NEKTAR® THERAPEUTICS

Nektar Therapeutics Corporate Presentation

January 2026

Bold immune science.
Transformative immune health.

Forward-looking statements

This presentation includes forward-looking statements regarding Nektar's proprietary drug candidates, the timing of the start and conclusion of ongoing or planned clinical trials, the timing and outcome of regulatory decisions, unaudited year-end cash and investments and sufficiency of working capital and future availability of clinical trial data.

Actual results could differ materially and these statements are subject to important risks detailed in Nektar's filings with the SEC including the Form 10-Q filed on November 7, 2025.

Nektar undertakes no obligation to update forward-looking statements as a result of new information or otherwise.

Targeting immunology and inflammation with immune modulating therapies

Deep Understanding of Immunology

Novel approaches to address the imbalance and dysfunction of regulatory T cells (Tregs) to restore the body's self-tolerance mechanisms and achieve immune homeostasis

Novel Targets and Differentiated Candidates

Lead candidate (Ph2), rezpegaldesleukin, is a first-in-class IL-2 agonist selective Treg therapy

Preclinical TNFR2 programs are designed to potentiate suppressive effects of Tregs

Compelling Proof-of-Concept Data

Promising clinical data for rezpegaldesleukin in atopic dermatitis suggest potential as a differentiated remittive and disease modifying therapy

Large Indications with High Unmet Need

Rezpegaldesleukin is being studied in two large, randomized Phase 2b studies in atopic dermatitis and alopecia areata and in one Phase 2 clinical trial in Type 1 diabetes mellitus

Well Capitalized Through Upcoming Catalysts

Expect to end the year with $240M in cash and cash equivalents

Cash runway into the second quarter of 2027

Strong opportunities exist within the I&I market

Blockbuster Potential Across I&I Markets

- **Over 70% of advanced therapies launched by 2022 have achieved over $1 billion in annual revenue within a single indication**, particularly in large, high-burden disease areas such as asthma, atopic dermatitis, Crohn's disease, psoriasis, rheumatoid arthritis, and ulcerative colitis

Attractive Market Dynamics within I&I Indications

- Multiple blockbuster therapies can **successfully coexist within the same indication**, reflecting the large patient population, heterogenous disease presentation, and evolving unmet needs
- **High-prevalence indications support sustained market expansion**, even in the presence of entrenched competitors

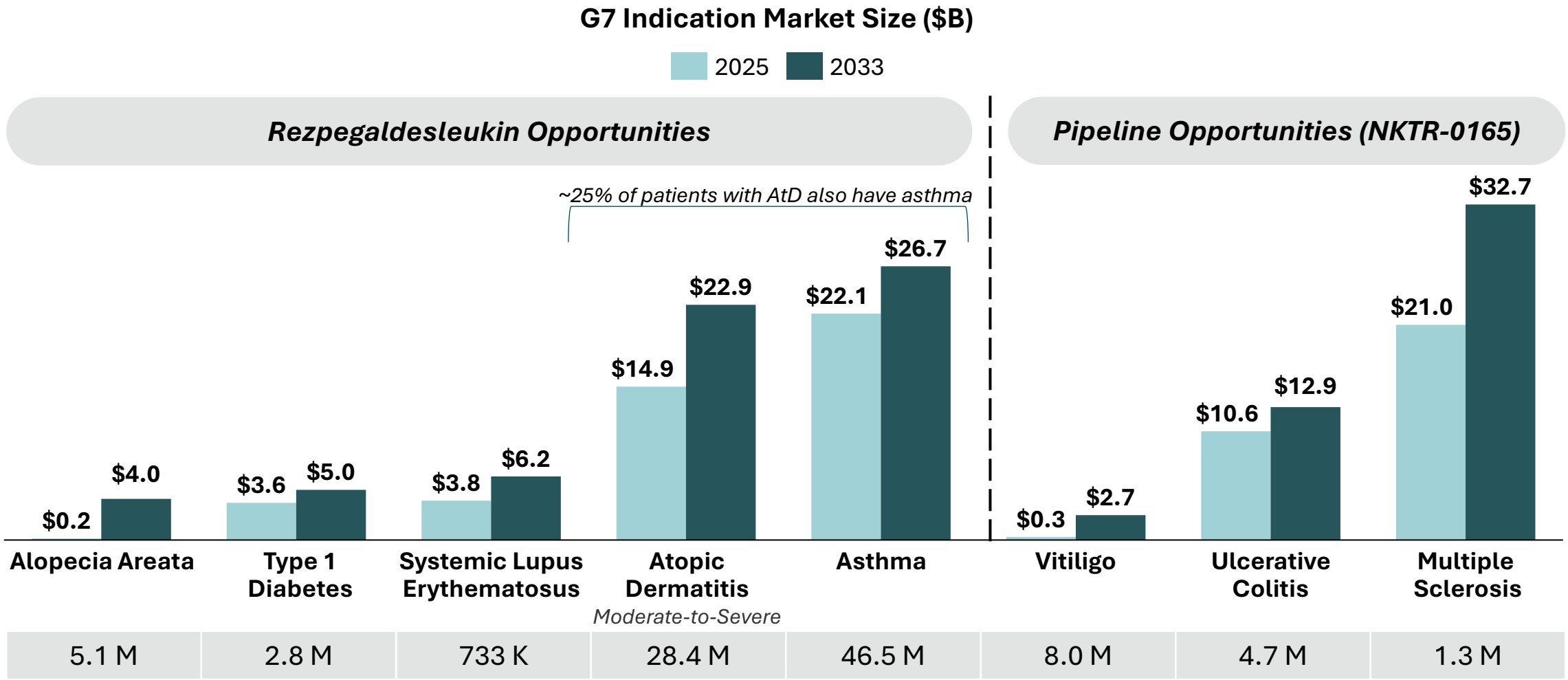
Innovation Drives Penetration and Pipeline Value

- **Novel mechanisms of action** continue to drive increased biologic penetration, particularly in traditionally underpenetrated indications like atopic dermatitis
- New entrants offering **improved efficacy, safety, or convenience** are capturing share despite competition from incumbent products
- Strong potential for **“pipeline-in-a-product” strategies**, with many agents demonstrating activity across **multiple autoimmune and inflammatory indications**

Nektar pipeline

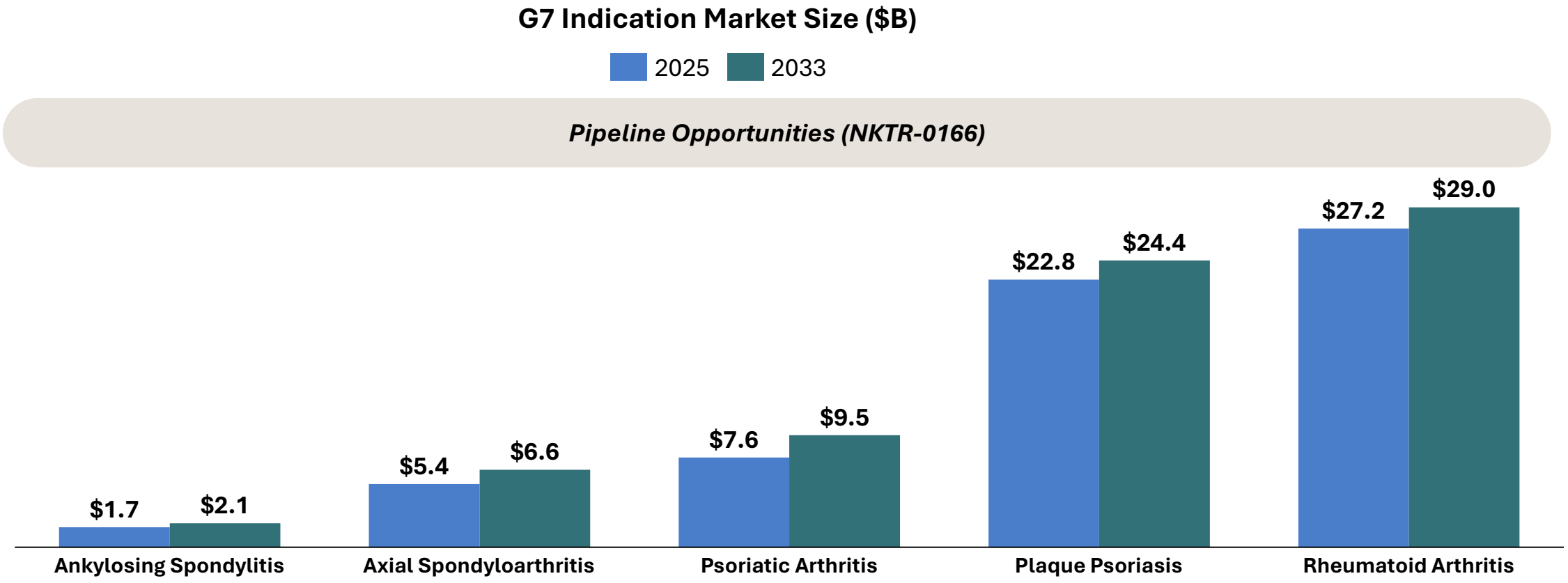
	Program	Indication	Stage	Preclinical	Phase 1	Phase 2	Phase 3	Partner
Immunology	**Rezpegaldesleukin** *(IL-2 T Regulatory Cell Stimulator)*	**Atopic Dermatitis**	**Completed Enrollment in Phase 2b Study (REZOLVE-AD)**			***Phase 2; Fast Track Designation***	***Maintenance Data Q1 2026***	
		Alopecia Areata	**Enrollment Completion in March 2025 (REZOLVE-AA)**			***Phase 2; Fast Track Designation***	***Follow-up/Extension Data Q2 2026***	
		Type 1 Diabetes (Stage 3)	**TrialNet P2 Study**			***Phase 2***	Type 1 Diabetes TrialNet	
	NKTR-0165 *(TNFR2 Agonist Antibody)*	**Multiple Sclerosis & Other I&I Indications**	**Preclinical**	***Preclinical***	BiolojicDesign			
	NKTR-0166 *(Bispecific Antibody)*	**I&I Indications**	**Preclinical**	***Preclinical***				
	NKTR-422 *(PEG-CSF)*	**Fibrotic Diseases & Other Indications**	**Preclinical**	***Preclinical***				
Oncology	**NKTR-255** *(IL-15 Receptor Agonist)*	**Oncology** *(LBCL, NSCLC, Bladder Cancer)*	**Multiple partnered and investigator-sponsored trials in various indications**			***Phase 2***		*Industry Partners* Merck KGaA Darmstadt, Germany; AbelZeta
	NKTR-288 *(Interferon Gamma)*	**Oncology**	**Preclinical**	***Preclinical***				

Nektar is focused on multiple immunology indications with projected growing markets



Sources: Clarivate; Evaluate Pharma WW Market Size Estimates (Alopecia Areata, Type 1 Diabetes, and Vitiligo – 2033 data projected from data available 2024-2030)

NKTR-0166 Expands I&I Breadth to Additional Indications

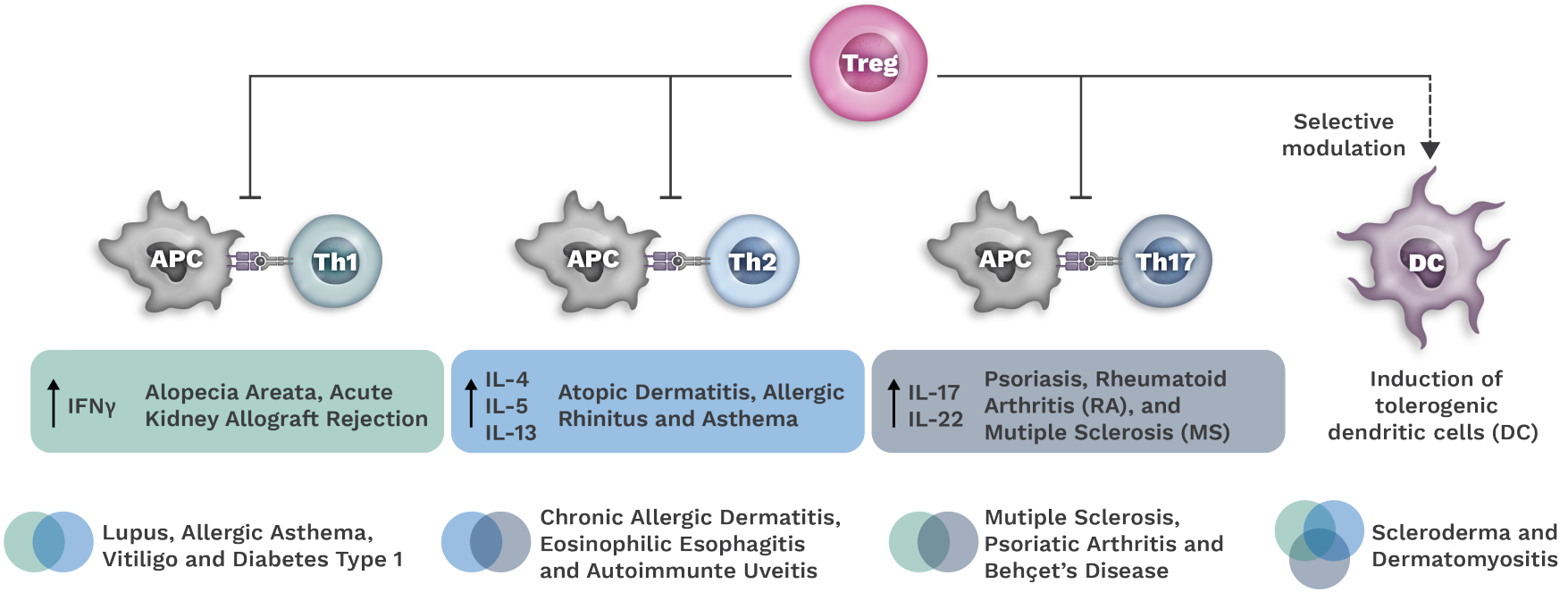


G7 Prevalence

2025	1.5 M	3.8 M	3.5 M	12.0 M	7.6 M

Sources: Clarivate; Evaluate Pharma WW Market Size Estimates (Alopecia Areata, Type 1 Diabetes, and Vitiligo – 2033 data projected from data available 2024-2030)

The central role of regulatory T cells in immune homeostasis



APC=Antigen-presenting cell; Th1 = Mature helper T cell (Th1); Th2 = Mature helper T cell (Th2); Th17 = Mature helper T cell (Th17)

Tregs are crucial for immune homoeostasis and the prevention of autoimmune conditions[1]

- ***IL-2 pathway agonism***
- ***TNFR2 agonism***

Sources: 1 Lykhopiy V, et al. Genes Immun. (2023)

Rezpegaldesleukin in Atopic Dermatitis

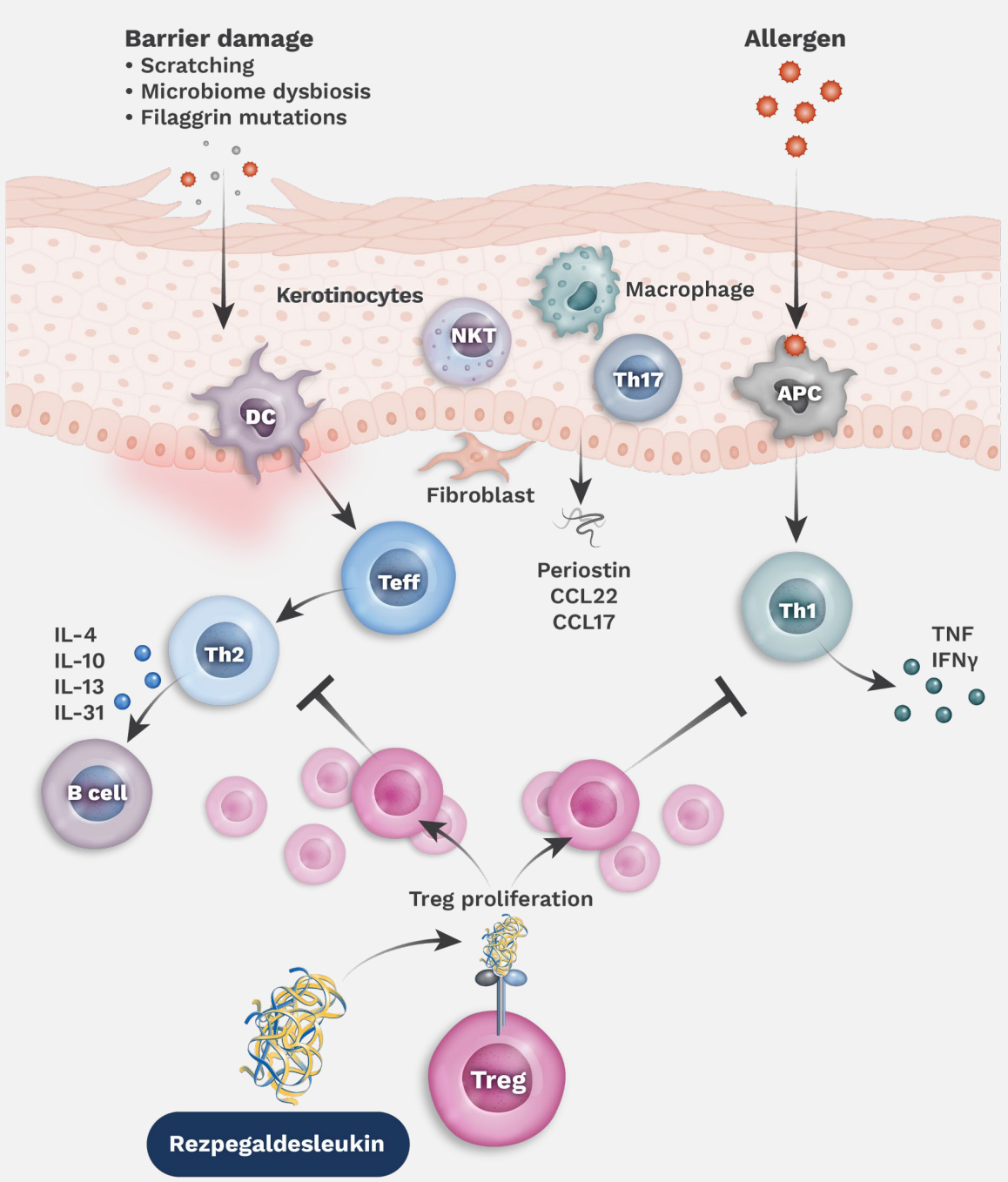
Rezpegaldesleukin
Atopic Dermatitis MOA

Scientific rationale

- By targeting receptors on regulatory T cells (Tregs), rezpegaldesleukin stimulates the proliferation of these cells, including FOX-P3+ Tregs

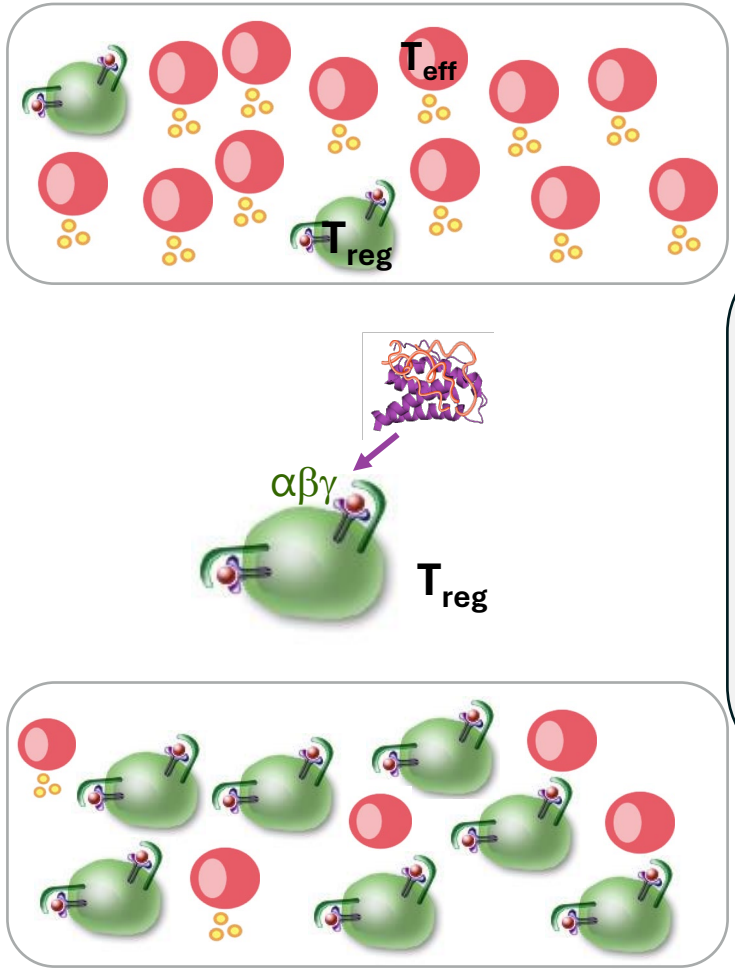
- Regulatory T cells act as a master immune-modulator upstream of the pro-inflammatory cytokine pathways, which drive Th1, Th2, Th17-mediated inflammatory disorders, such as atopic dermatitis

- By increasing the number and functionality of regulatory T cells, this investigational therapy aims to reduce inflammation more effectively than specific antagonist mechanisms that may target only one pathway



Rezpegaldesleukin is a potential first-in-class regulatory T cell mechanism to restore balance in immune system

- Many patients with moderate-to-severe atopic dermatitis (AD) **do not adequately achieve disease control** or have safety/tolerability issues with current therapies
- **Tregs play a central role in controlling AD** by dampening inflammatory cytokines and overactive T cells[1]
- **Granted Fast Track designation in Feb 2025** for treatment of adult and pediatric patients ≥12 years of age with moderate-to-severe AD whose disease is not adequately controlled with topical prescription therapies or when those therapies are not advisable



Increased activity and number of T effector cells shift the balance toward inflammation

Rezpegaldesleukin is a potential T-cell balancing therapy that acts on IL2 receptors and has been shown to[2,3]:

- **Proliferate** regulatory T cells
- **Restore** their functionality, reducing proinflammatory cytokines
- **Offer potential long-term control** of overactive immune responses

Treg expansion and activation restores the immunoregulatory balance

Sources: 1. Silverberg et al. 2024 Nature Communications, 15:9230; 2. Fanton et al. 2022 J. Translational Autoimmunity, 5:100152; 3. Dixit et al. 2021 J Translational Autoimmunity, 4:100103

Atopic dermatitis presents a multi-billion dollar market opportunity

Still high unmet need, especially for new therapies with potential for remittive effect

Atopic dermatitis (AD) is a chronic autoimmune condition that causes inflammation, redness and irritation of the skin. Moderate-to-severe AD is associated with unbearable itching that can result in significant negative impact to quality of life.

~30 million[1]

Adults with AD in U.S.

~220 million[2]

Adults with AD globally

~50%[3]

Adults with AD have moderate-to-severe disease

~8%[4]

Patients with moderate/severe AD are treated with a biologic

There is a high unmet need for a novel mechanism of action to:

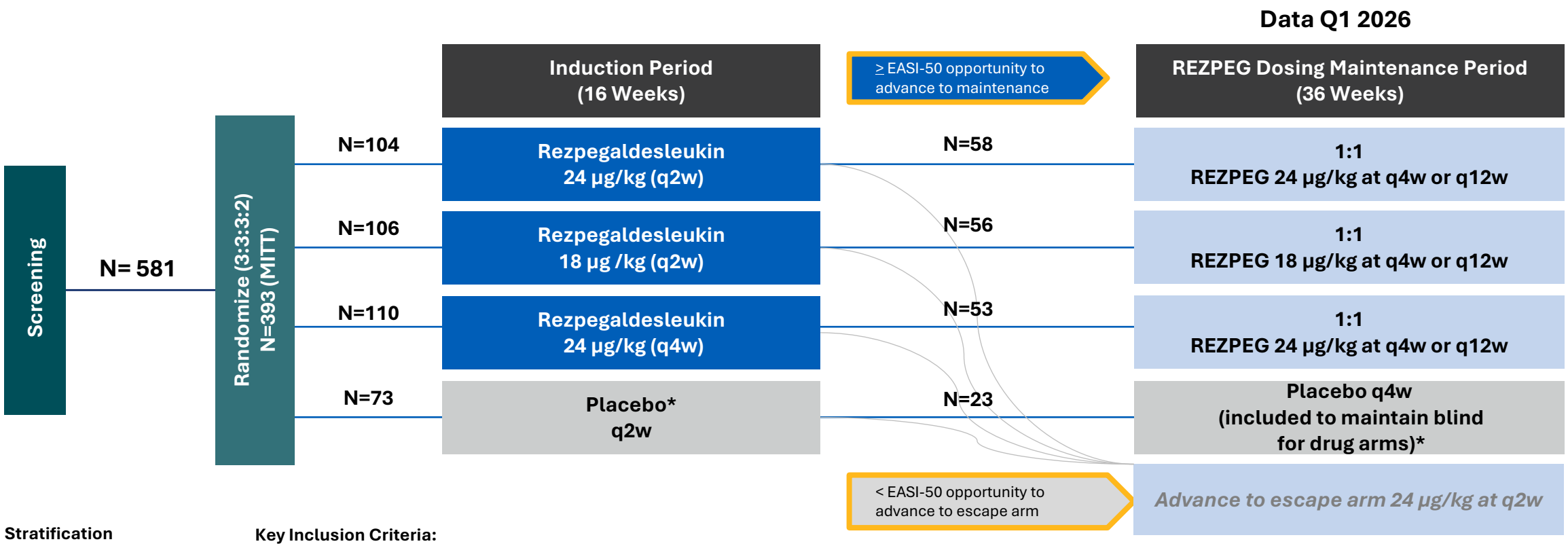
- **Offer dosing schedules without rebound effect**
- **Induce deep and potentially therapy-free remission**
- **Favorable safety and tolerability profile as compared to standard-of-care**

Dupixent: current market leader in atopic dermatitis exceeding $10.5 B in annual sales, **but 50% of patients fail on therapy**[5, 6]

Sources: 1. Eczema stats. National Eczema Association. (September 2022). https://nationaleczema.org/research/eczema-facts/; 2. Eczema council. (n.d.). https://www.eczemacouncil.org/assets/docs/global-report-on-atopic-dermatitis-2022.pdf; 3. ClarivateTM DRG Mature Markets Data 2023.; 4. DRG Epidemiology; 5. N Engl J Med 2016; 375:2335-2348 DOI: 10.1056/NEJMoa1610020; 6. EvaluatePharma, accessed January 2025

REZOLVE-AD: Phase 2b Trial Design

Patients with Moderate-to-Severe Atopic Dermatitis



Stratification

- ✓ Geographic region
- ✓ Disease severity by vIGA-AD

Key Inclusion Criteria:

- ✓ Age: ≥18 years
- ✓ Moderate/severe AD diagnosis for ≥ 12 months
  - EASI ≥ 16
  - vIGA-AD of 3 or 4
  - BSA ≥ 10%
- ✓ Biologic-naive (no prior biologic systemic therapy) and systemic JAKi-naïve
- ✓ Failure of prior therapy, including TCS of medium or higher potency, within last 6 months

MITT is defined as patients who were randomized and received at least one dose of study treatment or placebo.

*Placebo-treated clinical responders are not included in the efficacy analyses after week 16; however, they continued receiving placebo to maintain blinding (as was done in dupilumab Phase 3 SOLO-CONTINUE Study, amlitelimab Phase 2 STREAM-AD program, and tralokinumab Phase 3 ECZTRA Program)

REZOLVE-AD Phase 2b validates rezpegaldesleukin as a first-in-class novel regulatory T cell mechanism in atopic dermatitis

Novel Treg MoA differentiates from existing and in-development biologics

- **Up to 6-fold increase in Tregs**
- **Clear dose-dependent reduction in multiple AD biomarkers: IL-19, TARC/CCL17, Periostin, MDC/CCL22**

All 3 dose arms met primary endpoint

- **% improvement in EASI at 16 weeks** (p<0.001)
- **Clear dose-dependent response**
- **Rapid onset of action** (early separation from placebo)
- **Equal efficacy observed in severe patients as in moderate**

Highest dose met all six key secondaries

- **EASI-75** (p<0.001)
- **vIGA-AD 0/1** (p<0.05)
- **Itch-NRS** (p<0.01)
- **EASI-90** (p<0.05)
- **BSA** (p<0.001)

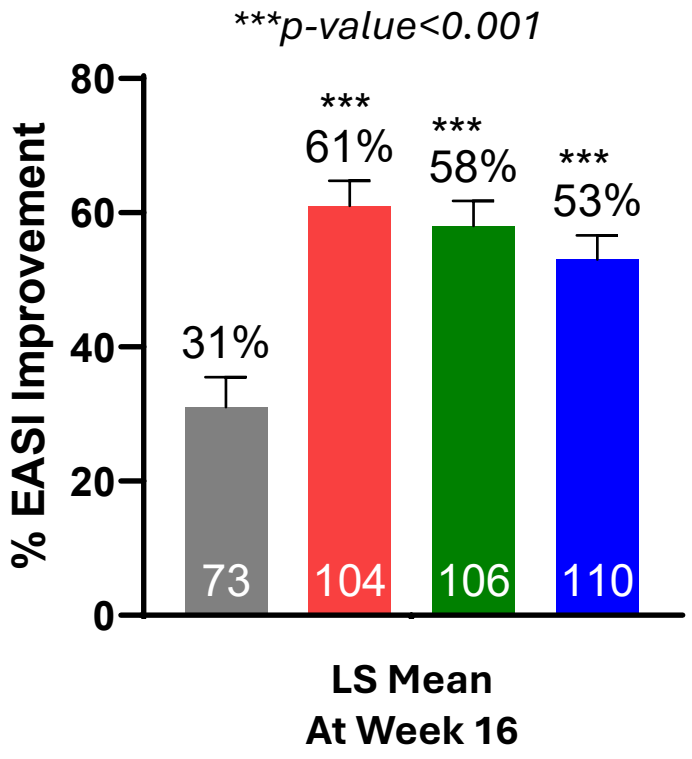
Other 2 doses also met multiple secondary endpoints

Safety consistent with previously-reported safety profile with no new safety concerns

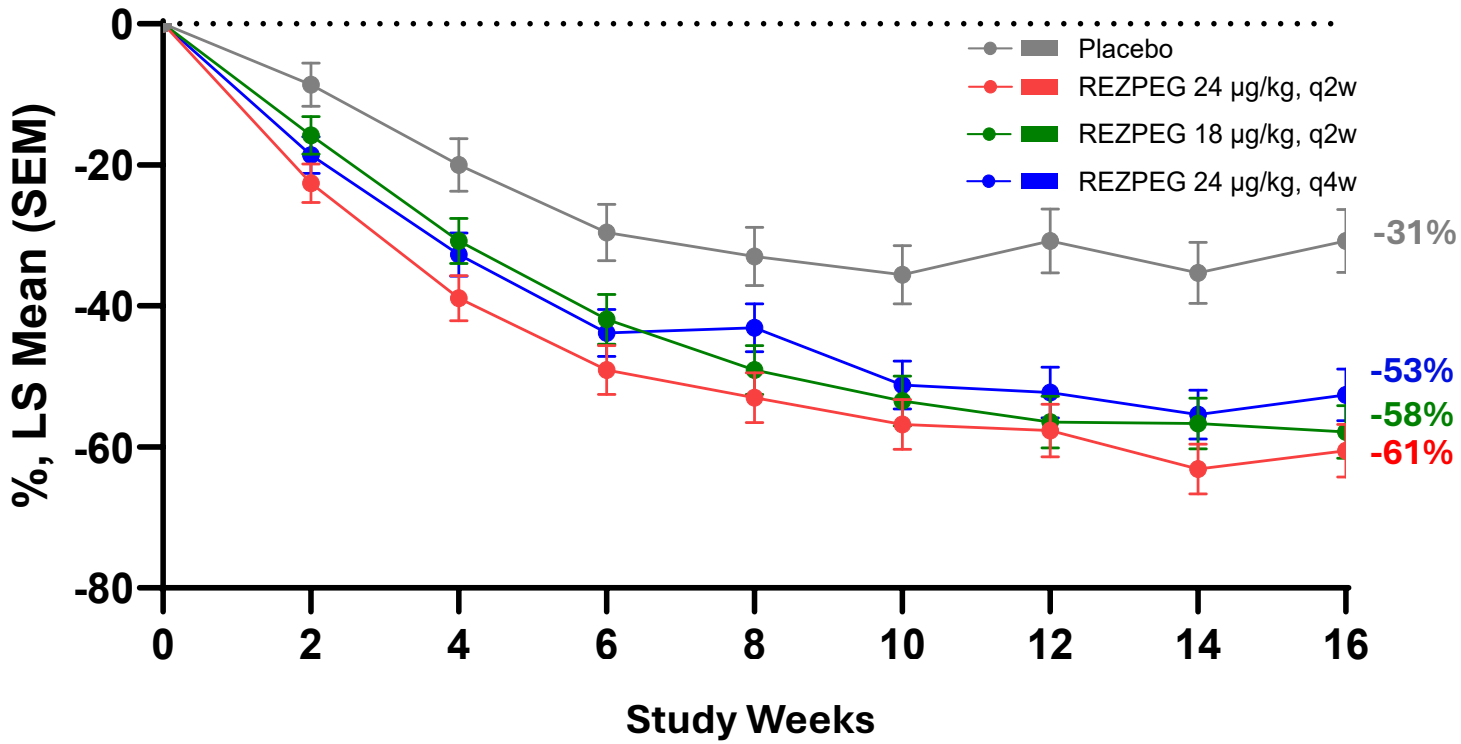
- No increased risk of conjunctivitis, oral ulcers, or infections, including oral herpes, in study treatment arms
- Most frequent AEs were mild injection site reactions (ISRs) that were self-resolving (<1% discontinuations due to ISRs)

Treatment arms showed a dose-dependent percentage reduction in EASI, with clear separation from placebo at all time points

All dose arms met primary endpoint with statistical significance p-value <0.001



% EASI Reduction from Baseline (Primary Estimand)

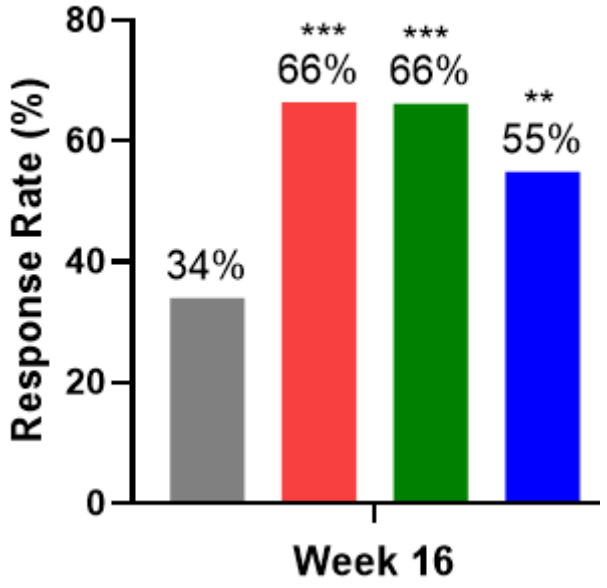


*Statistical significance: ***p-value<0.001; **p-value<0.01, *p-value<0.05; Sample size: placebo (n=73), 24 µg/kg Q2W (n=104), 18 µg/kg Q2W (n=106), and 24 µg/kg Q4W (n=110)*

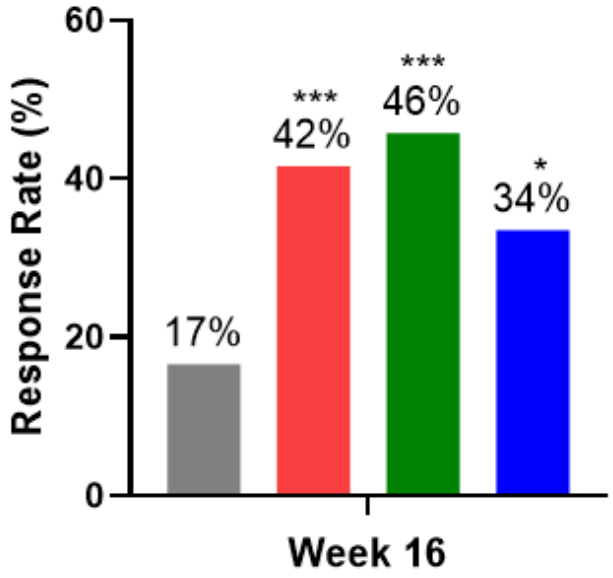
High dose treatment arm met all key secondary endpoints (*1 of 2*)

Multiple endpoints met for two additional dose arms

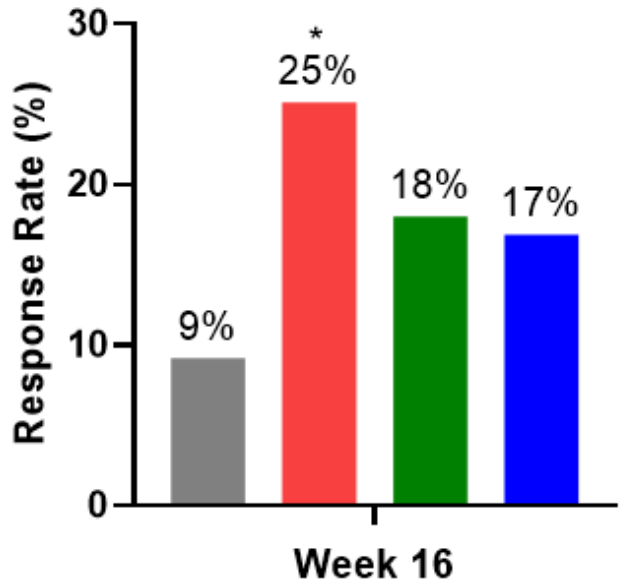
EASI-50



EASI-75



EASI-90



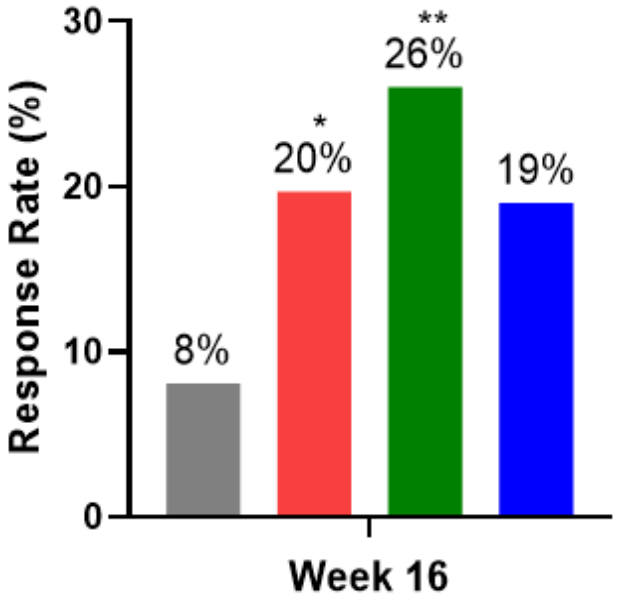
Placebo | Rezpeg 24 µg/kg Q2W | Rezpeg 18 µg/kg Q2W | Rezpeg 24 µg/kg Q4W

Data are modeled using logistic regression and primary estimand analysis. Patients that discontinue due to disease worsening or use rescue medication outside the protocol-specified period are imputed as non-responder. mITT population is used as the denominator (N=73, 104, 106, and 110 for the placebo, 24 µg/kg q2w, 18 µg/kg q2w, and 24 µg/kg q4w groups) for the EASI responder secondary endpoints.
*Statistical significance: ***p-value<0.001; **p-value<0.01, *p-value<0.05; Primary estimand analysis sample size: placebo (n=73), 24 µg/kg Q2W (n=104), 18 µg/kg Q2W (n=106), and 24 µg/kg Q4W (n=110)*

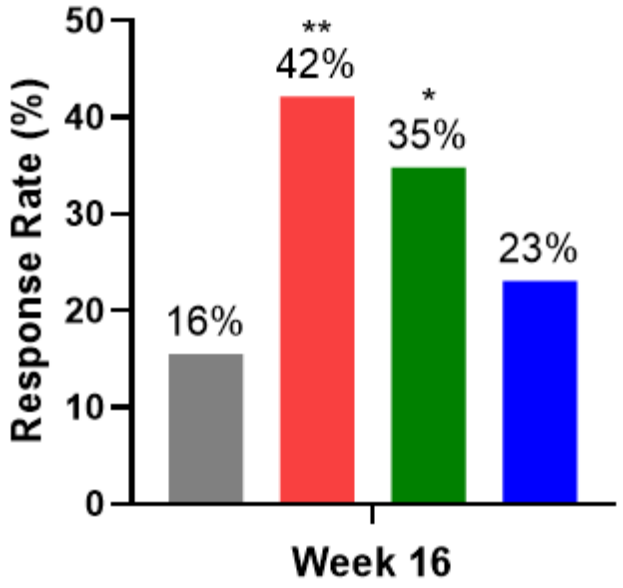
High dose treatment arm met all key secondary endpoints (*2 of 2*)

Multiple endpoints met for two additional dose arms

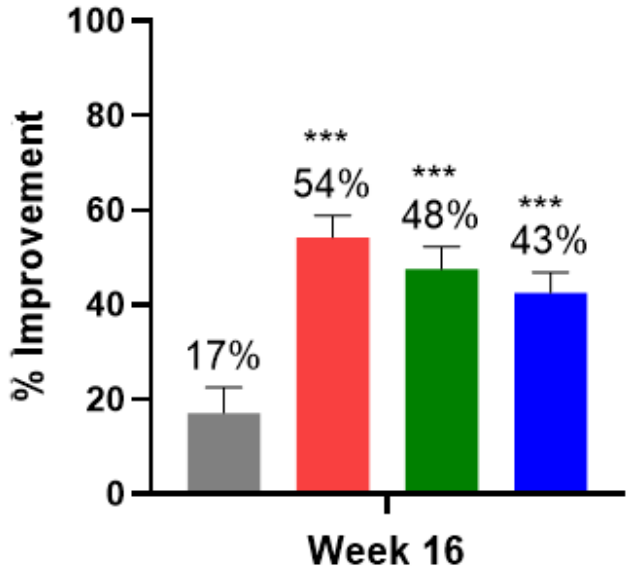
vIGA-AD 0/1



Itch NRS



BSA % Change

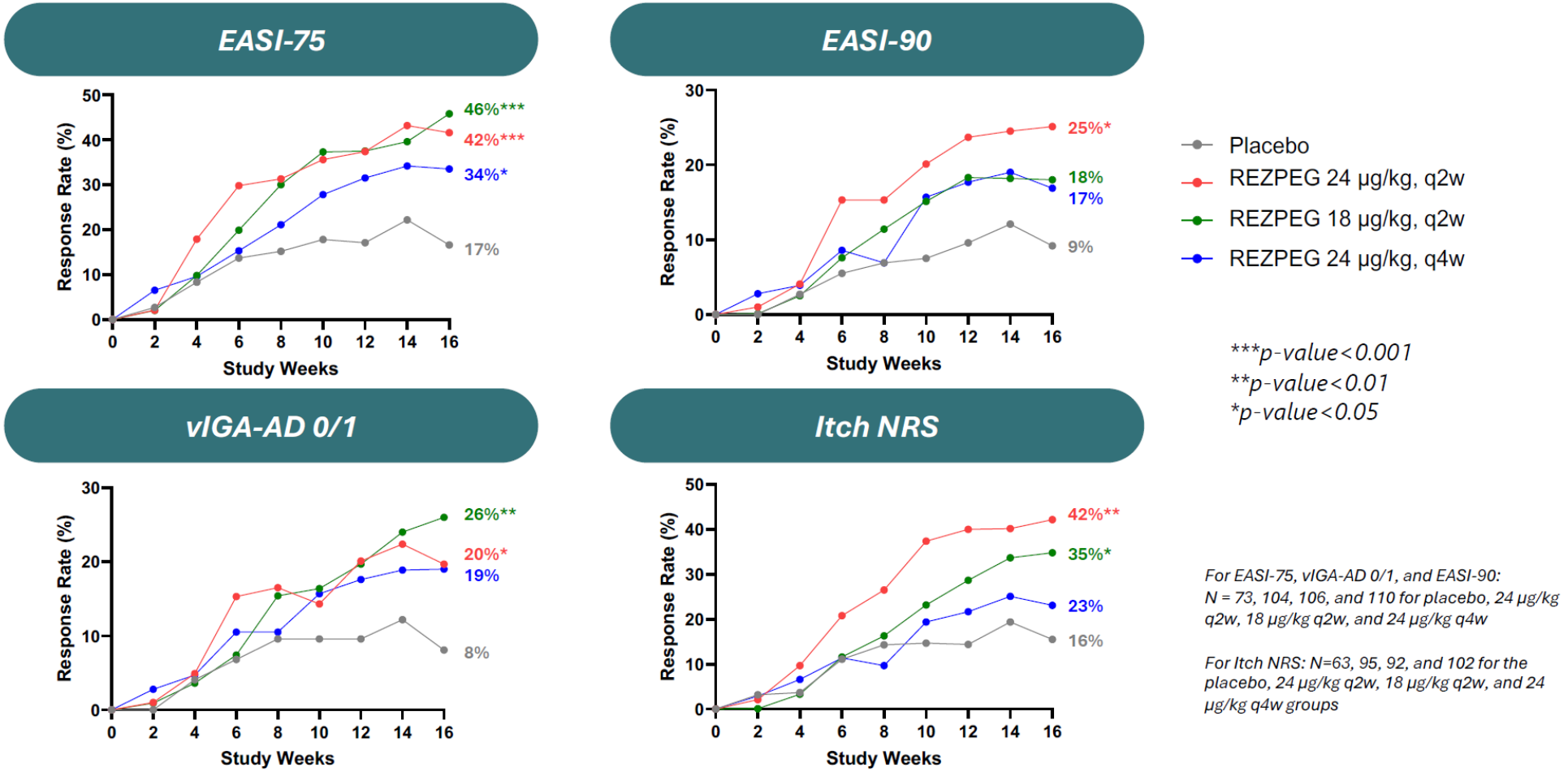


Placebo | Rezpeg 24 µg/kg Q2W | Rezpeg 18 µg/kg Q2W | Rezpeg 24 µg/kg Q4W

Data are modeled using logistic regression and primary estimand analysis for vIGA-AD 0/1 and Itch NRS, MMRM and primary estimand analysis for % BSA improvement. Patients that discontinue due to disease worsening or use rescue medication outside the protocol-specified period are imputed as non-responder. mITT population is used as the denominator (N=73, 104, 106, and 110 for the placebo, 24 µg/kg q2w, 18 µg/kg q2w, and 24 µg/kg q4w groups) for the vIGA-AD 0/1 and BSA endpoints. The mITT population with baseline itch ≥ 4 (N=63, 95, 92, and 102 for the placebo, 24 µg/kg q2w, 18 µg/kg q2w, and 24 µg/kg q4w groups) is used as the denominator for the Itch NRS endpoint
*Statistical significance: ***p-value<0.001; **p-value<0.01, *p-value<0.05; Primary estimand analysis sample size (vIGA-AD 0/1, BSA): placebo (n=73), 24 µg/kg Q2W (n=104), 18 µg/kg Q2W (n=106), and 24 µg/kg Q4W (n=110); MITT analysis sample size (itch): placebo (n=63), 24 µg/kg Q2W (n=95), 18 µg/kg Q2W (n=92), and 24 µg/kg Q4W (n=102)*

Fast Onset of Action Across All Key Secondary Endpoints

EASI-75, EASI-90, vIGA-AD 0/1, and Itch NRS (≥ 4-point Reduction) Responses Seen Early and Sustained Throughout



****p-value<0.001*
***p-value<0.01*
**p-value<0.05*

For EASI-75, vIGA-AD 0/1, and EASI-90: N = 73, 104, 106, and 110 for placebo, 24 µg/kg q2w, 18 µg/kg q2w, and 24 µg/kg q4w

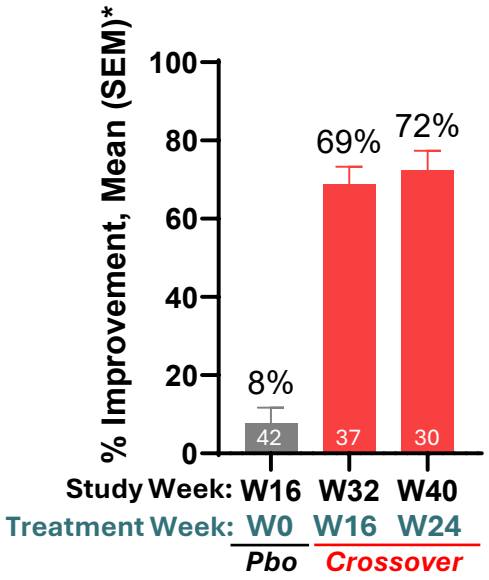
For Itch NRS: N=63, 95, 92, and 102 for the placebo, 24 µg/kg q2w, 18 µg/kg q2w, and 24 µg/kg q4w groups

The Primary Estimand analysis for binary endpoints use logistic regressions. Data after use of rescue therapy outside protocol specifications or discontinued treatment due to lack of efficacy were imputed as non-responders; data after patients who discontinued due to other reasons were set to missing and all missing data are imputed using the multiple imputation method.

Crossover from Placebo to Rezpegaldesleukin at Study Week 16

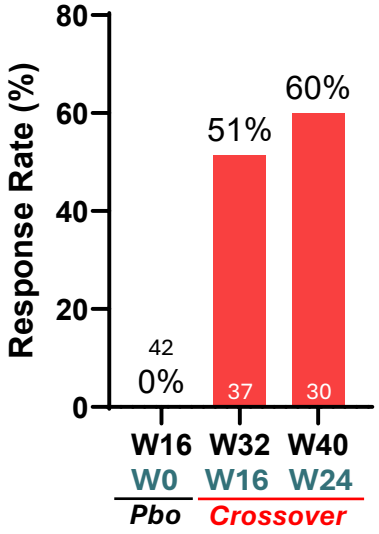
Deepening of Responses in Crossover Arm Support 24-Week Induction and Dose of 24 µg/kg q2w for Phase 3 Program

Mean EASI % Change

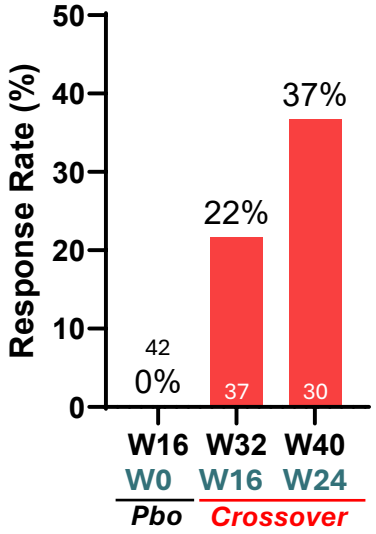


*Mean change from baseline at Week 16/24 post-induction phase of study for patients randomized to placebo and who are EASI<50 at the end of 16-week induction.

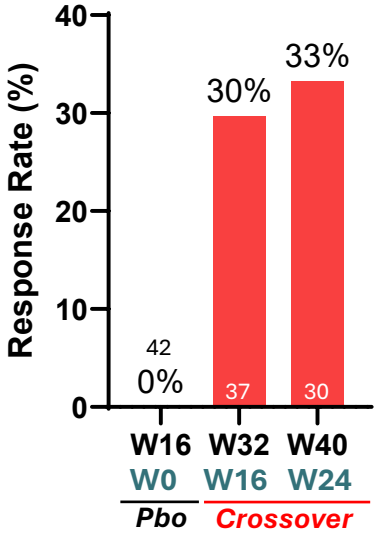
EASI-75



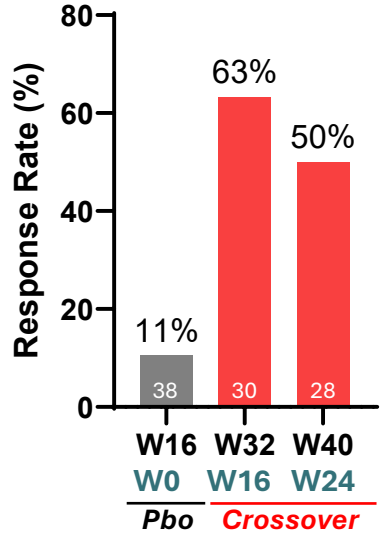
EASI-90



vIGA-AD 0/1



Itch NRS

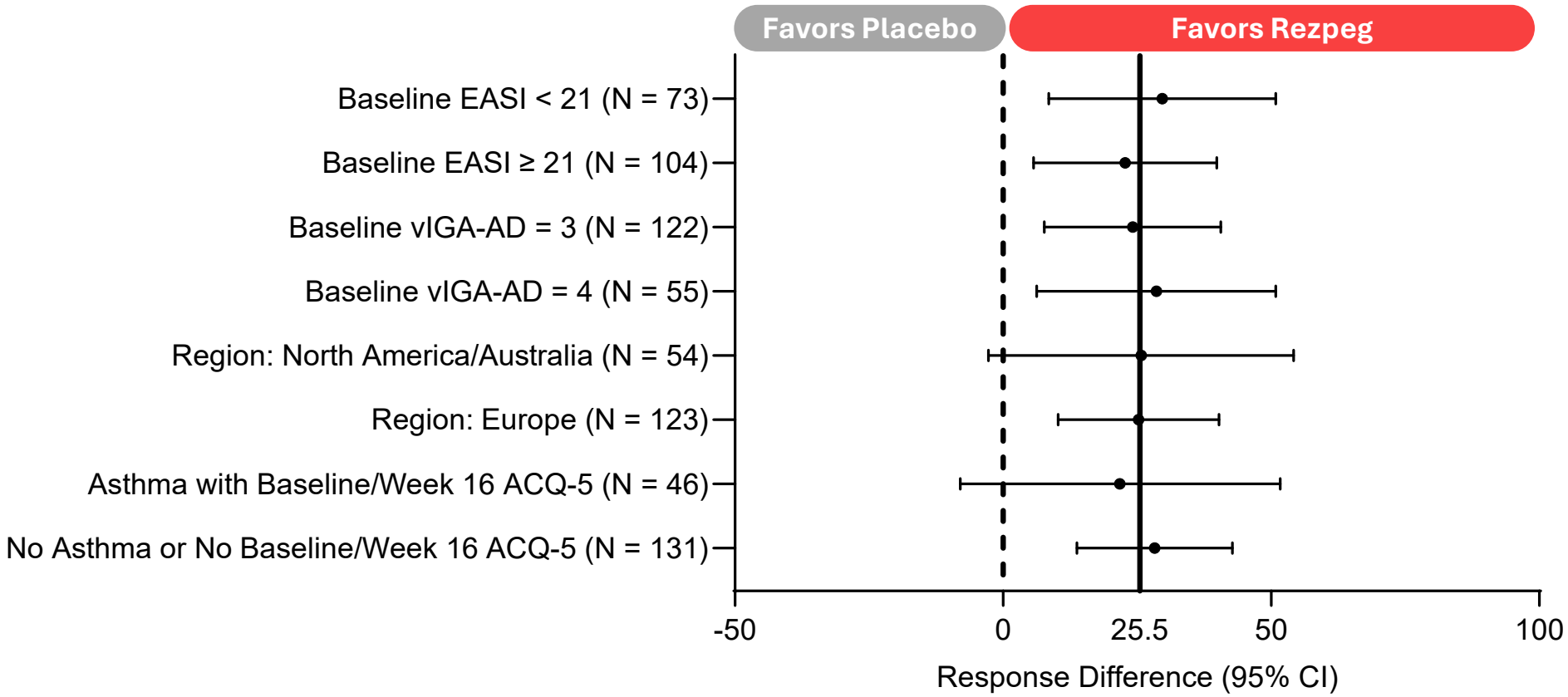


Interim analysis (15Oct2025 data cut), dosing up to study week 52 is ongoing.

As of 15Oct2025 data cut, 7 patients have discontinued up to week 24 (patient decision most common reason) and 1 patient has not yet reached week 24. Note 4 patients have missing data at week 24 but are ongoing and have data at later timepoints. The analysis of Mean EASI % Change for the crossover patients uses descriptive summary measures on observed data. The analysis of binary endpoints (EASI-75, EASI-90, vIGA-AD 0/1, and Itch NRS response) for the crossover patients uses descriptive summaries and number of patients with observed data as denominator.

EASI-75 Response for Rezpegaldesleukin 24 µg/kg q2w vs. Placebo

Consistent Treatment Effect Observed Across Baseline Severity of Disease, Geographical Region and Comorbidity of Asthma



The Primary Estimand analysis for binary endpoints use logistic regressions. Data after use of rescue therapy outside protocol specifications or discontinued treatment due to lack of efficacy were imputed as non-responders; data after patients who discontinued due to other reasons were set to missing and all missing data are imputed using the multiple imputation method.

Why we need additional therapies for Atopic Dermatitis

Current systemic treatment options fall short on safety and long-term disease control

- Majority of patients do not achieve adequate disease control by the end of the induction period[1]
- Even patients with a favorable response experience loss of disease control following cessation of therapy[2-3]
- The limited armamentarium of approved drugs with an adequate benefit–risk ratio represent major challenges in the field[4]
- New strategies aimed at inducing deep and potentially therapy-free remission are needed[5]

Currently available systemic therapies may be limited by their safety and efficacy profile:

IL-13 Biologics

- Side effects include conjunctivitis, facial erythema*, arthralgia*[6]
- No dose flexibility
- Lack of efficacy with 50% of patients failing Dupixent® therapy[1]
- Lack of long-term disease control with patients rebounding once off treatment

JAK Inhibitors

- Multiple black box warnings[7] (e.g. Serious infections, cardiovascular death, myocardial infarction, stroke, lymphoma, blood clots)
- Lab monitoring
- While treatment leads to rapid improvement in disease, once off therapy, patients rebound quickly

** Reported with dupilumab;*

Sources: 1Silverberg JI, et al. Dermatol Ther (Heidelb) (2022) 5:1181-1196; 2Gooderham et al. JAMA Derm (2019) 155(12): 137101379; 3Blauvelt et al. Am J Clin Dermatol. (2022) 23(3): 365-383; 4Bieber T. Nature Reviews Drug Discovery (2022) 21: 21–40; 5Bieber T. Nature Reviews Drug Discovery (2023) 22: 662–680; 6Torres T, et al. J Dermatolog Treat (2022) 33(5): 2554-2559; 7Mikhaylov D, et al. Ann Allergy, Asthma, Immuno (2023) 130(5) 577-592.

Study Design Comparisons for Maintenance Phase

Endpoint	Rezpegaldesleukin 18/24 µg/kg q4w/q12w 16/52W Nektar	Amlitelimab 250mg +LD/250mg/125 mg/62.5 mg q4w[1] 24/52W Sanofi	Rocatinlimab 150/600mg q4w 300/600 mg q2w[2] 16/36W Amgen**	Lebrikizumab 250mg q2w/q4w[3] Phase 3 (ADvocate 1&2) 16/52W Lilly/Dermira	Tralokinumab 300mg q2w/q4w[4] Phase 3 (ECZTRA 1&2) 16/52W Leo Pharma	Dupilumab 300mg q8w/q4w/qw/q2w[5] Phase 3 (SOLO CONTINUE) 16/52W Regeneron
Phase of trial	**2b**	**2b**	**2b**	**3**	**3**	**3**
Maintenance Duration (weeks)	**36 wks**	**28 wks**	**20 wks**	**36 wks**	**36 wks**	**36 wks**
MOA	**IL-2R agonist**	**OX40L**	**OX40**	**IL-13**	**IL-13**	**IL-4 & IL-13**
Re-randomization after Induction	Y	Y	N	Y	Y	Y
Re-randomization threshold	≥EASI-50	≥EASI-75 or IGA 0/1	NA	≥EASI-75 or IGA 0/1	≥EASI-75 or IGA 0/1	≥EASI-75 or IGA 0/1
Escape during Maintenance threshold	<EASI-25	<EASI-50	NA	<EASI-50	loss of adequate clinical response over ≥ 4-week period*	Not mentioned
Number of Maintenance dose Arms (analyzed arms)	6	4	4	2	2	3
Maintenance Dosing Frequency	Q4W, Q12W	Q4W	Q2W, Q4W	Q2W, Q4W	Q2W, Q4W	QW/Q2W, Q4W, Q8W
Placebo Induction to Placebo Maintenance to maintain the blind (non-analyzed arm)	Y	Y	N	N	Y	Y
Drug Withdraw Arm in Maintenance (analyzed arm)	N	Y	N	Y	Y	Y
Phase 3/label dose for Maintenance Phase	Q4W, Q12W	250mg Q4W+LD	300 mg Q4W	250mg Q4W	300mg Q2W	300mg Q2W

NA: Not applicable as Roca trial design allows all pts to be on Roca after W16; EOI: End Of Induction; EOM: End Of Maintenance
*For IGA=0 at W16, IGA≥2 and no EASI75; for IGA=1 at W16, IGA ≥3 and no EASI75; for IGA >1 at W16, no EASI75; **36-week parallel design

1. *Blauvelt, A et al. EADV 2025, FC08.1D; Weidinger et al. 2025, JACI 155:1264-75*
2. Guttman-Yassky et al. 2025, Dermatol Ther 15:3151-3171; Guttman-Yassky et al. Lancet 2023, 401:204-14
3. Blauvelt et al. 2023, JAMA Derm 156:411-20
4. Wollenberg et al. 2021, BJD 184:437-449
5. Worm et al. 2019, JAMA Derm 156:131-143

52 Week Benchmark Data for Atopic Dermatitis

Endpoint	Dupilumab 300mg qw/q2w[6] Phase 3 (SOLO CONTINUE) (IGA0/1 or EASI-75) 16/52W Regeneron	Lebrikizumab 250mg q4w[4] Phase 3 (ADvocate 1&2) (IGA0/1 or EASI-75) 16/52W Eli Lilly	Tralokinumab 300mg q2w/q4w Phase 3 (ECZTRA 1&2) 16/52W Lilly/Dermira	Amlitelimab 250mg q4w+LD[1] Phase 2b (IGA0/1 or EASI-75) 24/52W Sanofi
MOA	**IL-4 & IL-13**	**IL-13**	**IL-13**	**OX40**
Enrollment Completion, Total Sample Size	**2016, N=422**	**2021, N= 291**	**2018, N=412**	**2022, N=174**
Sample Size in Each Group (in maintenance)	**N=169, QW/Q2W**	**N=118, Q4W**	**N=159, Q2W**	**N=13, Q4W+LD**
% change in EASI during maintenance: EOI to Wk52	-92% to -91%	-89% to -84%	Not Reported	Not Reported
% EASI-75 who maintained EASI-75 at Week 52 from end of induction (n=number of week 16 responders)	72% (n=162)	82% (n=115)	57% (n=124)	67% (n=12)
% IGA 0/1 who maintained IGA 0/1 at Week 52 from end of induction (n=number of week 16 responders)	54% (n=126)	77% (n=77)	56% (n=93)	55% (n=11)
% Itch NRS with ≥4-point improvement at W52 amongst pts with Itch NRS ≥4-point improvement at Maintenance Baseline	Not Reported	85% (n=65)	Not Reported	Not Reported

Worm et al. 2019, JAMA Derm 156:131-143; 4. Blauvelt et al. 2023, JAMA Derm 156:411-20; Blauvelt, A et al. EADV 2025, FC08.1D; Weidinger et al. 2025, JACI 155:1264-75

Phase 2b AD Benchmark Data for the Induction Period (No 52-Week Maintenance Conducted Except for Amlitelimab)

Endpoint	Rezpegaldesleukin 18/24 µg/kg Q2W Phase 2b 16 Weeks (NEKTAR THERAPEUTICS)	Rezpegaldesleukin 24 µg/kg Q2W Ph2b Crossover 24 Weeks 15Oct2025 CCOD (NEKTAR THERAPEUTICS)	Amlitelimab 250mg Q4W[1] Phase 2b 16/24 Weeks (sanofi)	Rocatinlimab 150/600mg Q4W[2] Phase 2b 16 Weeks (AMGEN)	Nemolizumab 30mg Q4W[3] Phase 2b 24 Weeks (TCS Combo) (GALDERMA EST. 1981)	Lebrikizumab 250mg Q2W[4] Phase 2b 16 Weeks (Lilly)	Tralokinumab 300mg Q2W[5] Phase 2b 12 Weeks (TCS Combo) (LEO)	Dupilumab 300mg Q2W[6] Phase 2b 16 Weeks (REGENERON sanofi)
MoA	IL-2R agonist	IL-2R agonist	OX40L	OX40	IL-31	IL-13	IL-13	IL-4 & IL-13
Enrollment Completion Trial Size	2025 N=393	2025 N=30	2022 N=390	2020 N=274	2018 N=226	2019 N=280	2016 N=204	2014 N=380
EASI LS Mean % reduction from baseline (Placebo)	58/61% (31%)	72% (8%**)	62% (29%)	62/60% (32%)	69% (52%)	72% (41%)	Not Reported	68% (18%)
Placebo Adjusted	27/30%	64%	32%	30/28%	17%	31%	Not Reported	50%
EASI-75 (Placebo)	46/42% (17%)	60% (0%**)	40% (11%)	44/40% (11%)	46% (26%)	48% (12%)[b]	43% (16%)	52% (11%)[d]
Placebo Adjusted	29/25%	60%	29%	33/29%	20%	36%	27%	41%
vIGA-AD Responders (0/1) (Placebo)	26/20% (8%)	33% (0%**) *vIGA-AD*	22% (5%) *IGA-AD*	19/15% (2%)	37% (21%) *IGA-AD*	45% (15%) *IGA-AD*	27% (12%) *IGA-AD*	30% (2%) *IGA-AD*
Placebo Adjusted	18/12%	33%	17%	17/13%%	16%	30%	15%	28%
EASI-90 (Placebo)	18/25% (9%)	37% (0%**)	16% (4%)	19/12% (4%)	30% (11%)	44% (11%)	Not Reported	30% (4%)[d]
Placebo Adjusted	9/16%	37%	12%	15/8%	19%	33%	Not Reported	26%
Itch NRS ≥ 4 pt Responders (Placebo)	35/42% (16%)	50% (11%**) N=28	25% (5%)	37/46% (19%)	43% (24%)[a]	67% (39%)[c]	Not Reported	41% (8%)[e]
Placebo Adjusted	19/26%	39%	20%	18/27%	19%	28%	Not Reported	33%

Sources: 1. Weidinger et al. 2025, JACI 155:1264-75; 2. Guttman-Yassky et al. 2023, Lancet 401:204-14; 3. Silverberg et al. 2020, JACI 145:173-82; 4. Guttman-Yassky et al. 2020, JAMA Derm 156:411-20; 5. Wollenberg et al. 2019, JACI 143:135-41; 6. Thaci et al. 2016, Lancet 387:40-52
Notes: a. Estimated from Fig 4b; b. Sensitivity analysis 3, NRI for rescue meds & LOCF for other missing data (eTable 4); c. MCMC imputation (eFig 3b); d. estimated from Fig 3; e. 3pt responder scale
*** End of 16 week induction for the N=42 crossover population is the placebo result for this population*

Asthma in Atopic Dermatitis

Asthma commonly co-occurs with Atopic Dermatitis, reflecting substantial overlap in type 2 inflammatory disease

Patients with Atopic Dermatitis, particularly those with impaired skin-barrier function and a genetic predisposition toward IgE-mediated sensitization, are at increased risk of progressing to asthma as part of the "atopic march"

US Prevalence

~21 M ***Diagnosed AtD pts.***

~34% moderate-severe

~7.1 M ***Mod.-severe patients***

~26% asthmatic

~1.9 M ***Mod.-severe AtD / asthma patients***

Treatment Approach & Biologic Use

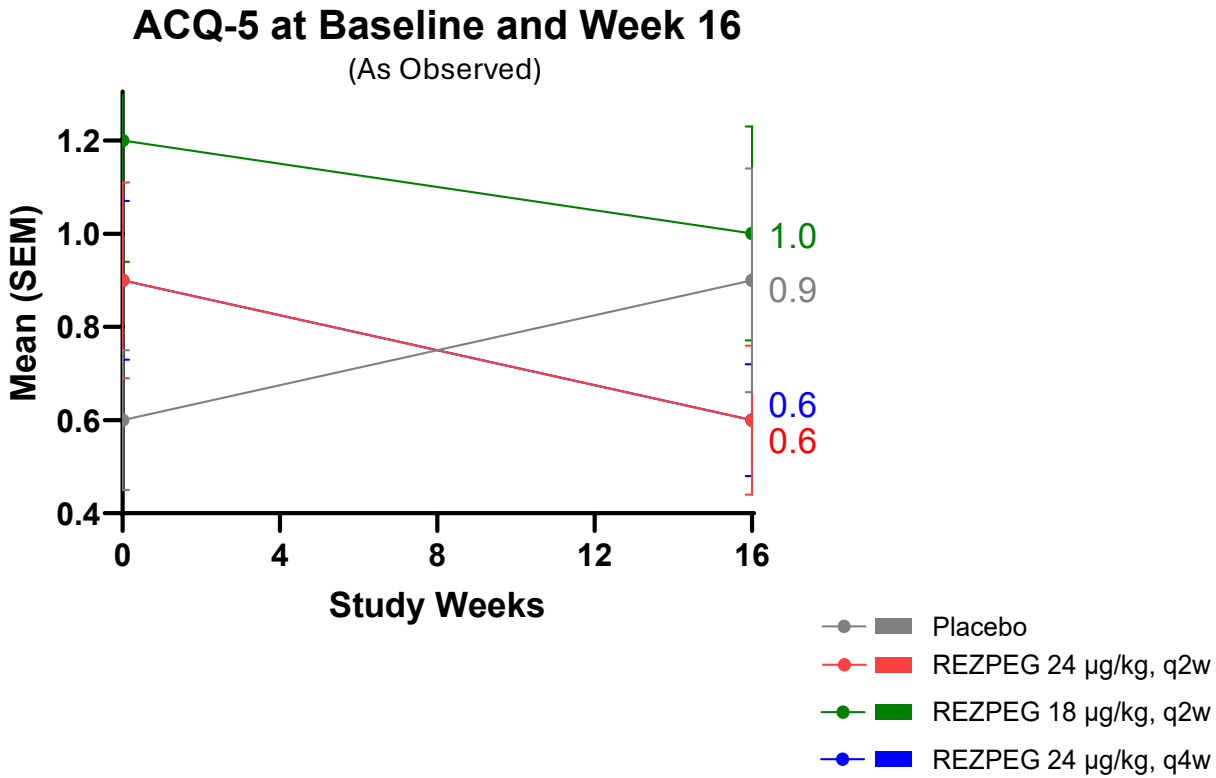
Dupilumab is the only biologic approved for both AtD and asthma; AtD guidelines highlight its benefits for patients with comorbid conditions such as asthma, and multiple peer-reviewed studies support its use as a first-line option in these patients

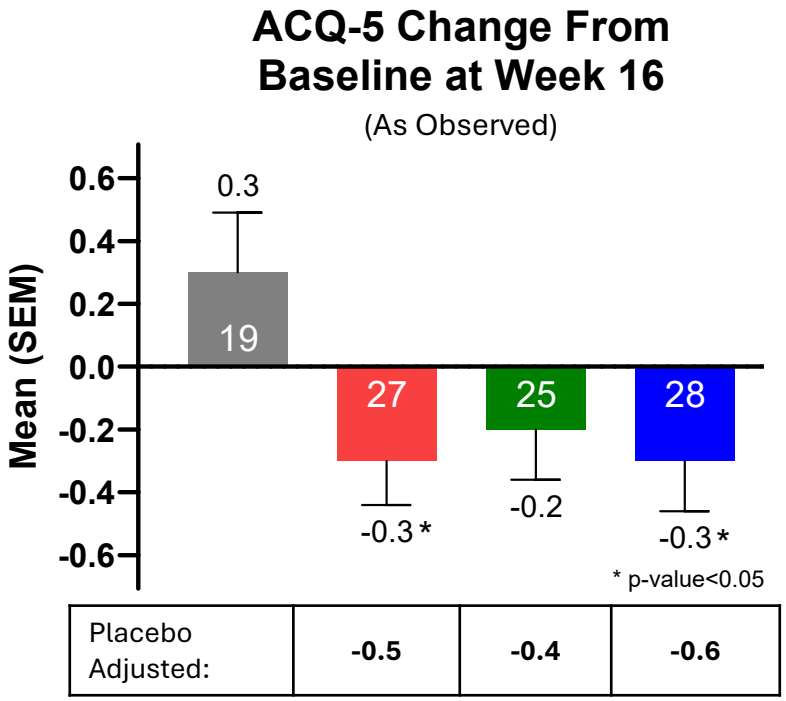
Indication	Treatment Landscape
Atopic Dermatitis	Approved biologics: **dupilumab**, tralokinumab, lebrikizumab, nemolizumab
	Guidelines recommend biologics for mod-severe patients who are refractory, intolerant, or unable to use topical treatments
Asthma	Approved biologics: **dupilumab**, omalizumab, tezepelumab, mepolizumab, benralizumab, reslizumab
	Biologics are recommended for mod-severe uncontrolled patients; several, including dupilumab, are indicated specifically for patients characterized by an eosinophilic phenotype

Sources: 1. Clarivate DRG Epidemiology (Diagnosed Population) 2. J Am Acad Dermatol 2025 DOI: 10.1016/j.jaad.2025.10.022 3. I Inv Dermatol 2019 DOI: 10.1016/j.jid.2018.08.028 4. Dermatitis 2014 DOI: 10.1097/DER.0000000000000034 5. J Am Acad Dermatol 2021; 84:471-478 DOI: 10.1016/j.jaad.2020.02.055 6. Ann Allergy Asthma Immunol 2023 DOI: 10.1016/j.anai.2023.11.009 7. Prim Care Respir Med 2023 DOI: 10.1038/s41533-023-00330-1 8. J Clin Med 2025 DOI: 10.3390/jcm14134749 9. UpToDate (Nov 2025)

ACQ-5 Response in Patients with Self-Reported Asthma History

Improvement in ACQ-5 Scores From Baseline to Week 16 in Rezpegaldesleukin Treated Patients with Asthma Comorbidity





Placebo Adjusted:	-0.5	-0.4	-0.6

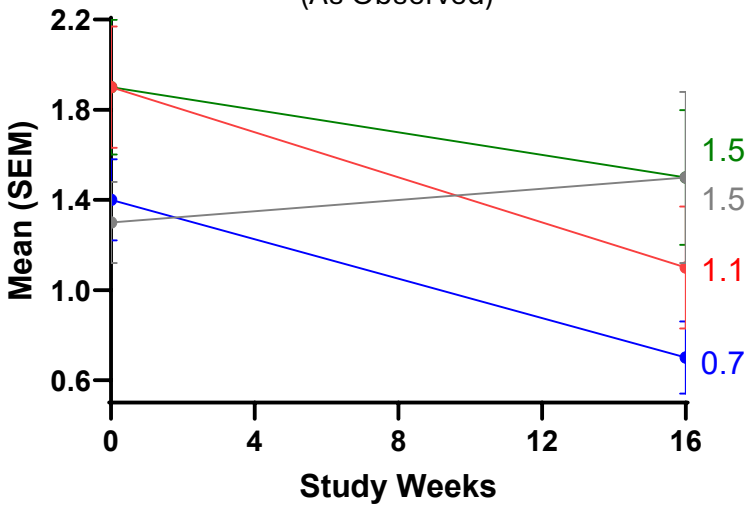
Only patients with both Baseline and Week 16 ACQ-5 data are included. The analysis for ACQ-5 data uses descriptive summary measures on observed data. P-value for change from baseline is from two-sample t-test.

Patients with Self-Reported Asthma History and Baseline ACQ-5 ≥0.5

Improvement in ACQ-5 From Baseline to Week 16 in Rezpegaldesleukin Treated Patients with Baseline ACQ-5 Score ≥ 0.5

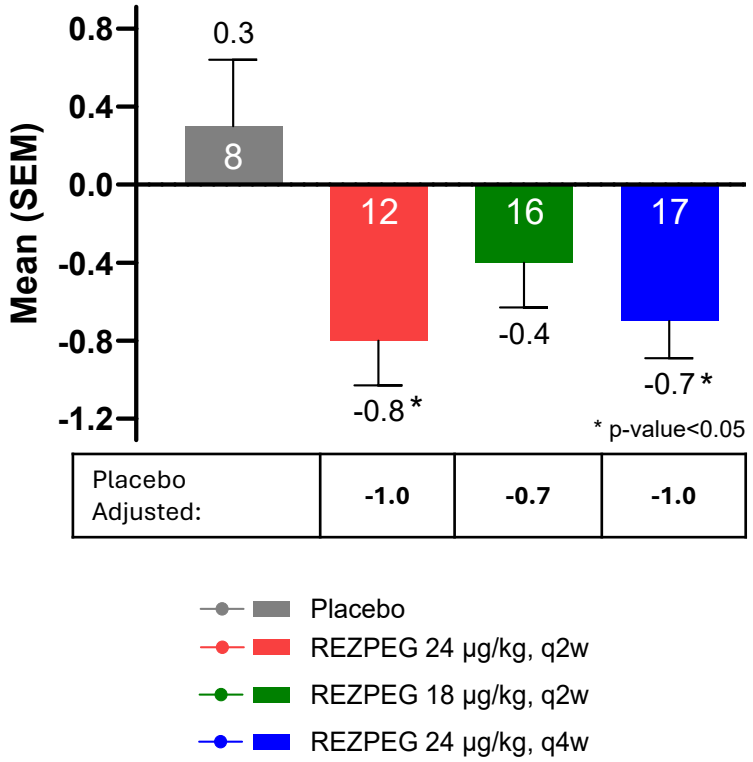
ACQ-5 at Baseline and Week 16

(As Observed)



ACQ-5 Change From Baseline at Week 16

(As Observed)

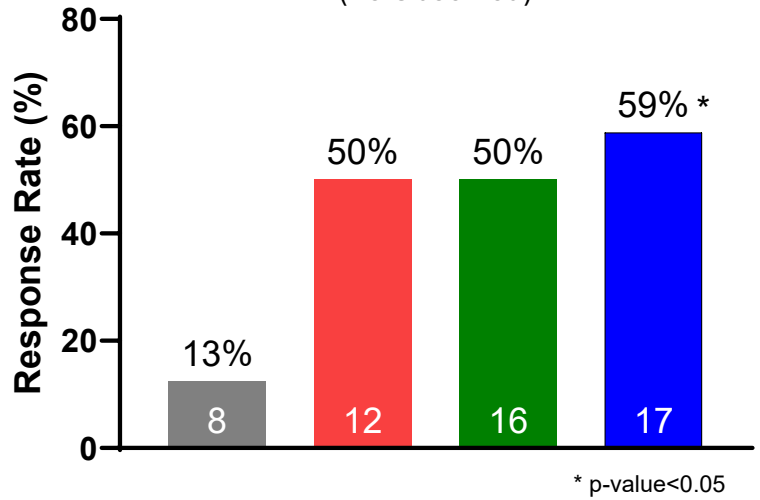


Placebo Adjusted:	-1.0	-0.7	-1.0

Placebo
REZPEG 24 µg/kg, q2w
REZPEG 18 µg/kg, q2w
REZPEG 24 µg/kg, q4w

Proportion of Patients with ACQ-5 Improvement of ≥ 0.5 at Week 16

(As Observed)



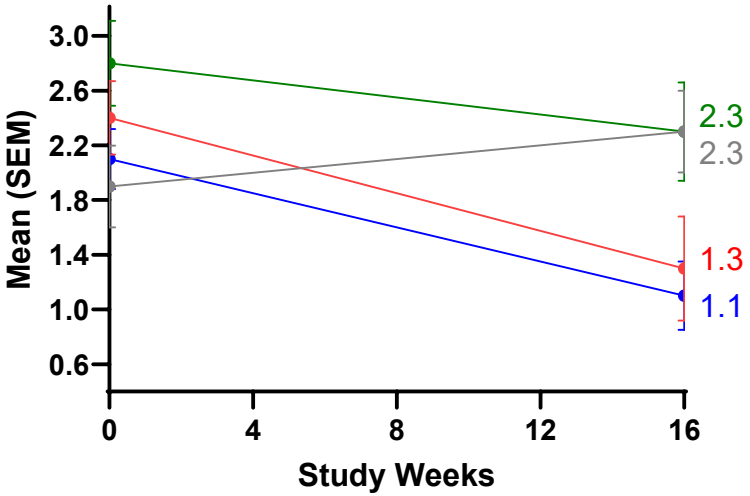
Only patients with both Baseline and Week 16 ACQ-5 data and baseline ACQ-5 ≥ 0.5 are included. The analysis for ACQ-5 data uses descriptive summary measures on observed data. P-value for change from baseline is from two-sample t-test and p-value for response is from Cochran-Mantel-Haenszel test.

ACQ-5 Response in Patients with Self-Reported *Uncontrolled* Asthma at Baseline

Improvement in ACQ-5 From Baseline to Week 16 in Rezpegaldesleukin Treated Patients with Baseline ACQ-5 Score ≥ 1.5

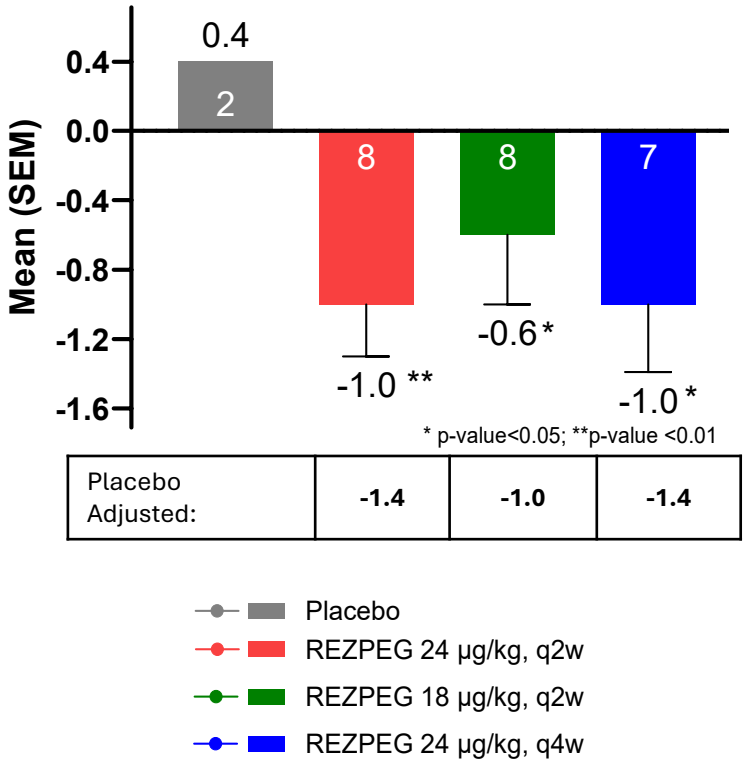
ACQ-5 at Baseline and Week 16

(As Observed)



ACQ-5 Change From Baseline at Week 16

(As Observed)

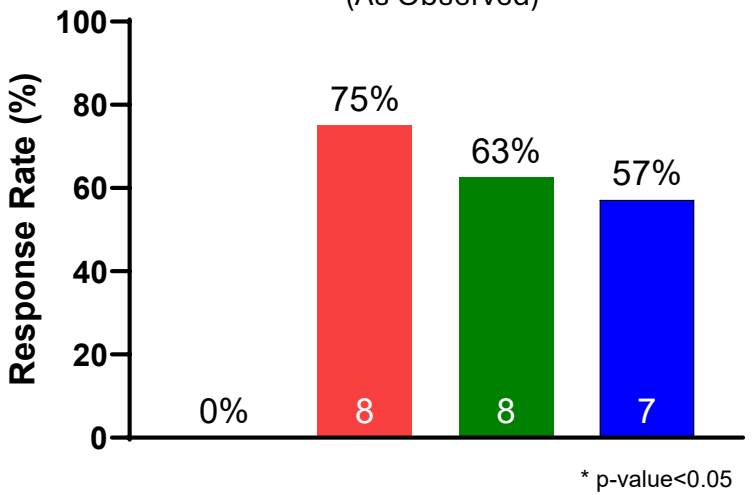


Placebo Adjusted:	-1.4	-1.0	-1.4

Placebo
REZPEG 24 µg/kg, q2w
REZPEG 18 µg/kg, q2w
REZPEG 24 µg/kg, q4w

Proportion of Patients with ACQ-5 Improvement of ≥ 0.5 at Week 16

(As Observed)



Only patients with both Baseline and Week 16 ACQ-5 data and baseline ACQ-5 ≥ 1.5 are included. The analysis for ACQ-5 data uses descriptive summary measures on observed data. P-value for change from baseline is from two-sample t-test and p-value for response is from Cochran-Mantel-Haenszel test.

REZOLVE-AD Phase 2b validates rezpegaldesleukin as a first-in-class novel regulatory T cell mechanism in atopic dermatitis

Novel Treg MoA differentiates from existing and in-development biologics

- **Up to 6-fold increase in Tregs**
- **Clear dose-dependent reduction in multiple AD biomarkers: IL-19, TARC/CCL17, Periostin, MDC/CCL22**

All 3 dose arms met primary endpoint

- **% improvement in EASI at 16 weeks** (p<0.001)
- **Clear dose-dependent response**
- **Rapid onset of action** (early separation from placebo)
- **Equal efficacy observed in severe patients as in moderate**

Highest dose met all six key secondaries

- **EASI-75** (p<0.001)
- **vIGA-AD 0/1** (p<0.05)
- **Itch-NRS** (p<0.01)
- **EASI-90** (p<0.05)
- **BSA** (p<0.001)

Other 2 doses also met multiple secondary endpoints

Safety consistent with previously-reported safety profile with no new safety concerns

- No increased risk of conjunctivitis, oral ulcers, or infections, including oral herpes, in study treatment arms
- Most frequent AEs were mild injection site reactions (ISRs) that were self-resolving (<1% discontinuations due to ISRs)

Overall Summary of Treatment Emergent Adverse Events

16-Week Induction Period

	Placebo q2w N = 73	Rezpeg 24 µg/kg q2w N = 104	Rezpeg 18 µg/kg q2w N = 106	Rezpeg, 24 µg/kg q4w N = 110	Rezpeg Total N = 320
Patients With at Least One TEAE	42 (57.5%)	89 (85.6%)	78 (73.6%)	90 (81.8%)	257 (80.3%)
Patients With at Least One TEAE (Excluding ISRs)	42 (57.5%)	69 (66.3%)	60 (56.6%)	64 (58.2%)	193 (60.3%)
Patients With at Least One Serious TEAE	0	1 (1.0%)	4 (3.8%)	0	5 (1.6%)
Patients With at Least One Severe TEAE	1 (1.4%)	3 (2.9%)	6 (5.7%)	1 (0.9%)	10 (3.1%)
Patients With at Least One TEAE Leading to Death*	0	0	0	0	0
TEAEs by System Organ Class and Preferred Term Over ≥ 5% in Any Arm					
General disorders and administration site conditions	7 (9.6%)	80 (76.9%)	67 (63.2%)	78 (70.9%)	225 (70.3%)
Proportion of patients with at least one Injection Site Reaction (ISR)	3 (4.1%)	79 (76.0%)	66 (62.3%)	78 (70.9%)	223 (69.7%)
Proportion of patients with ≤2 ISRs	73 (100.0%)	54 (51.9%)	68 (64.2%)	68 (61.8%)	190 (59.4%)
Injection site reaction (ISR) by Number of Events					
Proportion of ISR events-mild (%)	100%	65.5%	70.7%	69.9%	68.3%
Proportion of ISR events-moderate (%)	0%	33.9%	28.9%	30.1%	31.3%
Proportion of ISR events-severe (%)	0%	0.6%	0.4%	0%	0.4%
Pyrexia	2 (2.7%)	11 (10.6%)	5 (4.7%)	4 (3.6%)	20 (6.3%)
Infections and infestations	25 (34.2%)	29 (27.9%)	39 (36.8%)	32 (29.1%)	100 (31.3%)
Nasopharyngitis	10 (13.7%)	10 (9.6%)	14 (13.2%)	14 (12.7%)	38 (11.9%)
Upper respiratory tract infection	4 (5.5%)	7 (6.7%)	8 (7.5%)	4 (3.6%)	19 (5.9%)
Blood and lymphatic system disorders	3 (4.1%)	29 (27.9%)	6 (5.7%)	11 (10.0%)	46 (14.4%)
Eosinophilia**	2 (2.7%)	17 (16.3%)	4 (3.8%)	4 (3.6%)	25 (7.8%)
Lymphadenopathy	0	7 (6.7%)	1 (0.9%)	3 (2.7%)	11 (3.4%)
Musculoskeletal and connective tissue disorders	3 (4.1%)	19 (18.3%)	5 (4.7%)	11 (10.0%)	35 (10.9%)
Arthralgia	1 (1.4%)	10 (9.6%)	2 (1.9%)	4 (3.6%)	16 (5.0%)
Skin and subcutaneous tissue disorders	8 (11.0%)	12 (11.5%)	10 (9.4%)	13 (11.8%)	35 (10.9%)
Worsening atopic dermatitis	7 (9.6%)	2 (1.9%)	5 (4.7%)	6 (5.5%)	13 (4.1%)
Nervous system disorders	6 (8.2%)	10 (9.6%)	10 (9.4%)	9 (8.2%)	29 (9.1%)
Headache	3 (4.1%)	8 (7.7%)	6 (5.7%)	6 (5.5%)	20 (6.3%)
Gastrointestinal disorders	3 (4.1%)	8 (7.7%)	7 (6.6%)	11 (10.0%)	26 (8.1%)
Respiratory, thoracic and mediastinal disorders	1 (1.4%)	6 (5.8%)	5 (4.7%)	5 (4.5%)	16 (5.0%)
Investigations	1 (1.4%)	6 (5.8%)	4 (3.8%)	3 (2.7%)	13 (4.1%)

No increased risk of conjunctivitis, oral ulcers, asthma, infections or MACE

*Following 16-week induction, one death in a 38 y/o female occurred in the escape arm due to coronary thrombosis/heart failure. Patient had multiple, overlapping pre-existing cardiovascular risk factors. The death was assessed as unrelated to study treatment by the Sponsor Drug Safety Committee and independent external experts; **Eosinophilia was reported by the investigator based on the laboratory value being above the upper limit of normal. Only one patient discontinued in the study (at the mid-dose of 18 ug/kg q2w) due to increased eosinophil count.

Rezpegaldesleukin program: Next steps

End of Phase 2 Meeting with FDA to review Phase 3 development plan

↓

Full presentation of data to be submitted for presentation at a medical meeting in 2025

↓

Topline results from Phase 2b REZOLVE-AA (alopecia areata) in December 2025

↓

52-week maintenance data from Phase 2b REZOLVE-AD (atopic dermatitis) in early 2026

↓

52-week off-study treatment durability data from Phase 2b REZOLVE-AD in early 2027

Phase 2b REZOLVE-AA Study Evaluating REZPEG for Alopecia Areata

*Severe-to-very-severe alopecia areata (*NCT06340360) - Granted Fast Track Designation in July 2025

Key Eligibility Criteria	Induction Q2W (Weeks 0-36)	Week 36 Endpoints	Extension and Follow-up

- Adult patients (18+)
- Severe-to-very-severe AA
- SALT ≥50
- >6 months since diagnosis to exclude unstable AA
- No spontaneous improvement over the past 6 months
- 8-week washout of prior medication for AA

Stratification by baseline SALT score ≥50 to <95 vs ≥95 to 100

3:3:2 N=92

- **REZPEG 24 µg/kg n=35**
- **REZPEG 18 µg/kg n=37**
- **Placebo n=20**

Primary:
- Mean % SALT reduction at Week 36

Key Secondaries:
- SALT ≥ 30% reduction from baseline ($SALT_{30}$)
- Absolute SALT ≤ 30
- Absolute SALT ≤ 20
- Absolute SALT ≤ 10
- Clinician-Reported Outcome (ClinRO) Eyebrow and Eyelash (exploratory)
- Safety

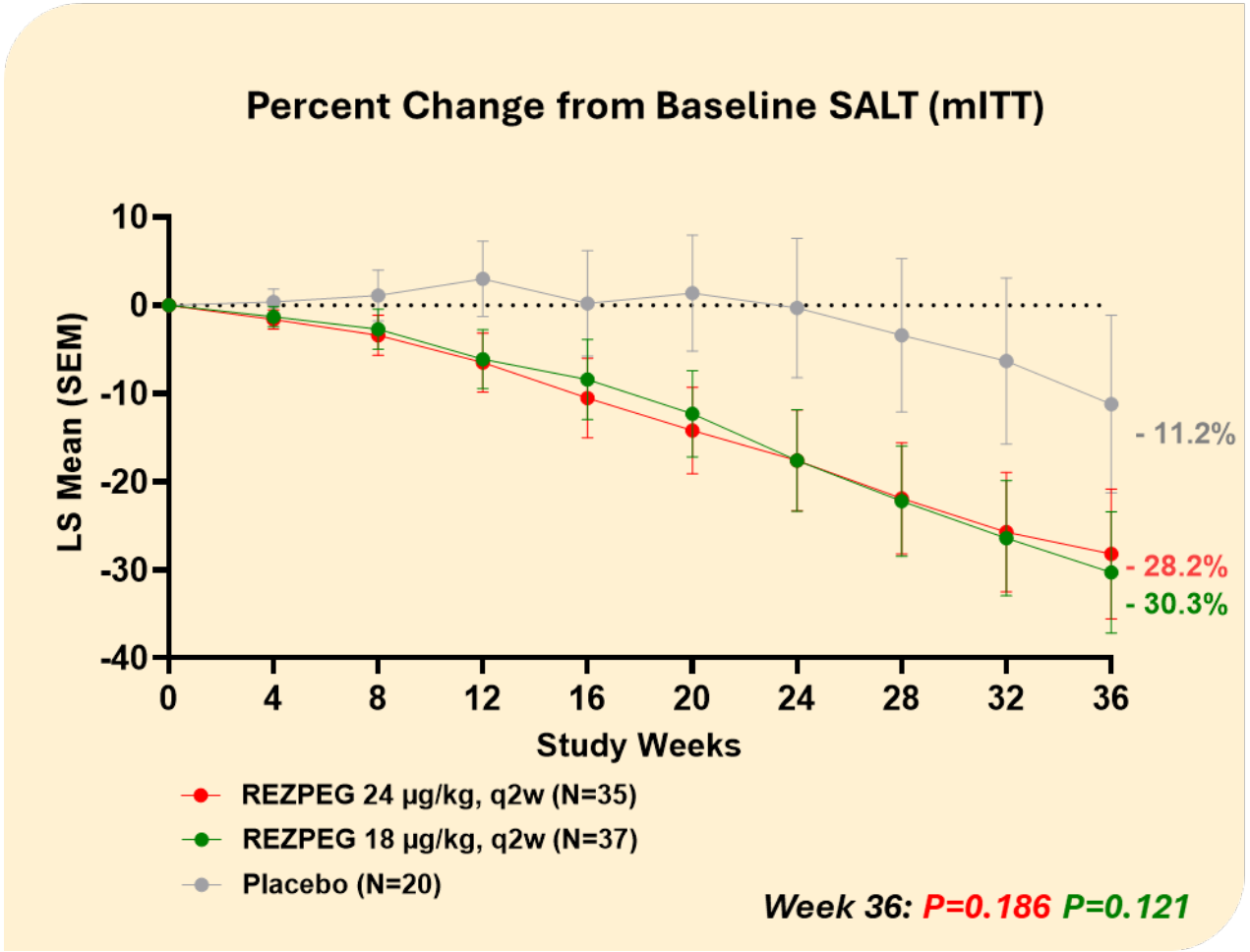
Blinded 16-week treatment extension for patients who demonstrated hair growth

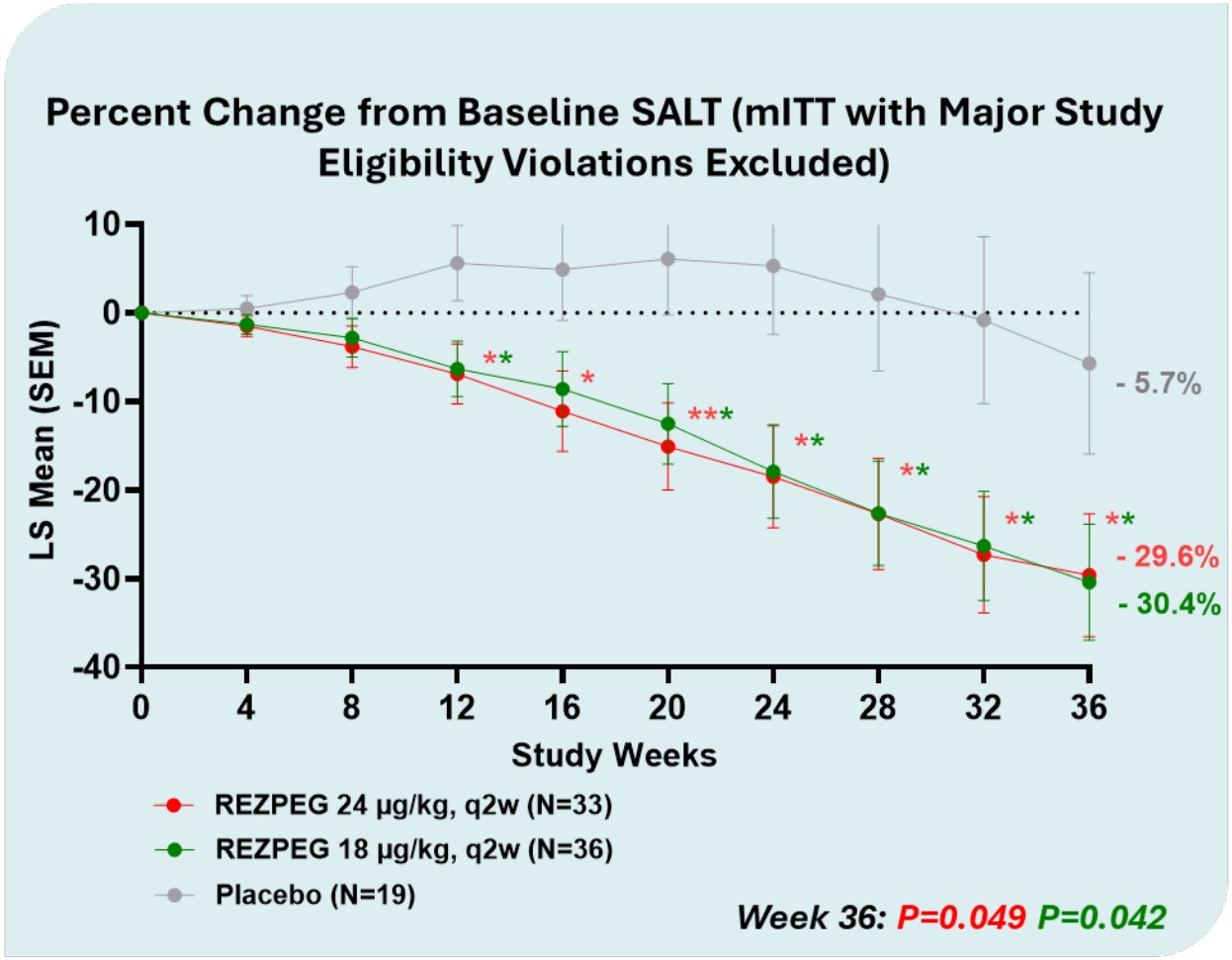
24-week follow-up period after treatment

Rezolve AA

Severity of Alopecia Tool (SALT) is a validated endpoint to assess the extent of scalp-hair loss in patients with alopecia areata

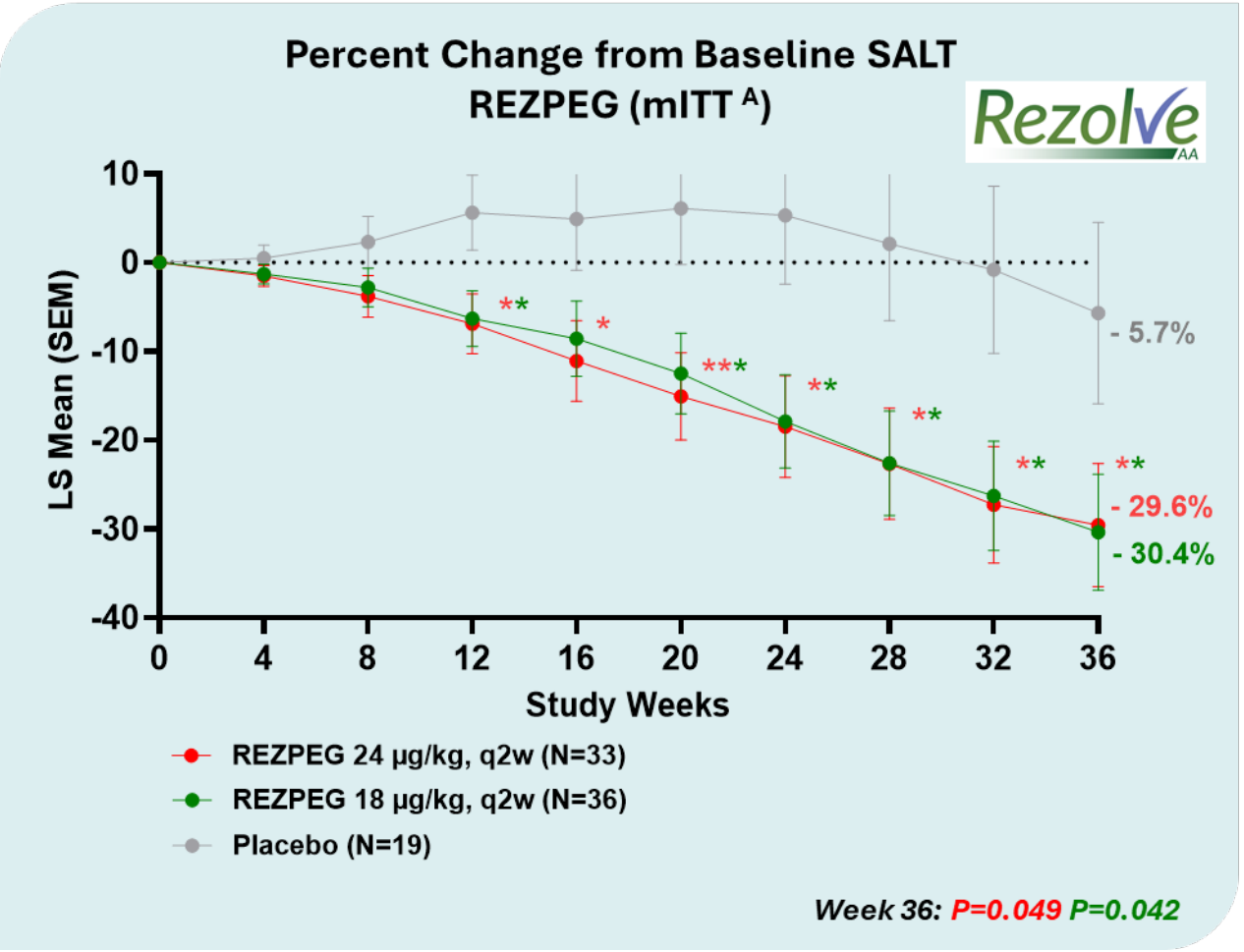
Both Treatment Arms Performed Equally With or Without Major Study Eligibility Violations

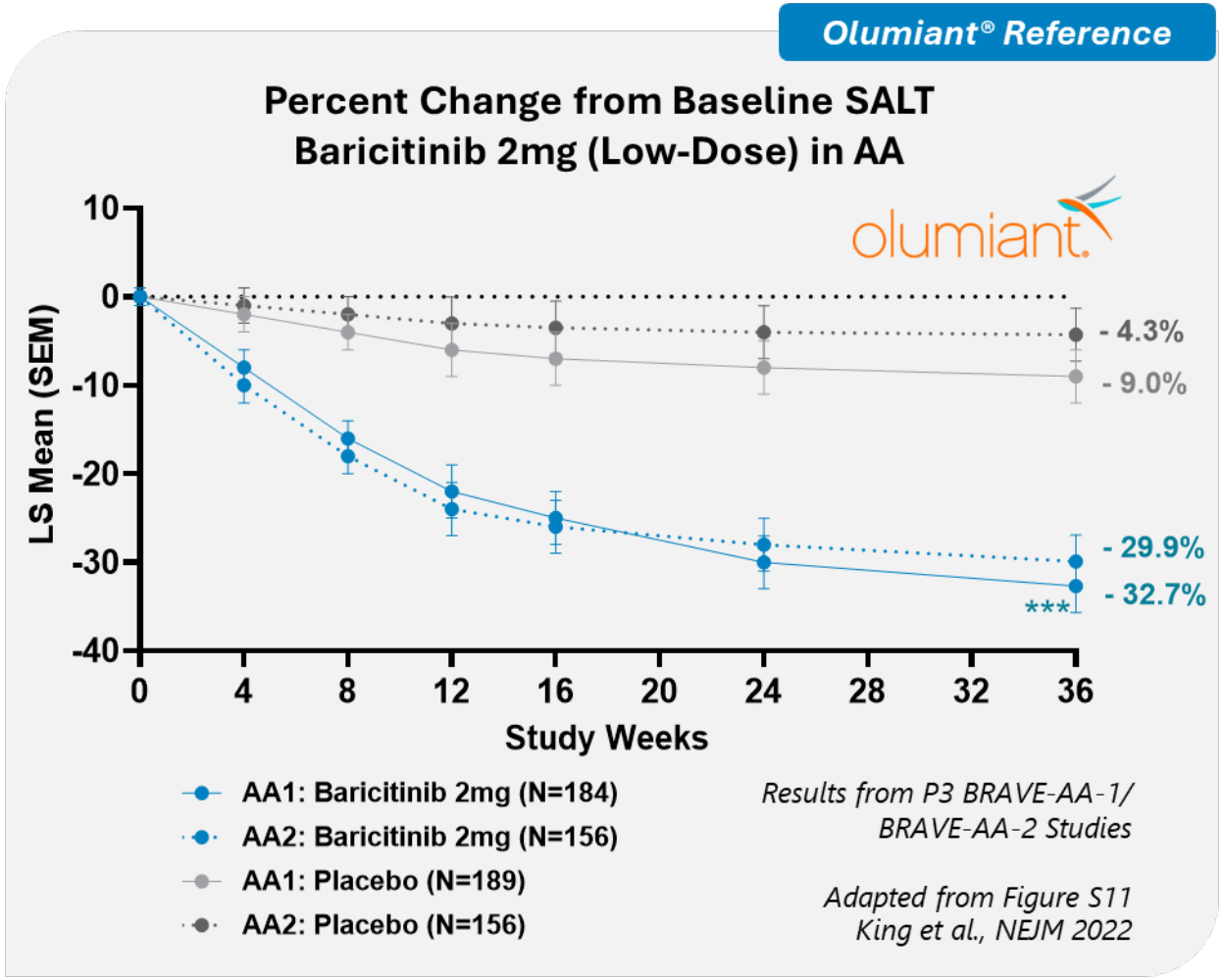




*p-value<0.05; **p-value<0.01
mITT excluding the 4 patients with the major study eligibility violations (post-hoc)

REZPEG Met Our Target Product Profile Which Was to Match Low-Dose JAKi at Week 36





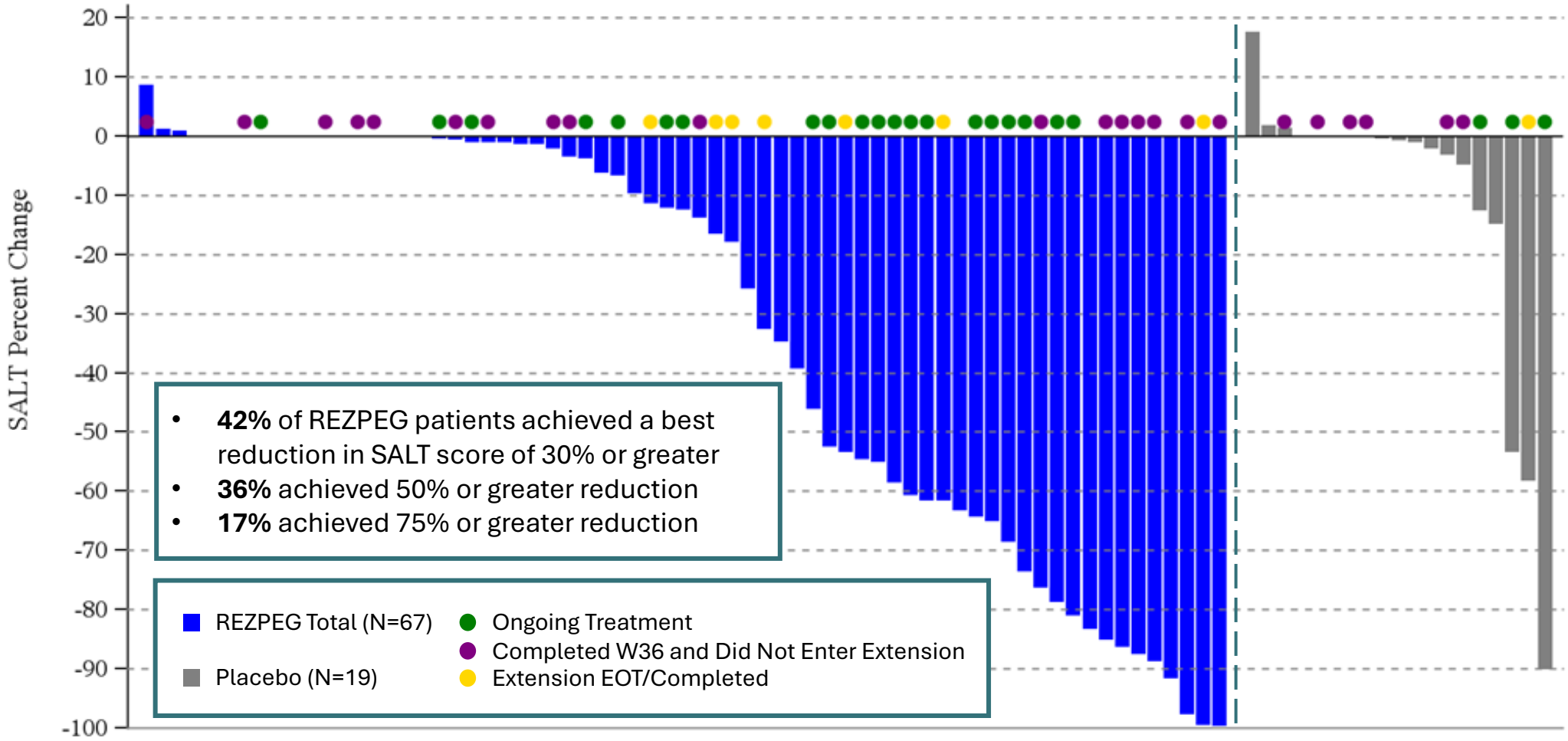
*p-value<0.05; **p-value<0.01
A. mITT excluding the 4 patients with the major study eligibility violations (post-hoc)

***p-value<0.001; Olumiant® is a registered trademark owned or licensed by Eli Lilly and Company, its subsidiaries, or affiliates.

This is a cross-trial comparison and includes data from different clinical studies and is not a head-to-head comparison.

REZPEG-Treated Patients Experienced Meaningful Hair Growth

Best Percent Change in SALT from Baseline (mITT [A]) at All Timepoints

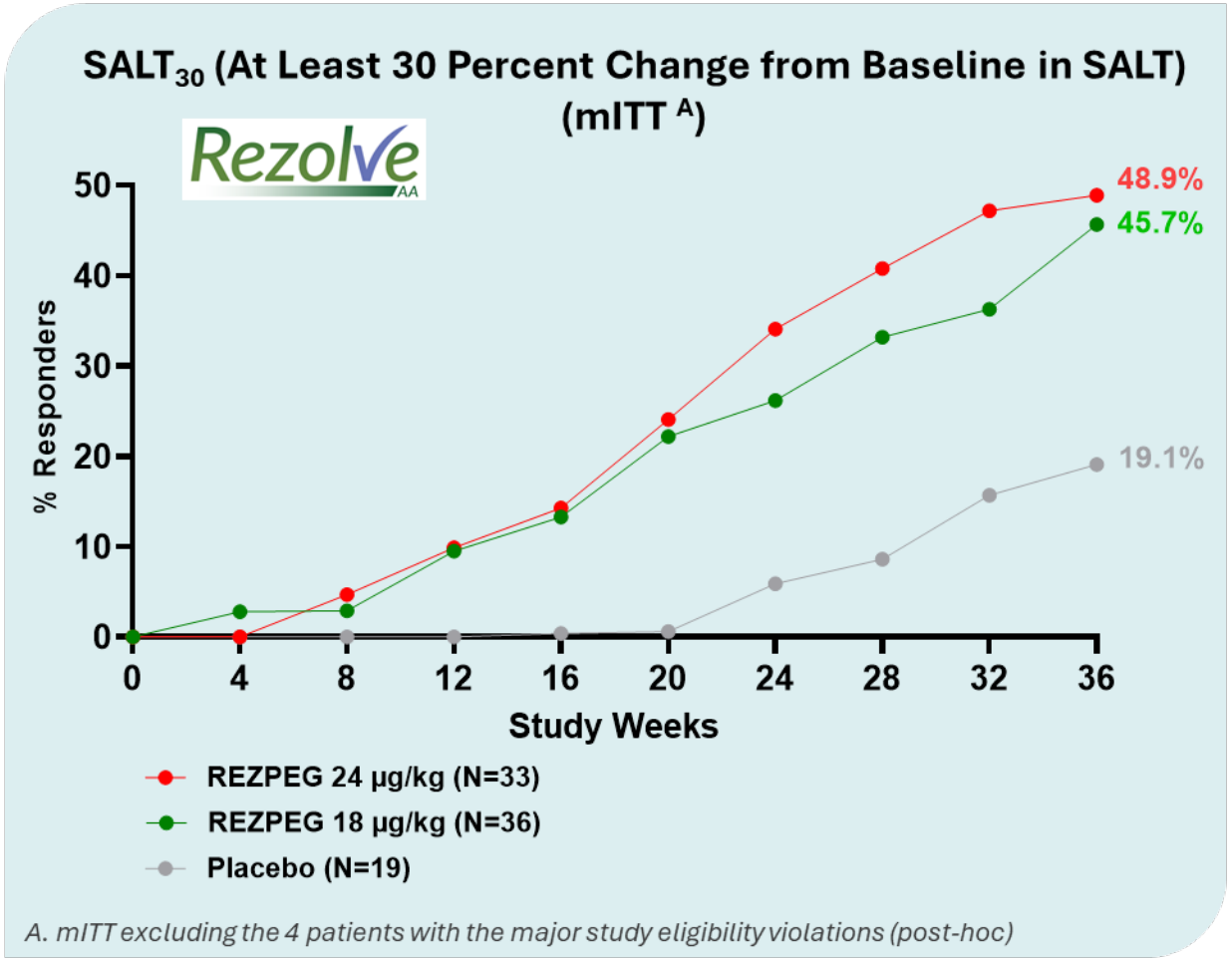


A. mITT excluding the 4 patients with the major study eligibility violations (post-hoc). Two patients in rezpeg group didn't have any post baseline assessment and therefore not included in this plot.

Each bar represents an individual patient

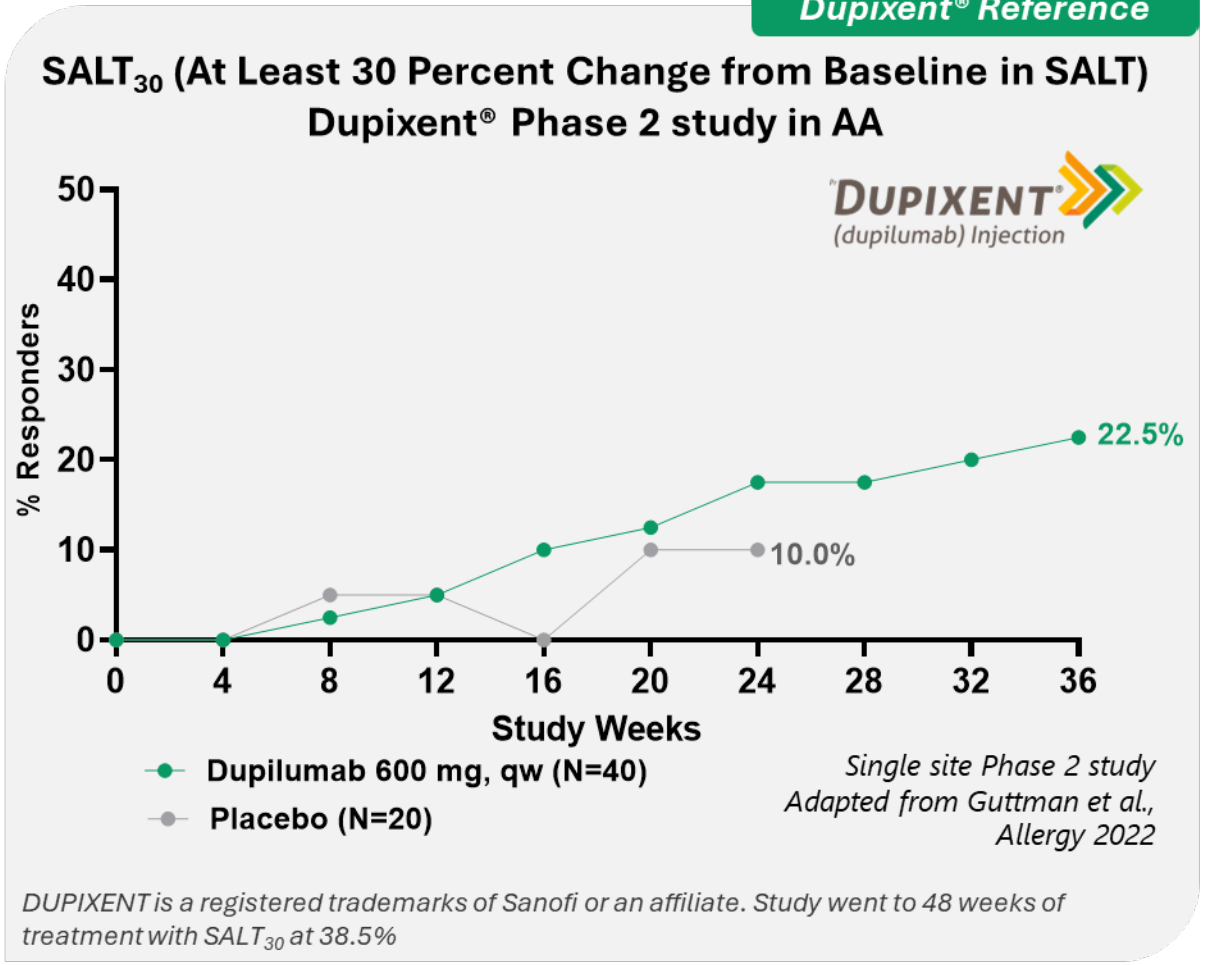
Promising Data for a Biologic in AA

$SALT_{30}$ (At Least 30 Percent Change from Baseline in SALT)



A. mITT excluding the 4 patients with the major study eligibility violations (post-hoc)

Dupixent® Reference

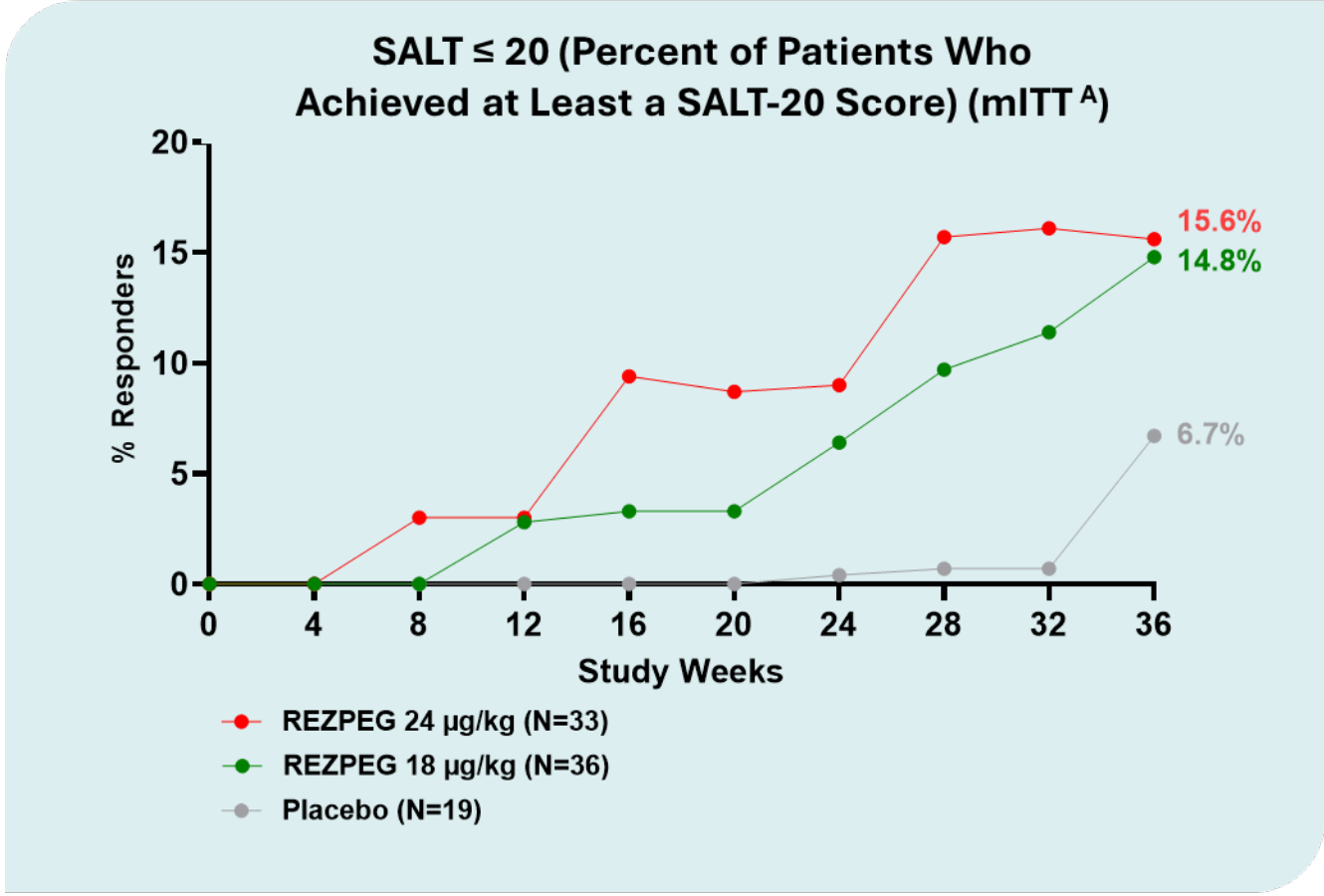


DUPIXENT is a registered trademarks of Sanofi or an affiliate. Study went to 48 weeks of treatment with $SALT_{30}$ at 38.5%

US DELPHI Consensus Guidelines "In patients with active atopy or history of atopic dermatitis, dupilumab may be considered as a long-term AA treatment" (Dec'25)

This is a cross-trial comparison and includes data from different clinical studies and is not a head-to-head comparison.

SALT ≤ 20: Clear Dose Response and Separation from Placebo



Among Rezpeg treated patients:

- 3 additional patients have already achieved a SALT ≤ 20 in the 16-week treatment extension with 2 patients ongoing treatment
- 7 additional patients who achieved SALT ≤ 30 are ongoing in the 16-week treatment extension

A. mITT excluding the 4 patients with the major study eligibility violations (post-hoc)

Rezpegaldesleukin in Alopecia Areata

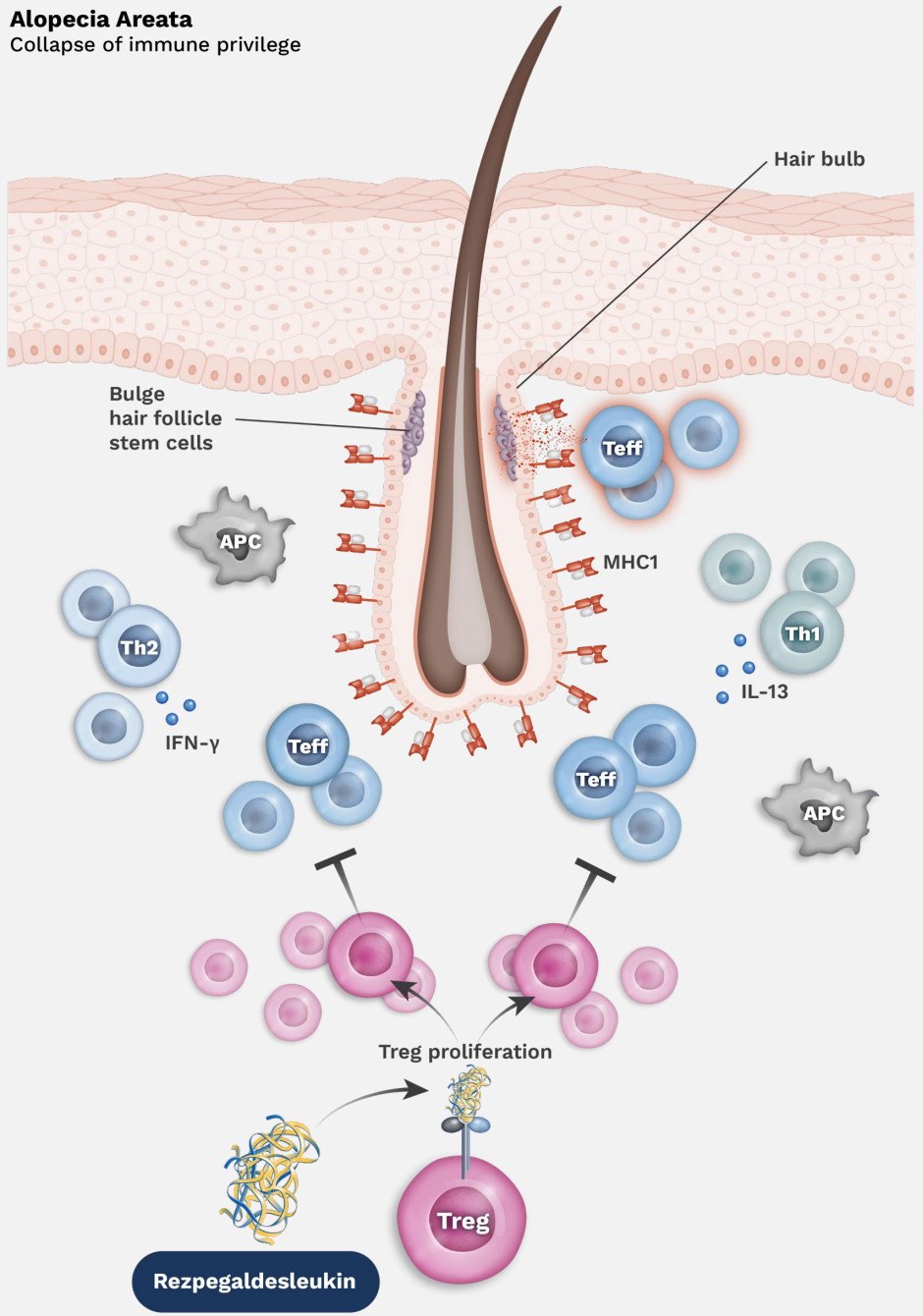
Rezpegaldesleukin
Alopecia Areata MOA

Scientific rationale

- By targeting receptors on regulatory T cells, rezpegaldesleukin stimulates the proliferation of regulatory T cells (Tregs), including FOX-P3+ Tregs

- In alopecia, a pro-inflammatory environment causes the collapse of immune privilege around the anagen hair bulb leading to hair follicle focal inflammation driven by NKG2D + T cells, NK cells, as well as auto-reactive CD8+ T cells

- Regulatory T cells act upstream of these inflammatory cytokines to reduce their activity; by increasing the number and functionality of regulatory T cells, rezpegaldesleukin aims to reduce the hair follicle local inflammation and restore immune privilege.



Rezpegaldesleukin in Alopecia Areata overview

Topline data from Phase 2b study in severe-to-very severe alopecia areata in December 2025

Alopecia areata (AA) is a disease that happens when the immune system attacks hair follicles and causes hair loss[1]

- Nearly 6.7 million people in the US have had it or will have it in their lifetime. ~700,000 people currently have alopecia areata in US. [2]
- ~160 million people worldwide have alopecia areata or have had, or will have it[2]
- 80% of patients show signs of alopecia before 40[2]
- Many patients are refractory to available therapies, and long-term use is associated with troublesome side effects and safety risks[3]
- Only systemic treatments approved for alopecia are JAK inhibitors with multiple Blackbox warnings. High relapse rates upon discontinuation of these therapies[3]

Represents additional opportunity to expand Rezpeg

Sources: 1. NIH; 2. National Alopecia Areata Foundation; 3. DRG – Unmet Needs of Alopecia Areata

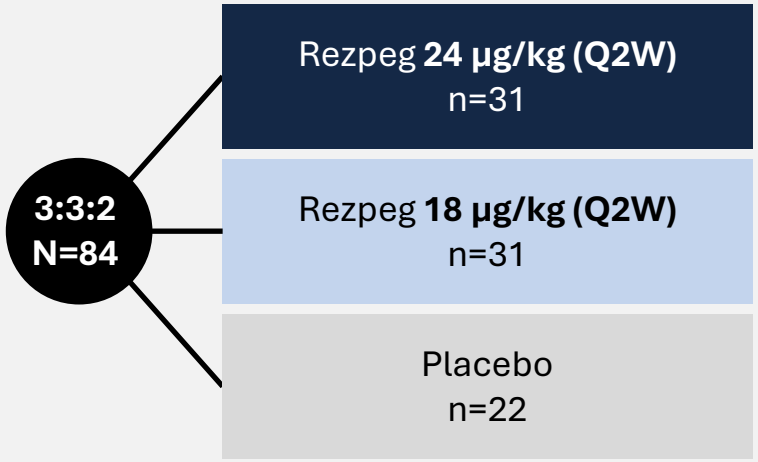
Phase 2b study – topline results expected December 2025

Severe-to-very severe Alopecia Areata

Screening	Induction (Weeks 0-36)	Blinded Observations 24 Weeks Follow-Up

Key Inclusion Criteria:

- 18+ years of age
- Severe-to-Very Severe AA
- SALT ≥50



Week 36 (End of Induction) Endpoints

Primary:

- Mean percent improvement in SALT

Secondary/Exploratory:

- Proportion with ≥50% reduction in SALT at Week 36 and other assessed timepoints
- Mean % improvement in SALT at other assessed timepoints
- Safety
- PK/PD

After week 36, a 16-week treatment extension is available to all cohorts that did not achieve a SALT score ≤20 and has demonstrated substantial hair regrowth

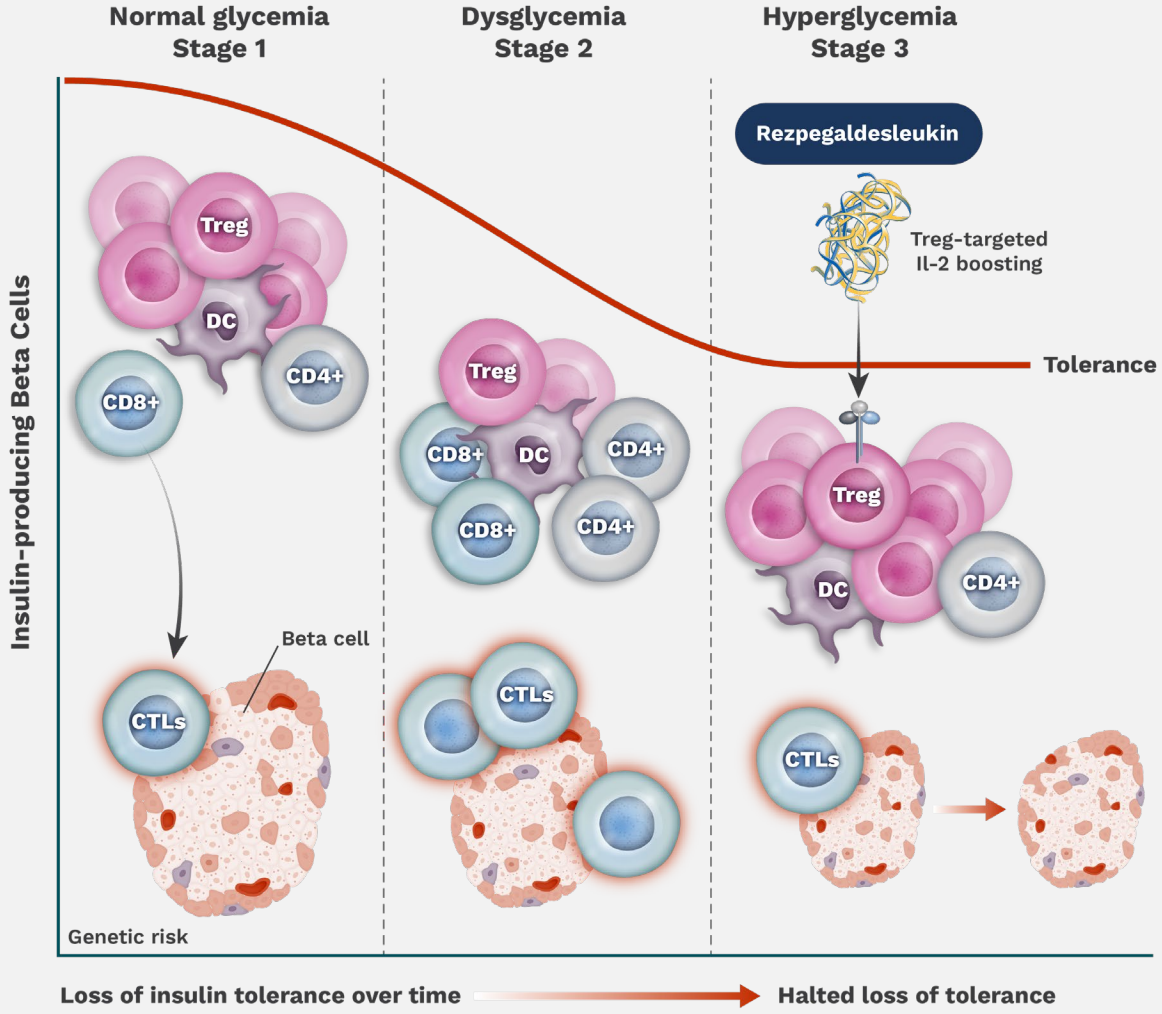
Severity of Alopecia Tool (SALT) is widely used to assess the extent of scalp-hair loss in patients with alopecia areata, with guidelines defining treatment success as a 50% improvement in scalp hair

Rezpegaldesleukin in Type 1 Diabetes

Rezpegaldesleukin
Type 1 Diabetes MOA

Scientific rationale

- By targeting receptors on regulatory T cells, rezpegaldesleukin stimulates the proliferation of regulatory T cells (Tregs), including FOX-P3+ Tregs
- In Type 1 diabetes, the destruction of insulin-producing pancreatic beta cells is caused by T cells of the immune system
- Regulatory T cells act upstream of these T cell and pro-inflammatory cytokines to reduce their activity; by increasing the number and functionality of regulatory T cells, this investigational therapy aims to slow the progression of new onset Type 1 diabetes.



Proposed study design

Type 1 Diabetes (Stage 3)

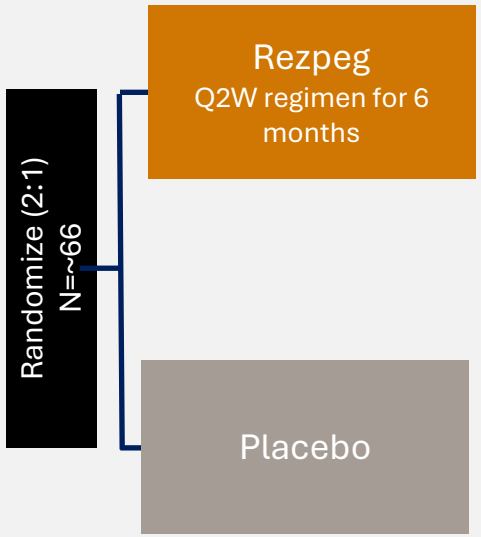
Type 1 Diabetes TrialNet

Eligibility

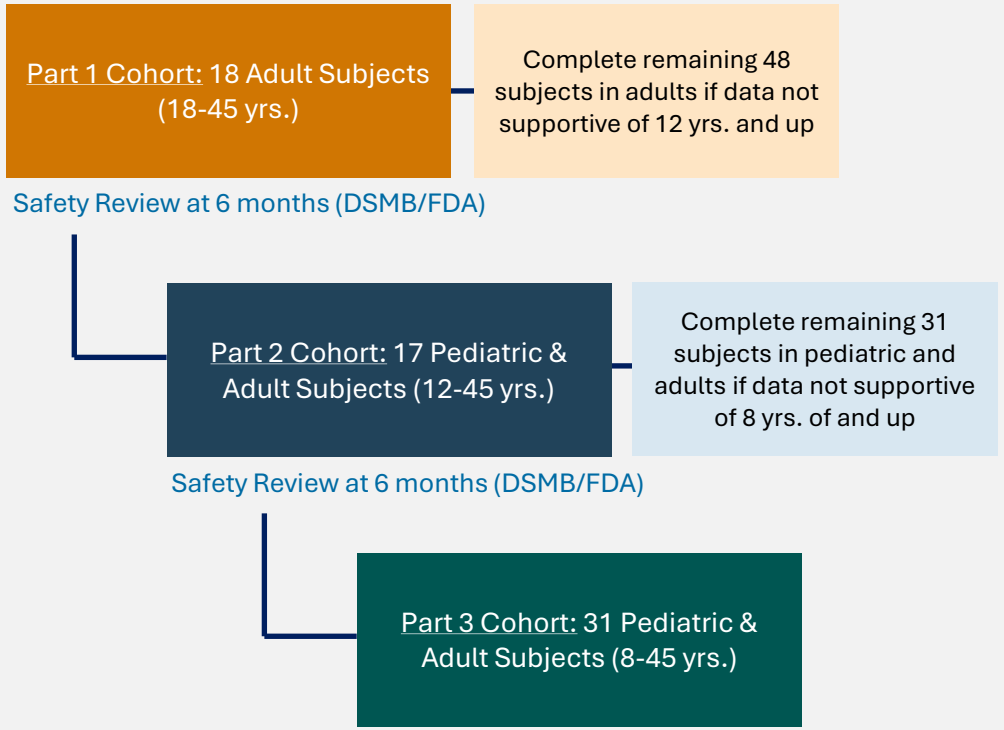
Key Inclusion:

- ✓ Ages starting at minimum of 18 and then decreasing to 12 and then 8 pending data review
- ✓ New onset T1D within 100 days
- ✓ Positive for ≥1 autoantibody (+AAB)
- ✓ Peak C-peptide >0.2pmol/mL

Treatment



Patient Enrollment



Outcome Measures

ENDPOINTS:

Primary:
C-peptide response to Mixed Meal Tolerance Test (MMTT) compared to placebo at 12 months

Secondary/Exploratory:

- Comparisons of MMTT-stimulated C-peptide and glucose AUCs at 3 and 6 months to placebo
- Comparisons of MMTT-stimulated C-peptide at 3, 6, and 12 months to historical model-adjusted placebo controls
- Glucose monitoring data and hypoglycemia events
- Adverse Events

Additional Assessments:

- Metabolic Assessments (HbA1c, insulin usage, etc.)
- Immunologic Assessments

NKTR-0165

TNFR2 Agonist Antibody

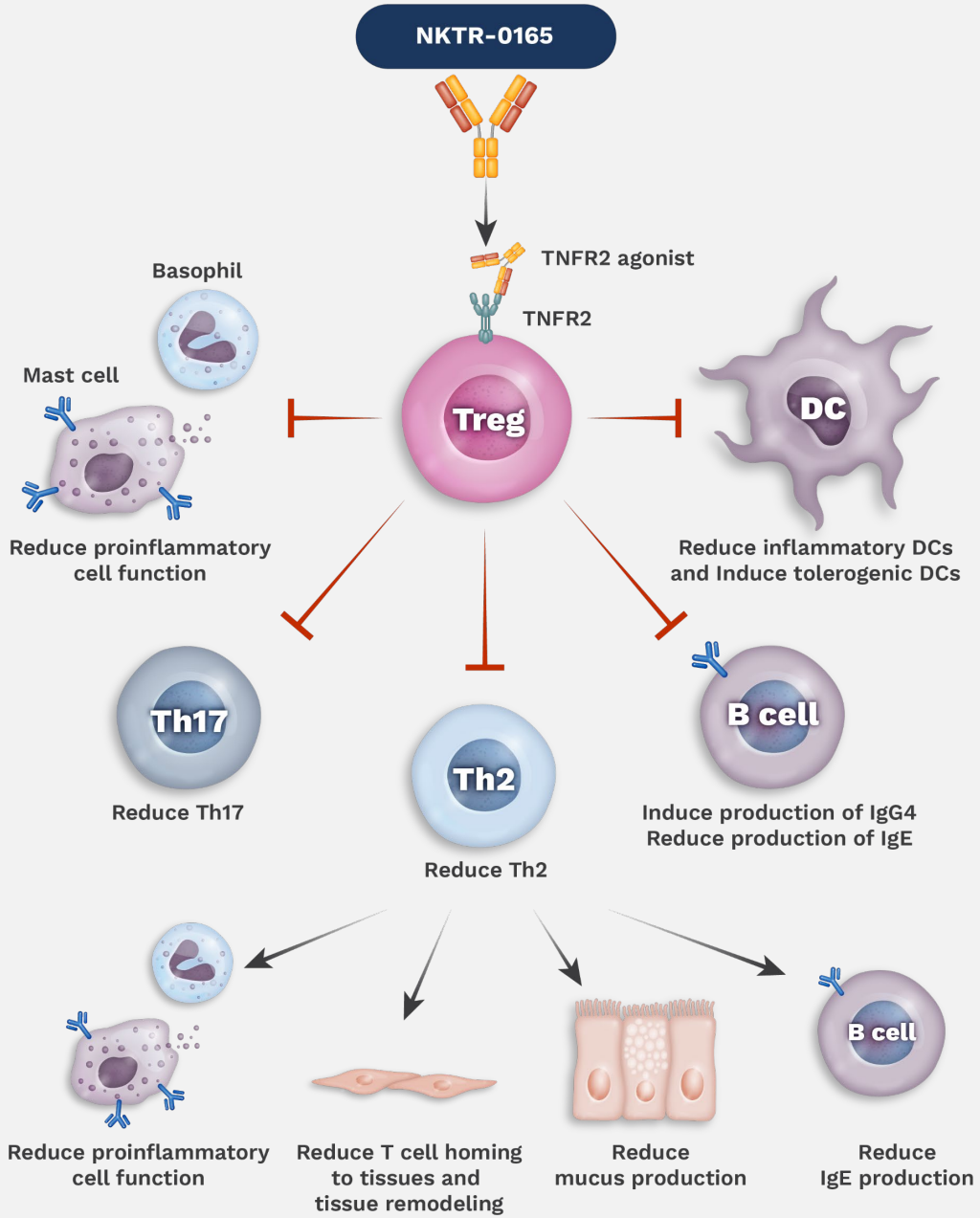
NKTR-0165/TNFR2 Agonist Bivalent Antibody

Scientific rationale

- By targeting TNFR2, a co-stimulatory receptor preferentially expressed on the surface of highly suppressive tissue-specific regulatory T cells, B-regulatory cells, neuronal cells and endothelial cells, NKTR-0165 has been shown to promote the activation, function, proliferation, and phenotypic stability of these cells

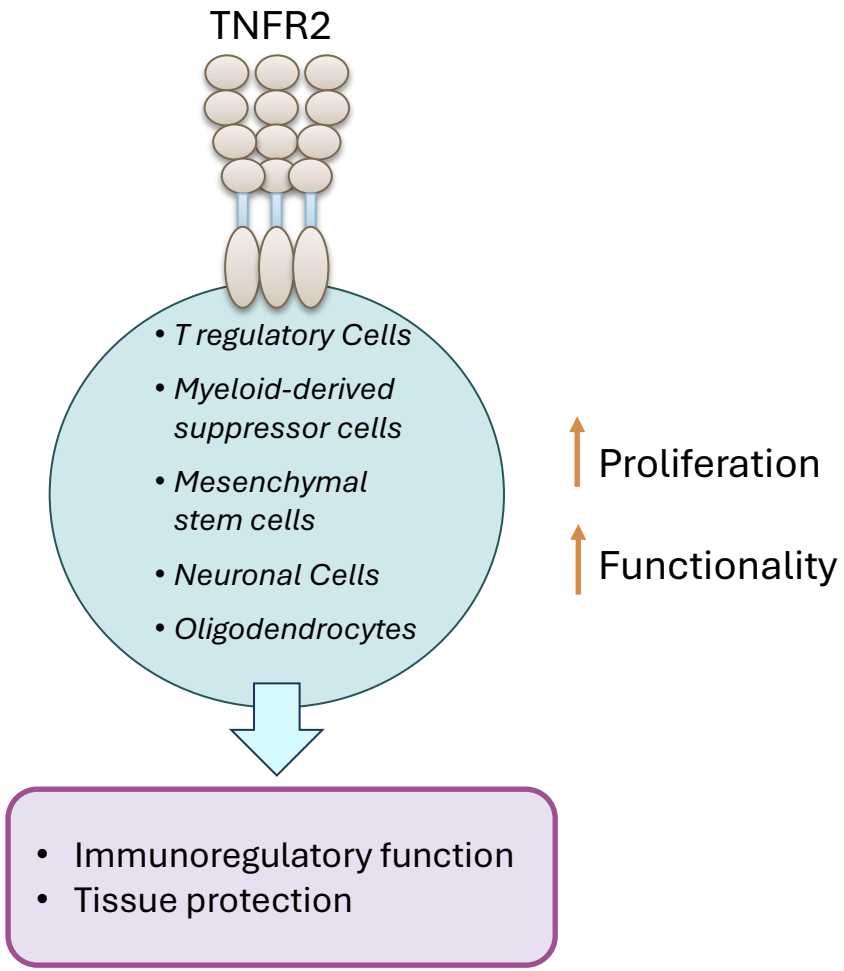
- TNFR2 signaling is an important gatekeeper of inflammation and its absence or deficit is associated with a broad range of autoimmune diseases

- As a monomeric bivalent antibody with a unique epitope, NKTR-0165 selectively stimulates TNFR2 receptor activity, without modulation of TNFR1 signaling; this novel first-in-class compound has the potential to modulate acute exacerbations and chronic trajectory of a number of autoimmune diseases



NKTR-0165: TNFR2 agonist antibody program

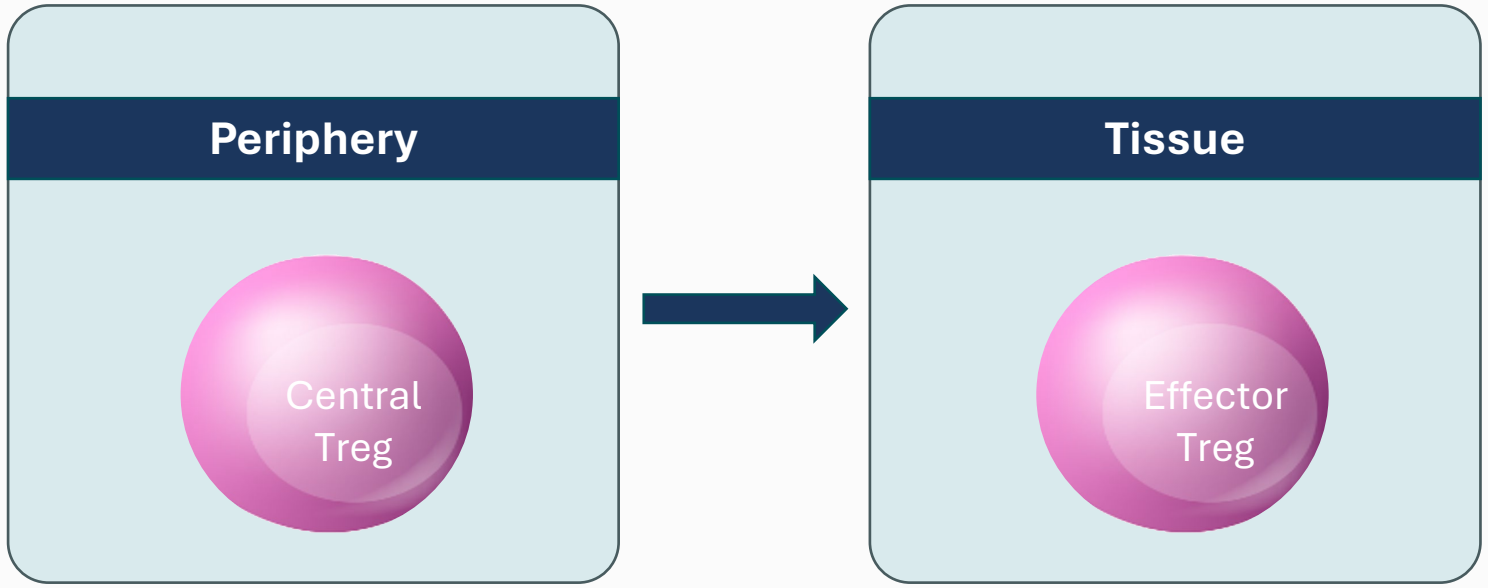
Targeting TNF Receptor 2 (TNFR2) for the treatment of autoimmune conditions



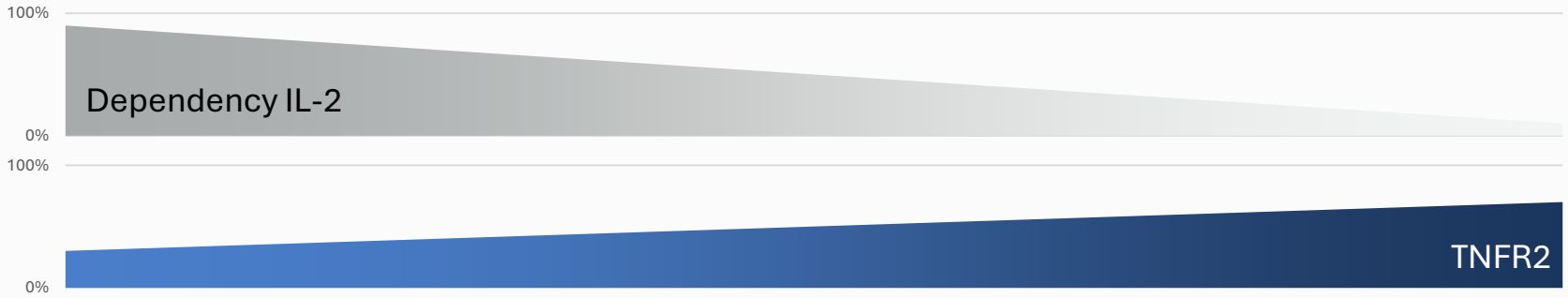
- TNFR2 signaling drives immunoregulatory function could provide direct protective effect for tissue cells
- Unique Nektar antibody candidates show selective T regulatory cell binding and signaling profiles enabling it to be developed for the treatment of autoimmune conditions
- Program targets multiple MOAs including suppression of inflammation, regrowth of myelin after demyelination (MS) and promotion of immune resolution.
  - Examples include Ulcerative Colitis, Multiple Sclerosis (i.e. myelin regrowth), Vitiligo and other autoimmune conditions
- Targeting IND submission in H2'26

TNFR2/NF-kB Axis is critical to maintain effector and tissue regulatory T cells

Rezpegaldesleukin (IL-2 T Regulatory Cell Stimulator)



NKTR-0165 (Bivalent Agonistic Antibody Targeting TNFR2)



Cell Reports 20, 2906-2920, September 19, 2017

Multiple near/medium-term inflection points

Rezpegaldesleukin in Atopic Dermatitis

- Presentation of full 16-week Phase 2 data for Rezpeg in atopic dermatitis at a major medical meeting
- Planned End-of-Phase 2 meeting with the FDA to align on Phase 3 development
- 36-week maintenance data from Phase 2 AD study expected in early 2026

Rezpegaldesleukin in Alopecia

- Topline data from Phase 2b study in severe-to-very severe alopecia areata in December 2025

Other programs

- Regulatory filing and potential approval from our partnered program dapirolizumab in SLE in 2026-2027
- NKTR-255 trial data updates with various combinations in 2025-2026