Bimekizumab in Psoriatic Arthritis

For proactive use by medical affairs personnel

Inspired by **patients.**
Driven by **science.**

Intended for healthcare professionals

Disclaimer

BIMZELX is indicated for the treatment of adult patients with active psoriatic arthritis. The recommended dosage is 160 mg by subcutaneous injection every 4 weeks. For psoriatic arthritis patients with coexistent moderate to severe plaque psoriasis, use the dosing regimen for adult patients with plaque psoriasis. The recommended dosage of BIMZELX is 320 mg (given as 2 subcutaneous injections of 160 mg each) at Weeks 0, 4, 8, 12, and 16, then every 8 weeks thereafter. For patients weighing ≥120 kg, consider a dosage of 320 mg every 4 weeks after Week 16.

US-BK-2401175 | Date of preparation: September 2024

BIMZELX® [prescribing information]. Smyrna, GA: UCB, Inc.

Scan for BIMZELX Prescribing Information

Publications of BKZ Phase 3 Trials in PsA

McInnes IB, et al. (**2023**)
Bimekizumab in patients with psoriatic arthritis, naïve to biologic treatment: a randomized, double-blind, placebo-controlled, phase 3 trial (**BE OPTIMAL**)

US-BK-2400557

Ritchlin CT, et al. (**2023**)
Bimekizumab treatment in biologic DMARD-naïve patients with active psoriatic arthritis: 52-week efficacy and safety results from the phase III, randomized, placebo-controlled, active reference **BE OPTIMAL** study

US-BK-2400561

Merola JF, et al. (**2023**)
Bimekizumab in patients with active psoriatic arthritis and previous inadequate response or intolerance to tumour necrosis factor-α inhibitors: a randomized, double-blind, placebo-controlled, phase 3 trial (**BE COMPLETE**)

US-BK-2400563

Coates LC, et al. (**2024**)
Bimekizumab treatment in patients with active psoriatic arthritis and prior inadequate response or intolerance to tumour necrosis factor inhibitors: 52-week safety and efficacy from the phase III **BE COMPLETE** study and its open-label extension **BE VITAL**

US-BK-2400567

BE OPTIMAL Study Design

Key inclusion criteria[1]

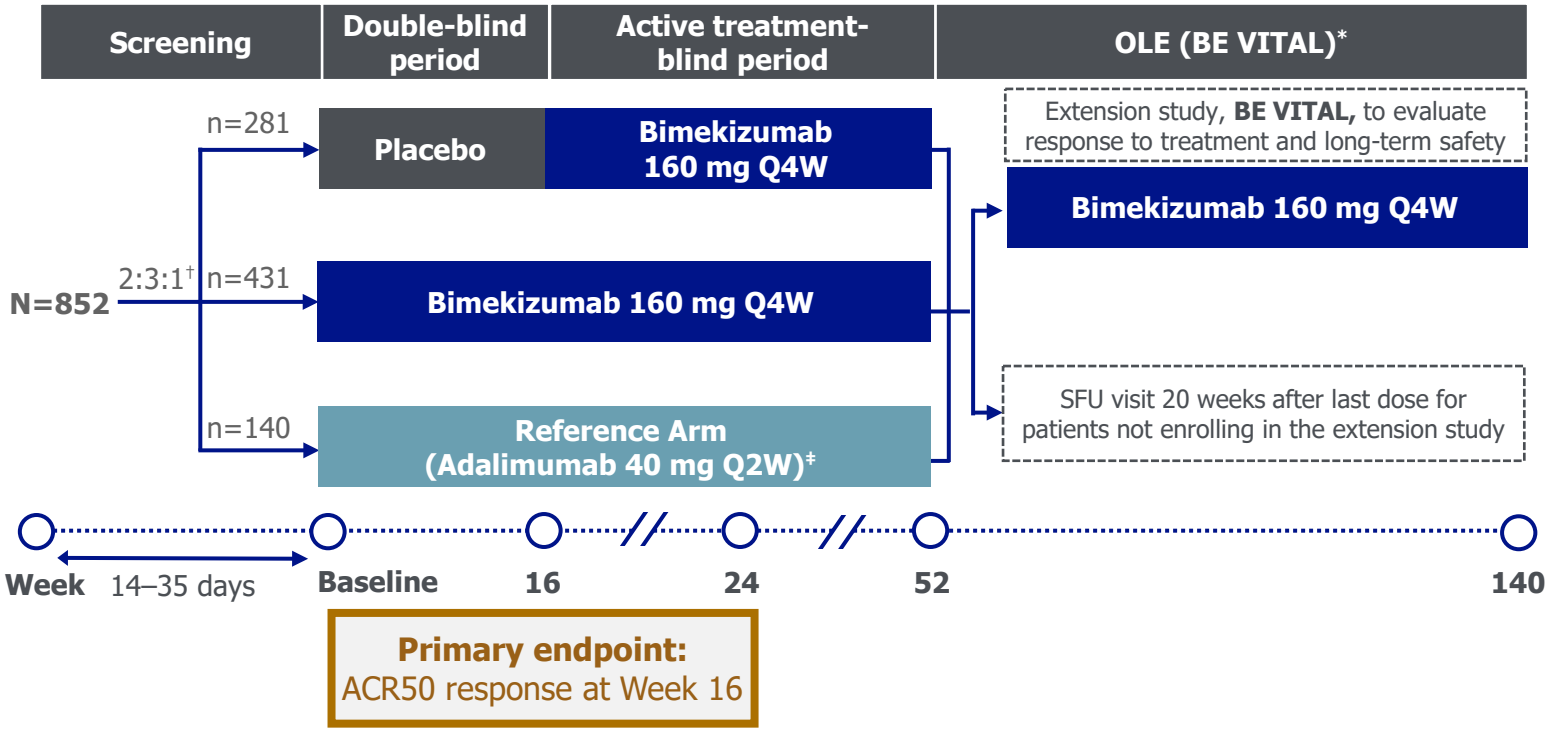
- ≥18 years of age with adult-onset PsA fulfilling CASPAR criteria with a duration of ≥6 months before screening
- TJC ≥3/68 and SJC ≥3/66
- ≥1 active psoriatic lesions and/or a documented history of PSO

Key exclusion criteria[1,2]

- Current or prior exposure to any biologics for treatment of PsA or PSO
- Active, symptomatic IBD at baseline or screening (prior history was not an exclusion criterion)

Patients were allowed concomitant NSAIDs, analgesics, oral corticosteroids, or conventional synthetic DMARDs at stable doses[1]

BE OPTIMAL (bDMARD-naïve patients)[1-5]



CASPAR; classification criteria for psoriatic arthritis; NSAIDs, non-steroidal anti-inflammatory drugs.
BKZ-treated patients were eligible to receive rescue therapy from Week 16 at the discretion of the investigator, while continuing to receive BKZ.[1] *Response to treatment to 140 weeks.[5] †Patients were stratified by bone erosions number at baseline (0 or ≥1) and region (North America, western Europe, eastern Europe, or Asia).[1] ‡The adalimumab 40 mg Q2W treatment arm served as an active reference.[1] The BE OPTIMAL study was not powered for statistical comparisons of adalimumab to bimekizumab or adalimumab to placebo.[1]

1. McInnes IB, et al. Lancet. 2023;401(10370):25–37. 2. McInnes IB, et al. Supplementary appendix. Lancet. 2023;401(10370):25-37. 3. Ritchlin CT, et al. Supplementary appendix. Ann Rheum Dis. 2023;82(1):1404-1414. 4. Ritchlin CT, et al. Ann Rheum Dis. 2023;82(1):1404-1414. 5. Coates LC, et al. RMD Open. 2024;1(0):e003855.

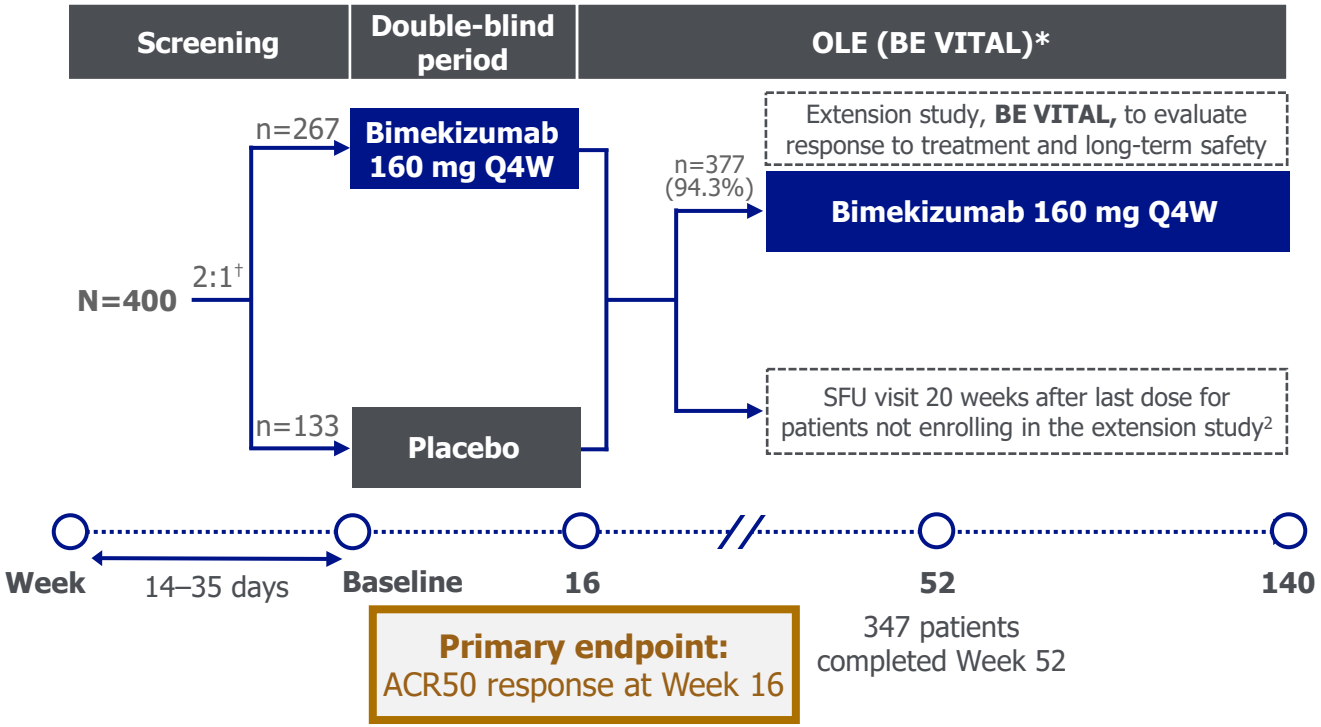
BE COMPLETE Study Design

Key inclusion criteria[1]
- ≥18 years of age with adult-onset PsA fulfilling CASPAR criteria with a duration of ≥6 months before screening
- TJC ≥3/68 and SJC ≥3/66
- ≥1 active psoriatic lesions and/or a documented history of PSO
- Inadequate response or intolerance to 1 or 2 TNFi for either PsA or PSO

Key exclusion criteria[1,2]
- Current or prior exposure to any biologics other than TNFi for treatment of PsA or PSO
- Active, symptomatic IBD at baseline or screening (prior history was not an exclusion criterion)

Patients were allowed concomitant NSAIDs, analgesics, oral corticosteroids, or conventional synthetic DMARDs at stable doses[1]

BE COMPLETE (TNFi-IR patients)[1-4]



CASPAR; classification criteria for psoriatic arthritis; NSAIDs, non-steroidal anti-inflammatory drugs.
BKZ-treated patients were eligible to receive rescue therapy from Week 16 at the discretion of the investigator, while continuing to receive BKZ.[2] *Response to treatment to 140 weeks.[3] †Patients were stratified by region previous exposure to TNFi (inadequate response to one or two TNFi, or intolerance to TNFi) and region (North America, western Europe, eastern Europe, or Asia).[1]

1. Merola JF, et al. Lancet. 2023;401(10370):38–48. 2. Merola JF, et al. Supplementary appendix. Lancet. 2023;401(10370):38-48. 3. Coates LC, et al. RMD Open. 2024;1(0):e003855. 4. Adapted from Coates LC, et al. Supplementary appendix. RMD Open. 2024;1(0):e003855.

US-BK-2401175 | Date of preparation: September 2024

Statistical Testing Hierarchy

BE OPTIMAL (bDMARD-naïve patients)[1]

Bimekizumab 160 mg Q4W α=0.05	Outcome	Test
H_1	Primary	ACR50 response at Week 16 superior to placebo
H_2	Secondary	Change from baseline in HAQ-DI at Week 16 superior to placebo
H_3	Secondary	PASI90 response at Week 16 superior to placebo*
H_4	Secondary	Change from baseline in SF-36 PCS at Week 16 superior to placebo
H_5	Secondary	MDA response at Week 16 superior to placebo
H_6	Secondary	Change from baseline in vdHmTSS at Week 16 superior to placebo†
H_7	Secondary	Enthesitis free response at Week 16 superior to placebo (in the pooled BE OPTIMAL/BE COMPLETE population)
H_8	Secondary	Dactylitis free response at Week 16 superior to placebo (in the pooled BE OPTIMAL/BE COMPLETE population)
H_9	Secondary	Change from baseline in vdHmTSS at Week 16 superior to placebo (overall population)

BE COMPLETE (TNFi-IR patients)[2]

Bimekizumab 160 mg Q4W α=0.05	Outcome	Test
H_1	Primary	ACR50 response at Week 16 superior to placebo
H_2	Secondary	Change from baseline in HAQ-DI at Week 16 superior to placebo
H_3	Secondary	PASI90 response at Week 16 superior to placebo*
H_4	Secondary	Change from baseline in SF-36 PCS at Week 16 superior to placebo
H_5	Secondary	MDA response at Week 16 superior to placebo

ACR: American College of Rheumatology; HAQ-DI: Health Assessment Questionnaire – Disability Index; MDA: Minimal Disease Activity; PASI: Psoriasis Area and Severity Index; PCS: Physical Component Summary; Q4W: every 4 weeks; SF-36: Short-Form 36-item Health Survey.
*In patients with psoriasis affecting at least 3% BSA at baseline.[1,2] †In patients with elevated hs-CRP (≥6mg/L) and/or at least one bone erosion.[1]

1. Adapted from McInnes IB, et al. Supplementary appendix. Lancet. 2023;401(10370):25-37. 2. Adapted from Merola JF, et al. Supplementary appendix. Lancet. 2023;401(10370):38-48.

BE OPTIMAL and BE COMPLETE: Baseline Characteristics (1/2)

	BE OPTIMAL[1]			BE COMPLETE[2]	
	Placebo (n=281)	BKZ 160 mg Q4W (n=431)	Reference Arm (ADA 40 mg Q2W; n=140)	Placebo (n=133)	BKZ 160 mg Q4W (n=267)
Age, years, mean (SD)	48.7 (11.7)	48.5 (12.6)	49.0 (12.8)	51.3 (12.9)	50.1 (12.4)
Sex, male, n (%)	127 (45)	201 (47)	71 (51)	60 (45)	130 (49)
BMI, kg/m^2, mean (SD)	29.6 (6.1)	29.2 (6.8)	28.4 (5.9)	29.0 (5.4)	30.1 (6.5)
Time since PsA diagnosis,* years, mean (SD)	5.6 (6.5)	6.0 (7.3)	6.1 (6.8)	9.2 (8.1)	9.6 (9.9)
csDMARDs at baseline, n (%)	194 (69)	301 (70)	99 (71)	63 (47)	139 (52)
Concomitant methotrexate, n (%)	163 (58)	252 (59)	82 (59)	51 (38)	119 (45)
Prior TNFi exposure, n (%)					
Inadequate response to 1 TNFi	-	-	-	103 (77)	204 (76)
Inadequate response to 2 TNFi	-	-	-	15 (11)	29 (11)
Intolerance to TNFi	-	-	-	15 (11)	34 (13)
TJC (of 68 joints), mean (SD)	17.1 (12.5)	16.8 (11.8)	17.5 (13.1)	19.3 (14.2)	18.4 (13.5)
SJC (of 66 joints), mean (SD)	9.5 (7.3)	9.0 (6.2)	9.7 (7.1)	10.3 (8.2)	9.7 (7.5)
hs-CRP ≥6 mg/L, n (%)	121 (43)	158 (37)	44 (31)	59 (44)	118 (44)

Randomized set.[1,2] BE OPTIMAL: the study was not powered for statistical comparisons of ADA to BKZ or ADA to PBO.[1] *BE OPTIMAL: data missing for 2 placebo patients, 8 BKZ patients and 1 ADA patient; BE COMPLETE: data missing for 1 placebo patient and 1 BKZ patient.[1,2]

1. Adapted from Ritchlin CT, et al. Ann Rheum Dis. 2023;82(11):1404–1414. 2. Adapted from Merola JF, et al. Lancet. 2023;401(10370):38–48.

US-BK-2401175 | Date of preparation: September 2024

BE OPTIMAL and BE COMPLETE: Baseline Characteristics (2/2)

	BE OPTIMAL[1]			BE COMPLETE[2]	
	Placebo (n=281)	BKZ 160 mg Q4W (n=431)	Reference Arm (ADA 40 mg Q2W; n=140)	Placebo (n=133)	BKZ 160 mg Q4W (n=267)
Affected BSA ≥3%, n (%)	140 (50)	217 (50)	68 (49)	88 (66)	176 (66)
PASI score,* mean (SD)	7.9 (5.6)	8.2 (6.8)	8.6 (7.6)	8.5 (6.6)	10.1 (9.1)
HAQ-DI,† mean (SD)	0.89 (0.61)	0.82 (0.59)	0.86 (0.54)	1.0 (0.69)	1.0 (0.59)
SF-36 PCS,‡ mean (SD)	36.9 (9.7)	38.1 (9.4)	37.6 (8.8)	35.9 (10.2)	36.4 (9.0)
PtAAP,† mean (SD)	56.8 (23.2)	53.6 (24.3)	56.7 (23.9)	61.7 (24.6)	58.3 (24.2)
vdHmTSS (at risk subgroup),§ mean (SD)	14.5 (23.9)	14.4 (32.0)	16.5 (28.4)	-	-
vdHmTSS (overall),‖ mean (SD)	12.3 (22.5)	12.5 (30.0)	13.8 (26.5)	-	-
Nail psoriasis,¶ n (%) mNAPSI score,** mean (SD)	156 (56) 4.0 (2.1)	244 (57) 4.1 (2.5)	75 (54) 3.7 (2.3)	83 (62) 4.5 (2.8)	159 (60) 4.3 (2.8)
Enthesitis,†† n (%) LEI score,‡‡ mean (SD)	70 (25) 2.9 (1.5)	143 (33) 2.5 (1.5)	36 (26) 2.3 (1.6)	36 (27) 2.9 (1.6)	106 (40) 2.6 (1.5)
Dactylitis,§§ n (%) LDI score,‖‖ mean (SD)	33 (12) 47.3 (41.1)	56 (13) 46.7 (54.3)	11 (8) 49.7 (31.9)	14 (11) 66.4 (127.6)	34 (13) 72.7 (114.4)

Randomized set.[1,2] *In patients with ≥3% BSA with PSO at baseline.[1,2] †Data missing for 1 BKZ patient in BE OPTIMAL.[1] ‡BE OPTIMAL: data missing for 1 BKZ patient and 1 ADA patient.[1] §At-risk subgroup defined as patients with elevated hs-CRP (≥6 mg/L) and/or ≥1 bone erosion at baseline; placebo n=227, BKZ n=361, ADA n=112; data not collected in BE COMPLETE.[1] ‖Radiographic set (patients with valid radiographs of hands and feet at baseline, as assessed by ≥2 reviewers); placebo n=269, BKZ n=420, ADA n=135; data not collected in BE COMPLETE.[1] ¶mNAPSI score >0, data missing for 7 BKZ patients in BE OPTIMAL and 1 placebo patient in BE COMPLETE.[1,2] **In patients with nail psoriasis at baseline (BE COMPLETE; placebo n=83, BKZ n=159).[2] ††Leeds Enthesitis Index >0; BE OPTIMAL: data missing for 6 BKZ patients and 1 ADA patient.[1,2] ‡‡In patients with enthesitis at baseline (BE COMPLETE: placebo n=36, BKZ n=106).[1,2] §§Leeds Dactylitis Index >0; BE OPTIMAL: data missing for 1 placebo patient, 7 BKZ patients and 1 ADA patient.[1,2] ‖‖In patients with dactylitis at baseline (BE COMPLETE: placebo n=14; BKZ n=34).[1,2]

1. Adapted from Ritchlin CT, et al. Ann Rheum Dis. 2023;82(11):1404–1414. 2. Adapted from Merola JF, et al. Lancet. 2023;401(10370):38–48.

US-BK-2401175 | Date of preparation: September 2024

BE OPTIMAL Met the Primary and All Ranked Secondary Endpoints at Week 16

	BE OPTIMAL			
	Placebo (n=281)	**BKZ 160 mg Q4W (n=431)**	**BKZ vs placebo p value***	**Reference Arm (ADA 40 mg Q2W; n=140)†**
ACR50 response‡	**28 (10%)**	**189 (44%)**	**<0.0001**	**64(46%)**
HAQ-DI CfB	-0.09 (0.03)	-0.26 (0.02)	<0.0001	-0.33 (0.04)
PASI90 response§	4 (3%) of 140	133 (61%) of 217	<0.0001	28 (41%) of 68
SF-36 PCS CfB	2.3 (0.5)	6.3 (0.4)	<0.0001	6.8 (0.8)
MDA response	37 (13%)	194 (45%)	<0.0001	63 (45%)
vdHmTSS CfB (at risk subgroup); number of patients, n	0.36 (0.10); 227	0.01 (0.04); 361	0.0012	-0.06 (0.08); 112
Complete resolution of enthesitis (pooled)‖	37 (35%) of 106	124 (50%) of 249	0.0083	18 (50%) of 36
Complete resolution of dactylitis (pooled)‖	24 (51%) of 47	68 (76%) of 90	0.0022	9 (82%) of 11
vdHmTSS CfB (overall); number of patients, n	0.31 (0.09); 269	0.01 (0.04); 420	0.0012	-0.03 (0.07); 135

Missing data were imputed using RBMI for continuous variables and NRI for proportions. Randomized set. Data are n (%) or mean CFB (SE) unless indicated. *For binary values, ORs, CIs, and p values were generated using logistic regression with treatment, bone erosion, and region as factors. For enthesitis and dactylitis resolution, where data were pooled from BE OPTIMAL and BE COMPLETE, the study was also included as a factor in the model, and bone erosion at baseline were excluded. For continuous variables, least squares mean, SE, difference in least squares means, and p values were generated using ANCOVA with treatment, bone erosion at baseline, and region as fixed effects, and the baseline value as covariate. †The adalimumab 40 mg every 2 weeks treatment group served as an active reference and the study was not powered for statistical comparisons of adalimumab to bimekizumab or placebo. ‡Primary endpoint. §In patients with psoriasis affecting 3% or more BSA at baseline. ‖Resolution of enthesitis and dactylitis data are reported for patients with enthesitis or dactylitis at baseline. Data for the placebo and bimekizumab groups are pooled from the BE OPTIMAL and BE COMPLETE trials; data for patients in the reference group are reported from BE OPTIMAL only.

Adapted from McInnes IB, et al. Lancet. 2023;401(10370):25–37.

US-BK-2401175 | Date of preparation: September 2024

BE COMPLETE Met the Primary and All Ranked Secondary Endpoints at Week 16

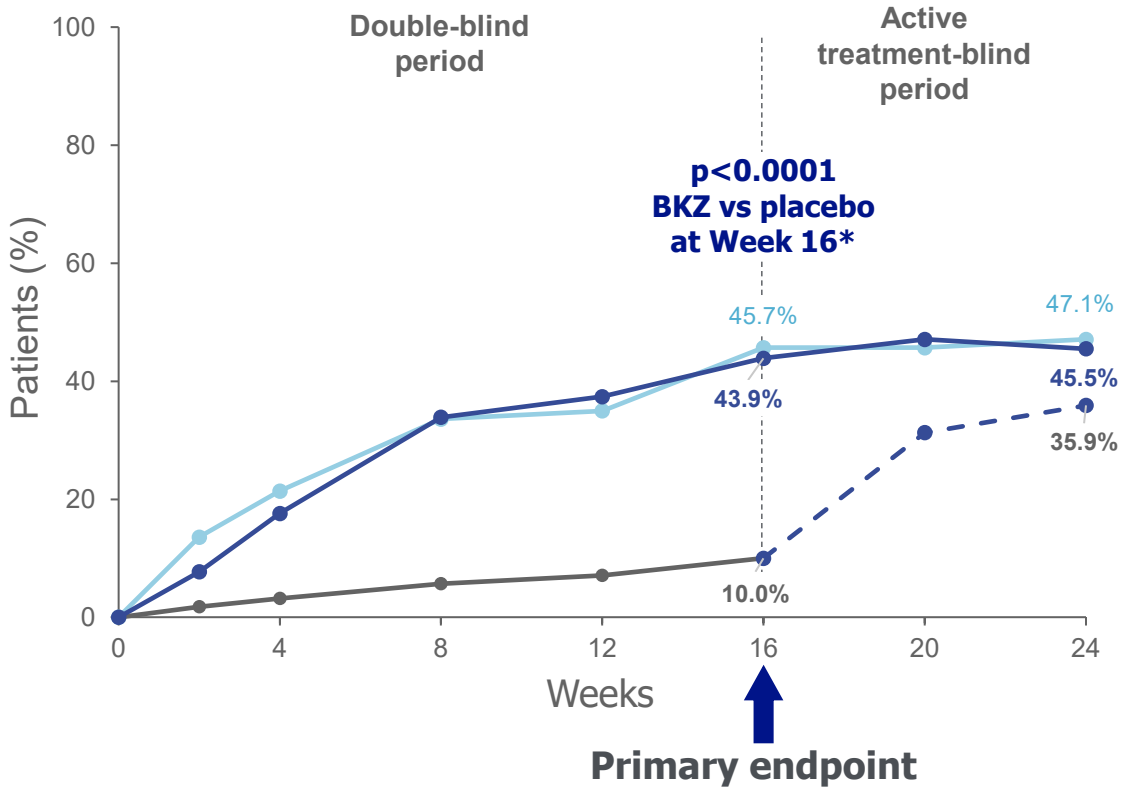
	BE COMPLETE		
	Placebo (n=281)	**BKZ 160 mg Q4W (n=431)**	**BKZ vs placebo p value***
ACR50 response†	**9 (7%)**	**116 (43%)**	**<0.0001**
HAQ-DI CfB	-0.07 (0.04)	-0.38 (0.03)	<0.0001
PASI90 response‡	6 (7%) of 88	121 (69%) of 176	<0.0001
SF-36 PCS CfB	1.4 (0.7)	7.3 (0.5)	<0.0001
MDA response	8 (6%)	118 (44%)	<0.0001

Missing data were imputed using RBMI for continuous variables and NRI for binary variables. Randomized set. Data are n (%) or mean CFB (SE) unless indicated. *For binary variables, ORs, CIs, and p values were generated using logistic regression with treatment, previous exposure to TNFα inhibitors, and region as factors. For continuous variables, least squares mean, SEs, difference in least squares means, and p values were generated using ANCOVA with treatment, previous exposure to TNFα inhibitors, and region as fixed effects and the baseline value of the outcome as covariate. †Primary endpoint. ‡In patients with psoriasis affecting 3% or more BSA at baseline.
Adapted from Merola JF, et al. Lancet. 2023;401(10370):38–48.

US-BK-2401175 | Date of preparation: September 2024

ACR50 Response with BKZ to Week 16 or Week 24 in bDMARD Naïve and TNFi-IR Patients (NRI)

BE OPTIMAL (bDMARD-naïve patients)[1]

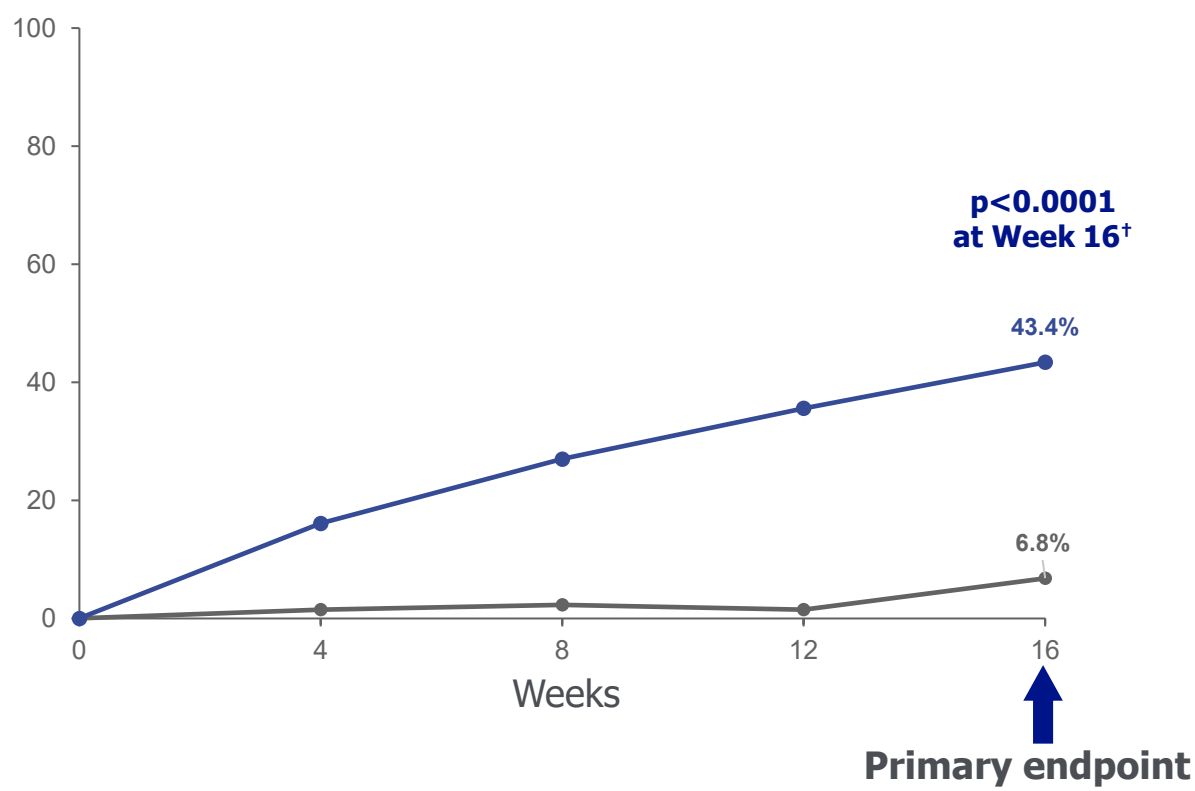


PBO/BKZ 160 mg Q4W (n=281)

BKZ 160 mg Q4W (n=431)

Reference arm; ADA 40 mg Q2W (n=140)

BE COMPLETE (TNFi-IR patients)[2]



PBO (n=133)

BKZ 160 mg Q4W (n=267)

US-BK-2401175 | Date of preparation: September 2024

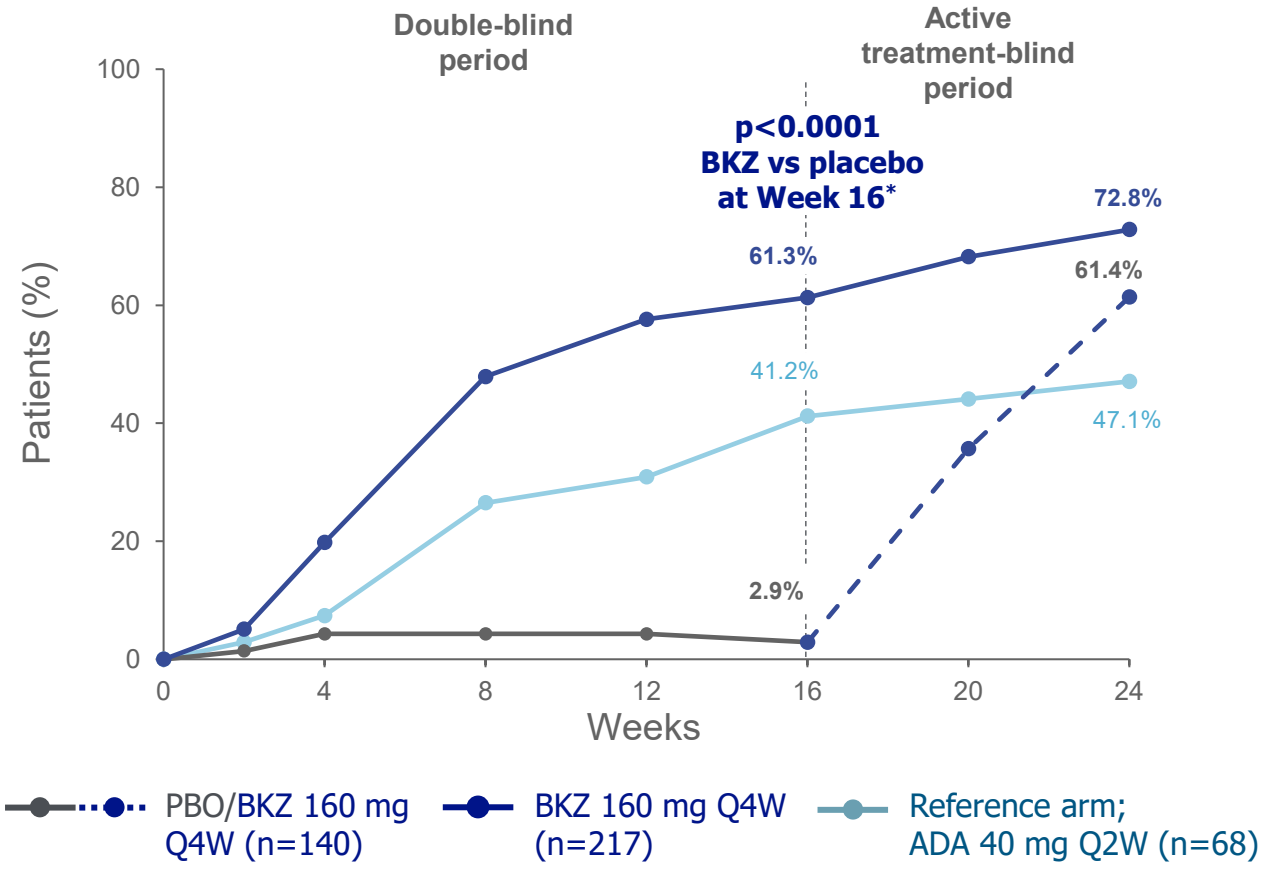
Inspired by patients. Driven by science.

Non-responder imputation.[1,2] **p values were only calculated for the primary endpoints (Week 16).**[1,2] Randomized set.[1,2] *p value was calculated using a logistic regression with treatment, bone erosion at baseline, and region as factors. The study was not powered for statistical comparisons of ADA to BKZ or ADA to placebo.[1] †p value obtained from logistic regression with treatment, prior TNFi exposure and region as factors.[2]

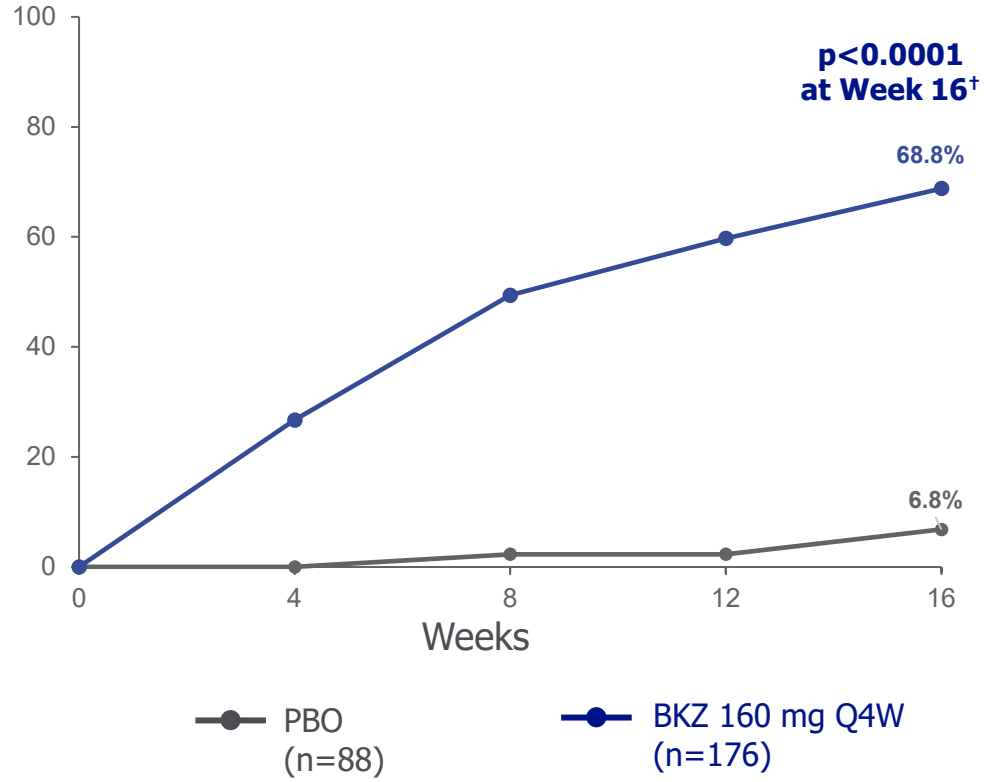
1. Adapted from McInnes IB, et al. Lancet. 2023;401(10370):25–37. 2. Adapted from Merola JF, et al. Lancet. 2023;401(10370):38–48.

PASI90 Response with BKZ to Week 16 or Week 24 in bDMARD Naïve and TNFi-IR Patients (NRI)

BE OPTIMAL (bDMARD-naïve patients)[1]



BE COMPLETE (TNFi-IR patients)[2]



US-BK-2401175 | Date of preparation: September 2024

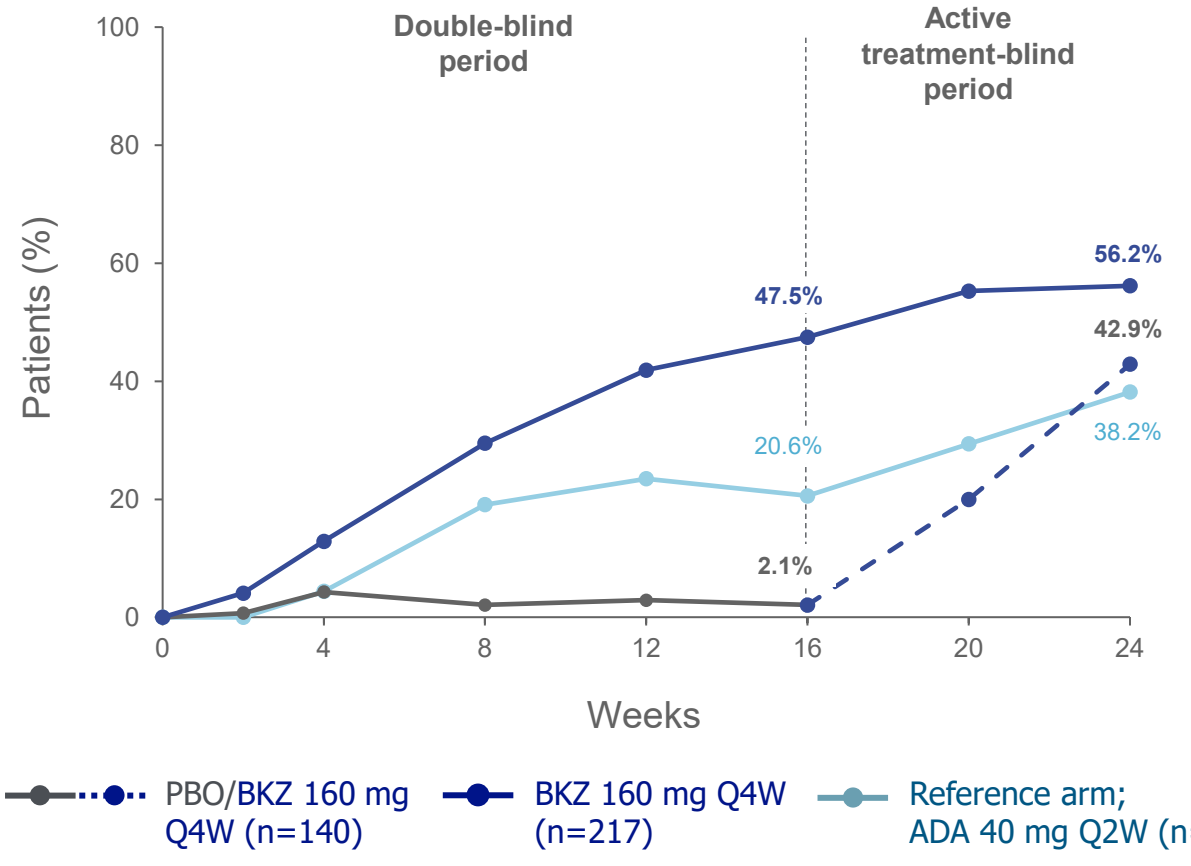
Inspired by patients. Driven by science.

Non-responder imputation.[1,2] **p values were only calculated for the ranked secondary endpoints (Week 16).**[1,2] Randomized set, in patients with PSO involving ≥3% BSA at baseline.[1,2] *p value was calculated using a logistic regression with treatment, bone erosion at baseline, and region as factors. The study was not powered for statistical comparisons of ADA to BKZ or ADA to placebo.[1] †p value obtained from logistic regression with treatment, prior TNFi exposure and region as factors.[2]

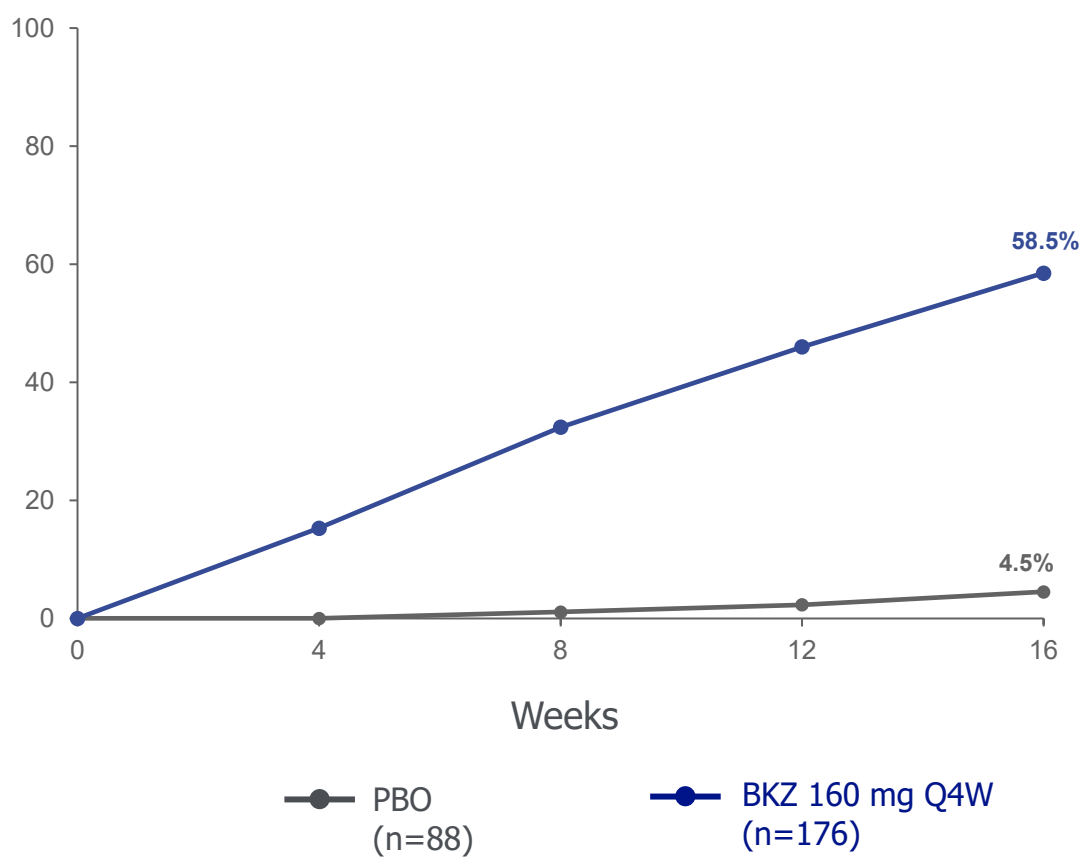
1. Adapted from McInnes IB, et al. Lancet. 2023;401(10370):25–37. 2. Adapted from Merola JF, et al. Lancet. 2023;401(10370):38–48.

PASI100 Response with BKZ to Week 16 or Week 24 in bDMARD Naïve and TNFi-IR Patients (NRI)

BE OPTIMAL (bDMARD-naïve patients)[1]



BE COMPLETE (TNFi-IR patients)[2]



US-BK-2401175 | Date of preparation: September 2024

Inspired by patients. Driven by science.

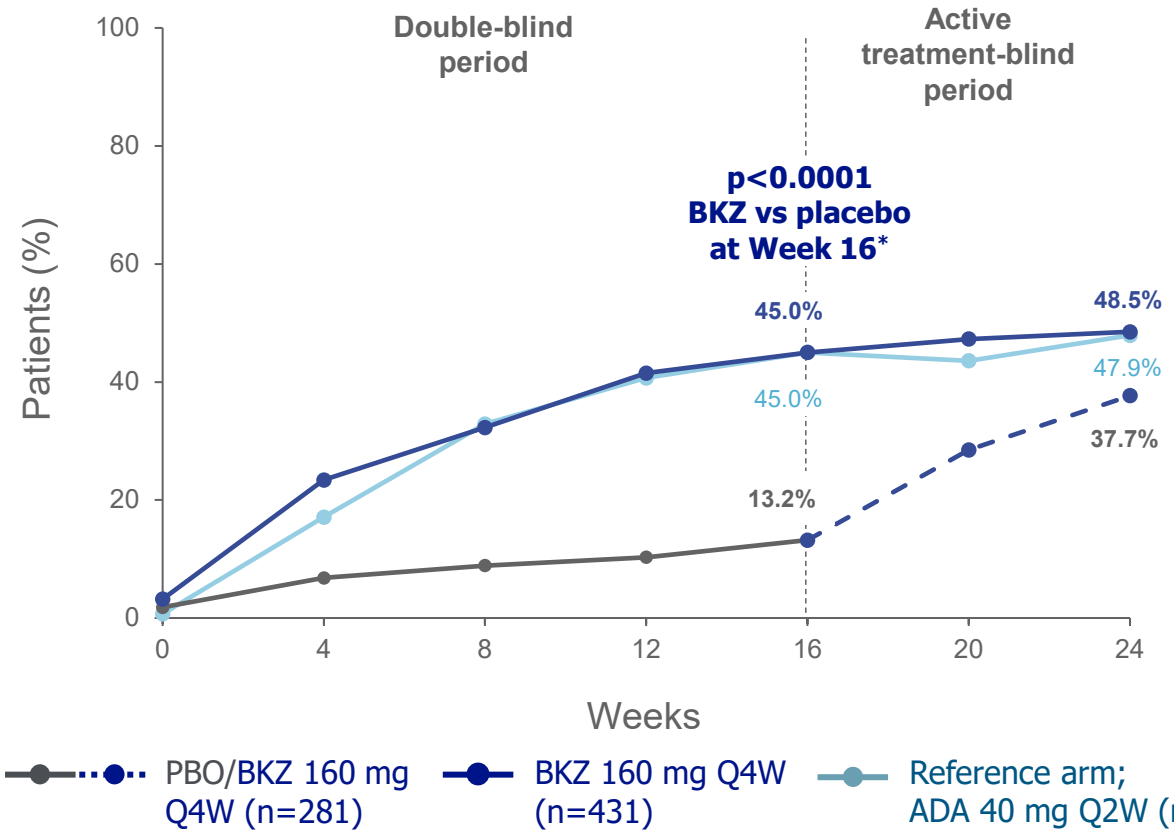
Non-responder imputation.[1,2] Randomized set, in patients with PSO involving ≥3% BSA at baseline.[1,2]

1. Adapted from McInnes IB, et al. Lancet. 2023;401(10370):25–37. 2. Adapted from Merola JF, et al. Lancet. 2023;401(10370):38–48.

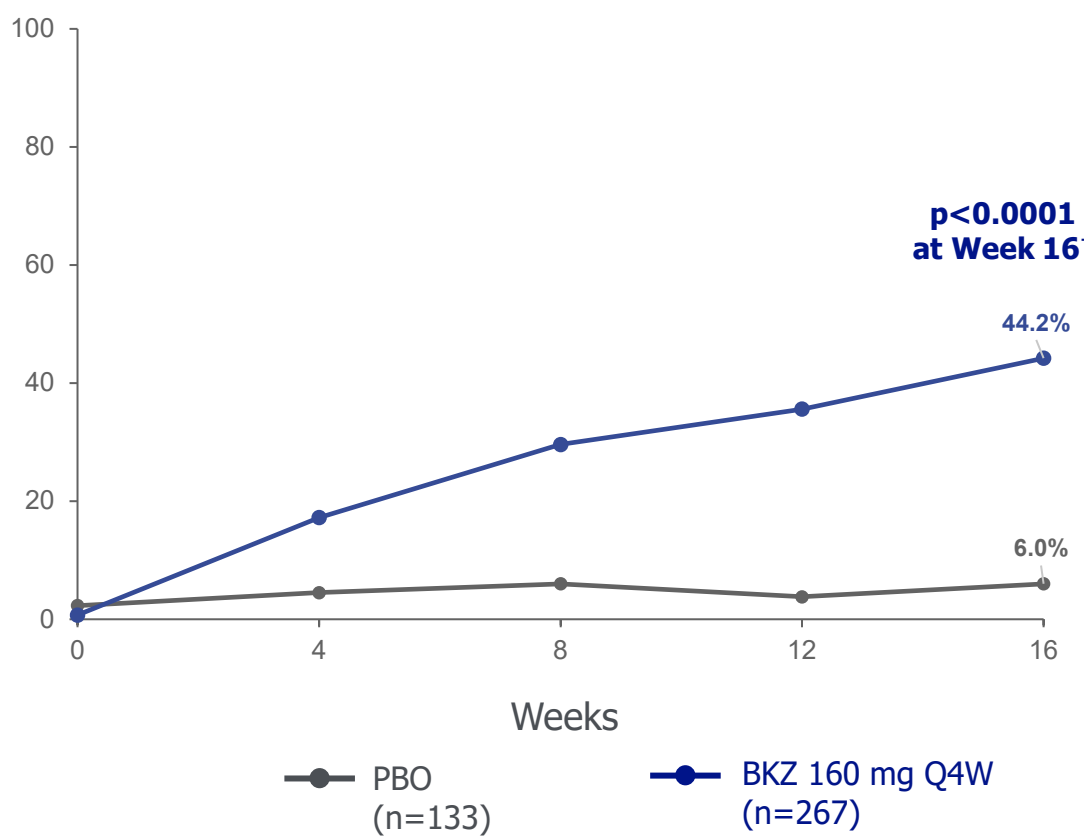
Minimal Disease Activity (MDA) Response with BKZ to Week 16 or Week 24 in bDMARD Naïve and TNFi-IR Patients (NRI)

MDA response defined as achievement of at least 5 of the 7 following criteria: TJC ≤1, SJC ≤1, PASI ≤1 or BSA ≤3%, Patient's assessment of arthritis pain (VAS) ≤15, patient global assessment for PsA (VAS) ≤20, HAQ-DI ≤0.5, and tender entheseal points (LEI) ≤1[1,2]

BE OPTIMAL (bDMARD-naïve patients)[1]



BE COMPLETE (TNFi-IR patients)[2]



US-BK-2401175 | Date of preparation: September 2024

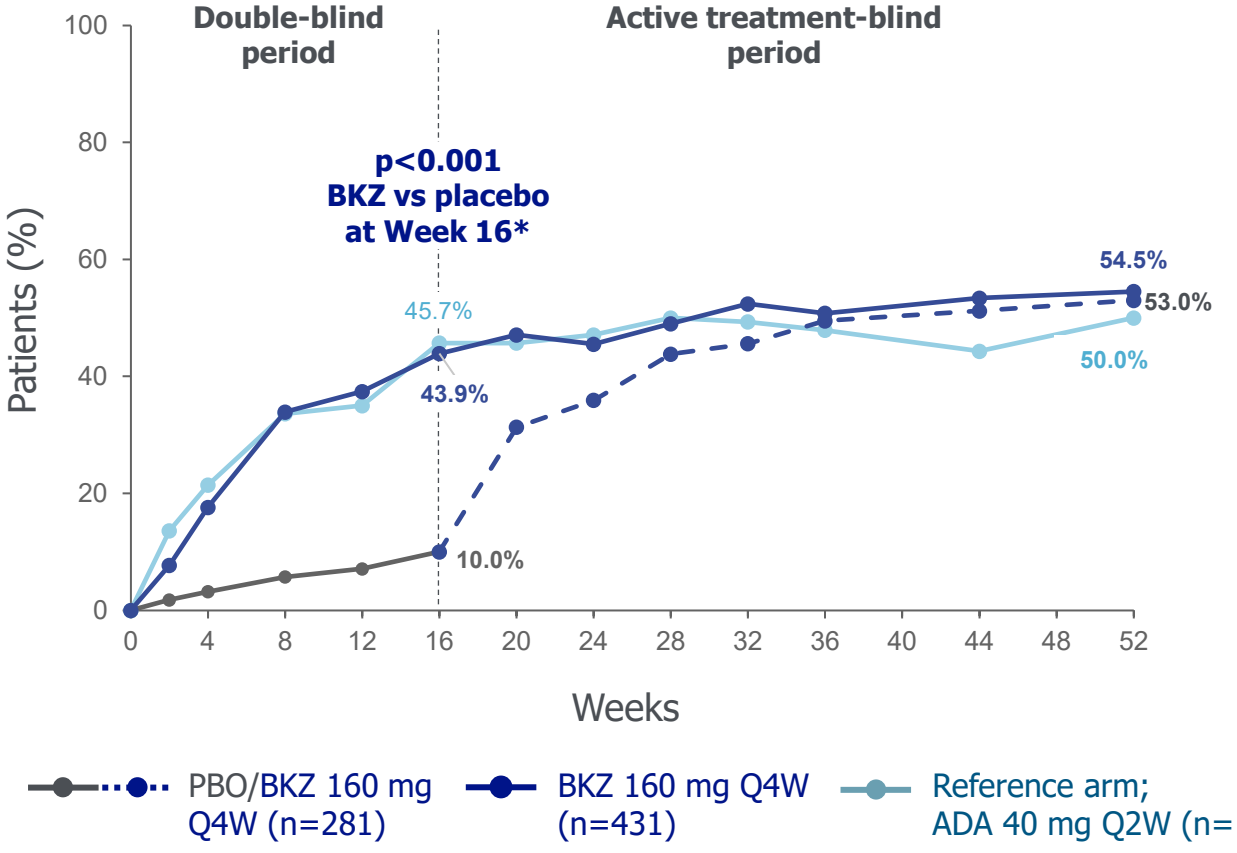
Inspired by patients. Driven by science.

Non-responder imputation.[1,2] p values were only calculated for the ranked secondary endpoints (Week 16).[1,2] Randomized set.[1,2] *p value was calculated using a logistic regression model with treatment, bone erosion at baseline, and region as factors. The study was not powered for statistical comparisons of ADA to BKZ or ADA to placebo.[1] †p value obtained from logistic regression with treatment, prior TNFi exposure and region as factors.[2]

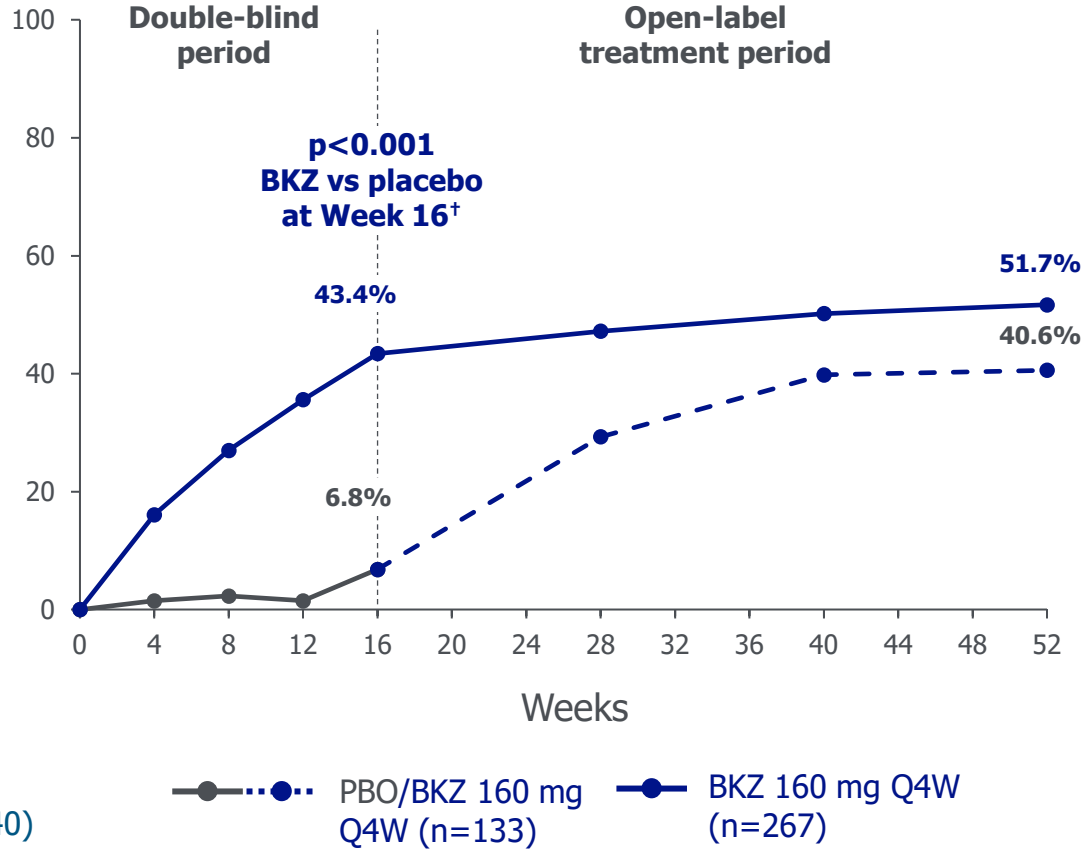
1. Adapted from McInnes IB, et al. Lancet. 2023;401(10370):25–37. 2. Adapted from Merola JF, et al. Lancet. 2023;401(10370):38–48.

ACR50 Responses with BKZ to Week 52 (NRI)

BE OPTIMAL (bDMARD-naïve patients)[1]



BE COMPLETE + OLE (TNFi-IR patients)[2]



US-BK-2401175 | Date of preparation: September 2024

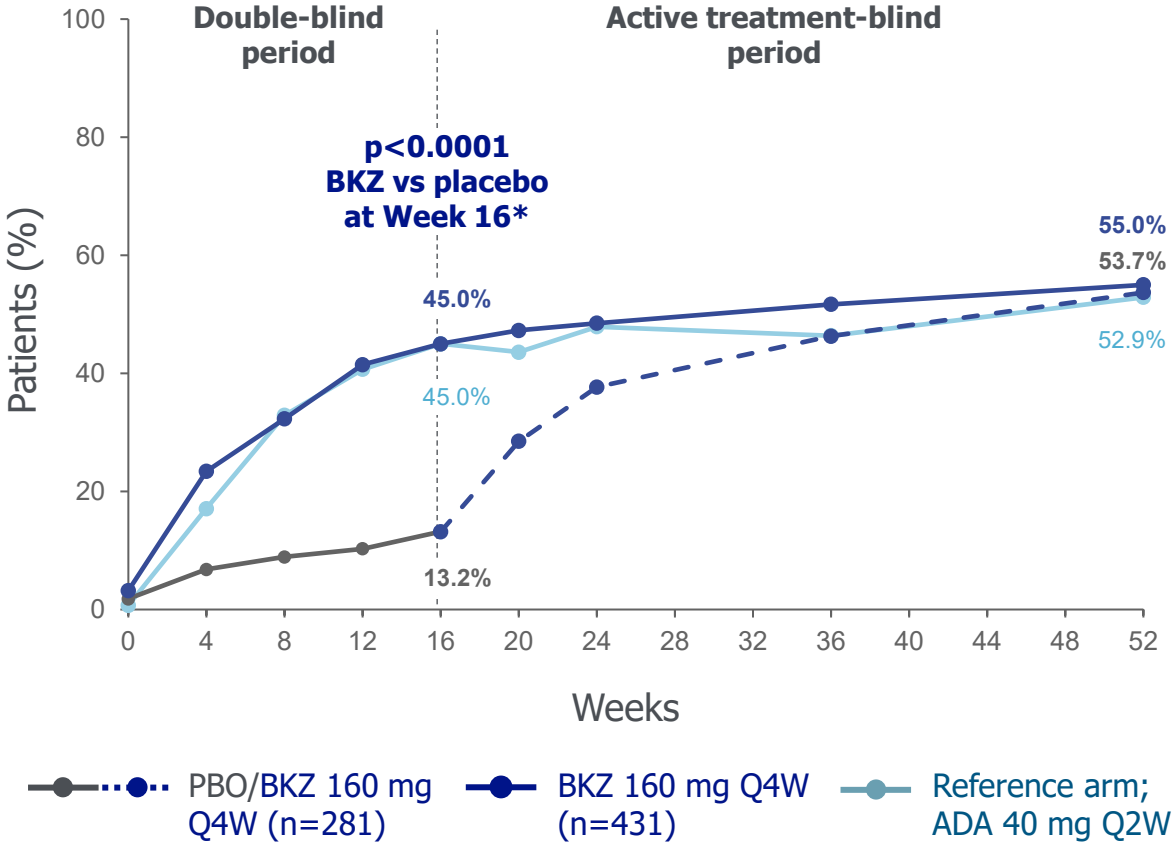
Non-responder imputation.[1,2] **p values were only calculated for the primary endpoints (Week 16).**[1-4] Randomized set.[1,2] *p value was calculated using a logistic regression model with treatment, bone erosion at baseline, and region as factors. The study was not powered for statistical comparisons of ADA to BKZ or ADA to placebo.[1] †p value obtained from logistic regression with treatment, prior TNFi exposure and region as factors.[2]

1. Adapted from Ritchlin CT, et al. 2023;82(1):1404-1414. 2. Adapted from Coates LC, et al. RMD Open. 2024;1(0):e003855. 3. McInnes IB, et al. Lancet. 2023;401(10370):25–37. 4. Merola JF, et al. Lancet. 2023;401(10370):38–48.

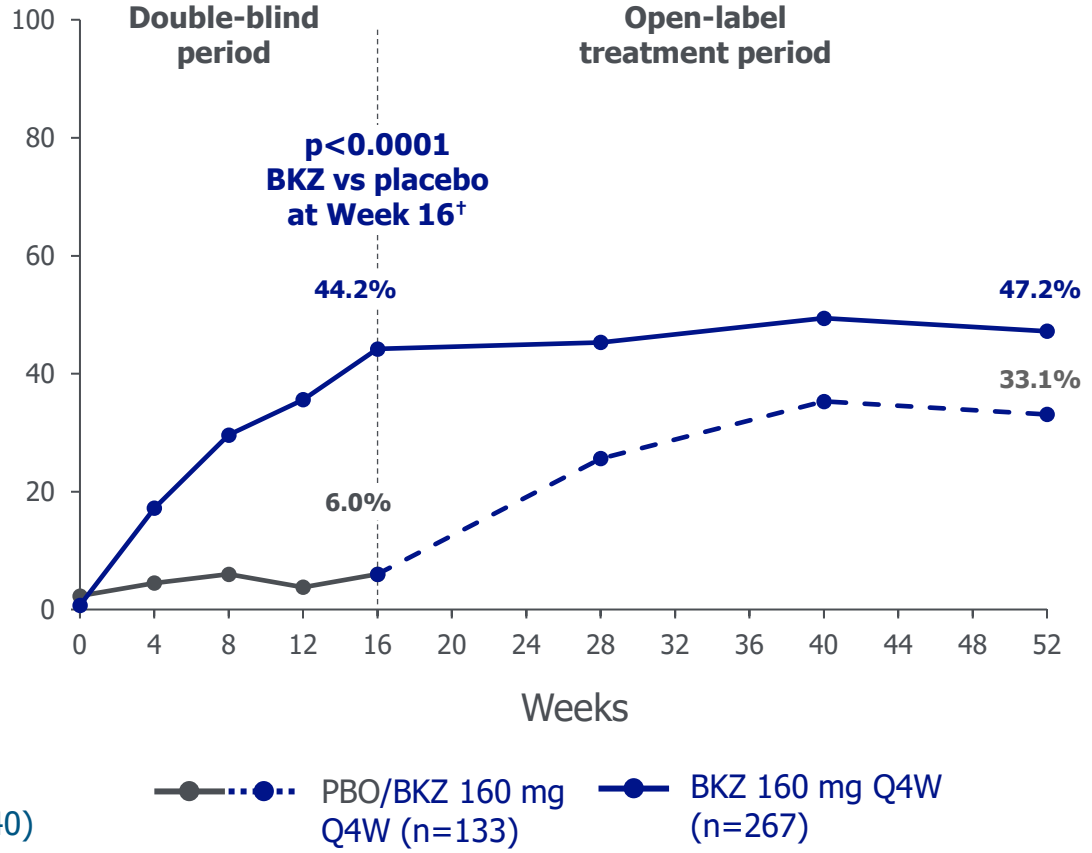
Minimal Disease Activity (MDA) Response with BKZ to Week 52 (NRI)

MDA response defined as achievement of at least 5 of the 7 following criteria:
TJC ≤1, SJC ≤1, PASI ≤1 or BSA ≤3%, Patient's assessment of arthritis pain (VAS) ≤15, patient global assessment for PsA (VAS) ≤20, HAQ-DI ≤0.5, and tender entheseal points (LEI) ≤1[1,2]

BE OPTIMAL (bDMARD-naïve patients)[1]



BE COMPLETE + OLE (TNFi-IR patients)[2]



US-BK-2401175 | Date of preparation: September 2024

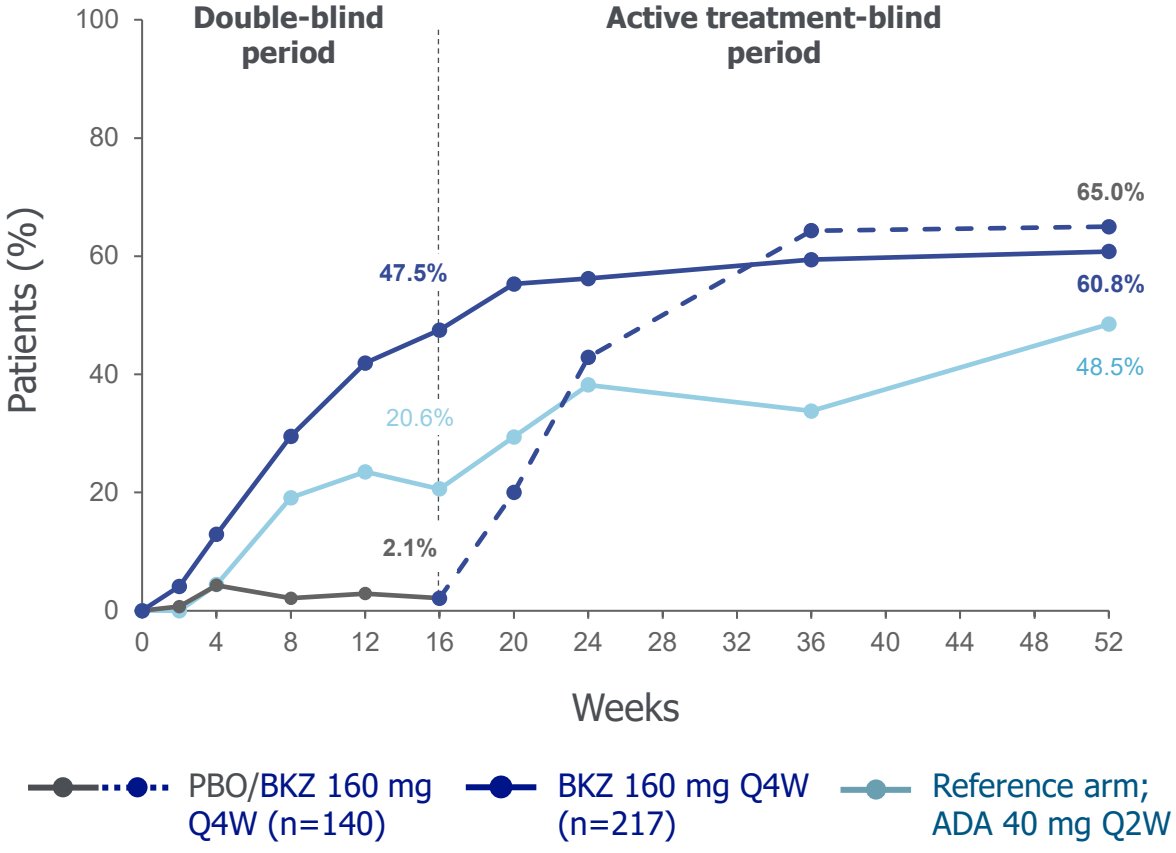
Inspired by patients. Driven by science.

Non-responder imputation.[1,2] **p values were only calculated for the ranked secondary endpoints (Week 16).**[1-4] Randomized set.[1,2] *p value was calculated using a logistic regression model with treatment, bone erosion at baseline, and region as factors. The study was not powered for statistical comparisons of ADA to BKZ or ADA to placebo.[1] †p value obtained from logistic regression with treatment, prior TNFi exposure and region as factors.[2]

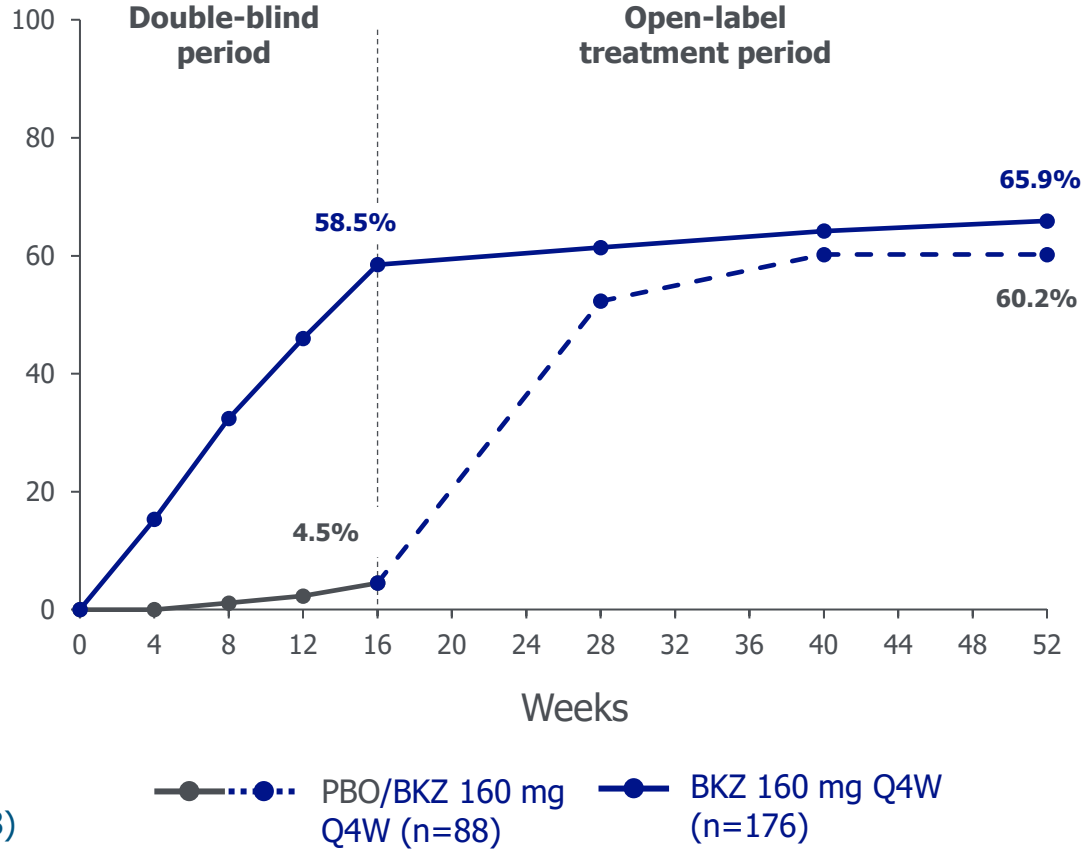
1. Adapted from Ritchlin CT, et al. 2023;82(1):1404-1414. 2. Adapted from Coates LC, et al. RMD Open. 2024;1(0):e003855.

PASI100 Responses with BKZ to Week 52 (NRI)

BE OPTIMAL (bDMARD-naïve patients)[1]



BE COMPLETE + OLE (TNFi-IR patients)[2]



US-BK-2401175 | Date of preparation: September 2024

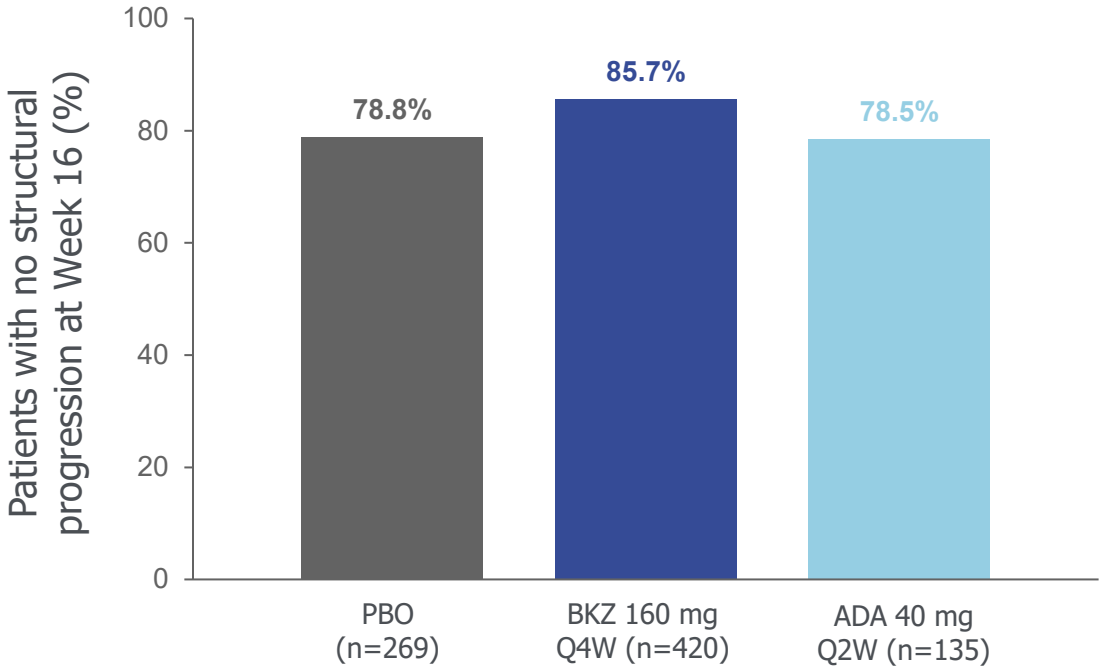
Inspired by patients. Driven by science.

Non-responder imputation.[1,2] Randomized set, in patients with psoriasis involving ≥3% of BSA at baseline.[1,2]

1. Adapted from Ritchlin CT, et al. 2023;82(1):1404-1414. 2. Adapted from Coates LC, et al. RMD Open. 2024;1(0):e003855.

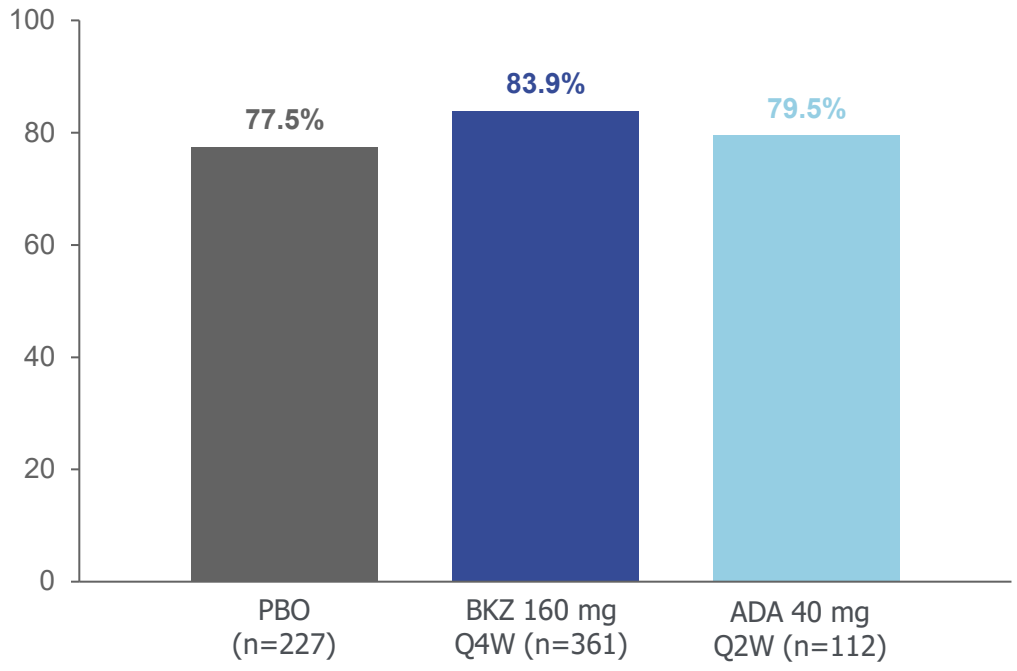
BE OPTIMAL: Inhibition of Structural Progression with BKZ at Week 16 in bDMARD-Naïve Patients (NRI)

Overall Population[1]



At-risk population[1,2,*]

Elevated hs-CRP (≥6 mg/L) and/or ≥1 bone erosion at baseline



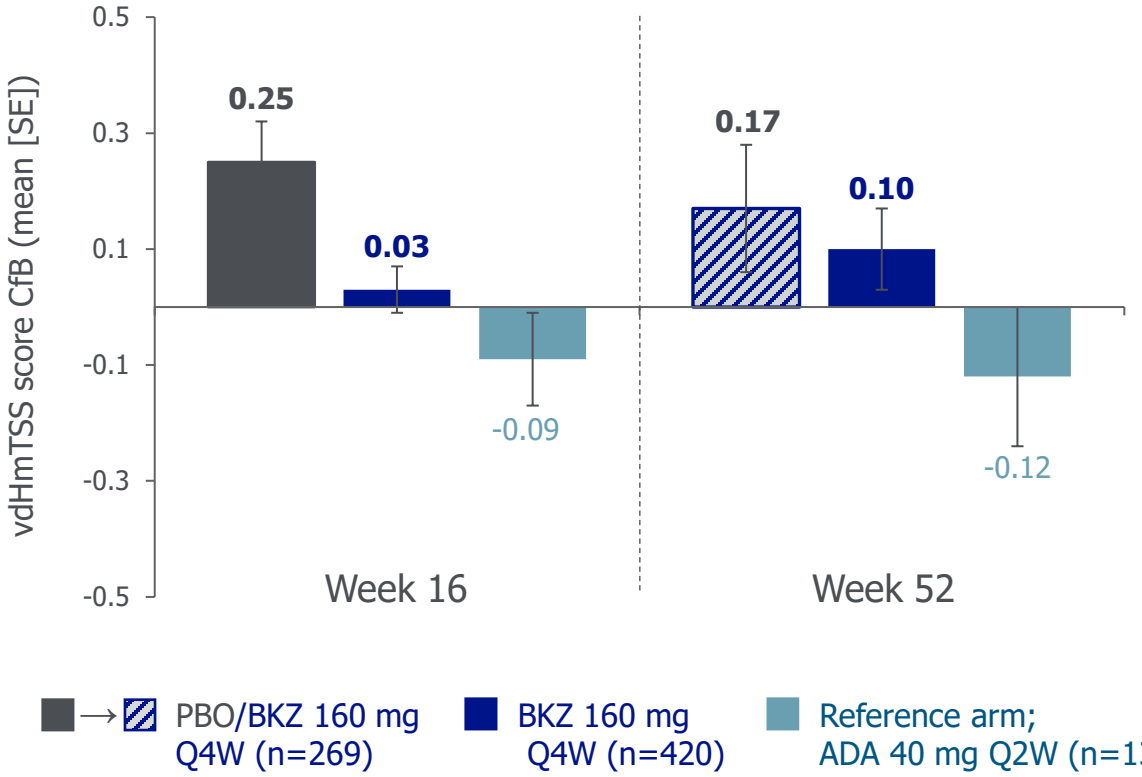
89.3% of patients treated with BKZ showed no radiographic progression at Week 52[3,4,†]

Non-responder imputation (Week 16); observed case (Week 52).[1,3,4] Radiographic set.[2,4] No structural progression defined as vdHmTSS score CfB ≤0.5.[1,3] This analysis was not performed for the BE COMPLETE study.[5] *Patients were stratified by bone erosions number at baseline (0 or ≥1) and region (North America, western Europe, eastern Europe, or Asia).[2] †BKZ: n=365 at Week 52.[3]

1. Adapted from McInnes IB, et al. Supplementary appendix. Lancet. 2023;401(10370):25–37. 2. McInnes IB, et al. Lancet. 2023;401(10370):25–37. 3. Ritchlin CT, et al. Supplementary appendix. Ann Rheum Dis. 2023;82(11):1404–1414. 4. Ritchlin CT, et al. Ann Rheum Dis. 2023;82(11):1404–1414. 5. Merola JF, et al. Lancet. 2023;401(10370):38–48.

BE OPTIMAL: Inhibition of Structural Progression with BKZ at Week 16 and Week 52 in bDMARD-Naïve Patients (MI)

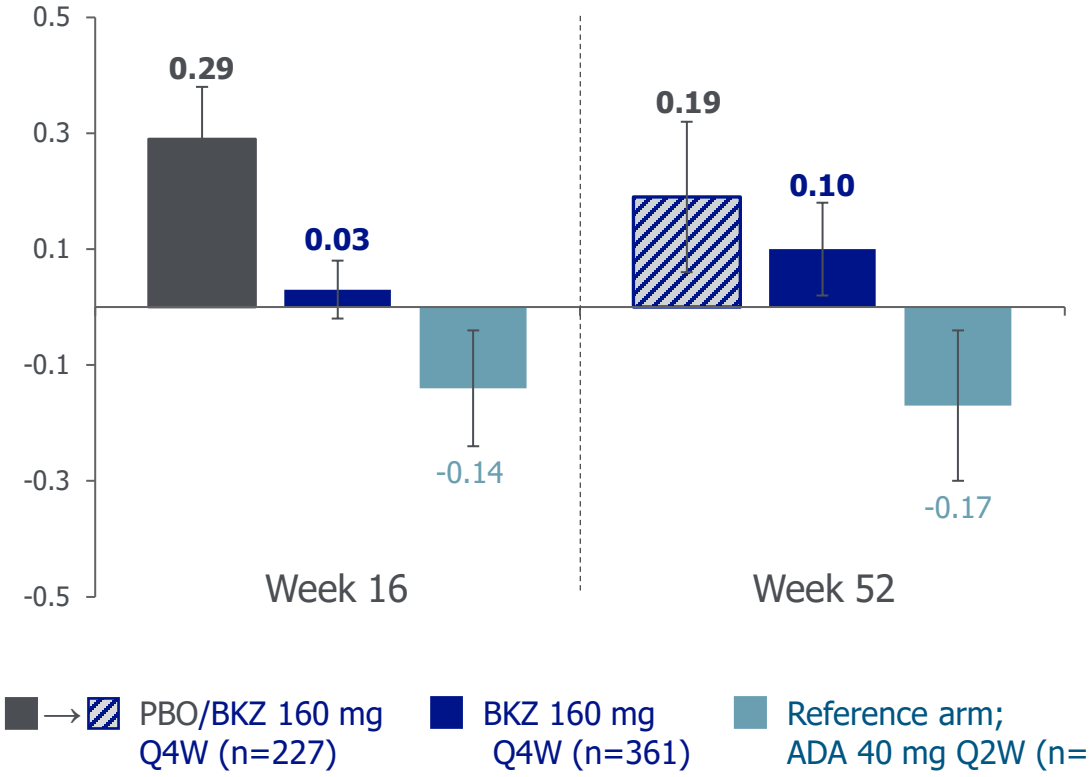
Overall Population[1]



PBO/BKZ 160 mg Q4W (n=269)
BKZ 160 mg Q4W (n=420)
Reference arm; ADA 40 mg Q2W (n=135)

At-risk population[1]

Elevated hs-CRP (≥6 mg/L) and/or ≥1 bone erosion at baseline



PBO/BKZ 160 mg Q4W (n=227)
BKZ 160 mg Q4W (n=361)
Reference arm; ADA 40 mg Q2W (n=112)

This data represents radiographs from Week 16 that were re-read at Week 52.[2] The data from the initial readings at Week 16 are presented here.

US-BK-2401175 | Date of preparation: September 2024

Multiple imputation.[1] Radiographic set.[1] This analysis was not performed for the BE COMPLETE study.[3]

1. Ritchlin CT, et al. Ann Rheum Dis. 2023;82(11):1404–1414. 2. McInnes IB, et al. Supplementary appendix. 2023;401(10370):25–37. 3. Merola JF, et al. Lancet. 2023;401(10370):38–48.

BE OPTIMAL: Adverse Event Summary to Week 16 and Week 52 (1/2)

n (%) [EAIR/100 PY]	Weeks 0–16			Weeks 0–52	
	Placebo n=281	BKZ 160 mg Q4W n=431	Reference Arm (ADA 40 mg Q2W) n=140	BKZ 160 mg Q4W Total n=702*	Reference Arm (ADA 40 mg Q2W) n=140
Any TEAE	139 (49.5)	258 (59.6)	83 (59.3)	555 (79.1) [222.5]	113 (80.7) [209.4]
Serious TEAEs	3 (1.1)	8 (1.9)	2 (1.4)	46 (6.6) [7.9]	10 (7.1) [7.5]
Discontinuation due to TEAEs	3 (1.1)	8 (1.9)	3 (2.1)	21 (3.0) [3.5]	7 (5.0) [5.2]
Drug-related TEAEs	35 (12.5)	100 (23.2)	34 (24.3)	224 (31.9)	54 (38.6)
Severe TEAEs	0	4 (0.9)	3 (2.1)	23 (3.3)	9 (6.4)
Deaths	0	0	0	1 (0.1)†	0

Safety set (Week 16) and active medication set for the overall study period (Week 52). For Weeks 0–52, only TEAEs occurring whilst receiving BKZ are reported; for patients initially randomized to PBO, only events after switching to BKZ at Week 16 are included. A safety follow-up was conducted 20 weeks after the last dose of BKZ for those not entering the OLE, or who discontinued early. *Includes patients who switched from placebo to BKZ (events after switch only). †Motorcycle accident. n: number of patients reporting at least one TEAE in that category.

Adapted from Ritchlin CT, et al. Ann Rheum Dis. 2023;82(11):1404–1414.

BE OPTIMAL: Adverse Event Summary to Week 16 and Week 52 (2/2)

n (%) [EAIR/100 PY]	Weeks 0–16			Weeks 0–52	
	Placebo n=281	BKZ 160 mg Q4W n=431	Reference Arm (ADA 40 mg Q2W) n=140	BKZ 160 mg Q4W n=702*	Reference Arm (ADA 40 mg Q2W) n=140
Most frequently reported TEAEs (≥5% in any study group at the Week 16 or Week 52 data cut-off)					
Nasopharyngitis	13 (4.6)	40 (9.3)	7 (5.0)	84 (12.0) [15.2]	12 (8.6) [9.4]
Upper respiratory tract infection	18 (6.4)	22 (5.1)	3 (2.1)	50 (7.1) [8.7]	8 (5.7) [6.1]
Headache	7 (2.5)	19 (4.4)	2 (1.4)	41 (5.8) [7.1]	6 (4.3) [4.5]
Diarrhea	7 (2.5)	16 (3.7)	5 (3.6)	36 (5.1) [6.2]	7 (5.0) [5.3]
Oral candidiasis†	0	9 (2.1)	0	38 (5.4) [6.5]	1 (0.7) [0.7]
Hypertension	11 (3.9)	12 (2.8)	4 (2.9)	29 (4.1) [4.9]	9 (6.4) [6.8]
Urinary tract infection	4 (1.4)	9 (2.1)	3 (2.1)	43 (6.1) [7.3]	5 (3.6) [3.7]
Increased ALT	2 (0.7)	3 (0.7)	7 (5.0)	16 (2.3) [2.7]	11 (7.9) [8.5]
Increased AST	2 (0.7)	1 (0.2)	4 (2.9)	14 (2.0) [2.3]	7 (5.0) [5.3]
Injection site erythema	0	1 (0.2)	4 (2.9)	6 (0.9) [1.0]	7 (5.0) [5.3]

Safety set (Week 16) and active medication set for the overall study period (Week 52). For Weeks 0–52, only TEAEs occurring whilst receiving BKZ are reported; for patients initially randomized to PBO, only events after switching to BKZ at Week 16 are included. A safety follow-up was conducted 20 weeks after the last dose of BKZ for those not entering the OLE, or who discontinued early. *Includes patients who switched from placebo to BKZ (events after switch only). †All infections were mild-to-moderate and none were serious, 1 BKZ-treated patient discontinued. n: number of patients reporting at least one TEAE in that category.
Adapted from Ritchlin CT, et al. Ann Rheum Dis. 2023;82(11):1404–1414.

US-BK-2401175 | Date of preparation: September 2024

BE OPTIMAL: Adverse Events of Special Interest to Week 16 and Week 52 (1/2)

n (%) [EAIR/100 PY]	Weeks 0–16[1]			Weeks 0–52[1]	
	Placebo n=281	BKZ 160 mg Q4W n=431	Reference Arm (ADA 40 mg Q2W) n=140	BKZ 160 mg Q4W Total n=702*	Reference Arm (ADA 40 mg Q2W) n=140
TEAEs of Special Interest					
Uveitis	0	0	0	0	0
Adjudicated MACE	0	0	0	4 (0.6) [0.7]†	0
Neutropenia	1 (0.4)	5 (1.2)	1 (0.7)	11 (1.6) [1.8]	2 (1.4) [1.5]
Infections					
Serious	0	1 (0.2)	1 (0.7)	6 (0.9) [1.0]‡	2 (1.4) [1.5]‡
Opportunistic	0	0	1 (0.7)§	9 (1.3) [1.5]	1 (0.7) [0.7]
Active TB	0	0	0	0	0
Candida infections[2]	2 (0.7)	11 (2.6)	0	54 (7.7)	1 (0.7)
Hypersensitivity	6 (2.1)	18 (4.2)	3 (2.1)	59 (8.4) [10.3]	7 (5.0) [5.3]
Injection-site reactions	3 (1.1)	5 (1.2)	7 (5.0)	15 (2.1) [2.5]	13 (9.3) [10.2]
Adjudicated SIB	0	0	0	0	0
Liver function test changes/enzyme elevations					
ALT >3 x ULN	0‖	5 (1.2)	2 (1.4)¶	15 (2.1)¶	7 (5.0)¶
AST or ALT >3 x ULN	0‖	5 (1.2)	3 (2.2)¶	24 (3.4)¶	9 (6.5)¶
Adjudicated IBD	0	0	0	2 (0.3) [0.3]**	0

Safety set (Week 16) and active medication set for the overall study period (Week 52).[1] For Weeks 0–52, only TEAEs occurring whilst receiving BKZ are reported; for patients initially randomized to PBO, only events after switching to BKZ at Week 16 are included.[1] A safety follow-up was conducted 20 weeks after the last dose of BKZ for those not entering the OLE, or who discontinued early.[1] *Includes patients who switched from placebo to BKZ (events after switch only).[1] †1 myocardial infarction; 1 cerebrovascular accident; 1 ischemic stroke; 1 thrombotic cerebral infarction.[1] ‡6 serious infections were reported on the BKZ treatment arm: 1 cellulitis, 1 gangrene, 1 pneumonia, 1 upper respiratory tract infection, 1 cystitis and 1 urinary tract infection; 2 ADA-treated patients reported serious infections: 1 otitis media and 1 reported both herpes zoster and atypical pneumonia.[1] §1 opportunistic infection event of herpes zoster was reported for the ADA treatment arm during the double-blind period.[1] ‖Data missing for 2 patients.[1] ¶Data missing for 1 patient.[1] **Both ulcerative colitis; 1 in a patient with a prior history of IBD, the other de novo. n: number of patients reporting at least one TEAE in that category.[1]

1. Adapted from Ritchlin CT, et al. Ann Rheum Dis. 2023;82(11):1404–1414. 2. Adapted from Ritchlin CT, et al. Supplementary appendix. Ann Rheum Dis. 2023;82(11):1404-1411.

US-BK-2401175 | Date of preparation: September 2024

BE OPTIMAL: Adverse Events of Special Interest to Week 16 and Week 52 (2/2)

n (%) [EAIR/100 PY]	Weeks 0–16			Weeks 0–52	
	Placebo n=281	BKZ 160 mg Q4W n=431	Reference Arm (ADA 40 mg Q2W) n=140	BKZ 160 mg Q4W Total n=702*	Reference Arm (ADA 40 mg Q2W) n=140
TEAEs of Special Interest					
Malignancies excluding nonmelanoma skin cancer					
Breast cancer stage 1	1 (0.4)	0	0	0	0
Colon cancer	0	0	0	1 (0.1) [0.2]	0
Chronic lymphocytic leukemia stage 0	0	0	0	1 (0.1) [0.2]	0
Papillary thyroid cancer	0	0	0	1 (0.1) [0.2]	0
Nonmelanoma skin cancer					
Squamous cell carcinoma	0	0	0	1 (0.1) [0.2]	0
Basal cell carcinoma	0	1 (0.2)	0	3 (0.4) [0.5]	0

Safety set (Week 16) and active medication set for the overall study period (Week 52). For Weeks 0–52, only TEAEs occurring whilst receiving BKZ are reported; for patients initially randomized to PBO, only events after switching to BKZ at Week 16 are included. A safety follow-up was conducted 20 weeks after the last dose of BKZ for those not entering the OLE, or who discontinued early. *Includes patients who switched from placebo to BKZ (events after switch only). n: number of patients reporting at least one TEAE in that category.
Adapted from Ritchlin CT, et al. Ann Rheum Dis. 2023;82(11):1404–1414.

US-BK-2401175 | Date of preparation: September 2024

BE COMPLETE: Adverse Event Summary to Week 16 and Week 52

n (%) [EAIR/100 PY]	Weeks 0–16[1]		Weeks 0–52[1]	
	Placebo N=132*	BKZ 160 mg Q4W n=267	BKZ 160 mg Q4W n=267	BKZ 160 mg Q4W Total* n=388
Any TEAE	44 (33.3)	108 (40.4)	175 (65.5) [125.4]	243 (62.6) [126.0]
Serious TEAEs	0	5 (1.9)†	15 (5.6) [6.0]	23 (5.9) [7.0]
Discontinuation due to TEAEs	0	2 (0.7)‡	10 (3.7) [3.9]	16 (4.1) [4.8]
Drug-related TEAEs	4 (3.0)	35 (13.1)	66 (24.7)	87 (22.4)
Severe TEAEs	0	5 (1.9)§	14 (5.2)	17 (4.4)
Deaths	0	0	0	1 (0.3)‖
Most frequently reported TEAEs (≥5% in any study group)				
Coronavirus infection	6 (4.5)	5 (1.9)	21 (7.9) [8.4]	28 (7.2) [8.5]
Oral candidiasis	0	7 (2.6)	17 (6.4) [6.8]	24 (6.2) [7.3]
Nasopharyngitis	1 (0.8)	10 (3.7)	19 (7.1) [7.7]	23 (5.9) [7.0]
Urinary tract infection	3 (2.3)	5 (1.9)	19 (7.1) [7.7]	23 (5.9) [7.0]

Safety set.[1] EAIRs are reported for Weeks 0-52 where available.[1] No cases of active tuberculosis, definite or probable adjudicated IBD, suicidal ideation and behavior, or uveitis were reported.[1] A safety follow-up was conducted 20 weeks after the last dose of BKZ for those not entering the OLE, or who discontinued early. One patient included in the randomized set was not counted in the safety set.[1] *Includes patients who switched from PBO to BKZ and only includes TEAEs occurring whilst receiving BKZ.[1] †1 intestinal obstruction, 1 bronchitis, 1 COVID-pneumonia, 1 joint injury, 1 toxic encephalopathy.[2] ‡1 stomatitis and 1 oral candidiasis.[2] §6 events in 5 patients: 1 bronchitis, 1 back pain, 1 toxic encephalopathy, 1 headache, 1 pruritus, 1 renal pain; 1 patient reported both severe back pain and renal pain.[2] ‖Sudden death in 54-year old patient with a history of hypertension, aortic regurgitation and electrocardiogram changes of coronary artery disease; no further information available; no autopsy was performed.[1]

1. Adapted from Coates LC, et al. RMD Open. 2024;1(0):e003855. 2. Adapted from Merola JF, et al. Lancet. 2023;401(10370):38–48

BE COMPLETE: Adverse Events of Special Interest to Week 16 and Week 52

n (%) [EAIR/100 PY]	Weeks 0–16[1]		Weeks 0–52[1]	
	Placebo N=132*	BKZ 160 mg Q4W n=267	BKZ 160 mg Q4W n=267	BKZ 160 mg Q4W Total* n=388
Serious infections	0	2 (0.7)†	4 (1.5) [1.6]	7 (1.8) [2.1]
Opportunistic infections	0	0	0	2 (0.5) [0.6]‡
Neutropenia	0	4 (1.5)§	5 (1.9) [2.0]‖	5 (1.3) [1.5]‖
Hypersensitivity	1 (0.8)	7 (2.6)	15 (5.6) [6.0]	19 (4.9) [5.8]
Injection-site reactions	0	3 (1.1)	6 (2.2) [2.4]	6 (1.5) [1.8]
Adjudicated MACE	0	0	0	2 (0.5) [0.6]¶
Malignancies excluding non-melanoma skin cancer	0	0	2 (0.7) [0.8]¶	3 (0.8) [0.9]**,††
Non-melanoma skin cancer	1 (0.8)‡‡	0	0	0

Safety set.[1] EAIRs are reported for Weeks 0-52 where available.[1] No cases of active tuberculosis, definite or probable adjudicated IBD, suicidal ideation and behavior, or uveitis were reported.[1] One patient included in the randomized set was not counted in the safety set.[1] *Includes patients who switched from PBO to BKZ and only includes TEAEs occurring whilst receiving BKZ.[1] †1 bronchitis, 1 COVID-pneumonia.[2] ‡2 oesophageal candidiasis.[1] §3 neutropenia; 1 neutrophil count decreased.[1] ‖4 neutropenia; 1 neutrophil count decreased.[1] ¶1 sudden death; 1 cerebral haemorrhage.[1] **1 endometrial cancer stage I; 1 gastric cancer recurrent.[1] ††1 prostate cancer.[1] ‡‡1 basal cell carcinoma.[1]

1. Adapted from Coates LC, et al. RMD Open. 2024;1(0):e003855. 2. Adapted from Merola JF, et al. Lancet. 2023;401(10370):38–48.

Limitations (1/2)

McInnes IB, et al.
Bimekizumab in patients with psoriatic arthritis, naïve to biologic treatment: a randomized, double-blind, placebo-controlled, phase 3 trial **(BE OPTIMAL)**[1]

- The patient population in this study may not reflect patients in real-world clinical practice as there were high proportions of patients with polyarticular versus oligoarticular psoriatic arthritis, patients with severe forms of comorbidities were excluded, and there was limited diversity in the demographics and characteristics in the study population
- The absence of a formal, statistical comparison between treatments in this study prevents direct comparison

Ritchlin CT, et al.
Bimekizumab treatment in biologic DMARD-naïve patients with active psoriatic arthritis: 52-week efficacy and safety results from the phase III, randomized, placebo-controlled, active reference **BE OPTIMAL** study[2]

- The subgroups of patients with enthesitis or dactylitis at baseline was relatively small and adequate power for statistical analysis in these subgroups were not possible with BE OPTIMAL data alone
- No comparisons between treatments can be made as the study was not powered for statistical comparison

US-BK-2401175 | Date of preparation: September 2024

1. McInnes IB, et al. Lancet. 2023;401(10370):25–37. 2. Ritchlin CT, et al. Ann Rheum Dis. 2023;82(1):1404-1414.

Limitations (2/2)

Merola JF, et al.
Bimekizumab in patients with active psoriatic arthritis and previous inadequate response or intolerance to tumour necrosis factor-α inhibitors: a randomized, double-blind, placebo-controlled, phase 3 trial **(BE COMPLETE)**[1]

- The patient population in this study may not reflect patients in real-world clinical practice since patients who have multiple active comorbidities
- The absence of a formal, statistical comparison between treatments in this study prevents direct comparison

Coates LC, et al.
Bimekizumab treatment in patients with active psoriatic arthritis and prior inadequate response or intolerance to tumour necrosis factor inhibitors: 52-week safety and efficacy from the phase III **BE COMPLETE** study and its open-label extension **BE VITAL**[2]

- A lack of comparator and the potential enrichment of the patient population in the OLE
- Radiographic outcomes were not assessed after Week 16
- The patient population in this study may not reflect patients in real-world clinical practice as there is more heterogeneity in the age, skin and musculoskeletal manifestations, comorbidities, and drug survival in the clinical population

US-BK-2401175 | Date of preparation: September 2024

1. Merola JF, et al. Lancet. 2023;401(10370):38–48. 2. Coates LC, et al. RMD Open. 2024;1(0):e003855.

Bimekizumab in patients with psoriatic arthritis, naïve to biologic treatment: a randomized, double-blind, placebo-controlled, phase 3 trial (**BE OPTIMAL**)

Iain B McInnes, Akihiko Asahina, Laura C Coates, Robert Landewé, Joseph F Merola, Christopher T Ritchlin, Yoshiya Tanaka, Laure Gossec, Alice B Gottlieb, Richard B Warren, Barbara Ink, Deepak Assudani, Rajan Bajracharya, Vishvesh Shende, Jason Coarse, Philip J Mease

Author Contributions: Substantial contributions to study conception and design, analysis and interpretation of the data, drafting the article or revising it critically for important intellectual content, final approval of the version of the article to be published: IBM, AA, LCC, RL, JFM, CTR, YT, LG, ABG, RBW, BI, DA, RB, VS, JC, and PJM. IBM, BI, DA, RB, VS, and JC accessed and verified the data.

Disclosures:
IBM: Consulting fees and honoraria **UCB Pharma** and research support from **UCB Pharma.**
AA: Honoria or research grants from **UCB Pharma**.
LCC: Reports grants or research support from **UCB Pharma**, paid consultant work for **UCB Pharma**, and paid speaker work for **UCB Pharma**.
RL: Reports consultancy fees from **UCB Pharma** and research grants from **UCB Pharma**.
JFM: Reports consultant or investigator work for **UCB Pharma**.
CTR: Reports research and consultancy for **UCB Pharma**.
LG: Reports non-financial support from UCB Pharma, during the conduct of the study, grants from **UCB Pharma**; personal fees from UCB Pharma, and non-financial support from **UCB Pharma**, outside the submitted work.
ABG: Reports honoraria as an advisory board member, non-promotional speaker, or consultant for **UCB Pharma**, and research or educational grants from **UCB Pharma** (all funds go to the Icahn School of Medicine at Mount Sinai).
RBW: Reports consulting fees from **UCB Pharma** and research grants to his institution from **UCB Pharma**.
BI: Employee and stockholder of **UCB Pharma.**
DA, RB, VS, and **JC** are all employees and stockholders of **UCB Pharma**.
PJM: Research grants from **UCB Pharma**; consultancy fees and speakers' bureau fees from **UCB Pharma.**

UCB Pharma contributed to study design, participated in data collection, completed the data analysis, and participated in data interpretation. UCB Pharma also participated in writing, review, and approval of the manuscript. All authors had full access to the data, reviewed and approved the final version, and were responsible for the decision to submit for publication. A medical writing agency, employed by UCB Pharma, assisted with manuscript preparation under the authors' direction.

US-BK-2401175 | Date of preparation: September 2024

Bimekizumab treatment in biologic DMARD-naïve patients with active psoriatic arthritis: 52-week efficacy and safety results from the phase III, randomized, placebo-controlled, active reference **BE OPTIMAL** study

Christopher T Ritchlin, Laura C Coates, Iain B McInnes, Philip J Mease, Joseph F Merola, Yoshiya Tanaka, Akihiko Asahina, Laure Gossec, Alice B Gottlieb, Richard B Warren, Barbara Ink, Rajan Bajracharya, Vishvesh Shende, Jason Coarse, Robert BM Landewé

Author Contributions: Substantial contributions to study conception and design: **CTR, LCC, IBM, PJM, JFM, YT, AA, LG, ABG, RBW, BI, RB, VS, JC, RBML**; substantial contributions to analysis and interpretation of the data: **CTR, LCC, IBM, PJM, JFM, YT, AA, LG, ABG, RBW, BI, RB, VS, JC, RBML**; drafting the article or revising it critically for important intellectual content: **CTR, LCC, IBM, PJM, JFM, YT, AA, LG, ABG, RBW, BI, RB, VS, JC, RBML**; final approval of the version of the article to be published: **CTR, LCC, IBM, PJM, JFM, YT, AA, LG, ABG, RBW, BI, RB, VS, JC, RBML**; manuscript guarantor: **BI**.

Disclosures:
CTR: Consultant for **UCB Pharma**.
LCC: Has received grants/research support from **UCB Pharma**; worked as a paid consultant for **UCB Pharma**; and has been paid as a speaker for **UCB Pharma.**
IBM: Consulting fees and honoraria from **UCB Pharma**; research support from **UCB Pharma**; Associate Editor at Annals of the Rheumatic Diseases.
PJM: Research grants from **UCB Pharma**; consultancy fees from **UCB Pharma**; speakers' bureau from **UCB Pharma.**
JFM: Consultant and/or investigator for **UCB Pharma**.
YT: Editorial Board Member at Annals of the Rheumatic Diseases.
AA: Honoraria and/or research grants from **UCB Pharma**.
LG: Research grants from **UCB Pharma**; consulting fees from **UCB Pharma**; Editorial Board Member at Annals of the Rheumatic Diseases.
ABG: Received honoraria as an advisory board member and consultant for **UCB Pharma;** has received research/educational grants from **UCB Pharma** (all paid to Mount Sinai School of Medicine).
RBW: Consulting fees from **UCB Pharma**; research grants to his institution from UC**B Pharma.**
BI: Employee of **UCB Pharma** and shareholder of **UCB Pharma.**
RB, VS, JC: Employees and shareholders of **UCB Pharma.**
RL: Consultancy fees from **UCB Pharma.** Research grants from **UCB Pharma.** Editorial Board Member at Annals of the Rheumatic Diseases.

UCB Pharma contributed to study design, participated in data collection, completed the data analysis, and participated in data interpretation. UCB Pharma also participated in writing, review, and approval of the manuscript. All authors had full access to the data, reviewed and approved the final version, and were responsible for the decision to submit for publication. A medical writing agency, employed by UCB Pharma, assisted with manuscript preparation under the authors' direction.

US-BK-2401175 | Date of preparation: September 2024

Bimekizumab in patients with active psoriatic arthritis and previous inadequate response or intolerance to tumour necrosis factor-α inhibitors: a randomised, double-blind, placebo-controlled, phase 3 trial (**BE COMPLETE**)

Joseph F Merola, Robert Landewé, Iain B McInnes, Philip J Mease, Christopher T Ritchlin, Yoshiya Tanaka, Akihiko Asahina, Frank Behrens, Dafna D Gladman, Laure Gossec, Alice B Gottlieb, Diamant Thaçi, Richard B Warren, Barbara Ink, Deepak Assudani, Rajan Bajracharya, Vishvesh Shende, Jason Coarse, Laura C Coates

Author Contributions: JFM, RL, IBM, PJM, CTR, YT, AA, FB, DDG, LG, ABG, DT, RBW, BI, DA, RB, VS, JC, and LCC made substantial contributions to study conception and design. JFM, RL, IBM, PJM, CTR, YT, AA, FB, DDG, LG, ABG, DT, RBW, BI, DA, RB, VS, JC, and LCC made substantial contributions to analysis and interpretation of the data. JFM, RL, IBM, PJM, CTR, YT, AA, FB, DDG, LG, ABG, DT, RBW, BI, DA, RB, VS, JC, and LCC drafted the article or revised it critically for important intellectual content. JFM, RL, IBM, PJM, CTR, YT, AA, FB, DDG, LG, ABG, DT, RBW, BI, DA, RB, VS, JC, and LCC approved the final version of the Article to be published. JFM, BI, DA, RB, VS, and JC accessed and verified the data. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Disclosures:
JFM: Consultant or investigator for **UCB Pharma**.
RL: Consultancy fees from **UCB Pharma;** research grants from **UCB Pharma**; and speaker's bureau fees from **UCB Pharma**.
IBM: Consulting fees and honoraria from **UCB Pharma;** and research support from **UCB Pharma**.
PJM: Reports research grants from UCB Pharma; consultancy fees from UCB Pharma; and speakers' bureau fees from **UCB Pharma**.
CTR: Reports research for **UCB Pharma**; and consultancy for **UCB Pharma**.
AA: Reports honoraria or research grants from **UCB Pharma.**
DDG: Received grants from **UCB Pharma;** and consulting fees from **UCB Pharma.**
LG: Reports non-financial support from **UCB Pharma** during the conduct of the study; grants from **UCB Pharma;** personal fees from **UCB Pharma;** and non-financial support from **UCB Pharma** outside the submitted work.
ABG: Reports honoraria as an advisory board member, non-promotional speaker, or consultant for **UCB Pharma**; and research or educational grants from **UCB Pharma** (all funds go to the Icahn School of Medicine at Mount Sinai).
DT: reports honoraria for participation on advisory boards as a speaker and for consultancy from **UCB Pharma.**
RBW: reports consulting fees from **UCB Pharma;** research grants to his institution from **UCB Pharma**
BI: is an employee of **UCB Pharma,** and stockholder of **UCB Pharma.**
DA, RB, VS, and JC: Employees and stockholders of **UCB Pharma.**
LCC: Reports grants and research support from **UCB Pharma;** paid consultancy for **UCB Pharma;** and speaker fees from **UCB Pharma.**

UCB Pharma contributed to study design, participated in data collection, completed the data analysis, and participated in data interpretation. UCB Pharma also participated in writing, review, and approval of the manuscript. All authors had full access to the data, reviewed and approved the final version, and were responsible for the decision to submit for publication. A medical writing agency, employed by UCB Pharma, assisted with manuscript preparation under the authors' direction.

US-BK-2401175 | Date of preparation: September 2024

Bimekizumab treatment in patients with active psoriatic arthritisand prior inadequate response or intolerance to tumour necrosis factor inhibitors: 52-week safety and efficacy from the phase III **BE COMPLETE** study and its open-label extension **BE VITAL**

Laura C Coates, Robert Landewé, Iain B McInnes, Philip J Mease, Christopher T Ritchlin, Yoshiya Tanaka, Akihiko Asahina, Frank Behrens, Dafna D Gladman, Laure Gossec, Ana-Maria Orbai, Alice B Gottlieb, Richard B Warren, Barbara Ink, Rajan Bajracharya, Vishvesh Shende, Jason Coarse, Joseph F Merola

Author Contributions: Substantial contributions to study conception and design, analysis and interpretation of the data, drafting the article or revising it critically for important intellectual content, final approval of the version of the article to be published: **LCC, RL, IBMcI, PJM, CTR, YT, AA, FB, DDG, LG, A-MO, ABG, RBW, BI, RB, VS, JC, JFM**.

Disclosures:
LCC: Grants/research support from **UCB Pharma**, consultant for **UCB Pharma**, speaking fees from **UCB Pharma**. Associate Editor of RMD Open.
RL: Consultancy fees from **UCB Pharma**, research grants from **UCB Pharma.**
IBM: Consulting fees and honoraria from **UCB Pharma**, research support from **UCB Pharma**.
PJM: Research grants from **UCB Pharma**, consultancy fees from **UCB Pharma**, speakers' bureau for **UCB Pharma**.
CTR: Consultant for **UCB Pharma**.
AA: Honoraria and/or research grants from **UCB Pharma**.
DDG: Consultant and/or received grant support from **UCB Pharma**.
LG: Personal fees from **UCB Pharma**.
A-MO: Research grants Consulting fees from UCB Pharma.
ABG: Research/educational grants from **UCB Pharma**, (all paid to Mount Sinai School of Medicine), Honoraria as an advisory board member and consultant for **UCB Pharma**.
RBW: Consulting fees from **UCB Pharma**, research grants to his institution from **UCB Pharma**.
BI: **UCB Pharma**, employee of **UCB Pharma**.
RB and JC: **Employees and shareholders of UCB Pharma.**
VS: **Employee of UCB Pharma**.
JFM: Consultant and/or investigator for **UCB Pharma**.

UCB Pharma contributed to study design, participated in data collection, completed the data analysis, and participated in data interpretation. UCB Pharma also participated in writing, review, and approval of the manuscript. All authors had full access to the data, reviewed and approved the final version, and were responsible for the decision to submit for publication. A medical writing agency, employed by UCB Pharma, assisted with manuscript preparation under the authors' direction

US-BK-2401175 | Date of preparation: September 2024

References

- BIMZELX® [prescribing information]. Smyrna, GA: UCB, Inc.
- Coates LC, et al. Bimekizumab treatment in patients with active psoriatic arthritis and prior inadequate response or intolerance to tumour necrosis factor inhibitors: 52-week safety and efficacy from the phase III BE COMPLETE study and its open-label extension BE VITAL. RMD Open. 2024;1(0):e003855.
- McInnes IB, et al. Bimekizumab in patients with psoriatic arthritis, naïve to biologic treatment: a randomized, double-blind, placebo-controlled, phase 3 trial (BE OPTIMAL). Lancet. 2023;401(10370):25–37.
- Merola JF, et al. Bimekizumab in patients with active psoriatic arthritis and previous inadequate response or intolerance to tumour necrosis factor-α inhibitors: a randomized, double-blind, placebo-controlled, phase 3 trial (BE COMPLETE). Lancet. 2023;401(10370):38–48.
- Ritchlin CT, et al. Bimekizumab treatment in biologic DMARD-naïve patients with active psoriatic arthritis: 52-week efficacy and safety results from the phase III, randomized, placebo-controlled, active reference BE OPTIMAL study. Ann Rheum Dis. 2023;82(1):1404-1414.

US-BK-2401175 | Date of preparation: September 2024