

Bimekizumab time to onset of PASI response in patients with psoriasis in three head-to-head phase 3/3b studies

Kenneth B. Gordon,¹ Annunziata Dattola,² Sascha Gerdes,³ Melinda Gooderham,^{4,5} Balint Szilagyi,⁶ Bengt Hoepken,⁶ Rhys Warham,^{7,8} Carle Paul⁹

¹Department of Dermatology, Medical College of Wisconsin, Milwaukee, Wisconsin, USA; ²Dermatology Clinic, Department of Clinical Internal, Anesthesiological and Cardiovascular Sciences, Sapienza University, Rome, Italy; ³Center for Inflammatory Skin Diseases, Department of Dermatology, Venereology and Allergology, University Medical Center Schleswig-Holstein, Campus Kiel, Kiel, Germany; ⁴SKiN Centre for Dermatology, Probody Medical Research, Peterborough, Ontario, Canada; ⁵Queen's University, Kingston, Ontario, Canada; ⁶UCB, Monheim am Rhein, Germany; ⁷Veramed, London, UK; ⁸UCB, Slough, UK; ⁹Toulouse University and CHU, Toulouse, France

Objective

To report the median time to onset of Psoriasis Area and Severity Index (PASI) responses in patients with moderate to severe plaque psoriasis treated with bimekizumab (BKZ) versus adalimumab (ADA), ustekinumab (UST) and secukinumab (SEC).

Introduction

- Rapid onset of treatment response is an important goal for patients with psoriasis and a treatment offering this is preferred over a treatment offering other highly desirable goals such as prolonged time to relapse.^{1,2}
- Fast-acting treatments that align with patients' treatment goals can help to improve patient-orientated psoriasis care.³
- BKZ, a humanised IgG1 monoclonal antibody that inhibits both interleukin (IL)-17F and IL-17A, has previously demonstrated earlier onset of ≥75%/≥90%/100% improvement from baseline in PASI (PASI 75/90/100) responses in comparison with ADA, UST and SEC.^{4–6}
- Absolute PASI scores can also be used to assess psoriasis; specifically, PASI ≤2 represents a clinically relevant disease endpoint to inform treat-to-target management strategies, and more stringent thresholds are becoming an increasingly achievable treatment goal.^{7–9}
- Assessing absolute PASI scores, rather than relative improvements, offers benefits in clinical practice, as absolute PASI outcomes are not influenced by baseline severity and provide a direct measure of the patient's current disease severity.^{8,10}

Methods

- Post hoc analyses were carried out using data from patients receiving BKZ 320 mg every 4 weeks (Q4W) to Week 16 vs comparators (dosed per label) in phase 3/3b studies: BE SURE (BKZ Q4W vs ADA), BE VIVID (BKZ Q4W vs UST) and BE RADIANT (BKZ Q4W vs SEC).^{11–13}
- Median times to PASI ≤1, PASI ≤2 and PASI ≤3 responses were estimated using Kaplan-Meier analysis.
- Patients who discontinued prior to achieving PASI ≤1/≤2/≤3 response, and those who reached Week 16 without PASI ≤1/≤2/≤3 response, were censored at the date of last PASI assessment.

Results

- Baseline characteristics for each study have been reported previously and were similar across treatment arms.^{11–13}
- In BE SURE, the median number of days to reach PASI ≤1 for BKZ Q4W (N=319) was 57, around 8 weeks less than it took for ADA (115 days [N=159]; **Figure 1A, Table 1**).
 - The median time taken to reach PASI ≤2 and PASI ≤3 was also shorter for BKZ Q4W than ADA: 55 days vs 86 days and 29 days vs 84 days, respectively (**Table 1**).
- In BE VIVID, the median number of days to reach PASI ≤1 for BKZ Q4W (N=321) was 57, around 8 weeks less than it took for UST (114 days [N=163]; **Figure 1B, Table 1**).
 - The median time taken to reach PASI ≤2 and PASI ≤3 was also shorter for BKZ Q4W than UST: 56 days vs 87 days and 30 days vs 85 days, respectively (**Table 1**).
- In BE RADIANT, the median number of days to reach PASI ≤1 for BKZ Q4W (N=373) was 58, around 4 weeks less than it took for SEC (83 days [N=370]; **Figure 1C, Table 1**).
 - The median time taken to reach PASI ≤2 and PASI ≤3 for BKZ Q4W was 56 days and 31 days, respectively, compared with 57 days to reach both for SEC (**Table 1**).

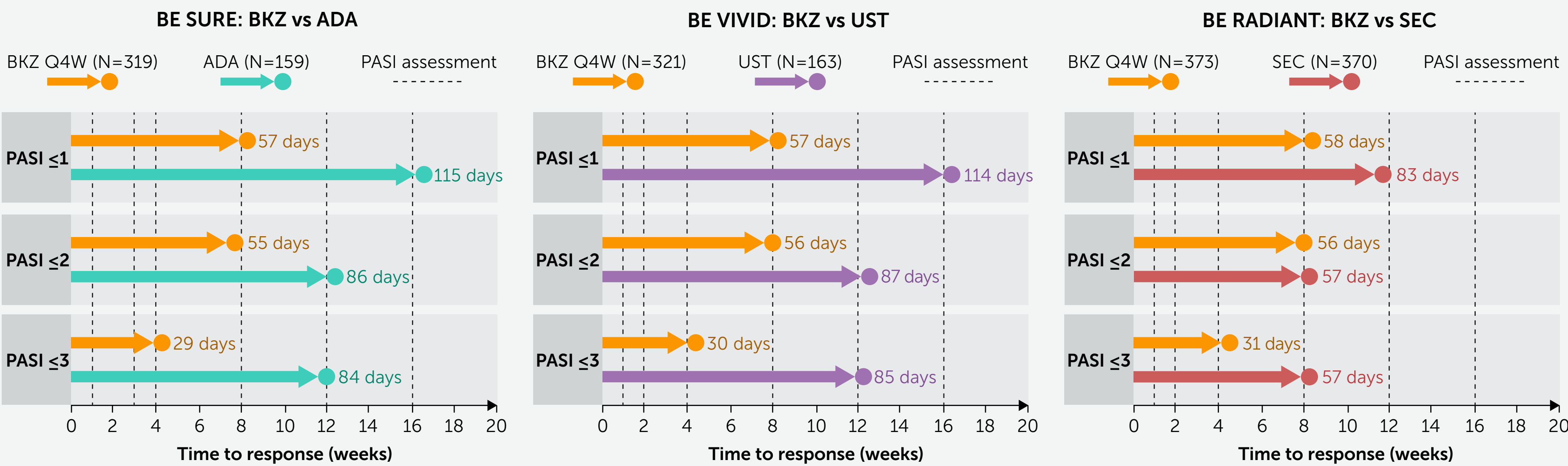
Conclusions

BKZ demonstrated a consistently rapid onset of clinically relevant absolute PASI responses (PASI ≤1/PASI ≤2/PASI ≤3), and this was generally faster than responses observed with ADA, UST and SEC.

These results demonstrate that BKZ is a fast-acting treatment option for psoriasis, which aligns with important patient treatment goals.

Summary

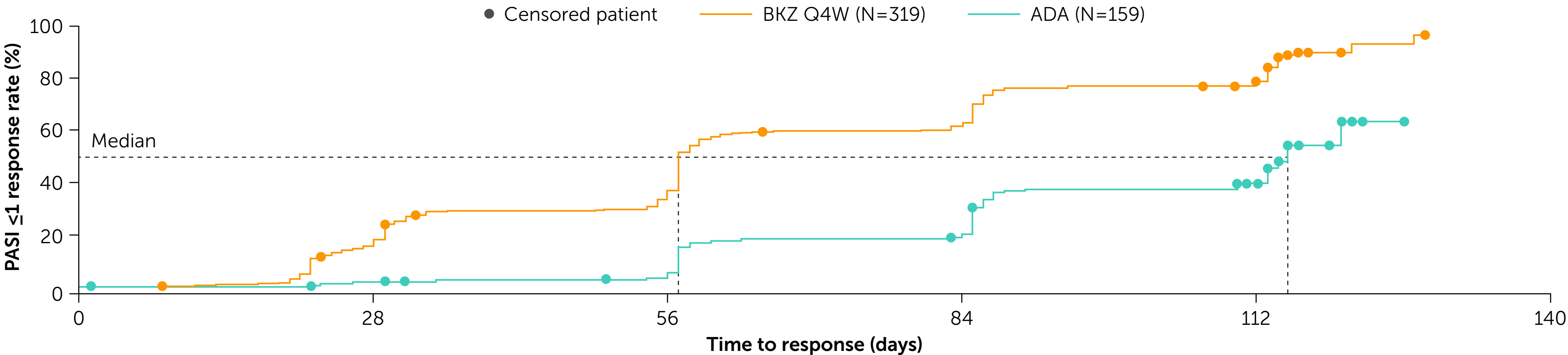
Median time to onset of PASI responses in three head-to-head phase 3/3b studies



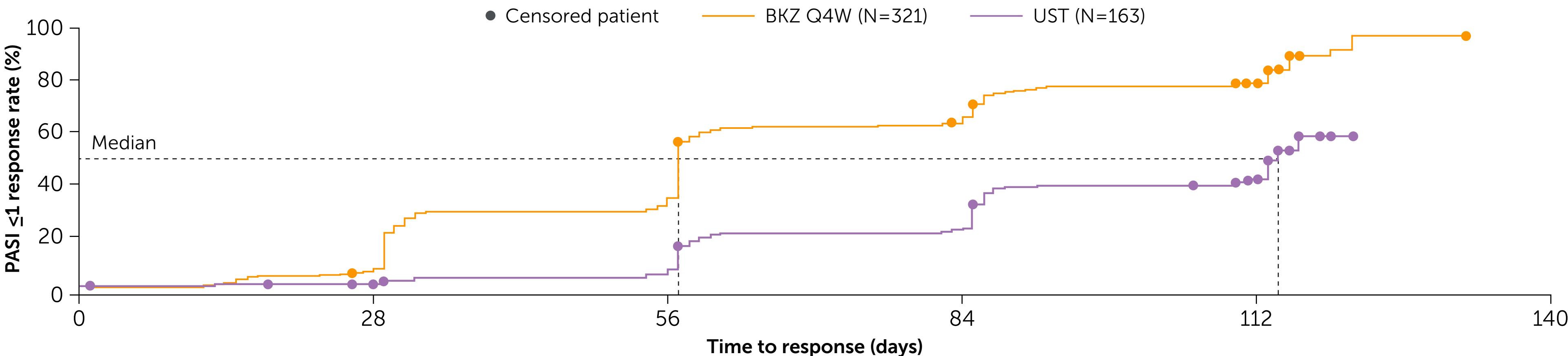
Treatment with BKZ resulted in **faster onset of clinically relevant PASI responses (PASI ≤1/≤2/≤3)** than with ADA, UST or SEC, demonstrating that BKZ treatment helps to achieve treatment goals that align with improved patient-orientated psoriasis care.

Figure 1 Time to absolute PASI ≤1 response in three head-to-head phase 3/3b studies

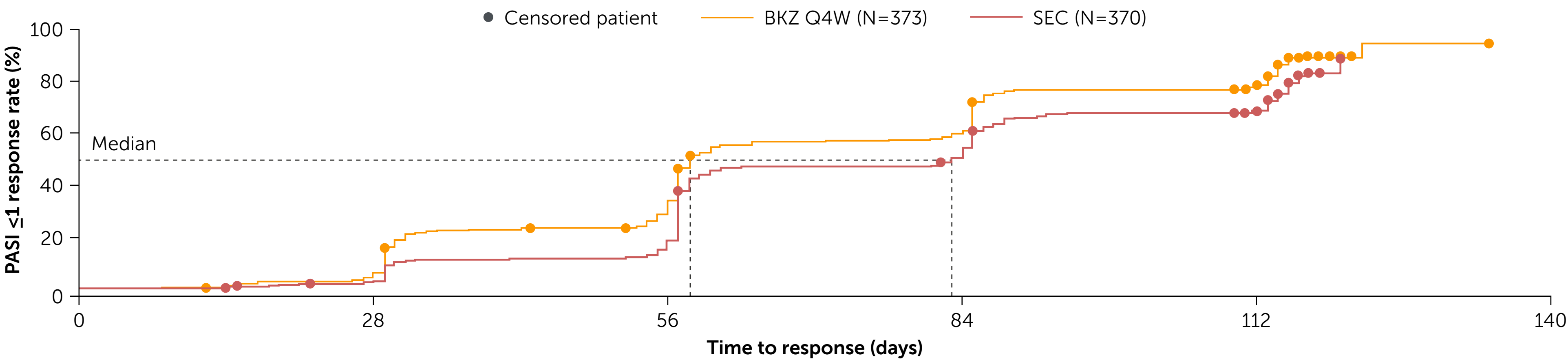
A) BE SURE: BKZ Q4W vs ADA



B) BE VIVID: BKZ Q4W vs UST



C) BE RADIANT: BKZ Q4W vs SEC



Randomised set (all study participants randomised to treatment). Time to PASI ≤1 response (in days) was calculated as min (date of first PASI ≤1 response, date of Week 16 visit) – date of baseline visit + 1. Patients who discontinued study treatment prior to achieving PASI ≤1 response were censored at the date of the last observed PASI assessment on or prior to the date of study treatment discontinuation. Patients who reached the Week 16 visit without achieving PASI ≤1 response were censored at the date of the last observed PASI assessment on or prior to the Week 16 visit. Comparator dosing was as follows: BE SURE patients received ADA 80 mg at baseline, then 40 mg Q2W from Week 1; BE VIVID patients received UST 45 mg/90 mg at baseline (weight-dependent) and Week 4, then Q12W thereafter; BE RADIANT patients received SEC 300 mg weekly to Week 4, then Q4W thereafter.^{11–13}

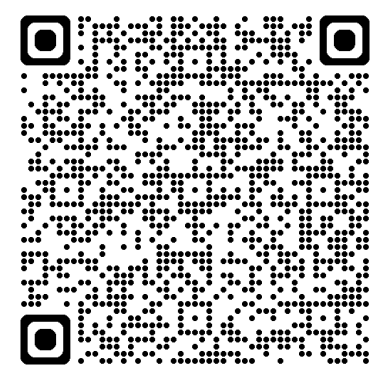
Table 1 Median time to onset of PASI threshold responses in three head-to-head phase 3/3b studies

Median time to response (days)	BE SURE		BE VIVID		BE RADIANT	
	BKZ Q4W (N=319)	ADA (N=159)	BKZ Q4W (N=321)	UST (N=163)	BKZ Q4W (N=373)	SEC (N=370)
PASI ≤1	57	115	57	114	58	83
PASI ≤2	55	86	56	87	56	57
PASI ≤3	29	84	30	85	31	57

Randomised set (all study participants randomised to treatment). Time to PASI response (in days) was calculated as min(date of first PASI response, date of Week 16 visit) – date of baseline visit + 1. Patients who discontinued study treatment prior to achieving PASI ≤1/≤2/≤3 response were censored at the date of the last observed PASI assessment on or prior to the date of study treatment discontinuation. Patients who reached the Week 16 visit without achieving PASI ≤1/≤2/≤3 response were censored at the date of the last observed PASI assessment on or prior to the Week 16 visit. Comparator dosing was as follows: BE SURE patients received ADA 80 mg at baseline, then 40 mg Q2W from Week 1; BE VIVID patients received UST 45 mg/90 mg at baseline (weight-dependent) and Week 4, then Q12W thereafter; BE RADIANT patients received SEC 300 mg weekly to Week 4, then Q4W thereafter.^{11–13}

ADA: adalimumab; BKZ: bimekizumab; IL: interleukin; PASI: Psoriasis Area and Severity Index; PASI 75/90/100: ≥75%/≥90%/100% improvement from baseline in PASI; Q2W: every 2 weeks; Q4W: every 4 weeks; Q12W: every 12 weeks; SEC: secukinumab; UST: ustekinumab.

References: ¹Blome C et al. Arch Dermatol Res 2016;308:69–78; ²Egeberg A et al. J Eur Acad Dermatol Venereol 2020;34:39–46; ³Uhlenhake EE et al. J Dermatolog Treat 2010;21:6–12; ⁴Adams R et al. Front Immunol 2020;11:1894; ⁵Feldman SR et al. Presented at AAD 2023. Poster 34310; ⁶Lebwohl M et al. Presented at AAD 2021. Poster 27376; ⁷Mahil SK et al. Br J Dermatol 2020;182:1075–76; ⁸Puig L et al. Acta Derm Venereol 2019;99:71–77; ⁹Augustin M et al. Dermatol Ther (Heidelb). 2024;14:2841–57; ¹⁰Gerdes S et al. J Dermatol Treat 2020;31:470–5; ¹¹Warren RB et al. N Engl J Med 2021;385:130–41 (NCT03412747); ¹²Reich K et al. Lancet 2021;397:487–98 (NCT03370133); ¹³Reich K et al. N Engl J Med 2021;385:142–52 (NCT03536884). **Author Contributions:** Substantial contributions to study conception/design, or acquisition/analysis/interpretation of data: **KBG, AD, SG, MG, BS, BH, RW, CP**; Drafting of the publication, or reviewing it critically for important intellectual content: **KBG, AD, SG, MG, BS, BH, RW, CP**; Final approval of the publication: **KBG, AD, SG, MG, BS, BH, RW, CP**. **Author Disclosures:** **KBG:** Received consulting fees from AbbVie, Almirall, Amgen, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Dermira, Eli Lilly and Company, Janssen, Novartis, Pfizer, Sun Pharma and UCB; research support from AbbVie, Bristol Myers Squibb, Celgene, Eli Lilly and Company, Janssen, Novartis and UCB. **AD:** Consultant for AbbVie, Amgen, Boehringer Ingelheim, Eli Lilly, LEO Pharma and UCB. **SG:** Advisor and/or received speakers' honoraria and/or participated in clinical trials of the following companies: AbbVie, Acelyrin Inc., Adimune Inc., Affibody AB, Akari Therapeutics Plc, Almirall-Hermal, Amgen, Apogee Therapeutics, Argenev BV, Aristeia Therapeutics, Bristol Myers Squibb, Boehringer Ingelheim, Celgene, Dermira, Eli Lilly and Company, Galderma, Hexal AG, Incyte Inc., Janssen-Cilag, Klinge Pharma, Kymab, LEO Pharma, Medac, MoonLake Immunotherapeutics, MSD, Neubourg Skin Care GmbH, Novartis, Pfizer, Pierre Fabre, Principia Biopharma, Regeneron Pharmaceutical, Sandoz Biopharmaceuticals, Sanofi-Aventis and UCB. **MG:** Investigator, speaker, consultant or advisory board member for AbbVie, Acelyrin, Akros, Amgen, AnaptysBio, Arcutis, Aristeia, ASLAN Pharmaceuticals, Bausch Health, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, Dermira, Eli Lilly and Company, Galderma, GSK, Incyte, JAMP Pharma, Janssen, Kyowa Kirin, L'Oréal, MedImmune, Meiji, MoonLake Immunotherapeutics, Nektar Therapeutics, Nimbus, Novartis, Pfizer, Regeneron, Reistone, Sanofi, Sun Pharma, Takeda, Tarsus, UCB, Union, Ventyx and Vyne. **BS, BH:** Employees and shareholders of UCB. **RW:** Veramed statistical consultant for UCB. **CP:** Received consulting fees and/or grants from AbbVie, Almirall, Amgen, Boehringer Ingelheim, Celgene, Eli Lilly and Company, GSK, Janssen Cilag, LEO Pharma, Novartis, Pierre Fabre, Pfizer, Sanofi Regeneron and UCB. **Acknowledgements:** These studies were funded by UCB. We thank the patients and their caregivers in addition to the investigators and their teams who contributed to this study. The authors acknowledge Inés Dueñas Pousa, PhD, UCB, Madrid, Spain for publication coordination, Oscar Jenkyn-Jones, MSc, Costello Medical, London, UK, and Rachel Mckeown, PhD, Costello Medical, Cambridge, UK, for medical writing and editorial assistance and the Costello Medical Creative team for graphic design assistance. All costs associated with development of this poster were funded by UCB.



To receive a copy of this poster, scan the QR code
Link expiration: 19 September 2025