



Bimekizumab durability of efficacy through 196 weeks and safety through 4 years in patients with moderate to severe plaque psoriasis: Results from the BE BRIGHT open-label extension trial

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Background: Patients with moderate to severe psoriasis experience significant burden on quality of life. Long-term management with latest-generation biologics can facilitate sustained complete skin clearance and improved patient well-being.

Objective: To report 4-year end-of-study bimekizumab efficacy and safety in patients with moderate to severe psoriasis.

Methods: Data were pooled from 3 phase 3 trials (BE VIVID, BE READY, and BE SURE) and their open-label extension (OLE; BE BRIGHT). Efficacy is reported for patients who received bimekizumab continuously from baseline into the OLE. Safety is reported for patients who received ≥ 1 bimekizumab dose.

Results: Seven hundred seventy-one patients received bimekizumab from baseline into the OLE. A high proportion achieved complete skin clearance (100% improvement from baseline in Psoriasis Area and Severity Index) at Week 52 (76.2%) and maintained this to Week 196 (64.7%). The rate of treatment-emergent adverse events over 4 years was 169.8/100 patient-years ($N = 1495$) and did not increase with longer exposure. The most common treatment-emergent adverse events were

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Funding sources: These studies were sponsored by UCB. This article was based on the original studies BE VIVID (NCT03370133), BE READY (NCT03410992), BE SURE (NCT03412747), and BE BRIGHT (NCT03598790) sponsored by UCB. Support for third-party writing assistance for this article, provided by Yasha Najafi, MSc, and Daniel Smith, MA, Costello Medical, UK, was funded by UCB in accordance with Good Publication Practice (GPP) guidelines (<https://www.ismpp.org/gpp-2022>).

Patient consent: Not applicable.

IRB approval status: Reviewed and approved by the relevant IRBs. Accepted for publication April 18, 2025.

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Published online April 24, 2025.

0190-9622

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<https://doi.org/10.1016/j.jaad.2025.04.038>

nasopharyngitis, oral candidiasis, and upper respiratory tract infection, consistent with bimekizumab's known safety profile.

Limitations: Eligibility criteria and ethnic representation.

Conclusion: Almost two-thirds of bimekizumab-treated patients achieved and maintained complete skin clearance through 4 years, making bimekizumab an effective, rapid, and durable long-term treatment option. (J Am Acad Dermatol 2025;93:644-53.)

Key words: bimekizumab; efficacy; long-term; plaque psoriasis; psoriasis; quality of life; safety.

INTRODUCTION

Patients with moderate to severe psoriasis face significant health and quality of life challenges,¹⁻³ including stigmatization and reduced self-esteem, which negatively impact social interactions.^{4,5} Effective control of systemic inflammation is required to limit disease progression and the development of joint disease and other comorbidities, such as cardiovascular disease.^{6,7} The chronic nature of psoriasis often means patients with moderate to severe disease receive long-term biologic treatment. Since some biologics lose effectiveness over time,⁸ understanding the long-term efficacy and safety of these medications is critical.

Bimekizumab, a relatively new biologic treatment approved for moderate to severe plaque psoriasis,^{9,10} inhibits interleukin (IL)-17F in addition to IL-17A,¹¹ key effector cytokines in psoriasis pathogenesis. Bimekizumab has demonstrated rapid and superior efficacy compared with placebo, adalimumab, ustekinumab, and secukinumab in phase 3/3b trials in psoriasis,¹²⁻¹⁵ with high efficacy levels maintained over 3 years.^{16,17} Notably, bimekizumab has shown high levels of complete skin clearance (100% improvement from baseline in Psoriasis Area and Severity Index; PASI 100),¹²⁻¹⁶ which is now an achievable goal for patients receiving newer biologics,¹⁸ and leads to greater benefits in patient well-being and symptoms versus near-complete skin clearance.^{18,19} Bimekizumab has a consistent safety profile and was well-tolerated through 3 years in clinical trials, with yearly rates of adverse events (AEs) not increasing with longer exposure.²⁰ Here, final end-of-study 4-year data for the global cohort from BE BRIGHT, an open-label extension (OLE) study assessing long-term efficacy

CAPSULE SUMMARY

- Long-term biologic treatment can improve psoriasis patient symptoms and well-being; investigating long-term efficacy and safety is important.
- Almost two-thirds of bimekizumab-treated patients achieved and maintained complete skin clearance through 196 weeks, and safety data were consistent with bimekizumab's known safety profile, making bimekizumab an effective, rapid, and durable long-term treatment.

and safety of bimekizumab in patients with moderate to severe plaque psoriasis, are reported.

METHODS

Study design and patients

Efficacy and safety data were pooled from 3 completed phase 3 feeder trials, BE VIVID (52 weeks; NCT03370133), BE READY (56 weeks; NCT03410992), and BE SURE (56 weeks; NCT03412747), and their OLE, BE BRIGHT (NCT03598790).^{12,13,15,16} BE BRIGHT lasted for 144 weeks (196/

200 weeks' total treatment, depending on feeder study) for the global cohort. Full eligibility criteria and study designs have been published previously.^{12,13,15,16} All studies were conducted in accordance with the principles of the Declaration of Helsinki,²¹ and approved by an Independent Review Board and Independent Ethics Committee. All participants provided informed written consent.

Upon completion of feeder studies, patients were eligible to enroll in BE BRIGHT, in which they received bimekizumab 320 mg every 4 weeks (Q4W) or every 8 weeks (Q8W), depending on treatment, dose, and PASI response (Supplementary Fig 1, available via Mendeley at <https://data.mendeley.com/datasets/24zjytjd7z/2>). Following protocol amendment, at OLE Week 48 (Week 100/104; end of the second year of treatment) or the next scheduled clinic visit, all patients receiving bimekizumab Q4W switched to Q8W dosing. An additional Japanese cohort with moderate to severe plaque psoriasis could enroll directly in BE BRIGHT; these patients are included in the safety analyses only.

Efficacy outcomes

Efficacy data were analyzed for patients randomized to bimekizumab at feeder study baseline, who received

Abbreviations used:

AE:	adverse event
BSA:	body surface area
DLQI:	Dermatology Life Quality Index
IBD:	inflammatory bowel disease
IGA:	Investigator's Global Assessment
IL:	interleukin
mNRI:	modified non-responder imputation
OLE:	open-label extension
PASI:	Psoriasis Area and Severity Index
PY:	patient-years
Q4W:	every 4 weeks
Q8W:	every 8 weeks

bimekizumab throughout the double-blinded study period and entered the OLE (bimekizumab total). This analysis focuses on patients randomized to bimekizumab, rather than those with specific Week 16 responses, as described previously in a 3-year efficacy report.¹⁶ Response rates for the patient subset who received bimekizumab Q4W from baseline to Week 16, then Q8W throughout double-blinded study periods and into the OLE (bimekizumab Q4W/Q8W; the approved dosing regimen for most patients with psoriasis)^{9,10} are also reported.

Proportions of patients achieving the following outcomes are reported to Week 196: $\geq 75\%/ \geq 90\%/ 100\%$ improvement from baseline in PASI (PASI 75/90/100); scores of 0/ ≤ 2 / ≤ 3 / ≤ 5 in PASI; scores of 0 and 0/1 in the Investigator's Global Assessment (IGA 0 [clear] and IGA 0/1 [clear or almost clear]); $\leq 1\%/ \leq 3\%$ body surface area affected (BSA); and a score of 0 or 1 in the Dermatology Life Quality Index (DLQI 0/1; indicating no effect of skin disease on patient quality of life).^{22,23}

Safety outcomes

Safety data were analyzed for all patients who received ≥ 1 bimekizumab dose in the phase 3 trials. Safety outcomes are presented for the full pooled trial period for all patients who received ≥ 1 bimekizumab dose and the bimekizumab Q4W/Q8W group, and separately by year of bimekizumab exposure: Years 1 (Weeks >0-52), 2 (Weeks >52-104), 3 (Weeks >104-156), and 4 (Weeks >156-208). Treatment-emergent AEs (TEAEs), serious and severe TEAEs, TEAEs leading to discontinuation, and TEAEs of interest are reported. TEAEs occurred during study treatment, including ≤ 140 days after last dose (safety follow-up period; 6 half-lives of bimekizumab). Further treatment-emergent adverse event definitions and assessment and adjudication details are reported in the Supplementary Methods, available via Mendeley at <https://data.mendeley.com/datasets/24zjytd7z/2>.

Statistical analysis

Missing efficacy data were accounted for using modified non-responder imputation (mNRI) and observed case analyses. For mNRI, patients discontinuing due to lack of efficacy or AEs considered treatment-related by the investigator were considered non-responders; multiple imputation was used for other missing data. Patients who entered the BE READY escape arm were considered non-responders from the date of escape until the end of BE READY, after which they were considered in the same way as all other non-escape patients during the BE BRIGHT OLE.¹² Safety data are reported up to the data cut-off (12 December 2023). Exposure-adjusted incidence rates/100 patient-years (PY) are reported with associated 95% confidence intervals.

RESULTS

Patients

In the efficacy population, out of 989 patients randomized to bimekizumab Q4W at feeder study baseline, 771 (78.0%) received bimekizumab continuously throughout the double-blinded study periods and entered the OLE (bimekizumab total); 105 bimekizumab Q4W-randomized patients in BE READY were re-randomized to placebo at Week 16 (treatment-withdrawal group; not included here).¹² Out of the 771 patients in the bimekizumab total group, 197 patients received bimekizumab Q4W/Q8W. In the bimekizumab total group, 645 of 771 (83.7%) completed Week 196/200; 1.2% discontinued due to lack of efficacy and 5.7% due to AEs (Supplementary Fig 2, available via Mendeley at <https://data.mendeley.com/datasets/24zjytd7z/2>). For the bimekizumab Q4W/Q8W subgroup, 167 of 197 (84.8%) completed Week 196/200; 2.0% discontinued due to lack of efficacy and 6.6% due to AEs. Baseline characteristics of both groups were generally similar (Supplementary Table I, available via Mendeley at <https://data.mendeley.com/datasets/24zjytd7z/2>).

In the safety population, 1495 patients received ≥ 1 dose of bimekizumab, totaling 4749 PY of bimekizumab exposure (Table I).

Efficacy outcomes

After 1 dose of bimekizumab (Week 4), efficacy and quality of life outcome responses were as follows (mNRI): 16.1% achieved PASI 100/PASI = 0, 43.3% achieved PASI 90, 77.5% achieved PASI 75, and 38.4% achieved DLQI 0/1. Similar Week 4 responses were seen in the bimekizumab Q4W/Q8W subgroup (PASI 100/PASI = 0: 18.0%; PASI 90: 40.5%; PASI 75: 80.7%; DLQI 0/1: 37.4%).

Table I. Treatment exposure and TEAEs in bimekizumab-treated patients

	By year				Over 4 y	
	≥1 BKZ dose Year 1 N = 1495	≥1 BKZ dose Year 2 N = 1373	≥1 BKZ dose Year 3 N = 1255	≥1 BKZ dose Year 4 N = 1129	≥1 BKZ dose N = 1495	BKZ 320 mg Q4W/Q8W N = 197
Total exposure (PY)	1384.4	1299.8	1213.3	823.4	4748.5	741.3
Summary of TEAEs, EAIR/100 PY (95% CI)						
Any TEAE	237.4 (224.4, 250.9)	150.3 (140.9, 160.1)	105.4 (98.1, 113.1)	100.5 (92.1, 109.5)	169.8 (161.0, 178.9)	155.5 (134.1, 179.4)
Serious TEAEs	6.7 (5.4, 8.3)	5.8 (4.6, 7.3)	5.8 (4.5, 7.4)	5.6 (4.1, 7.5)	5.5 (4.8, 6.2)	4.3 (2.9, 6.2)
Discontinuations due to TEAEs	4.8 (3.7, 6.1)	2.5 (1.7, 3.5)	2.2 (1.5, 3.3)	1.1 (0.5, 2.1)	2.8 (2.4, 3.4)	1.8 (0.9, 3.0)
Severe TEAEs	5.7 (4.5, 7.1)	4.9 (3.7, 6.3)	4.5 (3.3, 5.8)	5.1 (3.6, 6.9)	4.5 (3.9, 5.2)	3.5 (2.3, 5.2)
TEAEs leading to death	0.2 (0.0, 0.6)	0.4 (0.1, 0.9)	0.5 (0.2, 1.1)	0.2 (0.0, 0.9)	0.3 (0.2, 0.5)	0.5 (0.1, 1.4)
TEAEs of interest, EAIR/100 PY (95% CI)						
Serious infections	1.5 (0.9, 2.3)	0.7 (0.3, 1.3)	1.3 (0.8, 2.2)	1.1 (0.5, 2.1)	1.2 (0.9, 1.5)	0.7 (0.2, 1.6)
Active tuberculosis	0	0	0	0	0	0
Fungal infections	30.5 (27.4, 33.8)	19.8 (17.3, 22.5)	11.9 (10.0, 14.1)	8.5 (6.6, 10.8)	14.4 (13.2, 15.7)	13.2 (10.3, 16.7)
<i>Candida</i> infections	22.5 (19.9, 25.3)	14.1 (12.0, 16.3)	8.0 (6.5, 9.8)	5.6 (4.1, 7.6)	9.7 (8.8, 10.8)	8.8 (6.6, 11.6)
Oral candidiasis	19.3 (16.9, 21.9)	12.5 (10.6, 14.7)	7.1 (5.7, 8.8)	5.3 (3.8, 7.1)	8.6 (7.7, 9.5)	8.4 (6.2, 11.1)
Definite or probable adjudicated IBD	0.4 (0.1, 0.8)	0.1 (0.0, 0.4)	0.1 (0.0, 0.5)	0.1 (0.0, 0.7)	0.2 (0.1, 0.3)	0.1 (0.0, 0.8)
Adjudicated MACE	0.7 (0.3, 1.3)	0.2 (0.0, 0.7)	0.6 (0.2, 1.2)	1.1 (0.5, 2.1)	0.6 (0.4, 0.9)	1.0 (0.4, 2.0)
Malignancies	0.8 (0.4, 1.4)	0.8 (0.4, 1.5)	1.1 (0.6, 1.8)	1.0 (0.4, 1.9)	0.8 (0.6, 1.1)	1.4 (0.7, 2.5)
Malignancies (excluding NMSC)	0.4 (0.1, 0.8)	0.5 (0.2, 1.1)	0.9 (0.5, 1.6)	0.9 (0.3, 1.8)	0.6 (0.4, 0.9)	0.9 (0.4, 1.9)
Adjudicated SIB	0.1 (0.0, 0.4)	0.2 (0.0, 0.6)	0.2 (0.0, 0.6)	0	0.1 (0.0, 0.2)	0
Neutropenia	0.9 (0.5, 1.6)	0.3 (0.1, 0.8)	0.1 (0.0, 0.5)	0.2 (0.0, 0.9)	0.4 (0.2, 0.6)	0.1 (0.0, 0.8)
ALT or AST >3× ULN	2.7 (1.9, 3.7)	2.6 (1.8, 3.6)	2.2 (1.4, 3.2)	1.7 (0.9, 2.9)	1.9 (1.5, 2.4)	1.1 (0.5, 2.2)
ALT or AST >5× ULN	1.0 (0.6, 1.7)	0.4 (0.1, 0.9)	0.5 (0.2, 1.1)	0.6 (0.2, 1.4)	0.6 (0.4, 0.9)	0.3 (0.0, 1.0)
Serious hypersensitivity reactions	0.1 (0.0, 0.5)	0.2 (0.0, 0.6)	0	0	0.1 (0.0, 0.2)	0.1 (0.0, 0.8)
Injection site reactions	3.2 (2.3, 4.3)	1.2 (0.7, 2.0)	1.2 (0.7, 2.1)	0.4 (0.1, 1.1)	1.6 (1.3, 2.0)	2.1 (1.2, 3.5)

Data are presented for those who received ≥1 BKZ dose during the full pooled trial period over 4 years, and separately for Years 1 (Weeks 0-52), 2 (Weeks >52-104), 3 (Weeks >104-156), and 4 (Weeks >156-208). Data are also presented for the subset of patients who received BKZ Q4W from baseline to Week 16, then Q8W throughout the double-blinded study periods and into the OLE (BKZ Q4W/Q8W). At Year 2 (Week 100/104 or next scheduled clinic visit), all patients receiving BKZ Q4W switched to Q8W dosing.

ALT, alanine aminotransferase; AST, aspartate aminotransferase; BKZ, bimekizumab; CI, confidence interval; EAIR, exposure-adjusted incidence rate; IBD, inflammatory bowel disease; MACE, major adverse cardiac event; NMSC, non-melanoma skin cancer; PY, patient-years; Q4W, every 4 weeks; Q8W, every 8 weeks; SIB, suicidal ideation and behavior; TEAE, treatment-emergent adverse event; ULN, upper limit of normal.

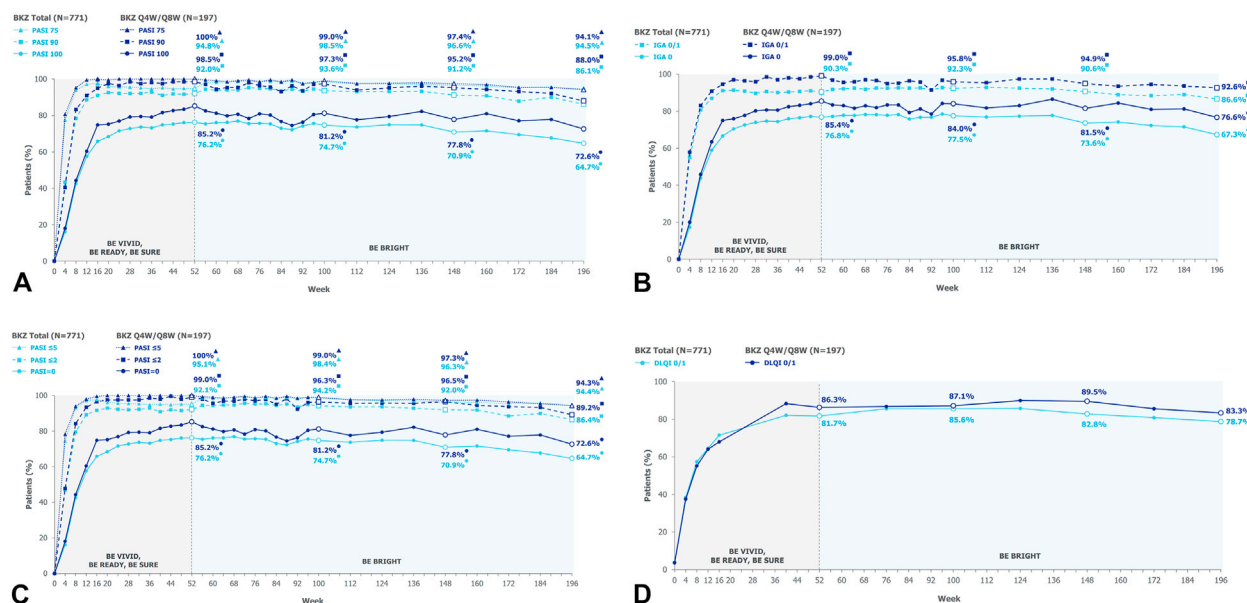


Fig 1. Efficacy outcome responses through 196 weeks (mNRI). **A**, PASI 100, PASI 90, and PASI 75; **B**, IGA 0 and IGA 0/1; **C**, PASI = 0, PASI ≤ 2, and PASI ≤ 5; **D**, DLQI 0/1. Observed case data for these efficacy outcome responses through 196 weeks are presented in the Supplementary Material, available via Mendeley at data.mendeley.com/datasets/24zjytd7z/2. The BKZ total group includes all patients who were randomized to BKZ Q4W at feeder study baseline, received BKZ Q4W or Q8W throughout the double-blinded study period, and entered the OLE. The BKZ Q4W/Q8W group includes the subset of patients who received BKZ Q4W from baseline to Week 16, then Q8W throughout the double-blinded study periods and into the OLE. BE VIVID lasted 52 weeks and BE SURE and BE READY lasted 56 weeks; to pool data across studies, Week 56 data were not included. For DLQI, Week 52 corresponds to the Week 48 assessment for BE SURE and BE READY, and Week 52 for BE VIVID, due to the lack of common visits at which DLQI 0/1 was recorded in these studies. *BKZ*, bimekizumab; *DLQI*, Dermatology Life Quality Index; *IGA*, Investigator's Global Assessment; *mNRI*, modified non-responder imputation; *OLE*, open-label extension; *PASI*, Psoriasis Area and Severity Index; *PASI 100/90/75*, 100%/≥90%/≥75% improvement from baseline in PASI; *Q4W*, every 4 weeks; *Q8W*, every 8 weeks.

Among bimekizumab total patients (mNRI), 76.2% achieved complete skin clearance (PASI 100/PASI = 0) at Week 52 and 64.7% sustained this to Week 196 (Fig 1, A and C); 92.0% achieved PASI 90 at Week 52 and 86.1% maintained this to Week 196 (Fig 1, A); 94.8% achieved PASI 75 at Week 52 and 94.5% at Week 196 (Fig 1, A). Regarding IGA, 76.8% achieved IGA 0 at Week 52 and 67.3% at Week 196, and 90.3% achieved IGA 0/1 at Week 52 and 86.6% at Week 196 (Fig 1, B). Absolute PASI responses were similar to relative PASI responses: 92.1%/93.9%/95.1% achieved PASI ≤ 2/≤ 3/≤ 5 at Week 52 and 86.4%/91.2%/94.4% at Week 196 (Fig 1, C); BSA ≤ 1%/≤ 3% data followed similar trends (Supplementary Fig 3, available via Mendeley at <https://data.mendeley.com/datasets/24zjytd7z/2>). Regarding quality of life, 81.2% achieved DLQI 0/1 at Week 52 and 78.7% at Week 196 (Fig 1, D). PASI, IGA, BSA, and DLQI responses were similar in the subgroup receiving bimekizumab Q4W/Q8W. Observed case data are shown in Supplementary Fig 4, available via

Mendeley at <https://data.mendeley.com/datasets/24zjytd7z/2>.

Safety outcomes

The rate of TEAEs was 169.8/100 PY over 4 years (Year 1: 237.4/100 PY; Year 2: 150.3/100 PY; Year 3: 105.4/100 PY; Year 4: 100.5/100 PY; Table I); the 4-year rate for bimekizumab Q4W/Q8W patients was 155.5/100 PY. Serious TEAEs (5.5/100 PY) and TEAEs leading to discontinuation (2.8/100 PY) were low and did not increase with longer exposure (Table I). The vast majority (97.1%) of TEAEs were mild or moderate; the rate of severe TEAEs was 4.5/100 PY.

The 3 most common TEAEs reported over the 4 years, by MedDRA preferred term, were nasopharyngitis (13.0/100 PY), oral candidiasis (8.6/100 PY), and upper respiratory tract infection (5.8/100 PY). Coronavirus infection was fourth most common (4.4/100 PY); the rate of serious coronavirus infections was 0.3/100 PY.

Over 4 years, 16 deaths (0.3/100 PY) were reported (Table I); none were reported as treatment-related by the investigator. All deaths over 4 years, including causes of death, have been included and described in the previous 3-year safety report.²⁰

Rates of infections and serious infections were 76.0 and 1.2/100 PY, respectively; serious infection rates were consistent through 4 years (Table I). The rate of *Candida* infections was 9.7/100 PY; most were oral candidiasis (8.6/100 PY). Yearly oral candidiasis rates did not increase with longer exposure (Table I) and the vast majority of events were mild or moderate in severity (99.4%); 5 were severe, none were serious. Five oral candidiasis events led to discontinuation over 4 years; 3 in Year 1, 1 in Year 2, and 1 in Year 3. Most cases were treated with nystatin and/or fluconazole; median (interquartile range) duration of anti-fungal therapy was 13.0 (7.0-26.0) days.

The rate of definite or probable adjudicated inflammatory bowel disease (IBD) was 0.2/100 PY and did not increase with longer bimekizumab exposure (Table I). Over 4 years, 8 patients had events adjudicated as definite IBD (2 Crohn's disease, 1 ulcerative colitis, 1 IBD unclassified by Adjudication Committee) or probable IBD (1 ulcerative colitis, 3 classified as IBD with no further differentiation possible by Adjudication Committee); one of these patients had a history of IBD.

Rates of adjudicated suicidal ideation and behavior and laboratory elevations in either alanine aminotransferase or aspartate aminotransferase were low and aligned with previous observations (Table I).^{20,24,25}

Four-year rates for other TEAEs of interest are reported in Table I. Rates of TEAEs of interest were similar in the subset of patients receiving bimekizumab Q4W/Q8W (Table I).

DISCUSSION

In these pooled analyses of long-term phase 3 data, bimekizumab demonstrated rapid onset and sustained efficacy over 4 years, across various efficacy and quality of life measures, including PASI, IGA, BSA, and DLQI. After 4 years of treatment, almost two-thirds of patients (64.7%) achieved complete skin clearance (PASI 100) and a large proportion (86.1%) achieved near-complete skin clearance (PASI 90); almost all (94.5%) had PASI 75. After 1 dose (Week 4), PASI 90 and PASI 100 rates were 43.3% and 16.1%, respectively, demonstrating rapid treatment response, an important outcome for patients.²⁶ Comparatively, reported Week 4 PASI 90 rates with the IL-17A inhibitors secukinumab and ixekizumab are 16.7% and 21% to 26%, and PASI 100 rates are 5.7% and 4% to 8%, respectively.^{27,28} These results reinforce bimekizumab's role as an effective, durable

treatment for the long-term management of patients with psoriasis.

Complete skin clearance is now attainable for many patients with moderate to severe psoriasis, due to high efficacy with latest-generation biologics. Not only is it the most stringent clinical outcome, but complete skin clearance also translates into the greatest benefits in health-related quality of life and patient-perceived symptoms.^{19,29} High levels of PASI 100 responses maintained over 4 years with bimekizumab may raise treatment goals for patients, thereby maximally improving their well-being. Additionally, a large majority of patients treated with bimekizumab (78.7%) reported their skin disease having no effect on quality of life (DLQI 0/1) after 4 years, further supporting bimekizumab's positive impact on patient well-being.

Clinical trials of the IL-17A inhibitors ixekizumab and secukinumab, and IL-23 inhibitors guselkumab and risankizumab, reported Year 4 PASI 90 response rates of 66.4%, 54.1%, 79.7%, and 83.6%, and Year 4 PASI 100 responses of 48.3%, 28.7%, 51.0%, and 52.8%, respectively (using mNRI, multiple imputation, or treatment failure rules).³⁰⁻³³ Since no long-term head-to-head studies have been conducted, direct comparisons cannot be made between treatments, as many variables differ, including study design and patient populations. Nonetheless, awareness of the differences in imputation methodology is important to properly interpret long-term psoriasis clinical trial data.³⁴ Long-term bimekizumab analyses used a strict mNRI definition, where patients who discontinued due to lack of efficacy or treatment-related AEs were considered non-responders. PASI responses over 4 years remained high despite the expected decline over time with the stringent mNRI imputation method.

Over 4 years of treatment, yearly treatment-emergent adverse event rates did not increase with longer exposure to bimekizumab, although a greater proportion of patients received Q8W dosing in Years 3 and 4 versus Years 1 and 2, which may have influenced rates. Bimekizumab's safety profile has remained consistent throughout 4 years, as compared with previous 2- and 3-year safety reports.^{20,35} Serious TEAEs and TEAEs leading to discontinuation remained low throughout 4 years. Serious infection rates were also low throughout, despite the study overlapping with the COVID-19 pandemic, and were consistent with long-term reports of other biologic treatments for psoriasis and the PSoriasis Longitudinal Assessment and Registry range.^{33,36} The COVID-19 pandemic may have altered 3- and 4-year AE rates, particularly respiratory infections like nasopharyngitis, due to confounding factors such as mask-wearing, social isolation, and

national lockdowns, although this impact is difficult to assess as infection control methods varied globally.

Treatments inhibiting the IL-17 pathway can increase risk of oral candidiasis, as IL-17 has a protective role against fungal infections in the oral mucosa.³⁷⁻⁴¹ Nevertheless, yearly oral candidiasis rates did not increase with longer exposure to bimekizumab. Over the 4-year period, nearly all cases of oral candidiasis were mild or moderate (99.4%), and only 5 (1.5%) led to discontinuation of bimekizumab, indicating that these events were well-tolerated.

Prolonged exposure to bimekizumab did not result in increased rates of liver transaminase elevations; most occurrences were transient and rarely led to discontinuation, aligning with a previous analysis on liver parameters.²⁵ Suicidal ideation and behavior rates were low through 4 years (0.1/100 PY), aligning with a previous in-depth study across 9 trials.²⁴ Malignancies (excluding non-melanoma skin cancer, 0.6/100 PY) and adjudicated major adverse cardiac events (0.6/100 PY) were low throughout 4 years and within PSORIASIS Longitudinal Assessment and Registry ranges.^{36,42} Meanwhile, the rate of definite or probable adjudicated IBD was 0.2/100 PY over 4 years, and yearly rates did not increase with longer exposure.

These analyses include data from the patient subgroup who received the bimekizumab dosing regimen approved for most patients with psoriasis (Q4W/Q8W),^{9,10} providing important supporting evidence for this regimen, which is now used in real-world clinical settings. However, this subgroup contained a smaller number of patients, so variability in results can be expected.

The large sample size overall, including data across 3 phase 3 trials and their OLE, strengthens these analyses. Moreover, the discontinuation rate among bimekizumab-randomized patients who entered the OLE was low (16.3% discontinued during the OLE), despite this overlapping with the COVID-19 pandemic (1.2% due to lack of efficacy and 5.7% due to AEs). Although, generalizability to real-world populations is limited by eligibility criteria and minimal racial diversity within the trials.

In conclusion, bimekizumab demonstrated high, durable levels of clinical and health-related quality of life responses over 4 years of treatment, with a large majority of patients achieving near-complete skin clearance and almost two-thirds achieving complete skin clearance. Bimekizumab was well-tolerated, as indicated by low rates of discontinuations due to TEAEs. Treatment-emergent adverse event rates did not increase with longer exposure to bimekizumab, and safety data were consistent with bimekizumab's

known safety profile. These results demonstrate that bimekizumab is an effective long-term treatment option for patients with moderate to severe plaque psoriasis.

ETHICS APPROVAL

All studies were conducted in accordance with the principles of the Declaration of Helsinki and were approved by an Independent Review Board and Independent Ethics Committee. All participants provided informed written consent.

DATA SHARING

Underlying data from this manuscript may be requested by qualified researchers 6 months after product approval in the US and/or Europe, or global development is discontinued, and 18 months after trial completion. Investigators may request access to anonymized individual patient-level data and redacted trial documents, which may include analysis-ready datasets, study protocol, annotated case report form, statistical analysis plan, dataset specifications, and clinical study report. Prior to use of the data, proposals need to be approved by an independent review panel at www.Vivli.org and a signed data sharing agreement will need to be executed. All documents are available in English only, for a prespecified time, typically 12 months, on a password protected portal.

CONSENT FOR PUBLICATION

All the results presented in this article are in aggregate form, and no personally identifiable information was used for this study.

The authors thank the patients, the investigators, and their teams who took part in this study. The authors also acknowledge Inés Dueñas Pousa, PhD, UCB, Madrid, Spain, for publication coordination, and Yasha Najafi, MSc, and Daniel Smith, MA, from Costello Medical, UK, for medical writing and editorial assistance based on the authors' input and direction.

Conflicts of interest

All details of authors' affiliation or involvement in an organization or entity with a financial or nonfinancial interest in the subject matter or materials discussed in this manuscript are disclosed. Dr Blauvelt has served as a speaker (received honoraria) for Eli Lilly and Company and UCB; has served as a scientific adviser (received honoraria) for AbbVie, Almirall, Alumis, Amgen, AnaptysBio, Apogee, Arcutis, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, Eli Lilly and Company, Galderma, GlaxoSmithKline, Incyte, IQVIA, Janssen, LEO Pharma, Lipidio, Merck, Novartis, Oruka, Paragon, Pfizer, Regeneron, Sanofi, Spherix Global Insights, Sun Pharma, Takeda, UCB, and Union; has acted as a clinical study

investigator (institution has received clinical study funds) for AbbVie, Acelyrin, Almirall, Alumis, Amgen, Arcutis, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, Eli Lilly and Company, Galderma, Incyte, Janssen, LEO Pharma, Merck, Novartis, Pfizer, Regeneron, Sanofi, Sun Pharma, Takeda, and UCB; and owns stock in Lipidio and Oruka. Dr Langley has been a principal investigator for AbbVie, Amgen, Boehringer Ingelheim, Celgene, Eli Lilly and Company, LEO Pharma, Merck, Novartis, Pfizer, and UCB; served on scientific advisory boards for AbbVie, Amgen, Boehringer Ingelheim, Celgene, Eli Lilly and Company, LEO Pharma, Merck, Novartis, Pfizer, and UCB; provided lectures for AbbVie, Amgen, Celgene, Eli Lilly and Company, LEO Pharma, Merck, Novartis, and Pfizer. Dr Lebwohl is an employee of Mount Sinai and receives research funds from AbbVie, Amgen, Arcutis, Avotres Therapeutics, Boehringer Ingelheim, Cara Therapeutics, Dermavant Sciences, Eli Lilly and Company, Incyte, Inozyme, Janssen Research & Development, LLC, Ortho Dermatologics, Pfizer, Sanofi-Regeneron, and UCB; consultant for Almirall, AltruBio Inc, AnaptysBio, Apogee, Arcutis, AstraZeneca, Atomwise, Avotres Therapeutics, Brickell Biotech, Boehringer-Ingelheim, Bristol Myers Squibb, Castle Biosciences, Celltrion, CorEvitas, Dermavant Sciences, EPI, Evommune Inc, Facilitation of International Dermatology Education, Forte Biosciences, Foundation for Research and Education in Dermatology, Galderma, Genentech, Incyte, LEO Pharma, Meiji Seika Pharma, Mindera, Pfizer, Sanofi-Regeneron, Seanergy, Strata, Takeda, Trevi, and Verrica. Dr Strober received consultancy honoraria from AbbVie, Acelyrin, Alamar, Almirall, Alumis, Amgen, Arcutis, Arena, Aristea, Asana, Boehringer Ingelheim, Bristol Myers Squibb, Capital One, Celltrion, CorEvitas, Dermavant, Eli Lilly and Company, Imagenebio, Janssen, Kangpu Pharmaceuticals, LEO Pharma, Maruho, Meiji Seika Pharma, Protagonist, Monte Carlo, Novartis, Pfizer, Rapt, Regeneron, Sanofi Genzyme, SG Cowen, Sun Pharma, Takeda, UCB, Union Therapeutics, Ventyxbio, and vTv Therapeutics; has stock options from Connect Biopharma, Mindera Health; was a speaker for AbbVie, Arcutis, Dermavant, Eli Lilly and Company, Incyte, Janssen, Regeneron, and Sanofi Genzyme; serves as a Scientific Co-Director (consulting fee) for CorEvitas Psoriasis Registry; serves as an investigator for CorEvitas Psoriasis Registry; serves as the editor-in-chief (honorarium) of *Journal of Psoriasis and Psoriatic Arthritis*. Dr Warren has received consulting fees from AbbVie, Almirall, Amgen, Arena, Astellas, Avillion, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Eli Lilly and Company, GSK, Janssen, LEO Pharma, Novartis, Pfizer, Sanofi, and UCB; research grants to his institution from AbbVie, Almirall, Janssen, LEO Pharma, Novartis, and UCB; honoraria from AbbVie, Almirall, Bristol Myers Squibb, Eli Lilly and Company, Galderma, Janssen, and Novartis. Dr Puig received consultancy/speaker's honoraria from and/or participated in trials sponsored by AbbVie, Almirall, Amgen, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, Eli Lilly and Company, Fresenius Kabi, Horizon,

Janssen, LEO Pharma, Novartis, Pfizer, Sandoz, STADA, Sun Pharma, and UCB. Dr Morita has received research grants, consulting fees, and/or speaker's fees from AbbVie, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Eisai, Eli Lilly and Company, Janssen, Kyowa Hakko Kirin, LEO Pharma, Maruho, Mitsubishi Tanabe Pharma, Nichi-Iko, Nippon Kayaku, Novartis, Pfizer, Sun Pharma, Taiho Pharmaceutical, Torii Pharmaceutical, UCB, and Ushio. Dr Gordon has received consulting fees from AbbVie, Almirall, Amgen, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Dermira, Eli Lilly and Company, Janssen, Novartis, Pfizer, Sun Pharma, and UCB; research support from AbbVie, Bristol Myers Squibb, Celgene, Eli Lilly and Company, Janssen, Novartis, and UCB. Dr Fernandez-Peñas has served on advisory boards for AbbVie, Amgen, Boehringer Ingelheim, Bristol Myers Squibb, Eli Lilly and Company, Janssen, L'Oreal, LEO Pharma, Merck, Merck Sharp & Dohme, Novartis, Pfizer, Sanofi, and UCB; has given educational lectures for AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly and Company, Galderma, Janssen, L'Oreal, LEO Pharma, Merck, Merck Sharp & Dohme, Novartis, Pfizer, Sanofi, UCB, and Zuellig Pharma; has conducted clinical trials for AbbVie, Akaal, Akesobio, Amgen, Arena, AstraZeneca, Boehringer Ingelheim, Bristol Myers Squibb, CSL, Eisai, Eli Lilly and Company, Galderma, Incyte, Janssen, Jiangsu Hengrui, KoBioLabs, Kyowa Hakko Kirin, Merck, Merck Sharp & Dohme, miRagen, Moderna, Nektar, Novartis, OncoSec, Pfizer, Regeneron, Sanofi, Sun Pharma, and UCB. Author Kavanagh is a consultant for Aclipse Therapeutics, Aliada Therapeutics, Allay Therapeutics, Autobahn Therapeutics, Cognition Therapeutics, Colorado Prevention Center, Karuna Therapeutics, Kisbee Therapeutics, LB Pharma, Nesos, Novartis, Onward, PharPoint Research, Summit Analytical, Tonix, Tornado Therapeutics, UCB, Whitsell Innovations, Worldwide Clinical Trials, and Zosano. Drs López Pinto, Lambert, Hoepken, Author Deherder, and Dr Cross are employees and shareholders of UCB. Dr Thaçi is an investigator and/or consultant/advisor for AbbVie, Almirall, Amgen, Boehringer Ingelheim, Bristol Myers Squibb, Celltrion, Eli Lilly and Company, Galderma, Janssen-Cilag, Kyowa Kirin, LEO Pharma, L'Oreal, New Bridge, Novartis, Pfizer, Regeneron, Samsung, Sanofi, Target-RWE, and UCB; has received grants from AbbVie, LEO Pharma, and Novartis.

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