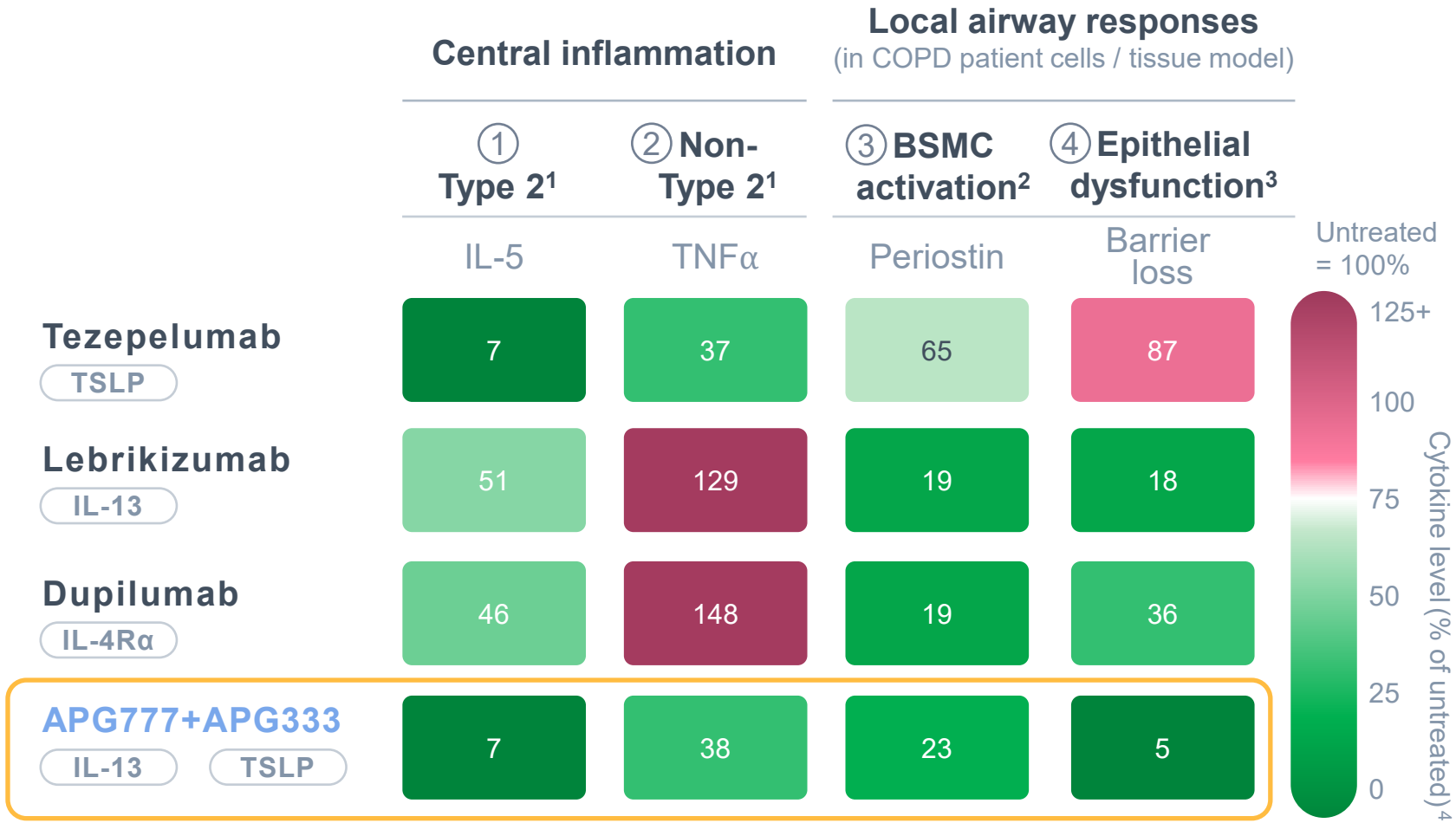
- **APG777+APG333,** lebrikizumab, and dupilumab **prevent barrier loss**
- Epithelial layer appears similar to control

NOTE: Human airway epithelial cells from a COPD donor were cultured at the air-liquid interface and treated with recombinant TSLP+IL-13 (except for unstimulated control). After 7 days, epithelial cultures were fixed and stained. Representative images of three replicates are shown.

APG777+APG333 has a broader effect on both central and local drivers of obstructive airway disease in our preclinical studies

APG777+APG333 combines orthogonal mechanisms for potentially best-in-class efficacy:

- **TSLP** inhibition to block **central inflammation**
- **IL-13** inhibition to address **local airway responses**

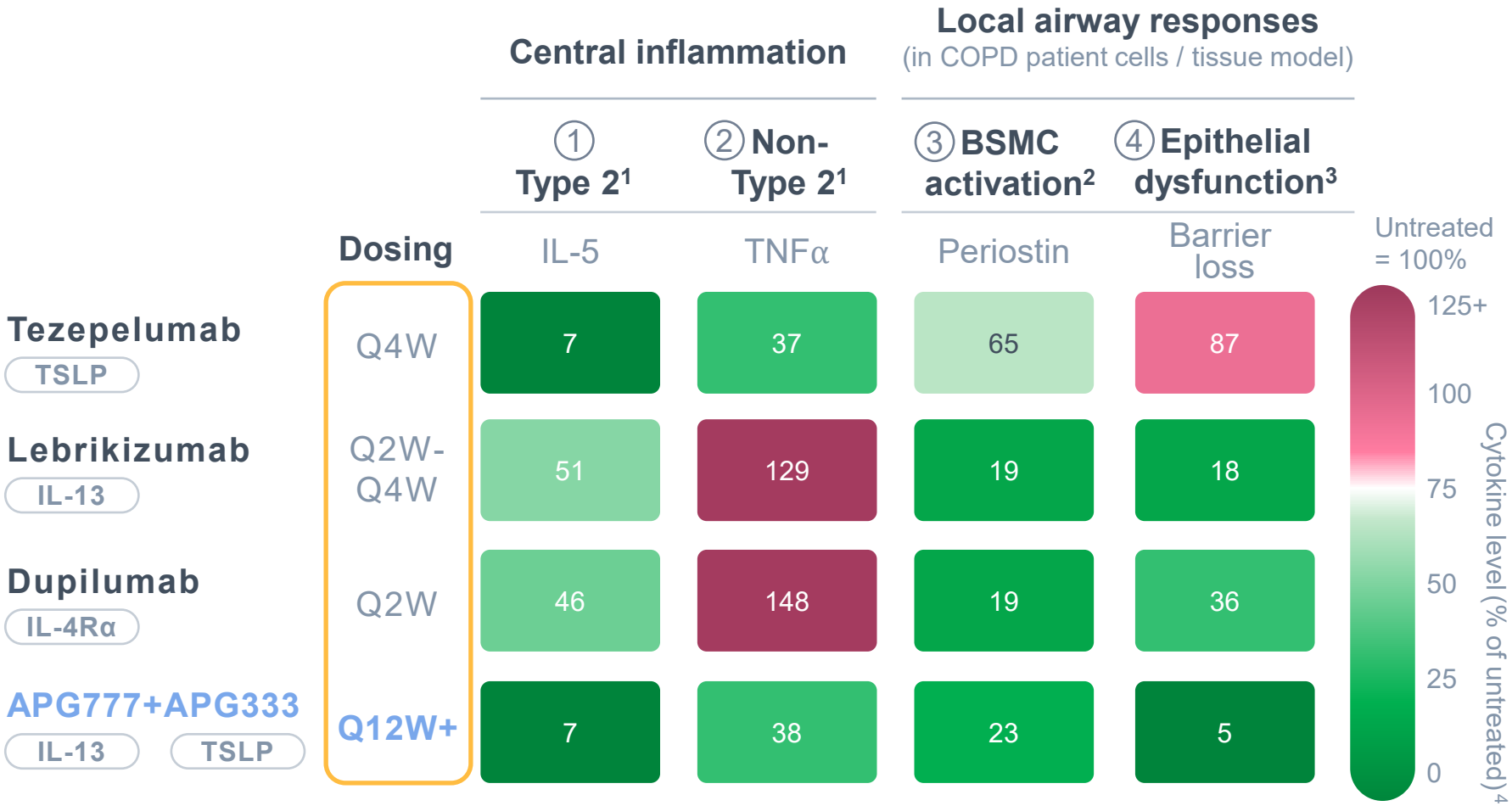


NOTE: [1] ALR performed using four donor pairs of TSLP-primed mDCs plus allogeneic CD4 cells for 5 days. [2] BSMCs from three COPD donors were stimulated with TSLP+IL-13 for 24 hours and cytokines were measured in the supernatant. [3] Human airway epithelial cells from one COPD donor were cultured at the air-liquid interface and treated with TSLP+IL-13. After 7 days, cytokines were measured in the basal supernatants and barrier integrity was measured using transepithelial electrical resistance. [4] Responses are reported as mean percent of control across all donors.

APG777+APG333 enables potentially best-in-class efficacy and dosing

- **APG777+APG333** has the potential to improve clinical outcomes while still **minimizing the injection burden**

- APG777+APG333 has the potential for **every three-month dosing** (or less frequent) vs. every 2- or 4-weeks for approved monotherapies



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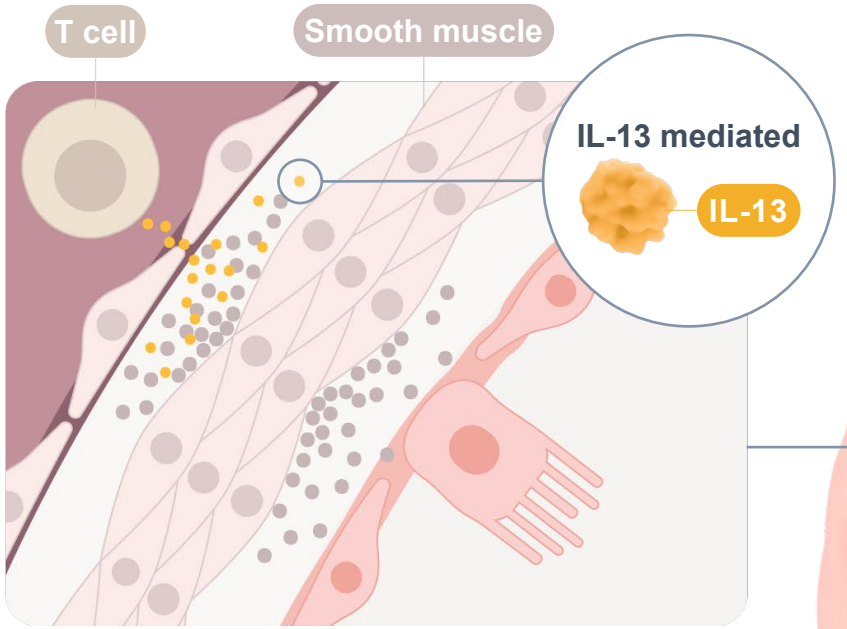
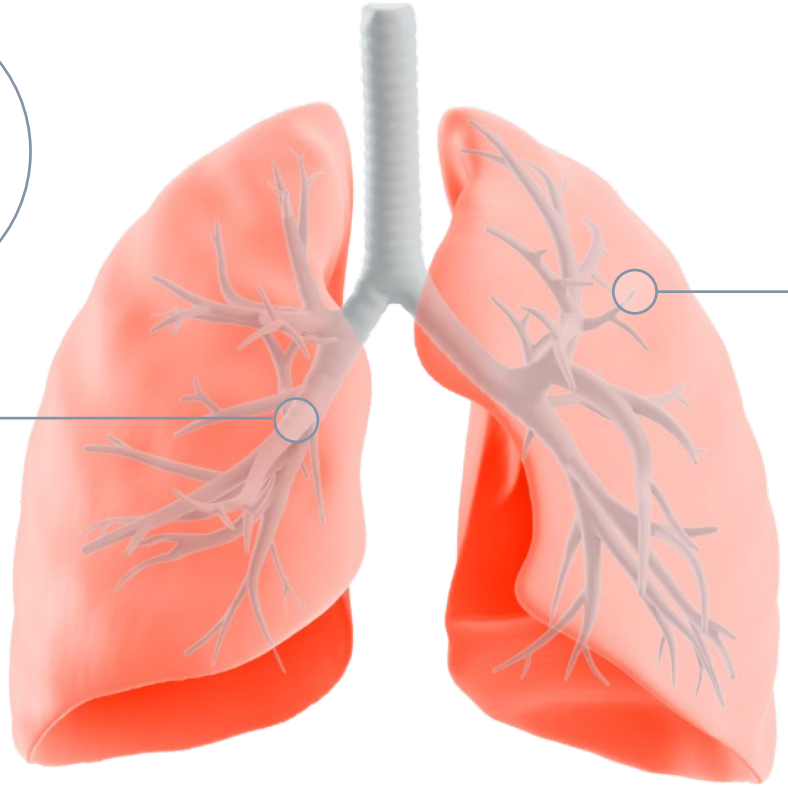
APG777+APG333 targets both central and local drivers of obstructive airway disease

Local Airway Inflammation



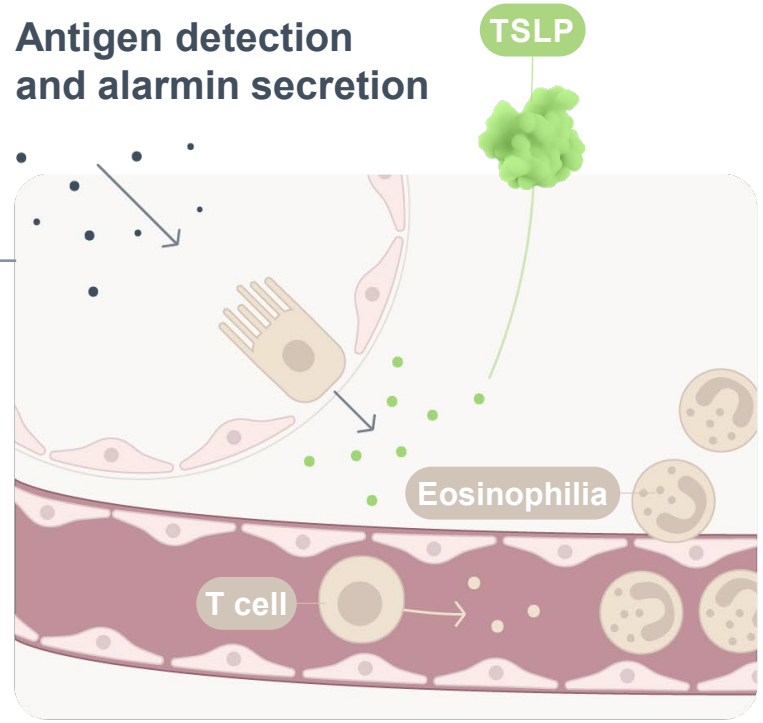


Bronchial smooth muscle activation

Inflammatory chemokine/ cytokine secretion

Airway epithelial barrier integrity loss

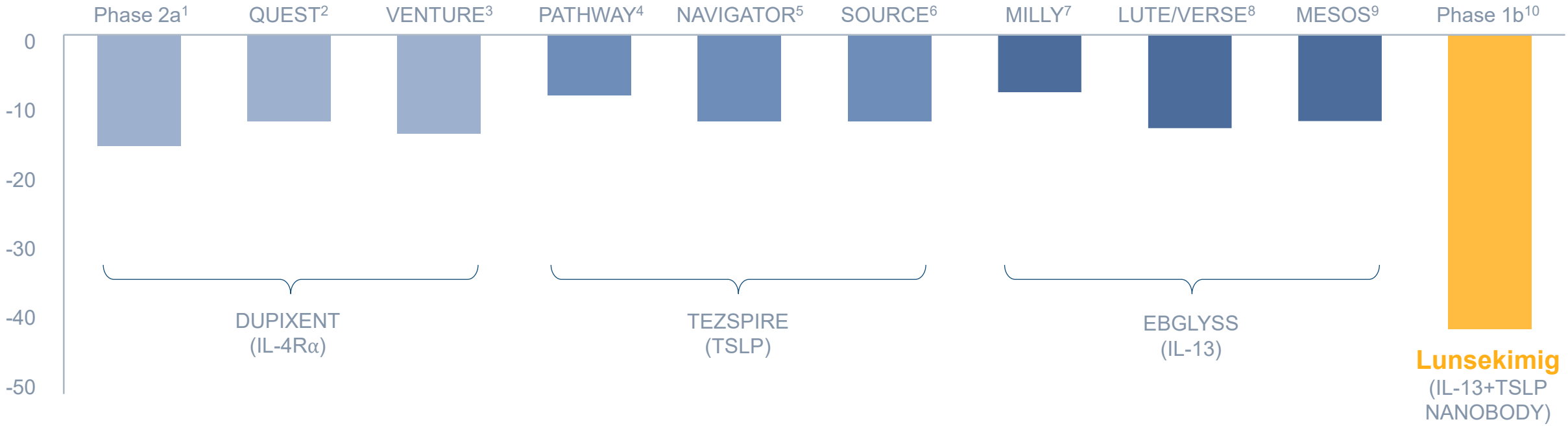
Central Inflammation



T cell activation and type 2 cytokine secretion ↑

Systemic eosinophilia and tissue infiltration ↑

Clinical POC for combining IL-13 and TSLP inhibition exists with lunsekimig, an every 2- to 4-week dosed NANOBODY currently in Phase 2 trials



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

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

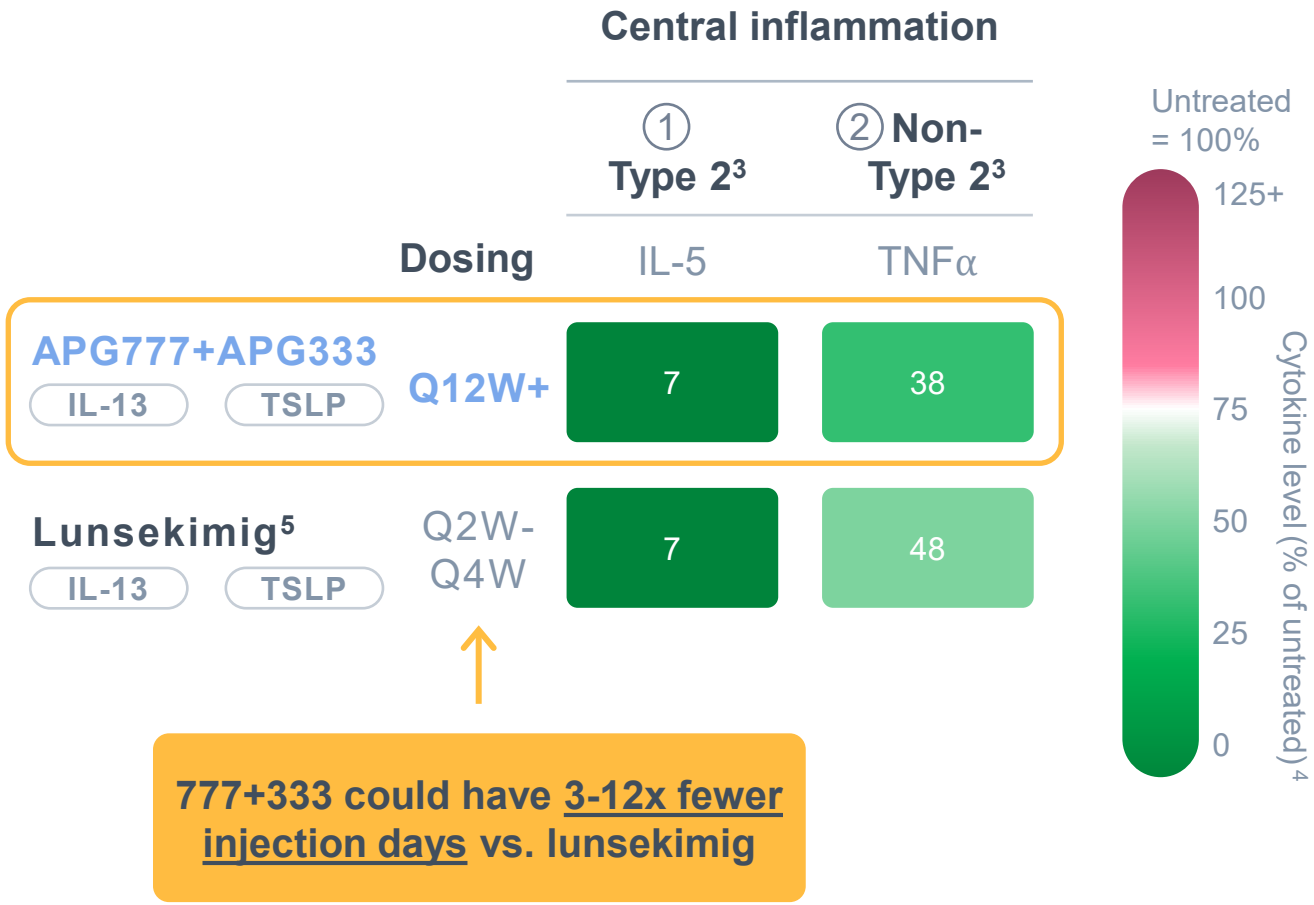
SOURCE: [1] Wenzel S, et al. NEJM, 2013. [2] Castro M, et al. NEJM, 2018. [3] Rabe KF et al. NEJM, 2018. [4] Corren JC, et al. NEJM, 2017. [5] Menzies-Gow A, et al. NEJM, 2021. [6] Weschler M, et al. Lancet Respir Med, 2022. [7] Corren JC, et al. NEJM., 2011. [8] Hanania NA, et al. Thorax, 2015. [9] Russell RJ, et al. Lancet Respir Med, 2018. [10] Deiteren A, et al. ATS, 2023.

APG777+APG333 performs similar to lunsekimig preclinically, but with potentially far fewer injections

- **Lunsekimig** is a single-domain multispecific antibody **targeting IL-13 and TSLP**

- Lunsekimig is currently in **Phase 2 for asthma** (all comers) with data expected in 2025

- With **~10-day half-life**[1], lunsekimig is expected to be **dosed every 2- to 4-weeks**[2]



SOURCE: [1] Deiteren et al. (2024) Clin Transl Sci. DOI: 10.1111/cts.13864. [2] Lunsekimig clinical trial design for High-risk Asthma (NCT06676319) and Chronic Rhinosinusitis With Nasal Polyps (NCT06454240).
NOTE: [3] ALR performed using four donor pairs of TSLP-primed mDCs plus allogeneic CD4 cells for 5 days. [4] Responses are reported as mean percent of control across all donors. [5] Lunsekimig produced and purified based on published sequence and evaluated at a concentration with an equivalent molarity of APG777+APG333.

Respiratory Development Plan

Amol Kamboj, MD
VP, Clinical Development

Monotherapy development could expand APG777's impact and enable potentially best-in-class respiratory combination

Wave 1: Prove out monotherapies

Initial safety and proof of concept

- **Phase 1b trials** in asthma for: **APG808, APG777, APG333**

Efficacy and dose optimization

- **APG777 Phase 2b trial** in asthma

  → Pipeline-in-a-product potential

Wave 2: Test potential best-in-class respiratory combination

Proof of concept

- **APG777+APG333 Phase 2 trial** in asthma
- **APG777+APG333 Phase 2 trial** in COPD

APG333 Phase 1 readout anticipated in 2025

Trial design elements

Double-blind, placebo-controlled, first-in-human trial
Single ascending dose in healthy volunteers

N ~ 32

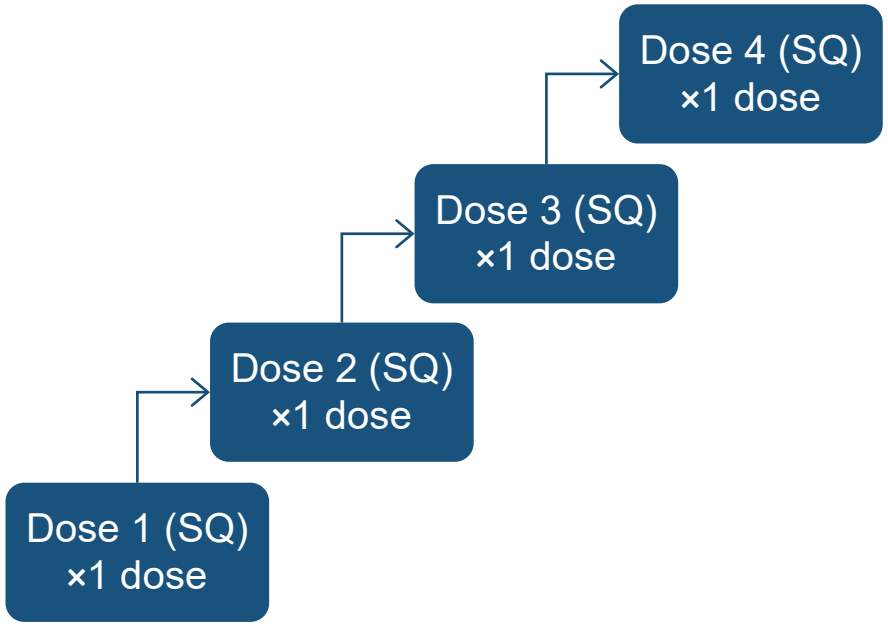
8 per cohort (6:2 active:placebo)

Key inclusion criteria: healthy adult volunteers

Primary endpoint: safety

Secondary endpoints: PK, ADA

Single ascending dose[1]



Study objectives

Confirm tolerable **safety profile to enable future combination trials**

Establish **optimized PK profile** with a half-life of at least ~60 days

Determine **dosing regimens** to sustain exposures similar to tezepelumab

Phase 1 readout in 2025 could confirm potential for best-in-class dosing (goal of every 3-months or less frequent)

NOTE: SQ = subcutaneous. [1] SAD dose ascension will ultimately be determined by the SRC (Scientific Review Committee) based on available data.

APG777 and APG333 Phase 1b trials in patients with asthma expected to initiate in 2025

Design elements for Phase 1b trials

Double-blind, placebo-controlled
Single dose regimen in patients with asthma

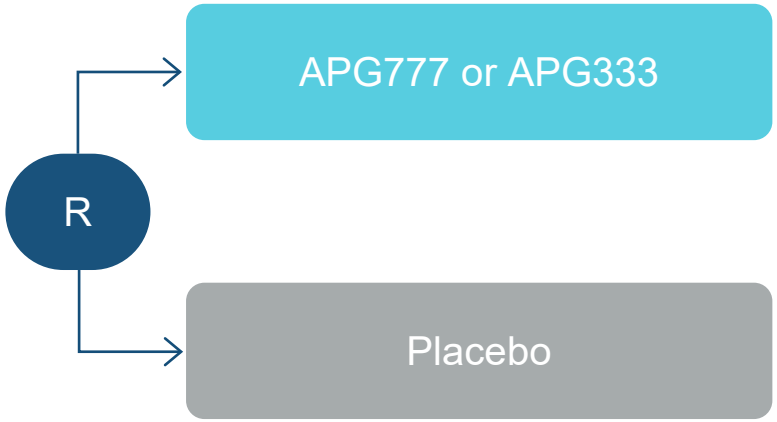
N ~ 20

Key inclusion criteria:
- Mild-to-moderate asthma
- FeNO >25 ppb

Primary endpoint: safety

Additional endpoints: PK, ADA, fractional exhaled nitric oxide (FeNO)

Schematic for Phase 1b trials



Study objectives

Confirm safety of APG777 and APG333 as monotherapies in **asthmatic patient population**

Demonstrate activity of APG777 and APG333 via **maximal suppression of FeNO in line with standard of care** (~10-15 parts per billion change from baseline)

Show **durable suppression of FeNO supporting every 3 months or less frequent**

Commercial opportunity and strategy

Jeff Hartness
Chief Commercial Officer

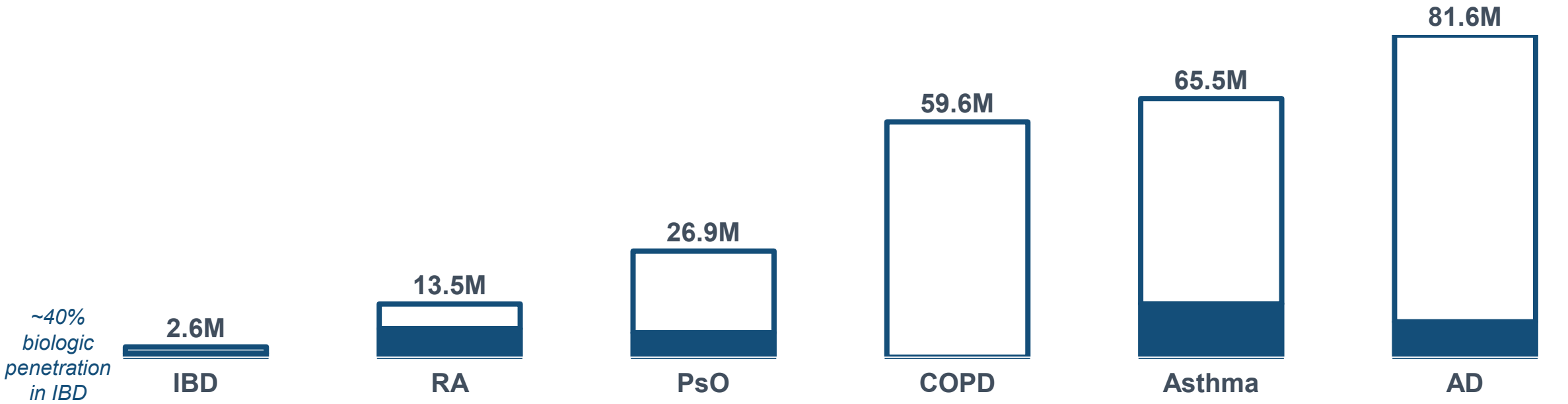
Apogee is focused on the largest I&I markets

Estimated population size. Moderate or severe, WW

US biologics penetration: 0% ⟷ 60%

Mature I&I markets have **consistently achieved high biologics penetration** (~25-60% after 15-20 years)

Apogee's current indications are the largest and least penetrated markets today

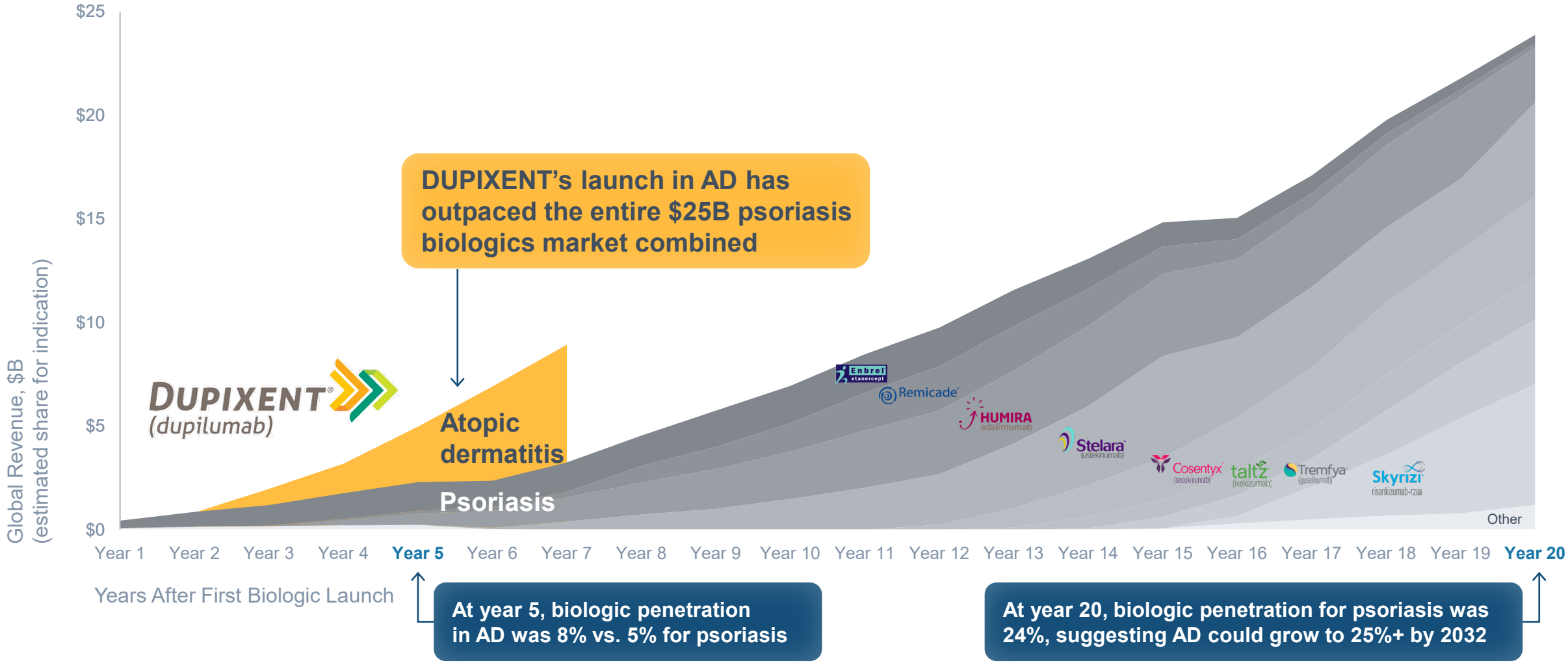


2023 WW Market Size: $23B $26B $27B

Significant future market opportunity

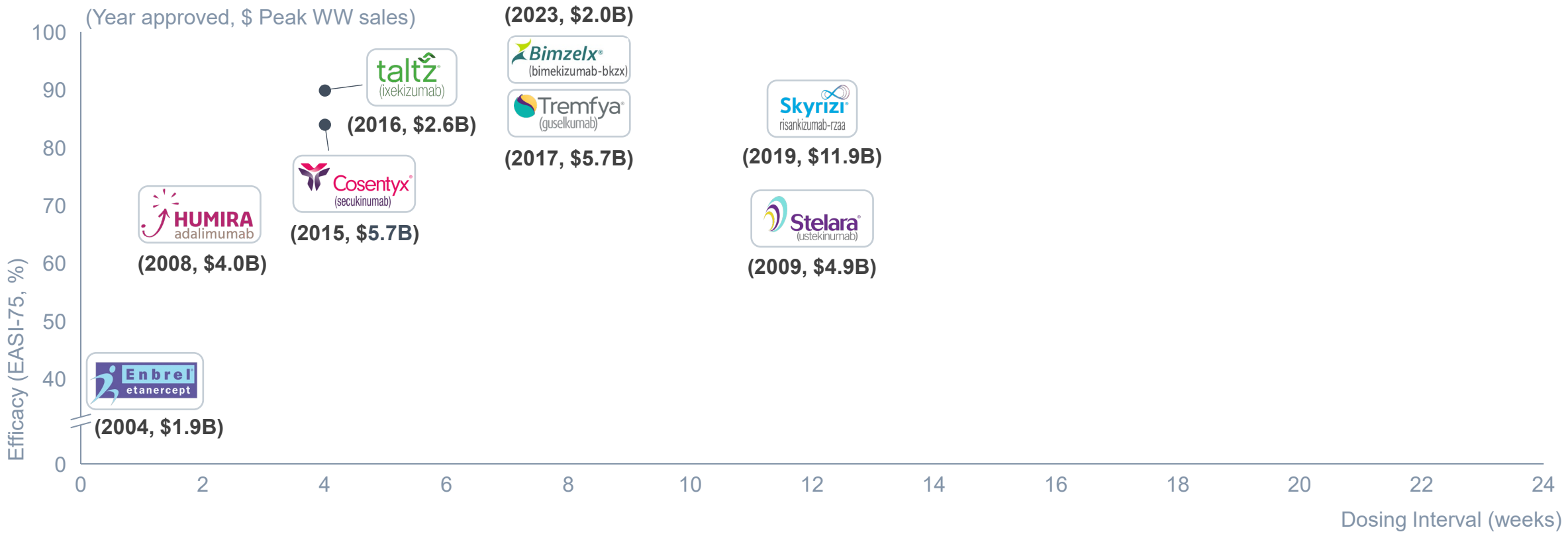
NOTE: IBD = Inflammatory bowel disease; RA = Rheumatoid arthritis; PsO = Psoriasis; COPD = Chronic obstructive pulmonary disease; AD = Atopic dermatitis.
SOURCE: Academic journals, disease foundations, WHO, CDC, census data, EvaluatePharma, analyst research.

Apogee has the potential of becoming a leader in a future $50B+ market that is in its early years and growing rapidly



SOURCE: EvaluatePharma
NOTE: Year 1 for DUPIXENT in AD is 2017 and for Biologics in PsO is 2004 (i.e., ENBREL approval). Other includes SILIQ, ILLUMYA, and CIMZIA, which all launched prior to SKYRIZI.

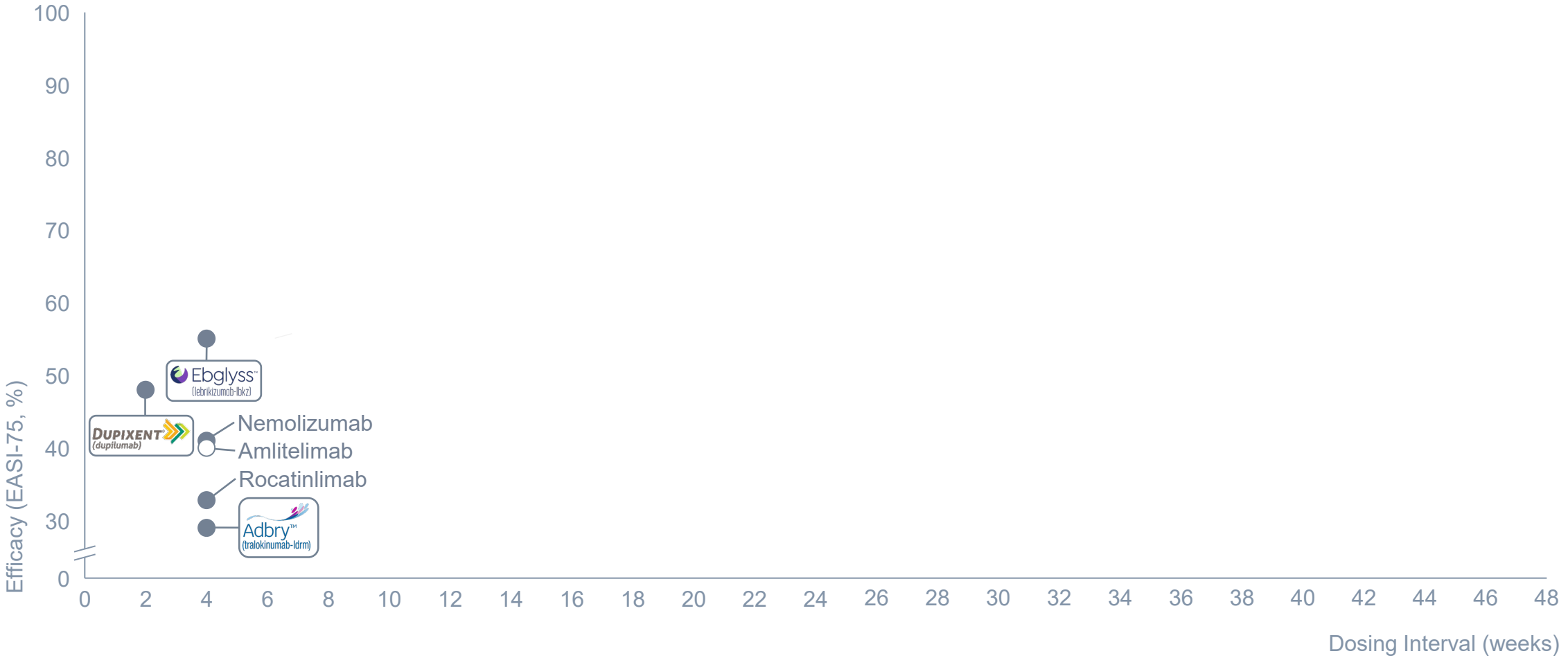
Psoriasis, a market analog to atopic dermatitis, has seen improved dosing and efficacy drive market success



- **Psoriasis is not a winner take all market — 8 blockbusters**
- **SKYRIZI, a late entrant, has #1 share due to quarterly dosing which improves adherence[1]**

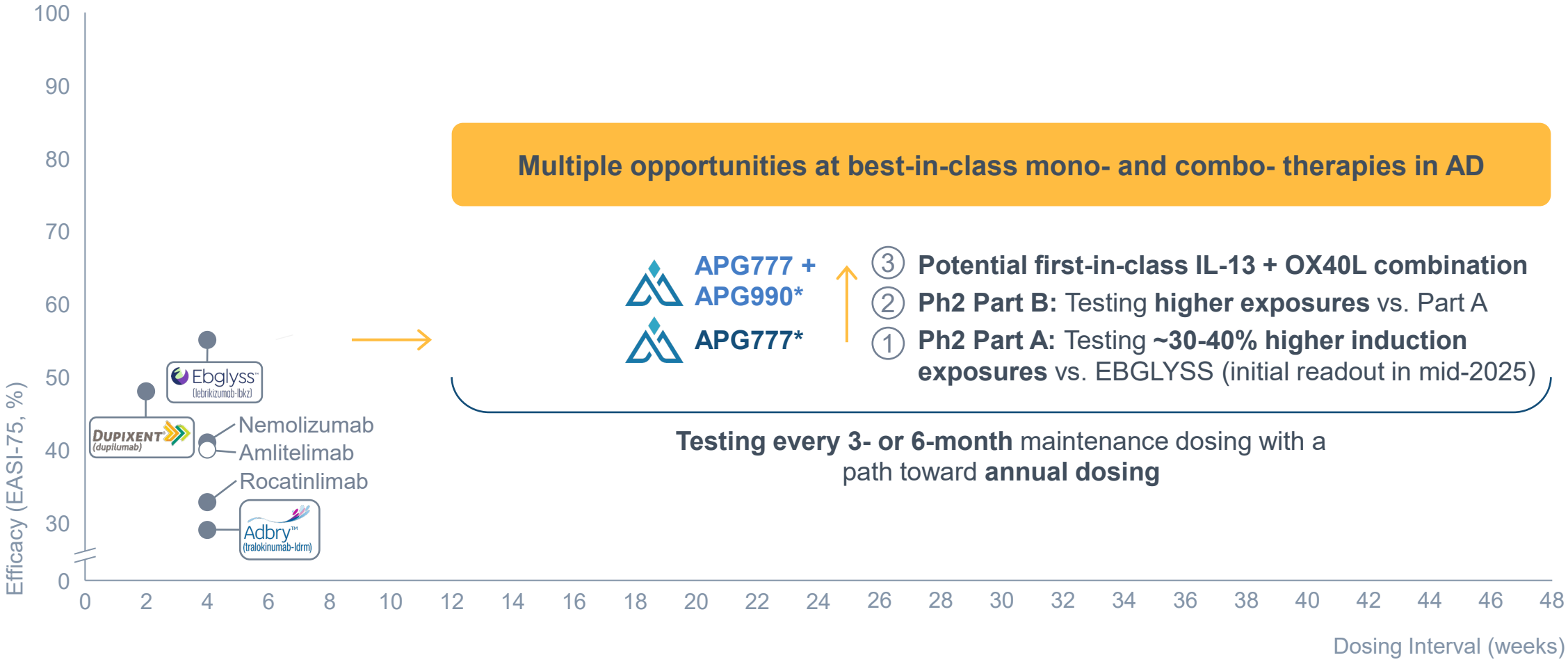
NOTE: PsO = Psoriasis. PsA = Psoriatic Arthritis. Year denotes US launch year for adults with moderate to severe plaque psoriasis. Efficacy data are derived from different clinical trials at different points in time, with differences in trial design, patient populations, and statistical analysis. As a result, cross-trial comparisons cannot be made. No head-to-head trials have been conducted among all biologics shown. Assumes 1 EUR = 1.07 USD.
[1] Real-world evidence shows SKYRIZI patients experienced fewer drug changes and a higher probability of drug survival compared with those treated with other biologic therapies for PsO and PsA.
SOURCE: Armstrong AW, et al JAMA Dermatol. 2020. Gordon KB, et al Lancet 2021. Reich K, et al Lancet 2021. GlobalData. EvaluatePharma. USPIs. Wall Street research and management projections. Erik L et al ACR Convergence 2023.

Atopic dermatitis, in contrast to psoriasis, has far fewer options for patients



NOTE: Only DUPIXENT, ADBRY, and EBGLYSS are approved in the US. 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] EBGLYSS 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] Nemolizumab 30 mg Q4W Ph3 avg. Silverberg J et al EADV 2023. [5] Rocatinlimab 150mg Q4W Ph2b Guttman-Yassky E et al Lancet 2023. [6] Amlitelimab 250mg Q4W Ph2b Weidinger S et al EADV oral presentation 2023.

Apogee is potentially the first in atopic dermatitis to provide transformational dosing and efficacy



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

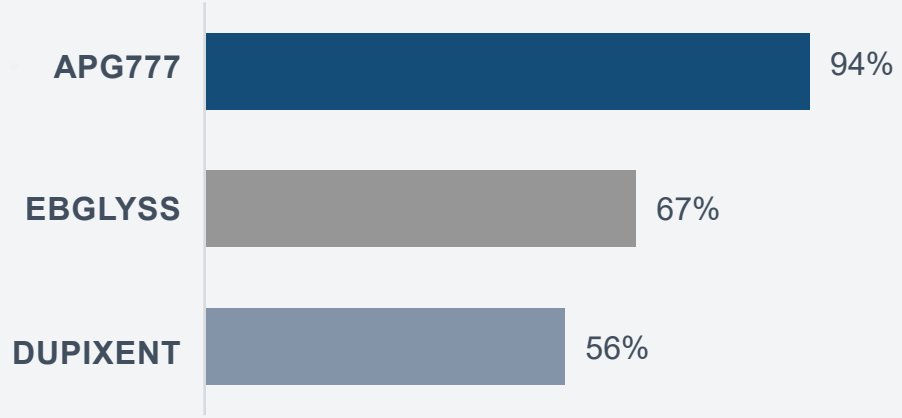
SOURCE: [1] EBGLYSS 250mg Q2W Ph3 avg. Silverberg JI et al. AAD 2022. [2] DUPIXENT 300 mg Q2W mono Ph3 avg. DUPIXENT USPI. [3] ADBRY 300 mg Q2W mono Ph3 avg. ADBRY USPI. [4] Nemolizumab 30 mg Q4W Ph3 avg. Silverberg J et al EADV 2023. [5] Rocatinlimab 150mg Q4W Ph2b Guttman-Yassky E et al Lancet 2023. [6] Amlitelimab 250mg Q4W Ph2b Weidinger S et al EADV oral presentation 2023.

Patients and physicians prefer APG777's quarterly dosing profile; payers support 1L biologic access

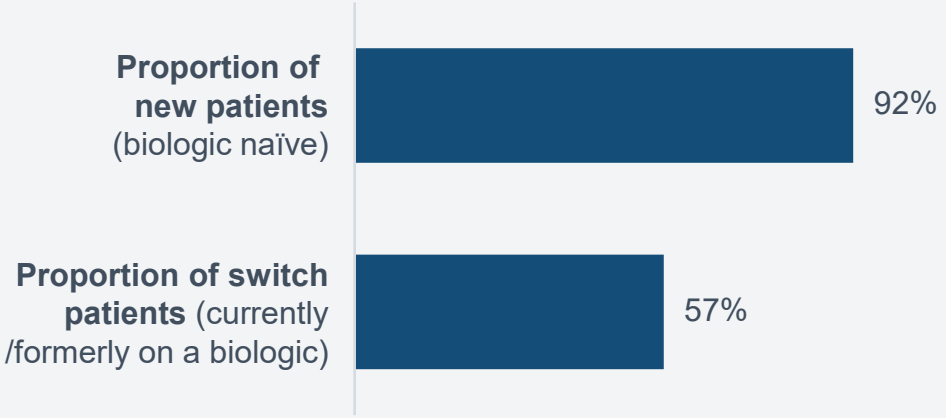
Market research supports APG777's differentiated profile

(based on blinded TPP with equivalent efficacy and safety as DUPIXENT but with every 3-month maintenance dosing)

Patient likelihood to take action for each treatment[1]



Physician intent to use a product with APG777 Target Product Profile[2]



> "**[APG777] would be placed** from a coverage perspective or a tiering perspective **at parity as DUPIXENT**"
>
> VP of Pharmacy, Large National PBM #1[3]

> "[APG777] would be **covered at parity**… if [APG777] shifts the market, then it may **move up to preferred**"
>
> VP of Pharmacy, Large National PBM #2[3]

> "[APG777] would be **parity and co-preferred** alongside DUPIXENT and ADBRY"
>
> VP of Pharmacy, National MCO[3]

SOURCE: Patients: TRINITY Qualitative Research with N=18 AD Patients, August 2024. Physicians: 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. Payers: Real Endpoints Qualitative Research with N=6 payers, February 2024. Charles River Associates research with N=10 payers, August 2024.
NOTE: [1] AD patients responding 6 or 7 on a scale from 1 to 7 rating their likelihood to take action after reviewing a blinded TPP for each treatment. APG777 TPP based on equivalent efficacy and safety as DUPIXENT. [2] For providers where likeliness to prescribe Product Y (equivalent efficacy and safety as DUPIXENT, but with Q3M dosing) 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. [3] Payer coverage expectations are based on a product with similar efficacy, safety, and net pricing as DUPIXENT, but with Q3M dosing.

Beyond APG777 in AD, multiple potential blockbuster expansions in dermatology, respiratory, and GI

Identify TA-specific dosing regimen(s) in Phase 2

↓

Option to launch directly to Ph3 for most promising expansions

↓

Opportunity to raise the bar through combos

Dermatology

Atopic dermatitis

↓

- ★ *AA*
- • **BP**
- ★ *HS*
- • **PN**

Respiratory

Asthma

↓

- ★ **AR**[1]
- • **ColdU**
- ★ **COPD**
- ★ **CRSwNP**
- ★ **CSU**

Gastroenterology

EoE

↓

- ★ *Celiac Disease*
- • *EGID*
- ★ *UC*[2]

BOLD = Established “direct to Phase 3” regulatory precedent[3]

★ Potential blockbuster indication

NOTE: [1] Perennial. [2] Eosinophilic subtypes. [3] Based on either DUPIXENT or EBGLYSS clinical development pathway.
PN = Prurigo Nodularis. BP = Bullous Pemphigoid. AA = Alopecia Areata. HS = hidradenitis suppurativa. CSU = Chronic Spontaneous Urticaria. CRSwNP = Chronic Rhinosinusitis with Nasal Polyps. AR = Allergic Rhinitis. ColdU = Cold Inducible Urticaria. EoE = Eosinophilic esophagitis. UC = Ulcerative Colitis. EGID = Eosinophilic Gastrointestinal Disorders (non-EoE).

Closing remarks

Michael Henderson, MD
Chief Executive Officer

Over the next 2 years, 8 clinical trial readouts expected across our pipeline

$754M in cash with runway into 2028

	★ KEY READOUT	2025	2026
Potential best-in-class monotherapy in AD	**APG777** IL-13	★ **Mid-2025: AD Phase 2 16-week PoC readout** • 1H: Asthma Phase 1b initiation • 2H: Asthma Phase 2b initiation	★ **1H: AD Phase 2 Part A 52-week readout** ★ **2H: AD Phase 2 Part B 16-week readout** • Asthma Phase 1b readout • EoE Phase 2 initiation
Potential first- or best-in-class combination approaches	**APG777+APG990** IL-13 OX40L	• AD Phase 1b PoC trial initiation (against DUPIXENT)	★ **2H: AD Phase 1b PoC readout (against DUPIXENT)**
	APG777+APG333 IL-13 TSLP	• Additional clinical plan announced	
Potential best-in-class mAbs for combinations	**APG808** IL-4Rα	• 1H: Asthma Phase 1b readout	
	APG990 OX40L	• 1H: Initial Phase 1 PK & safety in HVs	
	APG333 TSLP	• 2H: Initial Phase 1 PK & safety in HVs	

NOTE: As of September 30, 2024, Apogee had cash, cash equivalents and marketable securities of $754M.