Clinical Efficacy of Bimekizumab Treatment up to 2 Years Across Patients with Psoriatic Arthritis and Varying Baseline Joint Involvement: Results from a Post Hoc Analysis of Two Pooled Phase 3 Studies

1458

Philip J. Mease,[1] Laure Gossec,[2] Hideto Kameda,[3] David Nicholls,[4] Proton Rahman,[5] Barbara Ink,[6] Rajan Bajracharya,[6] Patrick Healy,[7] Laura C. Coates[8]

[1]Department of Rheumatology, Providence-Swedish Medical Center and University of Washington, Seattle, WA, USA; [2]Sorbonne Universite and Pitie-Salpetriere Hospital, Paris, France; [3]Department of Internal Medicine, Toho University, Tokyo, Japan; [4]Clinical Trials Unit, University of the Sunshine Coast, Queensland, Australia; [5]Division of Rheumatology, Craig L. Dobbin Genetics Research Centre, Discipline of Medicine, Memorial University of Newfoundland, St John's, NL, Canada; [6]UCB, Slough, UK; [7]UCB, Morrisville, NC, USA; [8]Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Diseases, University of Oxford and Oxford Biomedical Research Centre, Oxford University Hospitals NHS Trust, Oxford, UK.

Objective

To assess the efficacy of bimekizumab (BKZ) treatment up to 2 years in patients with psoriatic arthritis (PsA), stratified by baseline levels of swollen joint count (SJC), using data pooled from two phase 3 studies.

Introduction

- Patients in PsA clinical trials often present with greater joint involvement than in clinical settings due to trial inclusion criteria;[1] thus, treatment efficacy in patients with varying joint involvement should be assessed to ensure broader clinical applicability.
- BKZ, a monoclonal antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated long-term 2-year efficacy across disease domains in patients with PsA.[2]

Methods

- In this post hoc analysis, subcutaneous BKZ 160 mg every 4 weeks was assessed to 2 years in patients with PsA, stratified by levels of baseline joint involvement, as assessed by SJC.
- Patients were pooled from BE OPTIMAL (biologic disease-modifying anti-rheumatic drug [bDMARD]-naïve; NCT03895203) and BE COMPLETE (tumor necrosis factor inhibitor inadequate response/intolerance [TNFi-IR]; NCT03896581) phase 3 trials, which were double-blind and placebo (PBO)-controlled to Week 16.
  - In both trials, the primary outcome was ACR50 at Week 16.
  - Patients completing Week 52 of BE OPTIMAL or Week 16 of BE COMPLETE could enter BE VITAL (open-label extension; NCT04009499), where all patients received BKZ.
- In this analysis, patients were grouped by baseline SJC, with thresholds based on quartile boundaries at baseline (SJC ≤5; 6–≤7; 8–≤12; >12).
  - To meet study inclusion criteria, patients were required to have ≥3 tender and swollen joints.
  - Due to low patient numbers with 3 or 4 SJC at baseline, patients with 5 SJC were included in the first quartile; patient numbers in each quartile therefore vary.
  - As the trials included populations of different sizes (BE OPTIMAL: 431 BKZ, 281 PBO; BE COMPLETE: 267 BKZ, 133 PBO), proportions of bDMARD-naïve and TNFi-IR patients are not equal per quartile.
- We report efficacy across PsA domains, including joints, skin, composite, and patient-reported outcomes.
  - Data are reported by randomization group (PBO/BKZ or BKZ) as observed case (OC) or using non-responder imputation (NRI).

Results

- Demographics and disease characteristics by baseline SJC quartile subgroups are presented in the Table.
  - Baseline demographics were similar across all SJC quartile subgroups. Baseline disease characteristics suggested greater disease severity in patients with increased joint involvement.
- At Week 16, patients treated with BKZ demonstrated numerically greater improvements in joint, skin, composite, and patient-reported outcomes compared with PBO across patients with varying baseline joint involvement.
- For all domains assessed, improvements were sustained to 2 years in BKZ-randomized patients with varying baseline joint involvement (Figures 1–2).
- For patients who switched from PBO to BKZ at Week 16, improvements in efficacy were reported to Year 1 and sustained to Year 2, reaching similar responses to the BKZ-randomized group across patients with varying baseline joint involvement.

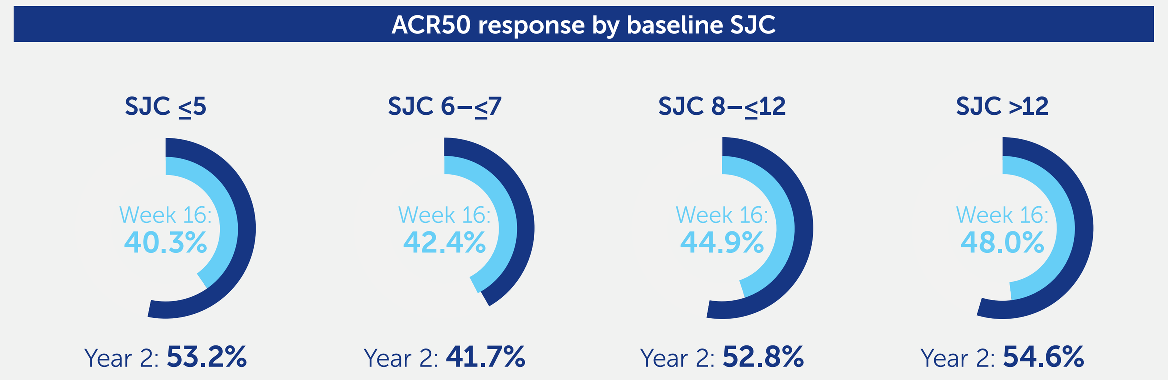
Conclusions

In patients with PsA, stratified by varying levels of baseline joint involvement, bimekizumab treatment resulted in greater improvements compared with placebo across disease domains at Week 16, with improvements sustained to 2 years.

Robust efficacy was observed across all groups of patients with varying baseline joint involvement, potentially reflecting the broader range of patients seen in clinical practice.

Summary

Post hoc analysis of **bimekizumab treatment** up to two years, across disease domains, in patients with **active PsA stratified by varying levels of baseline joint involvement**, using pooled data from two phase 3 trials



At Week 16, bimekizumab demonstrated **numerically greater responses versus placebo** in all assessed domains, **across patients with varying baseline swollen joint count**.

Improvements across domains were **sustained from Week 16 to 2 years**[a] with bimekizumab treatment, **across patients with varying baseline joint involvement**.[b]

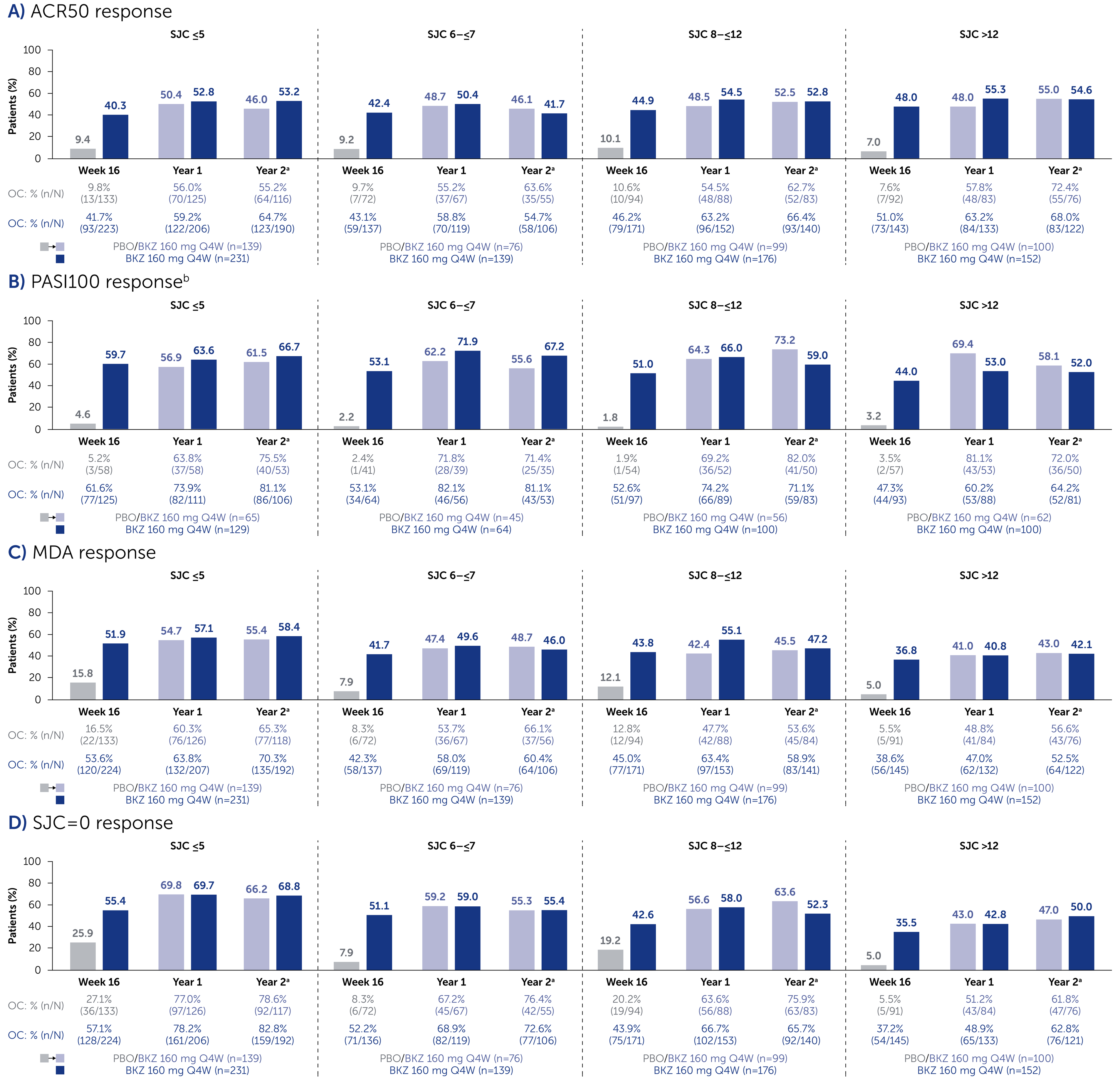
Data reported using NRI. [a] Year 2 refers to Week 104 (BE OPTIMAL) or Week 100 (BE COMPLETE); [b] Data reported for BKZ-randomized patients.

Table Baseline demographics and disease characteristics, reported by baseline joint involvement

	Joint Involvement							
	SJC ≤5		SJC 6–≤7		SJC 8–≤12		SJC >12	
Mean (SD) unless otherwise specified	PBO n=139	BKZ 160mg Q4W n=231	PBO n=76	BKZ 160mg Q4W n=139	PBO n=99	BKZ 160mg Q4W n=176	PBO n=100	BKZ 160mg Q4W n=152
Age, years	49.6 (13.2)	47.9 (13.2)	49.8 (12.0)	49.6 (12.8)	49.3 (11.1)	49.5 (11.6)	49.4 (12.0)	50.2 (12.1)
Sex, male, n (%)	65 (46.8)	112 (48.5)	34 (44.7)	54 (38.8)	43 (43.4)	82 (46.6)	45 (45.0)	83 (54.6)
BMI, kg/m²	28.0 (5.1)	28.4 (6.0)	31.0 (6.7)	29.7 (7.6)	30.1 (6.3)	30.6 (7.1)	29.5 (5.4)	30.0 (6.1)
Time since PsA diagnosis, years	6.0 (6.5)	6.4 (7.1)[a]	7.8 (7.8)	7.8 (9.1)[b]	7.2 (8.2)[c]	7.5 (9.4)[d]	6.7 (6.9)[c]	8.3 (9.0)[e]
cDMARD use at baseline, n (%)	73 (52.5)	147 (63.6)	46 (60.5)	85 (61.2)	61 (61.6)	111 (63.1)	77 (77.0)	97 (63.8)
bDMARD-naïve, n (%)	98 (70.5)	146 (63.2)	51 (67.1)	88 (63.3)	69 (69.7)	102 (58.0)	63 (63.0)	95 (62.5)
TNFi-IR, n (%)	41 (29.5)	85 (36.9)	25 (32.9)	51 (36.7)	30 (30.3)	74 (42.0)	37 (37.0)	57 (37.5)
BSA affected by psoriasis ≥3%, n (%)	65 (46.8)	129 (55.8)	45 (59.2)	64 (46.0)	56 (56.6)	100 (56.8)	62 (62.0)	100 (65.8)
PASI score[f]	8.6 (6.2)	9.2 (8.0)	8.2 (4.8)	8.1 (7.7)	7.2 (5.1)	9.0 (8.0)	8.4 (7.3)	9.5 (8.1)
SJC (of 66 joints)	4.2 (0.8)	4.1 (0.8)	6.4 (0.6)	6.4 (0.5)	9.6 (1.4)	9.6 (1.4)	20.2 (8.6)	19.2 (7.7)
TJC (of 68 joints)	10.7 (7.9)	10.8 (8.4)	13.5 (9.9)	15.3 (10.3)	18.1 (9.9)	18.0 (9.6)	30.6 (14.5)	28.8 (14.3)
Enthesitis (LEI >0), n (%)	37 (26.6)	64 (27.7)[g]	16 (21.1)	50 (36.0)[b]	20 (20.2)	71 (40.3)	33 (33.0)[h]	64 (42.1)[i]
LEI score[j]	2.5 (1.4)	2.4 (1.5)	3.3 (1.8)	2.3 (1.4)	2.9 (1.5)	2.5 (1.5)	3.2 (1.5)	3.0 (1.4)
Pain VAS[k]	52.3 (26.5)	49.3 (25.6)[l]	55.0 (21.6)	53.3 (24.6)	61.4 (22.4)	57.5 (22.9)	66.4 (19.7)	64.3 (20.7)
HAQ-DI	0.80 (0.60)	0.75 (0.58)[l]	0.81 (0.58)	0.87 (0.58)	1.03 (0.57)	0.92 (0.59)	1.14 (0.72)	1.03 (0.59)
PtGA	55.8 (24.8)	51.9 (24.4)[l]	56.1 (21.1)	53.9 (24.7)	62.0 (23.6)	59.1 (20.9)	66.9 (19.7)	64.0 (20.5)

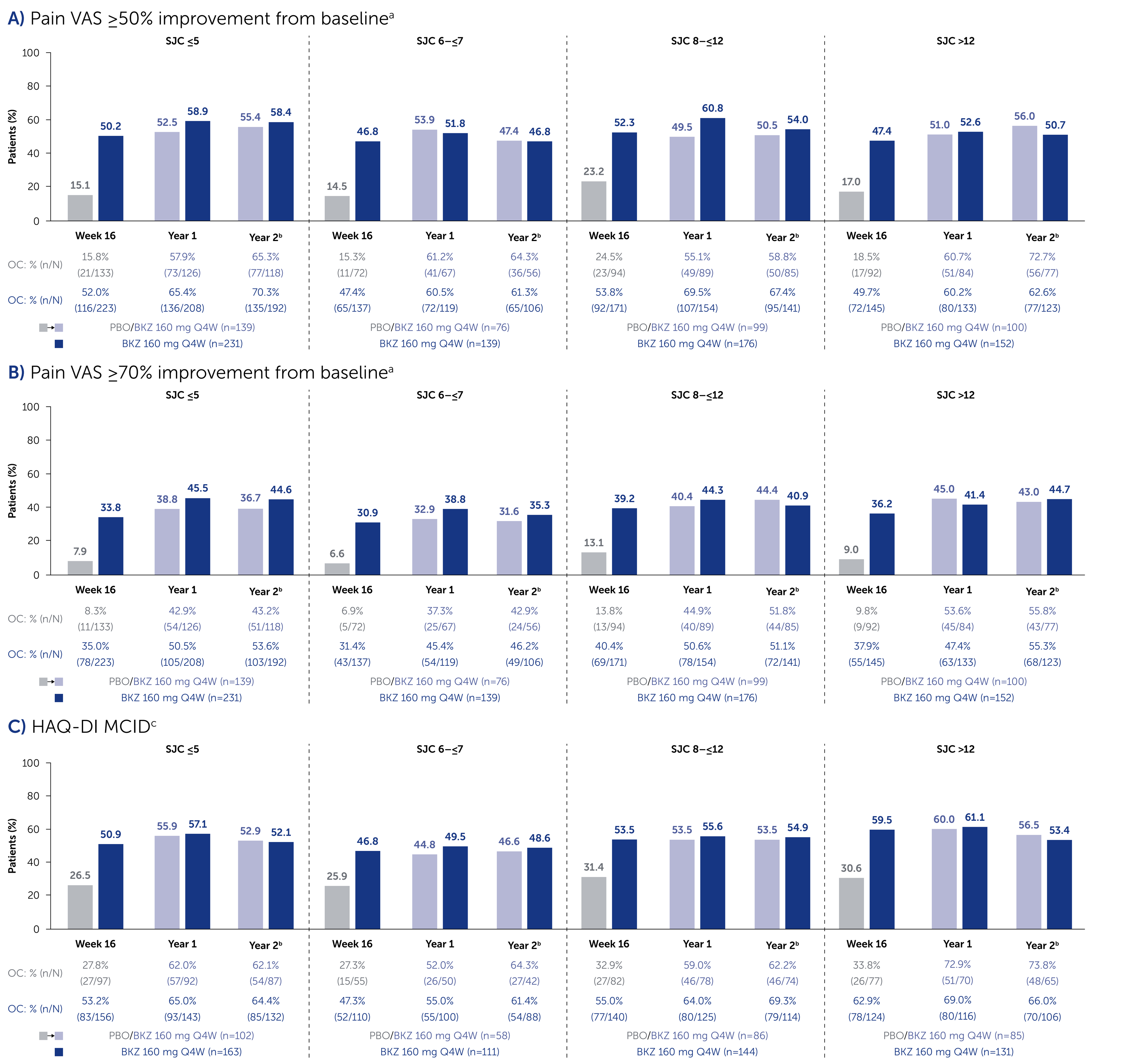
Randomized set. Due to the small number of patients with 3 SJC (n=85) and 4 SJC (n=143) at baseline, patients with 5 SJC (n=142) were included in the first quartile. [a] n=226; [b] n=138; [c] n=98; [d] n=174; [e] n=151; [f] In patients with BSA affected by psoriasis ≥3% at baseline; [g] n=228; [h] n=99; [i] n=150; [j] In patients with enthesitis at baseline (LEI >0); [k] Pain VAS assessed using Patient's Assessment of Arthritis Pain VAS (0 [no pain] to 100 [most severe pain]); [l] n=230.

Figure 1 Clinical efficacy outcomes at Week 16, Year 1, and Year 2, reported by baseline joint involvement (NRI, OC)



Randomized set. Data reported for all patients randomized to PBO or BKZ, stratified by joint involvement (based on SJC score quartiles). Missing data were imputed with NRI (bars) or reported using OC (text). [a] Data pooled at Year 2 from Week 104 in BE OPTIMAL and Week 100 in BE COMPLETE; [b] Assessed in patients with baseline psoriasis affecting ≥3% body surface area at baseline.

Figure 2 Patient-reported pain and physical function at Week 16, Year 1, and Year 2, reported by baseline joint involvement (NRI, OC)



Randomized set. Data reported for all patients randomized to PBO or BKZ stratified by joint involvement (based on SJC score quartiles). Missing data were imputed with NRI (bars) or reported using OC (text). [a] Pain VAS assessed using Patient's Assessment of Arthritis Pain VAS (0 [no pain] to 100 [most severe pain]); [b] Data pooled at Year 2 from Week 104 in BE OPTIMAL and Week 100 in BE COMPLETE; [c] HAQ-DI decrease ≥0.35 from baseline, assessed in patients with HAQ-DI ≥0.35 at baseline.

ACR50: ≥50% improvement from baseline in American College of Rheumatology response criteria; **bDMARD:** biologic disease-modifying antirheumatic drug; **BKZ:** bimekizumab; **BMI:** body mass index; **BSA:** body surface area; **cDMARD:** conventional disease-modifying antirheumatic drug; **HAQ-DI:** Health Assessment Questionnaire – Disability Index; **HRQoL:** health-related quality of life; **IL:** interleukin; **LEI:** Leeds Enthesitis Index; **MCID:** minimal clinically important difference; **MDA:** minimal disease activity; **NRI:** non-responder imputation; **OC:** observed case; **Pain50/70:** ≥50/70% improvement from baseline in Pain VAS; **PASI:** Psoriasis Area and Severity Index; **PASI100:** 100% improvement from baseline in PASI; **PBO:** placebo; **PsA:** psoriatic arthritis; **PtGA:** Patient's Global Assessment; **Q4W:** every 4 weeks; **SD:** standard deviation; **SJC:** swollen joint count; **TJC:** tender joint count; **TNFi-IR:** tumor necrosis factor inhibitor intolerance/inadequate response; **VAS:** visual analog scale.

References: [1]Alle G. J Rheum 2025;52:138–44; [2]Mease PJ. Rheumatol Ther 2024;11:1363–82.
Author Contributions: Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **PJM**, **LG**, **HK**, **DN**, **PR**, **BI**, **RB**, **PH**, **LCC**; Drafting of the publication, or reviewing it critically for important intellectual content: **PJM**, **LG**, **HK**, **DN**, **PR**, **BI**, **RB**, **PH**, **LCC**; Final approval of the publication: **PJM**, **LG**, **HK**, **DN**, **PR**, **BI**, **RB**, **PH**, **LCC**. **Author Disclosures: PJM:** Research grants from AbbVie, Acelyrin, Amgen, Bristol Myers Squibb, Eli Lilly and Company, Johnson & Johnson Innovative Medicine, Novartis, Pfizer Inc., Sana, and UCB; consulting fees from AbbVie, Acelyrin, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly and Company, Inmagene, Johnson & Johnson Innovative Medicine, MoonLake Immunotherapeutics, Novartis, Pfizer Inc., Spyre, Takeda, and UCB; speakers bureau fees from AbbVie, Amgen, Eli Lilly and Company, Johnson & Johnson Innovative Medicine, Novartis, Pfizer, and UCB. **LG:** Received grants or contracts from AbbVie, Biogen, Lilly, Novartis, and UCB; consulting fees from AbbVie, Amgen, BMS, Celltrion, Janssen, Lilly, Moonlake MSD, Novartis, Pfizer, Stada, and UCB. **HK:** Grants from Asahi-Kasei, Pfizer, and Taisho; advisory/consultant for AbbVie, Amgen, Asahi-Kasei, Eli Lilly, Janssen, Novartis, Pfizer, Sanofi, Taisho, and UCB; speaking fees from AbbVie, Amgen, Asahi-Kasei, BMS, Eisai, Eli Lilly, Janssen, Mitsubishi-Tanabe, and UCB. **DN:** Research support from AbbVie, BMS, Eli Lilly, Gilead, Incannex, Janssen, Novartis, Pfizer, Servatus, Sun Pharma, and UCB; advisory board or speaker fees from AbbVie, AstraZeneca, Janssen, Novartis, Pfizer, Sandoz, and UCB. **PR:** Consulting fees from Abbott, AbbVie, Amgen, Bristol Myers Squibb, Celgene, Eli Lilly and Company, Janssen, Novartis, Pfizer, and UCB; received research grants from Janssen, and Novartis. **BI:** Employee of UCB; shareholder of AbbVie, GSK, and UCB. **RB, PH:** Employees and shareholders of UCB. **LCC:** Grants/research support from AbbVie, Amgen, Celgene, Eli Lilly, Gilead, Janssen, Novartis, Pfizer, and UCB; consultant for AbbVie, Amgen, BMS, Boehringer Ingelheim, Celgene, Domain, Eli Lilly, Galapagos, Gilead, Janssen, MoonLake Immunotherapeutics, Novartis, Pfizer, and UCB; speaking fees from AbbVie, Amgen, Biogen, Celgene, Eli Lilly, Galapagos, Gilead, GSK, Janssen, medac, Novartis, Pfizer, and UCB. **Acknowledgments:** 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, Smyrna, Georgia, USA, for publication coordination, and study conceptualization and coordination, Lyes Derouiche, PhD, UCB, Brussels, Belgium, for publication coordination, Sona Popat, BA, Costello Medical, London, UK and Rebecca Light, BSc, Costello Medical, Manchester, UK for medical writing and editorial assistance, and the Costello Medical Creative team for design support. These studies were funded by UCB. All costs associated with development of this poster were funded by UCB.

To receive a copy of this poster, scan the QR code.
Link expiration:
January 27

Presented at ACR Convergence 2025 | October 24–29 | Chicago, IL