Maintenance of Response to Bimekizumab Over 3 Years of Treatment in Patients with Active Ankylosing Spondylitis: Post Hoc Analyses from the BE AGILE Study and its Open-Label Extension

V. Navarro-Compán,[1] M. Rudwaleit,[2] N. de Peyrecave,[3] C. Fleurinck,[3] M. Oortgiesen,[4] V. Taieb,[5] X. Baraliakos[6]

Presented at EULAR 2022 | 1–4 June

POS0938

Objective

To report on the maintenance of clinical response, including achievement of sustained inactive disease or low disease activity, to support and extend previous findings on the long-term efficacy of bimekizumab (BKZ) in patients with active ankylosing spondylitis (AS) treated for up to 3 years in the phase 2b BE AGILE study and its open-label extension (OLE).

Background

- BKZ, a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated clinical efficacy and was well tolerated in patients with active AS treated for up to 156 weeks in the phase 2b BE AGILE study.[1,2]
- Maintaining stringent disease control – such as sustained inactive disease or low disease activity – is an internationally recognised treatment target in spondyloarthritis and a relevant goal in clinical practice.[3]

Methods

- Study designs of the 48-week BE AGILE study (NCT02963506) and the ongoing 4-year OLE (NCT03355573) have been reported previously.[1,2]
- This post hoc analysis used data from patients randomised at baseline to subcutaneous BKZ 160 mg or 320 mg every 4 weeks (Q4W). At Week 48, patients receiving BKZ 320 mg Q4W switched to BKZ 160 mg Q4W.
- We report Ankylosing Spondylitis Disease Activity Score C-reactive protein (ASDAS-CRP), Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) and Assessment in SpondyloArthritis international Society 40% (ASAS40) response through 156 weeks.
- ASDAS scores used to assign disease activity were as follows: very high disease (VHD) activity: >3.5; high disease (HD) activity: ≥2.1–≤3.5; low disease (LD) activity: ≥1.3–<2.1; inactive disease (ID): <1.3.
- All reported analyses are observed case.

Results

- 121/303 patients were randomised to BKZ 160 mg (n=60) or 320 mg (n=61). At Week 156, relevant efficacy outcomes were recorded for 51/60 (85.0%) patients initially randomised to BKZ 160 mg, and 42/61 (68.9%; ASDAS and ASAS40) and 45/61 (73.8%; BASDAI) patients initially randomised to BKZ 320 mg.
- At baseline, mean (standard deviation [SD]) BASDAI was 6.3 (1.3) and 6.5 (1.6) while ASDAS was 3.9 (0.8) and 3.9 (0.7), respectively.
- At baseline, nearly all patients had ASDAS HD or VHD activity; by Week 48, more than half had achieved LD activity or ID (ASDAS <2.1: BKZ 160 mg, 55.4%; BKZ 320 mg, 63.6%; Figure 1).

Disease activity over time

- Improvements in ASDAS were consistent through 156 weeks across both BKZ 160 mg and 320 mg groups, with treatment response levels maintained to Week 156 following dose reduction to BKZ 160 mg at Week 48 in the BKZ 320 mg group (Figure 1; Figure 2A).
- Improvements in BASDAI scores were also sustained to Week 156 (Figure 2B), at which point mean (SD) BASDAI was 2.1 (1.8) and 2.2 (1.5) in patients initially randomised to BKZ 160 mg and 320 mg, respectively.

Maintenance of disease control in Week 12 responders

- Of those who achieved ASDAS <2.1 at Week 12 across both groups, 33/37 (89.2%) patients with an ASDAS assessment at Week 156 had maintained ASDAS <2.1, and 24/37 (64.9%) maintained this level of disease control at every assessment during Weeks 12–156.
- Of patients who achieved ASAS40 at Week 12, 44/47 (93.6%) patients with an assessment of the ASAS components at Week 156 had maintained ASAS40 response, and 36/47 (76.6%) had maintained ASAS40 response at every assessment during Weeks 12–156.

Conclusion

BKZ 160 mg Q4W provided long-term improvements in disease activity over 3 years in patients with active AS, irrespective of the initial dosing regimen (BKZ 160 mg or 320 mg Q4W). Robust maintenance of disease control over 3 years was observed in patients who had initially responded at Week 12.

Summary

Study design

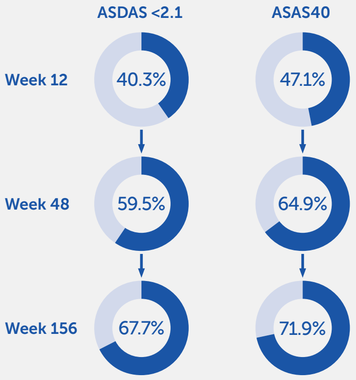
This analysis examined outcomes from the 121/303 patients with active **ankylosing spondylitis** in BE AGILE who were randomised to:

BKZ 160 mg Q4W (n=60) | BKZ 320 mg Q4W (n=61)

111 patients had a relevant efficacy assessment at Week 48

Patients received open-label BKZ 160 mg Q4W during Weeks 48–156

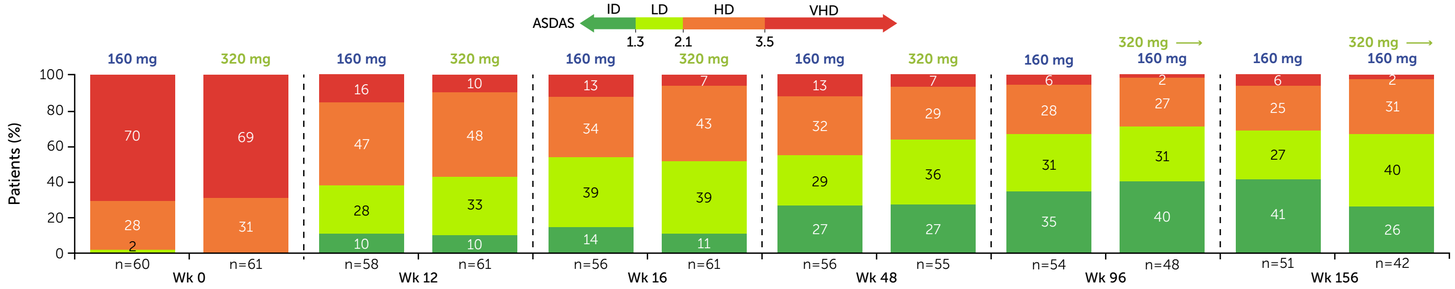
Maintenance of efficacy outcomes[a]



In Week 12 responders, maintenance of responses at Week 156 were:[a,b]

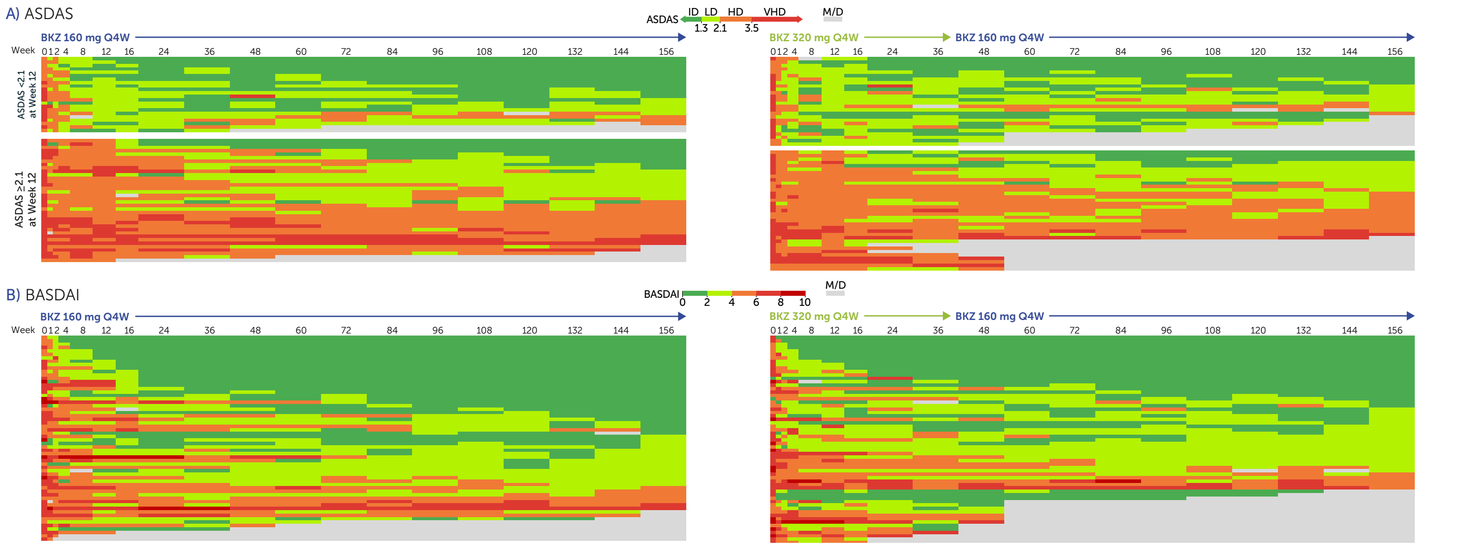
[a]Data reported as observed case; [b]Of patients with an assessment of ASDAS/ASAS at Week 156. Data for both patients initially randomised to BKZ 160 mg and 320 mg shown.

Figure 1 ASDAS states across Weeks 0–156 for patients randomised to BKZ 160 mg and 320 mg Q4W at baseline



Full analysis set. Data reported as observed case. VHD: ASDAS >3.5; HD: ASDAS ≥2.1–≤3.5; LD: ASDAS ≥1.3–<2.1; ID: ASDAS <1.3.

Figure 2 (A) ASDAS and (B) BASDAI individual results across Weeks 0–156 for patients randomised to BKZ 160 mg and 320 mg Q4W at baseline



Full analysis set. Data reported as observed case. VHD: ASDAS >3.5; HD: ASDAS ≥2.1–≤3.5; LD: ASDAS ≥1.3–<2.1; ID: ASDAS <1.3. ASDAS <2.1 includes both ASDAS-LD and ASDAS-ID.

AS: ankylosing spondylitis; ASAS40: Assessment of SpondyloArthritis international Society 40% response; ASDAS: Ankylosing Spondylitis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BKZ: bimekizumab; HD: high disease; ID: inactive disease; IL: interleukin; LD: low disease; OC: observed case; OLE: open-label extension; Q4W: every 4 weeks; SD: standard deviation; VHD: very high disease.

Institutions: [1]Department of Rheumatology, La Paz University Hospital, IdiPaz, Madrid, Spain; [2]University of Bielefeld, Klinikum Bielefeld, Bielefeld, Germany; [3]UCB Pharma, Brussels, Belgium; [4]UCB Pharma, Raleigh, North Carolina, USA; [5]UCB Pharma, Colombes, France; [6]Rheumazentrum Ruhrgebiet Herne, Ruhr-University Bochum, Bochum, Germany.

References: [1]van der Heijde D. Ann Rheum Dis 2020;79:595–604.[2]Gensler L. Arthritis Rheumatol 2021;73(suppl 10):0491; [3]Smolen J. Ann Rheum Dis 2018;77:3–17. **Author Disclosures: VNC:** Speakers bureau from AbbVie, Eli Lilly, Janssen, MSD, Novartis, Pfizer, UCB Pharma; Consultancy from AbbVie, Eli Lilly, MSD, Novartis, Pfizer, UCB Pharma; Research grants from AbbVie and Novartis; **MR:** Speakers bureau from AbbVie, BMS, Boehringer Ingelheim, Chugai, Eli Lilly, Janssen, Novartis, Pfizer, UCB Pharma; paid instructor for Janssen, Novartis, UCB Pharma; consultant of AbbVie, Novartis, UCB Pharma; **XB:** Speakers bureau from AbbVie, BMS, Chugai, Eli Lilly, Galapagos, Gilead, MSD, Novartis, Pfizer, UCB Pharma; paid instructor for AbbVie, BMS, Chugai, Eli Lilly, Galapagos, Gilead, MSD, Novartis, Pfizer, UCB Pharma; consultant of AbbVie, BMS, Chugai, Eli Lilly, Galapagos, Gilead, MSD, Novartis, Pfizer, UCB Pharma; **CF, MO, NdP, VT:** Employees of UCB Pharma. **Acknowledgements:** 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 Pharma, for publication coordination; Jane Spingardi, DPhil, Costello Medical, UK for medical writing and editorial assistance and the Costello Medical Design Team for design support. This study was funded by UCB Pharma. All costs associated with development of this presentation were funded by UCB Pharma.