



Data from Phase 2b REZOLVE-AD and REZOLVE-AA Studies of Repegaldesleukin Presented at 2026 American Academy of Dermatology Annual Meeting

March 28, 2026

Repegaldesleukin demonstrates statistically significant improvement in mean percent EASI improvement across both moderate and severe atopic dermatitis patients

Repegaldesleukin proof-of-concept data in alopecia areata patients presented as a late-breaking research oral presentation

SAN FRANCISCO, March 28, 2026 /PRNewswire/ -- Nektar Therapeutics (Nasdaq: NKTR) today showcased data in two presentations at the 2026 American Academy of Dermatology (AAD) Annual Meeting taking place in Denver, CO.

At AAD 2026, data from the global Phase 2b REZOLVE-AD study in 393 patients with moderate-to-severe atopic dermatitis were presented by Dr. Raj Chovatiya, Associate Professor at Rosalind Franklin University of Medicine and Science Chicago Medical School and Founder and Director of the Center for Medical Dermatology and Immunology Research, in an oral poster session entitled "Novel Regulatory T-cell enhancing Biologic Repegaldesleukin: Phase 2b Efficacy, Safety, and Baseline Severity-Dependent Treatment Response in Moderate-to-Severe Atopic Dermatitis" [\[link to presentation\]](#).

Patient randomization was stratified based on baseline disease severity measured by vIGA-AD[®] (validated Investigator's Global Assessment for Atopic Dermatitis) and geographic region. As presented at AAD, patients in the Phase 2b REZOLVE-AD study demonstrated consistent reduction in mean Eczema Area and Severity Index (EASI) scores over the 16-week induction period as compared to placebo regardless of baseline disease severity as measured by baseline vIGA-AD[®] scores of 3 or 4. During the 16-week induction period, patients also achieved comparable EASI-75 (at least a 75% improvement in EASI score from baseline) and EASI-90 response (at least a 90% improvement in EASI score from baseline). These disease improvement metrics were also comparable by geographic region.

"The consistency of EASI responses with repegaldesleukin across baseline disease severity further differentiates it from the standard of care biologic treatment, which can have lower response rates in more severe patients as compared to moderate patients," said Raj Chovatiya, MD, PhD, MSCI, FAAD. "We believe its novel agonist mechanism to expand regulatory T cells, which act as master regulators upstream of the cytokine-specific blockade mechanisms of other biologics to address multiple pathways, allows a potentially more consistent improvement across a broader patient population."

Based upon results from the Phase 2b REZOLVE-AD study of repegaldesleukin, Nektar is planning to initiate the Phase 3 ZENITH-AD program of repegaldesleukin in moderate-to-severe atopic dermatitis patients in the second quarter of 2026.

At AAD 2026, Dr. David Rosmarin presented a late-breaking research oral presentation highlighting previously-released data¹ titled: "Novel Regulatory T-cell Enhancing Biologic Repegaldesleukin: Phase 2b Efficacy and Safety Results Following 36-Weeks of Therapy in Severe-to-Very-Severe Alopecia Areata" [\[link to presentation\]](#).

On the primary endpoint of mean Severity of Alopecia Tool (SALT) reduction at 36 weeks of treatment, high dose repegaldesleukin, 24 µg/kg every two weeks (q2w), demonstrated a mean reduction in the SALT score of 28.2% in the 24 µg/kg arm versus 11.2% in the placebo arm. Mean percent reduction in SALT scores at 36 weeks was 30% for both treatment arms versus 6% in the placebo arm, achieving statistical significance (p<0.05) when excluding four patients that did not meet major study eligibility criteria at baseline. Repegaldesleukin was well tolerated and its safety profile was consistent with previously reported results.

"The clear activity of repegaldesleukin in alopecia areata builds on prior results in atopic dermatitis and reinforces the broader potential of this approach across T cell-driven inflammatory diseases," said David Rosmarin M.D., Chair, Department of Dermatology and Associate Professor of Dermatology, Indiana University School of Medicine. "I look forward to the upcoming results from the 16-week treatment extension to evaluate the potential for a deepening of SALT response over time."

About REZOLVE-AD Phase 2b Study

The global REZOLVE-AD ([NCT06136741](#)) Phase 2b study enrolled 393 patients with moderate to severe atopic dermatitis who have not previously been treated with a JAK inhibitor or other biologic. Patients were randomized (3:3:3:2) to receive subcutaneous treatment with three doses of repegaldesleukin: a high dose of 24 µg/kg every two weeks (Q2W), a middle dose of 18 µg/kg every two weeks (Q2W), and a low dose of 24 µg/kg every four weeks (Q4W), or placebo Q2W. The primary endpoint and secondary endpoints were assessed at the end of the 16-week induction period. Following the induction period, repegaldesleukin-treated patients who achieved EASI percent reductions of at least 50 were re-randomized (1:1) to continue at the same dose level on a Q4W or a Q12W regimen through Week 52 in a blinded maintenance period. Placebo patients

with EASI percent score reductions of at least 50 continue to receive placebo Q4W.

About REZOLVE-AA Phase 2b Study

The global REZOLVE-AA ([NCT06340360](#)) Phase 2b study enrolled 92 patients with severe-to-very-severe alopecia areata who have not previously been treated with a JAK inhibitor or other biologic. Patients were randomized (3:3:2) to receive one of two rezpegaldesleukin doses or placebo, administered as a subcutaneous injection twice-monthly. The primary endpoint was the mean percentage reduction from baseline in the SALT score at 36 weeks. Following 36 weeks of treatment, patients who demonstrated hair growth but had not yet reached SALT>20 had the option to continue for an additional 16 weeks of treatment through 52 weeks in a blinded extension period. Primary and secondary endpoints were assessed at the end of the 36-week induction treatment period.

About Rezpegaldesleukin

Autoimmune and inflammatory diseases cause the immune system to mistakenly attack and damage healthy cells in a person's body. A failure of the body's self-tolerance mechanisms enables the formation of the pathogenic T lymphocytes that conduct this attack. Rezpegaldesleukin is a potential first-in-class resolution therapeutic that may address this underlying immune system imbalance in people with many autoimmune and inflammatory conditions. It targets the interleukin-2 receptor complex in the body to stimulate proliferation of immune-modulating cells known as regulatory T cells. By activating these cells, rezpegaldesleukin may act to bring the immune system back into balance.

In February 2025, the U.S. Food and Drug Administration (FDA) granted Fast Track designation for rezpegaldesleukin for the treatment of adult and pediatric patients 12 years of age and older with moderate-to-severe atopic dermatitis whose disease is not adequately controlled with topical prescription therapies or when those therapies are not advisable. In July 2025, the FDA granted Fast Track designation for rezpegaldesleukin for the treatment of severe alopecia areata (AA) in adults and pediatric patients 12 years of age and older who weigh at least 40 kg.

Rezpegaldesleukin is being developed as a self-administered injection for a number of autoimmune and inflammatory diseases. It is wholly owned by Nektar Therapeutics.

About Nektar Therapeutics

Nektar Therapeutics is a clinical-stage biotechnology company focused on developing treatments that address the underlying immunological dysfunction in autoimmune and chronic inflammatory diseases. Nektar's lead product candidate, rezpegaldesleukin (REZPEG, or NKTR-358), is a novel, first-in-class regulatory T cell stimulator being evaluated in one Phase 2b clinical trial in atopic dermatitis, one Phase 2b clinical trial in alopecia areata, and one Phase 2 clinical trial in Type 1 diabetes mellitus. Nektar's pipeline also includes a preclinical bivalent tumor necrosis factor receptor type II (TNFR2) antibody and bispecific programs, NKTR-0165 and NKTR-0166, and a modified hematopoietic colony stimulating factor (CSF) protein, NKTR-422.

Nektar is headquartered in San Francisco, California. For further information, visit www.nektar.com and follow us on [LinkedIn](#).

Cautionary Note Regarding Forward-Looking Statements

This press release contains forward-looking statements which can be identified by words such as: "plan," "develop," "potential," "expand," "address," "may" and similar references to future periods. Examples of forward-looking statements include, among others, statements regarding the therapeutic potential of, and future development plans for, rezpegaldesleukin, NKTR-0165, NKTR-0166, and NKTR-422. Forward-looking statements are neither historical facts nor assurances of future performance. Instead, they are based only on our current beliefs, expectations and assumptions regarding the future of our business, future plans and strategies, anticipated events and trends, the economy and other future conditions. Because forward-looking statements relate to the future, they are subject to inherent uncertainties, risks and changes in circumstances that are difficult to predict and many of which are outside of our control. Our actual results may differ materially from those indicated in the forward-looking statements. Therefore, you should not rely on any of these forward-looking statements. Important factors that could cause our actual results to differ materially from those indicated in the forward-looking statements include, among others: (i) our statements regarding the therapeutic potential of rezpegaldesleukin, NKTR-0165, NKTR-0166 and NKTR-422 are based on preclinical and clinical findings and observations and are subject to change as research and development continue; (ii) rezpegaldesleukin, NKTR-0165, NKTR-0166 and NKTR-422 are investigational agents and continued research and development for these drug candidates is subject to substantial risks, including negative safety and efficacy findings in future clinical studies (notwithstanding positive findings in earlier preclinical and clinical studies); (iii) rezpegaldesleukin, NKTR-0165, NKTR-0166 and NKTR-422 are in clinical development and the risk of failure is high and can unexpectedly occur at any stage prior to regulatory approval; (iv) data reported from ongoing clinical trials are necessarily interim data only and the final results will change based on continuing observations; (v) the timing of the commencement or end of clinical trials and the availability of clinical data may be delayed or unsuccessful due to regulatory delays, slower than anticipated patient enrollment, manufacturing challenges, changing standards of care, evolving regulatory requirements, clinical trial design, clinical outcomes, competitive factors, or delay or failure in ultimately obtaining regulatory approval in one or more important markets; (vi) a Fast Track designation does not increase the likelihood that rezpegaldesleukin will receive marketing approval in the United States; (vii) patents may not issue from our patent applications for our drug candidates, patents that have issued may not be enforceable, or additional intellectual property licenses from third parties may be required; and (viii) certain other important risks and uncertainties set forth in our Annual Report on Form 10-K filed with the Securities and Exchange Commission on March 13, 2026. Any forward-looking statement made by us in this press release is

based only on information currently available to us and speaks only as of the date on which it is made. We undertake no obligation to update any forward-looking statement, whether written or oral, that may be made from time to time, whether as a result of new information, future developments or otherwise.

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1. Nektar Therapeutics, "REZOLVE-AA Phase 2b Study of Rezpegaldesleukin Establishes Proof-of-Concept in Patients with Severe-to-Very-Severe Alopecia Areata", press release, 12/16/2025, <https://ir.nektar.com/news-releases/news-release-details/rezolve-aa-phase-2b-study-rezpegaldesleukin-establishes-proof>

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