

Bimekizumab efficacy and safety in patients with psoriatic arthritis and psoriasis: Up to 2-year results from two phase 3 studies

Diamant Thaçi,¹ Alice B Gottlieb,² Akihiko Asahina,³ Mark Lebwohl,² Iain B McInnes,⁴ Richard B Warren,^{5,6} Wolf-Henning Boehncke,⁷ Barbara Ink,⁸ Rajan Bajracharya,⁸ Jason Coarse,⁹ Joseph F Merola¹⁰

Objective

To assess up to 2-year efficacy and safety of bimekizumab (BKZ) in patients with psoriatic arthritis (PsA) and psoriasis who were biologic disease-modifying antirheumatic drug (bDMARD)-naïve or had prior inadequate response or intolerance to tumour necrosis factor inhibitors (TNFi-IR).

Background

- BKZ, a humanised monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated efficacy and tolerability to 1 year in patients with PsA.^{1,2}
- PsA develops in up to 30% of patients with psoriasis,³ therefore investigating the efficacy and safety of treatments in patients with PsA and skin involvement is of clinical importance.

Methods

- BE OPTIMAL (NCT03895203; bDMARD-naïve) and BE COMPLETE (NCT03896581; TNFi-IR), both placebo (PBO)-controlled to Week 16, assessed subcutaneous BKZ 160 mg every 4 weeks (Q4W) in patients with PsA; PBO-randomised patients switched to BKZ (PBO/BKZ) at Week 16.^{1,2}
- BE OPTIMAL included a reference arm (adalimumab [ADA] 40 mg Q2W); these patients switched to BKZ at Week 52 (ADA/BKZ) with no washout between treatments.
- BE OPTIMAL Week 52 and BE COMPLETE Week 16 completers were eligible to enrol in the open-label extension BE VITAL (NCT04009499).
- Post hoc data are reported for patients with psoriasis affecting $\geq 3\%$ body surface area (BSA) at baseline; analyses were also conducted by psoriasis severity $\geq 3\%$ – $\leq 10\%$ and $>10\%$ BSA (data not shown).
- Efficacy outcomes are reported to Week 104 from BE OPTIMAL and Week 100 from BE COMPLETE. Missing data were imputed using non-responder (NRI; binary), multiple (MI; continuous) or worst category (WCI; categorical) imputation.

Results

Patient Characteristics

- Of patients with baseline psoriasis BSA $\geq 3\%$, 365/425 (85.9%) bDMARD-naïve and 216/264 (81.8%) TNFi-IR patients completed Week 104/100 of BE OPTIMAL and BE COMPLETE, respectively.
- Baseline characteristics were generally similar across treatment arms within trials.

Efficacy

- Efficacy responses seen at Week 52 with BKZ were sustained to Week 104/100, with high proportions of bDMARD-naïve and TNFi-IR patients achieving $\geq 50\%$ improvement from baseline in American College of Rheumatology (ACR50), 100% improvement from baseline in Psoriasis Area and Severity Index (PASI100), and minimal disease activity (MDA) responses (Figure 1).
- Efficacy responses were sustained following the switch from ADA to BKZ at Week 52, and a further improvement in PASI100 response was observed.
- Previously reported improvements in additional efficacy outcomes at Week 52 were also sustained to Week 104/100 (Table 1).
- Results were generally similar across the $\geq 3\%$ – $\leq 10\%$ and $>10\%$ BSA subgroups (data not shown).

Safety

- Table 2 presents safety data for patients treated with BKZ with baseline psoriasis $\geq 3\%$ BSA.
- In those treated with BKZ, the exposure-adjusted incidence rates per 100 patient-years (EAIR/100 PY) for ≥ 1 treatment-emergent adverse event (TEAE) were 157.2 and 84.4 for bDMARD-naïve and TNFi-IR patients for Week 0–104, respectively.
- Incidence rates (EAIR/100 PY) of serious TEAEs were 5.3 (bDMARD-naïve) and 4.6 (TNFi-IR).
- Over 2 years, 3 deaths occurred (bDMARD-naïve: 2 [1 before and 1 after Week 52], TNFi-IR: 1 [before Week 52]), all deemed unrelated to study treatment.
- Of reported *Candida* infections (EAIR/100 PY; bDMARD-naïve: 5.8, TNFi-IR: 2.5), none were serious or systemic; 3 infections led to study discontinuation (bDMARD-naïve: 2, TNFi-IR: 1).

Conclusions

Bimekizumab treatment resulted in sustained clinical efficacy up to 2 years, with observed improvements generally similar in bDMARD-naïve and TNFi-IR patients with PsA and psoriasis. Bimekizumab was well tolerated and no new safety signals were observed.^{1,2}

Summary

Efficacy and safety of bimekizumab treatment were assessed up to 2 years in patients with PsA and psoriasis who were bDMARD-naïve (BE OPTIMAL) or TNFi-IR (BE COMPLETE)

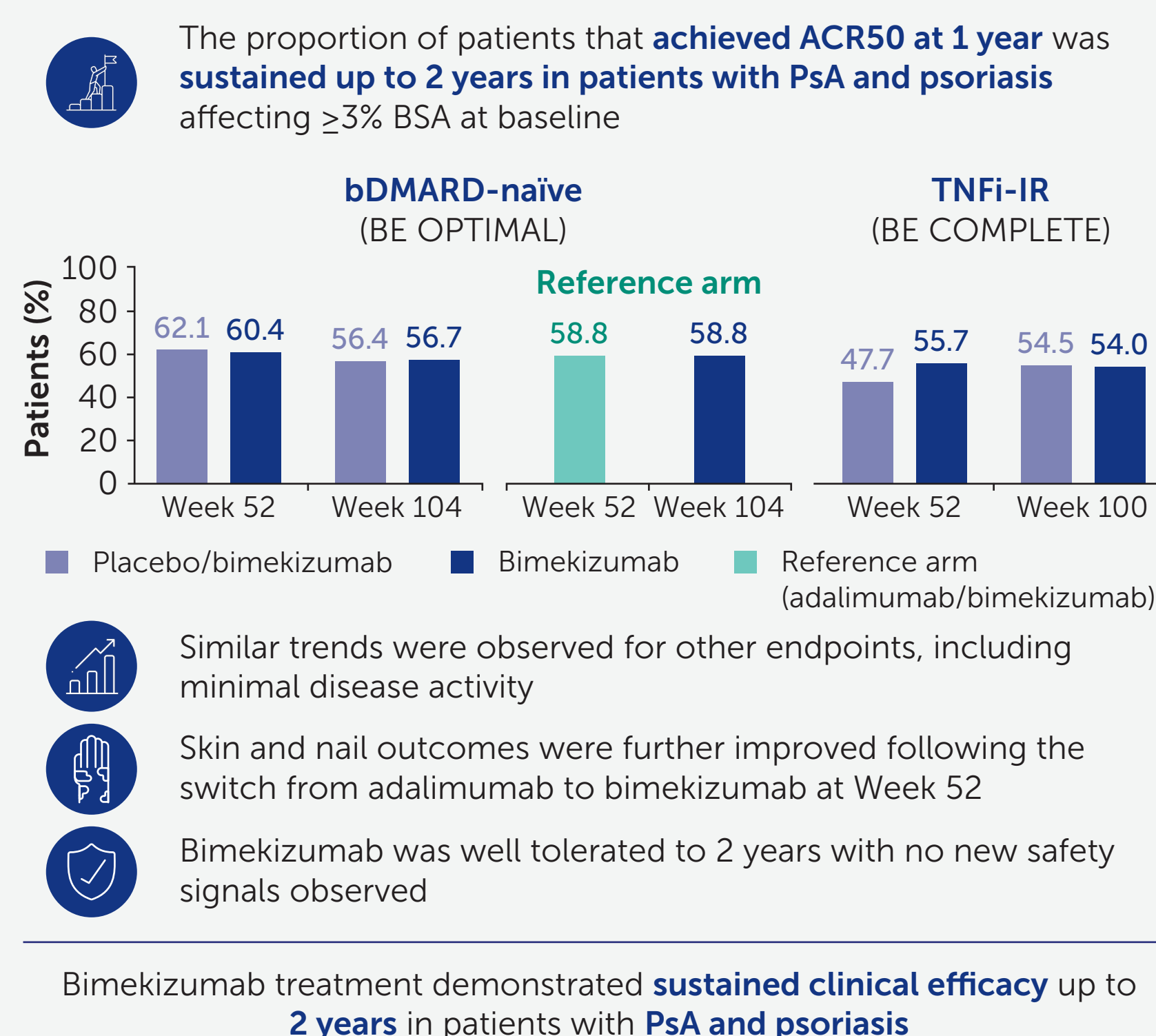
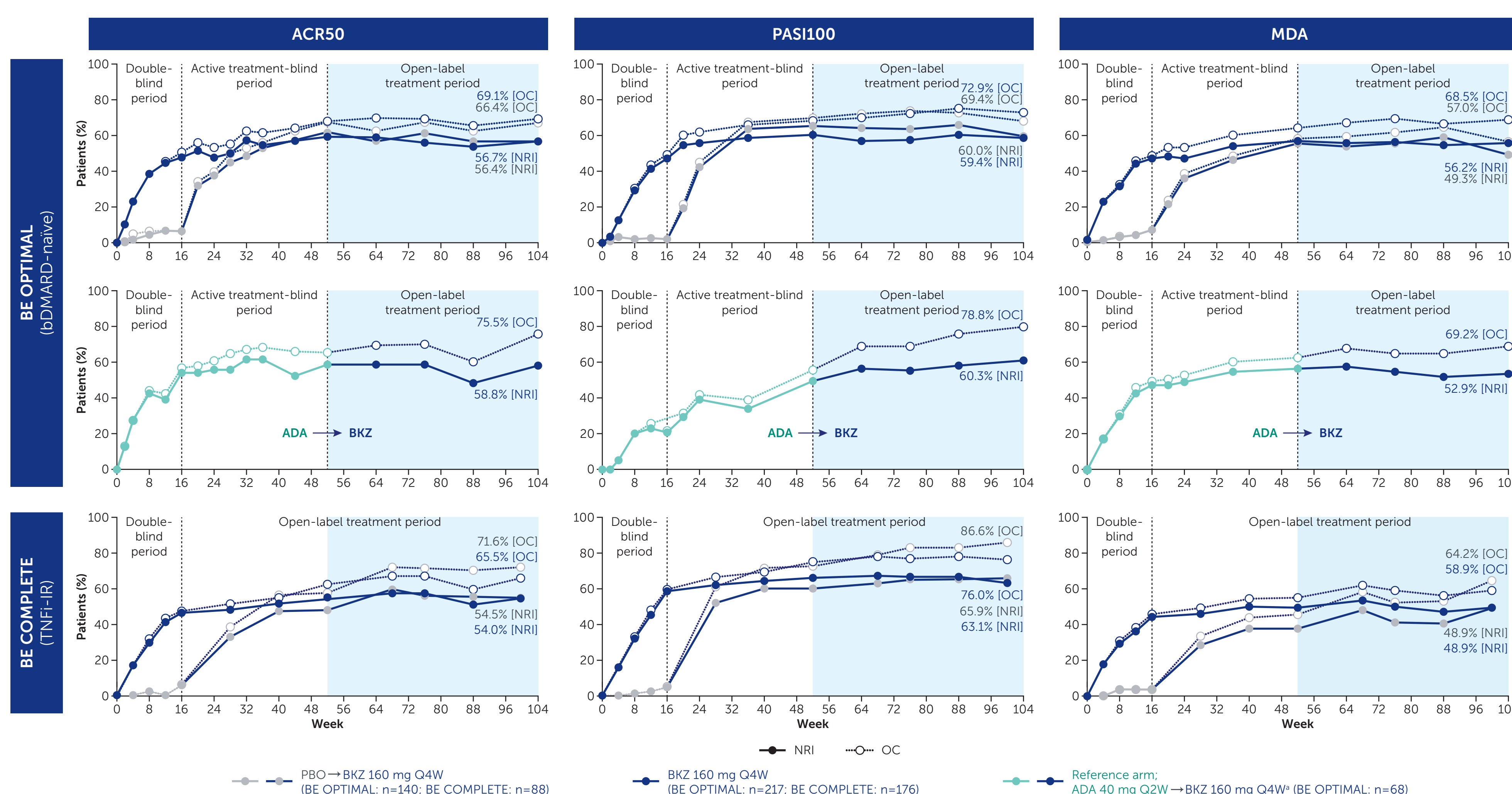


Table 1 Additional efficacy outcomes for patients with baseline psoriasis ($\geq 3\%$ BSA) at Week 104/100 (MI, NRI, WCI)

| | BE OPTIMAL (bDMARD-naïve) Week 104 | | | BE COMPLETE (TNFi-IR) Week 100 | |
|--|------------------------------------|----------------------|-----------------------------------|--------------------------------|----------------------|
| | PBO→BKZ 160 mg Q4W n=140 | BKZ 160 mg Q4W n=217 | ADA 40 mg Q2W→BKZ 160 mg Q4W n=68 | PBO→BKZ 160 mg Q4W n=88 | BKZ 160 mg Q4W n=176 |
| ACR20 responders, n (%) | 98 (70.0) | 151 (69.6) | 45 (66.2) | 64 (72.7) | 121 (68.8) |
| ACR70 responders, n (%) | 54 (38.6) | 90 (41.5) | 30 (44.1) | 33 (37.5) | 66 (37.5) |
| PASI75 responders, n (%) | 112 (80.0) | 162 (74.7) | 49 (72.1) | 63 (71.6) | 142 (80.7) |
| PASI90 responders, n (%) | 102 (72.9) | 153 (70.5) | 47 (69.1) | 61 (69.3) | 128 (72.7) |
| ACR50 + PASI100 responders, n (%) | 60 (42.9) | 93 (42.9) | 33 (48.5) | 42 (47.7) | 74 (42.0) |
| VLDA responders, n (%) | 39 (27.9) | 71 (32.7) | 24 (35.3) | 16 (18.2) | 44 (25.0) |
| DAPSA disease state [WCI], n (%) | | | | | |
| LDA+REM | 71 (50.7) | 122 (56.2) | 36 (52.9) | 50 (56.8) | 88 (50.0) |
| REM | 28 (20.0) | 54 (24.9) | 23 (33.8) | 14 (15.9) | 27 (15.3) |
| TJC=0 (of 68 joints), n (%) | 46 (32.9) | 77 (35.5) | 25 (36.8) | 21 (23.9) | 54 (30.7) |
| SJC=0 (of 66 joints), n (%) | 89 (63.6) | 145 (66.8) | 39 (57.4) | 55 (62.5) | 107 (60.8) |
| Enthesitis resolution ^a , n/N (%) | 26/34 (76.5) | 41/61 (67.2) | 9/15 (60.0) | 10/20 (50.0) | 39/63 (61.9) |
| Dactylitis resolution ^b , n/N (%) | 10/10 (100.0) | 21/27 (77.8) | 5/8 (62.5) | 4/7 (57.1) | 19/21 (90.5) |
| Nail psoriasis resolution ^c , n/N (%) | 63/88 (71.6) | 91/133 (68.4) | 32/42 (76.2) | 34/54 (63.0) | 72/105 (68.6) |
| HAQ-DI CIB [MI], mean (SE) | -0.42 (0.05) | -0.39 (0.04) | -0.50 (0.08) | -0.49 (0.07) | -0.45 (0.04) |
| SF-36 PCS CFB [MI], ^d mean (SE) | 10.0 (0.8) | 9.8 (0.7) | 11.8 (1.3) | 9.8 (1.2) | 9.6 (0.8) |

Randomised set, in patients with psoriasis affecting $\geq 3\%$ BSA at baseline. Missing data were imputed as NRI unless otherwise stated. [a] Reference arm; study not powered for statistical comparisons of ADA to BKZ or PBO. [b] In patients with baseline enthesitis (LEI >0). [c] In patients with baseline dactylitis (LDI >0). [d] In patients with baseline nail psoriasis (mNAPSI >0). [e] SF-36 PCS data collected to Week 88 only in BE COMPLETE.

Figure 1 Proportion of patients with baseline psoriasis ($\geq 3\%$ BSA) achieving ACR50, PASI100 and MDA over time to Week 104/100 (NRI, OC)



Randomised set, in patients with psoriasis affecting $\geq 3\%$ BSA at baseline. In BE OPTIMAL, patients were randomised 3:2:1 to BKZ 160 mg Q4W/PBO reference arm (ADA 40 mg Q2W), in BE COMPLETE patients were randomised 2:1 to BKZ 160 mg Q4W/PBO. In both studies, patients on PBO switched to BKZ 160 mg Q4W at Week 16. In BE OPTIMAL, patients on ADA switched to BKZ 160 mg Q4W at Week 52. The period highlighted in blue represents data not previously reported. [a] Reference arm; study not powered for statistical comparisons of ADA to BKZ or PBO.

Table 2 Safety to Week 52 and Week 104

| | BE OPTIMAL (bDMARD-naïve) | | | | BE COMPLETE (TNFi-IR) | |
|--|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|
| | Week 0–52 | Week 0–104 | Week 0–52 | Week 0–104 | Week 0–52 | Week 0–104 |
| EAIR/100 PY (95% CI) | | | | | | |
| Any TEAEs | 186.5 (164.8, 210.3) | 203.2 (153.1, 264.4) | 155.2 (138.3, 173.6) | 172.3 (124.7, 232.1) | 102.3 (86.1, 120.5) | 84.4 (72.7, 97.6) |
| Serious TEAEs | 6.0 (3.6, 9.5) | 6.1 (1.7, 15.6) | 5.5 (3.8, 7.7) | 1.7 (0.0, 9.6) | 6.9 (3.9, 11.4) | 4.6 (2.8, 7.2) |
| Severe TEAEs, n (%) | 12 (3.4) | 5 (7.4) | 24 (6.7) | 1 (1.7) | 12 (4.7) | 16 (6.3) |
| Study discontinuation due to TEAEs | 3.3 (1.6, 6.0) | 6.1 (1.7, 15.7) | 3.1 (1.9, 4.8) | 5.2 (1.1, 15.3) | 4.1 (1.9, 7.7) | 3.4 (1.9, 5.6) |
| Drug-related TEAEs, n (%) | 90 (25.3) | 25 (36.8) | 121 (34.0) | 18 (30.0) | 51 (20.0) | 70 (27.5) |
| Deaths, n (%) | 1 (0.3) ^a | 0 | 1 (0.3) ^a | 1 (1.7) ^a | 1 (1.0) ^a | 1 (1.0) ^a |
| Most frequent TEAEs ^b | | | | | | |
| SARS-CoV-2 (COVID-19) infection | 3.6 (1.8, 6.5) | 4.5 (0.9, 13.2) | 7.9 (5.8, 10.5) | 17.0 (7.8, 32.3) | 9.2 (5.6, 14.3) | 6.6 (4.4, 9.6) |
| Nasopharyngitis | 15.8 (11.4, 21.1) | 13.1 (5.6, 25.8) | 10.3 (7.8, 13.4) | 6.5 (3.0, 21.3) | 6.5 (3.6, 10.4) | 5.7 (3.6, 8.4) |
| Upper respiratory tract infection | 6.1 (3.6, 9.6) | 1.5 (0.0, 8.4) | 5.1 (3.4, 7.2) | 7.1 (1.9, 18.2) | 2.7 (1.0, 5.9) | 3.4 (1.9, 5.7) |
| Urinary tract infection | 5.6 (3.3, 9.0) | 1.5 (0.0, 8.5) | 4.3 (2.8, 6.4) | 3.6 (0.4, 12.9) | 7.9 (4.6, 12.7) | 5.0 (3.1, 7.6) |
| Oral candidiasis | 5.3 (3.0, 8.6) | 0 | 4.4 (2.8, 6.4) | 1.8 (0.0, 9.7) | 2.7 (1.0, 5.9) | 2.3 (1.1, 4.2) |
| Adjudicated MACCE | 0.3 (0.0, 1.3) ^b | 0 | 0.2 (0.0, 0.9) ^b | 1.7 (0.0, 9.6) | 0.9 (0.1, 3.2) ^b | 0.4 (0.1, 1.6) |
| Neutropenia | 2.3 (0.9, 4.7) ^c | 1.5 (0.0, 8.4) ^c | 1.3 (0.0, 2.6) ^c | 2.3 (0.7, 5.3) ^c | 2.3 (1.1, 4.2) ^c | 2.3 (1.1, 4.2) ^c |
| Serious infections | 0.7 (0.1, 2.4) ^d | 0 | 1.1 (0.5, 2.3) ^d | 0 | 2.3 (0.7, 5.3) ^d | 1.1 (0.4, 2.6) ^d |
| Opportunistic infections | 1.6 (0.5, 3.8) ^e | 0 | 1.1 (0.5, 2.3) ^e | 0 | 0.5 (0.0, 1.5) ^e | 0.5 (0.1, 1.6) ^e |
| Hypersensitivity | 8.1 (5.2, 12.1) | 3.1 (0.4, 11.1) | 6.1 (4.3, 8.5) | 14.9 (6.4, 29.4) | 2.7 (1.0, 5.9) | 2.5 (1.2, 4.5) |
| Dermatitis and eczema | 3.6 (1.8, 6.5) | 0 | 2.6 (1.5, 4.3) | 7.2 (2.0, 18.5) | 0.9 (0.1, 3.2) | 1.1 (0.4, 2.6) |
| Injection site reactions | 1.6 (0.5, 3.8) | 7.8 (2.5, 18.2) | 1.1 (0.5, 2.3) | 7.3 (2.0, 18.7) | 1.4 (0.3, 4.0) | 0.9 (0.3, 2.3) |
| Adjudicated suicidal ideation and behaviour | 0 | 0 | 0.3 (0.0, 1.2) | 0 | 0 | 0 |
| Hepatic adverse events including hepatic enzyme elevations | 11.7 (8.1, 16.3) | 27.5 (15.7, 44.7) | 8.3 (6.1, 11.0) | 7.2 (2.0, 18.4) | 6.9 (3.9, 11.4) | 5.4 (3.4, 8.1) |
| Malignancies excluding non-melanoma skin cancer | 0 | 0 | 0.3 (0.0, 1.2) ^f | 0 | 0.9 (0.1, 3.2) ^f | 0.7 (0.1, 2.0) ^f |
| Adjudicated IBD ^g | 0.3 (0.0, 1.8) | 0 | 0.2 (0.0, 0.9) ^g | 1.7 (0.0, 9.7) | 0 | 0 |

Safety set, in patients with psoriasis affecting $\geq 3\%$ BSA at baseline. No cases of active tuberculosis or uveitis were reported. [a] BKZ Total includes PBO/BKZ Week 16 switchers (for both BE OPTIMAL and BE COMPLETE), includes events after switch only. [b] BE OPTIMAL Weeks 0–104 ADA/BKZ includes events after Week 52 switch only. [c] EAIRs not available for full study period. [d] One death due to a motorcycle accident occurred before Week 52, reported as unrelated to the study treatment. [e] One death due to cardiac arrest occurred after Week 52, reported as unrelated to the study treatment. [f] Sudden death in 54-year old patient with a history of hypertension, aortic regurgitation and electrocardiogram changes of coronary artery disease occurred before Week 52, reported as unrelated to the study treatment; no further information available as no autopsy was performed. [g] Five most common TEAEs in any BKZ-treated group at the Week 104 data cut. [h] 1 thrombotic cerebral infarction; [i] 1 acute myocardial infarction; [j] 1 cerebral hemorrhage; 1 sudden death; [k] 6 neutropenia; 1 decreased neutrophil count; [l] 1 neutropenia; [m] 7 neutropenia; 1 decreased neutrophil count; [n] 4 neutropenia; 1 decreased neutrophil count; [o] 6 neutropenia; 5 decreased neutrophil count; [p] 1 pneumonia; 1 upper respiratory tract infection; [q] 3 pneumonia; 1 arthritis bacterial; 1 extracardiac abscess; 1 post-procedural infection; 1 staphylococcal bacteraemia; 1 upper respiratory tract infection; 1 UTI; [r] 1 buritis infective; 1 post-operative wound infection; 1 bronchitis; 1 pneumonia; 1 pyelonephritis acute; [s] 5 of which 3 were *Candida* infections; [t] 4 *Candida* infections; [u] 1 *Candida* infection; [v] 2 *Candida* infections; [w] 1 breast cancer; [x] 1 endometrial cancer; 1 gastric cancer; [y] 1 endometrial cancer; 2 gastric cancers; [z] Cases deemed definite or probable IBD by the investigator.

ACR20/50/70: $\geq 20/50/70\%$ improvement from baseline in American College of Rheumatology response criteria; ADA: adalimumab; bDMARD: biologic disease-modifying antirheumatic drug; BKZ: bimekizumab; BSA: body surface area; CFB: change from baseline; DAPSA: Disease Activity Index for Psoriatic Arthritis; EAIR: exposure-adjusted incidence rate; FACIT-Fatigue: Functional Assessment of Chronic Illness Therapy-Fatigue; HAO-DI: Health Assessment Questionnaire-Disability Index; IBD: inflammatory bowel disease; IL: interleukin; LDA: low disease activity; LEI: Leeds Dactylitis Index; LEI: Leeds Enthesitis Index; MACCE: major adverse cardiovascular event; MDA: minimal disease activity; MI: multiple imputation; mNAPSI: modified Nail Psoriasis Severity Index; NRI: non-responder imputation; OC: observed case; OLE: open-label extension; PASI75/90/100: $\geq 75/90/100\%$ improvement from baseline in Psoriasis Area and Severity Index; PBO: placebo; PsA: psoriatic arthritis; PsAID-12: Psoriatic Arthritis Impact of Disease-12; PY: patient-years; Q2W: every 2 weeks; Q4W: every 4 weeks; REM: remission; SD: standard deviation; SE: standard error; SF-36 PCS: Short-Form-36 Health Survey Physical Component Summary; SJC: swollen joint count; TEAE: treatment-emergent adverse event; TJC: tender joint count; TNFi-IR: prior inadequate response or intolerance to tumour necrosis factor inhibitors; UTI: urinary tract infection; VAS: visual analogue scale; VLDA: very low disease activity; WCI: worst category imputation.

¹Institute and Comprehensive Center for Inflammation Medicine, University of Lübeck, Lübeck, Germany; ²Department of Dermatology, The Icahn School of Medicine at Mount Sinai, New York, New York, USA; ³Department of Dermatology, The Jikei University School of Medicine, Tokyo, Japan; ⁴College of Medical Veterinary and Life Sciences, University of Glasgow, Glasgow, UK; ⁵Dermatology Centre, Northern Care Alliance NHS Foundation Trust, Manchester, UK; ⁶NIHR Manchester Biomedical Research Centre, Manchester University NHS Foundation Trust, Manchester Academic Health Science Centre, Manchester, UK; ⁷Division of Dermatology and Venerology, Department of Medicine, Geneva University Hospitals, and Department of Pathology and Immunology, Faculty of Medicine, University of Geneva, Geneva, Switzerland; ⁸UCB Pharma, Slough, UK; ⁹UCB Pharma, Morrisville, North Carolina, USA; ¹⁰Department of Dermatology and Department of Medicine, Division of Rheumatology, UT Southwestern Medical Center, Dallas, Texas, USA.

References: Ritchlin CT et al. Ann Rheum Dis 2023;82:1404–14; Coates LC et al. RMD Open 2024;10:e03955; Ritchlin CT et al. N Engl J Med 2017;376:957–70. **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: DT, ABG, AA, ML, IBM, RBW, W-HB, BI, RB, JC, JFM. Drafting of the publication, or reviewing it critically for important intellectual content: DT, ABG, AA, ML, IBM, RBW, W-HB, BI, RB, JC, JFM. **Disclosures:** DT: Investigator and/or consultant/advisor for AbbVie, Almiral, Amgen, BMS, Boehringer Ingelheim, Celtrion, Eli Lilly and Company, Galderma, Janssen-Cilag, Kyowa Kirin, LEO Pharma, L'Oréal, New Bridge, Novartis, Pfizer, Regeneron, Samsung, Sanofi, Target-RWE, UCB Pharma, and Vichy; received grants from AbbVie, LEO Pharma, and Novartis. ABG: Honoraria as an advisory board member and consultant for Amgen, AnaptysBio, Avotres Therapeutics, BMS, Boehringer Ingelheim, DICE Therapeutics, Eli Lilly and Company, Highlights Therapeutics, Janssen, Novartis, Sanofi, Teva, UCB Pharma, and Xbiotech (stock options for RA); research/educational grants from BMS, Highlights Therapeutics, Janssen, and UCB Pharma (all paid to Mount Sinai School of Medicine). AA: Honoraria and/or research grants from AbbVie, Amgen, BMS, Boehringer Ingelheim, Eisai, Eli Lilly and Company, Janssen, Kyowa Kirin, LEO Pharma, Maruho, Mitsubishi Tanabe Pharma, Novartis, Pfizer, Sun Pharma, Taiho Pharma, Tori Pharmaceutical Co., and UCB Pharma. ML: Employee of Mount Sinai; research funds from AbbVie, Amgen, Arcutis, Avotres Therapeutics, Boehringer Ingelheim, Cara Therapeutics, Dermavant Sciences, Eli Lilly and Company, Incyte, Inozyme, Janssen Research & Development, LLC, Novartis, Ortho Dermatologics, Regeneron, and UCB Pharma; consultant for Almiral, Atrius Inc., AnaptysBio, Arcutis Inc., Arena Pharmaceuticals, AstraZeneca, Avotres, BioMx, BMS, Boehringer Ingelheim, Brickell Biotech, Castle Biosciences, Celltrion, CorEvitas, Dermavant Sciences, Epi, Evomune Inc., Facilitation of International Dermatology Education, Forte Biosciences, Foundation for Research and Education in Dermatology, Galderma, Genentech, Incyte, LEO Pharma, Meiji Seika Pharma, Mintera, National Society of Cutaneous Medicine, New York College of Podiatric Medicine, Pfizer, Sanofi-Regeneron, Seaneer, Strata, Takeda, Trevi, and Verrica. IBM: Consulting fees and honoraria from AbbVie, AstraZeneca, BMS, Boehringer Ingelheim, Cabaletta, Causeway Therapeutics, Celgene, EVELO, Janssen, Eli Lilly and Company, Moonlake, Novartis, and UCB Pharma; research support from BMS, Boehringer Ingelheim, Celgene, Janssen, Novartis, and UCB Pharma. RBW: Consulting fees from AbbVie, Almiral, Amgen, Arena, Astellas, Avillion, Biogen, BMS, Boehringer Ingelheim, Celgene, Eli Lilly and Company, GSK, Janssen, LEO Pharma, Novartis, Pfizer, Sanofi, and UCB Pharma; research grants to his institution from AbbVie, Almiral, Janssen, LEO Pharma, Novartis and UCB Pharma; honoraria from Astellas, DICE Therapeutics, GSK, and Union Therapeutics. W-HB: Received honoraria as a speaker and/or advisor from AbbVie, Almiral, BMS, Eli Lilly and Company, Janssen, LEO Pharma, Novartis, and UCB Pharma. RB: Employee of UCB Pharma; shareholder of AbbVie, GSK, and UCB Pharma. RB, JC: Employees and shareholders of UCB Pharma. JFM: Consultant and/or investigator for AbbVie, Amgen, AstraZeneca, Biogen, BMS, Boehringer Ingelheim, Dermavant, Eli Lilly and Company, Incyte, Janssen, LEO Pharma, Moonlake Immunotherapeutics, Novartis, Pfizer, Sanofi-Regeneron, Sun Pharma, and UCB Pharma. **Acknowledgements:** We would like to thank the patients and their caregivers in addition to all the investigators and their teams who contributed to this study. The authors acknowledge Heather Edens, PhD, UCB Pharma, Smyrna, Georgia, USA for publication coordination, Orla Woodford, PhD, Costello Medical, London, UK for medical writing and editorial assistance, and the Costello Medical Creative team for design support. These studies were funded by UCB Pharma. All costs associated with development of this presentation were funded by UCB Pharma.



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