Br J Dermatol 2025; **00**:1–12
https://doi.org/10.1093/bjd/ljaf347
Advance access publication date: 2 September 2025

BJD
British Journal of Dermatology
Clinical Trial

Rademikibart monotherapy for moderate-to-severe atopic dermatitis in a 1-year, randomized phase II trial (SEASIDE CHINA): initial 2-week dosing, followed by 2-week or 4-week dosing

Jianzhong Zhang[1], **Jonathan I Silverberg**[2], **Jiawang Guo**[3], **Jili Yun**[3], **Wuban Pan**[3], **Zheng Wei**[4] and **Raúl Collazo**[4]

[1]Peking University People's Hospital, Department of Dermatology, Beijing, China
[2]Department of Dermatology, George Washington University School of Medicine and Health Sciences, Washington, DC, USA
[3]Suzhou Connect Biopharmaceuticals Ltd, Taicang, China
[4]Connect Biopharma LLC, San Diego, CA, USA

Correspondence: Raúl Collazo. Email: rcollazo@connectpharm.com

Abstract

Background Rademikibart (CBP-201) is a potent, next-generation human monoclonal antibody optimized to bind with high affinity to interleukin-4 receptor subunit alpha.

Objectives To evaluate rademikibart efficacy and safety, initially dosed every other week (Q2W), and Q2W or every fourth week (Q4W) from week 16, in Chinese adults/adolescents with moderate-to-severe atopic dermatitis (AD).

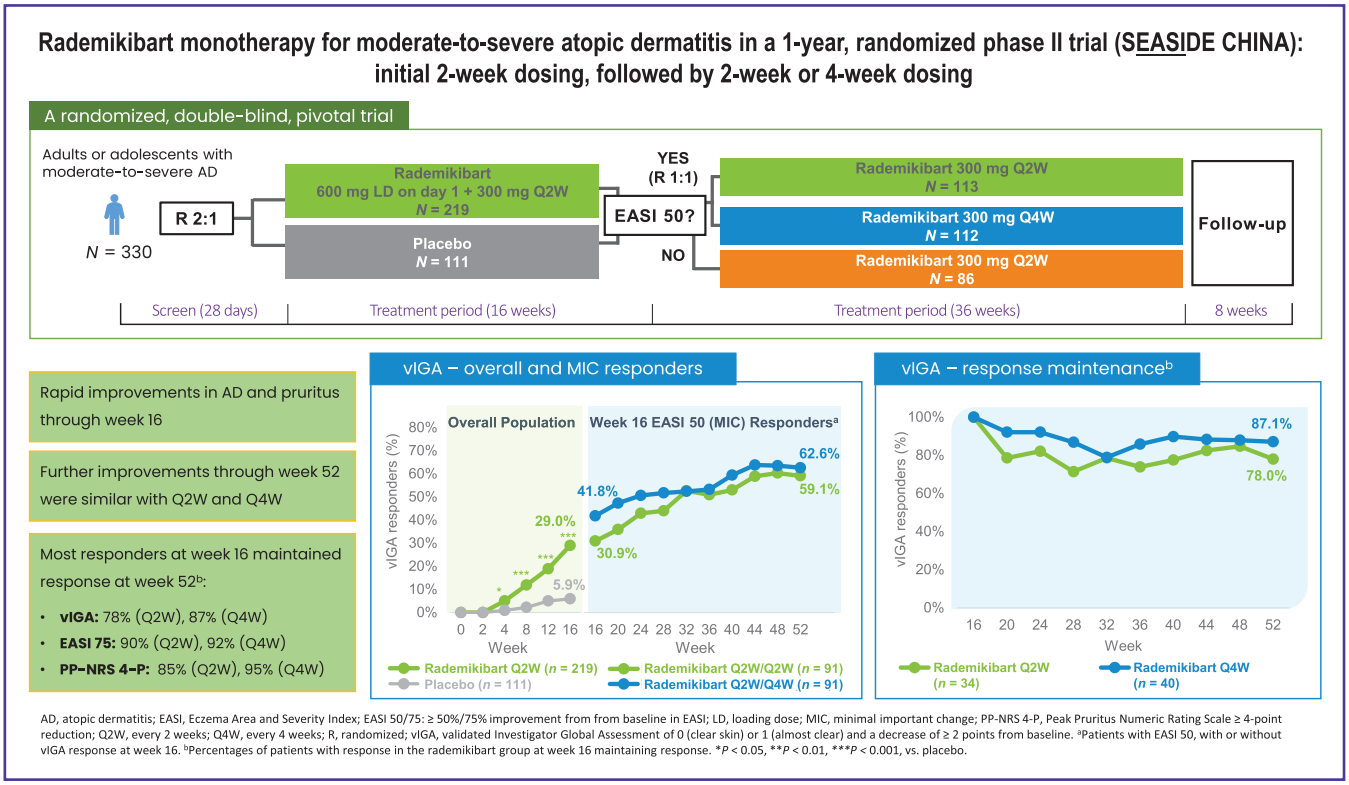
Methods SEASIDE CHINA (NCT05017480) was a phase II randomized trial: stage 1 was a 16-week treatment period, and stage 2 comprised 36-week treatment and 8-week follow-up periods. The primary endpoint was the proportion of patients with a validated Investigator Global Assessment Scale for AD score of 0 or 1 (vIGA 0/1) and ≥2-point reduction from baseline at week 16. Overall, 330 patients were randomized 2 : 1 double-blind to receive subcutaneous rademikibart 600 mg on day 1 and 300 mg Q2W from week 2–14, or placebo. Predose at week 16, patients were assessed for minimal important change [≥ 50% improvement from baseline in Eczema Area and Severity Index score (EASI 50)] and rerandomized 1 : 1 double-blind to rademikibart 300 mg Q2W or Q4W.

Results In stage 1, 29.0% of patients attained vIGA 0/1 and ≥2-point reduction with rademikibart Q2W ($P<0.001$) at week 16, without plateauing, vs. 5.9% with placebo. Rapid, significant improvements were also observed with all other rating scales in stage 1, including ≥75% improvement from baseline in EASI (EASI 75) (58.6% vs. 22.6%) and ≥4-point reduction in Peak Pruritus Numeric Rating Scale (PP-NRS) (36.3% vs. 10.5%) with rademikibart Q2W vs. placebo, respectively, at week 16. In stage 2, week 16 EASI 50 responders from stage 1 continued to improve through week 52 when treated with rademikibart Q2W or Q4W, including vIGA response (Q2W 59.1%; Q4W 62.6%), EASI 75 (Q2W 84.6%; Q4W 84.8%), and ≥4-point reduction in PP-NRS (Q2W 60.4%; Q4W 70.3%). Most patients with improvements during rademikibart Q2W therapy at week 16 maintained these responses through week 52 in both the rademikibart Q2W and Q4W groups: vIGA (Q2W 78.0%; Q4W 87.1%), EASI 75 (Q2W 90.1%; Q4W 92.3%) and ≥4-point reduction in PP-NRS (Q2W 85.3%; Q4W 94.8%). Injection site reactions were grade 1 (mild). No serious treatment-emergent adverse events (TEAEs) were treatment related. Three patients discontinued rademikibart owing to TEAEs (AD flare, vitiligo, pregnancy).

Conclusions Rademikibart Q2W induced rapid improvements in AD lesions and pruritus during the initial 16 weeks, which were maintained/improved further with rademikibart Q2W or Q4W across an additional 36 weeks. Rademikibart Q2W and Q4W were similarly efficacious and well tolerated. These findings are compatible with those from the published international phase II rademikibart trial, CBP-201-WW001.

Accepted: 26 August 2025

Graphical Abstract



AD, atopic dermatitis; EASI, Eczema Area and Severity Index; EASI 50/75: ≥ 50%/75% improvement from from baseline in EASI; LD, loading dose; MIC, minimal important change; PP-NRS 4-P, Peak Pruritus Numeric Rating Scale ≥ 4-point reduction; Q2W, every 2 weeks; Q4W, every 4 weeks; R, randomized; vIGA, validated Investigator Global Assessment of 0 (clear skin) or 1 (almost clear) and a decrease of ≥ 2 points from baseline. [a]Patients with EASI 50, with or without vIGA response at week 16. [b]Percentages of patients with response in the rademikibart group at week 16 maintaining response. *P < 0.05, **P < 0.01, ***P < 0.001, vs. placebo.

Lay summary

Atopic dermatitis (AD) is a common type of eczema, which can affect people of any age. People with moderate-to-severe AD can experience very sore and itchy skin. In some cases, they can develop depression and other mental health conditions. As the impact of eczema can be so deep, we need new medications that are effective and convenient to use.

We carried out this clinical trial in China. We investigated a possible new medication, rademikibart, in adults and adolescents with moderate-to-severe AD. There were 330 participants in the trial, who were treated for 1 year. Stage 1 lasted 16 weeks. Participants received either rademikibart or a placebo (an inactive lookalike drug). The drug or placebo was injected every 2 weeks through week 14. Stage 2 was from week 16. Participants received rademikibart either every 2 weeks or every 4 weeks. We found that most participants had great improvements in their AD during treatment with rademikibart. Their AD symptom improvements happened quickly, in the first few days and weeks. Improvements continued across 1 year of treatment. No serious adverse events were treatment related. Three patients did discontinue rademikibart due to adverse events: an AD flare, vitiligo (white patches on the skin) or pregnancy.

In our trial, AD symptoms improved for most participants during long-term treatment with rademikibart. This included relief from itching. Importantly, people had large improvements with rademikibart given once every 4 weeks, a convenient interval. However, a larger clinical trial is needed to further investigate rademikibart.

What is already known about this topic?

- Atopic dermatitis (AD) therapies are associated with loss of response.
- Rademikibart is optimized to bind to interleukin-4 receptor subunit alpha epitopes with high affinity.
- In a published international phase II trial, rademikibart resulted in rapid improvements in AD across a 16-week treatment period, when dosed every other week (Q2W) or every fourth week (Q4).

What does this study add?

- Rademikibart resulted in rapid improvements in eczematous lesions and pruritus through week 16 when dosed Q2W in Chinese patients.
- From weeks 16–52, patients experienced further improvements and ~90% of patients maintained their response, with similar efficacy when dosed Q2W or Q4W.
- Rademikibart may provide rapid and long-lasting relief from AD signs and symptoms with convenient dosing regimens.

Atopic dermatitis (AD) is a chronic inflammatory skin disease that affects a large proportion of the global population. One-year prevalence estimates for countries around the world range from 1–18% for adults, without clear geographical patterns, including ~6% in Chinese populations.[1–5] In China, an estimated 11.6 million adults and adolescents had AD in 2010.[6]

Moderate-to-severe AD manifests as recurrent eczematous lesions and intense, unrelenting pruritus,[7] associated with sleep disturbance and depression.[8–12]

Three systemic biologics, targeting either interleukin (IL)-4 receptor subunit alpha (IL-4Rα) (dupilumab) or IL-13 (tralokinumab and lebrikizumab), are approved treatments in the USA and Europe for moderate-to-severe AD when dosed every other week (Q2W) through 16 weeks, with tralokinumab and lebrikizumab maintenance therapy potentially administered every fourth week (Q4W).[13–26] Dupilumab is not approved for Q4W dosing.[25,26]

In China, the only systemic biologic approved for moderate-to-severe AD is dupilumab, when dosed Q2W.[27] Heterogeneous efficacy outcomes have been reported for AD medications, such as long-term loss of response, including when continuing with dupilumab once weekly (QW) or Q2W, or after switching to Q4W or Q8W dosing (33–54% of patients maintained response).[20,28] For patients who do not respond to biologic therapy, oral Janus kinase inhibitors are approved second-line treatments, although potential safety warnings include increased risk of herpes, serious infections, malignancy and cardiovascular events.[29–34] Thus, there is a need for medications with efficacy and safety demonstrated with particularly convenient dosing regimens for long-term AD management.

Rademikibart (formerly CBP-201) is a next-generation human IgG4 kappa monoclonal antibody targeting IL-4Rα. In preclinical experiments, rademikibart is optimized to bind to distinct IL-4Rα epitopes with higher affinity than dupilumab, resulting in potent downregulation of T helper (Th)2-driven inflammatory responses *in vitro*, *in vivo* and *ex vivo*.[35] Structural and molecular dynamics suggest that rademikibart is more stable when connected to IL-4Rα, demonstrating superior binding energy than dupilumab; thus rademikibart is expected to be efficacious at longer dosing intervals.[36] During the international phase II study CBP-201-WW001, in adults with moderate-to-severe AD rademikibart achieved rapid improvements in dermatitis and pruritus across 16 weeks of treatment.[37] Notably, improvements in eczematous lesions were similar with Q2W and Q4W dosing, and the lack of plateauing at week 16 indicated further room for improvement with longer treatment durations.[37] The clinical safety profile of rademikibart was generally comparable with placebo.[37,38]

Here we report key findings from SEASIDE CHINA, a phase II trial assessing short-term (16 weeks) and long-term (52 weeks) rademikibart monotherapy in adults and adolescents with moderate-to-severe AD.

Patients and methods

Study design

The SEASIDE CHINA phase II, randomized, double-blind, placebo-controlled trial (ClinicalTrials.gov NCT05017480) was conducted across 45 centres in China. This 64-week trial comprised of screening (4 weeks), stage 1 (16-week treatment period), and stage 2 (36-week treatment and 8-week follow-up periods) (Figure S1; see Supporting Information).

Patients were randomized (2 : 1) double-blind to receive subcutaneous rademikibart (600 mg) or matching placebo on day 1, and rademikibart (300 mg Q2W) or placebo from week 2 to week 14.

Predose at week 16, patients were assessed for minimal important change (MIC), defined as ≥50% improvement from baseline in Eczema Area and Severity Index score (EASI 50).[39] Week 16 EASI 50 responders were randomized (1 : 1) double-blind to receive rademikibart 300 mg Q2W or Q4W, with final doses administered at weeks 50 and 48, respectively.

Patients

Eligibility criteria are shown in Table S1 (see Supporting Information). Eligible patients were 12–75 years old, with moderate-to-severe AD [validated Investigator Global Assessment Scale for AD (vIGA-AD™) ≥3, EASI ≥16 and body surface area (BSA) involvement of AD ≥10%] inadequately controlled/not suitable for topical treatments. Patients were required to have an average Peak Pruritus Numeric Rating Scale (PP-NRS) score ≥4, based on at least four daily assessments for 7 days before randomization.

Procedures, assessments and endpoints

Patients were randomly assigned to rademikibart or placebo using a central randomization scheme and an interactive voice response system. Patients, site personnel, the sponsor and designees conducting/monitoring the study were unaware of treatment assignment. Patients were stratified according to baseline severity (moderate, vIGA=3; severe, vIGA=4).

Rademikibart was provided in 2 mL (150 mg mL^{-1}) subcutaneous injections. Injection frequency was identical in the rademikibart and placebo arms, and patients applied mild emollient twice daily from ≥7 days before baseline (ceasing 4 h before evaluations). No other concomitant topical AD treatment was allowed, except for permitted rescue medications (including topical corticosteroids, calcineurin inhibitors and, for patients without adequate response after ≥7 days, systemic medication). If a patient received systemic corticosteroid or nonsteroid immunosuppressive/immunomodulatory rescue therapy, study treatment was to be stopped immediately. If possible, the investigator was to assess efficacy and safety before administering rescue therapy. AD requiring rescue therapy was recorded as an adverse event (AE).

Investigator assessments of AD severity/extent (vIGA, EASI, BSA) were conducted at screening and on prespecified days (± 3 days) during stage 1 (predose day 1 and days 15, 29, 57, 85 and 113) and stage 2 (28-day intervals). Patients assessed peak pruritus severity during 24-h periods using the PP-NRS, with potential scores ranging from 0 (none) to 10 (worst imaginable). PP-NRS scores were recorded in daily diaries, commencing 7 days before baseline. Using the Patient Oriented Eczema Measure (POEM), patients assessed various aspects of AD in the past week

(dryness, itching, flaking, cracking, sleeplessness, bleeding and weeping/oozing), with potential scores ranging from 0 (none) to 28 (severe). The 10-item Dermatology Life Quality Index (DLQI) questionnaire was used to assess the impact of AD on quality of life (QoL) in the past week, with potential scores ranging from 0 (none) to 30 (severe).

Primary and secondary efficacy endpoints were assessed in stage 1, followed by additional assessments during stage 2. The primary endpoint was the proportion of patients with a vIGA score of 0 (clear skin) or 1 (nearly clear skin) and a ≥ 2-point reduction from baseline at week 16 (Figure S1). Secondary endpoints at week 16 included proportions of patients achieving EASI 75, EASI 90, and weekly average PP-NRS score decrease of ≥ 3 and ≥ 4 points from baseline; percentage change from baseline in EASI, BSA and PP-NRS scores (as well as at week 2 for PP-NRS); and change in POEM and DLQI. AEs were MedDRA coded (Version 26.0).

Statistical analysis

The sample size, assuming 15% dropout by week 16, would provide 90% power to detect a therapeutic effect on the primary endpoint for rademikibart vs. placebo. Power was calculated based on the assumption that the proportion of patients reaching the primary endpoint was 27% in the rademikibart group and 9% in the placebo group. The two-sided significance level was 0.05.

In stage 1, efficacy analyses were conducted with all randomized patients who received rademikibart or placebo. In stage 2, efficacy analyses were conducted in all patients who received rademikibart in stage 1 and achieved MIC (EASI 50) at week 16. Patients without EASI 50 in stage 1 were assessed for EASI 50 in stage 2.

In stage 1, binary responder endpoints were analysed by stratified Cochran–Mantel–Haenszel test, and score changes by a mixed-effect model for repeated measures (MMRM). The MMRM analysis included baseline value, treatment, visit, treatment by visit interaction and baseline AD severity (IGA 3 or 4). Data after intercurrent events were imputed based on the type of events (Table S2; see Supporting Information). If the target variable was still missing after application of the strategies for managing intercurrent events, missing data through week 16 in the rademikibart and placebo arms were imputed using jump to reference (J2R) and multiple imputation (MI), respectively. Non-responder imputation (NRI) sensitivity analyses were also conducted through week 16. In stage 2, for patients who achieved MIC (EASI 50 in stage 1), binary responses were analysed similar to stage 1 using NRI and MI for intercurrent events and missing data, while score changes were analysed by analysis of covariance (ANCOVA) and MI for intercurrent events and missing data. The ANCOVA model included baseline value, treatment and baseline severity (IGA 3 or 4).

Results

Baseline characteristics in stages 1 and 2

From August 2021, 330 patients with moderate-to-severe AD were randomly assigned 2 : 1 to receive rademikibart 300 mg Q2W or placebo. The trial completed in September 2023. Disease characteristics and demographics were well balanced per treatment arm at baseline in stages 1 and 2 (Tables S3, S4; see Supporting Information).

In stage 1, at week 16, 80.2% of patients achieved MIC (i.e. EASI 50) in the rademikibart 300 mg Q2W arm vs. 38.8% in the placebo group ($P < 0.001$). All week 16 MIC responders were rerandomized 1 : 1 to receive rademikibart 300 mg Q2W or Q4W for 36 weeks in stage 2 (Figure S1). Patients without MIC at week 16 were eligible for rademikibart 300 mg Q2W therapy in stage 2, and most of these patients subsequently achieved EASI 50 through week 52 (68.7% and 88.1% of those who in stage 1 received rademikibart Q2W or placebo, respectively).

Most patients completed stage 1 (95.4% rademikibart Q2W and 91.9% placebo) (Figure S2; see Supporting Information). Of the week 16 MIC responders to rademikibart Q2W in stage 1, 94.5% of those who continued with 300 mg Q2W and 93.4% of those who switched to 300 mg Q4W dosing during stage 2 completed the 36-week period (Figure S3; see Supporting Information).

Rescue medication rates in stage 1 were 10.0% (rademikibart Q2W) and 16.2% (placebo), including 7.3% and 10.8% for topical corticosteroids, respectively. In stage 2, rescue medication rates for week 16 MIC responders were 14.2% (rademikibart Q2W) and 21.4% (rademikibart Q4W), including 11.5% and 15.2%, respectively, for topical corticosteroids.

Rapid improvements in dermatitis and pruritus with rademikibart every 2 weeks through stage 1 (week 16)

In stage 1, 29.0% of patients on rademikibart Q2W vs. 5.9% on placebo achieved the primary endpoint of vIGA response of 0 (clear skin) or 1 (almost clear) and ≥ 2-point decrease from baseline at week 16 ($P < 0.001$). Response to rademikibart Q2W did not plateau at week 16 (Figure 1). This analysis of the primary endpoint, using J2R and MI methodology for the rademikibart and placebo arms, respectively, was supported by the NRI sensitivity analysis, in which 28.9% vs. 5.5% of patients achieved vIGA response (0/1 and ≥ 2-point decrease) with rademikibart Q2W vs. placebo, respectively, at week 16 ($P < 0.001$).

EASI, PP-NRS, POEM and DLQI outcomes improved rapidly, with continuous improvement across 16 weeks of treatment [Figures 2–4; Figure S4, Table S5 (see Supporting Information)]. In the rademikibart Q2W group, 58.6% of patients gained EASI 75 response at week 16 vs. 22.6% with placebo ($P < 0.001$) (Figure 2). Percentage change in PP-NRS at week 2 (a secondary endpoint) was significant in the rademikibart Q2W group compared with placebo (least squares mean −14.3% vs. −6.9%; $P < 0.01$), and sustained through week 16 (−40.0% vs. −13.5%; $P < 0.001$) (Figure 3).

Further improvements through stage 2 (week 52) were similar with 2- and 4-week dosing

In stage 2, when assessed in the week 16 MIC (EASI 50) responders to rademikibart Q2W from stage 1, the proportion of patients with clear/almost clear skin continued to increase across the 36-week treatment period and was comparable with Q2W and Q4W dosing (59.1% and 62.6%,

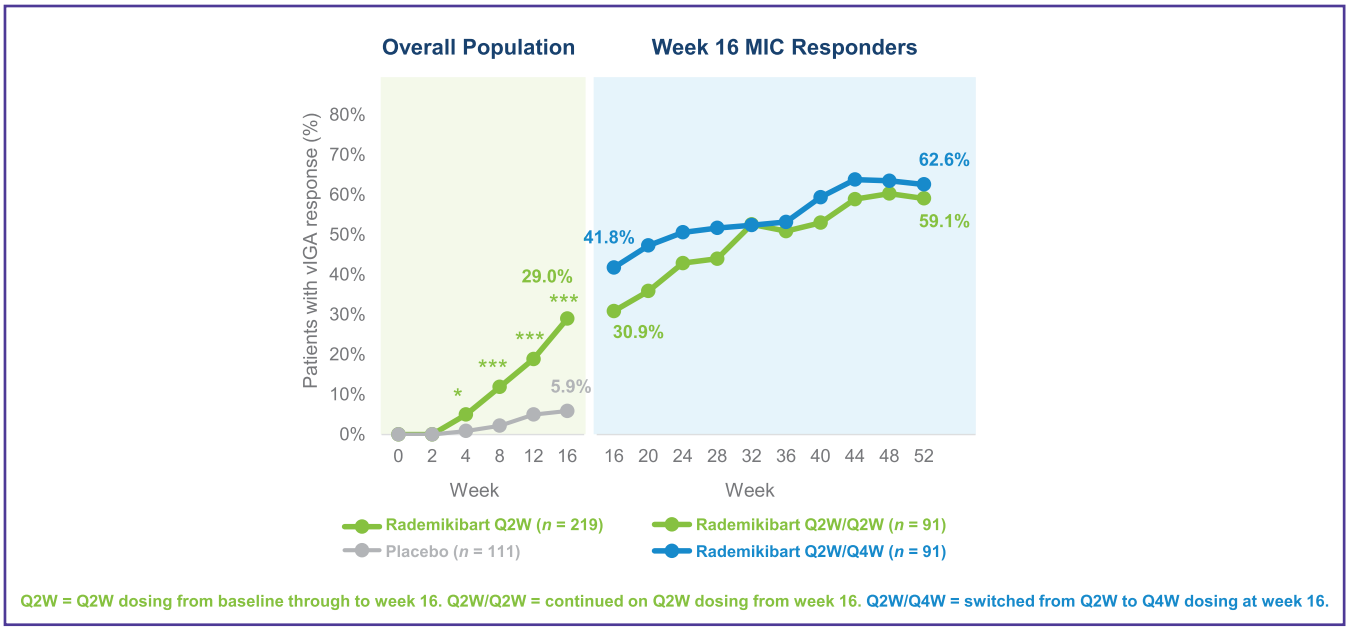


Figure 1 Validated Investigator Global Assessment (vIGA) response of 0 (clear skin) or 1 (almost clear skin) and a decrease of ≥2 points from baseline in the overall population and in week 16 minimal important change (MIC) responders, defined as those with ≥50% improvement in Eczema Area and Severity Index (EASI 50). Each *n* represents the number of patients in the analysis, which remained constant at each time point due to the use of statistical methodology to impute missing values. Up to week 16, binary response data were analysed by the Cochran–Mantel–Haenszel test. Through week 16, data after intercurrent events were imputed based on the type of events (Table S2; see Supporting Information). If the target variable was still missing after application of the strategies for managing intercurrent events, missing data through week 16 in the rademikibart and placebo arms were imputed using Jump to Reference and Multiple Imputation methodology, respectively. From week 16, binary response data were analysed by non-responder imputation and multiple imputation. Q2W, every 2 weeks; Q4W, every 4 weeks. **P*<0.05, ***P*<0.01, ****P*<0.001, vs. placebo.

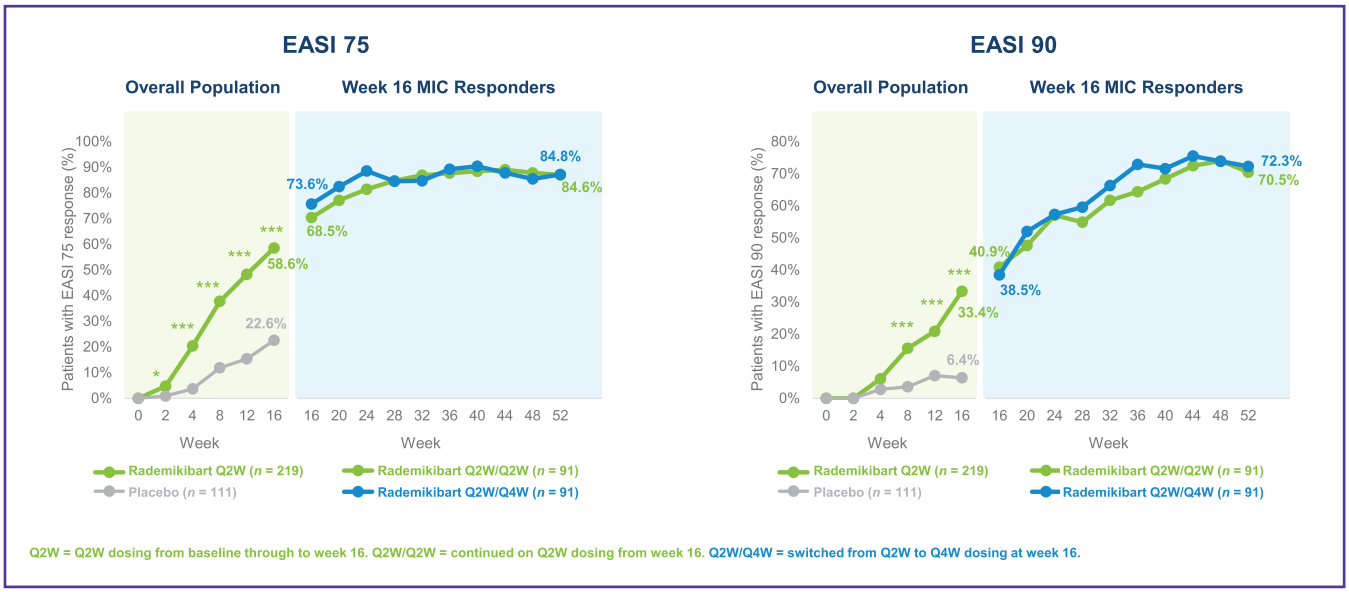


Figure 2 Response of ≥75% and ≥90% improvement from baseline in Eczema Area and Severity Index (EASI 75 and EASI 90) in the overall population and in week 16 minimal important change (MIC) responders, defined as those with ≥50% improvement in EASI (EASI 50) at week 16. Each *n* represents the number of patients in the analysis, which remained constant at each time point due to the use of statistical methodology to impute missing values. Up to week 16, binary response data were analysed by the Cochran–Mantel–Haenszel test. Through week 16, data after intercurrent events were imputed based on the type of events (Table S2; see Supporting Information). If the target variable was still missing after application of the strategies for managing intercurrent events, missing data through week 16 in the rademikibart and placebo arms were imputed using Jump to Reference and Multiple Imputation methodology, respectively. From week 16, binary response data were analysed by non-responder imputation and multiple imputation. Q2W, every 2 weeks; Q4W, every 4 weeks. **P*<0.05, ***P*<0.01, *P*<0.001, vs. placebo.

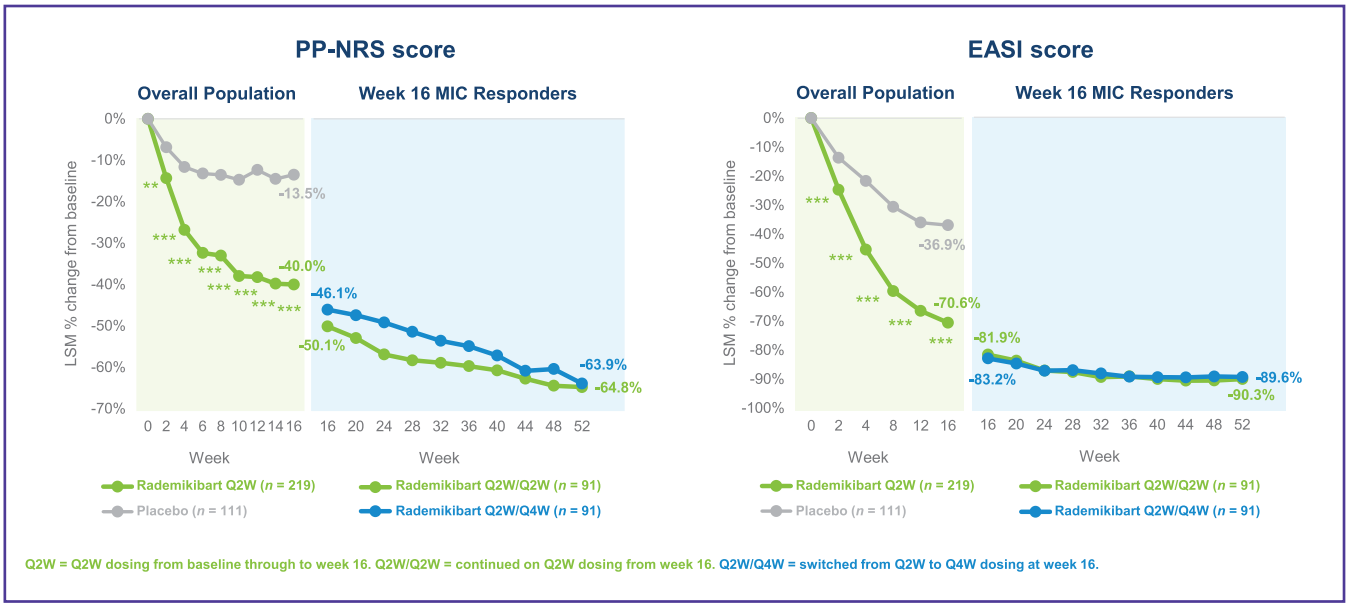


Figure 3 Peak Pruritus Numerical Rating Scale (PP-NRS) and Eczema Area and Severity Index (EASI) score change in the overall population and in week 16 minimal important change (MIC) responders, defined as those with ≥50% improvement in EASI (EASI 50) at week 16. Each *n* represents the number of patients in the analysis, which remained constant at each time point due to the use of statistical methodology to impute missing values. Up to week 16, score change was analysed by mixed-effect model repeat measurement (MMRM). The MMRM analysis included baseline value, treatment, visit, treatment by visit interaction, and baseline AD severity [Investigator Global Assessment (IGA) score of 3 or 4]. Through week 16, data after intercurrent events were imputed based on the type of events (Table S2; see Supporting Information). If the target variable was still missing after application of the strategies for managing intercurrent events, missing data through week 16 in the rademikibart and placebo arms were imputed using jump to reference and multiple imputation (MI) methodology, respectively. From week 16, score change was analysed by ANCOVA and MI. The ANCOVA model included baseline value, treatment and baseline severity (IGA 3 or 4). LSM, least squares mean; Q2W, every 2 weeks; Q4W, every 4 weeks. **P*<0.05, ***P*<0.01, ****P*<0.001, vs. placebo.

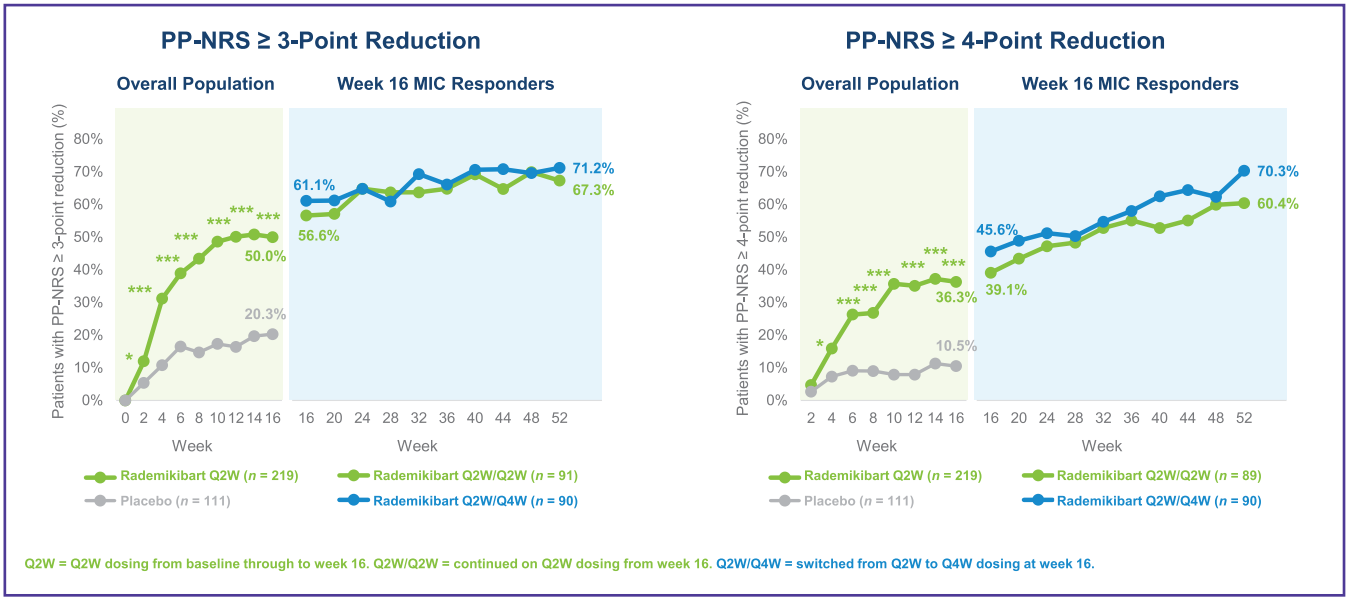


Figure 4 Peak Pruritus Numerical Rating Scale (PP-NRS) ≥3-point and ≥4-point response in the overall population and in week 16 minimal important change (MIC) responders, defined as those with ≥50% improvement in Eczema Area and Severity Index (EASI 50). Each *n* represents the number of patients in the analysis, which remained constant at each time point due to the use of statistical methodology to impute missing values. Up to week 16, binary response data were analysed by the Cochran–Mantel–Haenszel test. Through week 16, data after intercurrent events were imputed based on the type of events (Table S2; see Supporting Information). If the target variable was still missing after application of the strategies for managing intercurrent events, missing data through week 16 in the rademikibart and placebo arms were imputed using jump to reference and multiple imputation (MI) methodology, respectively. From week 16, binary response data were analysed by non-responder imputation and MI. Q2W, every 2 weeks; Q4W, every 4 weeks. **P*<0.05, ***P*<0.01, ****P*<0.001, vs. placebo.

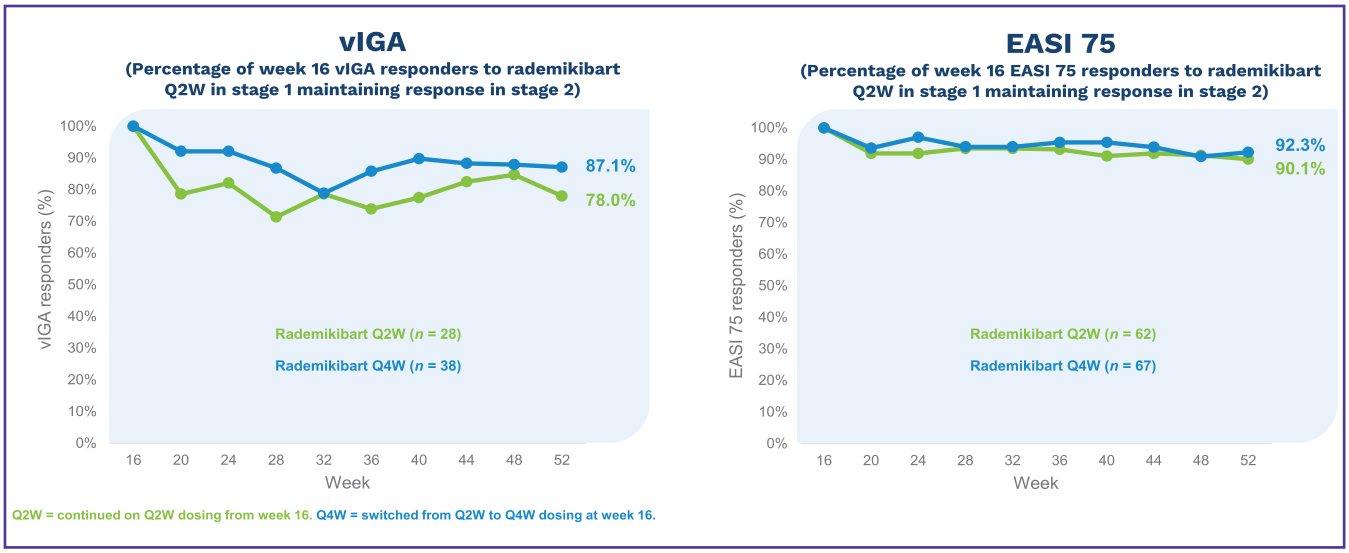


Figure 5 Maintenance of validated Investigator Global Assessment (vIGA) response of 0 (clear skin) or 1 (almost clear skin) and a decrease of ≥2 points from baseline and ≥75% improvement from baseline in Eczema Area and Severity Index (EASI 75) responses from week 16 through week 52. Data were analysed by non-responder imputation for rescue medications and multiple imputation for remaining missing data. Q2W, every 2 weeks; Q4W, every 4 weeks.

respectively, at week 52) (Figure 1). Further AD improvements in stage 2 were also obtained with the other AD rating scales, and were similar with Q2W and Q4W dosing, including the proportions of patients with EASI 75 (84.6% and 84.8%, respectively, at week 52) (Figures 2–4; Figure S4, Table S5).

Responders in stage 1 (week 16) maintained efficacy through stage 2 (week 52) with both 2-week and 4-week dosing

In stage 2, most patients who achieved response criteria during treatment with rademikibart Q2W at week 16 in stage 1 maintained these responses through week 52, with similar efficacy in the Q2W and Q4W groups [78–95% of patients for vIGA response (0/1 and ≥2-point decrease), EASI 75 or PP-NRS ≥4-point reduction] (Figures 5, 6).

Safety

Treatment-emergent adverse events (TEAEs) are summarized across stages 1 and 2 in Tables 1 and 2 [and Tables S6, S7 (see Supporting Information)]. In the rademikibart Q2W vs. placebo groups at week 16, the incidence of grade 3 TEAEs (1.8% vs. 4.5%) and serious TEAEs (0.5% vs. 2.7%) was lower with rademikibart, while the incidence of any TEAE was comparable (73.1% vs. 69.4%). No TEAEs were grade ≥4, and no serious TEAEs were related to study treatment.

Four patients discontinued treatment due to TEAEs, three in the rademikbart arms across stages 1 and 2 and one in the placebo arm in stage 1 (Tables 1, 2). Two patients discontinued owing to AD flares, one each in the rademikibart Q2W (grade 2) and placebo (grade 3) arms during stage 1. Two patients reported TEAEs that led to discontinuation of rademikibart in stage 2; one patient was pregnant (classified as a TEAE) and the other developed grade 2 vitiligo in stage 1 (rademikibart 300 mg Q2W arm) and the patient discontinued in stage 2.

Injection site reactions (ISRs), all grade 1 (mild) in severity, were experienced in stage 1 by 9.1% of patients (rademikibart Q2W) vs. 2.7% (placebo), and in stage 2 by 5.3% (Q2W) and 7.1% (Q4W) (Tables 1, 2). Other TEAEs of particular interest in the rademikibart Q2W and placebo groups, respectively, included ophthalmic TEAEs (conjunctivitis, 5.5% vs. 2.7%; keratitis, 0.9% vs. 0%) and herpes infections (1.8% vs. 1.8%) in stage 1, with a similar/lower incidence reported in stage 2 (see Tables 1 and 2 for the MedDRA Preferred Terms included under the umbrella terms 'conjunctivitis', 'keratitis' and 'herpes infections'). One TEAE of anaphylaxis was reported (nonserious, grade 1, unrelated to study treatment, and the patient continued treatment with rademikibart).

Discussion

In the SEASIDE CHINA phase II trial, most patients with moderate-to-severe AD experienced clinically meaningful reductions in the severity and extent of eczematous lesions as well as in burdensome pruritus, and reported substantially better health-related QoL. These improvements occurred rapidly, during the first 16 weeks of rademikibart 300 mg Q2W monotherapy (stage 1), and continued during the subsequent 36-week treatment period (stage 2) when, importantly, they were comparable for patients who remained on rademikibart Q2W or switched to Q4W monotherapy. Large proportions of patients with vIGA response (0/1 and ≥2-point decrease), EASI 75 and PP-NRS ≥4-point reduction at week 16 maintained these responses through week 52 with rademikibart Q2W or Q4W (78–95% of patients), and response maintenance was comparable with both dosing regimens. Although caution should be exercised when indirectly comparing studies (given that outcomes may be affected by

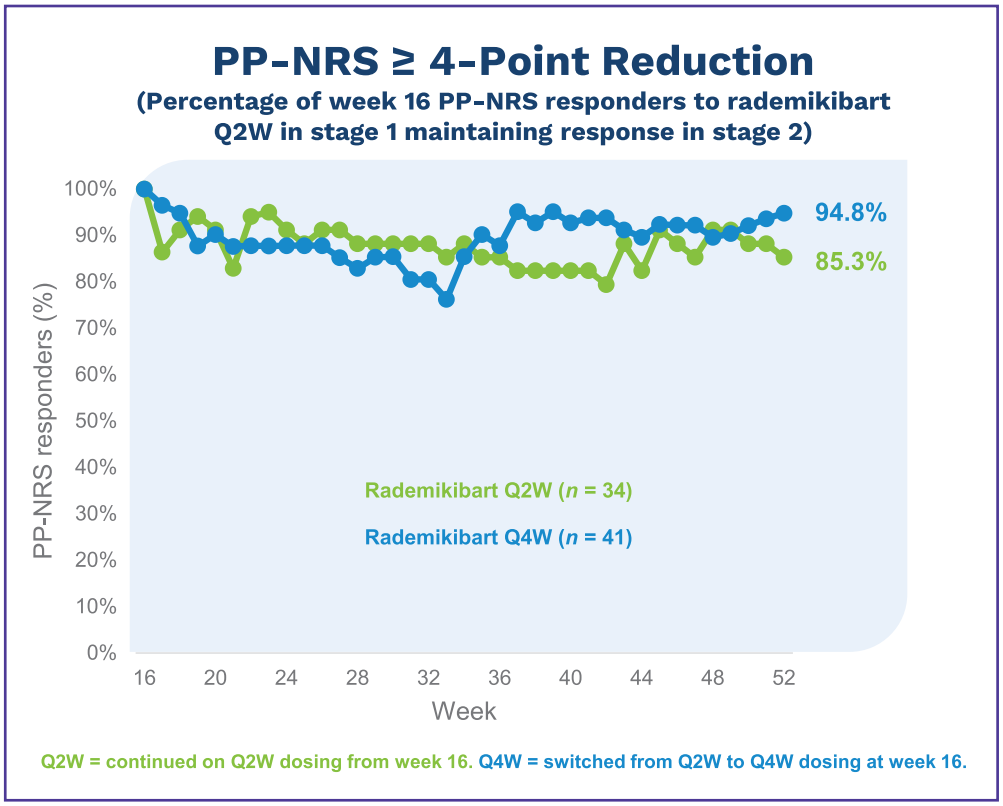


Figure 6 Maintenance of Peak Pruritus Numerical Rating Scale (PP-NRS) ≥4-point response from week 16 through week 52. Data were analysed by non-responder imputation for rescue medications and multiple imputation for remaining missing data. Q2W, every 2 weeks; Q4W, every 4 weeks.

Table 1 Overview of treatment-emergent adverse events (TEAEs) during stage 1

	Rademikibart 300 mg Q2W (*n* = 219)	Placebo Q2W (*n* = 111)	Total (*N* = 330)
TEAEs	160 (73.1)	77 (69.4)	237 (71.8)
Related to study drug	67 (30.6)	25 (22.5)	92 (27.9)
Serious TEAEs	1 (0.5)	3 (2.7)	4 (1.2)
Serious TEAEs related to study drug	0	0	0
Leading to death	0	0	0
Leading to study drug discontinuation	2 (0.9)[a]	1 (0.9)	3 (0.9)
Severe (grade 3) TEAEs	4 (1.8)	5 (4.5)	9 (2.7)
Injection site reactions (all grade 1)	20 (9.1)	3 (2.7)	23 (7.0)
Injection site erythema	11 (5.0)	1 (0.9)	12 (3.6)
Injection site induration	7 (3.2)	1 (0.9)	8 (2.4)
Injection site oedema	5 (2.3)	1 (0.9)	6 (1.8)
Injection site hematoma	1 (0.5)	1 (0.9)	2 (0.6)
Injection site reaction	2 (0.9)	0	2 (0.6)
Injection site inflammation	1 (0.5)	0	1 (0.3)
Injection site pain	1 (0.5)	0	1 (0.3)
Injection site pruritus	1 (0.5)	0	1 (0.3)
Injection site swelling	1 (0.5)	0	1 (0.3)
Conjunctivitis[b]	12 (5.5)	3 (2.7)	15 (4.5)
Keratitis[c]	2 (0.9)	0	2 (0.6)
Anaphylaxis[d]	1 (0.5)	0	1 (0.3)
Herpes infections[e]	4 (1.8)	2 (1.8)	6 (1.8)

All values are presented as *n* (%). TEAEs in stage 1 were defined as adverse events occurring or worsening on or after the first study dose and prior to the stage 2 dose or 70 days after the last dose in stage 1, whichever occurred first. EASI, Eczema Area and Severity Index; EASI 50, ≥50% reduction in EASI score from baseline; Q2W, every 2 weeks. [a]One patient discontinued in stage 2 (36-week treatment period) after onset of the grade 2 vitiligo in stage 1 (16-week treatment period). [b]'Conjunctivitis' includes the MedDRA Preferred Terms 'conjunctivitis' (2.7% vs. 2.7%) and 'conjunctivitis allergic' (3.2% vs. 0%) in the rademikibart Q2W vs. placebo arms, respectively. There were no reports of bacterial conjunctivitis, viral conjunctivitis, giant papillary conjunctivitis, eye irritation or eye inflammation. [c]'Keratitis' includes the MedDRA Preferred Term 'keratitis' (0.9% vs. 0%) in the rademikibart Q2W vs. placebo arms, respectively. There were no reports of ulcerative keratitis, allergic keratitis, atopic keratoconjunctivitis, or ocular herpes simplex. [d]The anaphylaxis TEAE was grade 1 and unrelated to study drug. [e]'Herpes infections' includes the following MedDRA Preferred Terms: herpes virus infection (0.5% vs. 0.9%), herpes zoster (0% vs. 0.9%), herpes simplex (0.5% vs. 0%), herpes simplex reactivation (0.5% vs. 0%), and oral herpes (0.5% vs. 0%) in the rademikibart Q2W vs. placebo arms, respectively.

Table 2 Overview of treatment-emergent adverse events (TEAEs) during stage 2

	Week 16 EASI 50 responders from stage 1[a]		Week 16 EASI 50 nonresponders from stage 1[a]	
	Rademikibart 300 mg			
	Q2W (*n* = 113)	Q4W (*n* = 112)	Q2W (*n* = 85)	Total (*N* = 310)
TEAEs	93 (82.3)	95 (84.8)	71 (83.5)	259 (83.5)
Related to study drug	28 (24.8)	28 (25.0)	25 (29.4)	81 (26.1)
Serious TEAEs	1 (0.9)	3 (2.7)	6 (7.1)	10 (3.2)
Serious TEAEs related to study drug	0 (0)	0 (0)	0 (0)	0 (0)
Leading to death	0 (0)	0 (0)	0 (0)	0 (0)
Leading to study drug discontinuation[b]	0 (0)	0 (0)	1 (1.2)	1 (0.3)
Severe (grade 3) TEAEs	3 (2.7)	5 (4.5)	6 (7.1)	14 (4.5)
Injection site reactions (all grade 1)	6 (5.3)	8 (7.1)	6 (7.1)	20 (6.5)
Injection site erythema	3 (2.7)	6 (5.4)	3 (3.5)	12 (3.9)
Injection site induration	3 (2.7)	3 (2.7)	1 (1.2)	7 (2.3)
Injection site oedema	1 (0.9)	1 (0.9)	3 (3.5)	5 (1.6)
Injection site inflammation	1 (0.9)	1 (0.9)	1 (1.2)	3 (1.0)
Conjunctivitis[c]	6 (5.3)	6 (5.4)	7 (8.2)	19 (6.1)
Keratitis[d]	1 (0.9)	0 (0)	0 (0)	1 (0.3)
AST/ALT > 5 × ULN[e]	0 (0)	1 (0.9)	0 (0)	1 (0.3)
Parasitic and opportunistic infection[f]	1 (0.9)	0 (0)	0 (0)	1 (0.3)
Herpes infections[g]	0 (0)	0 (0)	3 (3.5)	3 (1.0)

All values are presented as *n* (%). TEAEs in stage 2 were defined as adverse events occurring or worsening on or after the first study dose in stage 2 to the end of follow-up. ALT, alanine aminotransferase; AST, aspartate aminotransferase; EASI, Eczema Area and Severity Index; EASI 50, ≥50% reduction in EASI score from baseline; Q2W, every 2 weeks; Q4W, every 4 weeks; ULN, upper limit of normal. [a]Includes patients treated with rademikibart or placebo during stage 1 (16-week treatment period). [b]The patient discontinued due to a TEAE of pregnancy; another patient discontinued due to a TEAE in stage 2 (grade 2 vitiligo), although the TEAE began in stage 1, in the rademikibart 300 mg Q2W arm, thus the patient is reported in the stage 1 discontinuations in Table 1. [c]'Conjunctivitis' includes the MedDRA Preferred Terms 'conjunctivitis' (3.5% vs. 2.7% vs. 7.1%) and 'conjunctivitis allergic' (1.8% vs. 3.6% vs. 1.2%) in the respective treatment arms. There were no reports of bacterial conjunctivitis, viral conjunctivitis, giant papillary conjunctivitis, eye irritation, or eye inflammation. [d]'Keratitis' only includes the MedDRA Preferred Term 'keratitis'. There were no reports of ulcerative keratitis, allergic keratitis, atopic keratoconjunctivitis or ocular herpes simplex. [e]The hepatic enzyme TEAE was grade 3 and unrelated to the study drug. [f]The infection TEAE was grade 2, with the MedDRA Preferred Term 'otitis externa fungal'. [g]'Herpes infections' includes the following MedDRA Preferred Terms: herpes zoster (1.2%) and herpes simplex (2.4%) in the week 16 EASI 50 nonresponder arm.

various study design/conduct variables), the improvements through weeks 16 and 52 were similar to/greater than those obtained with Q2W or Q4W lebrikizumab, tralokinumab and dupilumab.[14–20,28,40–42] Treatment completion rates were also high across the 1 year of rademikibart therapy (generally more than 90% in stages 1 and 2). Based on these findings, after an initial 16 weeks of treatment with Q2W dosing, we would expect most patients to benefit greatly from rademikibart when administered at convenient once-monthly (Q4W) dosing frequencies for 1 year.

The SEASIDE CHINA efficacy findings are compatible with those from the WW001 international phase II trial of rademikibart.[37] However, efficacy responses were often larger than in WW001, even though all patients had moderate-to-severe AD in both trials. In SEASIDE CHINA, approximately half of patients had severe AD at baseline, compared with around a third in WW001. Post hoc analyses of multiple clinical trials suggest that placebo responses are larger in populations with less severe AD,[43–45] and it may be the case that baseline severity does not equally affect responses in active and placebo arms.[44,45] Baseline AD severity ratings in SEASIDE CHINA were similar, and AD improvements were greater/similar in magnitude, when indirectly compared with trials of other biologics.[14–20,28,40–42] However, it is notable that in phase II and III trials of dupilumab, EASI responses appeared to plateau by week 16.[14–16] In both SEASIDE CHINA and WW001, the rapid reductions in AD extent and severity did not plateau at week 16, suggesting further room for improvement, which in SEASIDE CHINA was demonstrated across 1 year of rademikibart treatment.

Rescue medication usage was numerically lower than/comparable with other AD biologic treatments.[17,18,20,28,40–42] The rescue medication rate at week 16 was 10.0% with rademikibart Q2W, whereas rates for dupilumab were 21.6% (QW) and 17.1% (Q2W) when pooled for two phase III international studies and 19.5% (Q2W) in a Chinese clinical trial.[17,18] Rescue medication rates for week 16 MIC (EASI 50) responders across the subsequent 36-week period were 14.2% (Q2W) and 21.4% (Q4W), compared with 19.5% (QW or Q2W dosing) and 30.2% (Q4W) for IGA 0/1 or EASI 75 responders treated with dupilumab.[28]

No treatment-related serious safety concerns were identified, three patients discontinued rademikibart due to TEAEs (AD flare, vitiligo, pregnancy), and most TEAEs were mild or moderate in severity. Across all AD trials of rademikibart, every ISR was mild in severity.[37,38] In SEASIDE CHINA, the incidence of ISRs with rademikibart 300 mg Q2W (9.1%) vs. placebo (2.7%) at week 16 was comparable with other biologics, including dupilumab 300 mg Q2W in international (12.3%)[17] and Chinese (8.5%) trials.[18] The incidence of ISRs was higher than in the WW001 international phase II trial (1.8% across the treatment arms) at week 16,[37] which we speculate may be related to the quantity of solution per injection (1 mL vs. 2 mL) in the WW001 and SEASIDE CHINA trials, respectively. It is also notable that, in SEASIDE

CHINA, ISR incidence with rademikibart was lower during the 36-week treatment period [5.3% (Q2W) and 7.1% (Q4W)] than during the initial 16 weeks [9.1% (Q2W)]. The incidence of conjunctivitis with rademikibart was generally comparable with/lower than reported for other AD biologics across 52 weeks.[14–20,28,40–42] However, these indirect comparisons are hampered by a lack of standardized and clear reporting of the AE MedDRA Preferred Terms included under the umbrella term 'conjunctivitis'.

The SEASIDE CHINA trial has several strengths and limitations. Firstly, the analyses were conducted in a sizeable population (330 adult and adolescent patients in stage 1), which to the best of our knowledge constitutes the largest AD clinical trial in China to date. Secondly, all patients were Chinese. It appears unlikely that efficacy varies according to ethnicity with IL-4Rα targeting medications; in post hoc analyses of three phase III trials of dupilumab, slightly greater responses in Asian patients vs. White and Black/African American patients were likely related to baseline differences in AD severity.[46] However, AD is heterogeneous, and the phenotype may vary according to ethnic background.[47] Thirdly, a strength of our study is that improvements in AD and QoL were assessed with several instruments by investigators (IGA, EASI, BSA) and patients (PP-NRS, POEM, DLQI). Other recently developed self-assessment instruments that were not used in the current study – Recap of Atopic Eczema (RECAP) and Atopic Dermatitis Control Test (ADCT) – are recommended by experts, to investigate long-term AD control.[48] Since the study was conducted, both RECAP and ADCT have been validated in Chinese patients and strongly correlated with POEM and DLQI.[48–50] Fourthly, MIC was defined as EASI 50;[39] alternative definitions of EASI MIC are available.[51] Finally, another notable strength is that outcomes were investigated across a longer duration of treatment (1 year) than in the WW001 international trial of rademikibart and Chinese trial of dupilumab (16 weeks).[18,37]

In summary, the SEASIDE CHINA phase II AD trial of rademikibart achieved all prespecified efficacy endpoints. Rapid improvements in AD signs and pruritus with rademikibart Q2W did not plateau through week 16, and were sustained through week 52 with comparable efficacy for Q2W and Q4W dosing. The efficacy findings of SEASIDE CHINA are greater/similar in magnitude to those obtained with lebrikizumab, tralokinumab and dupilumab.[14–20,28,40–42] Rademikibart was also well tolerated (> 90% of patients completed each treatment period). Taken together, these findings indicate that most patients with moderate-to-severe AD benefited from rapid improvements during initial treatment with rademikibart, and that these improvements were highly maintained during 1 year of treatment with a convenient once-monthly (Q4W) dosing regimen.

Acknowledgements

Writing support for this manuscript was provided by Michael Riley PhD (Fortis Pharma, a Bioscript company), funded by Connect Biopharma.

Funding sources

The study was funded by Connect Biopharma.

Conflicts of interest

J.Z. has served as an advisor, speaker or consultant for and receives honoraria from Akeso Biopharma Co. Ltd, Bristol-Myers Squibb China Investment Co. Ltd, Eli Lilly and Company (China), GlaxoSmithKline (China) Investment Co. Ltd, Keymed Biosciences Inc., Kintor Pharmaceutical Ltd, LEO Pharma China, Novartis Pharmaceuticals (China), Pfizer Investment Co. Ltd, Sanofi (China) and Xian Janssen Pharmaceutical Ltd. J.I.S. has acted as a researcher for Galderma, and as an advisor, speaker or consultant for AbbVie, AFYX Therapeutics, Arena Pharmaceuticals, Asana BioSciences, BiomX, Bluefin Biomedicine, Bodewell (P&G), Boehringer Ingelheim, Celgene, Connect Biopharma, Dermavant, Dermira, Eli Lilly, Galderma, GSK, Hoth Therapeutics, Incyte, Kiniksa Pharmaceuticals, LEO Pharma, Luna Pharma, Menlo Therapeutics, Novartis, Pfizer, RAPT Therapeutics, Regeneron Pharmaceuticals and Sanofi. All other authors are either current or former employees and shareholders of Suzhou Connect Biopharmaceuticals/Connect Biopharma.

Data availability

The data underlying this article are available in the article.

Ethics statement

The study protocol was approved by the respective institutional review boards and ethics committees. The trial complied with Good Clinical Practice guidelines and the Declaration of Helsinki.

Patient consent

All patients provided written informed consent before participating. Informed consent forms and the study protocol were approved by institutional review boards and ethics committees.

Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website.

References

1 Mathiesen SM, Thomsen SF. The prevalence of atopic dermatitis in adults: systematic review on population studies. *Dermatol Online J* 2019; **25**:13030/qt6nj0x5k0.
2 Bylund S, Kobyletzki LB, Svalstedt M, Svensson Å. Prevalence and incidence of atopic dermatitis: a systematic review. *Acta Derm Venereol* 2020; **100**:adv00160.
3 Chan TC, Wu NL, Wong LS *et al.* Taiwanese Dermatological Association consensus for the management of atopic dermatitis: a 2020 update. *J Formos Med Assoc* 2021; **120**:429–42.
4 Hwang CY, Chen YJ, Lin MW *et al.* Prevalence of atopic dermatitis, allergic rhinitis and asthma in Taiwan: a national study 2000 to 2007. *Acta Derm Venereol* 2010; **90**:589–94.
5 Mao D, Li J, Liu S *et al.* Prevalence and risk factors of atopic dermatitis in Chinese adults: a nationwide population-based cross-sectional study. *Chin Med J (Engl)* 2023; **136**:604–6.

6 Liu W, Cai J, Sun C *et al.* Time-trends for eczema prevalences among children and adults from 1985 to 2015 in China: a systematic review. *BMC Public Health* 2022; **22**:1294.
7 Boguniewicz M, Alexis AF, Beck LA *et al.* Expert perspectives on management of moderate-to-severe atopic dermatitis: a multidisciplinary consensus addressing current and emerging therapies. *J Allergy Clin Immunol Pract* 2017; **5**:1519–31.
8 Chiesa Fuxench ZC, Block JK, Boguniewicz M *et al.* Atopic dermatitis in America study: a cross-sectional study examining the prevalence and disease burden of atopic dermatitis in the US adult population. *J Invest Dermatol* 2019; **139**:583–90.
9 Dalgard FJ, Gieler U, Tomas-Aragones L *et al.* The psychological burden of skin diseases: a cross-sectional multicenter study among dermatological out-patients in 13 European countries. *J Invest Dermatol* 2015; **135**:984–91.
10 Schonmann Y, Mansfield KE, Hayes JF *et al.* Atopic eczema in adulthood and risk of depression and anxiety: a population-based cohort study. *J Allergy Clin Immunol Pract* 2020; **8**:248–57.e16.
11 Silverberg JI, Gelfand JM, Margolis DJ *et al.* Symptoms and diagnosis of anxiety and depression in atopic dermatitis in U.S. adults. *Br J Dermatol* 2019; **181**:554–65.
12 Xie QW, Dai X, Tang X *et al.* Risk of mental disorders in children and adolescents with atopic dermatitis: a systematic review and meta-analysis. *Front Psychol* 2019; **10**:1773.
13 Akinlade B, Guttman-Yassky E, de Bruin-Weller M *et al.* Conjunctivitis in dupilumab clinical trials. *Br J Dermatol* 2019; **181**:459–73.
14 Thaçi D, Simpson EL, Beck LA *et al.* Efficacy and safety of dupilumab in adults with moderate-to-severe atopic dermatitis inadequately controlled by topical treatments: a randomised, placebo-controlled, dose-ranging phase 2b trial. *Lancet* 2016; **387**:40–52.
15 Simpson EL, Bieber T, Guttman-Yassky E *et al.* Two phase 3 trials of dupilumab versus placebo in atopic dermatitis. *N Engl J Med* 2016; **375**:2335–48.
16 Tofte SJ, Papp K, Sadick N *et al.* Efficacy and safety of dupilumab for the treatment of moderate-to-severe atopic dermatitis in adults: a pooled analysis of two phase 2 clinical trials. *J Am Assoc Nurse Pract* 2018; **30**:529–41.
17 Thaçi D, Simpson EL, Deleuran M *et al.* Efficacy and safety of dupilumab monotherapy in adults with moderate-to-severe atopic dermatitis: a pooled analysis of two phase 3 randomized trials (LIBERTY AD SOLO 1 and LIBERTY AD SOLO 2). *J Dermatol Sci* 2019; **94**:266–75.
18 Zhao Y, Wu L, Lu Q *et al.* The efficacy and safety of dupilumab in Chinese patients with moderate-to-severe atopic dermatitis: a randomized, double-blind, placebo-controlled study. *Br J Dermatol* 2022; **186**:633–41.
19 Wollenberg A, Howell MD, Guttman-Yassky E *et al.* Treatment of atopic dermatitis with tralokinumab, an anti-IL-13 mAb. *J Allergy Clin Immunol* 2019; **143**:135–41.
20 Wollenberg A, Blauvelt A, Guttman-Yassky E *et al.* Tralokinumab for moderate-to-severe atopic dermatitis: results from two 52-week, randomized, double-blind, multicentre, placebo-controlled phase III trials (ECZTRA 1 and ECZTRA 2). *Br J Dermatol* 2021; **184**:437–49.
21 Adtralza. Summary of product characteristics. Available at: https://www.ema.europa.eu/en/documents/product-information/adtralza-epar-product-information_en.pdf (last accessed 15 September 2025).
22 ADBRY – tralokinumab-ldrm injection, solution. FDA label. Available at: https://www.accessdata.fda.gov/spl/data/b8446805-b24c-4d26-97e0-44a8518e3404/b8446805-b24c-4d26-97e0-44a8518e3404.xml#S2.2 (last accessed 15 September 2025).
23 Ebglyss. Summary of product characteristics. Available at: https://www.ema.europa.eu/en/documents/product-information/ebglyss-epar-product-information_en.pdf (last accessed 15 September 2025).
24 EBGLYSS (lebrikizumab-lbkz), injection, for subcutaneous use. FDA label. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2024/761306Orig1s000correctedlbl.pdf (last accessed 15 September 2025).
25 Dupixent. Summary of product characteristics. Available at: https://ec.europa.eu/health/documents/community-register/2019/20190801145601/anx_145601_en.pdf (last accessed 15 September 2025).
26 DUPIXENT® (dupilumab) injection, for subcutaneous use. FDA label. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2024/761055s064lbl.pdf (last accessed 15 September 2025).
27 Shi Y, Zhou Y, Li S *et al.* Short- and long-term cost-effectiveness of abrocitinib versus dupilumab in adults with moderate-to-severe atopic dermatitis in China. *J Med Econ* 2024; **27**:1180–9.
28 Worm M, Simpson EL, Thaçi D *et al.* Efficacy and safety of multiple dupilumab dose regimens after initial successful treatment in patients with atopic dermatitis: a randomized clinical trial. *JAMA Dermatol* 2020; **156**:131–43.
29 Fleischmann R. Recent issues in JAK inhibitor safety: perspective for the clinician. *Expert Rev Clin Immunol* 2022; **18**:295–307.
30 Svoboda SA, Johnson N, Phillips M. Dermatologic applications and safety considerations of Janus kinase inhibitors. *Skin Therapy Lett* 2020; **25**:6–11.
31 Wood H, Chandler A, Nezamololama N *et al.* Safety of Janus kinase (JAK) inhibitors in the short-term treatment of atopic dermatitis. *Int J Dermatol* 2022; **61**:746–54.
32 Singh R, Heron CE, Ghamrawi RI *et al.* Emerging role of Janus kinase inhibitors for the treatment of atopic dermatitis. *Immunotargets Ther* 2020; **9**:255–72.
33 Elmariah SB, Smith JS, Merola JF. JAK in the [black] box: a dermatology perspective on systemic JAK inhibitor safety. *Am J Clin Dermatol* 2022; **23**:427–31.
34 RINVOQ (upadacitinib) extended-release tablets, for oral use. FDA label. Available at: https://www.accessdata.fda.gov/drugsatfda_docs/label/2022/211675s004lbl.pdf (last accessed 15 September 2025).
35 Zhang L, Ding Y, Wang Q *et al.* Preclinical immunological characterization of rademikibart (CBP-201), a next-generation human monoclonal antibody targeting IL-4Rα, for the treatment of Th2 inflammatory diseases. *Sci Rep* 2023; **13**:12411.
36 Collazo R, Shi Y, Li H *et al.* Optimized second-generation IL-4Rα inhibition: structural and molecular dynamics properties of rademikibart Fab-IL-4Rα complex. *Am J Respir Crit Care Med* 2025; **211**:A1397 (abstract).
37 Silverberg JI, Strober B, Feinstein B *et al.* Efficacy and safety of rademikibart (CBP-201), a next-generation mAb targeting IL-4Rα, in adults with moderate to severe atopic dermatitis: a phase 2 randomized trial (CBP-201-WW001). *J Allergy Clin Immunol* 2024; **153**:1040–9.e12.
38 Wang J, White J, Sansone KJ *et al.* Rademikibart (CBP-201), a next-generation monoclonal antibody targeting human IL-4Rα: two phase I randomized trials, in healthy individuals and patients with atopic dermatitis. *Clin Transl Sci* 2023; **16**:2614–27.
39 Silverberg JI, Lei D, Yousaf M *et al.* What are the best endpoints for Eczema Area and Severity Index and Scoring Atopic Dermatitis in clinical practice? A prospective observational study. *Br J Dermatol* 2021; **184**:888–95.
40 Guttman-Yassky E, Blauvelt A, Eichenfield LF *et al.* Efficacy and safety of lebrikizumab, a high-affinity interleukin 13 inhibitor, in adults with moderate to severe atopic dermatitis: a phase 2b randomized clinical trial. *JAMA Dermatol* 2020; **156**:411–20.
41 Silverberg JI, Guttman-Yassky E, Thaçi D *et al.* Two phase 3 trials of lebrikizumab for moderate-to-severe atopic dermatitis. *N Engl J Med* 2023; **388**:1080–91.

42 Blauvelt A, Thyssen JP, Guttman-Yassky E *et al.* Efficacy and safety of lebrikizumab in moderate-to-severe atopic dermatitis: 52-week results of two randomized double-blinded placebo-controlled phase III trials. *Br J Dermatol* 2023; **188**:740–8.
43 Lee HH, Patel KR, Rastogi S *et al.* Placebo responses in randomized controlled trials for systemic therapy in atopic dermatitis: a systematic review and meta-analysis. *J Am Acad Dermatol* 2020; **82**:62–71.
44 Offidani A, Stingeni L, Neri I *et al.* Dupilumab treatment induced similar improvements in signs, symptoms, and quality of life in adults with moderate-to-severe atopic dermatitis with baseline Eczema Area and Severity Index Score <24 or ≥24. *Ital J Dermatol Venerol* 2022; **157**:39–46.
45 Silverberg JI, Ho S, Collazo R. A mini review of the impact of baseline disease severity on clinical outcomes: should we compare atopic dermatitis clinical trials? *Dermatol Ther (Heidelb)* 2023; **13**:3019–29.
46 Alexis AF, Rendon M, Silverberg JI *et al.* Efficacy of dupilumab in different racial subgroups of adults with moderate-to-severe atopic dermatitis in three randomized, placebo-controlled phase 3 trials. *J Drugs Dermatol* 2019; **18**:804–13.
47 Facheris P, Jeffery J, Del Duca E *et al.* The translational revolution in atopic dermatitis: the paradigm shift from pathogenesis to treatment. *Cell Mol Immunol* 2023; **20**:448–74.
48 Thomas KS, Apfelbacher CA, Chalmers JR *et al.* Recommended core outcome instruments for health-related quality of life, long-term control and itch intensity in atopic eczema trials: results of the HOME VII consensus meeting. *Br J Dermatol* 2021; **185**:139–46.
49 Yew YW, Phuan CZY, Zhao X *et al.* Validation of the Recap of Atopic Eczema (RECAP) measurement instrument for eczema control in adult patients in an Asian clinical setting. *Acta Derm Venereol* 2024; **104**:adv32323.
50 Luan TT, Peng CY, Song XT *et al.* [Validation study of the Chinese version of atopic dermatitis control tool]. *Zhonghua Yu Fang Yi Xue Za Zhi* 2023; **57**:422–6 (in Chinese).
51 Schram ME, Spuls PI, Leeflang MMG *et al.* EASI, (objective) SCORAD and POEM for atopic eczema: responsiveness and minimal clinically important difference. *Allergy* 2012; **67**:99–106.