

Bimekizumab improves draining tunnel outcomes in patients with moderate to severe HS regardless of prior biologic use: 3-year data from BE HEARD EXT

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Objective

To report the impact of bimekizumab on draining tunnel (DT) outcomes in patients with moderate to severe hidradenitis suppurativa (HS) over 3 years, including in prior biologic use patient subgroups.

Introduction

- HS is a chronic, inflammatory skin disease characterised by painful lesions, including DTs.¹
- DTs cause irreversible structural damage, negatively impact quality of life and are indicative of disease progression.^{2,3}
- Delays in initiating biologic treatment beyond the therapeutic 'window of opportunity' may lead to an increase in DT counts, thereby exacerbating this progression.⁴
 - Use of a greater number of prior biologic therapies may also reduce biologic responsiveness.⁵
- Interleukin (IL)-17F and IL-17A are highly expressed in patients with HS, with IL-17F more abundant in HS serum and lesional skin than IL-17A.^{6,7} IL-17F expression is significantly upregulated in lesions, with the highest expression in draining tunnels.⁸
- Bimekizumab is a humanised IgG1 monoclonal antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A.⁹

Methods

- Data were pooled from the phase 3 BE HEARD I&II (NCT04242446, NCT04242498) trials and their open-label extension, BE HEARD EXT (NCT04901195) for patients with moderate to severe HS.^{10,11}
- Data are reported for patients randomised to bimekizumab 320 mg from baseline in BE HEARD I&II who subsequently entered BE HEARD EXT and continued to receive bimekizumab (BKZ Total).
 - Data are also stratified by prior biologic use for HS (biologic-naïve or biologic-experienced).
- The following outcomes are reported:
 - Mean (standard deviation [SD]) DT count for all patients and those with ≥1 baseline DT over 3 years (Week 148);
 - Patients with ≥1 DT at baseline achieving DT counts of 0, 1–2, 3–5 or >5 at baseline, Year 1 (Week 48) and Year 3;
 - Patients with 1–2, 3–5 or >5 DTs at baseline achieving a DT count of 0 (DT=0) at Year 1 and Year 3.
- Data are reported as observed case (OC).

Results

- Of the 1,014 patients in BE HEARD I&II, 868 were randomised to bimekizumab at baseline; 556 patients completed Year 1 and entered BE HEARD EXT.
 - Of those, 444 patients were biologic-naïve and 112 were biologic-experienced; 425 had ≥1 DT at baseline.
- Baseline characteristics were generally similar between subgroups, though biologic-experienced patients had longer disease duration and more severe HS than those who were biologic-naïve (**Table 1**).
- Mean DT counts were generally higher in biologic-experienced versus biologic-naïve patients over 3 years; this trend was consistent in all patients with ≥1 DT at baseline (**Figures 1A and 1B**).
- In patients with ≥1 DT at baseline, the proportions achieving DT counts of 0, 1–2, 3–5 and >5 are presented in **Figure 2**.
 - At Year 3, over two thirds of biologic-naïve patients achieved DT=0, compared with approximately half of those who were biologic-experienced.
 - Similarly, the proportion of patients with >5 DTs reduced from baseline to Year 3, with greater reductions seen at Year 1 in biologic-naïve versus biologic-experienced patients.
- Achievement of DT=0 at Years 1 and 3 was highest in patients with 1–2 baseline DTs and lowest in those with >5 baseline DTs (**Figure 3**).
 - Across baseline DT counts, the proportions of patients achieving DT=0 at Year 3 were generally higher in biologic-naïve versus biologic-experienced patients.

Conclusions

Reductions in DT counts were maintained through 3 years of bimekizumab treatment for all subgroups, though greater achievement was observed in biologic-naïve versus biologic-experienced patients.

Lower baseline DT counts were associated with greater DT clearance over 3 years, irrespective of prior biologic use.

These results may support early bimekizumab initiation during the therapeutic 'window of opportunity', when prior biologic use is likely to be low and HS less severe, to prevent disease progression.

Summary

Biologic-naïve patients displayed **higher achievement of DT outcomes** with **bimekizumab** at Year 3 compared with biologic-experienced patients, though **improvements** were **maintained** through 3 years, **regardless of prior biologic use**

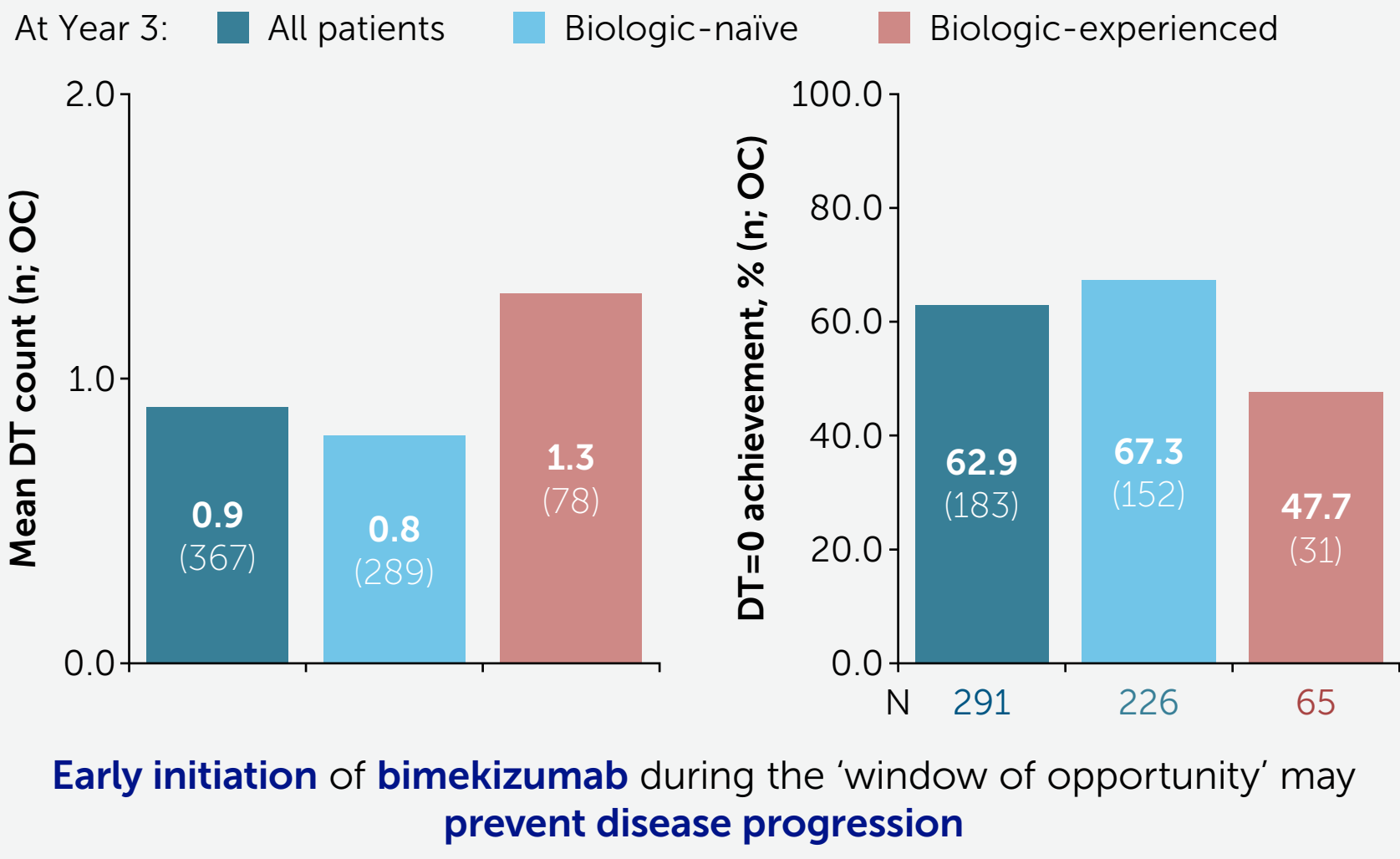
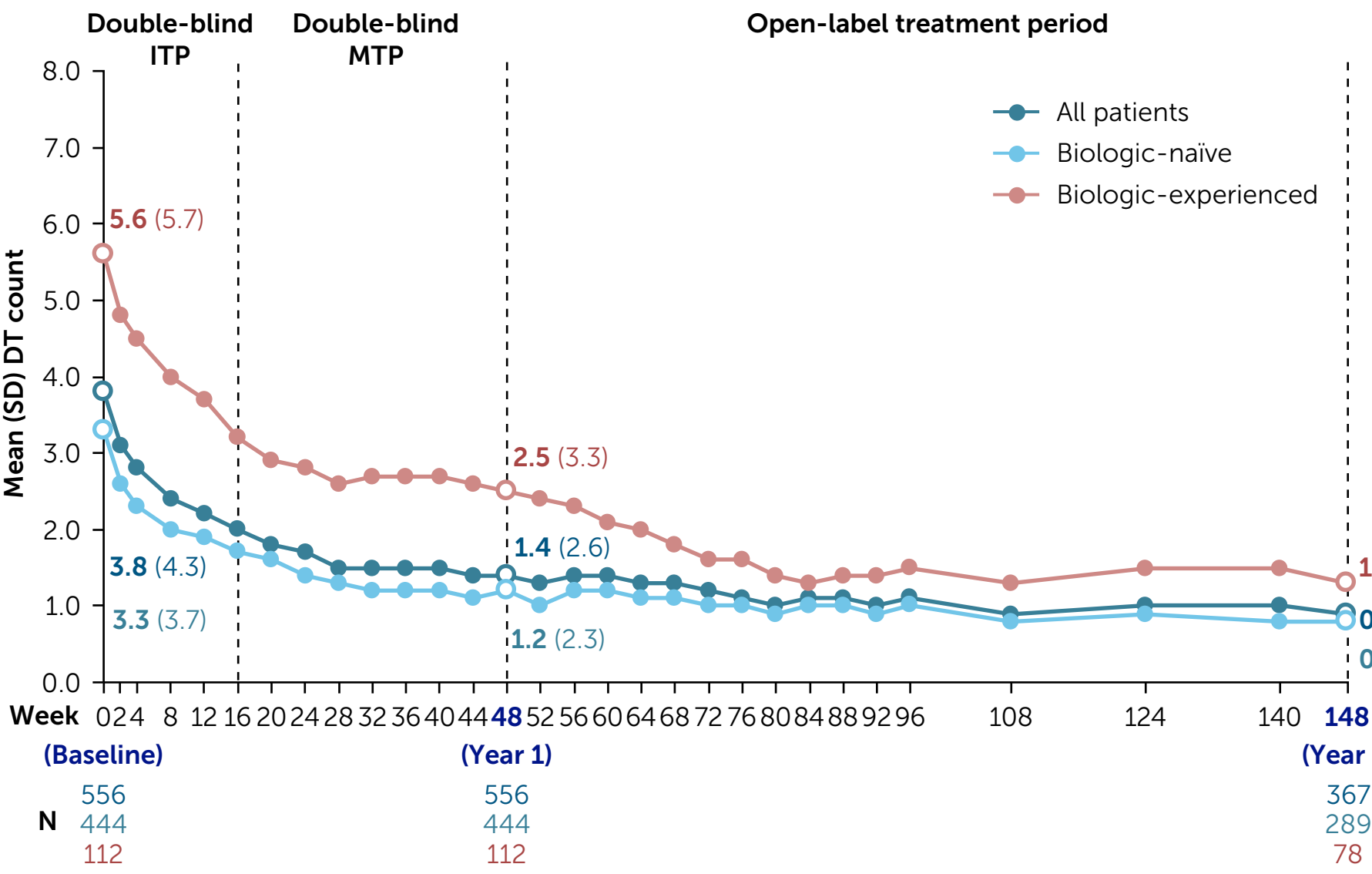


Figure 1 Mean DT count over 3 years

A) Mean DT count over 3 years



OC, N: N represents number of patients with a non-missing draining tunnel count assessment in the given week.

Table 1 Baseline characteristics

	All BKZ Total N=556	Biologic-naïve N=444	Biologic-experienced N=112
Age (years), mean (SD)	36.3 (12.2)	36.0 (12.0)	37.5 (12.6)
Sex (female), n (%)	299 (53.8)	244 (55.0)	55 (49.1)
Racial group, n (%)			
White	448 (80.6)	351 (79.1)	97 (86.6)
Black or African American	55 (9.9)	50 (11.3)	5 (4.5)
BMI (kg/m²), mean (SD)	32.5 (7.8)	32.5 (7.9)	32.4 (7.6)
Duration of disease (years), mean (SD)	7.4 (7.1)	7.2 (7.2)	8.3 (6.9)
IHS4 score, mean (SD)	35.6 (31.5)	33.3 (28.3)	44.7 (40.9)
AN count, mean (SD)	16.9 (18.5)	16.7 (15.6)	17.6 (27.3)
Hurley stage, ^a n (%)			
II	303 (54.5)	263 (59.2)	40 (35.7)
III	253 (45.5)	181 (40.8)	72 (64.3)
Baseline antibiotic use, n (%)	54 (9.7)	47 (10.6)	7 (6.3)
DLQI total score, mean (SD)	11.0 (6.8)	10.5 (6.6)	13.2 (7.1)
HisSQOL total score, mean (SD)	24.6 (12.8)	23.8 (12.7)	28.0 (12.6)

^a This refers to the worst overall Hurley stage derived from the Hurley stages recorded across all anatomical regions.

B) Mean DT count in patients with ≥1 DT at baseline over 3 years

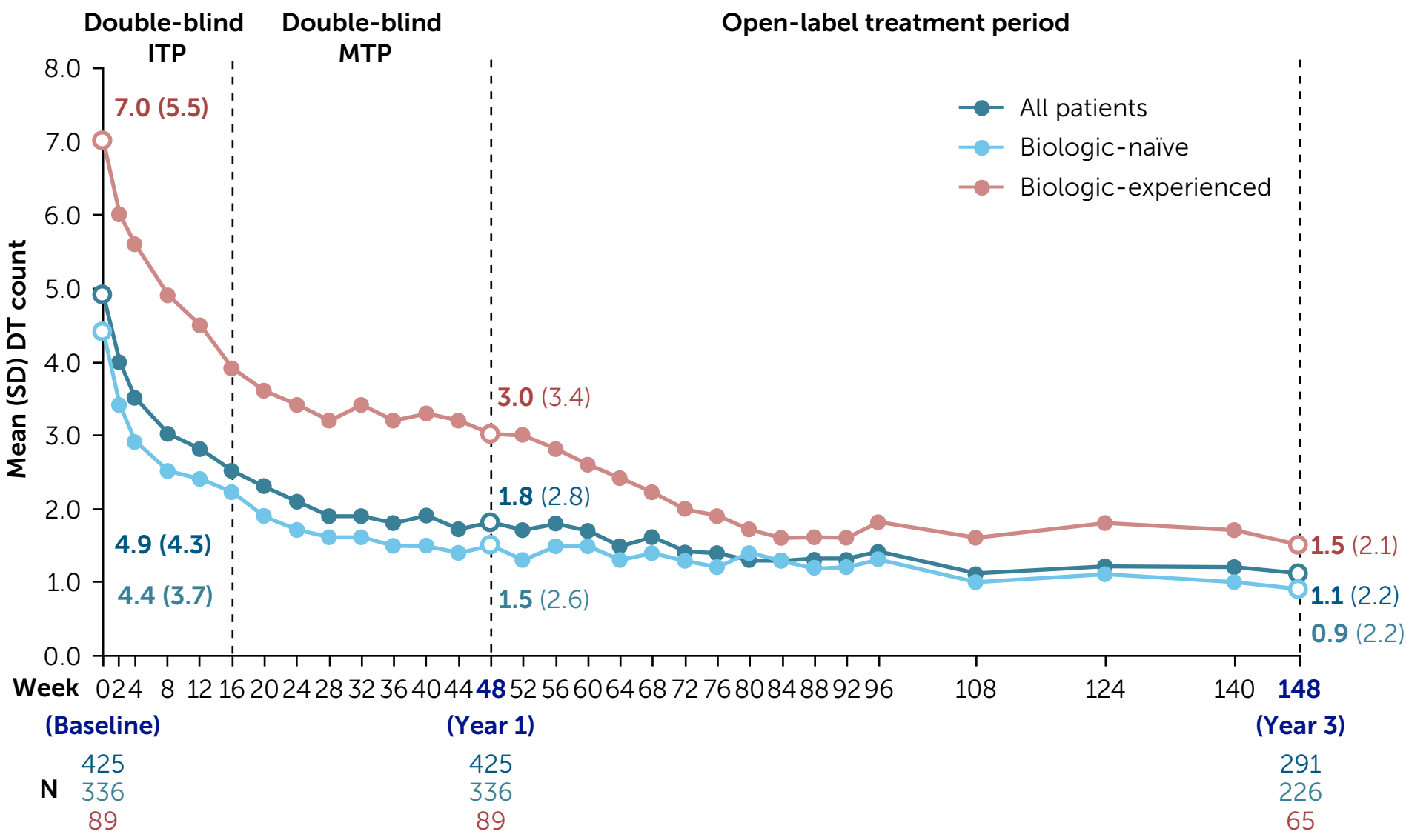
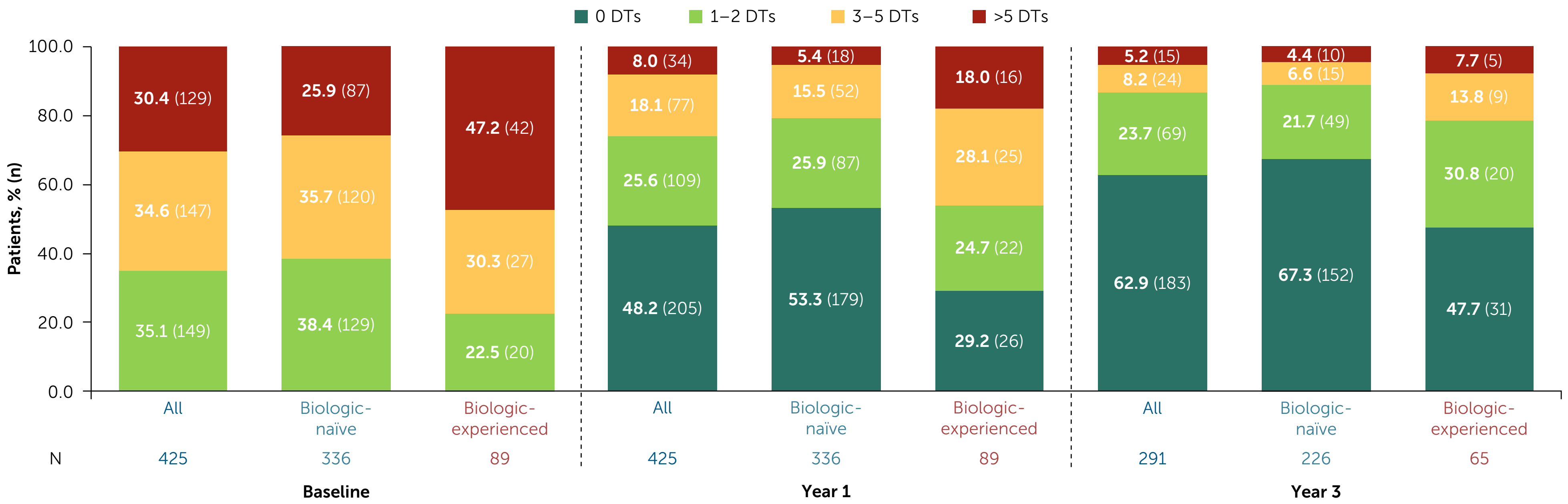
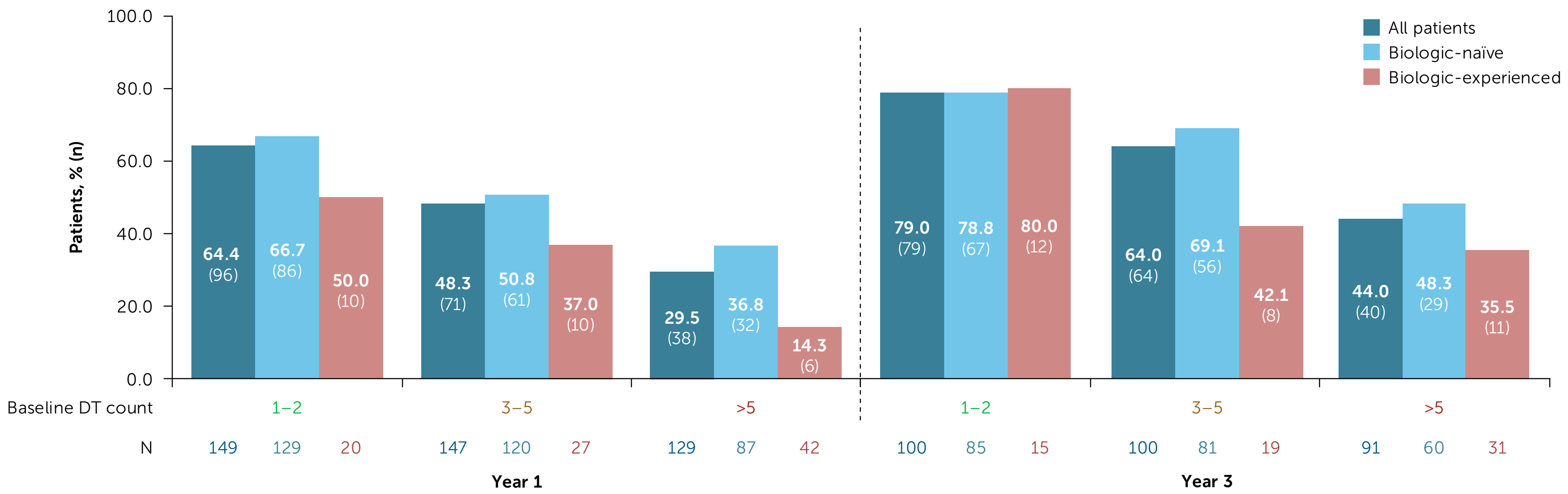


Figure 2 Patients with ≥1 DT at baseline with 0, 1–2, 3–5 or >5 DTs at baseline, Year 1, and Year 3



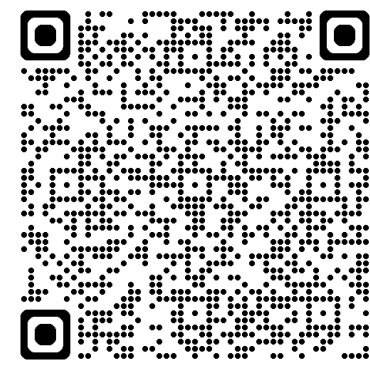
OC, N: N represents number of patients with a non-missing draining tunnel count assessment in the given week, and percentages are calculated accordingly.

Figure 3 Patients with 1–2, 3–5 or >5 DTs at baseline achieving DT=0 at Year 1 and Year 3



OC, N: N represents number of patients with a non-missing draining tunnel count assessment in the given week, and percentages are calculated accordingly.

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J Am Acad Dermatol 2026;94:967–78. **Author Contributions:** Substantial contributions to study conception/ design, or acquisition/analysis/interpretation of data: **AM, JH, FGB, CCZ, MG, TM, BL, CC, NT, MLP**; drafting of the publication, or reviewing it critically for important intellectual content: **AM, JH, FGB, CCZ, MG, TM, BL, CC, NT, MLP**; final approval of the publication: **AM, JH, FGB, CCZ, MG, TM, BL, CC, NT, MLP**. **Author Disclosures:** **AM:** Received honoraria and/or travel grants and/or acted as an advisory board member for AbbVie, Almiral, Amgen, Boehringer Ingelheim, Bristol Myers Squibb, Eli Lilly and Company, Janssen, Legit Health, LEO Pharma, L'Oréal, Novartis, Otsuka, Sanofi, and UCB; worked as a principal investigator in clinical trials supported by AbbVie, Bristol Myers Squibb, Cytiryl, Eli Lilly and Company, Galderma, Indero, Janssen, Kymera Therapeutics, Legit Health, Kymera Therapeutics, MSD, Novartis, Pfizer, Pivviant, Sanofi, Sun Pharma, Takeda, UCB, and Zura Bio; On the Board of Directors for the Hidradenitis Suppurativa Foundation (unpaid); served as an advisor, investigator and/or speaker for AbbVie, Aclaris, Amgen, Arcutis, AstraZeneca, Avano, Boehringer Ingelheim, Galderma, Incyte, Insmid, LEO Pharma, MoonLake Immunotherapeutics, Navigator Medicines, Novartis, Orla Therapeutics, Pfizer, Spyre, Sanofi, Regeneron and UCB. **FGB:** Received honoraria for participation in advisory boards, in clinical trials, and/or as a speaker from AbbVie Inc., AbbVie Deutschland GmbH & Co. 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