



NEKTAR[®]

NEW PATHWAYS TO
SMARTER MEDICINE™

Phase 2b REZOLVE-AD Topline Results from 16-Week Induction

Rezpegaldesleukin in Patients with Moderate-to-Severe Atopic Dermatitis

June 24, 2025

Forward-Looking Statements

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REZOLVE-AD Phase 2b Validates Rezpeg as a First-in-Class Novel T-Regulatory Mechanism in Atopic Dermatitis (AD)

Novel T-Reg MOA differentiates from existing and in-development biologics

- ✓ Up to 6-fold increase in T-regs
- ✓ 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 ($p < 0.001$)

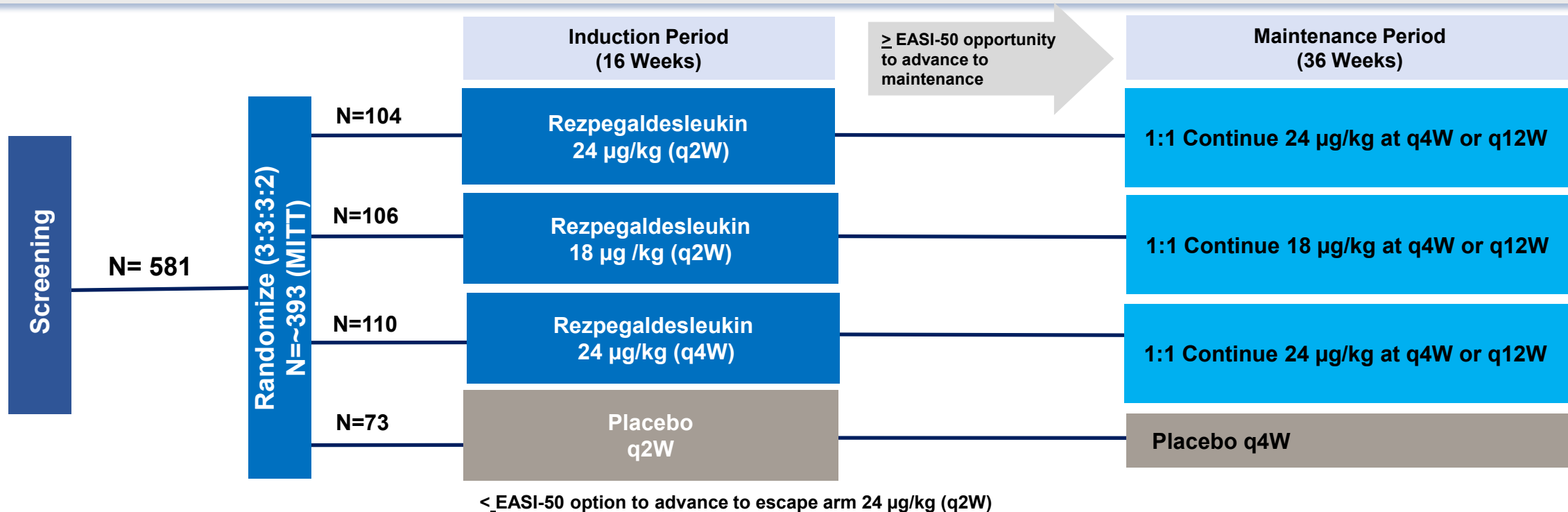
Other 2 doses also met multiple secondary endpoints

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)

REZOLVE-AD: Phase 2b Trial Design

Patients with Moderate-to-Severe Atopic Dermatitis



Stratification

- ✓ Geographic region
- ✓ Baseline severity

Key Inclusion Criteria:

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

Key Pharmacodynamic Biomarkers:

- T regulatory cell
- TARC/CC17
- Periostin
- MDC/CCL22
- IL-19

REZOLVE-AD: Phase 2b Trial Design

Primary and Secondary Endpoints, Use of Rescue Therapy and Statistical Design

Primary Endpoint:

- Mean % EASI improvement at Week 16

Key Secondary Endpoints at Week 16:

- vIGA-AD of 0 or 1 with ≥ 2 -point reduction from baseline (vIGA-AD 0/1)
- EASI-75, -90, -50
- Itch NRS, ≥ 4 -point reduction from baseline
- Mean % Body Surface Area (BSA) improvement

- **Primary Estimand analysis:** patients who used rescue therapy outside protocol specifications or who discontinue treatment due to lack of efficacy were considered **NONRESPONDERS** (using baseline observation carry forward (BLOCF) for continuous endpoints, and non responder imputation for binary endpoints), regardless of observed clinical response; data after patients who discontinue due to other reasons set to missing and all missing data are imputed using the multiple imputation method.

Statistical Analysis Methods

- The Primary Estimand analysis for continuous endpoints of %EASI improvement and %BSA improvement use a mixed model for repeated measures (MMRM) to estimate the treatment difference between dose arms and placebo
- The Primary Estimand analysis for binary endpoints (vIGA-AD 0/1, EASI-75, EASI-90, and Itch NRS) use a logistic regression model to estimate the treatment difference between dose arms and placebo
- Statistical methodologies **most similar** to Sanofi STREAM-AD Phase 2b Study

Baseline Demographics and Disease Characteristics

■ Patient Demographics

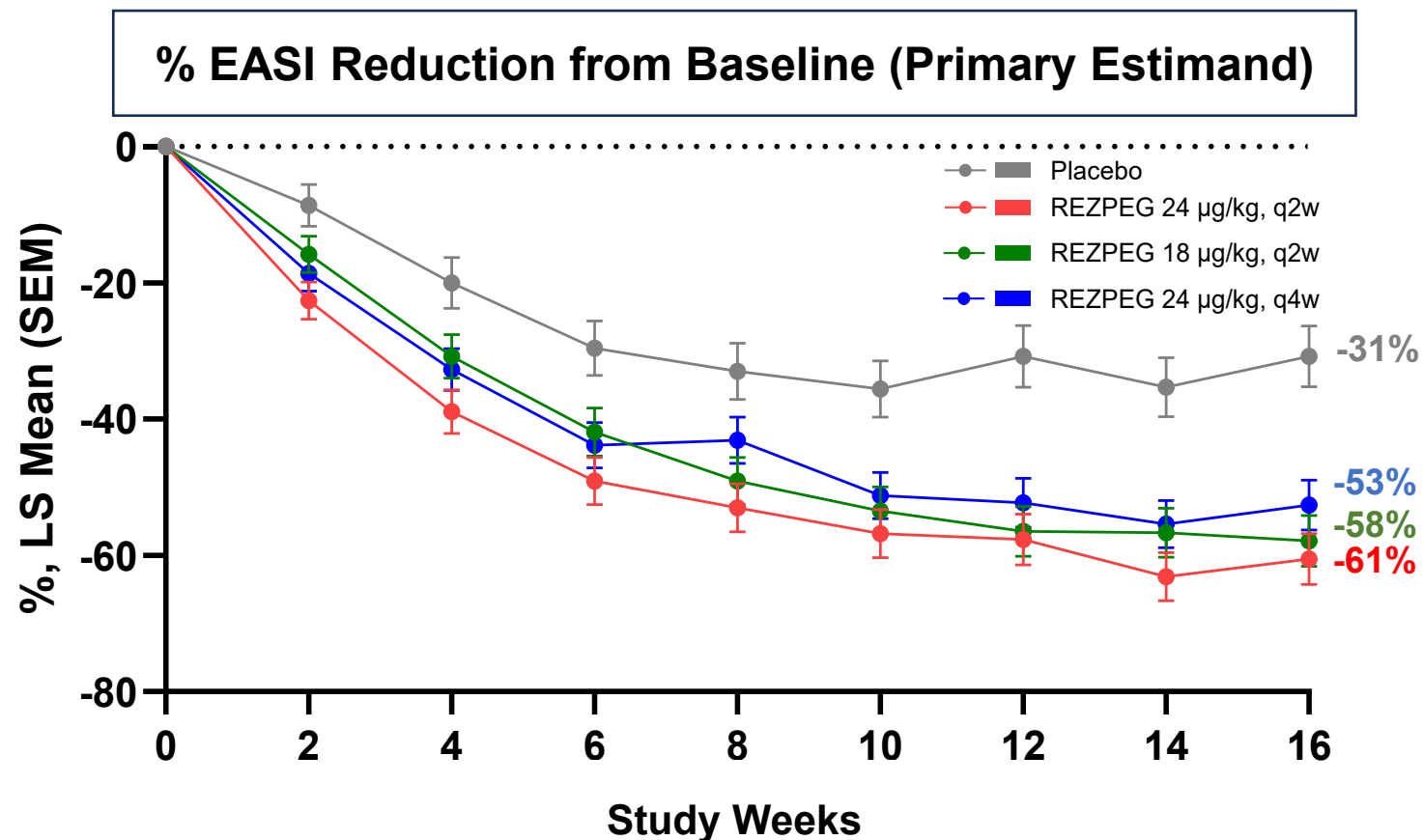
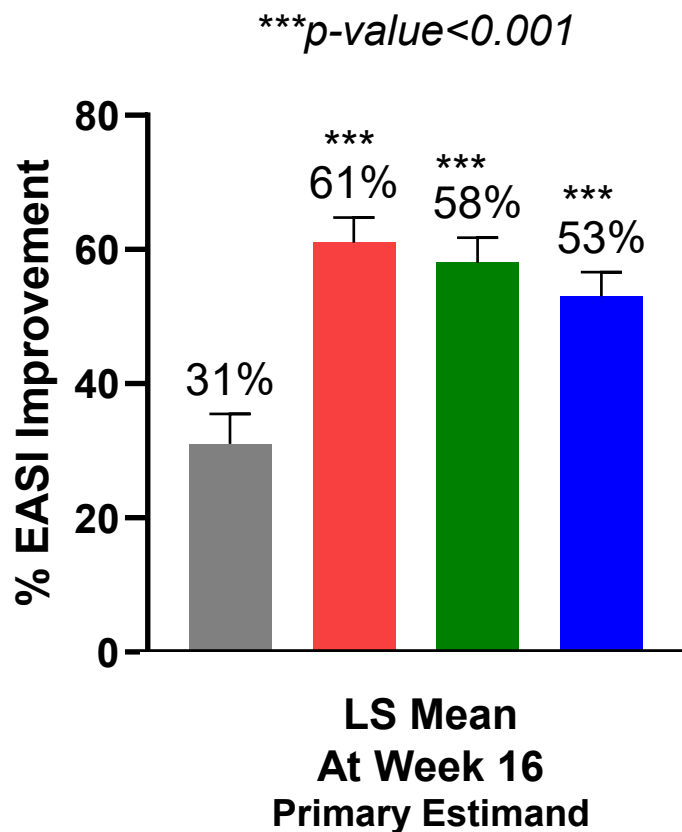
- Patients were predominantly recruited from Europe, but also from North America and Australia
- Stratification Factor: North America (27.5%) vs Rest of World (72.5%)
- Majority of patients were under 65 years-old, well balanced among men and women
- Majority were White (84.2%) with mean \pm SD disease duration of 21.5 ± 14.9 years

■ Baseline Disease Characteristics, mean \pm SD:

- EASI was 26.0 ± 9.8
 - EASI < 21 (40.7%) vs EASI ≥ 21 (59.3%)
- Stratification Factor: Baseline vIGA-AD 3 (67.9%) vs vIGA-AD 4 (32.1%)
- BSA was $39.5 \pm 20\%$
- Itch NRS was 6.8 ± 1.95
- Well-balanced across arms

Dose Dependent % EASI Reduction, Clear Separation from Placebo at All Timepoints for Study Treatment Arms

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



N=73, 104, 106, and 110 for the placebo, 24 µg/kg q2w, 18 µg/kg q2w, and 24 µg/kg q4w groups

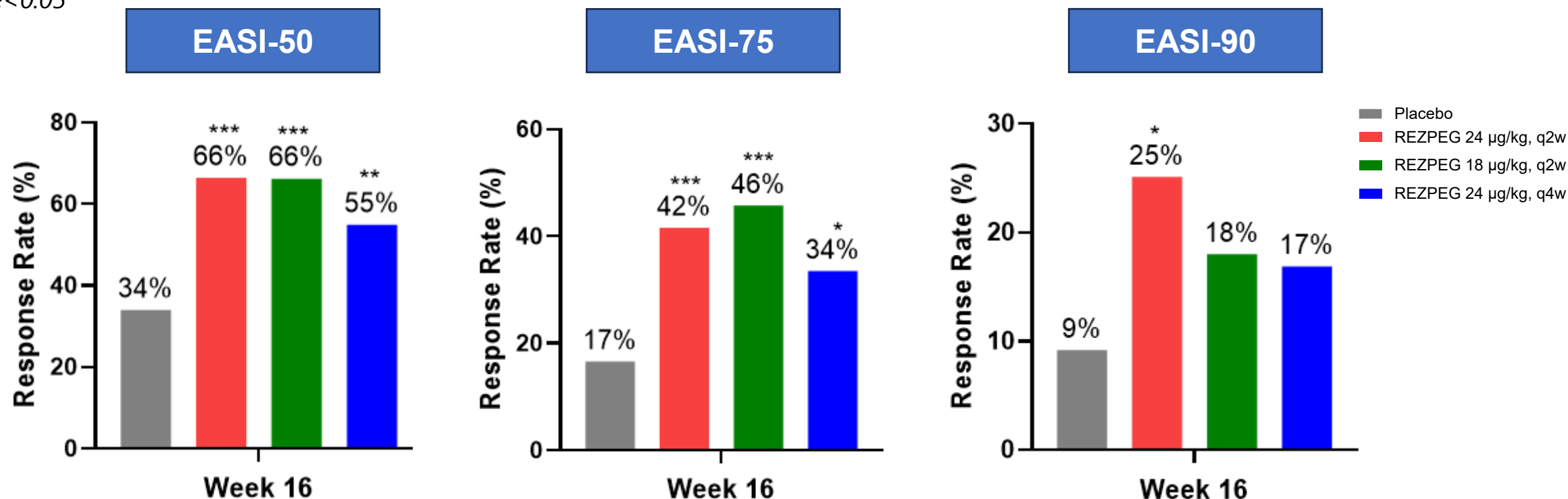
High Dose Met all Key Secondary Endpoints

Multiple endpoints met for 2 additional dose arms

*** p -value < 0.001

** p -value < 0.01

* p -value < 0.05



Primary Estimand: N=73, 104, 106, and 110 for the placebo, 24 µg/kg q2w, 18 µg/kg q2w, and 24 µg/kg q4w groups

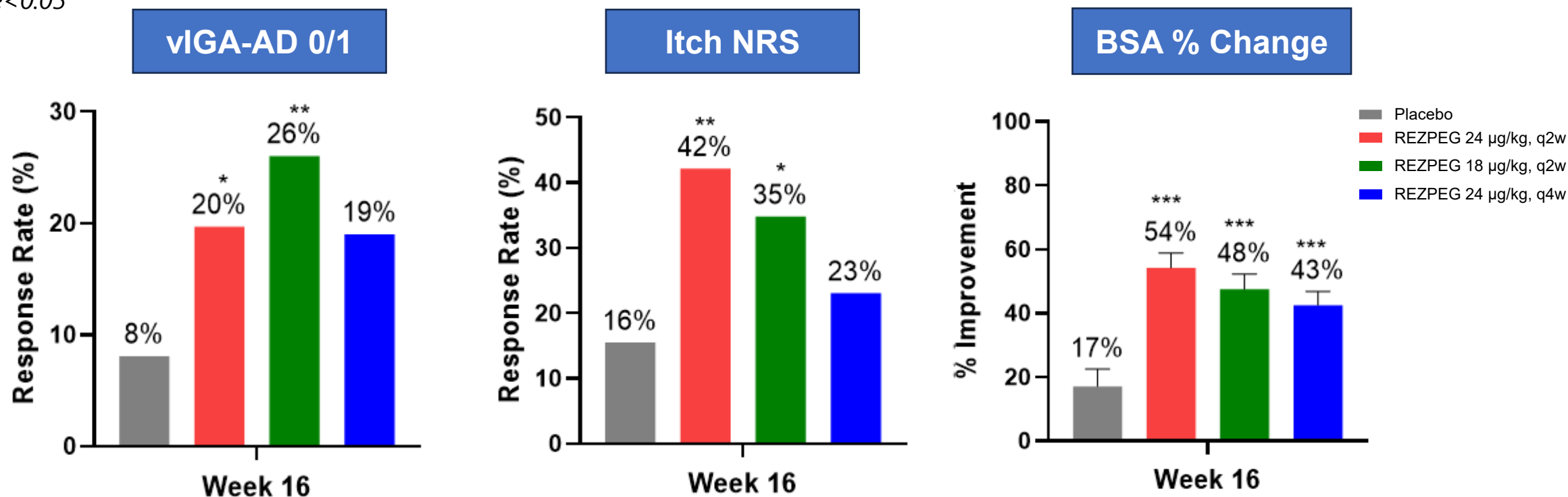
High Dose Met all Key Secondary Endpoints

Multiple endpoints met in 2 additional dose arms

*** p -value < 0.001

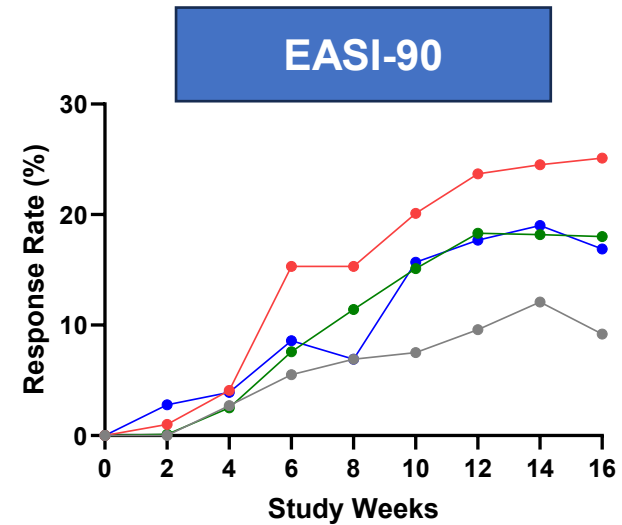
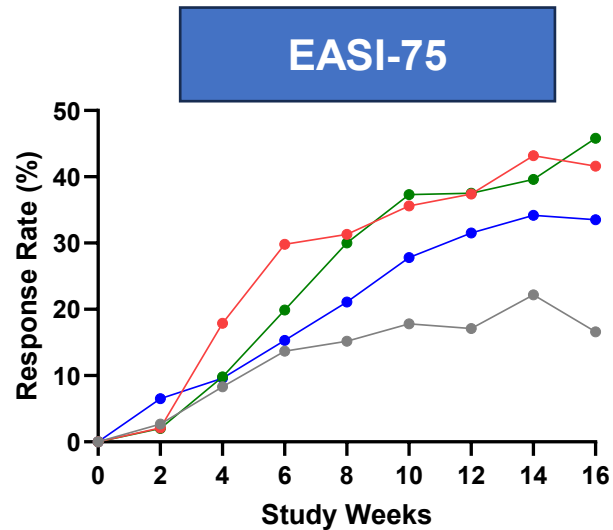
** p -value < 0.01

* p -value < 0.05

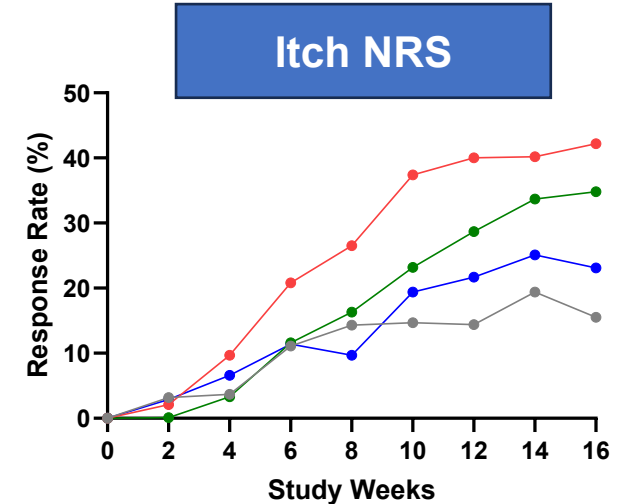
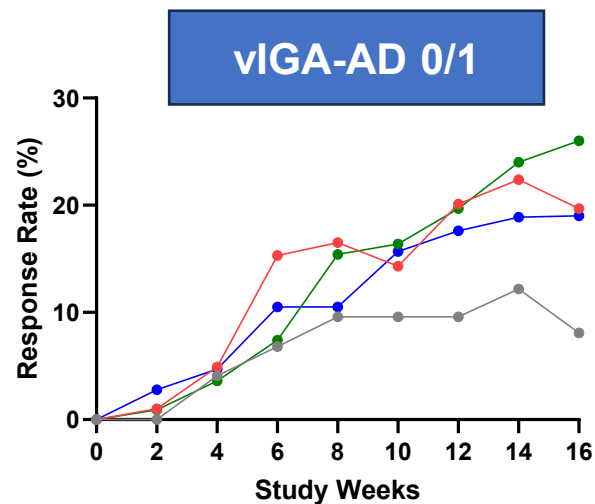


Primary Estimand: N=73, 104, 106, and 110 for the placebo, 24 µg/kg q2w, 18 µg/kg q2w, and 24 µg/kg q4w groups) for the vIGA-AD 0/1 and continuous BSA endpoint. The MITT population with baseline itch ≥ 4 (N=63, 95, 92, and 102 for the placebo, 24 µg/kg q2w, 18 µg/kg q2w, and 24 µg/kg q4w groups)

Fast Onset of Action Across All Key Secondary Endpoints



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



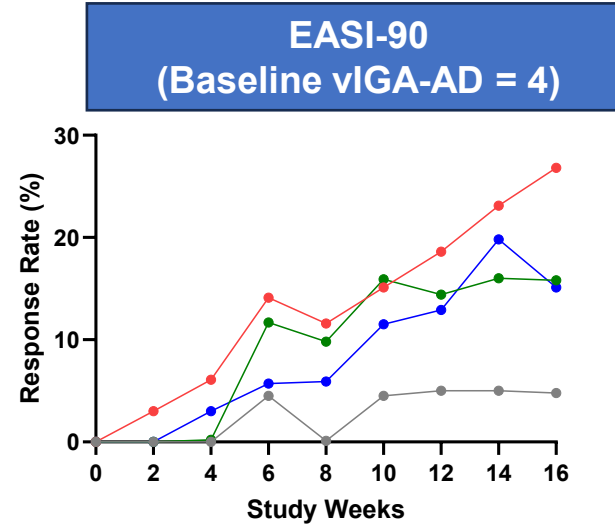
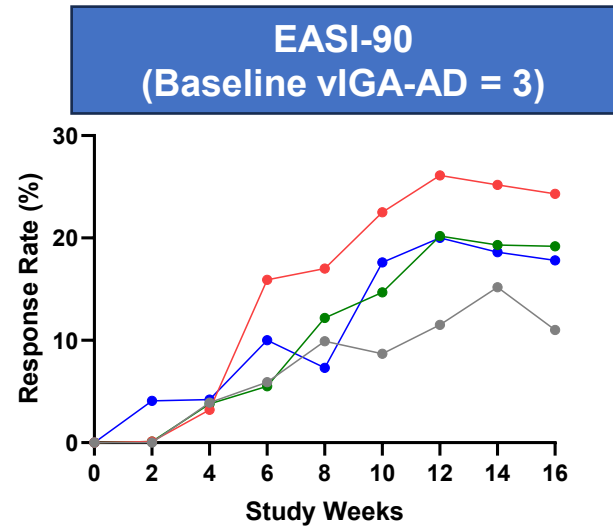
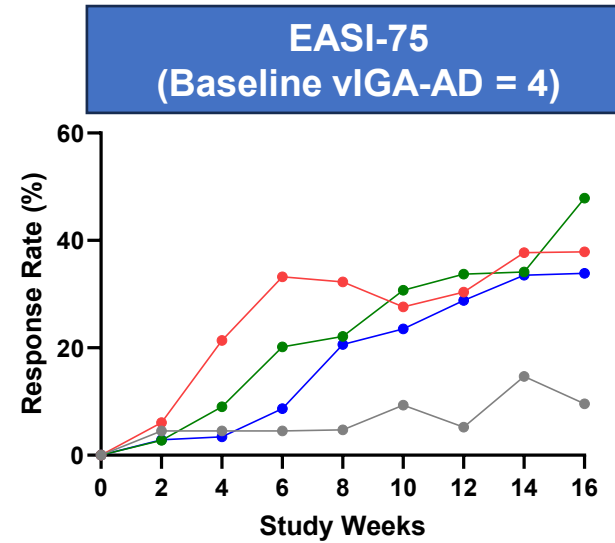
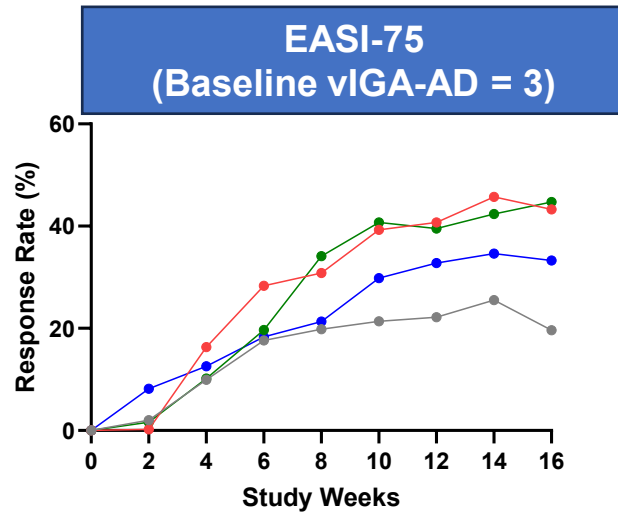
Primary Estimand Analysis

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

Similar Efficacy Observed in Severe Patients as in Moderate

EASI-75 and EASI-90 by baseline vIGA-AD score



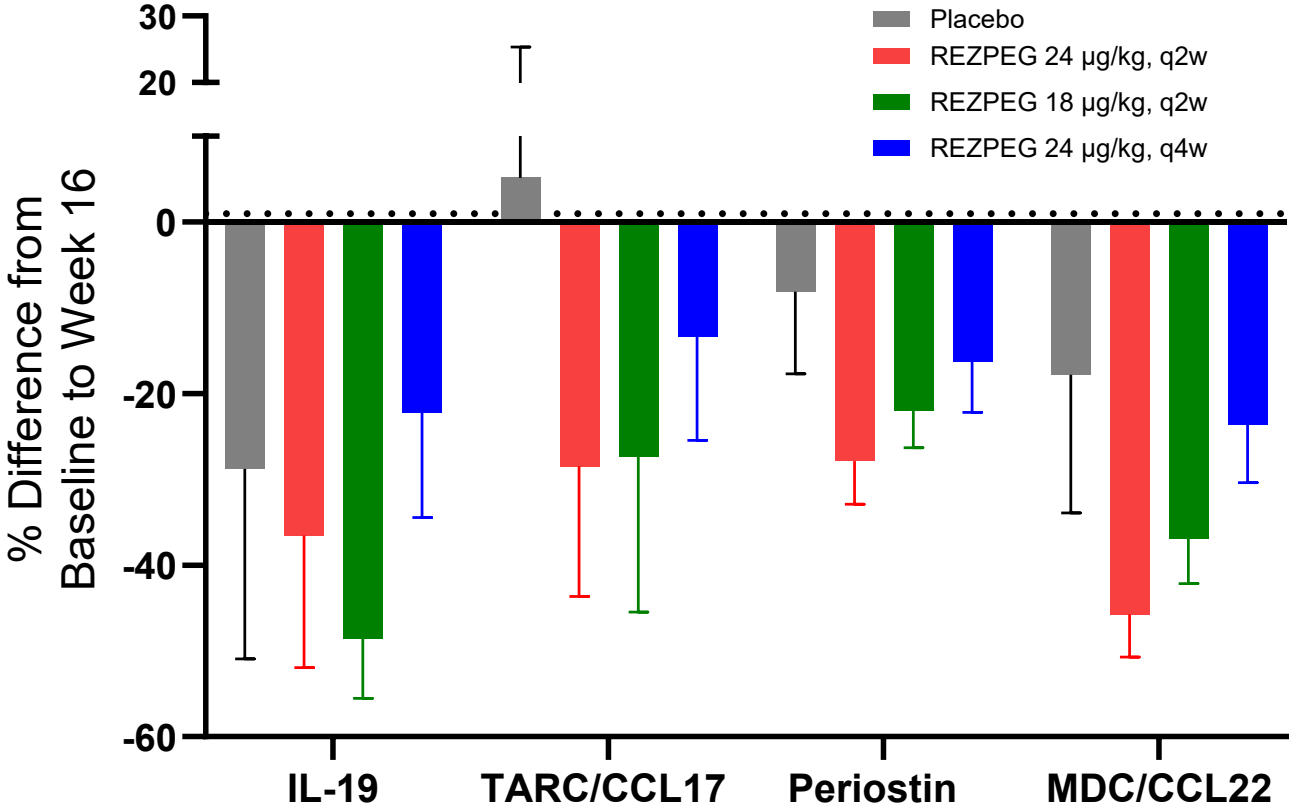
- Placebo
- 24 µg/kg, q2w
- 18 µg/kg, q2w
- 24 µg/kg, q4w

Arm	Sample Size	
	Baseline vIGA-AD =3	Baseline vIGA-AD =4
Placebo	51	22
24 µg/kg, q2w	71	33
18 µg/kg, q2w	70	36
24 µg/kg, q4w	75	35

Primary Estimand Analysis

Reductions Observed in Four Key Biomarkers of Atopic Dermatitis

*TARC/CCL17, Periostin, MDC/CCL22, IL-19 are key markers associated with atopic dermatitis**



Sample Size

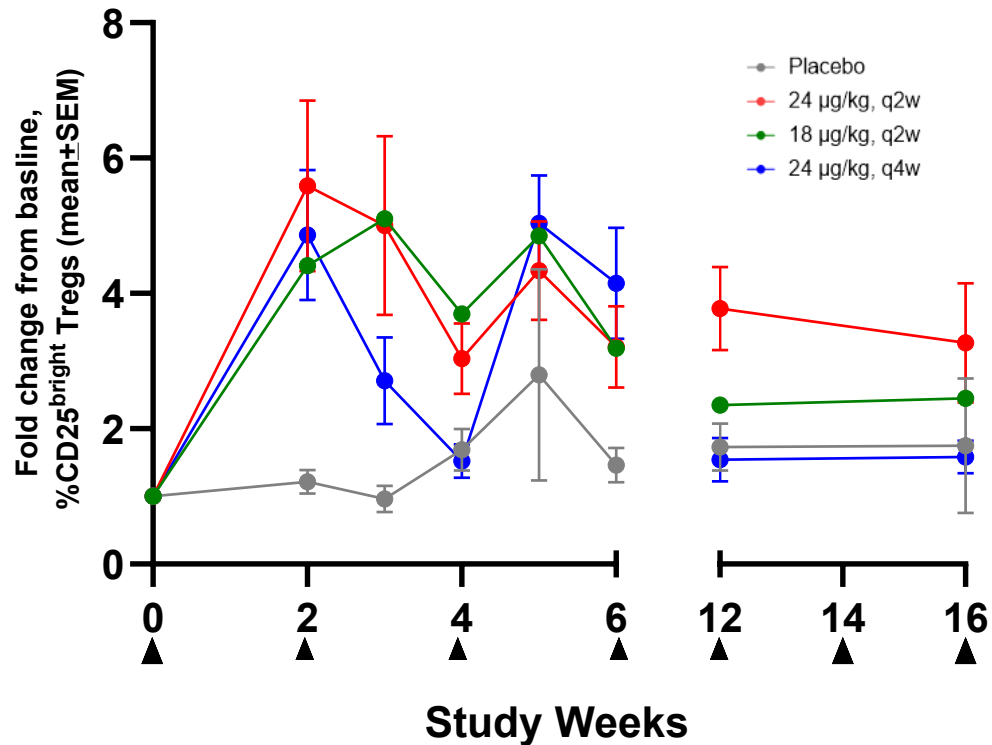
Arm	IL-19 (>51 pg/mL)	TARC/CCL17 (>0.94 ng/mL)	Periostin (>322 ng/mL)	MDC/CCL22 (>839 pg/mL)
Placebo	14	29	21	21
24 µg/kg, q2w	25	35	29	28
18 µg/kg, q2w	23	34	29	28
24 µg/kg, q4w	28	34	32	27

Patients with baseline values >ULN included in the analysis

PK/PD Profile Consistent with Prior Studies

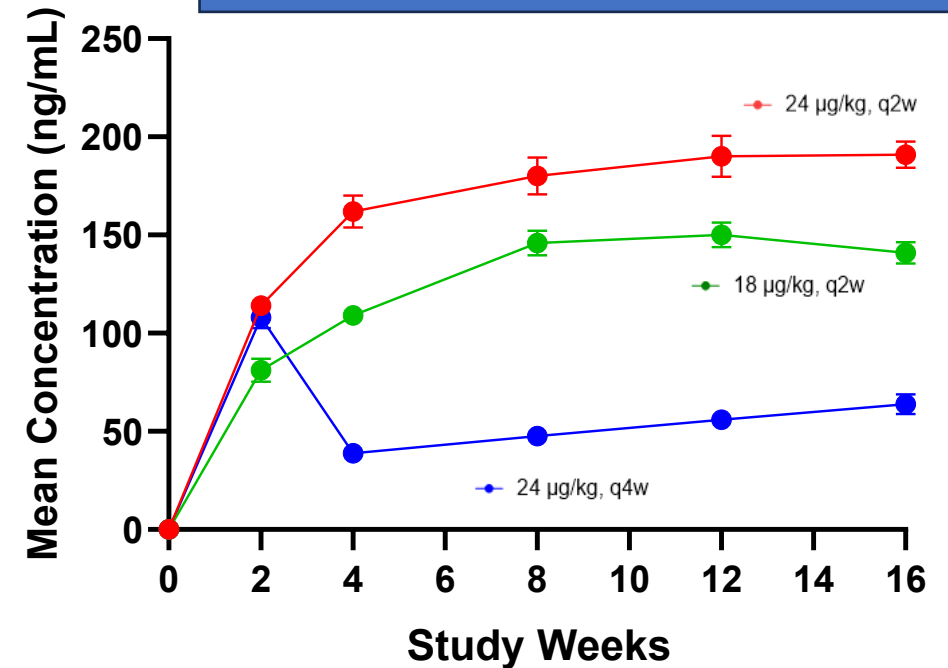
Strong relationship to dose-dependent clinical responses

Up to 6-fold increase in T-reg consistent with prior studies of REZPEG



▲ Administration q4w dosed on w0, w4, w8, w12, and w16

Dose-Dependent Pharmacokinetics



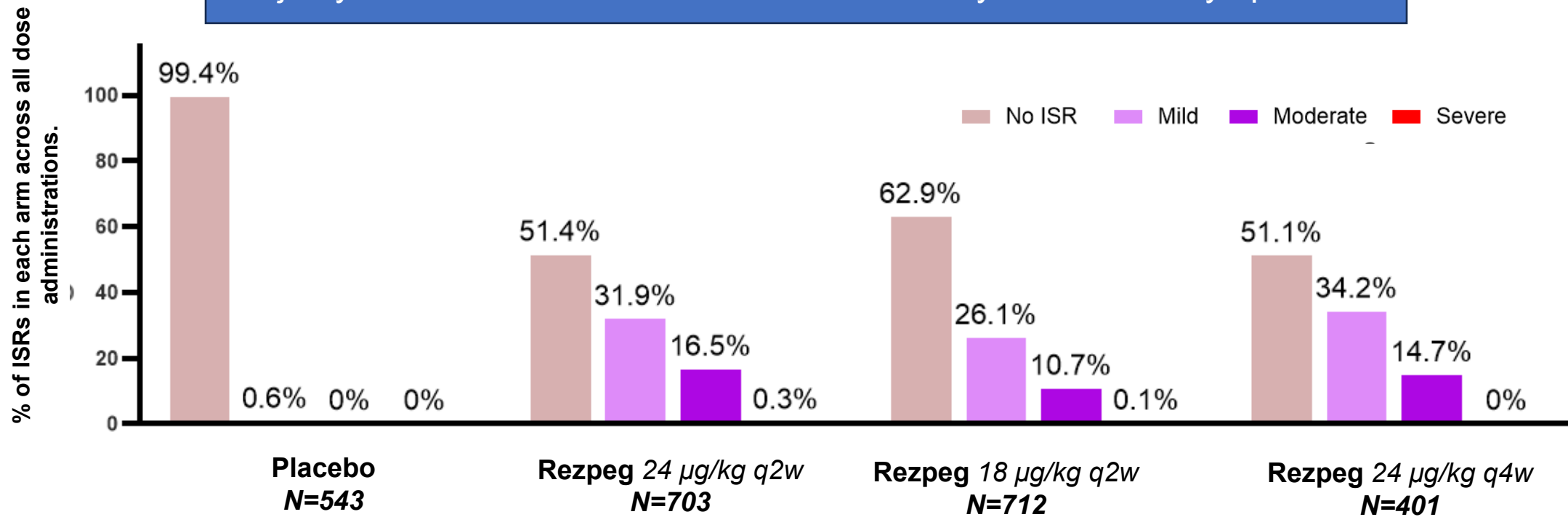
	0	2	3	4	5	8	12	16
N	95	83	39	85	37	79	75	75
N	102	84	45	84	41	82	74	71
N	107	84	31	78	38	81	83	78

REZOLVE-AD: Safety Summary

- **Safety of rezpeg for 16-week induction period in this Phase 2b study is consistent with previously observed and reported safety profile**
 - Serious and severe AEs were rare (1.6% and 3.1%, respectively) for rezpeg-exposed patients
 - Discontinuation rate due to AEs was low (5.6%) for rezpeg-exposed patients and was within the range of rates seen in contemporary Phase 2b studies
 - No imbalance to suggest an increased risk of infection over placebo
- **No increased risk of conjunctivitis, oral ulcers, or infections, including oral herpes, in study treatment arms**
- **The most frequently observed adverse event was injection site reactions (ISRs)**
 - Nearly all were mild-moderate in severity and self-resolving
 - The treatment discontinuation rate due to ISRs was very low (0.6%) for rezpeg exposed patients
 - Planning ISR mitigation strategy for commercialization

ISR Severity Breakdown Across All Dose Administrations By Severity Level Over 16-Week Induction

Majority of ISRs observed were mild with faint erythema and asymptomatic



N= number of study treatment or placebo administrations in arm (at 24 µg/kg q4w, only rezpeg administrations counted)

Mild: Faint erythema, asymptomatic, no or mild itch, no or mild tenderness

Moderate: Notable/great erythema, widespread itch, readily apparent induration, moderate pain

Severe: Widespread and constant itch limiting daily life, gross deviation of normal anatomic contour for induration, severe pain

Phase 2b Benchmarks

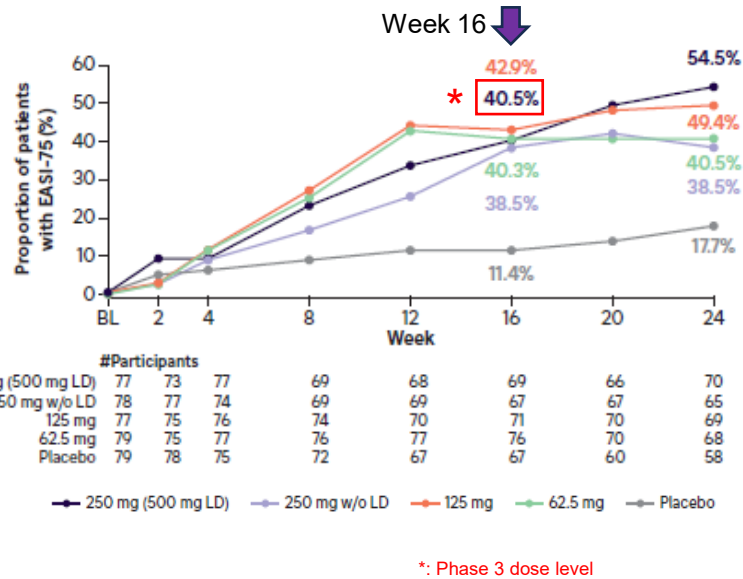
Endpoint	Repegaldesleukin 18/24 µg/kg q2w Phase 2b 16 Weeks Nektar	Amlitelimab 250mg q4w ¹ Phase 2b 16/24 Weeks Sanofi	Rocatinlimab 150/600mg q4w ² Phase 2b 16 Weeks Amgen	Nemolizumab 30mg q4w ³ Phase 2b 24 Weeks (TCS Combo) Galderma	Lebrikizumab 250mg q2w ⁴ Phase 2b 16 Weeks Lilly/Dermira	Tralokinumab 300mg q2w ⁵ Phase 2b 12 Weeks (TCS Combo) Leo Pharma	Dupilumab 300mg q2w ⁶ Phase 2b 16 Weeks Regeneron
MOA	IL-2R agonist	OX40L	OX40	IL-31	IL-13	IL-13	IL-4 & IL-13
Enrollment Completion Trial Size	2025 N=398	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%)	62% (29%)	62/60% (32%)	69% (52%)	72% (41%)	Not Reported	68% (18%)
Placebo Adjusted	27/30%	32%	30/28%	17%	31%	Not Reported	50%
EASI-75 (Placebo)	46/42% (17%)	40% (11%)	44/40% (11%)	46% (26%)	48% (12%) ^b	43% (16%)	52% (11%) ^d
Placebo Adjusted	29/25%	29%	33/29%	20%	36%	27%	41%
vIGA-AD Responders (0/1) (Placebo)	26/20% (8%)	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%	17%	17/13%	16%	30%	15%	28%
EASI-90 (Placebo)	18/25% (9%)	16% (4%)	19/12% (4%)	30% (11%)	44% (11%)	Not Reported	30% (4%) ^d
Placebo Adjusted	9/16%	12%	15/8%	19%	33%	Not Reported	26%
Itch NRS ≥ 4 pt Responders (Placebo)	35/42% (16%)	25% (5%)	37/46% (19%)	43% (24%) ^a	67% (39%) ^c	Not Reported	41% (8%) ^e
Placebo Adjusted	19/26%	20%	18/27%	19%	28%	Not Reported	33%

Benchmark to Phase 2b OX40/OX40L Class

Endpoint	Rezpegaldesleukin 18/24 µg/kg q2w Phase 2b 16 Weeks Nektar	Amlitelimab 250mg q4w ¹ Phase 2b 16 Weeks Sanofi	Rocatinlimab 150/600mg q4w ² Phase 2b 16 Weeks Amgen
MOA	IL-2R agonist	OX40L	OX40
Enrollment Completion Trial Size	2025 N=398	2022 N=390	2020 N=274
EASI LS Mean % reduction from baseline (Placebo)	58/61% (31%)	62% (29%)	62/60% (32%)
Placebo Adjusted	27/30%	32%	30/28%
EASI-75 (Placebo)	46/42% (17%)	40% (11%)	44/40% (11%)
Placebo Adjusted	29/25%	29%	33/29%
vIGA-AD Responders (0/1) (Placebo)	26/20% (8%)	22% (5%) IGA	19/15% (2%)
Placebo Adjusted	18/12%	17%	17/13%%
EASI-90 (Placebo)	18/25% (9%)	16% (4%)	19/12% (4%)
Placebo Adjusted	9/16%	12%	15/8%
Itch NRS ≥ 4 pt Responders (Placebo)	35/42% (16%)	25% (5%)	37/46% (19%)
Placebo Adjusted	19/26%	20%	18/27%

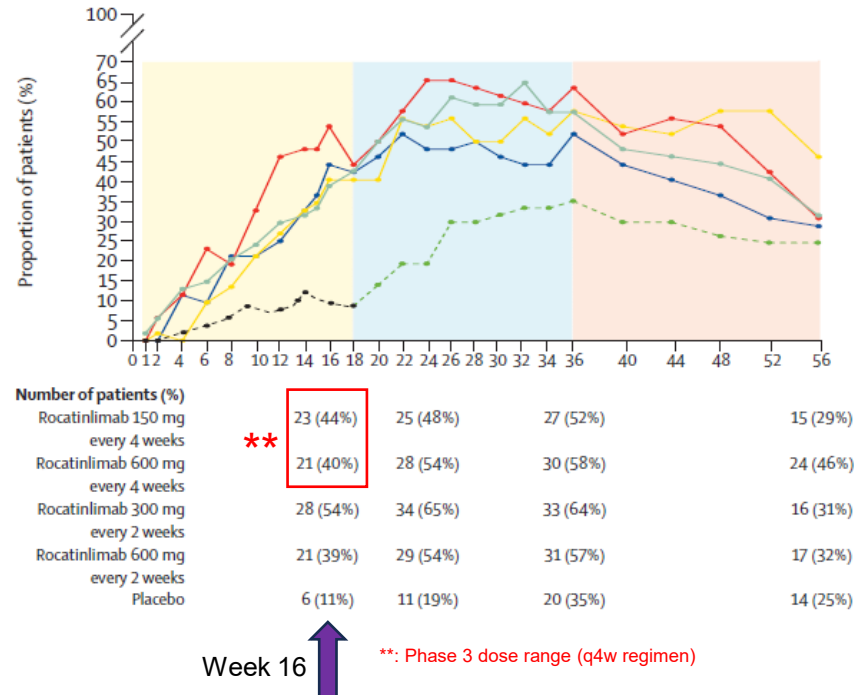
Rezpeg q2w Arms Show Similar Responses as OX40/OX40L Class (EASI-75 at Week 16)

Amlitelimab

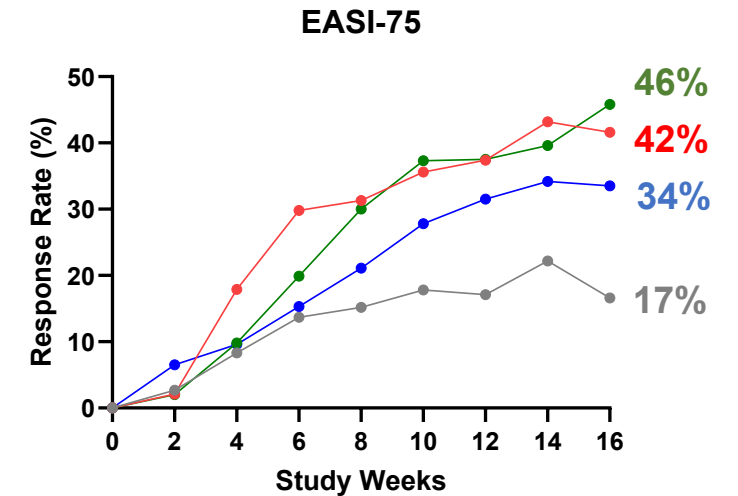


#Participants	250 mg (500 mg LD)	250 mg w/o LD	125 mg	62.5 mg	Placebo
250 mg (500 mg LD)	77	73	77	69	68
250 mg w/o LD	78	77	74	69	67
125 mg	77	75	76	74	70
62.5 mg	79	75	77	76	77
Placebo	79	78	75	72	67

Rocatinlimab



Rezpegaldesleukin



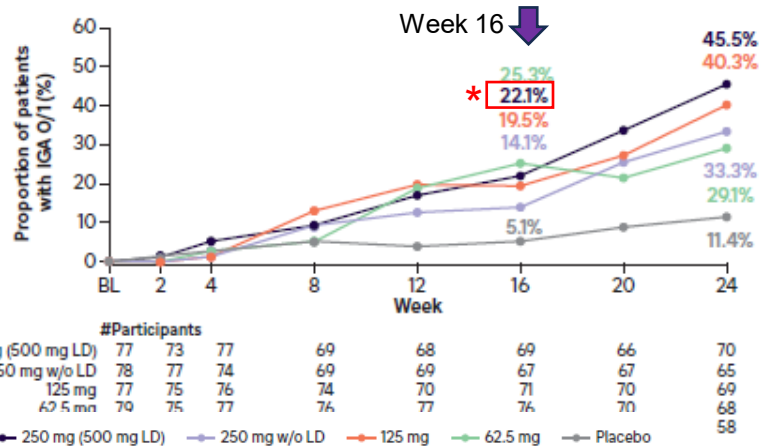
References:

Amlitelimab – Weidinger et al. 5th Inflammatory Skin Disease Summit, November 15-18; Vienna Austria (Fig. 3)

Rocatinlimab – Guttman-Yassky et al. 2023 Lancet 401:204-14 (Fig 2c)

Rezpeg q2w Arms Show Similar Responses as OX40/OX40L Class (vIGA-AD 0/1 at Week 16)

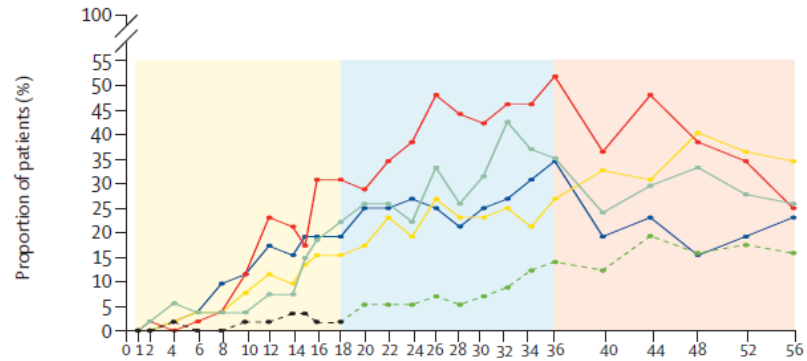
Amlitelimab



#Participants	77	73	77	69	68	69	66	70
250 mg (500 mg LD)	77	73	77	69	68	69	66	70
250 mg w/o LD	78	77	74	69	69	67	67	65
125 mg	77	75	76	74	70	71	70	69
62.5 mg	79	75	77	76	77	74	70	68

*: Phase 3 dose level

Rocatinlimab



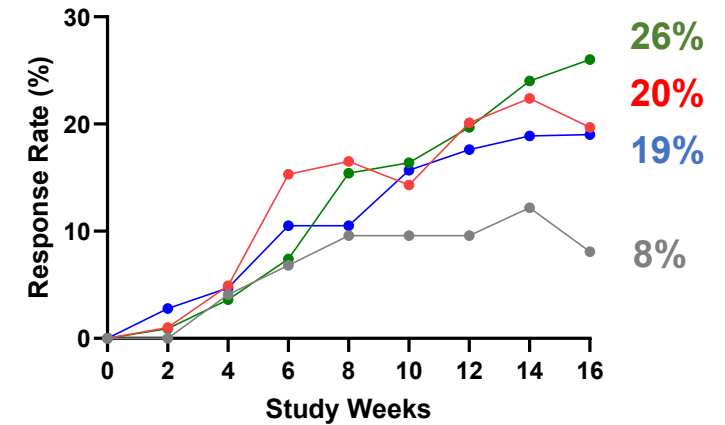
Number of patients (%)	Study week	16	20	24	28
Rocatinlimab 150 mg every 4 weeks	**	10 (19%)	14 (27%)	18 (35%)	12 (23%)
Rocatinlimab 600 mg every 4 weeks		8 (15%)	10 (19%)	14 (27%)	18 (35%)
Rocatinlimab 300 mg every 2 weeks		16 (31%)	20 (39%)	27 (52%)	13 (25%)
Rocatinlimab 600 mg every 2 weeks		10 (19%)	12 (22%)	19 (35%)	14 (26%)
Placebo		1 (2%)	3 (5%)	8 (14%)	9 (16%)

Week 16

** : Phase 3 dose range (q4w regimen)

Rezpegaldesleukin

vIGA-AD 0/1



References:

Amlitelimab – Weidinger et al. 5th Inflammatory Skin Disease Summit, November 15-18; Vienna Austria (Fig. 3)

Rocatinlimab – Guttman-Yassky et al. 2023 Lancet 401:204-14 (Fig 2c)



**Jonathan Silverberg, MD,
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Director of Clinical Research and
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Dr. Silverberg is Professor of Dermatology at The George Washington University School of Medicine and Health Sciences in Washington, DC. He is the Director of Clinical Research and Contact Dermatitis. Dr. Silverberg's area of clinical subspecialty is inflammatory skin disease, particularly atopic and contact dermatitis. Dr. Silverberg has also been a local, national and/or international principal investigator for numerous clinical trials for novel treatments in atopic dermatitis and other inflammatory disorders. Dr. Silverberg's research interests include drug development, clinical trial design, biomarkers, dermatology-epidemiology, health services research, patient-reported outcomes, comorbidities and burden of itch and inflammatory skin disease and evidence-based dermatology. His publications include more than 1000 peer-reviewed articles, abstracts and book chapters. He is an associate editor for the Journal of the American Academy of Dermatology, British Journal of Dermatology and Current Dermatology Reports.



David Rosmarin, MD

Chair of the Department of
Dermatology at Indiana University
School of Medicine

Kampen-Norins Scholar in
Dermatology

Dr. Rosmarin is Chair of the Department of Dermatology at Indiana University and is Kampen-Norins Scholar in Dermatology. He is nationally recognized and serves as a referral for physicians with difficult to manage inflammatory diseases such as atopic dermatitis. Previously, Dr. Rosmarin served as the Director of the Clinical Trials Unit in the Department of Dermatology at Tufts Medical Center. His research interests focus on development of novel therapeutics and investigating novel uses of established therapies, with a particular focus on chronic skin diseases such as atopic dermatitis, vitiligo, discoid lupus, and hidradenitis suppurativa. For his training, Dr. Rosmarin went to medical school at NYU, dermatology residency at Boston University-Tufts combined training program, and fellowship at Brigham and Women's Hospital.

REZOLVE-AD Phase 2b Validates Rezpeg as a First-in-Class Novel T-Regulatory Mechanism in Atopic Dermatitis

Novel T-Reg MOA differentiates from existing and in-development biologics

- Only T-regulatory mechanism to show compelling efficacy data across all endpoints in a large Phase 2b study
- Treg fold increase up to 6-fold
- Clear reduction in numerous AD markers of IL-19, TARC/CCL17, Periostin, MDC/CCL22

Highest dose arm significant on primary and all key secondary endpoints

- Fast onset of clinical benefit, observed within first several doses
- Clear dose-dependent efficacy across multiple dose arms
- No drop-off in treatment effect in severe AD population

Safety consistent with previously reported safety results

- No increased risk of conjunctivitis or oral herpes as found with other biologics and JAKis
- Most frequent observed AEs were mild injection site reactions (ISRs) that were self-resolving
- Less than 1% of discontinuations due to ISRs

- Rezpeg has potential for longstanding clinical benefit (remittive effect)
- Phase 2b results support advancement of a novel, first-in-class Treg-based immune-balancing mechanism

Rezpeg 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**

Beyond Rezpeg in Atopic Dermatitis, Potential Blockbuster Expansions for Treg MOA



Dermatology

Alopecia areata (Phase 2b underway, data December 2025)

Potential for expansion into vitiligo and other skin-related immune conditions



Immunology

Type 1 diabetes (Phase 2 starting in 2025)

Potential for expansion into systemic lupus and other auto-immune conditions



Second T Regulatory Cell Mechanism Entering Clinic in 2026

TNFR2 agonist antibody (planned IND 2026) with novel bispecifics combining TNFR2 agonism (Treg) with validated antibody mechanisms in auto-immune disease

Potential for development in Crohn's Disease, UC, MS



Appendix (data tables, additional materials)

REZOLVE-AD: Baseline Demographics

	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	Total N = 393
Age					
Mean (SD)	37.9 (14.39)	38.0 (13.73)	36.3 (15.41)	36.5 (14.30)	37.1 (14.44)
Median	35	36.5	31.5	33.5	34
Min, Max	18, 69	18, 70	18, 73	18, 69	18, 73
Age Category					
<65 yrs	70 (95.9%)	101 (97.1%)	100 (94.3%)	103 (93.6%)	374 (95.2%)
> = 65 yrs	3 (4.1%)	3 (2.9%)	6 (5.7%)	7 (6.4%)	19 (4.8%)
Sex					
Female	35 (47.9%)	49 (47.1%)	56 (52.8%)	63 (57.3%)	203 (51.7%)
Male	38 (52.1%)	55 (52.9%)	50 (47.2%)	47 (42.7%)	190 (48.3%)
Race					
White	58 (79.5%)	87 (83.7%)	90 (84.9%)	96 (87.3%)	331 (84.2%)
Black or African American	2 (2.7%)	7 (6.7%)	3 (2.8%)	5 (4.5%)	17 (4.3%)
Asian	9 (12.3%)	9 (8.7%)	11 (10.4%)	7 (6.4%)	36 (9.2%)
Other or not reported or Unknown	4 (5.5%)	1 (1.0%)	2 (1.9%)	2 (1.8%)	9 (2.3%)

REZOLVE-AD: Baseline Demographics

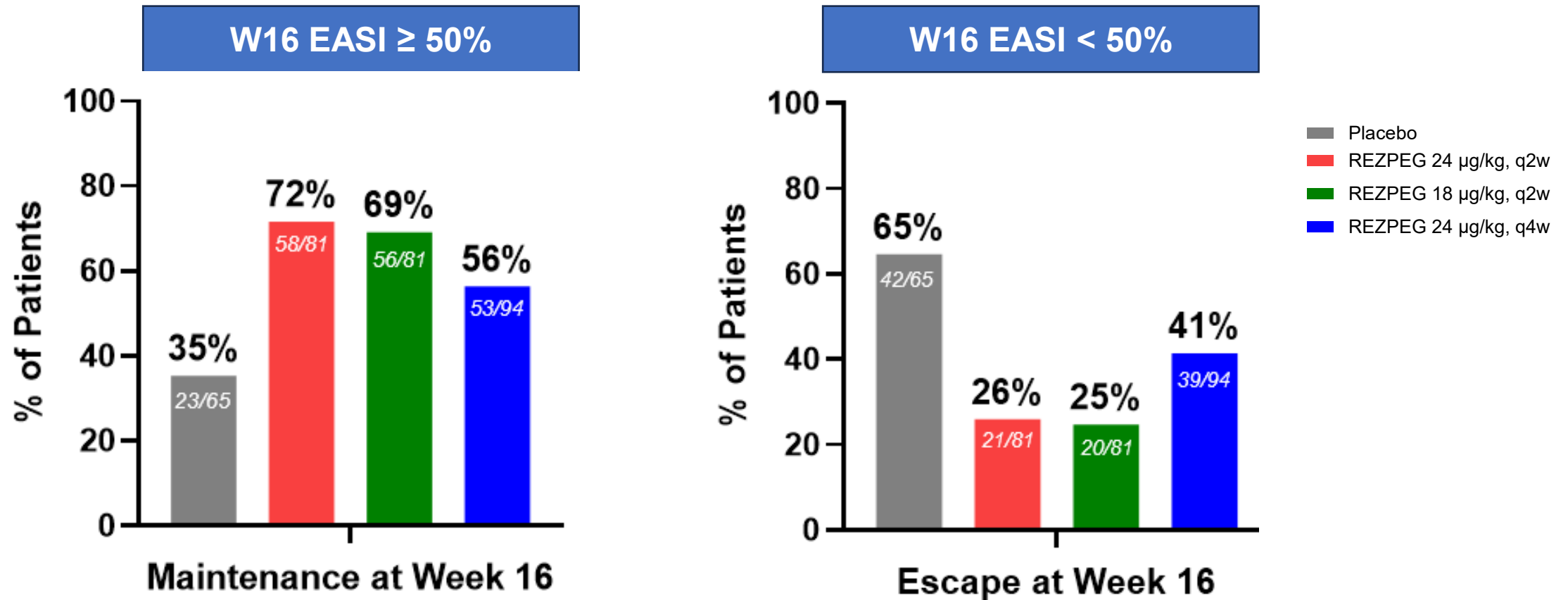
Country and Region Distribution

	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	Total N = 393
Country					
Australia	3 (4.1%)	3 (2.9%)	4 (3.8%)	8 (7.3%)	18 (4.6%)
Bulgaria	7 (9.6%)	8 (7.7%)	9 (8.5%)	16 (14.5%)	40 (10.2%)
Canada	7 (9.6%)	10 (9.6%)	14 (13.2%)	13 (11.8%)	44 (11.2%)
Croatia	1 (1.4%)	4 (3.8%)	0	1 (0.9%)	6 (1.5%)
Czechia	9 (12.3%)	7 (6.7%)	8 (7.5%)	9 (8.2%)	33 (8.4%)
Germany	8 (11.0%)	12 (11.5%)	10 (9.4%)	8 (7.3%)	38 (9.7%)
Hungary	0	0	0	1 (0.9%)	1 (0.3%)
Poland	21 (28.8%)	41 (39.4%)	42 (39.6%)	36 (32.7%)	140 (35.6%)
Spain	3 (4.1%)	2 (1.9%)	4 (3.8%)	0	9 (2.3%)
USA	14 (19.2%)	17 (16.3%)	15 (14.2%)	18 (16.4%)	64 (16.3%)
STRATA 1 for Region					
North America (USA, Canada)	21 (28.8%)	27 (26.0%)	29 (27.4%)	31 (28.2%)	108 (27.5%)
Rest of the world	52 (71.2%)	77 (74.0%)	77 (72.6%)	79 (71.8%)	285 (72.5%)

REZOLVE-AD: Baseline Disease Levels Balanced Across Arms

	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	Total N = 393
STRATA 1 for vIGA-AD					
3	51 (69.9%)	71 (68.3%)	70 (66.0%)	75 (68.2%)	267 (67.9%)
4	22 (30.1%)	33 (31.7%)	36 (34.0%)	35 (31.8%)	126 (32.1%)
EASI Total Score (0-72)					
Mean (SD)	25.2 (8.57)	25.4 (9.14)	27.2 (10.40)	26.1 (10.45)	26.0 (9.77)
Median	23.5	23	23.8	22.6	23.4
Min, Max	16.0, 59.1	16.2, 59.6	16.3, 62.0	16.1, 66.2	16.0, 66.2
EASI Total Score Category I					
<18	19 (26.0%)	22 (21.2%)	16 (15.1%)	24 (21.8%)	81 (20.6%)
>= 18	54 (74.0%)	82 (78.8%)	90 (84.9%)	86 (78.2%)	312 (79.4%)
EASI Total Score Category II					
<21	29 (39.7%)	44 (42.3%)	43 (40.6%)	44 (40.0%)	160 (40.7%)
>= 21	44 (60.3%)	60 (57.7%)	63 (59.4%)	66 (60.0%)	233 (59.3%)

REZOLVE-AD: Majority of Rezpeg-Treated Patients Continued into Maintenance



Percentages are calculated using completers at week 16 as denominator

REZOLVE-AD: Patient Populations and Disposition

	Placebo q2w	Rezpeg 24 µg/kg q2w	Rezpeg 18 µg/kg q2w	Rezpeg 24 µg/kg q4w	Total
Intent to Treat (ITT)	74	106	107	111	398
Modified Intent to Treat (MITT)	73	104	106	110	393
Discontinued before W16	8 (11.0%)	23 (22.1%)	25 (23.6%)	16 (14.5%)	72 (18.3%)
Completed W16 induction	65 (89.0%)	81 (77.9%)	81 (76.4%)	94 (85.5%)	321 (81.7%)
Continued to Maintenance (W16)	23 (31.5%)	58 (55.8%)	56 (52.8%)	53 (48.2%)	190 (48.3%)
Continue study to Escape (W16)	42 (57.5%)	21 (20.2%)	20 (18.9%)	39 (35.5%)	122 (31.0%)
Discontinued at W16	0	2 (1.9%)	5 (4.7%)	2 (1.8%)	9 (2.3%)

Discontinuation rates for all rezpeg arms comparable to treatment arms in Phase 2b studies for approved and late-stage biologics (others range from 3 – 24%)*

MITT Population count is used as the denominator to calculate the percentages in this table.

REZOLVE-AD: Treatment Discontinuation Reasons During 16-Week Induction

	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 Overall N = 320	Total N = 393
Discontinued before Week 16	8 (11.0%)	23 (22.1%)	25 (23.6%)	16 (14.5%)	64 (20.0%)	72 (18.3%)
Adverse Event	0	7 (6.7%)	5 (4.7%)	4 (3.6%)	16 (5.0%)	16 (4.1%)
ISR subset	0	1 (1.0%)	0	1 (0.9%)	2 (0.6%)	2 (0.5%)
Non-Compliance with Study Procedure	0	0	0	1 (0.9%)	1 (0.3%)	1 (0.3%)
Patient Decision	7 (9.6%)	14 (13.5%)	19 (17.9%)	9 (8.2%)	42 (13.1%)	49 (12.5%)
Lack of efficacy to study treatment	0	1 (1.0%)	0	1 (0.9%)	2 (0.6%)	2 (0.5%)
Other	1 (1.4%)	1 (1.0%)	1 (0.9%)	1 (0.9%)*	3 (0.9%)	4 (1.0%)
Discontinued at Week 16	0	2 (1.9%)	5 (4.7%)	2 (1.8%)	9 (2.8%)	9 (2.3%)
EASI-50 Responder	0	1 (1.0%)	4 (3.8%)	1 (0.9%)	6 (1.9%)	6 (1.5%)
Patient Decision	0	1 (1.0%)	3 (2.8%)	0	4 (1.3%)	4 (1.0%)
EASI-50 Non-responder	0	1 (1.0%)	1 (0.9%)	1 (0.9%)	3 (0.9%)	3 (0.8%)
Adverse Event	0	1 (1.0%)	0	0	1 (0.3%)	1 (0.3%)

*One patient cited reason for other specified as AE.

Overall discontinuation rates due to AEs for treatment arms across Phase 2b studies for approved and late-stage biologic therapies range from 0 – 15%*

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%)
Injection site reaction	3 (4.1%)	79 (76.0%)	66 (62.3%)	78 (70.9%)	223 (69.7%)
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%)



*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.

REZOLVE-AD: Serious TEAEs and TRAEs (All Cases)

16-Week Induction Period

System Organ Class Preferred Term	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 Overall N = 320	
	TEAE	TRAE	TEAE	TRAE	TEAE	TRAE	TEAE	TRAE	TEAE	TRAE
Patients With at Least One Serious AE	0	0	1 (1.0%)	0	4 (3.8%)	2 (1.9%)	0	0	5 (1.6%)	2 (0.6%)
Infections and infestations	0	0	1 (1.0%)	0	1 (0.9%)	1 (0.9%)	0	0	2 (0.6%)	1 (0.3%)
Gastroenteritis viral	0	0	1 (1.0%)	0	0	0	0	0	1 (0.3%)	0
Tonsillitis	0	0	0	0	1 (0.9%)	1 (0.9%)	0	0	1 (0.3%)	1 (0.3%)
Immune system disorders	0	0	0	0	1 (0.9%)	1 (0.9%)	0	0	1 (0.3%)	1 (0.3%)
Drug hypersensitivity	0	0	0	0	1 (0.9%)	1 (0.9%)	0	0	1 (0.3%)	1 (0.3%)
Nervous system disorders	0	0	0	0	1 (0.9%)	0	0	0	1 (0.3%)	0
Cerebrovascular accident	0	0	0	0	1 (0.9%)	0	0	0	1 (0.3%)	0
Skin and subcutaneous tissue disorders	0	0	0	0	1 (0.9%)	0	0	0	1 (0.3%)	0
Worsening atopic dermatitis	0	0	0	0	1 (0.9%)	0	0	0	1 (0.3%)	0

System organ class and preferred terms are coded based on the MedDRA v26.0. TEAEs in Induction are defined as AEs that start on or after the first dose date after randomization and up to one day before first dose date in maintenance or escape if patients receive at least one dose in maintenance or escape, or 30 days after last dose if patients EOT, otherwise, no ending date.

REZOLVE-AD: Severe TEAEs and TRAEs in ≥ 2 Patients in Any Arm

16-Week Induction Period

System Organ Class Preferred Term	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 Overall N = 320	
	TEAE	TRAE	TEAE	TRAE	TEAE	TRAE	TEAE	TRAE	TEAE	TRAE
Patients With at Least One Severe AE	1 (1.4%)	0	3 (2.9%)	3 (2.9%)	6 (5.7%)	3 (2.8%)	1 (0.9%)	0	10 (3.1%)	6 (1.9%)
General disorders and administration site conditions	0	0	3 (2.9%)	3 (2.9%)	2 (1.9%)	2 (1.9%)	0	0	5 (1.6%)	5 (1.6%)
Injection site reaction	0	0	2 (1.9%)	2 (1.9%)	1 (0.9%)	1 (0.9%)	0	0	3 (0.9%)	3 (0.9%)
Chest pain	0	0	0	0	1 (0.9%)	1 (0.9%)	0	0	1 (0.3%)	1 (0.3%)
Pyrexia	0	0	1 (1.0%)	1 (1.0%)	0	0	0	0	1 (0.3%)	1 (0.3%)
Skin and subcutaneous tissue disorders	1 (1.4%)	0	0	0	2 (1.9%)	0	0	0	2 (0.6%)	0
Worsening atopic dermatitis	1 (1.4%)	0	0	0	2 (1.9%)	0	0	0	2 (0.6%)	0
Immune system disorders	0	0	0	0	1 (0.9%)	1 (0.9%)	0	0	1 (0.3%)	0
Drug hypersensitivity	0	0	0	0	1 (0.9%)	1 (0.9%)	0	0	1 (0.3%)	0
Nervous system disorders	0	0	0	0	1 (0.9%)	0	0	0	1 (0.3%)	0
Cerebrovascular accident	0	0	0	0	1 (0.9%)	0	0	0	1 (0.3%)	0
Respiratory, thoracic and mediastinal disorders	0	0	0	0	0	0	1 (0.9%)	0	1 (0.3%)	0
Rhinitis allergic	0	0	0	0	0	0	1 (0.9%)	0	1 (0.3%)	0

Atopic Dermatitis Presents Potential 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 million¹

Adults with AD in U.S.



~220 million²

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

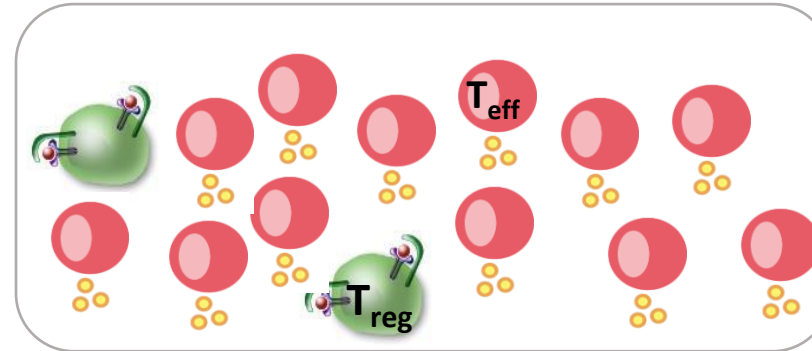
We believe there is a high unmet need for new mechanism of action with potential to:

- Offer dosing schedules without rebound effect
- Induce deep and potentially therapy-free remission
- Favorable safety and tolerability profile for ease of use

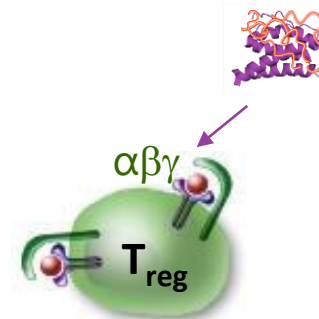
Dupixent®: current market leader in atopic dermatitis exceeding \$10.5B in annual sales, but 50% of patients fail on therapy^{5, 6}

Rezpegaldesleukin (Rezpeg) is Potential First-in-Class T-Regulatory Cell Mechanism to Restore Balance in Immune System

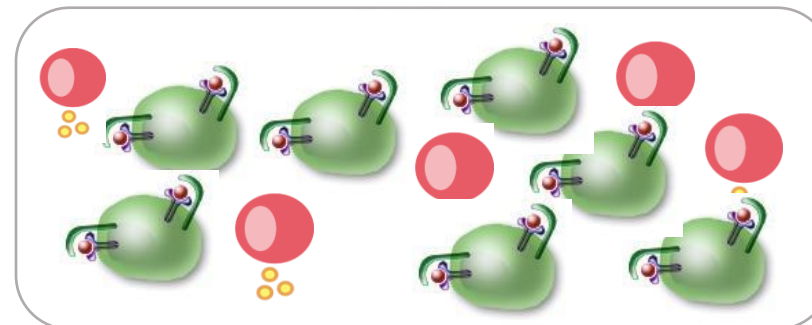
- Many patients with moderate-to-severe atopic dermatitis (AD) do not adequately achieve disease control or have safety/tolerability issues with current therapies
- Tregs play a central role in controlling AD by dampening inflammatory cytokines and overactive T-cells¹
- Rezpegaldesleukin is a potential T-cell balancing therapy that has been shown to^{2,3}:
 - **Enhance Treg numbers**
 - **Stimulate Tregs** thereby reducing proinflammatory cytokines
 - **Offer potential for long-term control** of over-active immune response



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



Rezpegaldesleukin acts on IL2 receptors to proliferate T-regulatory cells and restore their functionality



Treg expansion and activation restores the immunoregulatory balance

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