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Control of Inflammation and Sustained Improvements in Patient-Reported Outcomes in Patients With Psoriatic Arthritis: Bimekizumab Two-Year Results From Two Phase 3 Studies

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Objective. To assess two-year impact of bimekizumab on patient-reported outcomes (PROs), and their association with objective measures of inflammation, in patients with psoriatic arthritis (PsA) who were biologic disease-modifying antirheumatic drug (bDMARD)-naïve or had tumor necrosis factor inhibitor inadequate response or intolerance (TNFi-IR).

Methods. BE OPTIMAL (NCT03895203; bDMARD-naïve) and BE COMPLETE (NCT03896581; TNFi-IR) were phase 3 studies that assessed subcutaneous bimekizumab 160 mg every four weeks. Both were double-blind, placebo-controlled to week16, then placebo patients switched to bimekizumab. BE OPTIMAL week52 or BE COMPLETE week16 completers could enter BE VITAL (NCT04009499; open-label extension), where all patients received bimekizumab. PROs, disease impact (Psoriatic Arthritis Impact of Disease-12 questionnaire [PsAID-12]), and their association with inflammation (assessed using swollen joint count [SJC]) are reported to year 2.

Results. Among 712 bDMARD-naïve and 400 TNFi-IR patients, bimekizumab resulted in long-term sustained improvements in PROs for pain, fatigue, and function (Health Assessment Questionnaire–Disability Index) to year 2. Mean change from baseline (year 2) in bimekizumab-randomized patients for pain and disease impact (PsAID-12) was –33.9 to –29.2 and –2.5 to –2.2, respectively. An achievement of SJC = 0 was associated with the greatest reduction in pain. Decreased SJC was associated with improved pain, fatigue, function, and reduced disease impact to year 2. By year 2, 44.4% to 54.3% of patients originally randomized to placebo or bimekizumab reported no or low disease impact (PsAID-12 ≤1.95), and 88.0% to 92.0% of bimekizumab-randomized patients achieved a patient acceptable symptom state for disease impact (PsAID-12 ≤4), associated with concurrent improvement in SJC and skin involvement.

Conclusion. Bimekizumab treatment resulted in sustained clinically meaningful improvements in PROs and reduced disease impact to two years in bDMARD-naïve and TNFi-IR patients with PsA. Stringent inflammation control was associated with symptom relief and reduced disease impact.

INTRODUCTION

Psoriatic arthritis (PsA) is a chronic, inflammatory condition characterized by joint pain, swelling, and stiffness, and can manifest across the multiple clinical domains of the disease in individual patients, including peripheral and axial arthritis, enthesitis, dactylitis, skin, and nail involvement.[1–4] These manifestations have a profound and long-term impact on patients' health-related

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quality of life (HRQoL), affecting their physical, social, and emotional well-being.[4–7]

Symptoms caused by persistent, active inflammation, including joint pain, swelling, and damage, as well as skin problems and fatigue lead to decreased mobility and function which limit patients' ability to perform daily tasks.[5,8,9] PsA symptoms also contribute to a significant emotional burden for patients, including mood and behavioral changes, poor body image, and sleep disorders.[7] Social well-being is similarly affected, manifesting in reduced engagement in daily activities.[5,7]

There are a wide range of conventional synthetic and biologic disease-modifying antirheumatic drugs (cs/bDMARDs) now available to clinicians in the management of PsA.[10] Despite this, up to half of patients with PsA receiving bDMARD treatment switch therapy by one year,[11,12] highlighting the need for treatments with meaningful and sustainable effects. Some patients, even those who reach high treatment targets, experience residual symptoms including persistent joint pain and swelling, fatigue, and functional disability.[13] Evaluation of the effectiveness of new treatments should, therefore, include assessments based on the personal experiences of individuals living with PsA and the durability of symptom relief. Satisfactory long-term treatment solutions should improve symptoms across PsA domains, particularly those that have been identified as important to patients.[14–16] Bimekizumab is a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A.[17,18] In patients with active PsA, bimekizumab has demonstrated clinically meaningful improvements in efficacy outcomes to 16 weeks, which have been sustained up to two years in the phase 3 BE OPTIMAL (bDMARD-naïve patients) and BE COMPLETE (patients with inadequate response or intolerance to tumor necrosis factor inhibitors [TNFi-IR]) studies.[19–21] Clinically meaningful improvements in patient-reported symptoms, disease impact, and HRQoL have also been reported as early as four weeks, after a single dose of bimekizumab, and up to one year of treatment.[22–24]

Pain, skin problems, and fatigue are among the most important symptoms to patients receiving treatment for PsA.[14,15] Previous research has shown that symptoms including pain and fatigue in patients with PsA may be driven, at least in part, by inflammatory pathways.[25–27] Swollen joint count (SJC) is an accessible measure, readily performed in the clinic, and is considered a reliable and objective, physician-derived measure of joint inflammation.[28–30] Consequently, it is of interest to explore the association between clinically assessed inflammatory features, such as swollen joints, and patient-reported symptoms.

The objective of this study was to assess the two-year efficacy of bimekizumab on patient-reported symptoms and disease impact, and the association of these findings with inflammation as assessed by SJC, in patients with active PsA who were bDMARD-naïve or TNFi-IR. These data complement and expand on previous published efficacy and safety findings for bimekizumab[19,23,24] by reporting results for patient-reported outcomes (PROs) from baseline up to two years, including states of disease impact, for the first time. The association between SJC and improvements in PROs in these studies, and the association between SJC, skin involvement, and disease impact are also reported here for the first time.

PATIENTS AND METHODS

Study designs and patients. The efficacy and safety of bimekizumab was assessed in patients with active PsA in two phase 3, multicenter trials, BE OPTIMAL (bDMARD-naïve patients; NCT03895203) and BE COMPLETE (TNFi-IR patients; NCT03896581). Complete methodologies were reported to week 16 or week 24 in the primary publications of BE OPTIMAL and BE COMPLETE, and further details can be found in subsequent publications of one-year and two-year results.[19–21,31,32] Study design diagrams can be found in Supplementary Figure S1.

In brief, both studies were double-blind and placebo-controlled to 16 weeks. Patients in BE OPTIMAL were randomized 3:2:1 to receive either subcutaneous bimekizumab 160 mg every four weeks (Q4W), placebo or the reference (subcutaneous adalimumab 40 mg every two weeks). At week 16 in BE OPTIMAL, patients receiving placebo switched to receive bimekizumab 160 mg Q4W. Patients in BE COMPLETE were randomized 2:1 to subcutaneous bimekizumab 160 mg Q4W or placebo. Patients who completed week 52 of BE OPTIMAL or week 16 of BE COMPLETE were eligible to enter an open-label extension (OLE), BE VITAL (NCT04009499), in which all patients received bimekizumab 160 mg Q4W. Data are reported in-text for patients randomized to placebo or bimekizumab at baseline, up to two years. Data for reference arm (adalimumab)–randomized patients, who switched to bimekizumab at week 52, are reported in the

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Data from this article may be requested by qualified researchers six months after product approval in the United States and/or Europe, or global development is discontinued, and 18 months after trial completion. Investigators may request access to anonymized individual patient data and redacted study documents, which may include raw datasets, analysis-ready datasets, the study protocol, a blank case report form, an annotated case report form, the statistical analysis plan, dataset specifications, and the 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.

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Supplementary Material. Reference arm patients had received bimekizumab for only one year at the time of reporting and were, therefore, not included in this assessment of the two-year impact of bimekizumab treatment.

PROs. Change from baseline (CfB) and responder rates in PRO measures are reported to week 104 (year 2) in BE OPTIMAL, with reference to year 1 data at week 52, and to week 100 or week 88 (year 2) in BE COMPLETE, with reference to year 1 data at week 52 or week 40 (as some outcomes were not collected at week 52 or week 100 in BE COMPLETE). Hereafter, we refer to week 52/40 as year 1 and week 104/100/88 as year 2 across both trials; for further details of collection time points, the schedule of assessments is shown in Supplementary Table S1. Reported data include assessments of pain, fatigue, physical function, and disease impact; all PRO measures were captured electronically.

Pain was assessed using the Patient's Assessment of Arthritis Pain visual analog scale (VAS), in which study participants are asked to rate their level of pain resulting from arthritis from 0 representing "no pain" to 100 representing "most severe pain."[33] Pain VAS ≥50% improvement from baseline (Pain50) represents a substantial improvement in patient-reported pain.[34] Fatigue was assessed using the 13-item Functional Assessment of Chronic Illness Therapy (FACIT)-Fatigue subscale score. The FACIT-Fatigue score ranges from 0 to 52, with higher scores representing lower levels of fatigue.[35] FACIT-Fatigue minimal clinically important difference (MCID) was defined as an increase of ≥4 points in patients with a score of ≤48 at baseline.[35] Physical functioning was assessed using the Health Assessment Questionnaire–Disability Index (HAQ-DI). The HAQ-DI is a 20-item questionnaire measuring the level of difficulty patients experience with activities across eight categories; the score ranges from 0 to 3, with lower scores indicating better physical function.[36] HAQ-DI MCID was defined as a decrease of ≥0.35 points from baseline in patients with HAQ-DI ≥0.35 at baseline.[37] Physical function was also measured using the physical component summary (PCS) score derived from the Short Form 36-item Health Survey (SF-36, version 2, standard recall). CfB is reported here for the SF-36 PCS, mental component summary (MCS), and individual domain scores. Norm-based T-scores, standardized by setting the general US population (2009) to a mean score of 50 and an standard deviation (SD) of 10, are presented here. SF-36 scores range from 0 to 100, where higher scores indicate better HRQoL.[38]

Disease impact on HRQoL was assessed using the disease-specific, self-administered Psoriatic Arthritis Impact of Disease-12 (PsAID-12) which is formed of 12 physical, social, and psychological domains that cover a broad spectrum of clinical factors impacting HRQoL. Each item domain is assessed on a 0 to 10 numeric rating scale where higher scores indicate a worse impact of PsA.[39] To assess impact as a disease state, PsAID-12 levels were analyzed according to categories of patient-reported symptom or disease impact severity thresholds. Categories of no, low, moderate, or high symptom or disease impact are reported here, which were defined by PsAID-12 total scores of ≤1.15, >1.15 to ≤1.95, >1.95 to ≤3.60, or >3.60, respectively.[39] The proportion of patients achieving PsAID-12 patient acceptable symptom state (PASS), defined as a PsAID-12 total score ≤4 is also reported.[40]

Psoriasis was assessed using Psoriasis Area and Severity Index (PASI) scores to week 104 in BE OPTIMAL and week 100 in BE COMPLETE. The impact of skin involvement on patient's HRQoL was assessed using the PsAID-12 single-item domain of skin problems to week 104 in BE OPTIMAL and week 88 in BE COMPLETE.

SJC (of 66 joints) was assessed up to week 104 in BE OPTIMAL and week 100 in BE COMPLETE. The association of inflammation (using SJC as a proxy) with PROs (pain, fatigue, and function), and with disease impact (PsAID-12 total score) is reported here to week 104 in BE OPTIMAL and to week 100/88 in BE COMPLETE. The association of SJC, skin involvement (PASI), and disease impact (PsAID-12 PASS) is also reported to week 104 (BE OPTIMAL) and week 100/88 (BE COMPLETE), in bimekizumab-randomized patients only.

Statistical analysis. Statistical powering and sample size determination for BE OPTIMAL and BE COMPLETE were reported in the primary publications.[31,32] Nonresponder imputation (NRI) was used to impute missing data for binary end points, and multiple imputation was used to impute missing data for continuous outcomes. Data were imputed using original baseline patient numbers from randomization of the feeder studies (BE OPTIMAL and BE COMPLETE) as the denominators in the NRI method. Any patients who did not enter the BE VITAL OLE were imputed as nonresponders, as per the EULAR guidance for reporting clinical trial extension data.[41] Observed case (OC) data are also reported.

The strength of the association between absolute SJC and absolute scores for selected PROs was determined by the rate of change at each reported time point; this was assessed by calculating the slope value and Pearson correlation coefficient, both with a 95% confidence interval (CI). Slope and correlation values were calculated cross-sectionally and not adjusted for repeated observations; the slope was pooled across treatment arms with a separate slope and correlation generated for each visit. No adjustments were made for baseline disease severity, treatment assignment, or other clinical covariates. The slope line estimates the degree to which absolute scores for PROs would be expected to change with a decrease in absolute SJC; a positive slope indicates a direct relationship, and a negative slope indicates an inverse relationship, with the correlation coefficient reflecting the strength and direction of the linear relationship.

Ethics approval. Studies were conducted in accordance with the Declaration of Helsinki and the International Conference on Harmonization guidance for Good Clinical Practice. Ethical approval was obtained from the relevant institutional review boards at participating sites, and all patients provided written informed consent in accordance with local requirements.

RESULTS

Patient disposition and baseline characteristics. Patient disposition to year 2 of BE OPTIMAL (bDMARD-naïve) and BE COMPLETE (TNFi-IR) has been previously reported.[19] In brief, of the 852 bDMARD-naïve patients randomized in BE OPTIMAL, 254 placebo-randomized patients and 379 bimekizumab-randomized patients entered the OLE (BE VITAL) at week 52. Overall, 598 of 712 (84.0%) completed year 2 (week 104). Of the 400 TNFi-IR patients randomized in BE COMPLETE, 121 placebo-randomized patients and 256 bimekizumab-randomized patients entered the OLE at week 16. Overall, 322 of 400 (80.5%) patients completed year 2 (week 100).

Baseline patient demographics and clinical disease characteristics were reported in the primary manuscripts.[20,21,31,32] The average age of patients was 48.5 to 51.3 years with a mean disease duration between 5.6 and 9.6 years (Supplementary Table S2). Mean baseline pain VAS scores were 53.6 to 61.7, and mean baseline FACIT-Fatigue scores ranged between 35.3 and 37.8. Mean HAQ-DI scores were 0.82 to 1.04 at baseline, and mean baseline PsAID-12 total scores ranged between 3.9 and 4.5.

Bimekizumab efficacy on patient-reported symptoms, HRQoL, and disease impact (PsAID-12) up to two years. In patients randomized to bimekizumab at baseline, over half of patients achieved a substantial improvement in pain (Pain50) at year 1 (56.4% [bDMARD-naïve]; 56.9% [TNFi-IR]); these responses were 51.5% at year 2 in bDMARD-naïve patients and 56.2% in TNFi-IR patients. Similar results were observed in placebo-randomized patients who switched to bimekizumab at week 16 (Figure 1).

For bDMARD-naïve bimekizumab-randomized patients, 53.9% achieved FACIT-Fatigue MCID at year 1 and 50.0% at year 2, in TNFi-IR bimekizumab-randomized patients, 58.4% achieved FACIT-Fatigue MCID at year 1 and 52.4% at year 2. Similar results were observed in placebo-randomized patients who switched to bimekizumab (Figure 1). Absolute mean CfB in pain VAS was −29.2 in bDMARD-naïve patients and −33.9 in TNFi-IR bimekizumab-randomized patients at year 2. Mean CfB in fatigue at year 2 was 4.7 in bDMARD-naïve patients and 5.8 in TNFi-IR bimekizumab-randomized patients. Similar results were observed in placebo-randomized patients who switched to bimekizumab. Improvements from baseline in pain and fatigue, measured by absolute changes in the mean to year 2 can be found in Supplementary Figure S2.

Similar trends were observed for bimekizumab-randomized patients and placebo-randomized patients who switched to bimekizumab achieving HAQ-DI MCID across both trials (Figure 1). Values for all data points showing achievement of improvement in pain, fatigue, and function to year 2 shown in Figure 1 are reported in Supplementary Tables S3 to S5.

Improvements in SF-36 PCS scores achieved at year 1 were similar at year 2 in both studies. In bDMARD-naïve bimekizumab-randomized patients, mean PCS scores were 46.2 (95% CI 45.3–47.1) at year 1 and 45.3 (95% CI 44.1–46.5) in placebo-randomized patients who switched to bimekizumab. In TNFi-IR bimekizumab-randomized patients mean PCS scores were 44.8 (95% CI 43.6–46.0) at year 1 and 43.3 (95% CI 41.7–45.0) in placebo-randomized patients who switched to bimekizumab. Mean SF-36 MCS scores at year 1 were also similar at year 2 in both studies. Additional data reporting CfB at year 2 in SF-36 PCS, MCS, and individual domain scores (including the physical functioning domain) can be found in Supplementary Table S6.

Approximately half of patients across treatment arms in both BE OPTIMAL (bDMARD-naïve) and BE COMPLETE (TNFi-IR) reported no or low disease impact (PsAID-12 total score of ≤1.95), at two years of treatment (Figure 2). Proportions of bimekizumab-randomized patients and placebo-randomized patients who switched to bimekizumab reporting no symptom or disease impact at week 16 were sustained or improved further to year 1 and year 2, across both bDMARD-naïve and TNFi-IR patients. Absolute mean CfB in PsAID-12 total score at year 2 was −2.2 in bimekizumab-randomized bDMARD-naïve patients and −2.5 in TNFi-IR patients, with similar results observed in placebo-randomized patients who switched to bimekizumab. Improvements from baseline to week 16 and year 1 in absolute changes to the mean were observed for all mean PsAID-12 single-item domain scores and were sustained to year 2 in both bDMARD-naïve and TNFi-IR patients across treatment arms (Supplementary Figure S3 and Supplementary Table S7).

Specifically, reduced patient-reported impact of skin symptoms was observed at year 1 and sustained to year 2. Mean CfB in the single-item skin problems domain score in bDMARD-naïve bimekizumab-randomized patients was −3.1 (SE 0.2) at year 1, and −2.9 (SE 0.2) in placebo-randomized patients who switched to bimekizumab. In TNFi-IR bimekizumab-randomized patients, mean scores were −3.7 (SE 0.2) at year 1, and −3.6 (SE 0.3) in placebo-randomized patients who switched to bimekizumab (Supplementary Table S7). CfB in PROs including pain VAS, FACIT-Fatigue, HAQ-DI, PsAID-12, and SF-36 at year 1 and year 2 for reference arm–randomized patients can be found in Supplementary Table S8. Following the switch to bimekizumab at year 1, reference arm–randomized patients experienced improvements in PROs which, at year 2, were similar to improvements in

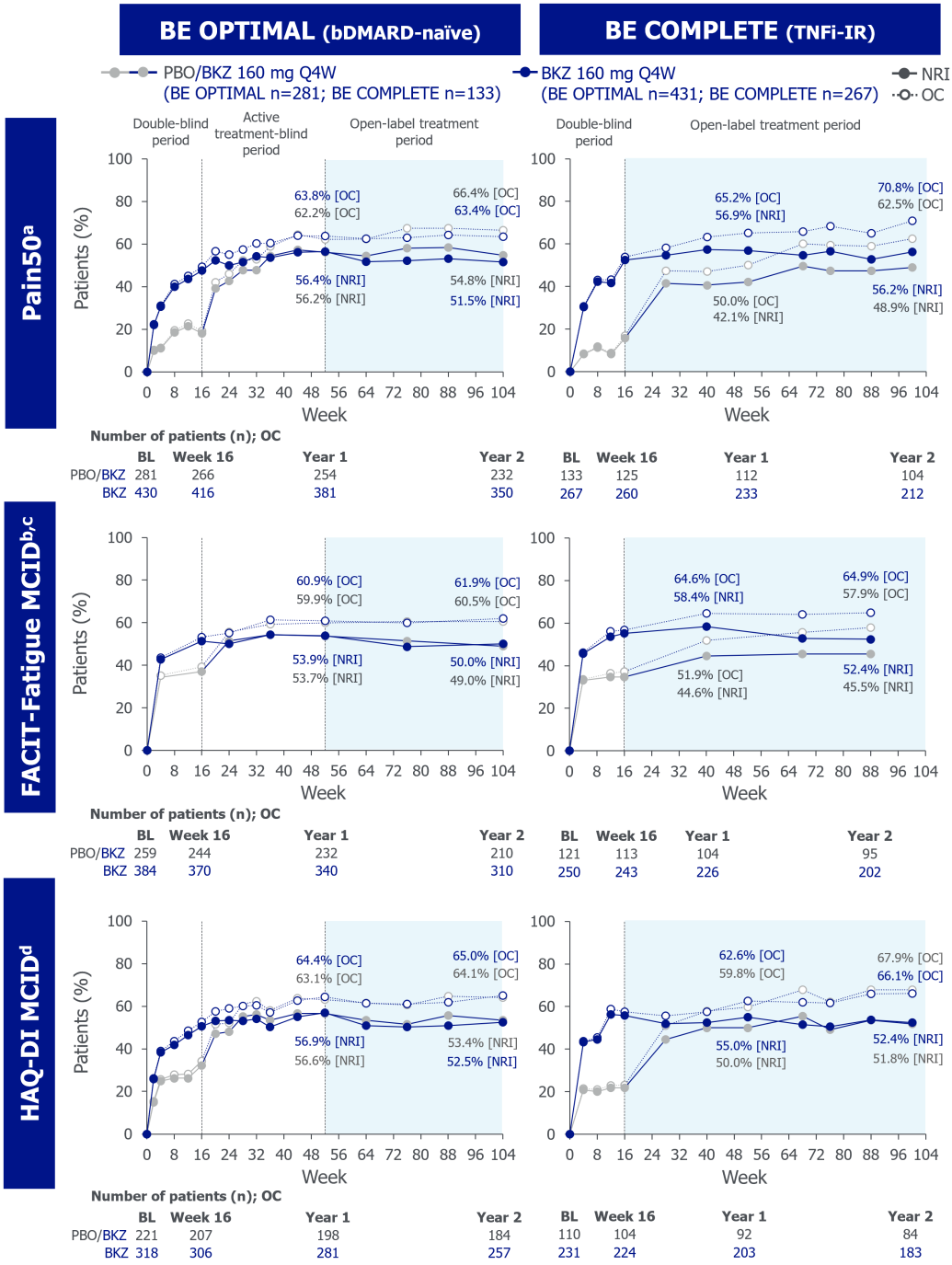

Figure 1. Clinically important improvements in pain, fatigue, and physical function to year 2 (NRI, OC). Randomized set: values for all data points can be found in Supplementary Tables S3 to S5. Data are reported at year 1 (week 52 in BE OPTIMAL and week 52 or week 40 in BE COMPLETE) and year 2 (week 104 in BE OPTIMAL and week 100 or week 88 in BE COMPLETE). [a]Pain50 defined as ≥50% improvement from baseline in pain VAS. Pain VAS was assessed using Patient's Assessment of Arthritis Pain VAS, which ranges from 0 (no pain) to 100 (most severe pain). Pain50 represents a substantial improvement in pain.[34] [b]Data collected at week 40 and week 88 in BE COMPLETE. [c]Fatigue was assessed using FACIT-Fatigue subscale score, which ranges from 0 to 52, with higher scores representing lower levels of fatigue. FACIT-Fatigue MCID defined as ≥4-point increase from baseline in patients with FACIT-Fatigue ≤48 at baseline (BE OPTIMAL placebo/bimekizumab n = 259, bimekizumab n = 384; BE COMPLETE placebo or bimekizumab n = 121, bimekizumab n = 250).[35] [d]HAQ-DI MCID represents a decrease from baseline of ≥0.35, reported in patients with HAQ-DI score ≥0.35 at baseline (BE OPTIMAL placebo or bimekizumab n = 221, bimekizumab n = 318; BE COMPLETE placebo or bimekizumab n = 110, bimekizumab n = 231). bDMARD, biologic disease-modifying antirheumatic drug; BKZ, bimekizumab; BL, baseline; FACIT-Fatigue, Functional Assessment of Chronic Illness Therapy-Fatigue; HAQ-DI, Health Assessment Questionnaire–Disability Index; MCID, minimal clinically important difference; NRI, nonresponder imputation; OC, observed case; PBO, placebo; Q4W, every four weeks; TNFi-IR, tumor necrosis factor inadequate response or intolerance; VAS, visual analog scale.

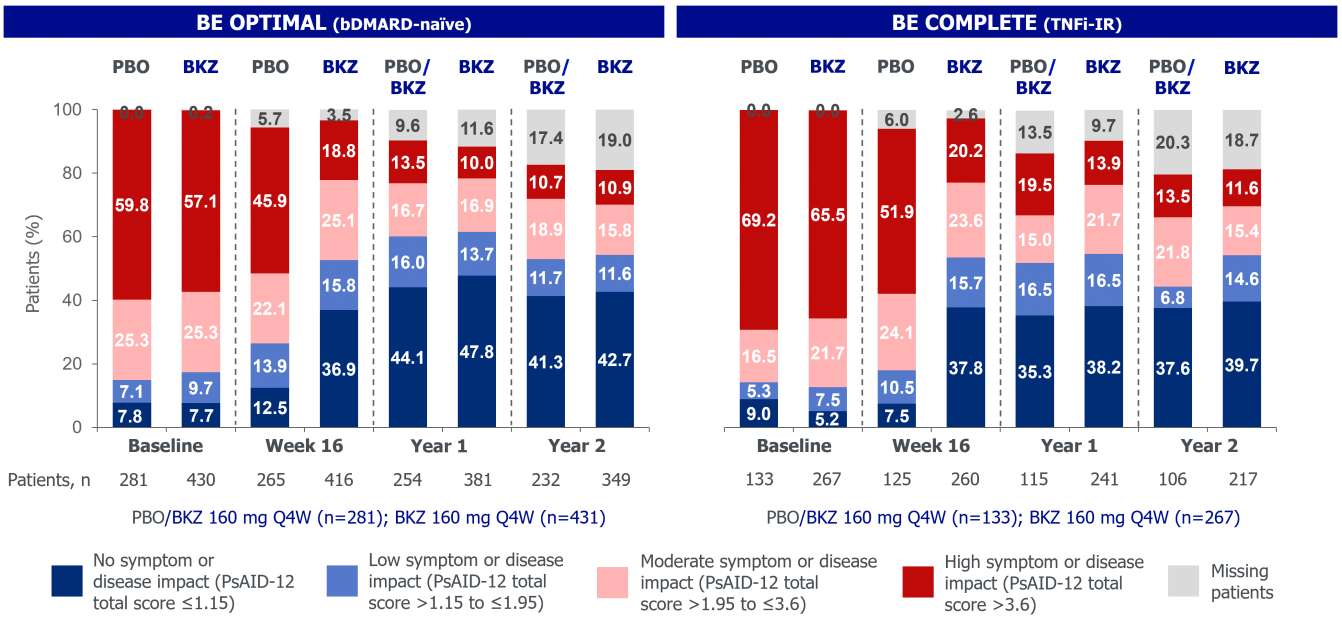

Figure 2. PsAID-12 total score symptom or disease impact by visit, at baseline, week 16, year 1, and year 2 (OC). Randomized set: data are reported at baseline, week 16, year 1 (week 52 in BE OPTIMAL and week 52 or week 40 in BE COMPLETE), and year 2 (week 104 in BE OPTIMAL and week 100 or week 88 in BE COMPLETE). PsAID-12 scores range from 0 to 10; higher scores indicate worse status.[39] Percentages may not sum to 100 as a result of rounding. PsAID-12 data collected at week 40 and week 88 in BE COMPLETE. bDMARD, biologic disease-modifying antirheumatic drug; BKZ, bimekizumab; OC, observed case; PBO, placebo; PsAID-12, Psoriatic Arthritis Impact of Disease-12; Q4W, every four weeks; TNFi-IR, tumor necrosis factor inadequate response or intolerance.

bimekizumab-randomized patients and placebo-randomized patients who switched to bimekizumab.

Association of SJC with PROs. Figure 3 shows the association of SJC = 0 with an achievement of Pain30/50/70 at week 16, year 1, and year 2. Patients were categorized by their pain improvement at week 16, and the patient flow between pain improvement categories is shown to year 2, represented by bands between the vertical bars. The proportion of patients who achieved a Pain30/50/70 response are shown in each colored bar; of those patients, the proportion who achieved SJC = 0 is also reported. These data show that the greatest improvements in patient-reported pain were associated with the resolution of swollen joints (SJC = 0) across both trials.

In bDMARD-naïve bimekizumab-randomized patients at year 1, 56.4% (243 of 431) achieved Pain50 or greater, with the majority of those patients achieving Pain70 (75.3% [183 of 243]). Resolution of swollen joints (SJC = 0) was associated with an achievement of greater improvement in pain; in patients who achieved Pain50, but not Pain70, 68.3% had SJC = 0, whereas in patients who achieved Pain70, 79.2% had SJC = 0. This was sustained to year 2, with 51.5% (222 of 431) of patients achieving Pain50 or greater, with the majority of these patients achieving Pain70 (82.4% [183 of 222]). In patients who achieved Pain50, but not Pain70, 84.6% had SJC = 0 and in patients who achieved Pain70, 83.1% had SJC = 0.

In placebo-randomized patients who switched to bimekizumab, 56.2% (158 of 281) achieved Pain50 or greater at year 1, with the majority of those patients achieving Pain70 (77.2% [122 of 158]; Figure 3). In patients who achieved Pain50, but not Pain70, 58.3% had SJC = 0 and in patients who achieved Pain70, 73.0% had SJC = 0. This was sustained to year 2, with 54.8% (154 of 281) of patients achieving Pain50 or greater and 74.0% of those patients (114 of 154) achieving Pain70. Of patients who achieved Pain50, but not Pain70 at year 2, 65.0% had SJC = 0, and of patients who achieved Pain70, 79.8% had SJC = 0.

Similar results were observed at year 1 in TNFi-IR bimekizumab-randomized patients, with 56.9% (152 of 267) achieving Pain50 or greater and 77.0% (117 of 152) of those achieving Pain70. In patients who achieved Pain50 but not Pain70, 65.7% had SJC = 0, whereas in patients who achieved Pain70, 77.8% had SJC = 0. This was sustained to year 2, with 56.2% (150 of 267) of patients achieving Pain50 or greater and 72.7% (109 of 150) of those achieving Pain70. For patients who achieved Pain50, 70.7% had SJC = 0, and for those who achieved Pain70, 74.3% had SJC = 0.

In TNFi-IR placebo-randomized patients who switched to bimekizumab, 42.1% (56 of 133) achieved Pain50 or greater with 75.0% (42 of 56) of those achieving Pain70 at year 1. For patients who achieved Pain50, 71.4% had SJC = 0, and for those who achieved Pain70, 83.3% achieved SJC = 0. This was sustained to year 2, with 48.9% (65 of 133) of patients achieving Pain50 or

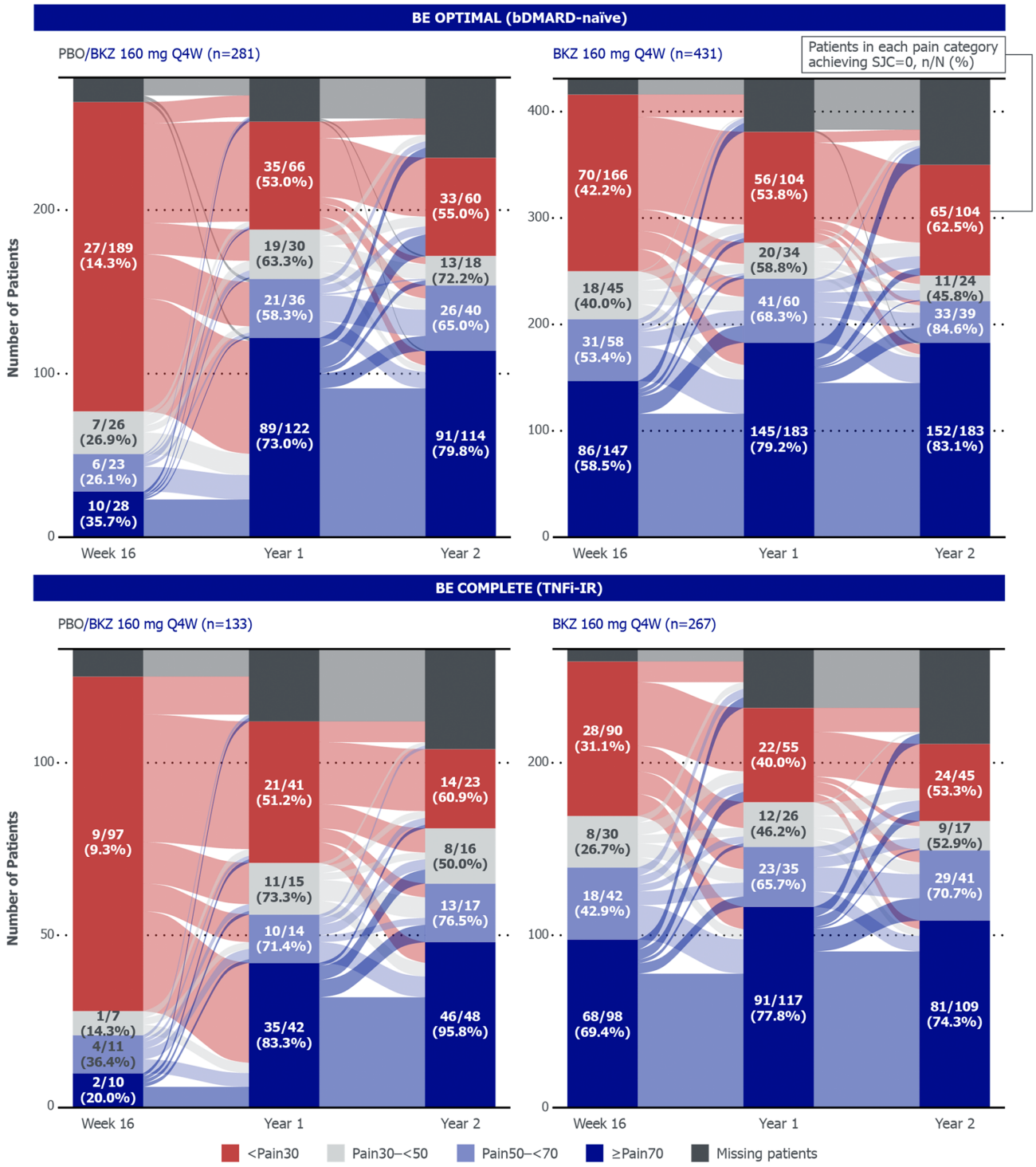

Figure 3. Association of resolution of swollen joints (SJC = 0) with an achievement of ≥30/50/70% improvement from baseline in pain (Pain30/50/70) at week 16, year 1, and year 2 (OC). Randomized set: data are reported at week 16, year 1 (week 52 in BE OPTIMAL and BE COMPLETE), and year 2 (week 104 in BE OPTIMAL and week 100 in BE COMPLETE). Pain30/50/70 defined as ≥30/50/70% improvement from baseline in pain VAS. Pain VAS was assessed using Patient's Assessment of Arthritis Pain VAS, which ranges from 0 (no pain) to 100 (most severe pain). Patients were categorized by their pain improvement at week 16, and the flow of patients between pain improvement categories, represented by bands between the vertical bars, is shown to year 2. The proportion of patients who achieved a Pain30/50/70 response is shown in each colored bar; of those patients, the proportion who achieved SJC = 0 is reported. For example, at week 104 of BE OPTIMAL, 42.5% (183 of 431) of BKZ-randomized patients achieved Pain70. Of those patients, 83.1% (152 of 183) achieved resolution of swollen joints (SJC = 0). bDMARD, biologic disease-modifying antirheumatic drug; BKZ, bimekizumab; OC, observed case; PBO, placebo; Q4W, every four weeks; SJC, swollen joint count; TNFi-IR, tumor necrosis factor inadequate response or intolerance; VAS, visual analog scale.

greater and 73.8% (48 of 65) of those achieving Pain70. Of patients who achieved Pain50, 76.5% had SJC = 0, and for those who achieved Pain70, 95.8% had SJC = 0.

Supplementary Table S9 reports the association of improvement in pain and fatigue in patients with and without resolution of SJC to two years.

Individual patient-level data showed that a reduction in absolute SJC correlated with an improvement (decrease) in HAQ-DI total score in both bDMARD-naïve and TNFi-IR patients (Figure 4). The rate of change, which was unadjusted and pooled across treatment arms, increased from baseline to week 16, and was sustained to year 1 and year 2 for bDMARD-naïve bimekizumab-randomized patients and placebo-randomized patients who switched to bimekizumab. Similar increased rates of change were observed in TNFi-IR bimekizumab-randomized patients and placebo-randomized patients who switched to bimekizumab, which were also sustained to year 2.

Correlations between absolute SJC and PsAID-12 total score, determined by increased rates of change, were also reported for both bDMARD-naïve and TNFi-IR bimekizumab-randomized patients and placebo-randomized patients who switched to bimekizumab from baseline to year 1, and these were sustained or increased further to year 2 (Figure 5).

Similar correlations were also found between absolute SJC and pain (Supplementary Figure S4) and between SJC and fatigue (Supplementary Figure S5) to year 2.

Concurrent improvement of joint (SJC), skin (PASI) disease, and reduced disease impact (PsAID-12 total score) was observed from baseline to week 16, with improvements sustained out to two years. Figure 6 shows the distribution of individual

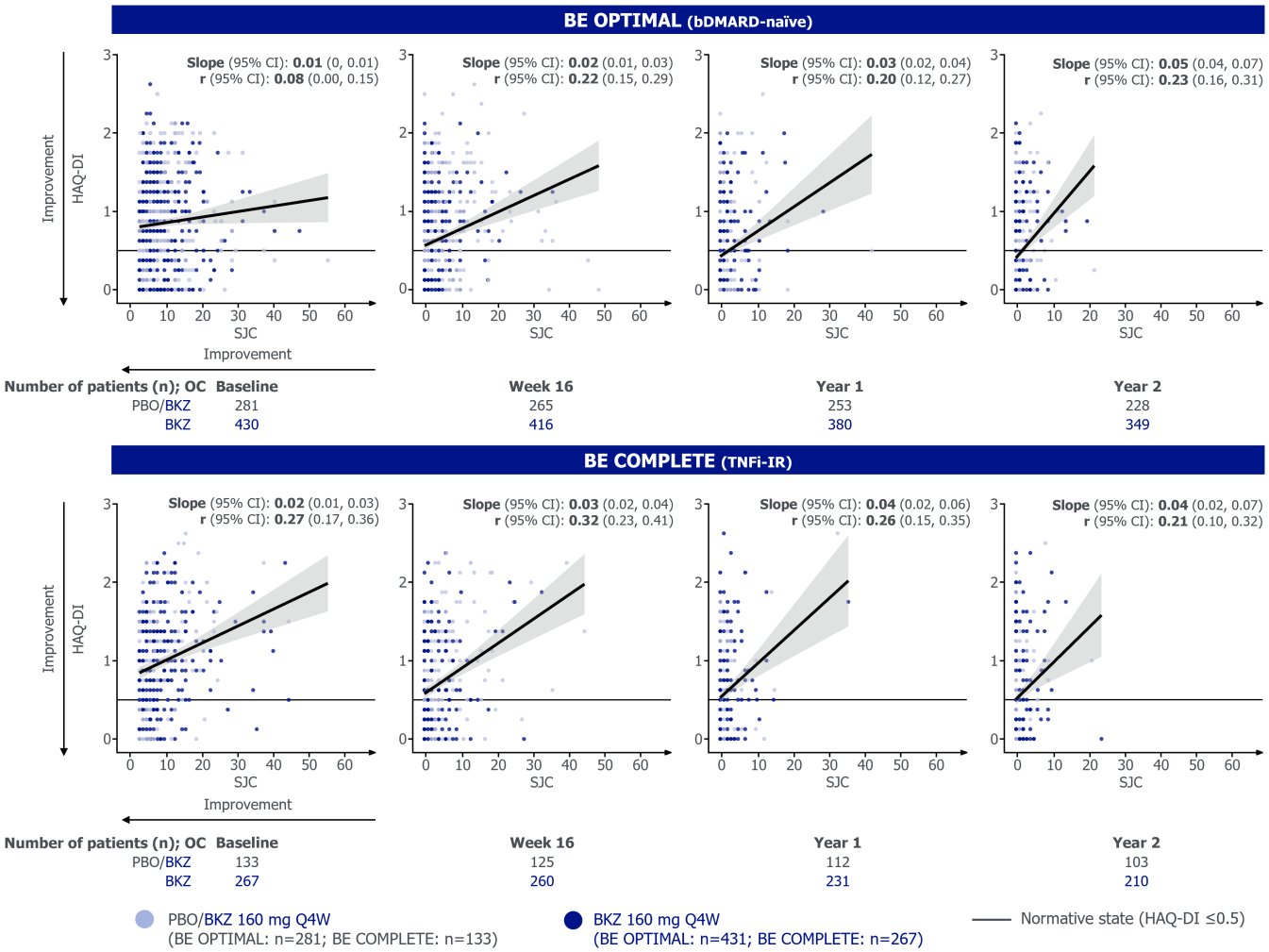

Figure 4. Association of absolute SJC with HAQ-DI total score at baseline, week 16, year 1, and year 2 (OC). Randomized set: data are reported at baseline, week 16, year 1 (week 52 in BE OPTIMAL and BE COMPLETE), and year 2 (week 104 in BE OPTIMAL and week 100 in BE COMPLETE). Values for the slope (95% CI) and Pearson's correlation coefficient (r; 95% CI) apply to all patients at that time point regardless of treatment arm. The threshold line represents HAQ-DI normative state, defined as HAQ-DI ≤ 0.5. bDMARD, biologic disease-modifying antirheumatic drug; CI, confidence interval; HAQ-DI, Health Assessment Questionnaire–Disability Index; OC, observed case; Q4W, every four weeks; SJC, swollen joint count; TNFi-IR, inadequate response or intolerance to tumor necrosis factor inhibitors.

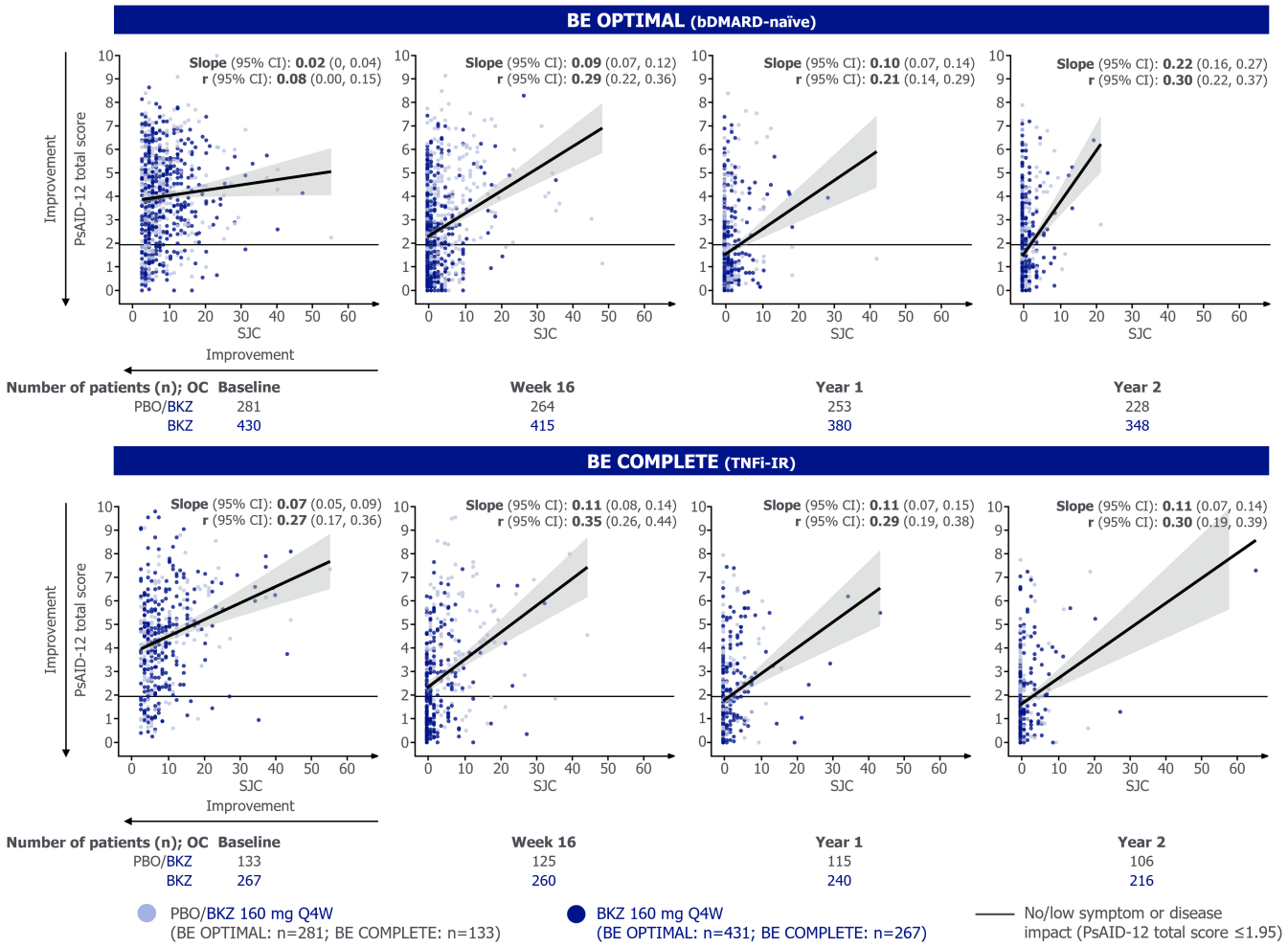

Figure 5. Association of absolute SJC with PsAID-12 total score at baseline, week 16, year 1, and year 2 (OC). Randomized set: data are reported at baseline, week 16, year 1 (week 52 in BE OPTIMAL and week 40 in BE COMPLETE), and year 2 (week 104 in BE OPTIMAL and week 88 in BE COMPLETE). Values for the slope (95% CI) and Pearson's correlation coefficient (r; 95% CI) apply to all patients at that time point regardless of treatment arm. PsAID-12 scores range from 0 to 10, where higher scores indicate worse status.[39] The threshold line represents no or low symptom or disease impact, defined as PsAID-12 score ≤1.95.[39] bDMARD, biologic disease-modifying antirheumatic drug; CI, confidence interval; OC, observed case; PsAID-12, Psoriatic Arthritis Impact of Disease-12; Q4W, every four weeks; SJC, swollen joint count; TNFi-IR, tumor necrosis factor inadequate response or intolerance.

patient-level results in bimekizumab-randomized patients between SJC, PASI, and PsAID-12 at baseline, week 16, year 1, and year 2, with a reference plane at the PASS threshold (PsAID-12 = 4); the proportion of patients above and below the reference plane is also shown. Only patients with a complete data set at each visit are shown; patients missing any PASI, SJC, or PsAID-12 value(s) at the visit were excluded from the analysis. The proportion of bimekizumab-randomized patients achieving PsAID-12 PASS with reduction of SJC and PASI, reported using OC data, was 50.5% (bDMARD-naïve) and 40.1% (TNFi-IR) at baseline. This improved to week 16 with 85.5% of bDMARD-naïve patients and 83.1% of TNFi-IR patients achieving PsAID-12 PASS and was sustained up to two years (92.0% [bDMARD-naïve] and 88.0% [TNFi-IR]). Still images of 3D plots from one angle are shown in Figure 6; videos of individual 3D plots can be found in the Supplementary Material.

The proportion of bimekizumab-randomized patients and placebo-randomized patients who switched to bimekizumab and achieved PsAID-12 PASS up to two years was 69.9% to 73.7% (NRI). PsAID-12 PASS responders at week 16, year 1, and year 2 for bDMARD-naïve and TNFi-IR patients across all treatment arms are provided in Supplementary Figure S6.

DISCUSSION

Long-term treatment with bimekizumab led to sustained, clinically meaningful improvements in patient-reported symptoms and overall disease impact up to two years in patients with PsA. These findings support previously published data and provide additional evidence that patients receiving bimekizumab achieve

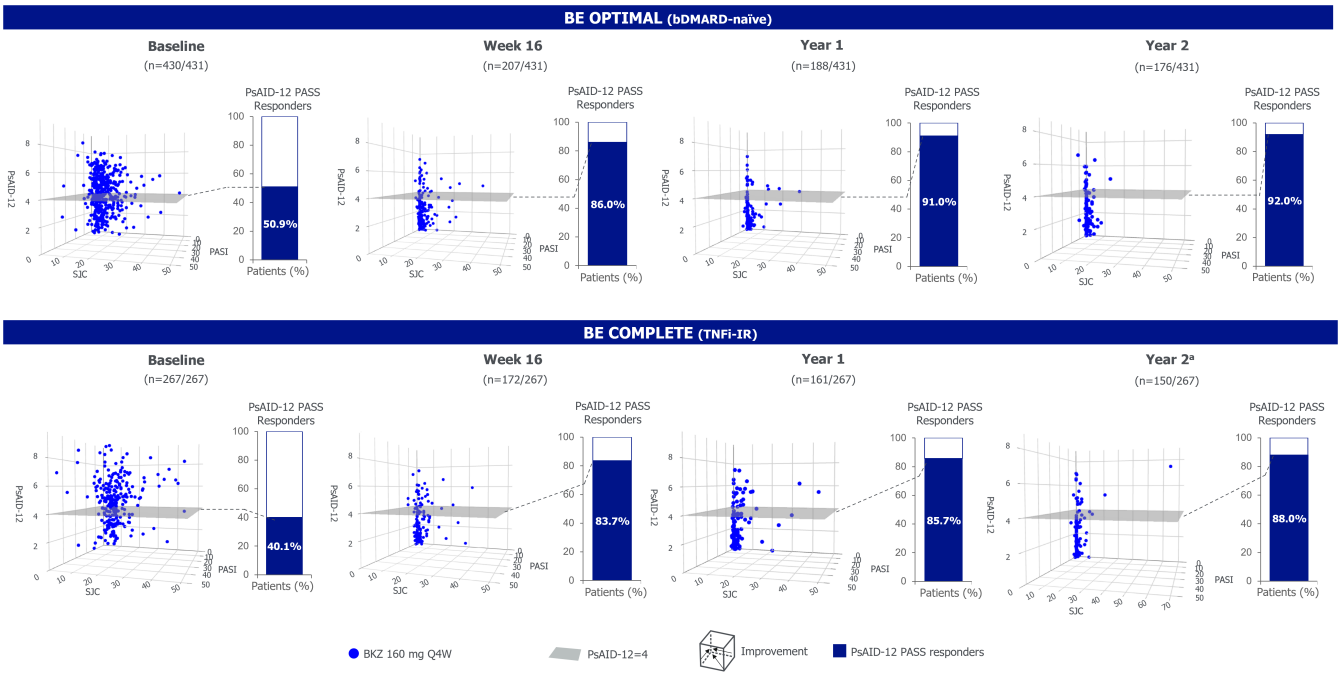

Figure 6. Association of SJC, PASI, and PsAID-12 at baseline, week 16, year 1, and year 2 (observed case). Randomized set: data are reported at baseline, week 16, year 1 (week 52 in BE OPTIMAL and week 40 in BE COMPLETE), and year 2 (week 104 in BE OPTIMAL and week 88 in BE COMPLETE). Only patients with a complete data set at each visit are shown; patients missing any PASI, SJC, or PsAID-12 value(s) at the visit were excluded from the analysis. The threshold plane represents the cutoff for the PsAID-12 PASS, where PsAID-12 PASS is defined as a PsAID-12 total score ≤4[40]; proportions of patients equal to or below this threshold (PsAID-12 PASS responders) are reported as percentages of the total number of patients assessed at each time point. [a]SJC axis is reported on a different scale (0–70) at year 2 compared with other time points to capture outlier data. bDMARD, biologic disease-modifying antirheumatic drug; n/N, number of patients who had available PASI, SJC, and PsAID-12 data available at a given time point divided by the total number of bimekizumab-randomized patients; PASI, Psoriasis Area and Severity Index; PASS, patient acceptable symptom state; PsAID-12, Psoriatic Arthritis Impact of Disease-12; Q4W, every four weeks; SJC, swollen joint count; TNFi-IR, tumor necrosis factor inadequate response or intolerance.

durable control of inflammation across multiple domains of disease, translating into meaningful relief from the symptoms that patients prioritize most.[14–16] Disease impact states (PsAID-12) by visit up to two years are reported here for the first time. We have also reported, for the first time, the association between SJC and improved PROs and the association between SJC, skin involvement (PASI), and disease impact (PsAID-12). PsA treatment goals advocate for evaluating as many disease domains as possible, including those best captured by PROs.[3] These results represent a thorough evaluation of the effects of bimekizumab on patient-reported symptoms and disease impact, in line with these recommendations. The associations-based analyses highlight the importance of achieving inflammation control and the continued impact of that control on symptom alleviation, from the patients' perspective, up to two years.

The greatest improvements in patient-reported pain were observed in patients who achieved complete resolution of swollen joints (SJC = 0). Reductions in SJC were associated with improvements in physical function, as measured by HAQ-DI, and overall disease impact, assessed by PsAID-12 at 16 weeks; these associations were sustained through two years of bimekizumab treatment. SJC was selected as a surrogate for inflammation control because, in comparison to other multidimensional disease activity measures that may be influenced by several components, it is a reliable, objective, standalone metric, frequently performed both in clinical practice and trial settings. These analyses support clinical decision-making by quantifying the relationship between objective clinical measures such as SJC and PROs, providing an evidence-based understanding of how patient experiences reflect underlying disease activity. These findings could help clinicians interpret discordances, tailor treatment discussions, and better integrate PROs into routine patient management.

High proportions of patients receiving bimekizumab achieved PsAID-12 PASS with lower SJC and PASI scores at two years, compared with baseline, showing that control of inflammation in the skin and joints are associated with a meaningful reduction in disease impact. This association is further shown in other studies; a study of ixekizumab treatment in PsA similarly found that treatment of both joint and skin symptoms resulted in optimal improvements in HRQoL, and another ixekizumab study reported that improvements in PROs were associated with

synergistic improvements in synovitis and enthesitis.[42,43] These results further support the importance of inflammation control for patients, both in achieving outcomes such as SJC resolution, and in the accompanying symptom improvement and reduced impact on HRQoL that patients experience.

Reductions in SJC observed with bimekizumab treatment were also correlated with improvements in both patient-reported pain and fatigue, with a stronger correlation observed between SJC and pain. Some patients' HAQ-DI, PsAID-12, or fatigue scores remained high, despite SJC reduction across the population, which could be indicative of noninflammatory contributors, such as central pain or structural damage. High levels of fatigue have been linked with a greater number of affected joints and high pain levels.[18] Despite this, the relationship between inflammation and fatigue in PsA has not been well defined and reductions in disease activity have not been shown to consistently correlate with reductions in fatigue.[27,44,45] There is a more prominent relationship between pain and inflammation, and many bDMARDs have been found to have a positive impact on reducing inflammation and pain symptoms in PsA.[27] However, pain has been shown to be persistent in PsA and can remain even in patients who respond to clinical disease activity measures.[27,46] The evolving concepts of difficult-to-treat (D2T) and complex-to-manage (C2M) PsA reflect the multifaceted nature of disease burden in patients with PsA. Patients with D2T PsA continue to experience treatment failure, clinically meaningful disease activity and patient perceived impact, even after receiving multiple DMARDs or biologics. C2M PsA encompasses patients whose disease management is complicated by noninflammatory mechanisms, comorbidities, and psychosocial factors, despite apparent clinical control.[47–49] Pain can also show a disconnect from measured inflammation, with patients reporting pain even when inflammatory activity is low, which may also be driven by noninflammatory pain mechanisms.[49,50] These factors were not explored in this analysis which primarily focused on the strong link between the reduction in clinically assessed inflammation and improvements in PROs, emphasizing that achieving control of inflammation is associated with reduced pain in patients with PsA.

Sustained treatment effects are critically needed in PsA as many patients require switching or cycling of bDMARDs due to suboptimal treatment response or loss of efficacy,[51,52] a challenge compounded by the chronic nature of the disease. Previous studies have shown that patients who were bDMARD-naïve and TNFi-IR experienced similar improved responses in patient-reported symptoms and disease impact outcomes with bimekizumab treatment up to one year.[23,24] These results expand these findings to two years, demonstrating that bimekizumab provides durable symptom relief and reduced disease burden, irrespective of prior TNFi exposure.

These studies assessed multiple PROs and measures of disease impact in patients with PsA receiving bimekizumab. Reporting the long-term impact of bimekizumab treatment on outcomes which patients have identified as meaningful to their HRQoL is important given the chronic nature of PsA. These results establish a link between control of inflammation, assessed using SJC, and improvements in patient-reported symptoms. This emphasizes the relationship between the achievement of clinical efficacy outcomes and patients' experience of symptom relief, and the impact of PsA on their HRQoL.

An additional strength of this analysis is that response data were imputed using NRI from baseline of BE OPTIMAL and BE COMPLETE and were not modified at any later time point which is a more conservative method. Bimekizumab treatment was also evaluated in both bDMARD-naïve and TNFi-IR populations over a duration of two years.

The OLE study, BE VITAL, was not designed to compare treatment groups from BE OPTIMAL and BE COMPLETE; in BE COMPLETE, data reported after week 16 are from BE VITAL. Several efficacy outcomes, including PROs, were not collected at the same time points in BE OPTIMAL and BE COMPLETE, limiting the pooling of data and the opportunity to directly compare trial groups at specific time points. Reference arm patients in BE OPTIMAL switched from adalimumab to bimekizumab at week 52 as per the study design and had received bimekizumab for one year at the time of reporting. These patients were therefore not included in this two-year evaluation of bimekizumab. The results achieved with bimekizumab treatment do not have a numerical or visual comparator reference, therefore limiting interpretation of whether bimekizumab treatment can maintain responses achieved by other treatments.

Another limitation of the study is that it reported data from a clinical trial population and, therefore, it may not be suitable to generalize these results to a patient population typically seen in clinical practice. Additionally, clinical trial cohorts usually include active inflammatory disease as part of their inclusion criteria; therefore, the association between pain and inflammation may be weaker when considering all patients with PsA and particularly patients with D2T or C2M PsA. There is also limited generalizability to patients with advanced therapy failures beyond one TNF inhibitor. Furthermore, assessment of patient-reported skin symptoms was limited to the PsAID-12 single-item skin question; additional patient-reported skin-focused outcomes such as the Dermatology Life Quality Index were not collected in these studies.

For the association analyses, no assessment of causality was performed and correlations were unadjusted. These analyses considered the relationship between improvements in PROs including disease impact, a skin outcome (PASI), and inflammation via SJC; however, the observed improvements and reduced disease impact are most likely multifactorial. Contributing factors are likely, and include psychological well-being, enthesitis, and physical functioning, which may have noninflammatory mechanisms in addition to inflammatory ones. These factors were not accounted for in our estimation of the relationship between

reduced SJC and improvements in PROs, therefore this analysis may represent a slight overestimation in treatment benefit. Treatment effect should also be interpreted with caution due to the impact of nonlinearity on efficacy outcomes. As SJC approaches zero, further reductions become unlikely, compressing early SJC changes and creating a nonlinear relationship with improvements in PROs, especially for patients with a low baseline SJC. Real-world evidence has also shown substantial PRO improvements in the first six months following initiation of a TNFi, with improvements sustained thereafter.[53]

Future work is needed to further explore the relationships between clinical outcomes, objective measures of inflammation and PROs, with the goal of better characterizing how inflammation control can translate into symptom relief and reduced disease burden. Assessing the impact of achieving control of inflammation across additional disease domains and the correlation with PROs could also be explored and may further elucidate the effect of bimekizumab treatment on inflammation control and patients' HRQoL in clinical practice. Analysis of HRQoL measures and work productivity, including evaluation of economic impact, should be considered to assess the impact of treatment with bimekizumab on the ability of patients to undertake and maintain paid work. Future research may also benefit by exploring the specific characteristics of patients who are in a state of moderate or high disease activity yet remain in the study receiving bimekizumab.

Clinically meaningful improvements in patient-reported symptoms and reduced disease impact with bimekizumab treatment were sustained up to two years, with findings consistent between bDMARD-naïve and TNFi-IR patients with PsA. Stringent control of inflammation, measured by reduced swollen joints, was associated with symptom relief and reduced disease impact. These findings demonstrate the impact of long-term controlled inflammation with bimekizumab treatment for patients in improving their symptoms and should be explored further in real-world evidence studies.

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AUTHOR CONTRIBUTIONS

All authors contributed to at least one of the following manuscript preparation roles: conceptualization AND/OR methodology, software, investigation, formal analysis, data curation, visualization, and validation AND drafting or reviewing/editing the final draft. As corresponding author, Dr Gossec confirms that all authors have provided the final approval of the version to be published and takes responsibility for the affirmations regarding article submission (eg, not under consideration by another journal), the integrity of the data presented, and the statements regarding compliance with institutional review board/Declaration of Helsinki requirements.

ROLE OF THE STUDY SPONSOR

The study sponsor (UCB) had a role in the study design and in the collection, analysis, and/or interpretation of the data provided. The study sponsor had input during manuscript development, including editorial suggestions and interpretation, under the direction of the authors. The authors retained final responsibility for the content, interpretation of data, and the decision to publish. Support for third-party medical writing and editorial assistance provided by Alice Di Vincenzo, MSc, and Laura Mawdsley, MSc of Costello Medical, United Kingdom, based on the authors' input and direction and was funded by UCB in accordance with Good Publication Practice guidelines.

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