Validation of a Disease Activity Index for Psoriatic Arthritis Based on 44 Joints (DAPSA44) Using Data from Phase 3 Clinical Trials of Bimekizumab in Psoriatic Arthritis

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Objective

To validate the use of the DAPSA44 as a composite measure to assess disease activity due to peripheral arthritis, using data from phase 3 randomised controlled trials of bimekizumab (BKZ), a monoclonal IgG1 antibody that inhibits interleukin (IL)-17F in addition to IL-17A, in psoriatic arthritis (PsA).

Background

- The original DAPSA is a composite measure used to assess disease activity due to peripheral arthritis in PsA, based on the 66 swollen joint count (SJC66) and 68 tender joint count (TJC68).[1]
- For situations where only 44 swollen (SJC44) and tender joint counts (TJC44) are collected, DAPSA44 has been developed and validated for use in spondyloarthritis (SpA) using data from an observational cohort study in patients with PsA, axial spondyloarthritis (axSpA) and peripheral SpA.[2,3]
- However, DAPSA44 has not yet been validated against original-DAPSA in the context of clinical trials.

Methods

- This analysis used data from patients with active PsA randomised to subcutaneous BKZ 160 mg every 4 weeks (Q4W) or placebo in Weeks 0–16 (double-blind period) of the phase 3 trials, BE OPTIMAL (NCT03895203; biological disease-modifying antirheumatic drug-naïve patients) and BE COMPLETE (NCT03896581; patients with intolerance/inadequate response to tumour necrosis factor inhibitors).[4,5] Patients randomised to adalimumab in BE OPTIMAL were excluded.
- DAPSA44 was calculated using the original-DAPSA formula, with a 1.3× conversion factor applied to SJC44/TJC44:
  - (1.3 × SJC44) + (1.3 × TJC44) + Patient Global Assessment (PGA) + patient pain assessment + high-sensitivity C-reactive protein (hs-CRP; mg/dL).[1,3]
- Agreement between continuous DAPSA44 and original-DAPSA was assessed using a Bland-Altman plot and absolute agreement intra-class correlation coefficient (ICC). An ICC value of >0.90 indicates 'excellent' agreement between instruments.[3]
- Agreement between disease activity states for DAPSA44 vs original-DAPSA was assessed using the weighted kappa coefficient.
- To assess clinical trial discrimination, mean change from baseline and proportion of patients achieving different disease states according to DAPSA44 and original-DAPSA at Week 16 are reported by treatment group.
  - Discrimination between treatment groups was calculated using standardised mean differences (SMD) for continuous scores and odds ratios (ORs) for disease states.
- All data are reported as observed case (OC), except for OR (non-responder imputation; NRI).

Results

- Data pooled from 1,112 patients across BE OPTIMAL and BE COMPLETE were included in this analysis (placebo: n=414; BKZ: n=698). Baseline DAPSA scores and components were similar between treatment groups (**Table 1**).

Construct validity assessment

- The Bland-Altman plot revealed minimal differences between DAPSA44 and original-DAPSA and consistent agreement across the range of measurements (**Figure 1**).
- The agreement between continuous DAPSA44 and original-DAPSA at Week 16 was excellent, with an ICC (95% confidence interval [CI]) of 0.98 (0.98, 0.98; n=1,061). ICC remained consistent when stratified by oligo/polyarthritis and distal interphalangeal joint (DIP) involvement at Week 16:
  - Oligoarthritis (<5 SJC; n=756): 0.98 (0.98, 0.98); polyarthritis (≥5 SJC; n=305): 0.96 (0.95, 0.97).
  - 0 DIP (n=937): 0.98 (0.98, 0.99); ≥1 DIP (n=124): 0.97 (0.92, 0.99).
- The agreement between DAPSA44 and original-DAPSA disease activity states at Week 16 was almost perfect, indicated by a weighted kappa of 0.92 (95% CI: 0.90, 0.93; **Table 2**).

Clinical trial discrimination

- Change from baseline values were comparable between DAPSA44 and original-DAPSA within the treatment groups (**Figure 2**).
  - SMDs for BKZ vs placebo (DAPSA44: 0.87; original-DAPSA: 0.85) corresponded to large discrimination between treatment groups for both instruments.
- The distribution of patients across different disease states at Week 16 was similar between DAPSA44 and original-DAPSA within the treatment groups (**Figure 3A**).
  - ORs for BKZ vs placebo indicated a larger proportion of patients receiving BKZ compared with placebo achieved remission (REM) or REM/low disease activity (LDA), with 95% CIs showing clear discrimination between treatment groups, similar for both instruments (OR >1; **Figure 3B**).

Conclusions

This analysis supports the validation of DAPSA44 as a comparable instrument to original-DAPSA for assessing disease activity due to peripheral arthritis. These findings, together with prior results from observational and post hoc analyses in axSpA,[2,3,6] suggest that DAPSA44 may offer a valid alternative composite measure in axSpA clinical trials where limited joint counts are common. While dedicated validation in axSpA remains necessary, the current results from PsA studies provide good evidence on the measurement properties of DAPSA44.

Summary

We validate the use of **DAPSA44** against original-DAPSA to assess **peripheral arthritis** using **phase 3 clinical trial data** of bimekizumab in patients with PsA

DAPSA44 = (1.3 × SJC44) × (1.3 x TJC44) + PGA + pain + hs-CRP (mg/dL)

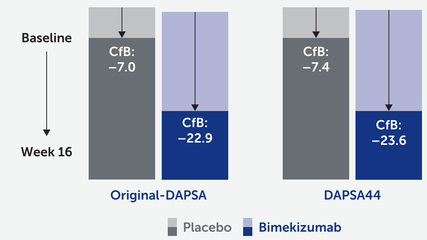
Using Week 0–16 data in bimekizumab- and placebo-randomised patients, we found:

Strong agreement between continuous DAPSA44 and original-DAPSA, and their disease states, consistent across **subgroups** for joint involvement at Week 16:

Oligo/polyarthritis
(<5 SJC vs ≥5 SJC)

DIP involvement
(0 DIP vs ≥1 DIP)

DAPSA44 showed **good discrimination** between bimekizumab vs placebo, **comparable** to original-DAPSA



This analysis validates the use of **DAPSA44 as a comparable instrument** to **original-DAPSA** for assessing disease activity due to **peripheral arthritis**

Table 1 DAPSA score and DAPSA components at baseline

Mean (SD), unless otherwise stated	PBO n=414	BKZ 160 mg Q4W n=698
Original-DAPSA	40.3 (21.1)	38.8 (19.5)[a]
DAPSA44	40.9 (20.5)	39.6 (19.4)[a]
SJC (0–66)	9.7 (7.6)	9.2 (6.7)
TJC (0–68)	17.8 (13.1)	17.4 (12.5)
SJC (0–44)	8.0 (5.8)	7.7 (5.3)[a]
TJC (0–44)	13.6 (9.4)	13.5 (9.2)[a]
PGA	60.0 (23.1)	56.8 (23.3)[a]
Patient pain assessment	58.4 (23.8)	55.4 (24.3)[a]
hs-CRP, mg/L		
Geometric mean (geometric CV, %)	4.5 (2.5)	4.1 (2.6)
Median (Q1, Q3)	4.6 (1.7, 11.8)	4.1 (1.5, 12.3)

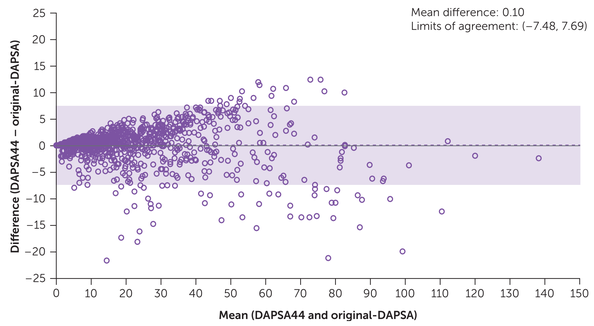
Pooled randomised set (N=1,112). PGA and patient pain assessment were measured on a 0–100 scale and converted to a 0–10 scale for the calculation of original-DAPSA and DAPSA44. **[a]** n=697.

Table 2 Agreement between DAPSA44 and original-DAPSA disease activity states at Week 16 (OC)

		Original-DAPSA[a] REM	LDA	MDA	HDA	DAPSA44 total, n
DAPSA44[a]	REM	**136 (91.3%)**	14 (4.4%)	1 (0.3%)	0	151
	LDA	13 (8.7%)	**289 (90.0%)**	15 (5.1%)	0	317
	MDA	0	18 (5.6%)	**254 (86.7%)**	15 (5.0%)	287
	HDA	0	0	23 (7.8%)	**283 (95.0%)**	306
Original-DAPSA total, n		149	321	293	298	1,061
Weighted Kappa (95% CI) for DAPSA44 vs original-DAPSA:[b] **0.92 (0.90, 0.93)**						

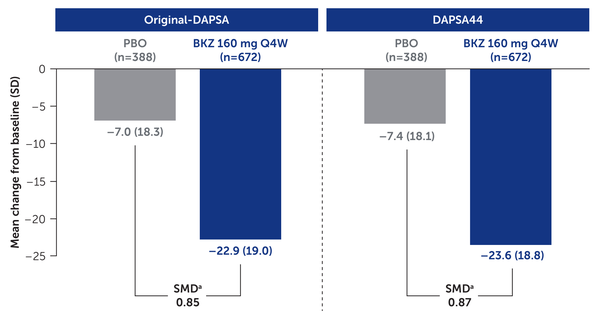
Pooled randomised set (N=1,112). Disease activity states are defined as DAPSA score ≤4 (REM), >4–≤14 (LDA), >14–≤28 (MDA) or >28 (HDA). **[a]** Values in the table show number of patients (n) within both respective disease activity states, and percentage agreement (%) within original-DAPSA disease activity states. The 13 patients who achieved DAPSA44 LDA but original-DAPSA REM had Week 16 scores close to the threshold for REM and LDA (mean scores, original-DAPSA: 3.7; DAPSA44: 4.3). One patient with primarily swollen or tender DIP and PIP joints achieved DAPSA44 REM but had original-DAPSA MDA; **[b]** A weighted kappa value of ≥0.91 indicates 'almost perfect' agreement.[3]

Figure 1 Bland-Altman plot of agreement between DAPSA44 and original-DAPSA at Week 16 (OC)



Pooled randomised set (N=1,112). The shaded area shows the limits of agreement which are calculated by: *mean difference +/− (1.96 × SD)*. The dashed line represents the mean difference.

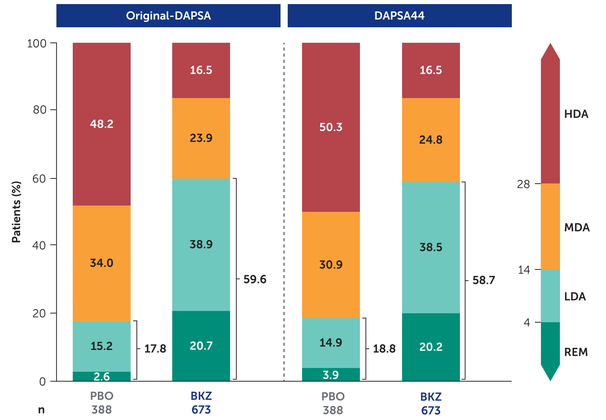
Figure 2 Mean change from baseline in DAPSA scores at Week 16, by treatment group (OC)



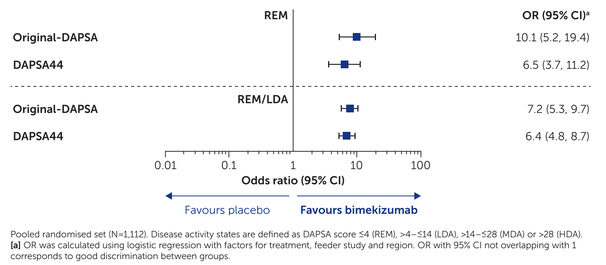
Pooled randomised set (N=1,112). **[a]** SMD was calculated as the difference between the group score means divided by the pooled standard deviation, where a value of >0.8 corresponds to large discrimination between known groups.[2,3]

Figure 3 DAPSA disease activity states at Week 16, by treatment group

A) Proportion of patients achieving DAPSA disease activity states at Week 16, by treatment group (OC)



B) OR of patients achieving REM or REM/LDA at Week 16 with bimekizumab vs placebo (NRI)



Pooled randomised set (N=1,112). Disease activity states are defined as DAPSA score ≤4 (REM), >4–≤14 (LDA), >14–≤28 (MDA) or >28 (HDA). **[a]** OR was calculated using logistic regression with factors for treatment, feeder study and region. OR with 95% CI not overlapping with 1 corresponds to good discrimination between groups.

axSpA: axial spondyloarthritis; **BKZ:** bimekizumab; **CfB:** change from baseline; **CI:** confidence interval; **CV:** coefficient of variation; **DAPSA:** Disease Activity index for PSoriatic Arthritis; **DAPSA44:** DAPSA based on 44 joints; **DIP:** distal interphalangeal joint; **HDA:** high disease activity; **hs-CRP:** high-sensitivity C-reactive protein; **ICC:** intra-class correlation coefficient; **IL:** interleukin; **LDA:** low disease activity; **MDA:** moderate disease activity; **NRI:** non-responder imputation; **OC:** observed case; **OR:** odds ratio; **PBO:** placebo; **PIP:** proximal interphalangeal joint; **PsA:** psoriatic arthritis; **PGA:** Patient Global Assessment; **Q1/Q3:** first/third quartile; **Q4W:** every 4 weeks; **REM:** remission; **SD:** standard deviation; **SJC:** swollen joint count; **SMD:** standardised mean differences; **SpA:** spondyloarthritis; **TJC:** tender joint count.

References: [1]Schoels M. Ann Rheum Dis 2010;69:1441–7; [2]Capelusnik D. Ann Rheum Dis 2025;84:1324–34; [3]Capelusnik D. Ann Rheum Dis 2025;85:297–307; [4]McInnes IB. Lancet 2023;401:25–37; [5]Merola JF. Lancet 2023;401:38–48; [6]Ramiro S. RMD Open 2025;11:e005969. **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **SR**, **DC**, **CLM**, **DvdH**, **JSS**, **DA**, **BI**, **VT**, **AM**; Drafting of the publication, or reviewing it critically for important intellectual content: **SR**, **DC**, **CLM**, **DvdH**, **JSS**, **DA**, **BI**, **VT**, **AM**; Final approval of the publication: **SR**, **DC**, **CLM**, **DvdH**, **JSS**, **DA**, **BI**, **VT**, **AM**. **Author Disclosures: 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. **DC:** Nothing to disclose. **CLM:** Speakers bureau for AbbVie, Eli Lilly, Janssen, Johnson & Johnson, MSD, Novartis and UCB; consultant for Eli Lilly, Johnson & Johnson, Novartis and UCB; grant/research support from AbbVie, Eli Lilly, Novartis and UCB. **DvdH:** Consultant for Alfasigma, BMS, GreyWolf Therapeutics, Janssen, Takeda and UCB; stocks of Altis Medicines Inc; associate editor Annals of the Rheumatic Diseases and director of Imaging Rheumatology BV. **JSS:** Research grants from AbbVie, AstraZeneca, Eli Lilly and Company, Novartis and Roche; honoraria from AbbVie, Amgen, AstraZeneca, Astro, Bristol Myers Squibb, Celgene, Celltrion, Chugai, Eli Lilly and Company, Gilead, ILTOO, Janssen, Merck Sharp & Dohme, Pfizer, R-Pharma, Roche, Samsung, Sandoz, Sanofi and UCB; editor of Annals of the Rheumatic Diseases; co-editor of Rheumatology 7E/8E; convenor of EULAR Task Forces and T2T Task Forces. DA: Grants from Janssen and Lilly; speakers' bureau/consultancy from Advanz, AstraZeneca, Janssen/Johnson & Johnson, Mitsubishi Tanabe, Sanofi and UCB. **BI:** Shareholder of AbbVie, GlaxoSmithKline and UCB; employee of UCB. **VT:** Employee and shareholder of UCB. **AM:** Grant/research support from AbbVie, Biogen, MSD, Pfizer and UCB; consultant for AbbVie, Alfasigma, Eli Lilly, Johnson & Johnson, MSD, Novartis, Pfizer and UCB. **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 also thank Celia Menckeberg, PhD, of UCB, for editorial review during poster development and publication coordination, Syauqina Fakhira, MSc, of Costello Medical, Cambridge, 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.

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