Bimekizumab was Efficacious in Patients with Axial Spondyloarthritis Regardless of Age, BMI, CRP or HLA-B27 Status: 1-Year Results from Two Phase 3 Studies

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Objective

To assess the impact of patient baseline characteristics, including age, body mass index (BMI), C-reactive protein (CRP) levels and human leukocyte antigen B27 (HLA-B27) status, on clinical outcomes in patients with axial spondyloarthritis (axSpA) treated with bimekizumab (BKZ) at Week 16 versus placebo (PBO), and longer term up until 1 year.

Introduction

- BKZ is a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A.
- BKZ has demonstrated sustained long-term efficacy and safety in patients with non-radiographic (nr-) and radiographic (r-)axSpA in the phase 3 studies BE MOBILE 1 and 2,[1] as well as comparable efficacy at 1 year across sexes and at 2 years across varying symptom durations.[2,3]
- Baseline characteristics (age, BMI, CRP, HLA-B27) may predict clinical response in axSpA.[4] Here, we examine BKZ efficacy across these subgroups through 1 year.

Methods

- In the BE MOBILE 1 (nr-axSpA; NCT03928704) and 2 (r-axSpA; NCT03928743) trials, patients were randomised to receive subcutaneous BKZ 160 mg every 4 weeks (Q4W) or PBO. From Week 16–52, all patients received BKZ 160 mg Q4W.
- The proportions of patients achieving Assessment of Spondyloarthritis international Society 40% (ASAS40; non-responder imputation [NRI]) and Axial Spondyloarthritis Disease Activity Score (ASDAS) <2.1, and mean change from baseline in ASDAS, Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), Ankylosing Spondylitis Quality of Life (ASQoL; all multiple imputation [MI]) and objective signs of inflammation (OSI; MRI Spondyloarthritis Research Consortium of Canada [SPARCC] sacroiliac joint [SIJ] and MRI Berlin spine; observed case [OC] for patients enrolled in MRI sub-studies) were assessed from Week 16–52 for the pooled nr-/r-axSpA populations, stratified by baseline:
  - **Age** (≤35, >35–≤45, >45 years)
  - **BMI** (<25, ≥25–<30, ≥30 kg/m^2)
  - **hs-CRP** (≤5, >5 mg/L)
  - **HLA-B27** (negative, positive)
- To compare the Week 16 treatment effect of BKZ versus PBO across subgroups, adjusted odds ratios and least squares (LS) mean differences were calculated using logistic regression and ANCOVA, respectively; p-values are not reported due to the post hoc nature of these analyses.

Results

- Of 586 pooled patients across BE MOBILE 1 and 2, 349 and 237 were randomised to BKZ and PBO at baseline, respectively. Baseline characteristics were largely similar across treatment groups (**Table**).
- Generally, larger improvements in clinical outcomes were seen for BKZ versus PBO across all subgroups at Week 16 (**Figures 1–3**).
  - At Week 16, a larger proportion of patients in the BKZ group achieved ASAS40 and ASDAS <2.1 compared with PBO across all subgroups.
    - A significant difference, indicated by 95% confidence intervals (CIs) for adjusted odds ratios that did not cross 1, was observed for BKZ versus PBO across most subgroups, with the exception of patients aged >45 years, with BMI ≥30 kg/m^2 and HLA-B27 negative patients (ASDAS <2.1 only; **Figure 1**).
  - At Week 16, greater LS mean reductions (i.e., improvements) from baseline in ASDAS, BASDAI and ASQoL were detected for BKZ compared with PBO across all subgroups.
    - A significant difference, indicated by 95% CIs for LS mean differences that did not cross 0, was observed for BKZ versus PBO across most subgroups, with the exception of patients aged >45 years (BASDAI only) and with BMI ≥30 kg/m^2 (BASDAI and ASQoL only; **Figure 2**).
  - At Week 16, greater LS mean reductions from baseline in OSI were detected for BKZ versus PBO across all subgroups.
    - A significant difference, indicated by 95% CIs for LS mean differences that did not cross 0, was observed for BKZ versus PBO across most subgroups, with the exception of patients aged >45 years and HLA-B27 negative patients (all outcomes), those with BMI ≥30 kg/m^2 (MRI SPARCC SIJ only), and those with BMI <25 kg/m^2 and CRP ≤5 mg/L (MRI Berlin spine only; **Figure 3**).
- At Week 52, improvements were sustained or further improved across all subgroups and endpoints in the "All BKZ" group, which included patients randomised to BKZ and PBO-randomised patients who switched to BKZ at Week 16 (**Figures 1–3**).

Conclusions

Bimekizumab demonstrated greater improvements in clinical outcomes compared with placebo at Week 16 in patients with axSpA, regardless of patient demographics and baseline clinical presentation. Improvements were sustained or further improved to 1 year across all subgroups.

Summary

This post hoc analysis assessed the impact of **age**, **BMI**, **CRP** and **HLA-B27** on clinical responses in patients with **axSpA** treated with **bimekizumab** at Week 16 vs placebo, and longer-term up until 1 year, using pooled data from two phase 3 trials.

Week 16

Greater **improvements** in **clinical outcomes** were seen for **bimekizumab** versus placebo **across all subgroups** at Week 16.

ASAS40 · ASDAS · BASDAI · ASQoL · MRI Outcomes (SPARCC SIJ and Berlin spine)

Differences between bimekizumab and placebo were **significant** at Week 16 across **most subgroups** for each clinical outcome.

Week 52

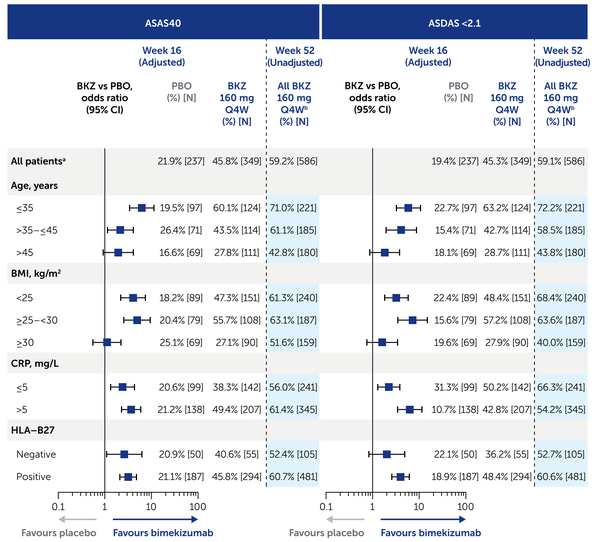
Improvements in clinical responses with bimekizumab treatment were **sustained** or further improved to 1 year, **regardless of baseline characteristics**.

Table Baseline demographics and characteristics by treatment group for patients pooled across BE MOBILE 1 and 2

Mean (SD) unless otherwise specified	PBO (N=237)	BKZ 160 mg Q4W (N=349)
Age, years	38.8 (12.1)	40.0 (11.8)
Sex, male, n (%)	145 (61.2)	233 (66.8)
BMI, kg/m^2	27.4 (5.7)	26.9 (5.9)
HLA-B27, positive, n (%)	187 (78.9)	294 (84.2)
hs-CRP, mg/L, geometric mean (geometric CV, %)	5.7 (216.6)	5.8 (286.5)
hs-CRP >5 mg/L (ULN), n (%)	138 (58.2)	207 (59.3)
ASDAS	3.7 (0.7)	3.7 (0.8)[a]
BASDAI	6.6 (1.3)	6.6 (1.3)
ASQoL	9.0 (4.3)	9.2 (4.7)
MRI SPARCC SIJ[b]	7.4 (10.8)[c]	6.7 (9.2)[d]
MRI Berlin spine[b]	2.2 (3.5)[e]	2.5 (3.9)[f]

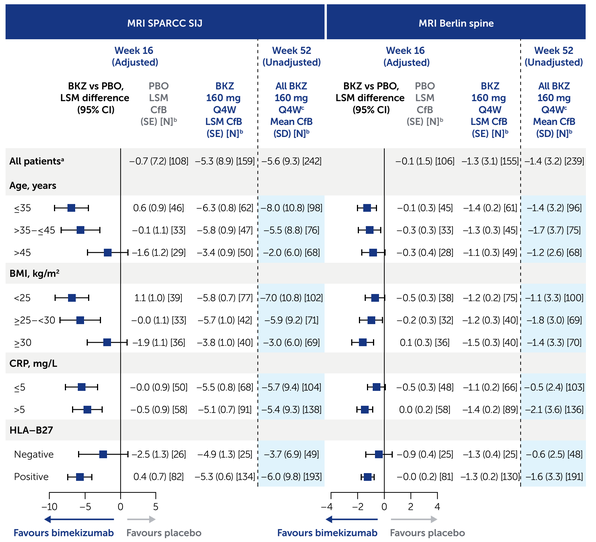
Pooled randomised set. The total ranges of the MRI SPARCC SIJ and MRI Berlin spine scores are 0–72 and 0–69, respectively. **[a]** n=348; **[b]** Only includes patients from the MRI sub-studies; **[c]** n=118; **[d]** n=172; **[e]** n=115; **[f]** n=168.

Figure 1 Patients achieving ASAS40 (NRI) and ASDAS <2.1 (MI) at Week 16 and Week 52, stratified by baseline characteristic subgroups



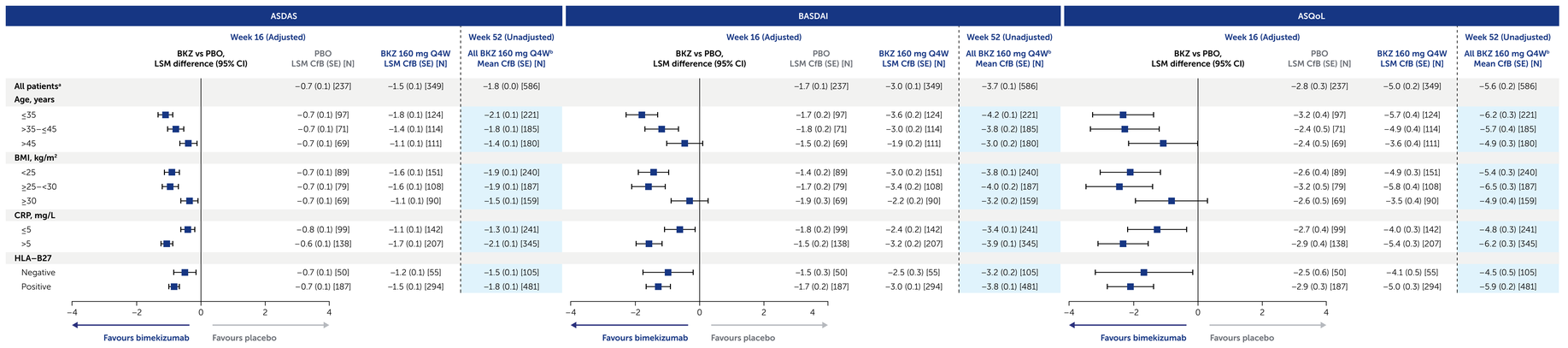
Pooled randomised set (ASAS40 reported with NRI; ASDAS <2.1 reported with MI). N numbers indicate total number of patients available in the treatment group. Adjusted responder rates and odds ratios calculated using logistic regression with factors for treatment, feeder study (BE MOBILE 1 or BE MOBILE 2), region, subgroup, and treatment × subgroup. **[a]** Unadjusted data reported for the 'all patients' row at Week 16 and Week 52; **[b]** All BKZ group includes BKZ-randomised patients and PBO-randomised patients who switched to BKZ at Week 16.

Figure 3 Mean change from baseline in MRI SPARCC SIJ and MRI Berlin spine scores at Week 16 and Week 52, stratified by baseline characteristic subgroups (OC)



Patients enrolled in MRI sub-studies within the pooled randomised set (OC). For MRI SPARCC SIJ and MRI Berlin spine scores, larger decreases indicate larger improvements. Adjusted least squares mean differences calculated using ANCOVA with factors for treatment, region, feeder study (BE MOBILE 1 or BE MOBILE 2), baseline continuous variable value, subgroup, and treatment × subgroup. **[a]** Unadjusted mean CfB (SD) [N] reported for the 'all patients' row at Week 16 and Week 52; **[b]** N numbers reflect the number of patients included in each analysis, rather than the total number of patients in the treatment group; **[c]** All BKZ group includes BKZ-randomised patients and PBO-randomised patients who switched to BKZ at Week 16.

Figure 2 Mean change from baseline in ASDAS, BASDAI and ASQoL at Week 16 and Week 52, stratified by baseline characteristic subgroups (MI)



Pooled randomised set (MI). N numbers indicate total number of patients available in the treatment group. For ASDAS, BASDAI and ASQoL, larger decreases indicate larger improvements. Adjusted least squares mean differences calculated using ANCOVA with factors for treatment, region, feeder study (BE MOBILE 1 or BE MOBILE 2), baseline continuous variable value, subgroup, and treatment × subgroup. **[a]** Unadjusted mean CfB (SE) [N] reported for the 'all patients' row at Week 16 and Week 52; **[b]** All BKZ group includes BKZ-randomised patients and PBO-randomised patients who switched to BKZ at Week 16.

ACR: American College of Rheumatology; **ANCOVA:** analysis of covariance; **ASAS40:** Assessment of Spondyloarthritis International Society 40%; **ASDAS:** Axial Spondyloarthritis Disease Activity Score; **ASQoL:** Ankylosing Spondylitis Quality of Life; **axSpA:** axial spondyloarthritis; **BASDAI:** Bath Ankylosing Spondylitis Disease Activity Index; **BKZ:** bimekizumab; **BMI:** body mass index; **CfB:** change from baseline; **CI:** confidence interval; **CRP:** C-reactive protein; **CV:** coefficient of variation; **HLA–B27:** human leukocyte antigen B27; **IL:** interleukin; **LS:** least square; **LSM:** least squares mean; **MI:** multiple imputation; **MRI:** magnetic resonance imaging; **nr-axSpA:** non-radiographic axSpA; **NRI:** non-responder imputation; **OC:** observed case; **OSI:** objective signs of inflammation; **PBO:** placebo; **Q4W:** every 4 weeks; **r-axSpA:** radiographic axSpA; **SD:** standard deviation; **SE:** standard error; **SIJ:** sacroiliac joint; **SPARCC:** Spondyloarthritis Research Consortium of Canada; **ULN:** upper limit of normal.

References: [1]Baraliakos X. Rheumatology (Oxford) 2025;64:3534–46; [2]Rudwaleit M. Ann Rheum Dis 2025;84 (suppl 1):1047–9; [3]Ramiro S. Arthritis Res Ther 2026;28:40; [4]Wang R. JAMA Netw Open 2022;5:e222312. **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **MM**, **HMO**, **YK**, **DN**, **MR**, **AD**, **GV**, **CP**, **SK**, **VNC**; Drafting of the publication, or reviewing it critically for important intellectual content: **MM**, **HMO**, **YK**, **DN**, **MR**, **AD**, **GV**, **CP**, **SK**, **VNC**; Final approval of the publication: **MM**, **HMO**, **YK**, **DN**, **MR**, **AD**, **GV**, **CP**, **SK**, **VNC**. **Author Disclosures: MM:** Consultancy fees from AbbVie, BMS, Eli Lilly, Novartis, Pfizer and UCB; research grants from AbbVie, BMS and UCB. **HMO:** Speaking honoraria and/or consultancy fees from AbbVie, Advanz Pharma, Amgen, Biogen, Eli Lilly, Janssen, MoonLake, Novartis, Pfizer, Takeda and UCB; research grants from Janssen, Novartis, Pfizer and UCB. **YK:** Speaker fees from AbbVie, BMS, Eli Lilly, Gilead, Incannex, Janssen, Novartis, Pfizer, Servatus, Sun Pharma and UCB; advisory board or speaker fees from AbbVie, AstraZeneca, Janssen, Novartis, Pfizer, Sandoz and UCB. **MR:** Speakers bureau from AbbVie, Boehringer Ingelheim, Eli Lilly, Janssen, Novartis, Pfizer and UCB; consultant for AbbVie, Eli Lilly, Janssen, Novartis and UCB. **AD:** Speaker for Eli Lilly, J&J, Novartis, Pfizer and UCB; consultant for BMS, Eli Lilly, J&J, MoonLake, Novartis, Pfizer and UCB; grant/research support from BMS, Eli Lilly, J&J, Novartis, Pfizer and UCB. **GV:** Speaker or consulting fees from AbbVie, Amgen, Alfasigma, Celltrion, Eli Lilly, Galapagos, Janssen, Novartis, Pfizer and UCB; research support from AbbVie, Celltrion, EG, Galapagos, MSD, Pfizer and Takeda. **CP:** Contractor for UCB and employee of Veramed. **SK:** Consultant for Aclipse Therapeutics, Aliada Therapeutics, Allay Therapeutics, Autobahn Therapeutics, Biolacuna, Calibr-Skaggs Institute of Innovative Medicines, Cognition Therapeutics, Cytonics Corp, Karuna Therapeutics, Kisbee Therapeutics, LB Pharmaceuticals, Lotus Clinical, Nesos, Neuralink, Novartis, Onward Medical, PharPoint Research, Summit Analytical, Therini Bio, Tonix Pharmaceuticals, Tornado Therapeutics, UCB and Worldwide Clinical Trials. **VNC:** Speakers bureau for AbbVie, Alfasigma, Eli Lilly, Fresenius Kabi, Janssen, MSD, Novartis, Pfizer and UCB; consultant for AbbVie, Alfasigma, Eli Lilly, Galapagos, MoonLake, MSD, Novartis, Pfizer and UCB; grant/research support from AbbVie and Novartis. **Acknowledgements:** The authors thank the patients, the investigators and their teams who took part in these studies. The authors also thank Celia Menckeberg, PhD, of UCB for editorial review during poster development and publication coordination, Libby Sadler, MSc, Costello Medical, London, UK for medical writing, Roshni Patel, BSc, Costello Medical, London, UK for editorial assistance and the Costello Medical Creative team for design support. Funded by UCB. All costs associated with development of this poster were funded by UCB.

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