Impact of Bimekizumab on Spinal MRI Inflammation and Structural Lesions in Patients with Radiographic Axial Spondyloarthritis: 2-Year CANDEN Scoring Results from a Phase 3 Study and Its Open-Label Extension

0428

Walter P. Maksymowych,[1] Robert G. W. Lambert,[2] Victoria Navarro-Compán,[3] Xenofon Baraliakos,[4] Jason Coarse,[5] Natasha de Peyrecave,[6] Mikkel Østergaard[7,8]

[1]Department of Medicine, University of Alberta, 568 Heritage Building, Edmonton, AB, Canada; [2]University of Alberta, Department of Radiology & Diagnostic Imaging, Edmonton, AB, Canada; [3]Department of Rheumatology, La Paz University Hospital, IdiPaz, Madrid, Spain; [4]Rheumazentrum Ruhrgebiet Herne, Ruhr-University Bochum, Germany; [5]UCB, Morrisville, NC, USA; [6]UCB, Brussels, Belgium; [7]Department of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark; [8]Copenhagen Center for Arthritis Research, Center for Rheumatology, Rigshospitalet, Glostrup, Denmark.

Objective

To evaluate the effects of 2-year treatment with bimekizumab (BKZ) on spinal MRI inflammatory and structural lesions in patients with radiographic axial spondyloarthritis (r-axSpA) from a phase 3 study and its open-label extension (OLE) using Canada-Denmark (CANDEN) scoring.

Background

- Spinal inflammation and structural progression are key features of r-axSpA.[1,2] CANDEN scoring enables anatomical-based assessments of MRI inflammatory and structural lesions by spinal compartment.[3]
- BKZ, a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has shown sustained efficacy and safety to 3 years in patients across the full spectrum of axSpA in the phase 3 studies BE MOBILE 1 and BE MOBILE 2 and their open-label extension (OLE), and to 5 years in r-axSpA in the phase 2b study BE AGILE.[4,5]
  - Minimal spinal radiographic progression with two years of BKZ treatment has previously been demonstrated in r-axSpA, evaluated using the modified Stoke Ankylosing Spondylitis Spinal Score (mSASSS).[6]
- Here, we present the first 2-year evaluation of the effects of a biologic treatment (i.e., BKZ) on active MRI inflammation and structural lesions across the whole spine of patients with r-axSpA using CANDEN scoring.

Methods

- In BE MOBILE 2 (NCT03928743), patients with r-axSpA were randomized to subcutaneous BKZ 160 mg every 4 weeks or placebo (PBO); from Week 16, all patients received BKZ. At Week 52, eligible patients could enroll in the OLE (NCT04436640) and continue receiving BKZ.[4]
- Post hoc CANDEN scoring assessments were conducted in patients included in the MRI sub-study who had evaluable spinal MRIs at all four timepoints: baseline, Week 16, Week 52, and Week 104; all readers were blinded to timepoint.
- We report mean change from baseline (CfB) at Week 16, Week 52, and Week 104 in total inflammation score and subscores for vertebral bodies (corners and non-corners), posterolateral elements, and facet joints, as well as the cumulative probability for mean CfB in total inflammation score at Week 16; in patients with CANDEN assessments at baseline and Week 16. Mean CfB of total structural lesion scores (fat lesions, erosions, and new bone formation) at Week 16, Week 52, and Week 104 are also presented.
- All data are reported using observed case (OC).

Results

Patients

- 140/332 (42.2%) patients in BE MOBILE 2 had MRIs eligible for CANDEN scoring. 102 patients (PBO/BKZ: 38; BKZ: 64) had CANDEN scores at baseline, Week 16, Week 52, and Week 104.
  - The baseline characteristics of these patients were largely comparable between treatment arms and with the overall population (**Table**).

Inflammation

- At Week 16, a greater reduction from baseline in the total inflammation score was seen with BKZ versus PBO (**Figures 1A and 2**).
  - At Week 52, after switching to BKZ at Week 16, PBO-randomized patients had an improvement from baseline comparable to BKZ-randomized patients, both of which were largely sustained at Week 104.
- Similar patterns of improvement were observed for the vertebral body (corners only) and posterolateral elements subscores (**Figures 1B and 1D**).
- At Week 104, minimal mean CfB was observed in both treatment arms in the vertebral body (non-corners only) and facet joint subscores (**Figures 1C and 1E**).

Structural lesions

- At Week 16, a greater increase from baseline in the total fat lesion score was seen with BKZ versus PBO (**Figure 3**).
  - From Week 16 to Week 104, minimal further change in the total fat lesion score in BKZ-randomized patients was observed. After switching to BKZ at Week 16, PBO-randomized patients followed the pattern of an increase in the total fat lesion score at Week 52 followed by minimal change at Week 104.
- No changes were observed in the total erosion score to Week 104.
- Minimal changes were observed in the total new bone formation score to Week 104 (**Figure 4**).

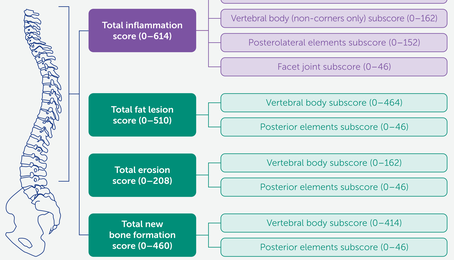
Conclusions

In patients with r-axSpA, comprehensive scoring of MRI spinal inflammatory and structural lesions using CANDEN showed that two years of bimekizumab treatment led to a rapid and sustained reduction in inflammation, an initial increase in fat lesions with minimal subsequent change, and minimal changes in erosions and new bone formation.

These results build on previous findings that showed minimal spinal radiographic progression in r-axSpA with two years of bimekizumab treatment, possibly suggesting that bimekizumab leads to tissue repair in the spine without resulting in new bone formation.

Summary

The **CANDEN** scoring system allows for **detailed, anatomical evaluations** of **MRI inflammatory and structural lesions in the spine**, using the following scores and subscores:



In patients with r-axSpA, **two years of treatment with bimekizumab** led to:

- Rapid and sustained **reductions** from baseline in **inflammation**
- **Increases** from baseline in **fat lesions**
- **No changes** in **erosions**
- **Minimal changes** in new **bone formation**

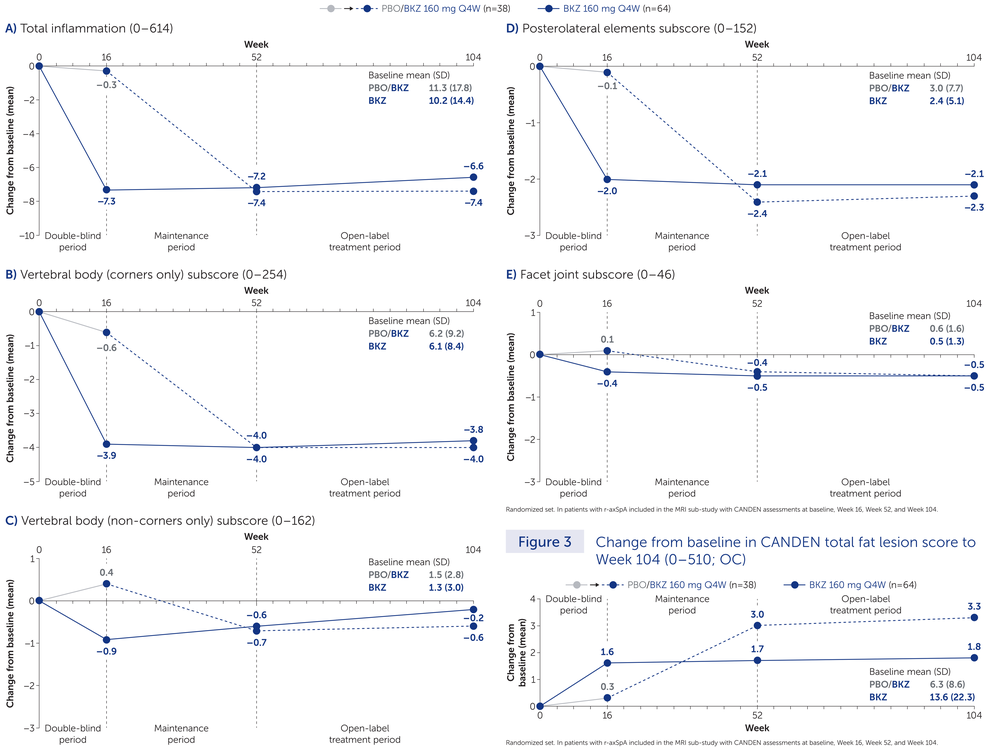
Table Patient demographics and baseline characteristics

Mean (SD), unless otherwise specified	Patients with CANDEN assessments at all four timepoints[a]		Overall population	
	PBO (n=38)	BKZ 160 mg Q4W (n=64)	PBO (n=111)	BKZ 160 mg Q4W (n=221)
Age, years	39.3 (11.7)	40.5 (11.9)	39.2 (12.6)	41.0 (12.1)
Sex, male, n (%)	25 (65.8)	47 (73.4)	80 (72.1)	160 (72.4)
HLA–B27 positive, n (%)	29 (76.3)	59 (92.2)	93 (83.8)	191 (86.4)
Symptom duration, years	12.1 (9.0)	13.1 (10.9)	11.9 (8.6)	14.2 (11.0)
ASDAS	3.6 (0.7)	3.8 (0.9)	3.7 (0.8)	3.7 (0.8)[b]
BASDAI	6.5 (1.3)	6.5 (1.3)	6.5 (1.3)	6.5 (1.3)
BASFI	5.0 (1.9)	5.1 (2.1)	5.2 (2.0)	5.3 (2.2)
ASQoL	7.3 (3.9)	8.5 (4.9)	8.5 (4.3)	9.0 (4.7)
MRI Berlin spine score	3.8 (5.7)	3.5 (4.3)	3.2 (4.1)[c]	3.3 (4.5)[d]
hs-CRP, mg/L, median (Q1, Q3)	6.1 (2.2, 11.2)	8.2 (2.5, 22.7)	6.3 (2.7, 17.2)	8.2 (2.4, 17.7)
Prior TNFi exposure, n (%)	4 (10.5)	10 (15.6)	17 (15.3)	37 (16.7)

Randomized set. **[a]** Includes patients with CANDEN assessments at baseline, Week 16, Week 52, and Week 104; **[b]** n=220; **[c]** n=48; **[d]** n=89.

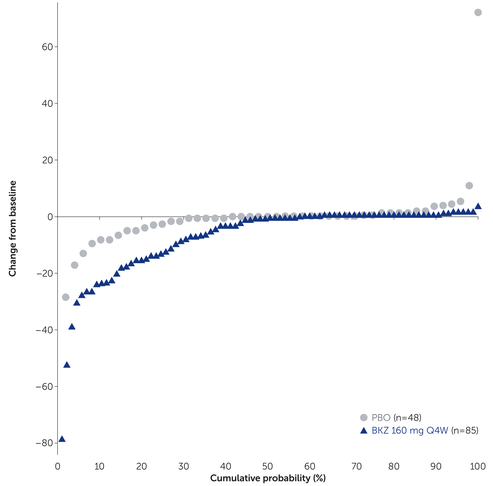
ASDAS: Axial Spondyloarthritis Disease Activity Score; **ASQoL:** Ankylosing Spondylitis Quality of Life; **BASDAI:** Bath Ankylosing Spondylitis Disease Activity Index; **BASFI:** Bath Ankylosing Spondylitis Functional Index; **BKZ:** bimekizumab; **CANDEN:** Canada–Denmark; **CfB:** change from baseline; **HLA:** human leukocyte antigen; **hs-CRP:** high-sensitivity C–reactive protein; **IL:** interleukin; **MRI:** magnetic resonance imaging; **mSASSS:** modified Stoke Ankylosing Spondylitis Spinal Score; **OC:** observed case; **OLE:** open-label extension; **PBO:** placebo; **Q4W:** every 4 weeks; **r-axSpA:** radiographic axial spondyloarthritis; **SD:** standard deviation; **TNFi:** tumor necrosis factor inhibitor.

Figure 1 Change from baseline to Week 104 in CANDEN total inflammation score and subscores (OC)



Randomized set. In patients with r-axSpA included in the MRI sub-study with CANDEN assessments at baseline, Week 16, Week 52, and Week 104.

Figure 2 Cumulative probability plot for change from baseline in CANDEN total inflammation score at Week 16 (0–614; OC)

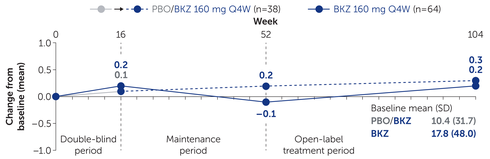


Randomized set. In patients with r-axSpA included in the MRI sub-study with CANDEN assessments at baseline and Week 16.

Figure 3 Change from baseline in CANDEN total fat lesion score to Week 104 (0–510; OC)

Randomized set. In patients with r-axSpA included in the MRI sub-study with CANDEN assessments at baseline, Week 16, Week 52, and Week 104.

Figure 4 Change from baseline in CANDEN total new bone formation score to Week 104 (0–460; OC)



Randomized set. In patients with r-axSpA included in the MRI sub-study with CANDEN assessments at baseline, Week 16, Week 52, and Week 104.

References: [1]Navarro-Compán V. Lancet 2025;405:159–72; [2]Lu L. Front Immunol 2021;12:700260; [3]Krabbe S. RMD Open 2019;5:e001057; [4]Baraliakos X. Ann Rheum Dis 2025;84(suppl 1):947–8; [5]Deodhar A. RMD Open 2025;11:e005081; [6]Baraliakos X. ACR Convergence 2024 [Presentation 1758].

Author Contributions: Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **WPM**, **RGWL**, **VNC**, **XB**, **JC**, **NdP**, **MØ**; Drafting of the publication, or reviewing it critically for important intellectual content: **WPM**, **RGWL**, **VNC**, **XB**, **JC**, **NdP**, **MØ**; Final approval of the publication: **WPM**, **RGWL**, **VNC**, **XB**, **JC**, **NdP**, **MØ**. **Author Disclosures: WPM:** Honoraria/consulting fees from AbbVie, BMS, Boehringer-Ingelheim, Celgene, Eli Lilly, Galapagos, Janssen, Novartis, Pfizer, and UCB; educational grants from AbbVie, BMS, Eli Lilly, Galapagos, Pfizer, and UCB; research grants from AbbVie, Novartis, and Pfizer; Chief Medical Officer for CARE ARTHRITIS. **RGWL:** Consultant for AbbVie, CARE Arthritis and Image Analysis Group. **VNC:** Speakers bureau for AbbVie, 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. **XB:** Speakers bureau for AbbVie, Advanz, Alexion, Alphasigma, Amgen, BMS, Cesas, Celltrion, Clarivate, Galapagos, J&J, Eli Lilly, MoonLake, Novartis, Peervoice, Pfizer, Roche, Sandoz, Springer, Stada, Takeda, UCB, and Zuellig; grant/research support from AbbVie, Celltrion, Janssen, MoonLake, and Novartis. **JC:** Employee and shareholder of UCB. **NdP:** 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. **Acknowledgments:** We would like to thank the patients and their caregivers in addition to all the investigators and their teams who contributed to this study. The authors acknowledge Celia Menckeberg, PhD, UCB, for publication coordination, Sneha Krishnamurthy, MSc, Costello Medical, London, UK for medical writing and editorial assistance, and the Costello Medical Creative team for design support. ~~Funded by UCB.~~ All costs associated with development of this ~~presentation~~ were funded by UCB.

To receive a copy of this poster, scan the QR code.
Link expiration:
January 27, 2026