Σύγχρονη διαχείριση σε PsA και axSpA, βασισμένη σε real-world δεδομένα και κλινική εμπειρία διπλής αναστολής των IL-17A και IL-17F

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Disclosures

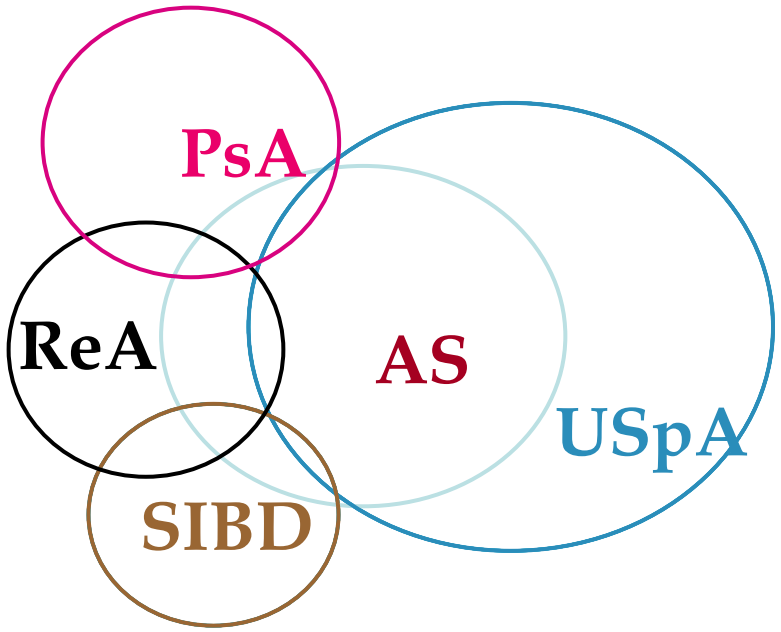
- COI: speaker has received honoraria/speaker fees from: Pfizer, UCB, Abbvie, Lilly, Novatis, Janssen, Farran, Boheringer-Ingelheim, Vianex,

Τιμητική αμοιβή από UCB για την συγκεκριμένη ομιλία

Spondyloarthritis

Psoriatic Arthritis

SpA: Overlapping entities



AS **Ankylosing spondylitis**

PsA **Psoriatic arthritis**

USpA **Undifferentiated SpA**

ReA **Reactive arthritis**

SIBD **SpA με IBD**

Davis JC & Mease PJ Semin Arthr & Rheum 2008

The Prevalence of EMMs and MSK Manifestations Varies Across SpA Subtypes, Yet There Are Many Overlapping Clinical Features[1]

1 | UVEITIS

axSpA*[2]	PsA†[3]
30–40%	7–18%

2 | IBD

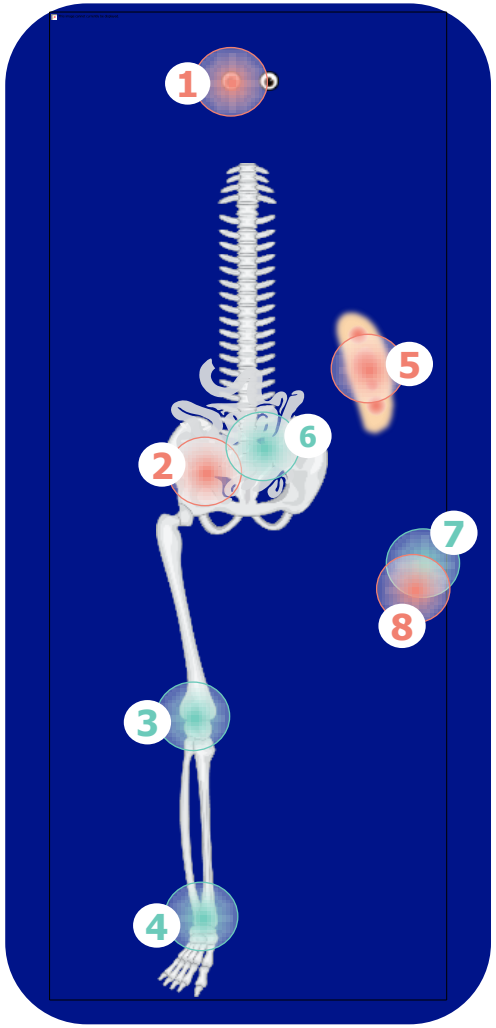
axSpA[2]	PsA[4,5]
5–10%	0.5–4%

3 | PERIPHERAL ARTHRITIS

axSpA[6]	PsA[7–11]
36%	Up to 100%

4 | ENTHESITIS

axSpA[12]	PsA[13,14]
~25%	35–57%



5 | PSORIASIS

axSpA[15,16]	PsA[17]
~10–27%	92%

6 | DACTYLITIS

axSpA[20]	PsA[21]
~6%	16–48%

7 | NAIL DISEASE

axSpA	PsA[22]
–	Up to 80%

■ Extra-musculoskeletal manifestations (EMMs)
■ Musculoskeletal (MSK) manifestations

*Data for acute anterior uveitis.[2]
†Data for anterior/posterior uveitis.[3] ‡Sacroiliitis on MRI.[18]

1. McGonagle DG, et al. Ann Rheum Dis. 2019;78(9):1167–1178. 2. Taurog JD, et al. N Engl J Med. 2016;374(26):2563–2574. 3. Chen H and Chou C. Curr Rheumatol Rev. 2008;4:111–114. 4. Williamson L, et al. J Rheumatol. 2004;31(7):1469–1470. 5. López-Medina C, et al. RMD Open. 2021;7(1):e001450. 6. López-Medina C, et al. Arthritis Res Ther. 2019;21(1):139. 7. Moll JMH and Wright V. Semin Arthritis Rheum. 1973;3(1):55–78. 8. Torre Alonso JC, et al. Br J Rheumatol. 1991;30(4):245–250. 9. Helliwell PS and Taylor WJ. Ann Rheum Dis. 2005;64(Suppl II):ii3–ii8. 10. Gladman DD. Ann Rheum Dis. 2006;65(Suppl 3):iii22–iii24. 11. Acosta Felquer ML and FitzGerald O. Clin Exp Rheumatol. 2015;33(Suppl 93):S26–S30. 12. Mease PJ, et al. ACR Open Rheumatol. 2020;2(7):449–456. 13. D'Agostino MA, et al. Arthritis Rheum. 2003;48(2):523–533. 14. Kaeley GS, et al. Semin Arthritis Rheum. 2018;48(1):35–43. 15. Navarro-Compán V, et al. Ann Rheum Dis. 2021;80(12):1511–1521. 16. Lucasson F, et al. RMD Open. 2022;8:e001986. 17. Kane D, et al. Rheumatology (Oxford). 2003;42(12):1460–1468. 18. van den Berg R, et al. Rheumatology (Oxford). 2013;52(8):1492–1499. 19. Battistone MJ, et al. Skeletal Radiol. 1999;28(4):196–201. 20. de Winter JJ, et al. Arthritis Res Ther. 2016;18(1):196. 21. Helliwell PS, et al. J Rheumatol. 2005;32(9):1745–1750. 22. Sobolewski P, et al. Reumatologia. 2017;55(3):131–135.

Patients With SpA Have Many Overlapping Comorbidities

Prevalence of Comorbidities

OSTEOARTHRITIS

PsA[1]
22%

FIBROMYALGIA

axSpA[10]	PsA[11,12]
16%	34–53%

CVD

axSpA[2,3]	PsA[3,4]
1–12%	~8–19%

OBESITY

axSpA[13]	PsA[14,15]
14%	28–33%

OSTEOPOROSIS

axSpA[5,6]	PsA[7,8]
12–50%	~12–27%

DEPRESSION/ANXIETY

axSpA[3,16,17]	PsA[3,18]
11–38%	12–29%

HS

axSpA[9]
9%

Many of these clinical features are exacerbated **by chronic inflammation associated with SpA pathobiology**[2,3,13]

Patients with SpA who have **multiple comorbidities tend to have higher disease activity**; >50% of patients with PsA have at least one comorbidity, with up to 40% having more than three[3,19]

Due to the interplay between comorbidities and disease activity, it is **crucial to manage comorbidities effectively to optimise treatment outcomes**[3,12,15]

1. Charlton RA, et al. J Rheumatol. 2021;48(6):841–846. 2. Duruöz MT, et al. Rheumatol Int. 2024;44(4):631–642. 3. Bosch P, et al. Best Pract Res Clin Rheumatol. 2023;37(3):101857. 4. Husted JA, et al. J Rheumatol. 2013;40(8):1349–1356. 5. Moltó A and Nikiphorou E. Front Med (Lausanne). 2018;5:62. 6. Ramírez J, et al. Semin Arthritis Rheum. 2018;48(1):44–52. 7. Martínez-Vidal MP, et al. Reumatol Clin (Engl Ed). 2024;20(1):8–13. 8. Takami K, et al. Mod Rheumatol. 2024;34(6):1252–1257. 9. Rondags A, et al. Semin Arthritis Rheum. 2019;48(4):611–617. 10. Jones GT, et al. Rheumatol Int. 2020;40(10):1581-1591. 11. Ulus Y, et al. Adv Rheumatol. 2019;60(1):1. 12. Magrey MN, et al. Arthritis. 2013;2013:762921. 13. Guła Z, et al. Rheumatol Int. 2024;44(12):2817–2828. 14. Queiro R, et al. Medicine (Baltimore). 2019;98(28):e16400. 15. Jafri K, et al. Arthritis Care Res (Hoboken). 2017;69(1):51–57. 16. Redeker I, et al. Arthritis Res Ther. 2020;22(1):210. 17. Reddy KN, et al. Eur J Rheumatol. 2022;9(1):8–13. 18. Zhao SS, et al. Clin Rheumatol. 2020;39(1):217–225. 19. Karmacharya P, et al. Ther Adv Musculoskelet Dis. 2021;13:1–15.

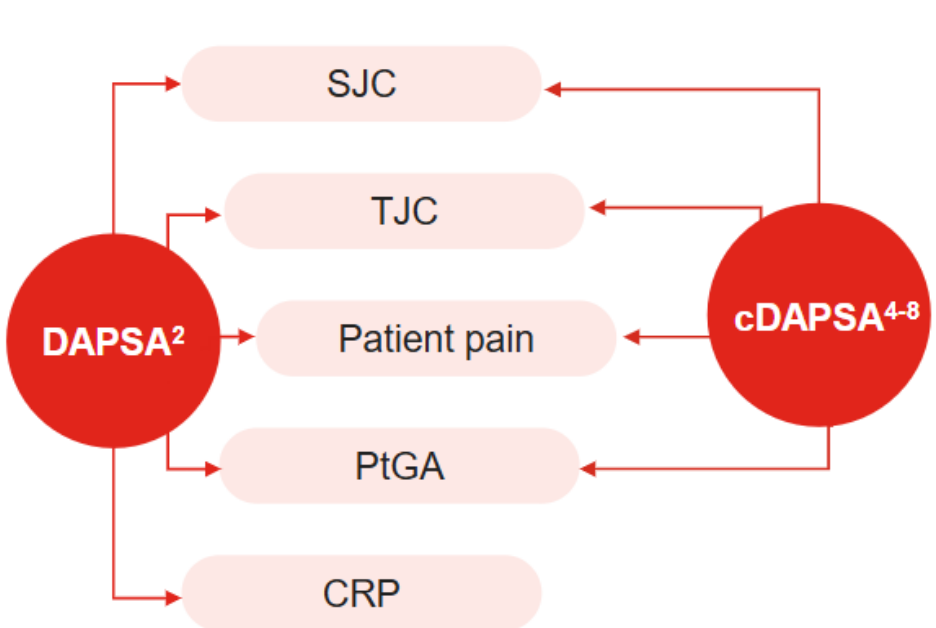
One size fits all?

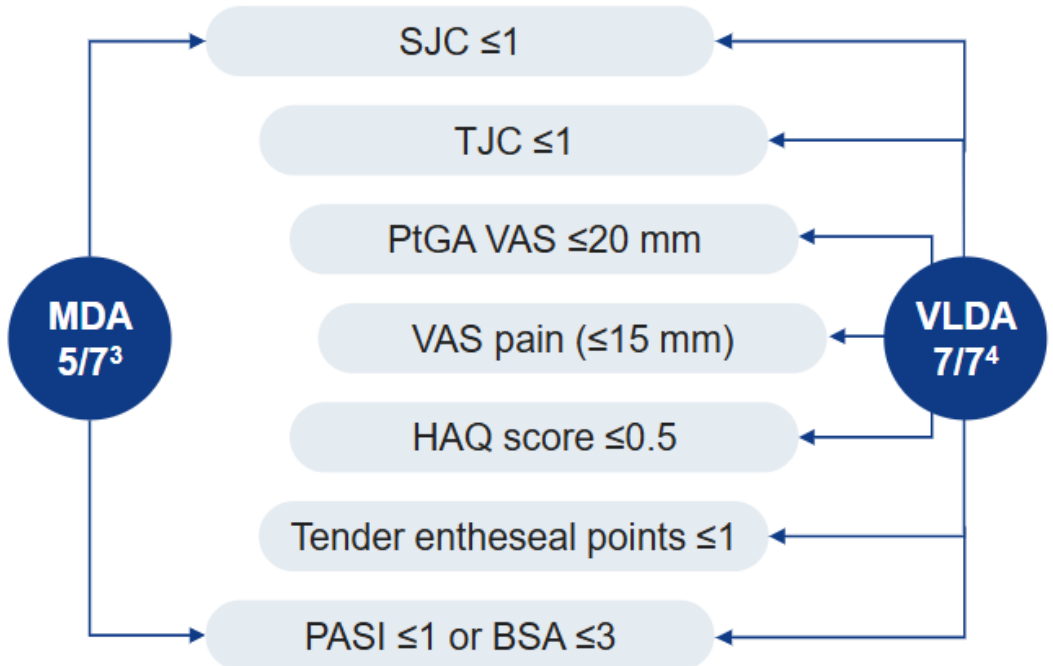
Table 1 Composite indices for PsA

	Synovitis TJC, SJC	Axial involvement	Enthesitis	Dactylitis	Skin	Pain	CRP	PtGA	PGA	Function/QoL
Disease activity										
DAPSA	+					+	+	+		
CPDAI	+	+	+	+	+					+
PASDAS	+		+	+			+	+	+	+
GRACE/AMDF	+				+	+		+		+
PsARC	+							+	+	
PsAJAI	+					+	+	+	+	+
MDA/VLDA	+		+		+	+		+		+

PsA, psoriatic arthritis; *TJC*, Tender Joint Count; *SJC*, Swollen Joint Count; *CRP*, C-reactive protein; *PtGA*, Patient Global Assessment; *PGA*, Physician Global Assessment; *QoL*, quality of life; *DAPSA*, Disease Activity for Psoriatic Arthritis; *PsARC*, Psoriatic Arthritis Response Criteria; *PsAJAI*, Psoriatic Arthritis Joint Activity Index; *GRACE*, Group for Research and Assessment of Psoriasis and Psoriatic Arthritis Composite Exercise; *AMDF*, Arithmetic Mean of Desirability Function; *PASDAS*, Psoriatic Arthritis Disease Activity Score; *CPDAI*, Composite Psoriatic Disease Activity Index; *MDA*, minimal disease activity; *VLDA*, very low disease activity

Gialouri et al. Clin Rheum 2o21


SJC
TJC
DAPSA[2]
Patient pain
cDAPSA[4-8]
PtGA
CRP


SJC ≤1
TJC ≤1
PtGA VAS ≤20 mm
MDA 5/7[3]
VAS pain (≤15 mm)
VLDA 7/7[4]
HAQ score ≤0.5
Tender entheseal points ≤1
PASI ≤1 or BSA ≤3

Treatment Recommendations Set Control of Inflammation as a Primary Goal…

…In PsA

GOAL

EULAR 2023 Recommendations

Abrogation of inflammation is important to achieve treatment goals[1]

Remission should be seen as an "abrogation of inflammation" as the most favourable outcomes are achieved when inflammation is fully controlled[1,2]

TARGET

MDA, VLDA and DAPSA LDA/REM disease activity scores are recommended as valid targets for low disease activity/remission in clinical trials or clinical practice[3–8]

…In axSpA

GOAL

ASAS-EULAR 2022 Recommendations

Emphasise control of inflammation as one important treatment goal[9]

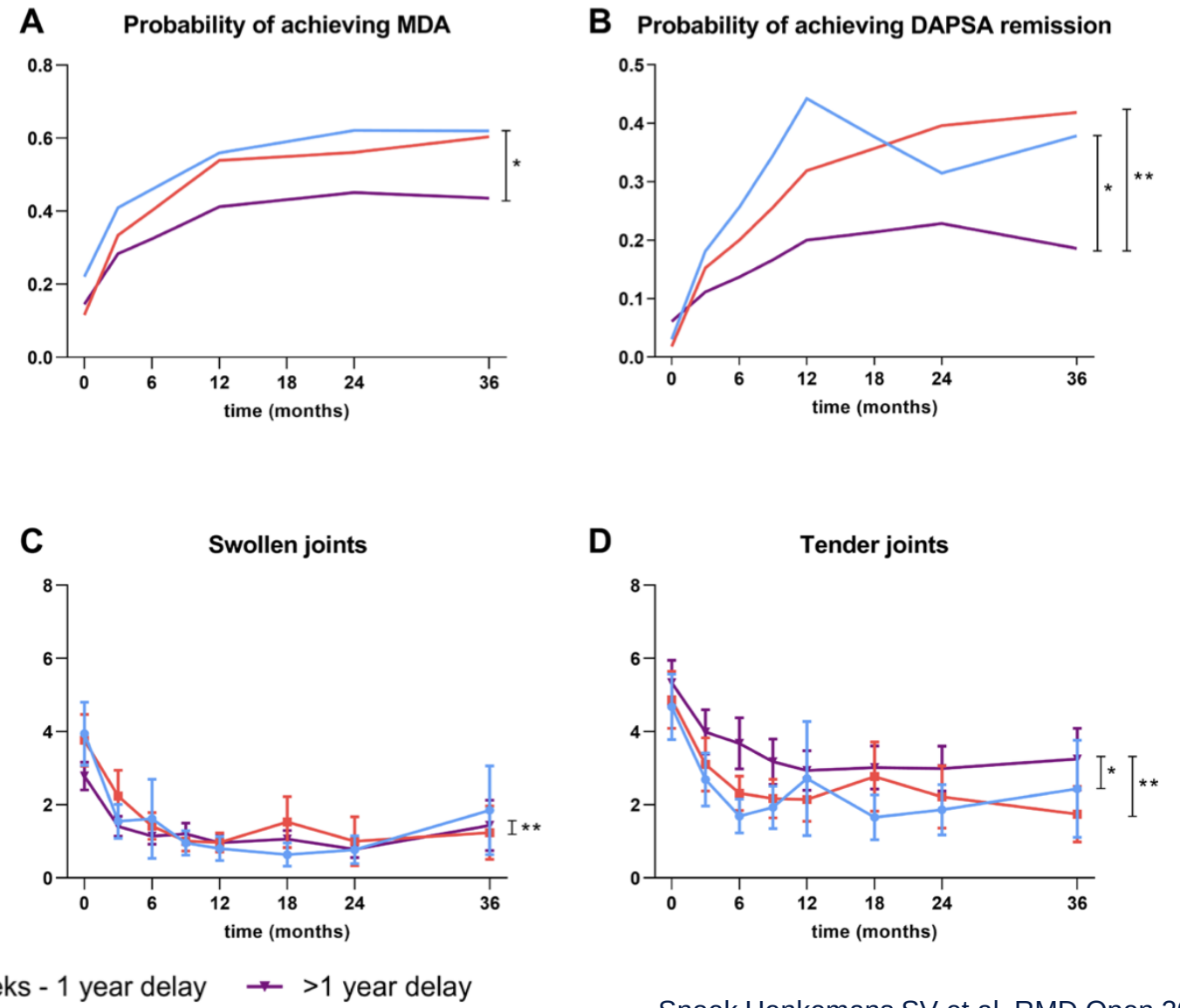
TARGET

ASDAS LDA and ASDAS ID are appropriate targets for low disease activity/remission in clinical trials or clinical practice[9]

1. Gossec L, et al. Ann Rheum Dis. 2024;83(6):706–719. 2. McInnes IB and Gravallese EM. Nat Rev Immunol. 2021;21(10):680–686. 3. Smolen JS, et al. Clin Exp Rheumatol. 2015;33(5 Suppl 93):S48–S50. 4. Coates LC, et al. RMD Open. 2019;5(2):e001002. 5. Gossec L, et al. J Rheumatol. 2018;45(1):6–13. 6. Wervers K, et al. Arthritis Care Res (Hoboken). 2018;70(12):1764–1770. 7. Aletaha D, et al. Ann Rheum Dis. 2017;76(2):418–421. 8. Schoels MM, et al. Ann Rheum Dis. 2016;75(5):811–818. 9. Ramiro S, et al. Ann Rheum Dis. 2023;82(1):19–34.

Window of opportunity: the earlier, the better?

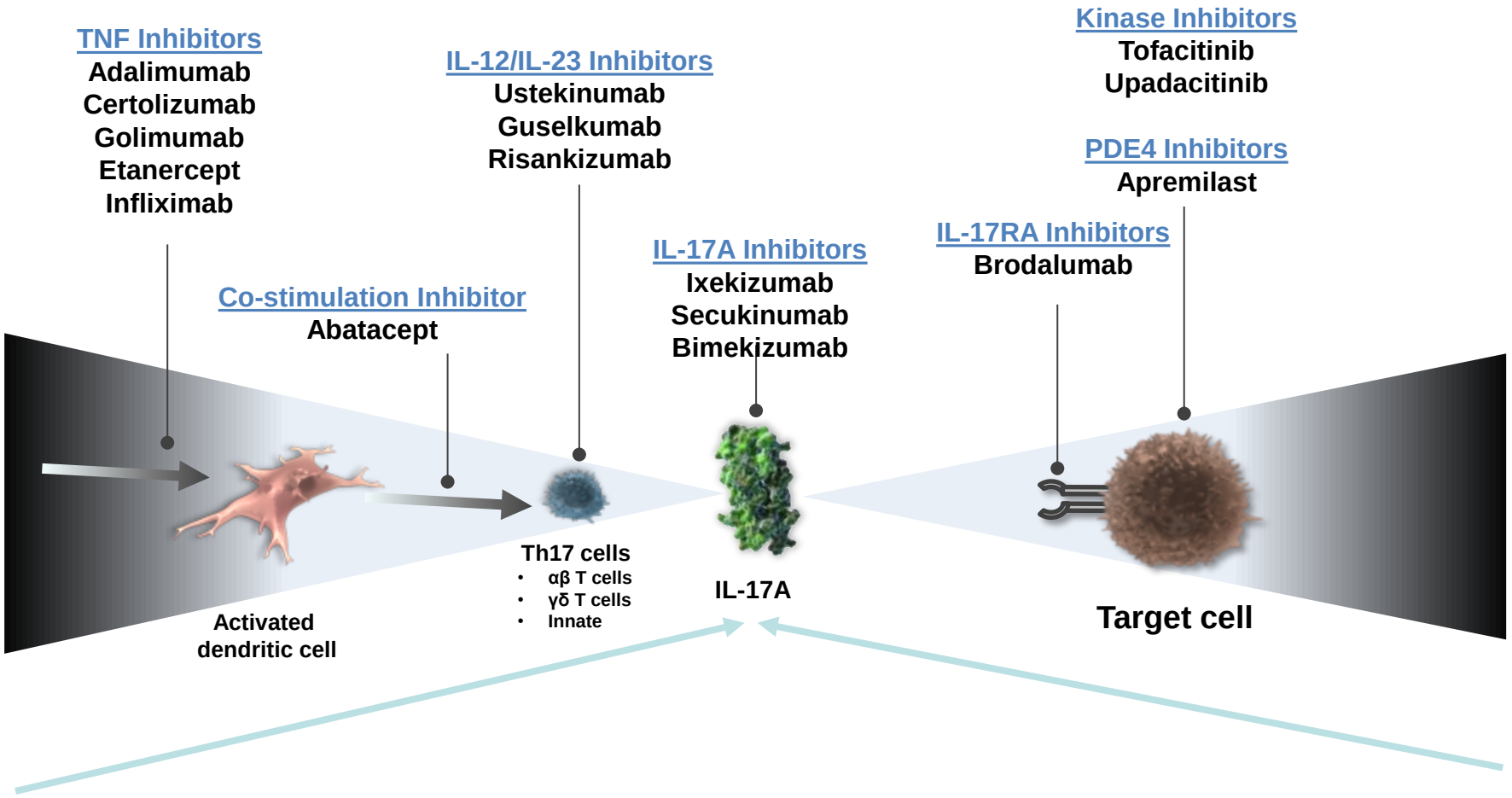
All new PsA in the Dutch cohort
The total delay
*short (<12 weeks)
*intermediate (12 weeks to 1 year)
*long (>1 year)



Snoek Henkemans SV et al, RMD Open 2024

Treatment – the major players

tsDMARDs & Biologics



Nestle F et al. *N Engl J Med.* 2009
Kopf M et al. *Nat Rev Drug Discov.* 2010
Garber K. *Nat Biotechnol.* 2011

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journal homepage: https://www.sciencedirect.com/journal/eular-rheumatology-open

Psoriatic arthritis

Prevalence, outcomes, and predictive factors: a systematic literature review to inform the development of EULAR Points to Consider for the definition of Difficult-to-Manage and Treatment-Refractory psoriatic arthritis

Stephanie R. Harrison[1,2], George E. Fragoulis[3], Xabier Michelena[4], Cristina Macía-Villa[5], Louise Falzon[6], Stefan Siebert[7], Helena Marzo-Ortega[1], Alexandre Sepriano[8,9], Pedro M. Machado[10,11,12,*]

Table 2

Summary of the prevalence of D2M/TR psoriatic arthritis in real-world studies

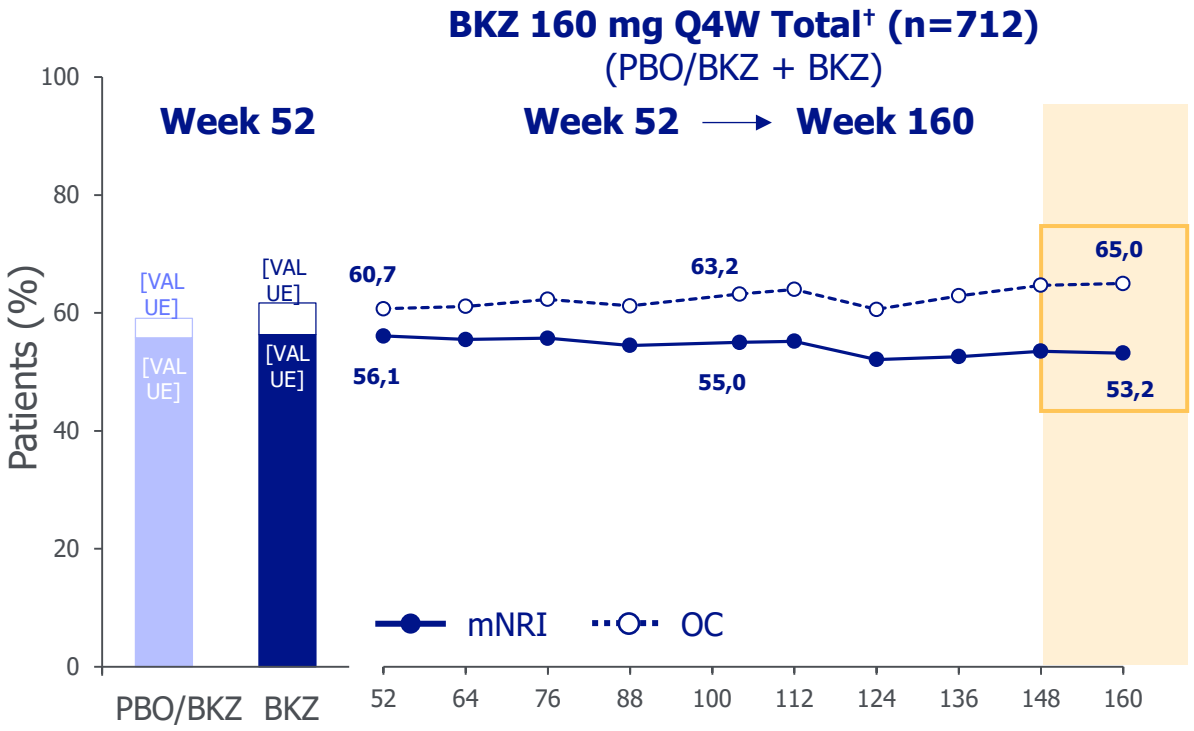
	Criteria used to define D2M/TR			
	Failed 1 b/tsDMARDs		Persistent disease activity	Increased disease activity despite 2 b/tsDMARDs
	Treatment-naïve	Treatment-IR		
Number of studies (number of abstracts included)	8 (0)	52 (3)	15 (2)	7 (2)
Prospective/retrospective (%)	25/75	27/73	40/60	14/86
RoB % (low/moderate/high)	13/62/25	48/42/10	33/60/7	43/57/0
Prevalence (range)	3 mo: 1%-22% 6 mo: 2%-51% 12 mo: 3%-74% 24 mo: 27%-80%		6 mo: 57%-97% (did not achieve DAPSA remission) 12 mo: 57%-70% did not achieve MDA, 90% did not achieve VLDA	8%-34%[a]

b/tsDMARDs, biologic/targeted synthetic disease-modifying antirheumatic drugs; DAPSA, disease activity psoriatic arthritis; D2T, difficult-to-treat; D2M, difficult-to-manage; TR, treatment-refractory; IR, inadequate responders; MDA, minimal disease activity; RoB, risk of bias; VLDA, very low disease activity.

[a] In studies with low risk of bias.

Bimekizumab Achieved ACR50 in ~50% of Patients, Irrespective of Previous Treatment, Sustained From 1 to 3 Years

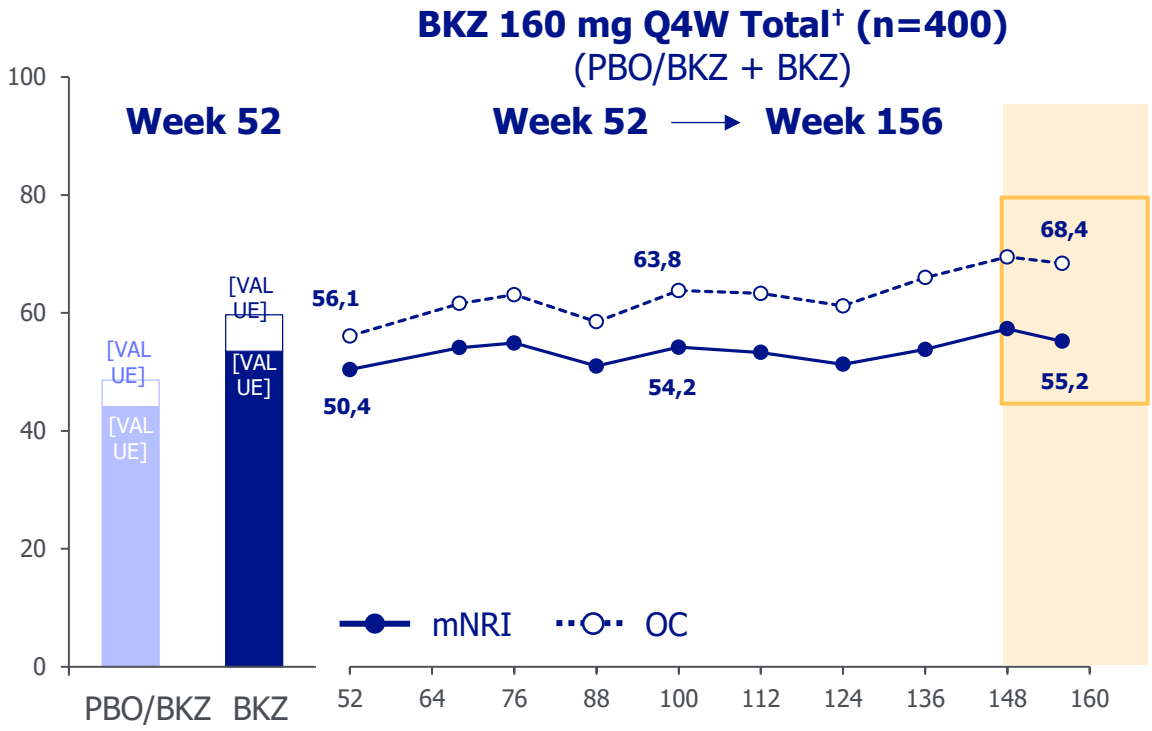
bDMARD-naïve patients[1]



	PBO/BKZ	BKZ
OC n/N:	149/252	234/379
mNRI N:	281	431

BKZ Total (OC), n/N	Year 1	Year 2	Year 3
	383/631	364/576	341/525

TNFi-IR patients[2]



	PBO/BKZ	BKZ
OC n/N:	54/111	138/231
mNRI N:	133	267

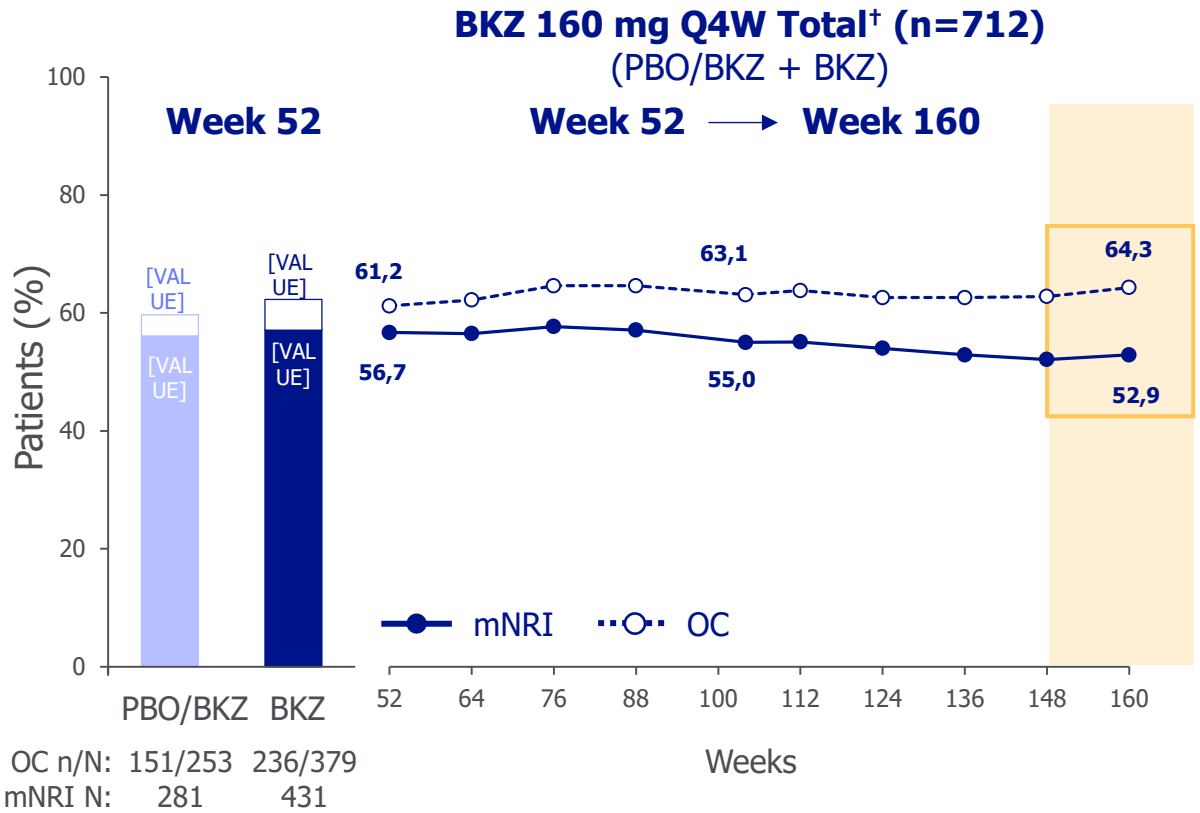
BKZ Total (OC), n/N	Year 1	Year 2	Year 3
	192/342	199/312	199/291

Modified non-responder imputation; observed case. Randomised set.[1,2] Long-term data are highlighted to emphasise the sustained response. The filled portion of the bar indicates mNRI data and the unfilled portion indicates OC data. mNRI considered all visits following discontinuation due to adverse events or lack of efficacy as non-response.[1,2] Data from BE OPTIMAL + OLE (bDMARD-naïve patients) and BE COMPLETE + OLE (TNFi-IR patients).[1,2] BE OPTIMAL: Year 2 data are reported to Week 104 and Year 3 data are reported to Week 160; BE COMPLETE: Year 2 data are reported to Week 100 and Year 3 data are reported to Week 156.[1,2] †BKZ Total group includes BKZ-randomised patients and PBO patients that switched to BKZ at Week 16.[1,2]

1. Adapted from Gossec L, et al. Ann Rheum Dis. 2025;84(Suppl 1):1337.
2. Adapted from McInnes IB, et al. Ann Rheum Dis. 2025;84(Suppl 1):400.

Bimekizumab Achieved MDA in ~50% of Patients, Irrespective of Previous Treatment, Sustained From 1 to 3 Years

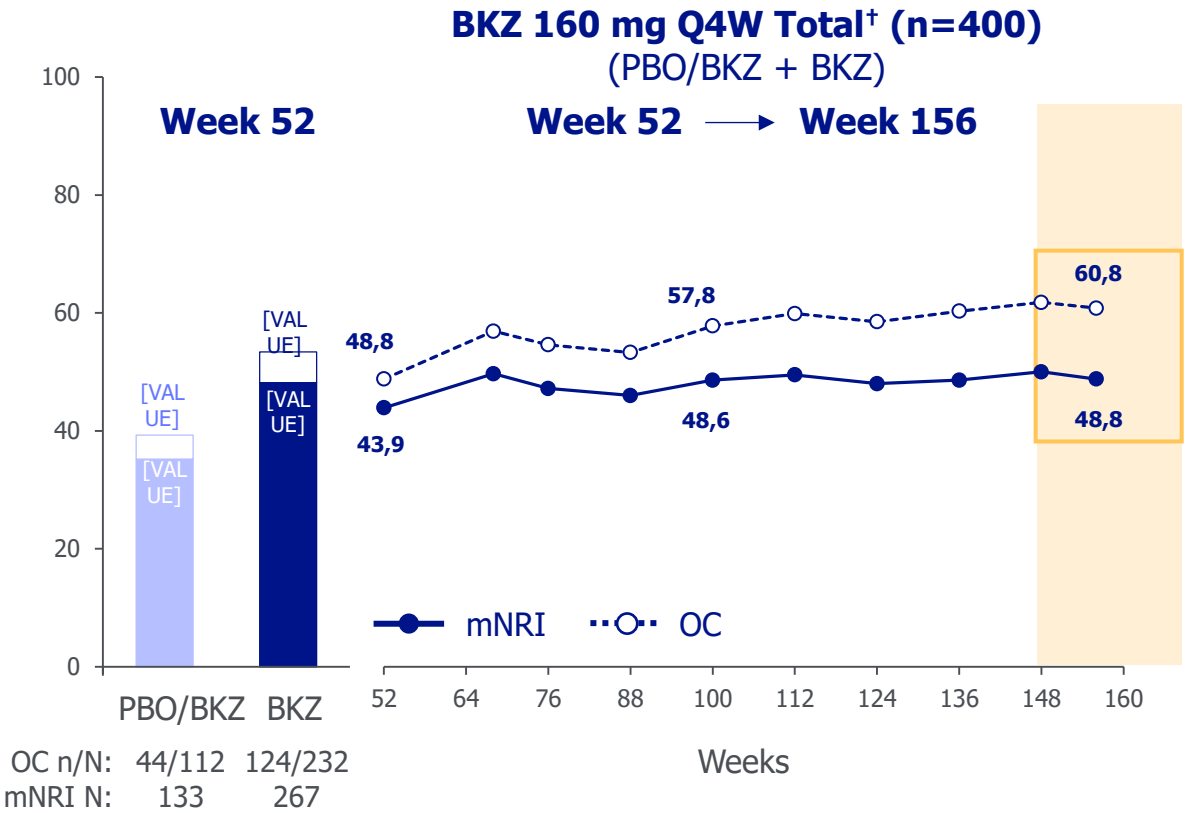
bDMARD-naïve patients[1]



OC n/N: 151/253 236/379
mNRI N: 281 431

	Year 1	Year 2	Year 3
BKZ Total (OC), n/N	387/632	366/580	338/526

TNFi-IR patients[2]



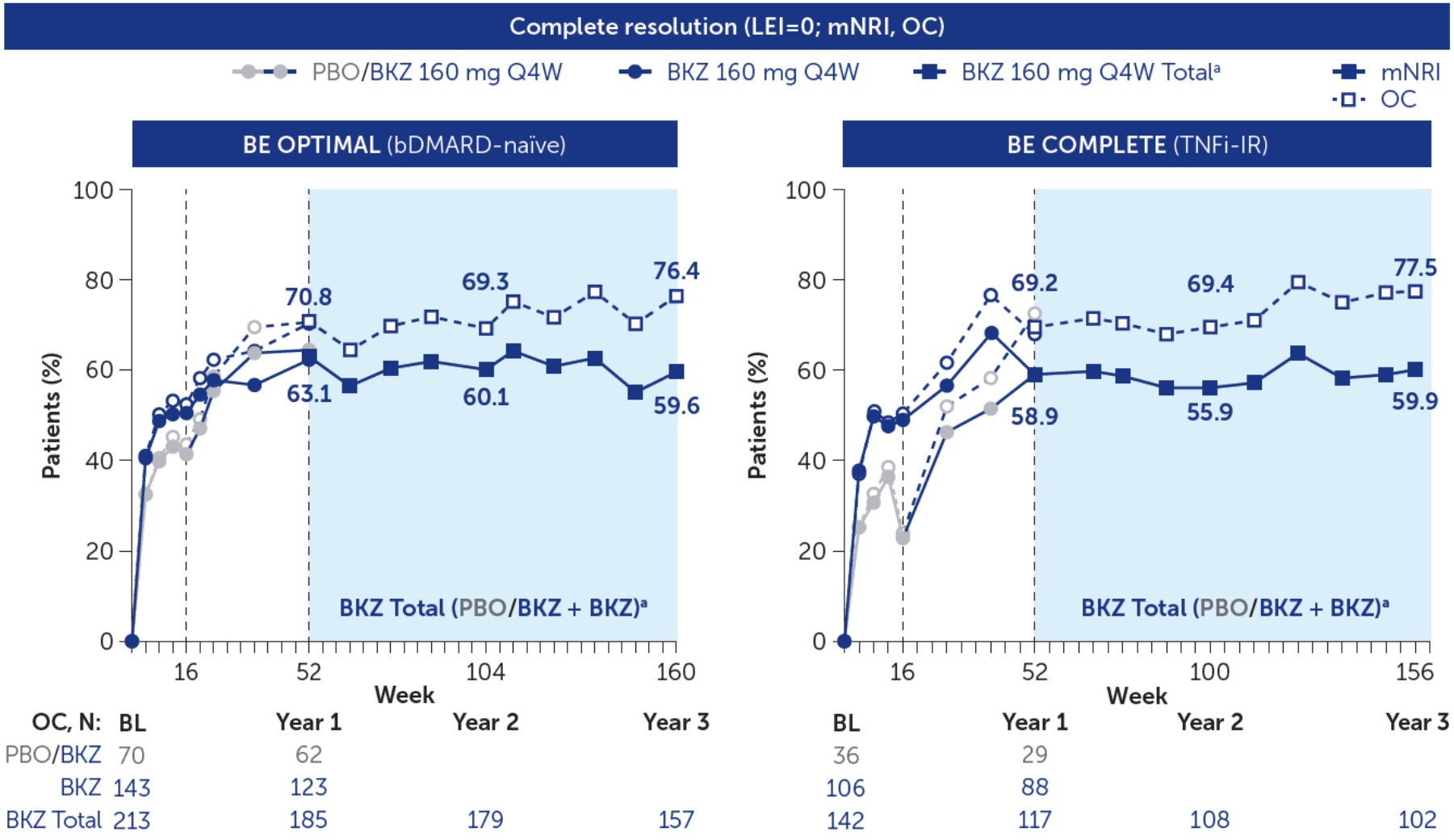
OC n/N: 44/112 124/232
mNRI N: 133 267

	Year 1	Year 2	Year 3
BKZ Total (OC), n/N	168/344	182/315	178/293

Modified non-responder imputation; observed case. Randomised set.[1,2] Long-term data are highlighted to emphasise the sustained response. The filled portion of the bar indicates mNRI data and the unfilled portion indicates OC data. mNRI considered all visits following discontinuation due to adverse events or lack of efficacy as non-response.[1,2] Data from BE OPTIMAL + OLE (bDMARD-naïve patients) and BE COMPLETE + OLE (TNFi-IR patients).[1,2] BE OPTIMAL: Year 2 data are reported to Week 104 and Year 3 data are reported to Week 160; BE COMPLETE: Year 2 data are reported to Week 100 and Year 3 data are reported to Week 156.[1,2] †BKZ Total group includes BKZ-randomised patients and PBO patients that switched to BKZ at Week 16.[1,2]

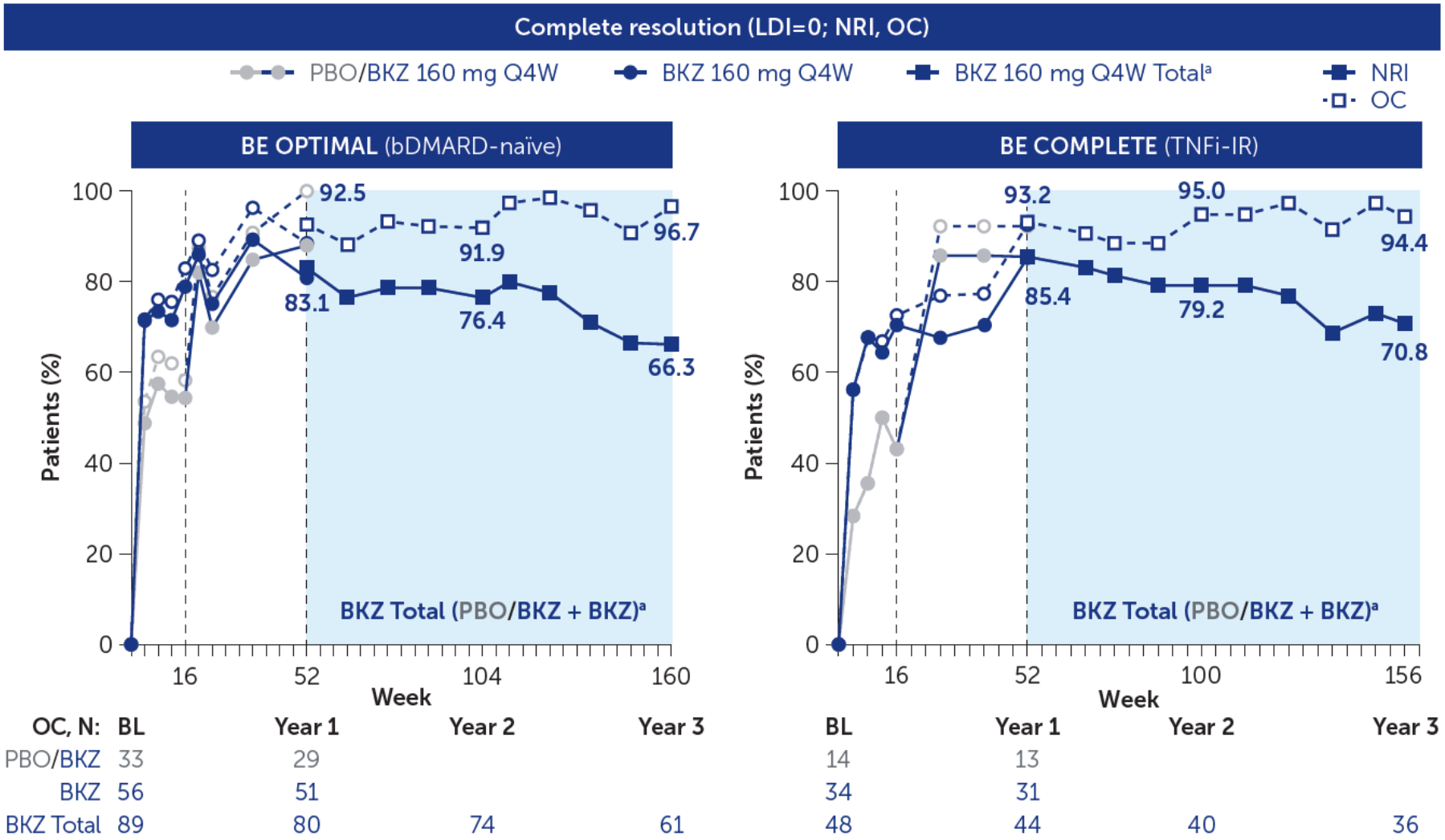
1. Adapted from Gossec L, et al. Ann Rheum Dis. 2025;84(Suppl 1):1337.
2. Adapted from McInnes IB, et al. Ann Rheum Dis. 2025;84(Suppl 1):400.

59.6%–59.9% had Complete Resolution of Enthesitis at Year 3 (mNRI)



Merola JF, et al. ACR 2025. Poster 2365.
Randomized set, in patients with enthesitis at baseline (LEI >0). All patients randomized to PBO received BKZ 160 mg Q4W from Week 16 in BE OPTIMAL and BE COMPLETE (PBO/BKZ).
Data reported to 3 years (Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE). BL N numbers refer to the full population size; N numbers for Years 1, 2, and 3 refer to the number of patients with observations at the respective time point. [a]BKZ 160 mg Q4W Total includes patients originally randomized to PBO up to Week 16. bDMARD: biologic disease-modifying antirheumatic drug; BKZ: bimekizumab; BL: baseline; LEI: Leeds Enthesitis Index; mNRI: modified non-responder imputation; OC: observed case; PBO: placebo; Q4W: every 4 weeks;
TNFI-IR: prior inadequate response or intolerance to tumor necrosis factor inhibitors.

66.3%–70.8% had Complete Resolution of Dactylitis at Year 3 (NRI)

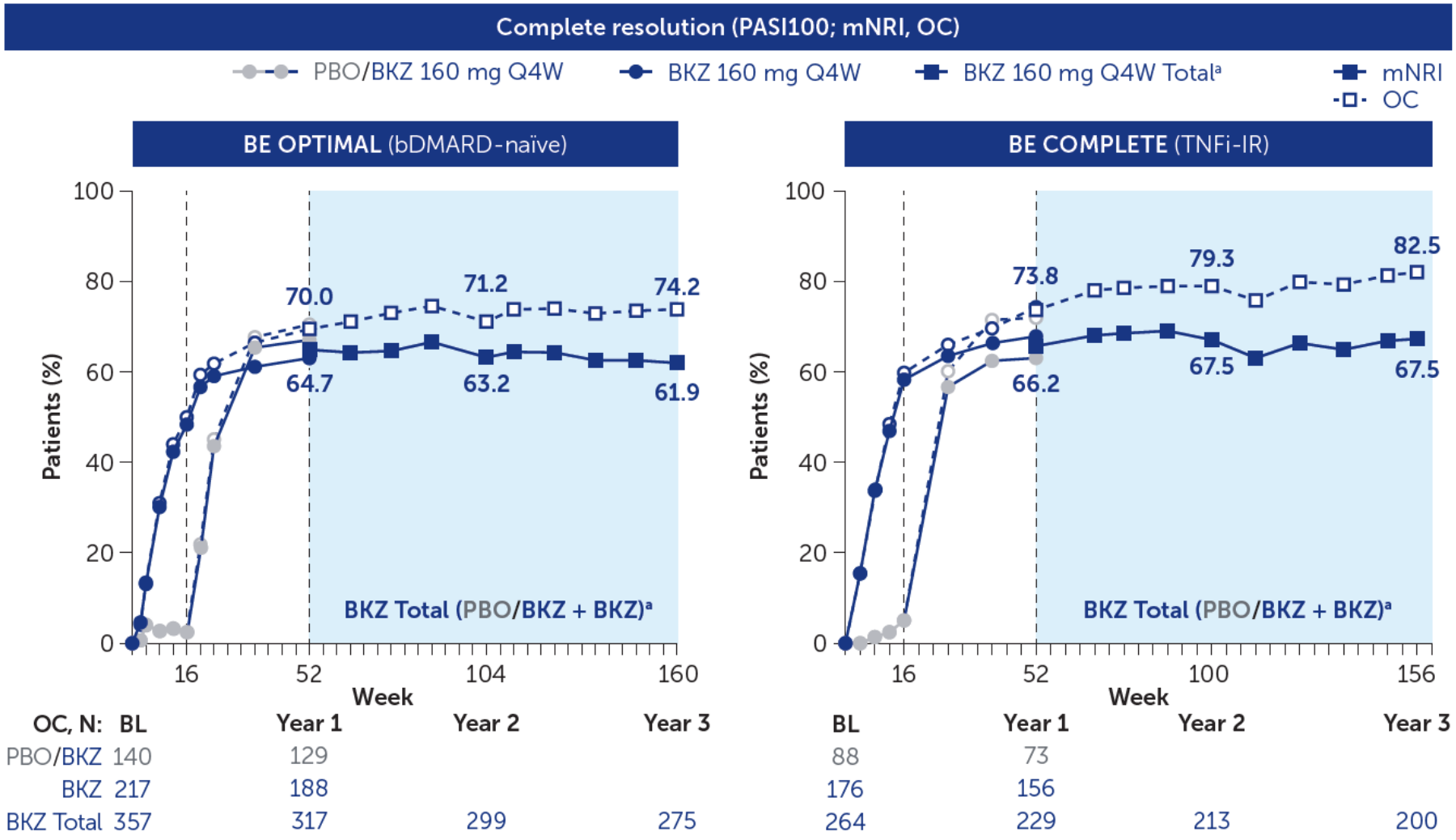


OC, N:	BL	Year 1	Year 2	Year 3
PBO/BKZ	33	29		
BKZ	56	51		
BKZ Total	89	80	74	61

BL	Year 1	Year 2	Year 3
14	13		
34	31		
48	44	40	36

Merola JF, et al. ACR 2025. Poster 2365.
Randomized set, in patients with dactylitis at baseline (LDI >0). All patients randomized to PBO received BKZ 160 mg Q4W from Week 16 in BE OPTIMAL and BE COMPLETE (PBO/BKZ).
Data reported to 3 years (Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE). BL N numbers refer to the full population size; N numbers for Years 1, 2, and 3 refer to the number of patients with observations at the respective time point. [a]BKZ 160 mg Q4W Total includes patients originally randomized to PBO up to Week 16. bDMARD: biologic disease-modifying antirheumatic drug; BKZ: bimekizumab; BL: baseline; LDI: Leeds Dactylitis Index; NRI: non-responder imputation; OC: observed case; PBO: placebo; Q4W: every 4 weeks;
TNFI-IR: prior inadequate response or intolerance to tumor necrosis factor inhibitors.

61.9%–67.5% had Complete Resolution of Skin Psoriasis at Year 3 (mNRI)

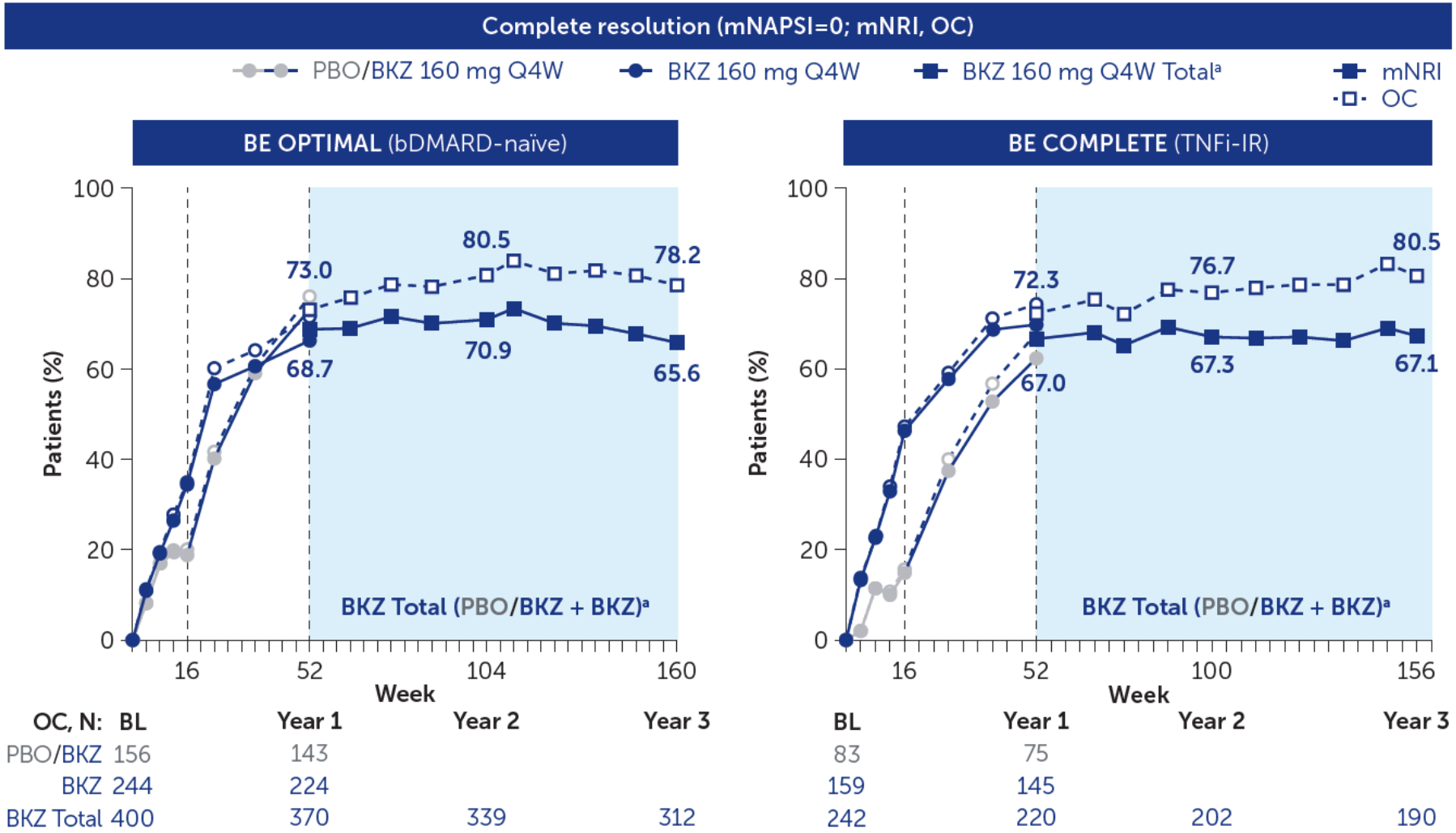


OC, N:	BL	Year 1	Year 2	Year 3
PBO/BKZ	140	129		
BKZ	217	188		
BKZ Total	357	317	299	275

BL	Year 1	Year 2	Year 3
88	73		
176	156		
264	229	213	200

Merola JF, et al. ACR 2025. Poster 2365.
Randomized set, in patients with ≥3% body surface area affected by psoriasis at baseline. All patients randomized to PBO received BKZ 160 mg Q4W from Week 16 in BE OPTIMAL and BE COMPLETE (PBO/BKZ). Data reported to 3 years (Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE). BL N numbers refer to the full population size; N numbers for Years 1, 2, and 3 refer to the number of patients with observations at the respective time point. [a]BKZ 160 mg Q4W Total includes patients originally randomized to PBO up to Week 16.
bDMARD: biologic disease-modifying antirheumatic drug; BKZ: bimekizumab; BL: baseline; mNRI: modified non-responder imputation; OC: observed case; PASI: Psoriasis Area and Severity Index; PASI100: 100% improvement from baseline in PASI; PBO: placebo; Q4W: every 4 weeks; TNFi-IR: prior inadequate response or intolerance to tumor necrosis factor inhibitors.

65.6%–67.1% had Complete Resolution of Nail Psoriasis at Year 3 (mNRI)

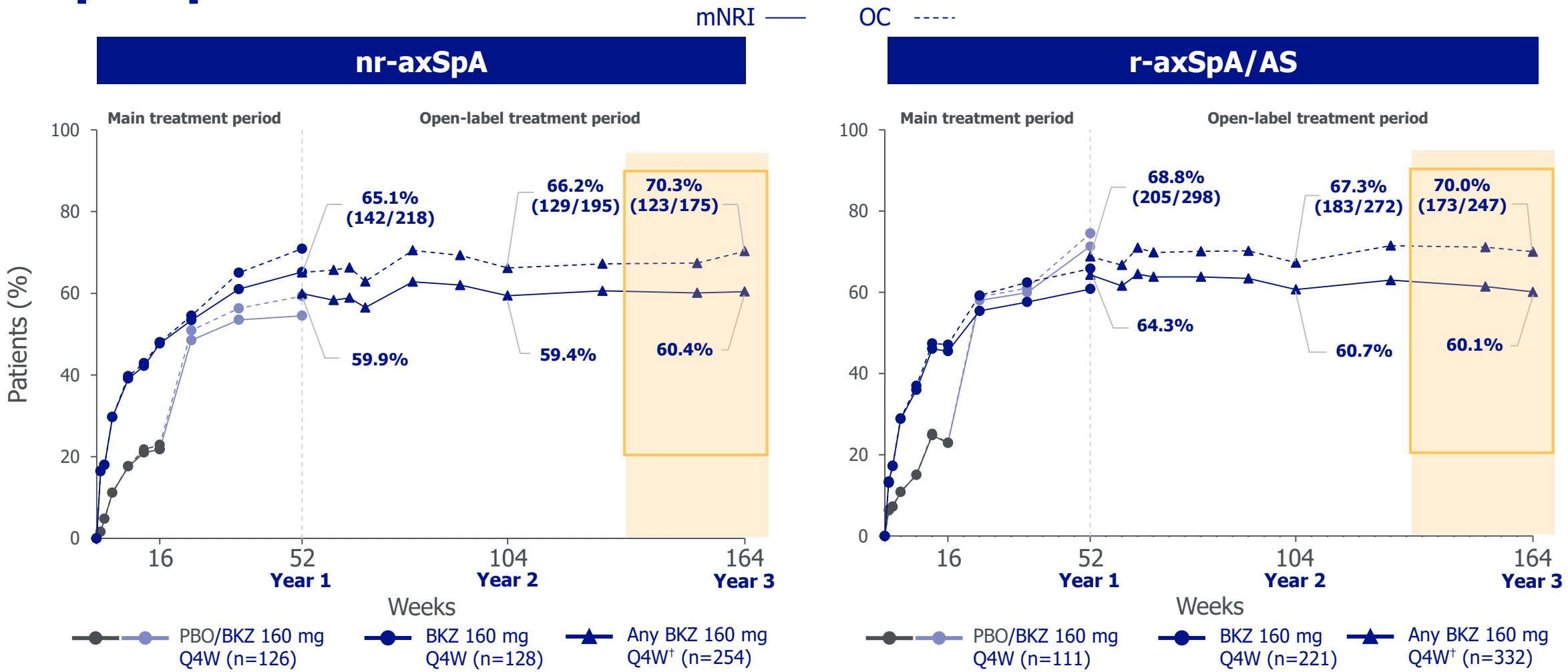


OC, N:	BL	Year 1	Year 2	Year 3
PBO/BKZ	156	143		
BKZ	244	224		
BKZ Total	400	370	339	312

BL	Year 1	Year 2	Year 3
83	75		
159	145		
242	220	202	190

Merola JF, et al. ACR 2025. Poster 2365.
Randomized set, in patients with nail psoriasis at baseline (mNAPSI >0). All patients randomized to PBO received BKZ 160 mg Q4W from Week 16 in BE OPTIMAL and BE COMPLETE (PBO/BKZ). Data reported to 3 years (Week 160 in BE OPTIMAL and Week 156 in BE COMPLETE). BL N numbers refer to the full population size; N numbers for Years 1, 2, and 3 refer to the number of patients with observations at the respective time point. [a]BKZ 160 mg Q4W Total includes patients originally randomized to PBO up to Week 16. bDMARD: biologic disease-modifying antirheumatic drug; BKZ: bimekizumab; BL: baseline; mNAPSI: Modified Nail Psoriasis Severity Index; mNRI: modified non-responder imputation; OC: observed case; PBO: placebo;
Q4W: every 4 weeks; TNFI-IR: prior inadequate response or intolerance to tumor necrosis factor inhibitors.

Bimekizumab Achieved ASAS40 In Over Half of Patients at 1 Year, Which was Sustained Up to 3 Years Across the axSpA Spectrum

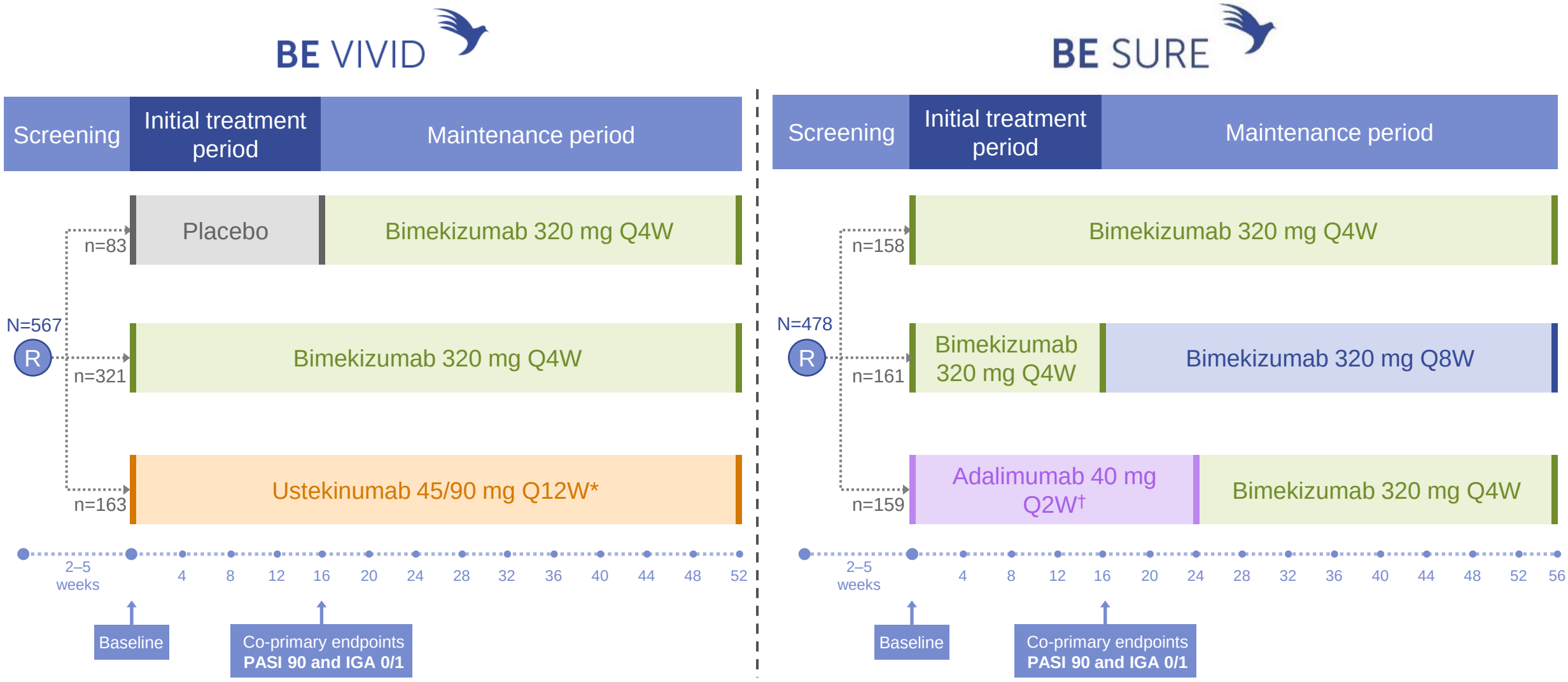


Modified non-responder imputation and observed case. Randomised sets. Long-term data are highlighted to emphasise the sustained response. Data from BE MOBILE 1 + BE MOVING OLE (nr-axSpA) and BE MOBILE 2 + BE MOVING OLE (r-axSpA/AS). mNRI considered all visits following discontinuation due to adverse events or lack of efficacy as non-response; all other missing data were imputed with MI and the response derived from the imputed values. Data labels at Week 52 are related to the Any BKZ group. †Includes patients originally randomised to placebo; all patients were treated with BKZ 160 mg Q4W from Week 16.

Adapted from Baraliakos X, et al. EULAR 2025. Poster POS0788.

Pivotal Phase 3 study design

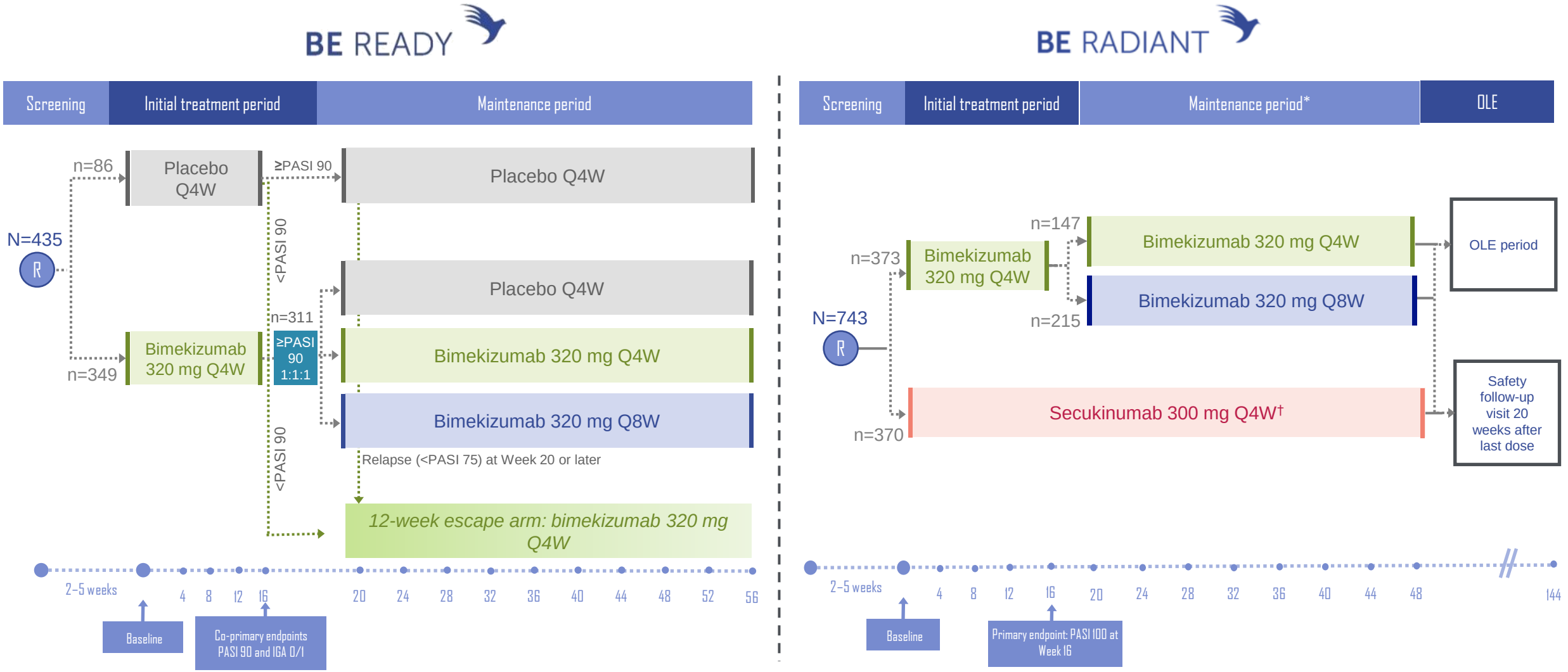
Active comparator studies[1,2]



- *Ustekinumab was dosed per label, with loading doses at Weeks 0 and 4, and weight-based dosing such that patients with weight ≤100 kg received 45 mg doses, whereas patients with body weight >100 kg received 90 mg doses. †All adalimumab patients received 80 mg at Week 0, 40 mg at Week 1 then 40 mg Q2W until Week 24, when they were all switched to bimekizumab. IGA, Investigators Global Assessment; PASI 90, ≥90% improvement from baseline in Psoriasis Area and Severity Index; Q2W, every 2 weeks; Q4W, every 4 weeks, Q8W, every 8 weeks; Q12W, every 12 weeks; R, randomisation. Figures adapted from references 1 and 2. 1. Reich K, et al. Lancet. 2021;397:487–98. 2. Warren RB, et al. N Engl J Med. 2021;385:130–41.

BE RADIANT and BE READY study design

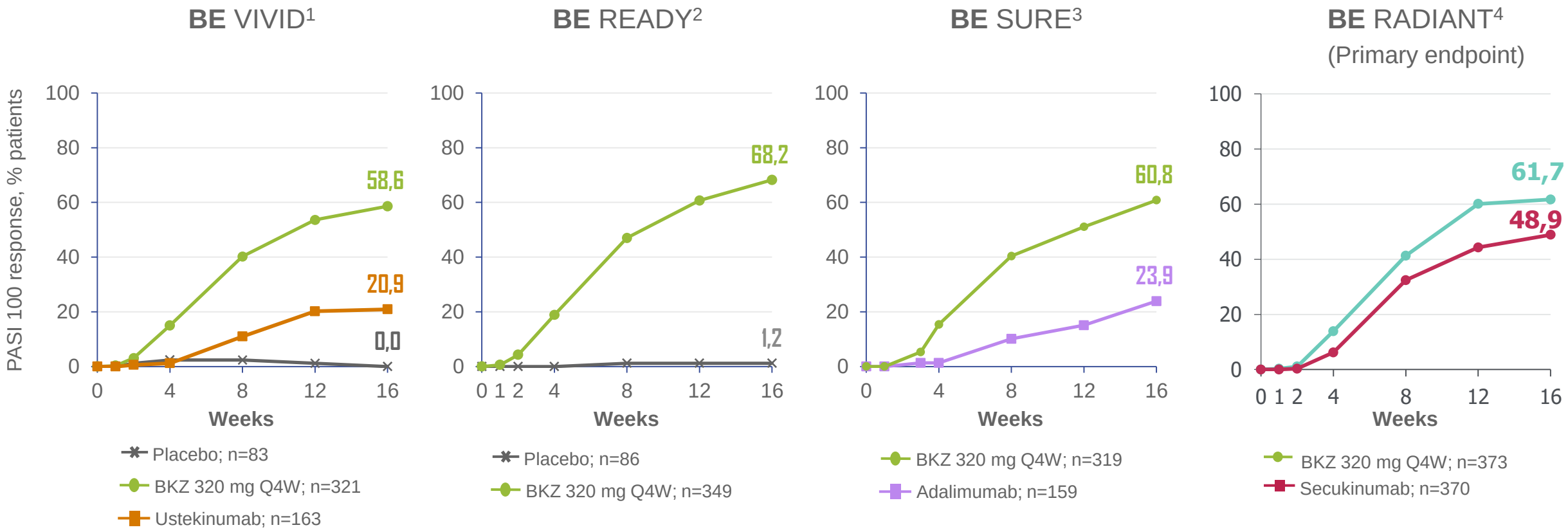
Active comparator and withdrawal studies[1,2]



- *BE RADIANT: At Week 16, patients receiving bimekizumab Q4W were re-randomised 1:2 to receive either bimekizumab 320 mg Q4W or Q8W to Week 48. The Q8W maintenance regimen was added via protocol amendment, with the first re-randomisation ~7 months after the trial began, after 82 patients completed Week 16. †QW to Week 4, Q4W thereafter. IGA, Investigators Global Assessment; OLE, open-label extension; PASI 75/90/100, ≥75/≥90/100% improvement from baseline in Psoriasis Area and Severity Index; QW, once weekly; Q4W, every 4 weeks; Q8W, every 8 weeks; R, randomisation. Figures adapted from references 1 and 2. 1. Reich K, et al. N Engl J Med. 2021;385:142–52. 2. Gordon KB, et al. Lancet. 2021;397:475–86.

BKZ consistently demonstrated high levels of complete skin clearance across studies (NRI)

PASI 100 at Week 16 shows % patients who are 'clear'



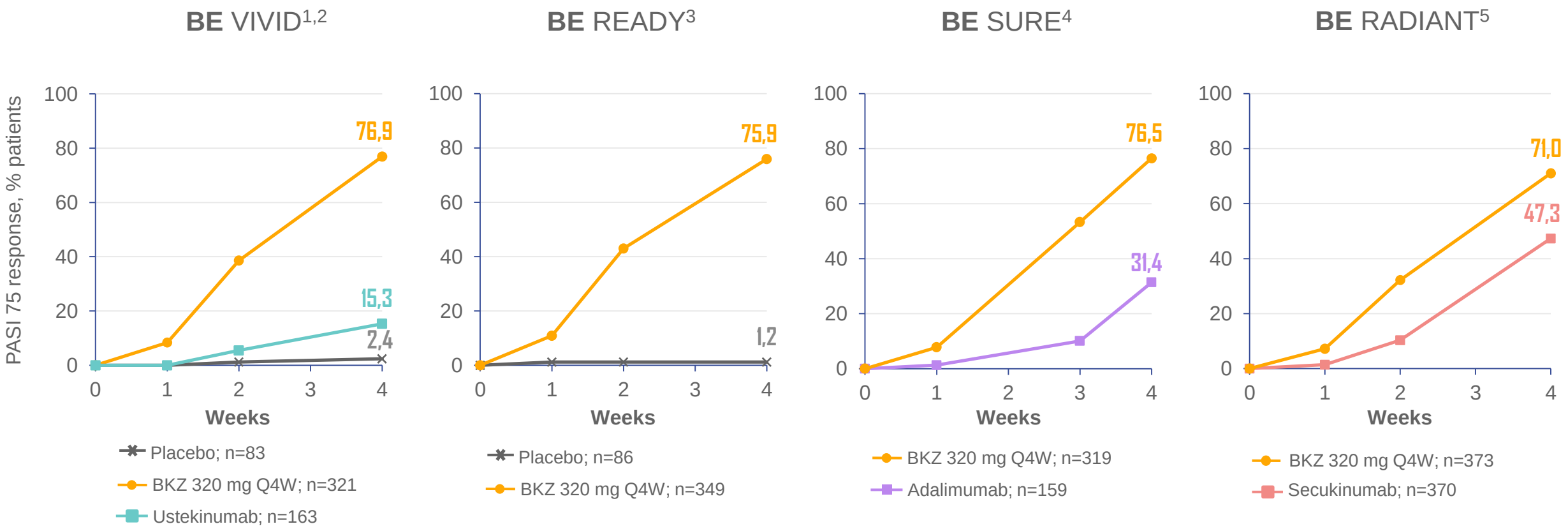
All comparisons (in all trials) p<0.001 (BE VIVID: p<0.0001 BE READY: p<0.0001; p value for ustekinumab comparison is nominal)

- PASI 100 through Week 16 was a ranked secondary endpoint in BE VIVID, BE READY, BE SURE. PASI 100 through Week 16 was the primary endpoint in BE RADIANT.
- NRI, non-responder imputation; PASI 100, 100% improvement from baseline in Psoriasis Area and Severity Index; Q4W, every 4 weeks.
  Figures adapted from references 1–4. 1. Reich K, et al. Lancet. 2021;397:487–98. 2. Gordon KB, et al. Lancet. 2021;397:475–86. 3. Warren RB, et al. N Engl J Med. 2021;385:130–41. 4. Reich K, et al. N Engl J Med. 2021;385:142–52.

BKZ demonstrated a faster onset of response vs active comparators or placebo (NRI)

PASI 75 at Week 4* shows superior efficacy after one dose of BKZ

BKZ superiority: all comparisons (in all trials) p<0.001



- *PASI 75 through Week 4 was a ranked secondary endpoint in BE VIVID, BE READY, BE SURE and BE RADIANT. BKZ, bimekizumab; NRI, non-responder imputation; PASI 75, ≥75% improvement from baseline in Psoriasis Area and Severity Index; Q4W, every 4 weeks. Figures adapted from references 1–5. 1. Reich K, et al. Lancet 2021;397:487–98. 2. PS0009 Table 6.3.1.6.1 NRI RS. 3. Gordon KB, et al. Lancet 2021;397:475–86. 4. Warren RB, et al. N Engl J Med. 2021;385:130–41. 5. Reich K, et al. N Engl J Med. 2021;385:142–52.

Interim Adelphi Real-World Data in Patients With axSpA Treated With Bimekizumab (1/2)

PsA
axSpA
PSO

The Adelphi Real-World BKZ axSpA Plus Disease Specific Programme™ is an ongoing, cross-sectional, partially retrospective*, multinational† real-world study to assess patients with axSpA treated with bimekizumab

DATA SOURCE

Rheumatologists (N=73) and their consulting patients with axSpA (N=325)

PATIENT DEMOGRAPHICS

r-axSpA/AS
53% (N=171)

nr-axSpA
47% (N=154)

Germany
40%
(N=130)

Spain
29%
(N=95)

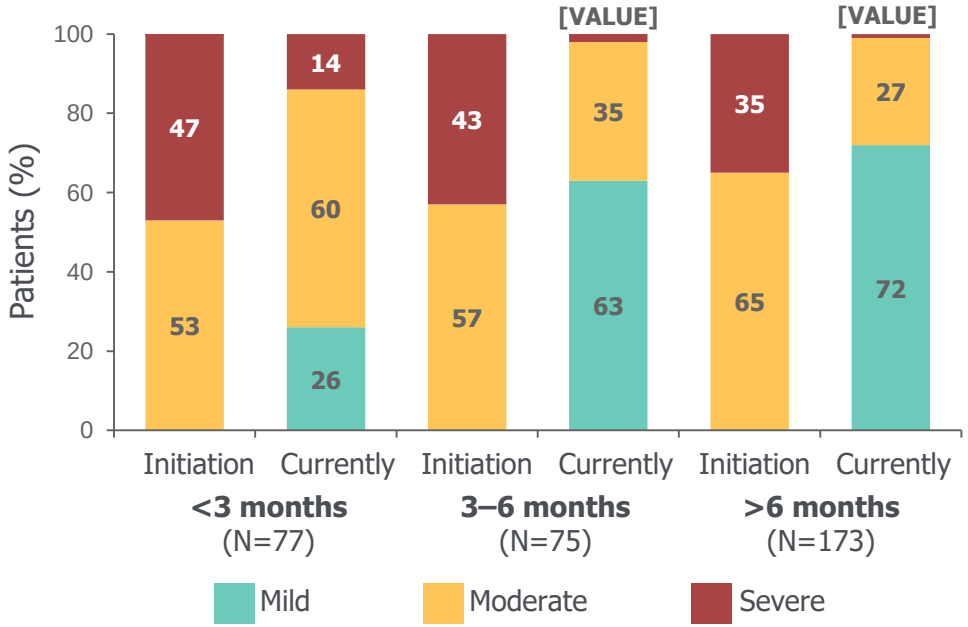
UK
31%
(N=100)

DISEASE

SATISFACTION

Physician-Perceived‡ Disease Severity at Initiation and Most Recent Consultation

Results stratified by bimekizumab treatment duration



CONCLUSION

- In real-world settings, physician-perceived disease severity was seen to **improve in patients with axSpA treated with bimekizumab**, even **when prescribed for less than three months**
- **Sustained treatment** of three months or more **yielded the largest improvements**

Observed case. All patients had moderate-to-severe disease at bimekizumab initiation. Data collection began in July 2024, following a staggered approach by country, based on bimekizumab reimbursement, and is expected to be completed in February 2026. *Retrospective at the point of initiating the current treatment. †France, Germany, Italy, Spain, UK, Canada, Japan and the USA. ‡No clinical definition applied; Rheumatologists may have considered multiple factors when subjectively assessing a patient's disease.

Adapted from Poddubnyy D, et al. Ann Rheum Dis. 2025;84(Suppl 1):2164–2165.

Interim Adelphi Real-World Data in Patients With PsA Treated With Bimekizumab (1/3)

PsA
axSpA
PSO

The Adelphi Real-World BKZ PsA Plus Disease Specific Programme™ is an ongoing, cross-sectional, partially retrospective, multinational* real-world study to assess patients with PsA treated with bimekizumab

DATA SOURCE

Rheumatologists (N=72), dermatologists (N=27) and their consulting patients with PsA (N=342)

PATIENT DEMOGRAPHICS

80% of patients (N=274) had a diagnosis of PSO prior to PsA

Germany **35%** (N=120)

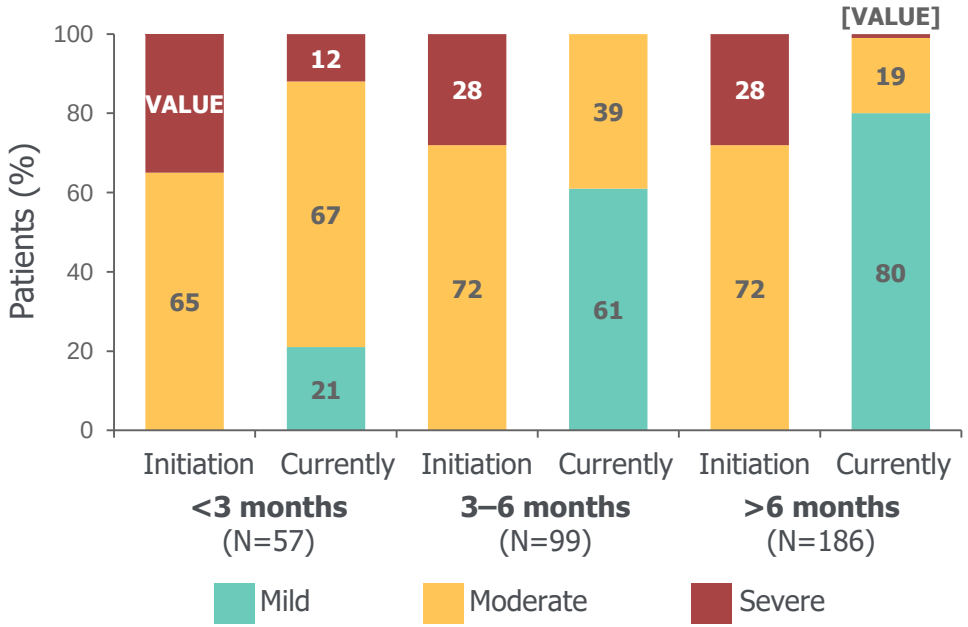
Spain **40%** (N=137)

UK **25%** (N=85)

DISEASE | SKIN | SATISFACTION

Physician-Perceived Disease Severity at Initiation and Most Recent Consultation

Results stratified by bimekizumab treatment duration



CONCLUSION

- **Physician-perceived overall disease severity improved** in patients with PsA treated with bimekizumab
- These findings confirmed the **effectiveness of bimekizumab in PsA in routine clinical practice**

Observed case. All patients had moderate-to-severe disease at bimekizumab initiation. Data collection began in July 2024, following a staggered approach by country, based on bimekizumab reimbursement, and was expected to be completed in September 2025. *France, Germany, Italy, Spain, UK, Canada, Japan and the USA.

Adapted from Proft F, et al. Ann Rheum Dis. 2025;84(Suppl 1):2124–2125.

Comparison of Bimekizumab efficacy in bDMARD-naïve versus IL-17i-experienced patients with Psoriatic Arthritis. A real-world prospective study.

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[1] 1. First Department of Propedeutic and Internal Medicine, Joint Academic Rheumatology program, National and Kapodistrian University of Athens, Athens, Greece, [2] Rheumatology and Clinical Immunology Unit, Attikon University Hospital, National and Kapodistrian University of Athens, Medical School, Athens, Greece, [3] Rheumatology department, KAT General Hospital of Attica, Athens, Greece, [4] 4th Department of Internal Medicine, Aristotle University, Thessaloniki, Greece, [5] Department of Pathophysiology, Joint Academic Rheumatology program, National and Kapodistrian University of Athens, Athens, Greece, [6] Rheumazentrum Ruhrgebiet, Ruhr-University Bochum, Herne, Germany, [7] Department of Rheumatology, Evangelismos General Hospital, Athens, Greece, [8] First Department of Internal Medicine, University Hospital of Alexandroupolis, Democritus University of Thrace, Alexandroupolis, Greece, [9] Third Department of Medicine, National and Kapodistrian University of Athens, School of Medicine, Sotiria Hospital, Athens, Greece, [10] Department of Rheumatology, School of Health Sciences, Faculty of Medicine, University of Ioannina, Ioannina, Greece, [11] Department of Rheumatology, Patras University Hospital, University of Patras Medical School, Patras, Greece, [12] Rheumatology Department, Asklipio Hospital, Athens, Greece, [13] Division of Rheumatology, General Hospital of Ioannina "Hatzikosta", Ioannina, Greece, [14] Department of Rheumatology and Clinical Immunology, University of Thessaly, Larissa, Greece, [15] Rheumatology department, Genimatas General Hospital, Athens, Greece

Table 1. Cohort characteristics

	Naïve (n= 48)	Experienced (n=78)	p-value
Descriptives			
Age (years) mean ± S.D.	52 ± 12	52 ± 12	0.741
Sex (female), n (%)	30 (63)	56 (72)	0.276
Disease duration (years)	2 (7-1)	6 (12-3)	0.001
BMI (kg/cm^2)	31 (35-25)	30 (33-27)	0.676
Smoking (current), n (%)	18 (38)	29 (37)	0.995
HLA B27, n (%)	2 (4)	3 (4)	0.910
Disease characteristics			
Axial disease, n (%)	14 (29)	32 (41)	0.162
Dactylitis, n (%)	14 (29)	17 (22)	0.351
Enthesitis, n (%)	11 (23)	38 (49)	0.004
Skin disease (psoriasis), n (%)	41 (85)	67 (86)	0.940
Nail involvement, n (%)	16 (33)	33 (42)	0.315
Eye involvement, n (%)	1 (2)	2 (3)	0.864
Bowel disease, n (%)	0 (0)	1 (1)	0.431
Number of previous b/tsDMARDs	0	2 (4-2)	NA
Discontinuation of bimekizumab	5 (10)	10 (13)	0.686

Values are presented as median (Q3-Q1) unless otherwise indicated. p-value in bold indicates statistically significant difference (p<0.05)

BMI: Body Mass Index, b/tsDMARD: biologic/target synthetic DMARD

Poulia V. et al., unpublished data, 2

Table 2. Comparison of Disease Activity Between time-0, month-3 and month-6 in b/tsDMARD-naïve and IL-17Ai-Experienced Patients with Psoriatic Arthritis (PsA)

	b/tsDMARD-naïve			p (0-3)	p (0-6)	IL-17Ai-experienced			p (0-3)	p (0-6)
	Time-0 (n=48)	Time-3 (n=46)	Time-6 (n=33)			Time-0 (n=79)	Time-3 (n=65)	Time-6 (n=43)		
TJC	4 (9-2)	0 (1-0)	0 (1-0)	**<0.001**	**<0.001**	4 (10 -1)	1 (3-0)	0 (5-0)	**<0.001**	**<0.001**
SJC	2 (6-1)	0 (0-0)	0 (0-0)	**<0.001**	**<0.001**	2 (4-0)	0 (1-0)	0 (0-0)	**<0.001**	**<0.001**
Enthesitis, n (%)	15 (31)	7 (15)	2 (6)	**0.035**	**0.011**	35 (44)	21 (32)	14 (35)	**0.033**	**0.007**
Dactylitis, n (%)	9 (19)	1 (2)	0 (0)	**0.008**	**0.025**	9 (11)	5 (8)	5 (12)	0.317	**0.083**
BSA	5 (10-1)	0 (1-0)	0 (0-0)	**<0.001**	**<0.001**	1 (6-0)	0 (2-0)	0 (1-0)	**<0.001**	**<0.001**
Nail, n (%)	12 (25)	8 (17)	5	0.102	0.256	17 (22)	14 (22)	1 (2)	0.257	**0.025**
DAPSA	22 (35 -16)	8 (11-5)	5 (11 -3)	**<0.001**	**<0.001**	17 (27-12)	8 (13 -2)	7 (17-3)	**<0.001**	**<0.001**
ASDAS*	0 (2.1 -0)	0 (1.4 -0)	0 (1-0)	0.208	0.152	2.2 (3-0)	0.4 (2-0)	1.5 (1.9-0)	**0.005**	**<0.001**
ESR	20 (35-9)	11 (24-4)	12 (20-8)	**0.005**	**0.02**	19 (30-10)	12 (19-6)	11 (13-5)	**0.001**	**0.001**
CRP	2.5 (8.5 - 1.0)	1.15 (2.8-0.3)	1.0 (2.4-0.6)	**<0.001**	**<0.001**	2.9 (5.8-0.5)	1.2 (5-0.3)	1 (4-0.4)	**0.02**	**0.004**
cDMARDs, n (%)	12 (25)	8 (18)	6 (18)	0.102	0.157	27 (34)	17 (26)	13 (30)	**0.014**	0.157
NSAIDs, n (%)	7 (15)	1 (2)	1 (3)	**0.025**	**0.034**	13 (16)	5 (8)	2 (5)	**0.034**	**0.008**

Values are presented as median (Q3-Q1) unless otherwise indicated. p-value in bold indicates statistically significant difference (p<0.05)

b/tsDMARD: biologic/target synthetic DMARD; DAPSA: Disease Activity in Psoriatic Arthritis; TJC: Tender Joint Count; SJC: Swollen Joint Count; BSA: Body Surface Area affected by psoriasis; ASDAS: Ankylosing Spondylitis Disease Activity Score;

Table 3 – Comparison of differences in clinical and laboratory parameters from time-0 to month-3 and month-6 between b/tsDMARDs-naïve and IL17i-experienced patients

Period 0-3 months			
	b/tsDMARD-naïve	IL-17Ai-experienced	p (0-3)
Δtjc	4 (8-1)	3 (6-0)	0.314
Δsjc	2 (4-2)	1 (3-0)	0.099
ΔBSA	5 (8-1)	1 (3-0)	**0.001**
ΔDAPSA	13 (22-6)	9 (14-3)	**0.015**
ΔASDAS	0.4 [1.1- (-0.2)]	0.8 (1.1-0.4)	0.377
ΔESR	5 [22-(-1)]	5 (18-0)	0.971
ΔCRP	1.5 (5.2-0)	0.2 [2.3- (-0.1)]	0.021
Period 0-6 months			
	b/tsDMARD-naïve	IL-17Ai-experienced	p (0-6)
Δtjc	4 (9-1)	3 (5-1)	0.457
Δsjc	2 (5-0)	2 (4-0)	**0.269**
ΔBSA	5.5 (10-2)	1 (4-0)	**<0.001**
ΔDAPSA	14 (29 -7)	10 (16-4)	**0.030**
ΔASDAS	0.5 (0.7-0.3)	1.2 (1.8-1.1)	**0.037**
ΔESR	4.5 [19- (-5)]	11 (10-5)	0.287
ΔCRP	1.2 (4.8-0.2)	0.4 [3- (-0.1)]	0.175

Values are presented as median (Q3–Q1). p-value in bold indicates a statistically significant difference (p<0.05). b/tsDMARD: biologic/target synthetic DMARD; DAPSA: Disease Activity in Psoriatic Arthritis; TJC: Tender Joint Count; SJC: Swollen Joint Count; BSA: Body Surface Area affected by psoriasis; ESR: Erythrocyte Sedimentation Rate; CRP: C-reactive protein; Δ: indicates difference between value in baseline minus value in the follow-up time point as it is noted.

Poulia V. et al., unpublished data, 2026

***ASDAS is calculated based on CRP**

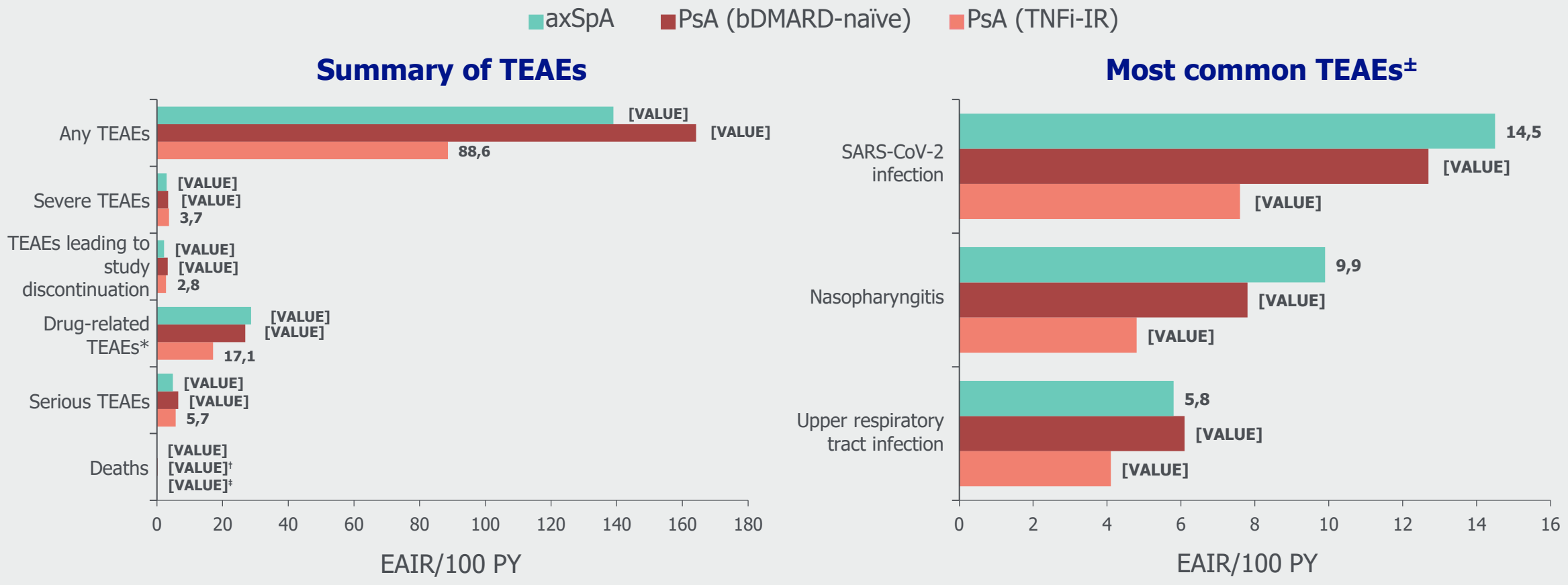
Bimekizumab Has Shown a Generally Well-Tolerated Safety Profile[1–3]

574 axSpA patients (1,664.7 PY)
823 bDMARD-naïve PsA patients (2,022.1 PY)
388 TNFi-IR PsA patients (985.3 PY)

Data from Phase 3 Studies
axSpA: BE MOBILE 1, BE MOBILE 2, BE MOVING (OLE, pooled)
PsA: BE OPTIMAL/BE COMPLETE, BE VITAL (OLE)

The long-term safety profile of bimekizumab was consistent with prior studies

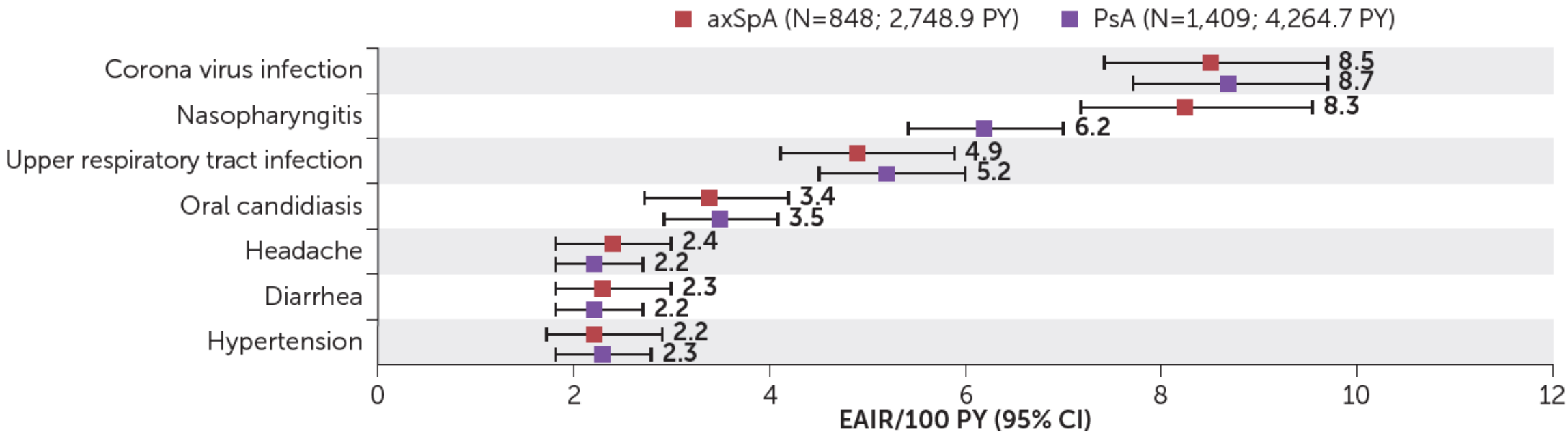
GL-BK-2500197



Safety sets.[1–3] Data include all patients who received ≥1 dose of BKZ 160 mg Q4W (axSpA, N=574; bDMARD-naïve PsA patients, N=823; TNFi-IR PsA patients, N=388).[1–3] TEAEs defined by MedDRA v19.0. *Per investigator assessment.[1–3] †One death due to traumatic shock (motorcycle accident), unrelated to treatment (Week 0–52). One death due to cardiac arrest, unrelated to treatment (Week 104–156). One death due to acute myocardial infarction, unrelated to treatment (Week 52–104).[1] ‡One sudden death (Week 0–52), deemed unrelated to treatment.[2] ±Most frequent TEAEs are the top three adverse events occurring in all patients.[1–3]

1. Gossec L, et al. EULAR 2025. Poster POS1294.
2. McInnes IB, et al. EULAR 2025. Poster POS0105.
3. Baraliakos X, et al. EULAR 2025. Poster 0788.

Most Common TEAEs in the Phase 2b/3 Studies



patient with PsA.

Most fungal infections were *Candida* infections (EAIR/100 PY for axSpA: 4.3; PsA: 4.7), the majority of which were reported as oral candidiasis (EAIR/100 PY for patients with axSpA: 3.5; PsA: 3.8); most cases of

Mease PJ, et al. ACR 2025. Poster 1454.
Data from the September 2024 (axSpA) and August 2024 (PsA) data-cuts shown, including all patients who received ≥1 dose of BKZ 160 mg Q4W. TEAEs defined by MedDRA v19.0 preferred term, occurring in ≥6% of patients in both the axSpA and PsA patient pools. axSpA: axial spondyloarthritis; BKZ: bimekizumab; CI: confidence interval; EAIR: exposure-adjusted incidence rate; MedDRA: Medical Dictionary for Regulatory Activities; PsA: psoriatic arthritis; PY: patient-years; Q4W: every 4 weeks; TEAE: treatment-emergent adverse event.

Mease et al RMDOpen 2026

Recurrence of Oral Candidiasis Within Individual Patients During First 3 Years of Treatment With Bimekizumab in the Phase 3 Studies

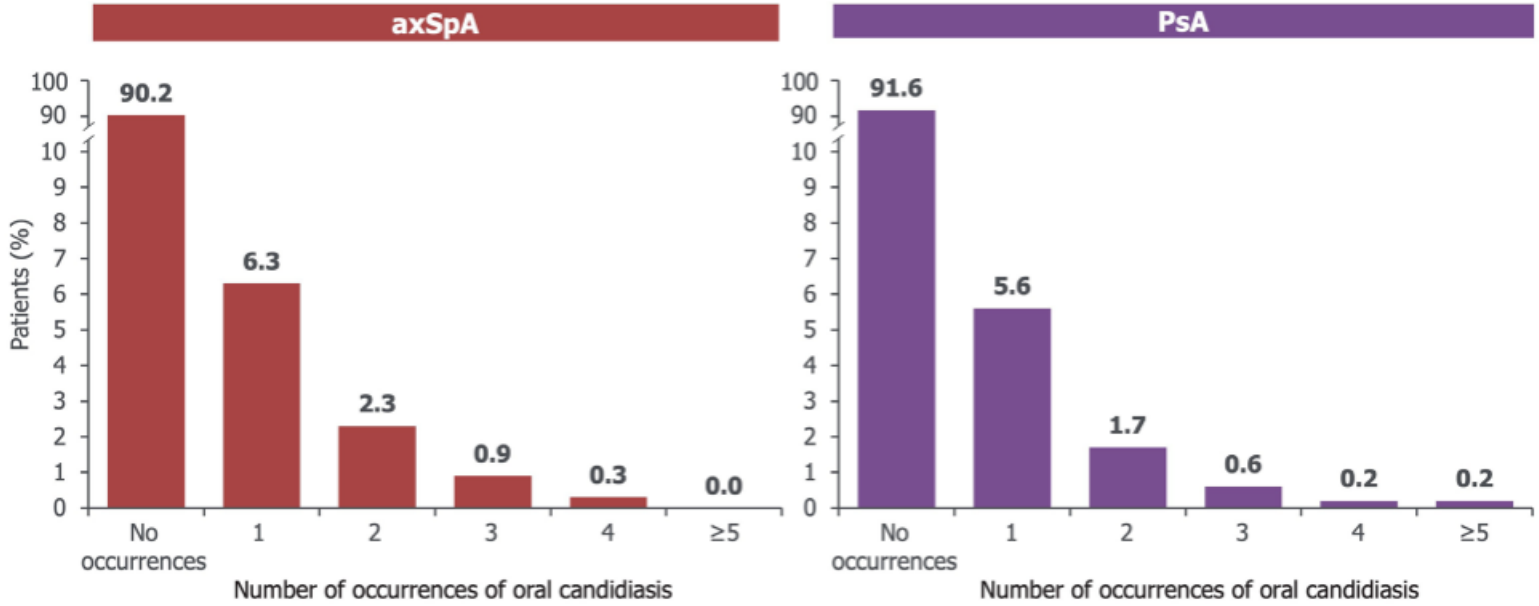
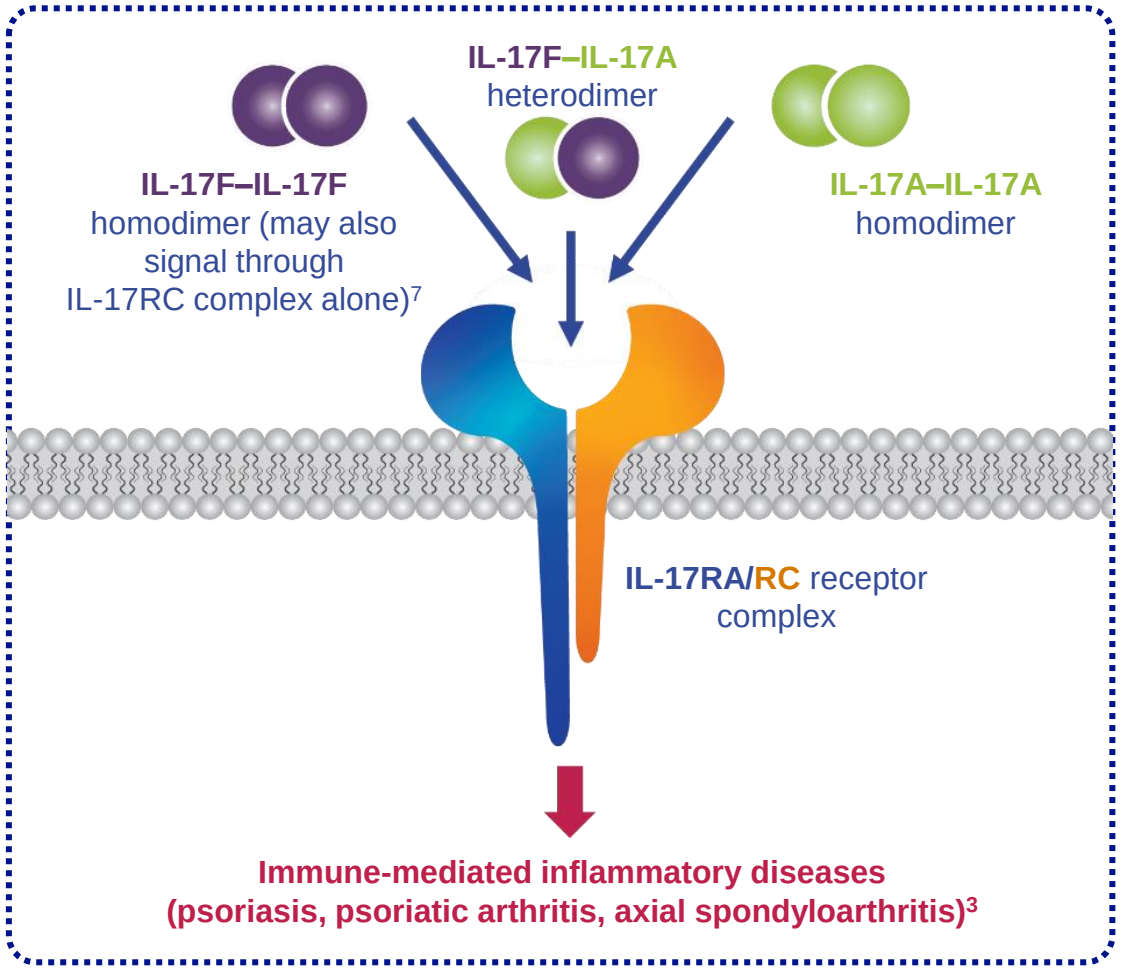


Figure 1 Recurrence of oral candidiasis TEAEs during the first 2 years of treatment with bimekizumab in the phase III studies. Pooled axSpA and PsA safety sets from the phase III studies; includes all patients who received ≥1 dose of BKZ 160 mg Q4W in the phase III studies (axSpA N=574; PsA N=1211). Data from the most recent data cut (July 2023) are shown. Data labels indicate the percentage of patients reporting the respective number of occurrences of oral candidiasis. TEAEs (preferred term according to MedDRA V.19.0) with relative day of onset during weeks 0–104 of treatment with BKZ. axSpA, axial spondyloarthritis; BKZ, bimekizumab; PsA, psoriatic arthritis; Q4W, every 4 weeks; TEAE, treatment-emergent adverse event.

Mease et al RMDOpen 2026

IL-17F Has Overlapping Biology With IL-17A and Forms Hetero- and Homo-dimers; Both Are Produced By Adaptive and Innate Immune Cells and Signal Through the Same Receptor[1–4]

IL-17A and IL-17F[1]



~50% shared structural homology between IL-17F and IL-17A and overlapping biological functions[1,2]

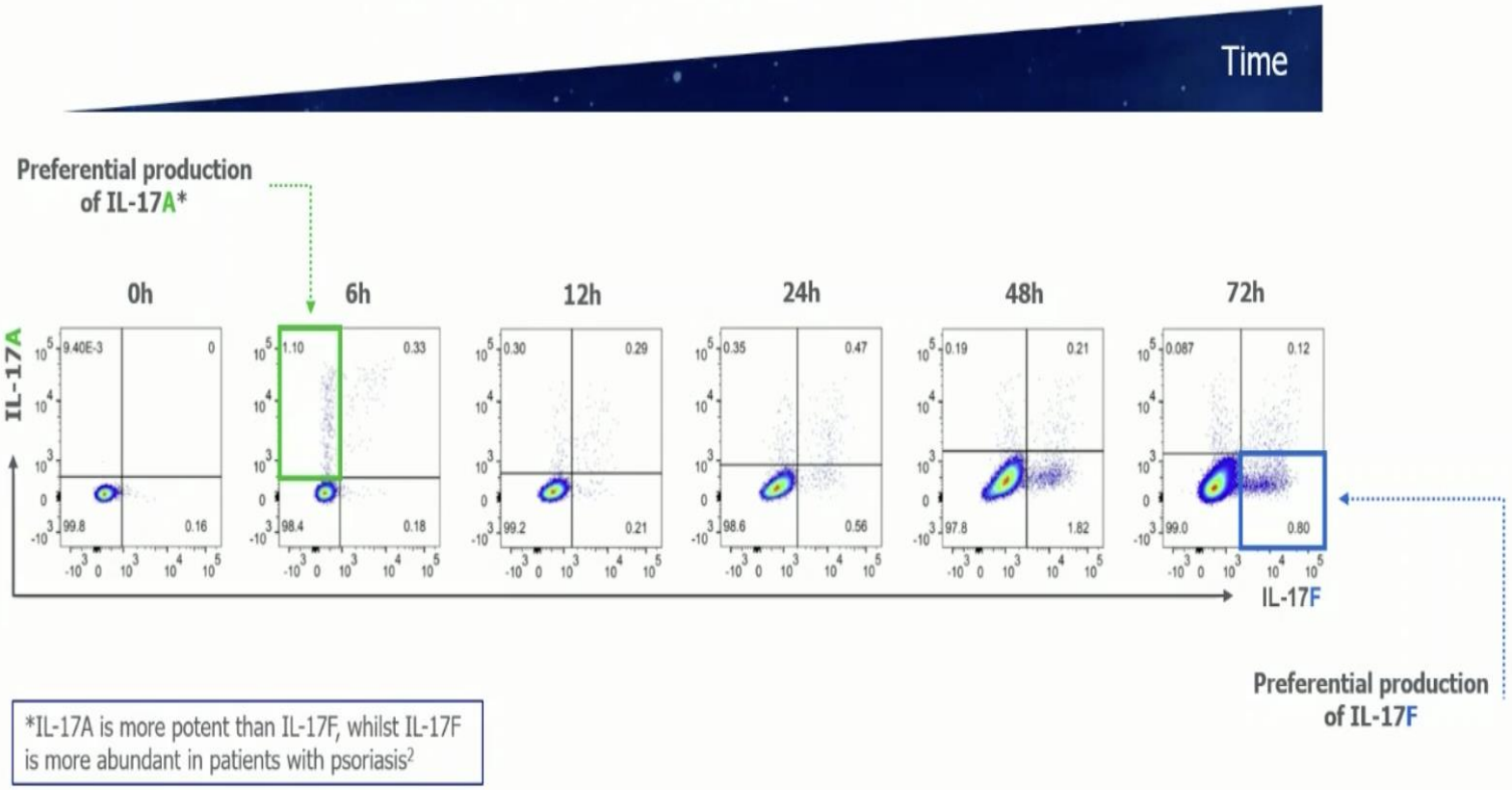
IL-17F and IL-17A are produced by various innate and adaptive immune cells[4]

- IL-17R: Interleukin 17 receptor.

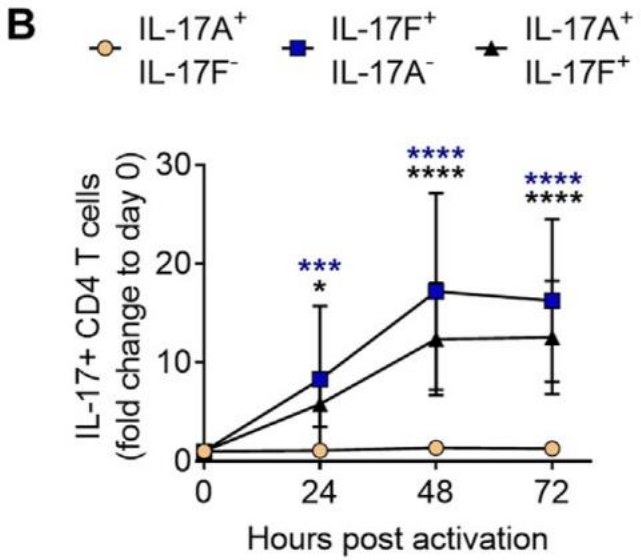
1. Yang XO et al. J Exp Med. 2008;1063–1075. 2. Hymowitz SG et al. EMBO. 2001;20, 5332–5341. 3. Glatt S et al. Ann Rheum Dis. 2018;77:523–532. 4. Jin W and Dong C. Emerg Microbes Infect. 2013;2:e60. 5. van Baarsen LGM et al. Arthritis Res Ther. 2014;16:426. 6. Maroof A et al. ESDR 2017. Poster P426. 7. Goepfert A et al. Immunity. 2020;52(3):499–512.e5.

In vitro, IL-17F Became the Dominant Cytokine Expressed by Th17 Cells Within 72 Hours[1]

***In vitro*, activated Th17 initially express more IL-17A upon stimulation but switch to preferential IL-17F expression within 72 hours[1†]**

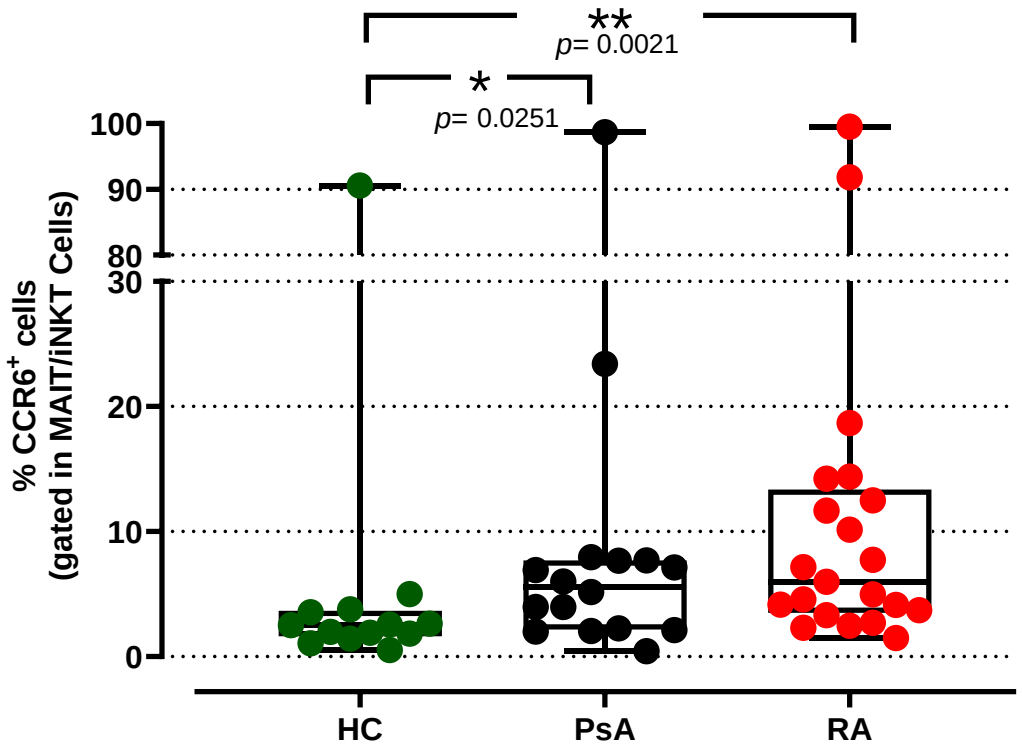


*IL-17A is more potent than IL-17F, whilst IL-17F is more abundant in patients with psoriasis[2]



Cole s et al. J Allergy Clin Innumol 2023:152:783-98

MAIT/iNKT
Peripheral blood CCR6 (Increased in PsA)



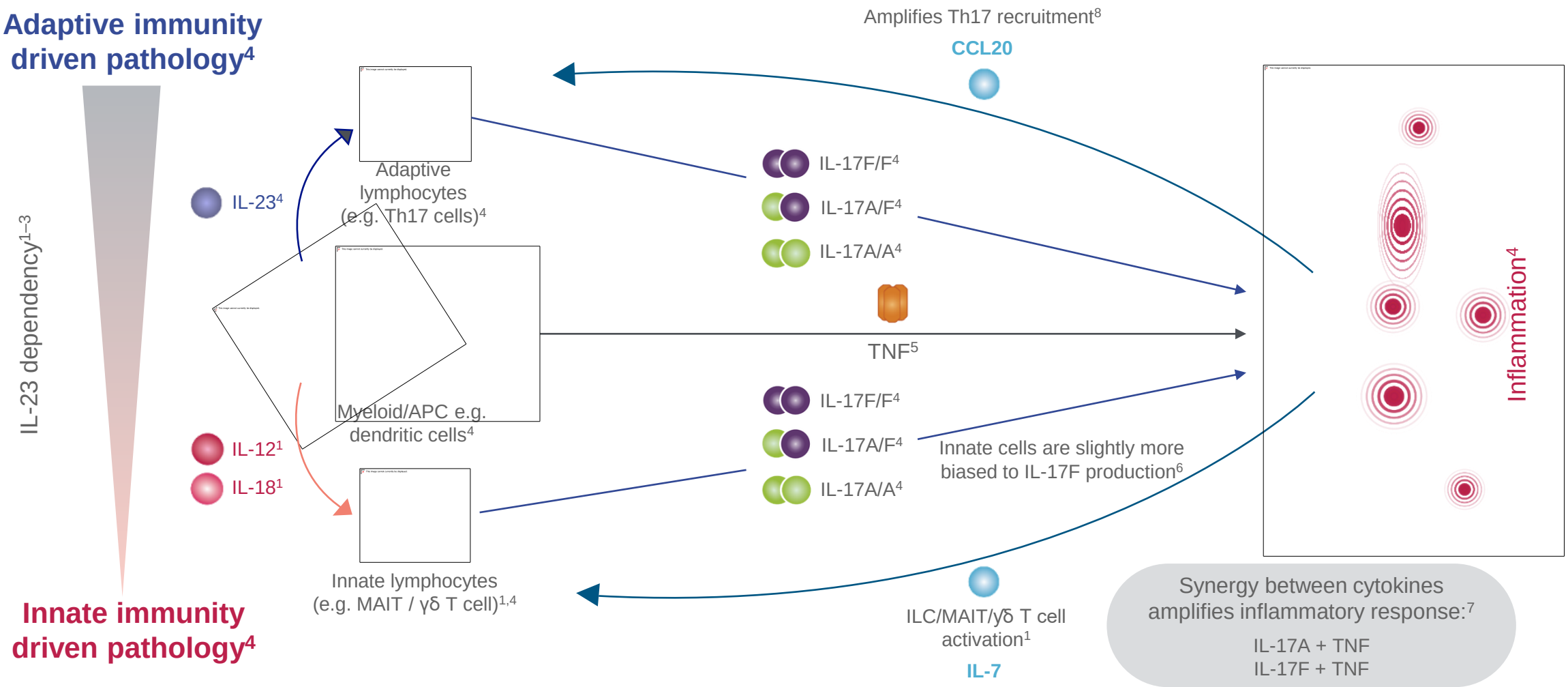
ELSEVIER

Clinical Immunology
Volume 253, August 2023, 109679

Distinct innate and adaptive immunity phenotypic profile at the circulating single-cell level in Psoriatic Arthritis

George E. Fragoulis [a c], Eleni-Kyriaki Vetsika [b], Maria Kyriakidi [b], Kleio-Maria Verrou [b], George Kollias [b], Maria G. Tektonidou [a], Iain B. McInnes [c], Petros P. Sfikakis [a b]

IL-23 Appears to be Less Important in axSpA Where IL-17-Producing Innate Cells May Play an Important Role[1–3]



- Example cell types are shown; not an exhaustive list. APC: antigen-presenting cell; CCL20: C-C motif chemokine ligand 20; ILC: innate lymphoid cell; MAIT: mucosal-associated invariant T cell; Th17: T helper 17 cell; TNF: tumour necrosis factor; γδT: gamma delta T cell.

1. Rosine N, Miceli-Richard C. Front Immunol. 2021;11:553742. 2. McGonagle DG et al. Ann Rheum Dis. 2019;78(9):1167–1178. 3. McGonagle D et al. Front Immunol. 2021;12:614255. 4. Tsukazaki H, Kaito T. Int J Mol Sci. 2020;21(17):6401. 5. Blanco P et al. Cytokine Growth Factor Rev. 2008;19(1):41–52. 6. Cole et al. Front Immunol. 2020;11:585134.2. 7. Glatt S et al. Ann Rheum Dis. 2018;77:523–532. 8. Russell T et al. Cells. 2021;10(2):341.

Dual Inhibition of IL-17F in Addition to IL-17A With Bimekizumab Modulates Downstream Cytokine Production

Inhibiting IL-17A and IL-17F with bimekizumab suppresses the PsA transcriptome[1]

Pro-inflammatory gene expression*

	PsA	+Anti-IL-17A	+BKZ
CXCL1	14.88	4.97	2.19
CXCL8	14.06	5.65	4.26
C3	8.90	2.63	2.31
CXCL3	10.07	3.14	1.52
CXCL6	10.85	6.88	2.98
CCL20	4.91	1.66	1.41
IL-6	4.71	3.38	2.79
CCL7	8.65	6.32	5.66
CXCL10	55.04	59.51	40.76
IL-15RA	8.56	8.97	8.53
LIF	4.52	2.66	2.62

Inhibiting IL-17F in addition to IL-17A results in suppression of synoviocyte proinflammatory genes that are elevated in PsA[1]

Glatt S et al. Ann Rheum Dis. 2018;77:523–532.

Take home messages

- IL-17 A+F inhibition: a new mechanism of intervention
  - Solid underlying pathogenetic mechanism
  - Effective
    - Some additional benefits
    - Safe
- Real world evidence
  - Accumulating ☺
    - Consolidating the efficacy/safety profile of the drug

ΕΛΛΗΝΙΚΗ ΡΕΥΜΑΤΟΛΟΓΙΚΗ ΕΤΑΙΡΕΙΑ
& ΕΠΑΓΓΕΛΜΑΤΙΚΗ ΕΝΩΣΗ
ΡΕΥΜΑΤΟΛΟΓΩΝ ΕΛΛΑΔΟΣ

Registries for Early PsA & axSpA

- **Στόχος:** Καταγραφή **Νεοδιαγνωσθέντων** ασθενών (b/tsDMARDs naive) και ετήσια παρακολούθηση
- **Πλατφόρμες:**

  PsA: https://psa.ere.gr/

  axSpA: https://axspa.ere.gr/

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Thank you for your attention ☺