Impact of Bimekizumab on MRI Inflammatory and Structural Lesions in the Sacroiliac Joints of Patients with Non-Radiographic Axial Spondyloarthritis: 2-Year Results from a Phase 3 Study and its Open-Label Extension

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Objective

To evaluate the impact of two years of bimekizumab (BKZ) treatment on MRI inflammatory and structural lesions in the sacroiliac joints (SIJ) of patients with non-radiographic axial spondyloarthritis (nr-axSpA) in the phase 3 study, BE MOBILE 1, and its open-label extension (OLE).

Background

- AxSpA is a chronic inflammatory disease mainly affecting the SIJ and spine.[1] MRI is used to detect active inflammatory and structural lesions in the axial skeleton.[2] Evaluating SIJ inflammation and structural lesions using MRI is particularly important in nr-axSpA, where structural changes are not yet visible on radiographs.[2]
- BKZ, a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated sustained efficacy and was well tolerated over three years in patients across the full spectrum of axSpA in the phase 3 studies BE MOBILE 1 (nr-axSpA) and BE MOBILE 2 (radiographic [r-]axSpA) and their combined OLE.[3]
- Treatment with BKZ has previously been shown to lead to rapid reductions in MRI inflammation and have a considerable impact on structural lesions in the SIJ at Week 16, compared with placebo (PBO).[4]
- The long-term effects of BKZ on MRI inflammatory and structural lesions in axSpA have not been reported.

Methods

- In BE MOBILE 1 (NCT03928704), patients with nr-axSpA were randomised to subcutaneous BKZ 160 mg every 4 weeks or PBO; from Week 16, all patients received BKZ. At Week 52, eligible patients could enrol in the OLE (NCT04436640) and continue receiving BKZ to Week 164.
- MRI assessments are reported in patients in the MRI sub-study with evaluable MRIs at baseline, Week 52 and Week 104. MRIs were evaluated centrally by two independent expert readers blinded to timepoint and any clinical data, with an adjudicator for disagreements.
- MRI Spondyloarthritis Research Consortium of Canada (SPARCC) SIJ inflammation score was used to assess inflammation, using short tau inversion recovery (STIR) sequences, and SPARCC SIJ Structural Scores (SSS: erosion, backfill, fat lesions, ankylosis) were used to assess structural lesions, using T1-weighted sequences.[2]
- At Weeks 52 and 104, we report mean change from baseline (CfB) in SPARCC SIJ inflammation score, the proportion of patients with a baseline SPARCC SIJ inflammation score ≥2 achieving MRI remission (SPARCC SIJ score <2) and mean CfB and cumulative probability of CfB in SPARCC SSS components.
- All data are pooled across treatment arms and reported as observed case.

Results

- In total, 254 patients were randomised in BE MOBILE 1, of which 152/254 (59.8%) were included in the MRI sub-study. Of these, 90/152 (59.2%) patients had evaluable MRI SPARCC SIJ inflammation assessments and 103/152 (67.8%) patients had evaluable MRI SPARCC SSS assessments at all three timepoints.
- Baseline characteristics in the MRI sub-study were comparable to the overall study population (**Table**).

MRI inflammation

- A substantial mean reduction from baseline in SPARCC SIJ inflammation score was observed at Week 52, with a largely similar reduction observed at Week 104 (**Figure 1A**).
- In patients with SPARCC SIJ score ≥2 at baseline (n=57), more than half of patients achieved MRI remission (SPARCC SIJ score <2) at Week 52; this proportion was similar at Week 104 (**Figure 1B**).
  - Of these patients, 26/57 (45.6%) achieved MRI remission at both Week 52 and Week 104.

MRI structural lesions

- A mean reduction from baseline in erosions, evaluated using SPARCC SSS, was observed at Week 52, with a largely similar reduction observed at Week 104 (**Figure 2A**).
- Mean increases from baseline in backfill and fat lesions were observed at Week 52, with minimal further change at Week 104. Overall, no ankylosis was observed at any of the three timepoints (**Figure 2B–D**).
- The cumulative probabilities of CfB in each SPARCC SSS component at Week 52 and Week 104 are depicted in **Figure 3**.

Conclusions

Two years of bimekizumab treatment led to substantial reduction in MRI SIJ inflammation in patients with nr-axSpA, with more than half of patients achieving MRI SIJ remission at Weeks 52 and 104. A decrease from baseline in erosions and increases from baseline in backfill and fat lesions were observed at Week 52 with minimal further change at Week 104. No ankylosis was observed at either timepoint.

These results build on previous findings, indicating sustained control of inflammation and potential tissue repair in the SIJ of patients with nr-axSpA with two years of bimekizumab.

Summary

MRI SIJ evaluations demonstrated that, in patients with **nr-axSpA**, **two years** of dual IL-17A and IL-17F inhibition with **bimekizumab** led to:

Inflammation

A substantial **reduction** from baseline in MRI **inflammation** at Week 52, with a similar reduction at Week 104

More than half of patients achieving MRI **remission** at Week 52 and Week 104[a]

Structural Lesions

Decreases from baseline in **erosions** at Week 52 and Week 104

Increases from baseline in **backfill and fat lesions** at Week 52 and Week 104

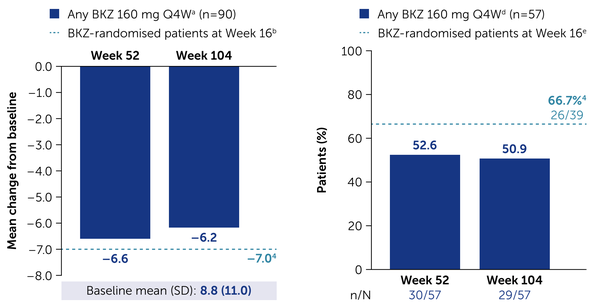
No ankylosis at Week 52 or Week 104

[a] MRI SIJ remission was defined as SPARCC SIJ score <2.

Table Baseline characteristics in BE MOBILE 1 and its MRI sub-study

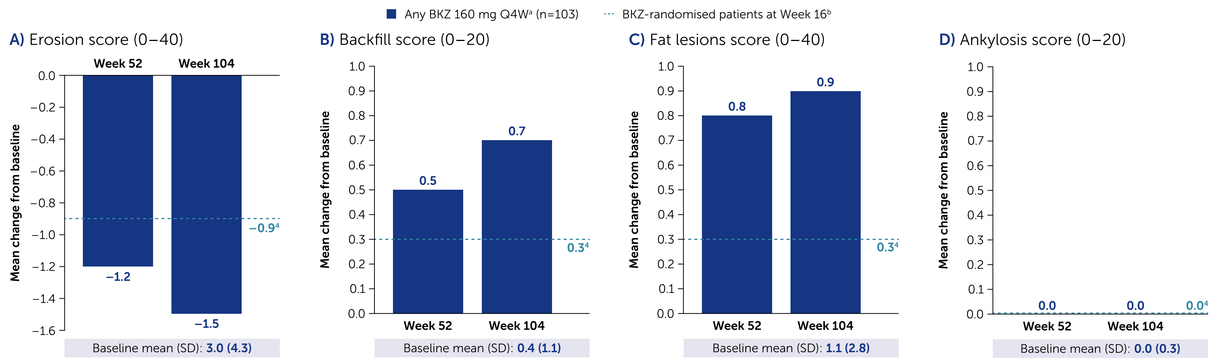
Mean (SD), unless otherwise stated	Overall BE MOBILE 1 population N=254	BE MOBILE 1 MRI sub-study n=152
Age, years	39.4 (11.5)	39.4 (12.0)
Sex, male, n (%)	138 (54.3)	81 (53.3)
HLA-B27 positive, n (%)	197 (77.6)	112 (73.7)
Symptom duration, years	9.0 (8.8)	8.9 (8.8)
ASDAS	3.7 (0.7)	3.6 (0.7)
BASDAI	6.8 (1.3)	6.7 (1.3)
hs-CRP, mg/L, geometric mean (geometric CV, %)	4.8 (261.8)	4.3 (243.2)
Prior TNFi exposure, n (%)	27 (10.6)	13 (8.6)

Figure 1 Mean change from baseline in MRI SPARCC SIJ inflammation score and achievement of SPARCC SIJ remission at Week 52 and Week 104 (OC)



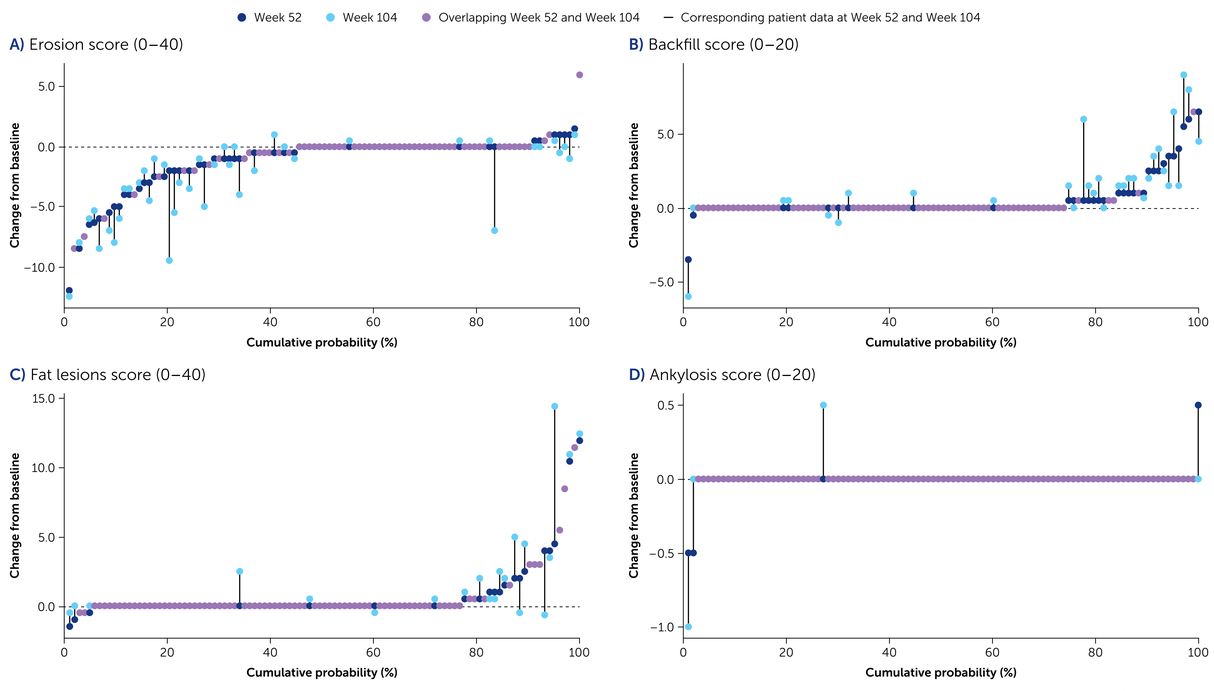
Includes patients in the MRI sub-study of BE MOBILE 1 with SPARCC SIJ assessments available at baseline, Week 52 and Week 104. **[a]** Includes patients originally randomised to placebo who switched to BKZ at Week 16; **[b]** Previously published BE MOBILE 1 Week 16 data, scored at Week 52;[4] n=64; **[c]** Assessed in patients with SPARCC SIJ score ≥2 at baseline; **[d]** Includes patients originally randomised to placebo who switched to BKZ at Week 16 and had SPARCC SIJ score ≥2 at baseline; **[e]** Previously published BE MOBILE 1 Week 16 data, scored at Week 52.[4]

Figure 2 Mean change from baseline in MRI SPARCC SSS components at Week 52 and Week 104 (OC)



Includes patients in the MRI sub-study of BE MOBILE 1 with SPARCC SSS assessments available at baseline, Week 52 and Week 104. **[a]** Includes patients originally randomised to placebo who switched to BKZ at Week 16; **[b]** Previously published BE MOBILE 1 Week 16 data, scored at Week 52;[4] n=69.

Figure 3 Cumulative probability of change from baseline in MRI SPARCC SSS components at Week 52 and Week 104 (OC)



Includes patients in the MRI sub-study of BE MOBILE 1 with SPARCC SSS assessments available at baseline, Week 52 and Week 104. Data presented include all patients receiving BKZ, including patients originally randomised to placebo who switched to BKZ at Week 16 (n=103).

ASDAS: Axial Spondyloarthritis Disease Activity Score; **axSpA:** axial spondyloarthritis; **BKZ:** bimekizumab; **CfB:** change from baseline; **hs-CRP:** high-sensitivity C-reactive protein; **HLA:** human leukocyte antigen; **IL:** interleukin; **MRI:** magnetic resonance imaging; **nr-axSpA:** non-radiographic axial spondyloarthritis; **OC:** observed case; **OLE:** open-label extension; **PBO:** placebo; **Q4W:** every 4 weeks; **r-axSpA:** radiographic axial spondyloarthritis; **SD:** standard deviation; **SIJ:** sacroiliac joint; **SPARCC:** Spondyloarthritis Research Consortium of Canada; **SSS:** SIJ Structural Scores; **TNFi:** tumour necrosis factor inhibitor.

References: [1]Navarro-Compán V. Lancet 2025;405:159–72; [2]Sieper J. Lancet 2017;390:73–84; [3]Baraliakos X. Ann Rheum Dis 2025;84(suppl 1):947–8; [4]Maksymowych W. Arthritis Rheumatol 2024;76(suppl 9). **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **WPM**, **SR**, **DP**, **LSG**, **RGWL**, **CP**, **AM**, **MØ**, **XB**. Drafting of the publication, or reviewing it critically for important intellectual content: **WPM**, **SR**, **DP**, **LSG**, **RGWL**, **CP**, **AM**, **MØ**, **XB**. Final approval of the publication: **WPM**, **SR**, **DP**, **LSG**, **RGWL**, **CP**, **AM**, **MØ**, **XB**. **Author Disclosures: WPM:** Honoraria/consulting fees from AbbVie, BMS, Boehringer-Ingelheim, Celgene, Eli Lilly, Galapagos, Janssen, Novartis, Pfizer and UCB; research grants from AbbVie, BMS, Eli Lilly, Galapagos, Pfizer and UCB; educational grants from AbbVie, Novartis and Pfizer; Chief Medical Officer for CARE ARTHRITIS. **SR:** Consultant for AbbVie, Alfasigma, Eli Lilly, Johnson & Johnson, MSD, Novartis, Pfizer, Sanofi, Takeda and UCB; Grants from AbbVie, Alfasigma, Eli Lilly, MSD, Novartis, Pfizer and UCB. **DP:** Speaker for AbbVie, BMS, Eli Lilly, MSD, Novartis, Pfizer and UCB; Consultant for AbbVie, Biocad, Eli Lilly, Gilead, GSK, Moonlake, MSD, Novartis, Pfizer, Samsung Bioepis and UCB; Grant/research support from: AbbVie, Lilly, MSD, Novartis and Pfizer. **LSG:** Grants from UCB paid to institution; consulting fees from Acelyrin, Eli Lilly, Janssen, Novartis, Pfizer and UCB. **RGWL:** Consultant for AbbVie, CARE Arthritis and Image Analysis Group. **CP:** Contractor for UCB and employee of Veramed. **AM:** Employee of UCB. **MØ:** Research grants from AbbVie, Amgen, BMS, Celgene, Eli Lilly, Merck, Novartis and UCB; speaker fees from AbbVie, BMS, Celgene, Eli Lilly, Galapagos, Gilead, Janssen, MEDAC, Merck, Novartis, Pfizer, Sandoz and UCB; consultancy fees from AbbVie, BMS, Celgene, Eli Lilly, Galapagos, Gilead, Janssen, Merck, Novartis, Pfizer and UCB. **XB:** Speakers bureau for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, JbJ, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; paid instructor for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, JbJ, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; consultant for AbbVie, Advanz, Alexion, Alfasigma, Amgen, BMS, Celltrion, Cesas, Clarivate, Galapagos, JbJ, Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB and Zuellig; grant/research support from AbbVie, Celltrion, Janssen, MoonLake and Novartis.
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