Onset of clinical response with deucravacitinib in patients with moderate to severe scalp psoriasis: a post hoc analysis of the phase 3b/4 PSORIATYK SCALP trial

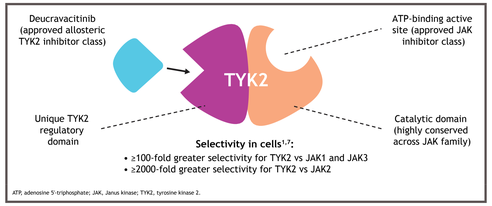
Matthias Hoffmann,[1] Andrew Blauvelt,[2] Eugene Balagula,[3] Andrew Napoli,[3] Chun-Yen Cheng,[3] Rachel Dyme,[3] Virginia Hala,[3] Stephen Tyring,[4] Jerry Bagel,[5] Mark Lebwohl[6]

[1]Private Practice, Witten, Germany; [2]Blauvelt Consulting, Annapolis, MD, USA; [3]Bristol Myers Squibb, Princeton, NJ, USA; [4]University of Texas Health Center and Center for Clinical Studies, Houston, TX, USA; [5]Psoriasis Treatment Center of New Jersey, East Windsor, NJ, USA; [6]Icahn School of Medicine at Mount Sinai, New York, NY, USA

Synopsis

- Tyrosine kinase 2 (TYK2) is an intracellular enzyme that mediates signaling of select inflammatory cytokines (eg, interleukin [IL]-23, IL-12, Type I interferons [IFNs])[1]
  - IL-23 and Type I IFNs are involved in psoriasis pathogenesis[1]
- Deucravacitinib, an oral, selective, allosteric TYK2 inhibitor, is approved in the US, EU, and other countries for the treatment of adults with moderate to severe plaque psoriasis who are candidates for systemic therapy[2-6]
- Deucravacitinib uniquely binds to the TYK2 regulatory domain rather than to the catalytic domain where Janus kinase (JAK) 1,2,3 inhibitors bind[1,7] (**Figure 1**), driving its selectivity for TYK2 and representing the first in a new class of oral drugs

Figure 1. Mechanism of action of deucravacitinib



ATP, adenosine 5′-triphosphate; JAK, Janus kinase; TYK2, tyrosine kinase 2.

- Deucravacitinib demonstrated a robust and rapid clinical efficacy and was well tolerated in the global phase 3 POETYK PSO-1 (NCT03624127) and POETYK PSO-2 (NCT03611751) trials in patients with moderate to severe plaque psoriasis[8-10]
- Scalp psoriasis, which occurs in up to 80% of patients with psoriasis and is associated with itching, flaking, pain, and bleeding, disproportionately reduces quality of life and is challenging to treat with topical agents[11-17]
- Patients with scalp psoriasis have an increased risk of developing psoriatic arthritis[18]
- The PSORIATYK SCALP (NCT05478499) trial evaluated deucravacitinib in patients with moderate to severe scalp psoriasis, including those with more limited overall psoriasis
  - PSORIATYK SCALP achieved its primary endpoint and all key secondary endpoints, with a significantly greater proportion of patients treated with deucravacitinib achieving scalp-specific Physician Global Assessment score of 0 (clear) or 1 (almost clear) (ss-PGA 0/1; 48.5%), ≥90% reduction from baseline in the Psoriasis Scalp Severity Index (PSSI 90; 38.8%), static Physician Global Assessment score of 0 (clear) or 1 (almost clear) (sPGA 0/1; 51.0%), and a mean decrease from baseline in the scalp-specific numeric rating scale (ss-NRS) itch score (-3.2) at Week 16 compared with patients receiving placebo[19]

Objective

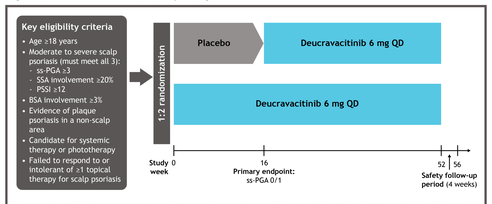
- This post hoc analysis of the PSORIATYK SCALP trial evaluated the onset of clinical response to deucravacitinib treatment in patients with moderate to severe scalp psoriasis and in subgroups of patients with total baseline BSA involvement of 3%-10% vs >10%

Methods

Study design

- PSORIATYK SCALP was a phase 3b/4, multicenter, randomized, double-blinded, placebo-controlled trial designed to evaluate the efficacy and safety of deucravacitinib in patients with moderate to severe scalp psoriasis, including those with more limited overall psoriasis (body surface area [BSA] involvement ≥3%)[18] (**Figure 2**)
  - 154 patients were randomized 1:2 to oral placebo (n = 51) or deucravacitinib 6 mg once daily (QD) (n = 103); 90% of patients in each group completed treatment at Week 16
  - At Week 16, all patients were switched to open-label deucravacitinib 6 mg QD through Week 52
  - Stratification factors were prior use of biologic therapy for psoriasis, psoriatic arthritis, or other inflammatory disease (yes/no) and body weight (≥90 kg/<90 kg)
- PSORIATYK SCALP included patients with moderate to severe scalp psoriasis defined by more focused and objective inclusion criteria (ss-PGA ≥3; scalp surface area [SSA] involvement ≥20%; PSSI ≥12) and with more limited overall psoriasis as compared with the POETYK trials[18]

Figure 2. PSORIATYK SCALP study design



BSA, body surface area; PSSI, Psoriasis Scalp Severity Index; QD, once daily; SSA, scalp surface area; ss-PGA, scalp-specific Physician Global Assessment; ss-PGA 0/1, ss-PGA score of 0 (clear) or 1 (almost clear).

Analysis populations

- Full analysis set (BSA involvement ≥3%): all patients randomized to study treatment
- Patients with BSA involvement of 3%-10%
- Patients with BSA involvement >10%

Outcomes

- In each analysis population (Weeks 0-16):
  - Achievement of ss-PGA 0/1
  - Achievement of PSSI 75, PSSI 90, and/or PSSI 100
  - Adjusted mean percent change and mean change from baseline in PSSI
  - Adjusted mean percent change and mean change from baseline in SSA involvement

Statistical analysis

- Efficacy was analyzed after all randomized patients had completed their Week 16 visit or had discontinued treatment prior to Week 16
- BSA subgroups were defined based on categorized BSA involvement at baseline: 3%-10% and >10%
- Nonresponder imputation (NRI) was used for binary efficacy endpoints for patients who had missing endpoint data
  - 95% confidence intervals (CIs) for the individual response rates were estimated using the Clopper-Pearson (exact binomial) method
  - *P* values of the odds ratios were obtained using a stratified Cochran-Mantel-Haenszel test (full analysis set) or an unstratified chi-squared test (BSA subgroups)
- Modified baseline observation carried forward (mBOCF) was used to impute missing data for continuous outcomes; patients who discontinued treatment due to lack of efficacy or adverse events had the baseline observation carried forward for all subsequent analysis weeks after the point of discontinuation, and patients who discontinued study treatment due to other reasons or a missing value had the most recent valid observation carried forward
  - Adjusted means, 95% CIs, and *P* values for the overall population were derived from an analysis of covariance model (ANCOVA) with randomization stratification factors (prior biologic use and body weight) as fixed effects and baseline value as a covariate
    - Subgroup ANCOVA values were unstratified
- All analyses are post hoc and all *P* values are nominal

Results

Baseline patient demographics and clinical characteristics

- Baseline patient demographics and clinical characteristics were similar in the placebo and deucravacitinib groups (**Table 1**)
- Mean baseline BSA involvement was similar in the placebo and deucravacitinib groups (10.0% vs 10.5%, respectively), and most patients had total BSA involvement in the 3%-10% range (placebo, 74.5%; deucravacitinib, 68.0%) (**Table 1**)
- Patients in the BSA >10% subgroup treated with deucravacitinib had longer disease duration (19.8 years [standard deviation (SD), 13.3]) compared with those receiving placebo (13.2 [SD, 9.6]) and were more likely to have had prior biologic therapy (42.4% vs 30.8%, respectively) (**Table 2**)

Table 1. Baseline patient demographics and clinical characteristics: full analysis set (BSA involvement ≥3%)

Parameter	Placebo (n = 51)	Deucravacitinib (n = 103)
Age, mean (SD), y	43.2 (13.1)	42.8 (15.7)
Weight, mean (SD), kg	88.3 (27.6)	89.3 (23.8)
Body mass index, mean (SD), kg/m²	29.2 (7.0)	30.1 (7.1)
Female, n (%)	20 (39.2)	45 (43.7)
Race, n (%)		
White	47 (92.2)	93 (90.3)
Asian	2 (3.9)	3 (2.9)
Black or African American	2 (3.9)	5 (4.9)
Other	0	2 (1.9)
Psoriasis vulgaris duration, mean (SD), y	11.9 (9.8)	16.1 (11.4)
Scalp psoriasis duration, mean (SD), y	12.4 (9.6)	16.4 (11.7)
Prior systemic therapy, n (%)		
Yes	27 (52.9)	54 (52.4)
Biologic	16 (31.4)	37 (35.9)
Nonbiologic	11 (21.6)	17 (16.5)
No	24 (47.1)	49 (47.6)
ss-PGA score, n (%)		
3 (moderate)	32 (62.7)	76 (73.8)
4 (severe)	19 (37.3)	27 (26.2)
PSSI, mean (SD)	32.2 (13.7)	33.5 (12.5)
ss-NRS itching score, mean (SD)	6.4 (1.8)	6.4 (2.3)
sPGA score, n (%)		
2 (mild)	4 (7.8)	7 (6.8)
3 (moderate)	42 (82.4)	81 (78.6)
4 (severe)	5 (9.8)	15 (14.6)
SSA involvement, mean (SD), %	53.0 (24.0)	57.6 (23.1)
BSA involvement, mean (SD), %	10.0 (8.1)	10.5 (9.6)
3%-10%, n (%)	38 (74.5)	70 (68.0)
>10%, n (%)	13 (25.5)	33 (32.0)
PASI, mean (SD)	9.4 (5.6)	10.2 (6.7)

BSA, body surface area; PASI, Psoriasis Area and Severity Index; PSSI, Psoriasis Scalp Severity Index; SD, standard deviation; sPGA, static Physician Global Assessment; SSA, scalp surface area; ss-NRS, scalp-specific numeric rating scale; ss-PGA, scalp-specific Physician Global Assessment.

Table 2. Baseline patient demographics and clinical characteristics by baseline BSA involvement: 3%-10% vs >10%

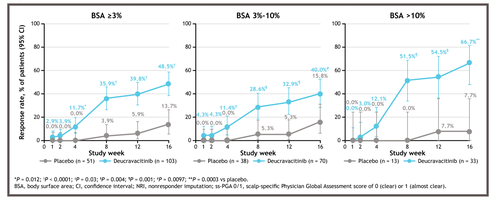
Parameter	Baseline BSA involvement			
	3%-10%		>10%	
	Placebo (n = 38)	Deucravacitinib (n = 70)	Placebo (n = 13)	Deucravacitinib (n = 33)
Age, mean (SD), y	41.8 (13.1)	42.0 (15.2)	47.5 (12.5)	44.3 (16.9)
Weight, mean (SD), kg	86.3 (24.3)	88.7 (25.0)	93.8 (36.2)	90.6 (21.3)
Body mass index, mean (SD), kg/m²	28.7 (6.4)	29.8 (7.3)	30.8 (8.8)	30.6 (6.5)
Female, n (%)	18 (47.4)	33 (47.1)	2 (15.4)	12 (36.4)
Race, n (%)				
White	36 (94.7)	64 (91.4)	11 (84.6)	29 (87.9)
Asian	1 (2.6)	1 (1.4)	1 (7.7)	2 (6.1)
Black or African American	1 (2.6)	3 (4.3)	1 (7.7)	2 (6.1)
Other	0	2 (2.9)	0	0
Psoriasis vulgaris duration, mean (SD), y	11.4 (9.9)	14.4 (10.1)	13.2 (9.6)	19.8 (13.3)
Scalp psoriasis duration, mean (SD), y	12.0 (9.7)	15.4 (11.1)	13.7 (9.8)	18.5 (12.9)
Prior systemic therapy, n (%)				
Yes	16 (42.1)	35 (50.0)	11 (84.6)	19 (57.6)
Biologic	12 (31.6)	23 (32.9)	4 (30.8)	14 (42.4)
Nonbiologic	4 (10.5)	12 (17.1)	7 (53.9)	5 (15.2)
No	22 (57.9)	35 (50.0)	2 (15.4)	14 (42.4)
ss-PGA score, n (%)				
3 (moderate)	23 (60.5)	56 (80.0)	9 (69.2)	20 (60.6)
4 (severe)	15 (39.5)	14 (20.0)	4 (30.8)	13 (39.4)
PSSI, mean (SD)	32.4 (13.8)	32.1 (11.8)	31.5 (14.0)	36.5 (13.7)
ss-NRS itching score, mean (SD)	6.4 (1.8)	6.3 (2.4)	6.5 (2.0)	6.5 (2.0)
sPGA score, n (%)				
2 (mild)	4 (10.5)	6 (8.6)	0	1 (3.0)
3 (moderate)	31 (81.6)	56 (80.0)	11 (84.6)	25 (75.8)
4 (severe)	3 (7.9)	8 (11.4)	2 (15.4)	7 (21.2)
SSA involvement, mean (SD), %	54.4 (24.4)	53.8 (22.5)	48.8 (23.0)	65.6 (22.7)
BSA involvement, mean (SD), %	6.1 (2.1)	5.8 (2.0)	21.5 (8.1)	20.5 (11.5)
PASI, mean (SD)	7.0 (3.1)	6.9 (3.1)	16.5 (5.1)	17.4 (6.6)

BSA, body surface area; NRS, numeric rating scale; PASI, Psoriasis Area and Severity Index; PSSI, Psoriasis Scalp Severity Index; SD, standard deviation; sPGA, static Physician Global Assessment; ss-NRS, scalp-specific NRS; ss-PGA, scalp-specific Physician Global Assessment.

ss-PGA 0/1

- In patients with BSA involvement ≥3% and in the BSA 3%-10% subgroup, a significant difference from placebo was seen by Week 4 (**Figure 3**)
  - In patients with BSA involvement of >10%, a significant difference was seen by Week 8

Figure 3. ss-PGA 0/1 response rates by baseline BSA involvement, Weeks 0-16 (NRI)

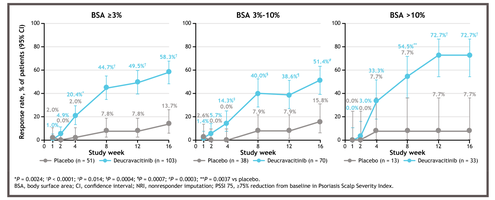


*P = 0.012; †P < 0.0001; ‡P = 0.0213; §P = 0.0046; ¶P < 0.0001; #P = 0.0097; **P = 0.0003 vs placebo.
BSA, body surface area; CI, confidence interval; NRI, nonresponder imputation; ss-PGA 0/1, scalp-specific Physician Global Assessment score of 0 (clear) or 1 (almost clear).

PSSI

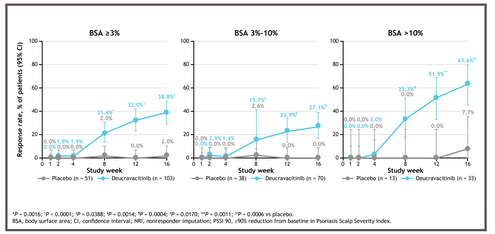
- For patients with BSA involvement ≥3% and those in the BSA 3%-10% subgroup, a significant difference from placebo in PSSI 75 rates was seen by Week 4 (**Figure 4**)
  - A significant difference was seen by Week 8 in the BSA >10% subgroup
- The onset of clinical response was similar for all groups for PSSI 90 (Week 8), PSSI 100 (Week 12), and percent change from baseline PSSI (Week 4) (**Figures 5-7**)
- For mean change from baseline PSSI, patients with BSA involvement ≥3% and patients in the BSA 3%-10% subgroup saw an earlier onset of response (Week 1) compared with those with BSA >10% (Week 2) (**Figure 8**)

Figure 4. PSSI 75 response rates by baseline BSA involvement, Weeks 0-16 (NRI)



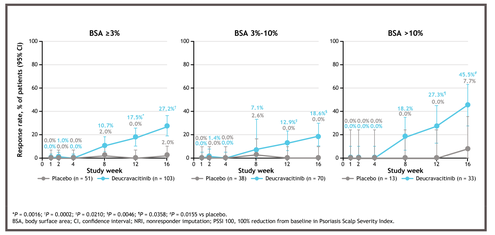
*P = 0.0024; †P < 0.0001; ‡P = 0.014; §P = 0.0004; ¶P = 0.0007; #P = 0.0003; **P = 0.0037 vs placebo.
BSA, body surface area; CI, confidence interval; NRI, nonresponder imputation; PSSI 75, ≥75% reduction from baseline in Psoriasis Scalp Severity Index.

Figure 5. PSSI 90 response rates by baseline BSA involvement, Weeks 0-16 (NRI)



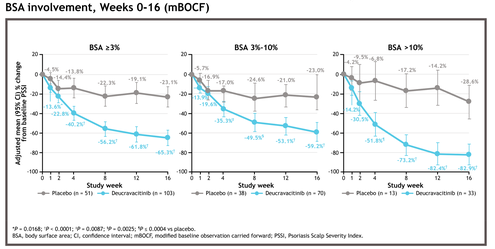
*P = 0.0016; †P < 0.0001; ‡P = 0.0388; §P = 0.0014; ¶P = 0.0004; #P = 0.0170; **P = 0.0011; ††P = 0.0006 vs placebo.
BSA, body surface area; CI, confidence interval; NRI, nonresponder imputation; PSSI 90, ≥90% reduction from baseline in Psoriasis Scalp Severity Index.

Figure 6. PSSI 100 response rates by baseline BSA involvement, Weeks 0-16 (NRI)



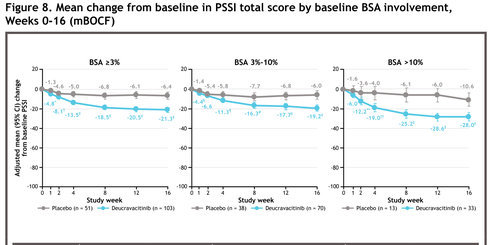
*P = 0.0016; †P = 0.0002; ‡P = 0.0210; §P = 0.0046; ¶P = 0.0358; #P = 0.0155 vs placebo.
BSA, body surface area; CI, confidence interval; NRI, nonresponder imputation; PSSI 100, 100% reduction from baseline in Psoriasis Scalp Severity Index.

Figure 7. Mean percent change from baseline in PSSI total score by baseline BSA involvement, Weeks 0-16 (mBOCF)



*P = 0.0016; †P < 0.0001; ‡P = 0.0087; §P = 0.0025; ¶P = 0.0004 vs placebo.
BSA, body surface area; CI, confidence interval; mBOCF, modified baseline observation carried forward; PSSI, Psoriasis Scalp Severity Index.

Figure 8. Mean change from baseline in PSSI total score by baseline BSA involvement, Weeks 0-16 (mBOCF)



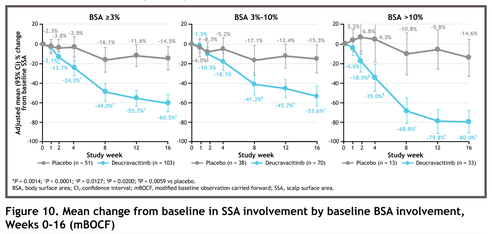
Baseline	BSA ≥3%		BSA 3%-10%		BSA >10%	
	Placebo	Deucravacitinib	Placebo	Deucravacitinib	Placebo	Deucravacitinib
Mean (SD)	32.2 (13.7)	33.4 (12.7)	32.4 (13.8)	32.1 (11.8)	31.5 (14.0)	36.2 (14.3)

*P = 0.0049; †P = 0.0364; ‡P < 0.0001; §P = 0.0259; ¶P = 0.0099; #P = 0.0010; **P = 0.0251; ††P = 0.0004 vs placebo.
BSA, body surface area; CI, confidence interval; mBOCF, modified baseline observation carried forward; PSSI, Psoriasis Scalp Severity Index; SD, standard deviation.

SSA involvement

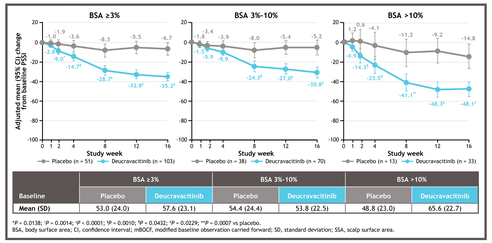
- Significant differences from placebo were seen by Week 2 in the BSA >10% subgroup, Week 4 in the BSA ≥3% subgroup, and Week 8 in the BSA 3%-10% subgroup for both percent and mean change from baseline SSA involvement (**Figure 9** and **Figure 10**)

Figure 9. Mean percent change from baseline in SSA involvement by baseline BSA involvement, Weeks 0-16 (mBOCF)



*P < 0.0014; †P < 0.0001; ‡P = 0.0127; §P = 0.0200; ¶P < 0.0059 vs placebo.
BSA, body surface area; CI, confidence interval; mBOCF, modified baseline observation carried forward; SSA, scalp surface area.

Figure 10. Mean change from baseline in SSA involvement by baseline BSA involvement, Weeks 0-16 (mBOCF)



Baseline	BSA ≥3%		BSA 3%-10%		BSA >10%	
	Placebo	Deucravacitinib	Placebo	Deucravacitinib	Placebo	Deucravacitinib
Mean (SD)	53.0 (24.0)	57.6 (23.1)	54.4 (24.4)	53.8 (22.5)	48.8 (23.0)	65.6 (22.7)

*P < 0.0138; †P < 0.0014; ‡P < 0.0001; §P = 0.0010; ¶P = 0.0432; #P = 0.0229; **P = 0.0007 vs placebo.
BSA, body surface area; CI, confidence interval; mBOCF, modified baseline observation carried forward; SD, standard deviation; SSA, scalp surface area.

Conclusions

- In the phase 3b/4 PSORIATYK SCALP trial, deucravacitinib significantly improved scalp-specific efficacy outcomes compared with placebo as early as Week 4, including in patients with more limited overall psoriasis (BSA involvement ≥3%)
- Greater and more rapid improvements were seen in both BSA subgroups (3%-10% and >10%) with deucravacitinib vs placebo from baseline through Week 16
  - Differences in responses in the BSA subgroups were likely driven by the differences in baseline severity

References

1. Burke JR, et al. *Sci Transl Med*. 2019;11:eaaw1736. **2.** Sotyktu [package insert]. Princeton, NJ: Bristol Myers Squibb; September 2022. **3.** Sotyktu [European summary of product characteristics]. Dublin, Ireland: Bristol Myers Squibb EEIG; December 2023. **4.** Sotyktu [package insert]. Tokyo, Japan: Bristol Myers Squibb K.K.; September 2022. **5.** Sotyktu [product monograph]. Montreal, QC, Canada: Bristol Myers Squibb Canada Co.; November 2022. **6.** Sotyktu [product information]. Mulgrave, VIC, Australia: Bristol Myers Squibb Australia Pty. Ltd.; December 2022. **7.** Wrobleski ST, et al. *J Med Chem*. 2019;62:8973-8995. **8.** Armstrong AW, et al. *J Am Acad Dermatol*. 2023;88:29-39. **9.** Strober B, et al. *J Am Acad Dermatol*. 2023;88:40-51. **10.** Korman NJ, et al. *J Derm Treatment*. 2024;35:2371045. **11.** Kragballe K. *Curr Probl Dermatol*. 2009;38:160-171. **12.** Chan CS, et al. *J Am Acad Dermatol*. 2009;60:962-971. **13.** Rich P, et al. *J Am Acad Dermatol*. 2016;74:134-142. **14.** Papp K, et al. *J Eur Acad Dermatol Venereol*. 2007;21:1151-1160. **15.** Kragballe K, et al. *J Dermatol Treat*. 2013;24:188-192. **16.** Mason AR, et al. *Br J Dermatol*. 2013;169:519-527. **17.** Blauvelt A, et al. *J Am Acad Dermatol*. 2024;90:775-782. **18.** Wilson FC, et al. *Arthritis Rheum*. 2009;61:233-239. **19.** Callis Duffin K, et al. Presented at the 33rd EADV Congress; 25-28 September 2024; Amsterdam, Netherlands.

Acknowledgments

- This study was sponsored by Bristol Myers Squibb
- Writing and editorial assistance was provided by Kimberly MacKenzie, PhD, of Peloton Advantage, LLC, an OPEN Health company, funded by Bristol Myers Squibb

Disclosures

- **MH:** Advisory boards: AbbVie, Almirall, Dermasence, Galderma, Incyte, Janssen, Leo Pharma, Leti Pharma, Novartis, Pfizer, Sanofi, and UCB; Consultant: AbbVie, Almirall, Dermasence, Galderma, Incyte, Janssen, Leo Pharma, Leti Pharma, Novartis, Pfizer, Sanofi, and UCB; Investigator: AbbVie, ALK, Almirall, Alumis, Amgen, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, Dermasence, Dermira, Eli Lilly, Forward, Galderma, Incyte, Janssen, Kinnikua, Leo Pharma, Leti Pharma, Meda Pharma, Medac, Meiji Pharma, MSD, Nektar Therapeutics, Novartis, Pierre Fabre, Pfizer, Sanofi, Schering-Plough, Smerud Medical Research, Stallergenes Greer, and UCB
- **AB:** Speaker (with honoraria): Eli Lilly and UCB; Scientific adviser (with honoraria): AbbVie, Almirall, Alumis, Amgen, Anaptysbio, Apogee, Arcutis, Boehringer Ingelheim, Bristol Myers Squibb, Celltrion, Corvus, Dermavant, Eli Lilly, Galderma, GlaxoSmithKline, Incyte, IQVIA, Janssen, Leo Pharma, Lipidio, Merck, Novartis, Oruka, Paragon, Pfizer, Regeneron, Sanofi, Spherix Global Insights, Sun Pharma, Takeda, UCB, and Union; Clinical study investigator (institution received clinical study funds): AbbVie, Acelyrin, Almirall, Alumis, Amgen, Arcutis, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, Eli Lilly, Galderma, Incyte, Janssen, Leo Pharma, Merck, Novartis, Pfizer, Regeneron, Sanofi, Sun Pharma, Takeda, and UCB; Stock owner: Lipidio and Oruka
- **EB, AN, C-YC, RD,** and **VH:** Employees and shareholders: Bristol Myers Squibb
- **ST:** Clinical trial investigator: Bristol Myers Squibb
- **JB:** Research funds payable to the Psoriasis Treatment Center of New Jersey: AbbVie, Amgen, Arcutis, Boehringer Ingelheim, Bristol Myers Squibb, CorEvitas Psoriasis Registry, Dermavant, Dermira/UCB, Eli Lilly, Glenmark, Janssen Biotech, Kadmon, Leo Pharma, Lycera, Menlo Therapeutics, Novartis, Pfizer, Regeneron, Sun Pharma, Taro, and Valeant; Consultant: AbbVie, Amgen, Eli Lilly, Janssen Biotech, Novartis, Sun Pharma, and Valeant; Speaker: AbbVie, Eli Lilly, Janssen Biotech, and Novartis
- **ML:** Received research funds on behalf of Mount Sinai: AbbVie, Amgen, Arcutis, Avotres, Boehringer Ingelheim, Cara Therapeutics, Dermavant, Eli Lilly, Incyte, Janssen, Ortho Dermatologics, Regeneron, and UCB; Consultant: Almirall, AltruBio, AnaptysBio, Arcutis, Avotres, Brickell Biotech, Boehringer Ingelheim, Bristol Myers Squibb, Castle Biosciences, Celltrion, CorEvitas Psoriasis Registry, Dermavant, EPI Health, Evommune, Forte Biosciences, Galderma, Genentech, Incyte, Leo Pharma, Meiji Seika Pharma, Mindera Health, Pfizer, Seanergy, Strata, Trevi, and Verrica

Presented at the Winter Clinical Dermatology Conference - Hawaii*; February 14-19, 2025; Waikoloa, HI

Email: drhoff68@t-online.de