Two Phase 3, Randomized, Placebo-Controlled Trials of Povorcitinib for Nonsegmental Vitiligo: Results From STOP-V1 and STOP-V2

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Objective

- To evaluate the efficacy and safety of povorcitinib 30 mg in adults with moderate-to-severe[8] NSV over 52 weeks in 2 parallel phase 3 studies: STOP-V1 (NCT06113445) and STOP-V2 (NCT06113471)

Conclusions

- **In adults with moderate-to-severe NSV, povorcitinib 30 mg QD demonstrated statistically significant and clinically meaningful improvements vs placebo over 52 weeks in both STOP-V1 and STOP-V2, including F-VASI75, facial VNS of "a lot less noticeable" or "no longer noticeable," T-VASI50, and percentage change in T-VASI**
- **Improvements in F-VASI and T-VASI with povorcitinib continued through Week 52 without plateau, suggesting that further repigmentation may be observed with additional treatment**
- **Povorcitinib 30 mg QD was well tolerated, with results consistent with its known safety profile[6,9,10]**
- **These findings demonstrate that povorcitinib is an effective systemic treatment option for adults with moderate-to-severe NSV**

Abbreviations

ALT, alanine aminotransaminase; AST, aspartate aminotransferase; BSA, body surface area; CPK, creatine phosphokinase; F-BSA, facial body surface area; F-VASI, facial Vitiligo Area Scoring Index; F-VASI75, ≥75% improvement from baseline in F-VASI; GGT, gamma-glutamyl transferase; IFN-γ, interferon gamma; JAK, Janus kinase; LSM, least squares mean; NSV, nonsegmental vitiligo; QD, once daily; STAT, signal transducer and activator of transcription; T-BSA, total body surface area; T-VASI, total Vitiligo Area Scoring Index; T-VASI50/75, ≥50%/≥75% improvement from baseline in T-VASI; TEAE, treatment-emergent adverse event; UTI, urinary tract infection; VNS, Vitiligo Noticeability Scale.

Disclosures

KE is a consultant for AbbVie, Incyte Corporation, La Roche-Posay, Pfizer, Pierre Fabre, Sanofi, and Valeo Bio; HL has served as an advisor, consultant, investigator, and/or speaker for AbbVie, Incyte Corporation, L'Oréal, Pfizer, and Vita Imaging; VE has served as a consultant, investigator, and/or speaker for AbbVie, Almirall, Belenstof, Boehringer Ingelheim, Cerahle, Galderma, Genesis Pharma, Incyte Corporation, La Roche-Posay, L'Oréal, Merck/MSD, Novartis, Pfizer, and Takeda; is the representative for clinical trials of the Research Subcommittee of the Executive Committee of the British Association of Dermatologists (BAD); is a West Midlands BAD Representative (trustee of the BAD); has held a position of interest for the Trial Generation and Prioritisation Panel (UK Dermatology Clinical Trials Network); and is the NIHR West Midlands RIEON Dermatology Specialty Lead. SRD has received fees and/or honoraria as a consultant for Almirall, Avita, Bristol Myers Squibb, Casiopea SpA, Dermavant Sciences, Derma, Ferndale Laboratories, Foamix, Galderma Laboratories LP, Incyte Corporation, MC2 Therapeutics, Ortho Dermatologics, Pfizer, Scientis, Sente Labs, SkinCeuticals LLC, UCB, and Vertex Pharmaceuticals; has received stock options as a consultant for Core Range Capital; has received honoraria as a speaker for Almirall and Ortho Dermatologics; has received grants/research funding as an investigator for AbbVie, ACBiome LLC, Atacama Therapeutics, Brickell Biotech, Dermavant Sciences, Incyte Corporation, Novan, and Sonoma Biotherapeutics; has served as an advisory board member for Almirall, Avita, Dermavant Sciences, Ferndale Laboratories, Foamix, Galderma Laboratories LP, Incyte Corporation, Novan, and Sonoma Biotherapeutics; has stock/stock options in Brickell Biotech, Atlas Immunotherapeutics, and Sonoma Biotherapeutics. PG has served as a speaker for Bristol Myers Squibb, Cerave, Galderma, GSK, Lilly, Pfizer, Sanofi, Sun Pharma, and UCB. JRL has received honoraria from AbbVie, Alumis, Amgen, Apogee, Arcutis, ASLAN, Bristol Myers Squibb, Celgene, Concert Pharmaceuticals, Dice Therapeutics, Eli Lilly, Incyte Corporation, Innovaderm, Janssen, Kymera, Kyowa Kirin, LEO Pharma, Merck, Novartis, Oculis, Pfizer, Sanofi Genzyme, Sun Pharma, Takeda, and UCB; has received grants and/or is a consultant and/or investigator for AbbVie, Boehringer Ingelheim, Bristol Myers Squibb, First Tracks Biotherapeutics, Idorsia, Incyte Corporation, Merck/MSD, Novartis, Pfizer, Takeda, and Teva and is chair of the Vitiligo Task Force for the European Academy of Dermatology and Venereology (EADV). AW has served as consultant and/or investigator for AbbVie, Incyte Corporation, Merck/MSD, Novartis, Pfizer, Philips, and UCB; has received equipment from Humeca and Perfliction; and has served on advisory boards for AbbVie and Incyte Corporation. CL has served as a speaker or consultant or advisor or investigator or received honoraria from AbbVie, Allius, Amgen, Arpeal, Arcutis, Bausch Health, Bayer, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Cipher, Dermavant, Dermirian, Eli Lilly, Fresenius Kabi, Galderma, GSK, Innovaderm, Intrigg Skin, Janssen, Kyowa Kirin, La Roche-Posay, LEO Pharma, L'Oréal, Medexus, Merck, Merck, Novartis, P&G, Pedopharm, Pfizer, Pierre Fabre, Sanofi, Sun Pharma, Takeda, Teva, UCB, and Vyne Therapeutics. MA has served as a consultant, speaker, or investigator for AbbVie, Almirall, Amgen, Arena, Astria, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Concert, CSL Behring, Biogen, Boehringer Ingelheim, Celgene, Centocor, Eli Lilly, GSK, Hexal, Janssen, LEO Pharma, Medac, Merck, MSD, Mundipharma, Novartis, Pfizer, Sandoz, and UCB. JLLE has served as a consultant, participated in clinical trials, and/or received lecture fees from AbbVie, Almirall, Babé, Bioderma, Galderma, Incyte Corporation, Invesa, Isdin, Janssen, LEO Pharma, Lilly, Novartis, Pierre Fabre, and UCB. PG has served as a consultant for Aclaris Therapeutics, Clarity Medical, Dermaframe, Incyte Corporation, Procter & Gamble, and Verocytar Technologies and as a principal investigator for Aclaris Therapeutics, Allergan/SkinMedica, Cinuvel Pharmaceuticals, Incyte Corporation, Johnson & Johnson, L'Oréal, Merz Pharma, Pfizer, Thyric Global Inc., and VT Cosmetics. DR has served as a consultant, speaker, or investigator for AbbVie, Alcura, Almirall, Altrubio, Amgen, Arena, Astria, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Concert, CSL Behring, Dermavant Sciences, Derma, Dualtas, EMD Serono, Galderma. Incyte Corporation, Janssen, Kymera, Kyowa Kirin, Lilly, Merck, Nektar, Novartis, Pfizer, RAPT Therapeutics, Reckitt, Regeneron, Revolo Biotherapeutics, Sanofi, Sun Pharma, UCB, Vaxa Bio, and Zura Bio. KB, SW, DK, and DK are employees and shareholders of Incyte Corporation. AGP has served as an investigator for Avita, Clinuvel, and Incyte Corporation; has served as a consultant for AbbVie, Avita, Boehringer Ingelheim, Incyte Corporation, Lilly, Pfizer, Sun Pharma, Thelocan, Villana, Vimela, Vyne, and WCG; and is a shareholder of Tara Medical.

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Introduction

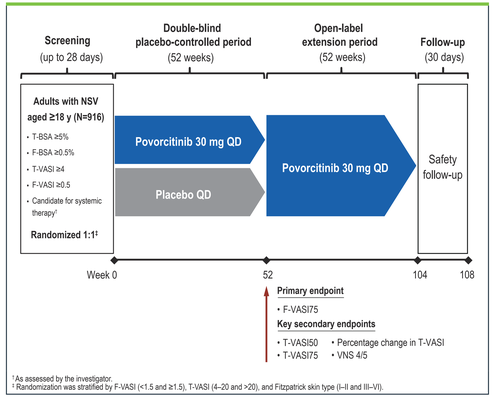
- Melanocyte loss in vitiligo is largely driven through the activation of the JAK-STAT signaling pathway by IFN-γ, leading to chemokine production and recruitment of cytotoxic T cells[1]
- Ruxolitinib (selective JAK1/JAK2 inhibitor) cream was the first approved treatment for repigmentation in NSV[2] and is indicated for patients aged ≥12 years with ≤10% affected BSA in the US and other regions[3,4]
- Povorcitinib (oral, highly selective JAK1 inhibitor)[5] demonstrated substantial repigmentation over 52 weeks and was well tolerated in a phase 2 dose-ranging study (15, 45, and 75 mg QD) in adults with more extensive NSV[6]
  - Povorcitinib 30 mg QD, a dose not tested in phase 2, was selected for use in phase 3 studies based on exposure-response modeling[7]

Methods

Patients and Study Design

- STOP-V1 and STOP-V2 were identically designed phase 3, randomized, double-blind, placebo-controlled trials of QD oral povorcitinib in adults with moderate-to-severe NSV (**Figure 1**)

Figure 1. STOP-V Study Design



† As assessed by the investigator.
‡ Randomization was stratified by F-VASI (<1.5 and ≥1.5), T-VASI (4–20 and >20), and Fitzpatrick skin type (I–II and III–VI).

Statistical Analyses

- For primary and key secondary endpoints, patients who initiated prohibited therapy with potential impact on vitiligo outcomes before Week 52 were considered nonresponders (binary outcomes) or imputed using the baseline score (T-VASI percentage change from baseline); patients with missing data at Week 52 were imputed using multiple imputation
- All other outcomes are reported as observed and summarized using descriptive statistics

Results

Patients

- Demographics and baseline clinical characteristics were generally similar across treatment groups and studies (**Table 1**)

Table 1. Patient Demographics and Baseline Clinical Characteristics

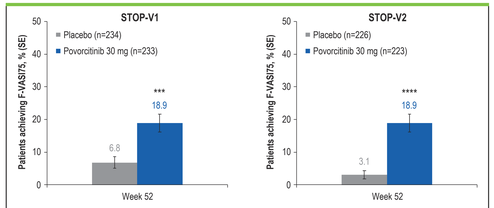
	STOP-V1		STOP-V2	
Characteristic	Placebo (n=234)	Povorcitinib 30 mg (n=233)	Placebo (n=226)	Povorcitinib 30 mg (n=223)
Age, median (range), y	50.0 (19–80)	46.0 (18–89)	46.0 (19–72)	47.0 (18–79)
Female, n (%)	127 (54.3)	130 (55.8)	125 (55.3)	131 (58.7)
Race, n (%)				
White	187 (79.9)	184 (79.0)	193 (85.4)	188 (84.3)
Asian	27 (11.5)	24 (10.3)	13 (5.8)	25 (11.2)
Black	7 (3.0)	13 (5.6)	15 (6.6)	5 (2.2)
Hispanic/Latino, n (%)	42 (17.9)	42 (18.0)	26 (11.5)	31 (13.9)
Fitzpatrick skin type, n (%)				
I	11 (4.7)	17 (7.3)	5 (2.2)	3 (1.3)
II	88 (37.6)	82 (35.2)	66 (29.2)	69 (30.9)
III	83 (35.5)	77 (33.0)	97 (42.9)	86 (38.6)
IV	37 (15.8)	42 (18.0)	37 (16.4)	44 (19.7)
V	6 (2.6)	13 (5.6)	16 (7.1)	18 (8.1)
VI	8 (3.4)	2 (0.9)	5 (2.2)	3 (1.3)
F-VASI, mean (SD)	1.06 (0.61)	1.04 (0.61)	1.18 (0.67)	1.16 (0.74)
<1.5, n (%)	181 (77.4)	178 (76.4)	162 (71.7)	159 (71.3)
≥1.5, n (%)	53 (22.6)	55 (23.6)	64 (28.3)	64 (28.7)
T-VASI, mean (SD)	19.5 (15.0)	19.5 (15.5)	23.0 (19.6)	23.1 (18.7)
4–20, n (%)	153 (65.4)	155 (66.5)	130 (57.5)	132 (59.2)
>20, n (%)	81 (34.6)	78 (33.5)	96 (42.5)	91 (40.8)
Disease duration, median (range), y	15.2 (0.1–57.7)	17.8 (0.1–61.9)	19.2 (0.1–55.4)	17.2 (0.2–59.3)
Active disease, n (%)†	100 (42.7)	81 (34.8)	62 (27.4)	70 (31.4)
Prior medication for vitiligo, n (%)	95 (40.6)	88 (37.8)	93 (41.2)	96 (43.0)

† Signs of active disease included the presence of confetti pigmentation, trichrome appearance of vitiligo, and Koebner phenomenon.

Efficacy

- Significantly more patients achieved the primary endpoint of F-VASI75 at Week 52 with povorcitinib vs placebo in both studies (**Figure 2**)
- Patients had greater mean percentage improvements from baseline in F-VASI with povorcitinib vs placebo at Week 52 (STOP-V1, –36.8% vs –10.1%; STOP-V2, –36.4% vs –8.3%)

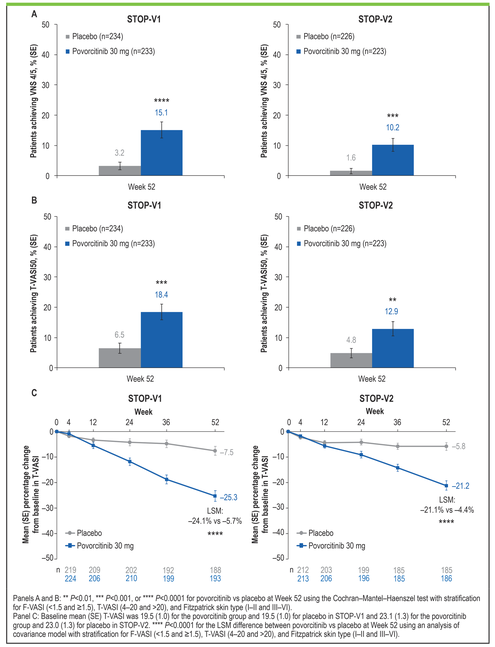
Figure 2. Primary Endpoint: Achievement of F-VASI75



*** $P<0.001$ or **** $P<0.0001$ for povorcitinib vs placebo at Week 52 using the Cochran–Mantel–Haenszel test with stratification for F-VASI (<1.5 and ≥1.5), T-VASI (4–20 and >20), and Fitzpatrick skin type (I–II and III–VI).

- Significantly more patients achieved facial VNS 4/5 (**Figure 3A**) and T-VASI50 (**Figure 3B**) at Week 52 with povorcitinib vs placebo in both studies
- Few patients achieved T-VASI75 at Week 52 (STOP-V1, 2.6% vs 1.3% [P=0.35]; STOP-V2, 2.3% vs 0% [P<0.05])
  - Longer treatment duration may be needed to achieve this stringent threshold for repigmentation
- Patients had significantly greater mean percentage improvement from baseline in T-VASI at Week 52 with povorcitinib vs placebo in both studies and showed continued improvement over 52 weeks without plateau (**Figure 3C**)
- In post hoc analyses, more patients who received povorcitinib vs placebo had no increase in T-VASI at Week 52 in both studies, indicating no worsening or progression of disease (STOP-V1, 94.3% vs 71.8%; STOP-V2, 87.6% vs 69.2%)

Figure 3. Key Secondary Endpoints: (A) Achievement of VNS 4/5 (A Lot Less or No Longer Noticeable), (B) Achievement of T-VASI50, and (C) Percentage Change From Baseline in T-VASI



Panels A and B: ** $P<0.01$, *** $P<0.001$, or **** $P<0.0001$ for povorcitinib vs placebo at Week 52 using the Cochran–Mantel–Haenszel test with stratification for F-VASI (<1.5 and ≥1.5), T-VASI (4–20 and >20), and Fitzpatrick skin type (I–II and III–VI).
Panel C: Baseline mean (SE) T-VASI was 19.5 (1.0) for the povorcitinib group and 19.5 (1.0) for placebo in STOP-V1 and 23.1 (1.3) for the povorcitinib group and 23.0 (1.3) for placebo in STOP-V2. **** $P<0.0001$ for the LSM difference between povorcitinib vs placebo at Week 52 using an analysis of covariance model with stratification for F-VASI (<1.5 and ≥1.5), T-VASI (4–20 and >20), and Fitzpatrick skin type (I–II and III–VI).

- Progressive facial and body repigmentation was observed in patients treated with povorcitinib (**Figure 4**)

Figure 4. Selected Clinical Images of (A) Facial and (B) Body Repigmentation in Patients Treated With Povorcitinib

Safety

- TEAEs were consistent between studies, with no new safety signals identified (**Table 2**)
  - Serious TEAEs were generally single events
  - Increased blood CPK was the most common grade ≥3 TEAE; none were serious
- There were no clinically meaningful trends in ALT/AST or hematology parameters associated with povorcitinib and no indicators of drug-induced liver injury
- JAK inhibitor class–specific TEAEs were absent or rare with povorcitinib

Table 2. Summary of TEAEs Through Week 52

	STOP-V1		STOP-V2	
n (%)	Placebo (n=234)	Povorcitinib 30 mg (n=233)	Placebo (n=225)	Povorcitinib 30 mg (n=222)
Any TEAE	149 (63.7)	160 (68.7)	141 (62.7)	148 (66.7)
Most common TEAEs†				
Nasopharyngitis	23 (9.8)	26 (11.2)	25 (11.1)	25 (11.3)
Acne	7 (3.0)	25 (10.7)	5 (2.2)	24 (10.8)
Upper respiratory tract infection	21 (9.0)	16 (6.9)	24 (10.7)	31 (14.0)
Blood CPK increased	8 (3.4)	16 (6.9)	15 (6.7)	14 (6.3)
Headache	13 (5.6)	12 (5.2)	13 (5.8)	15 (6.8)
Any treatment-related TEAE	50 (21.4)	72 (30.9)	42 (18.7)	74 (33.3)
Any grade ≥3 TEAE‡	14 (6.0)	21 (9.0)	24 (10.7)	20 (9.0)
Any serious TEAE§	5 (2.1)	5 (2.1)	7 (3.1)	8 (3.6)
Any fatal TEAE	0	0	0	0
Any TEAE leading to discontinuation of study drug¶	10 (4.3)	12 (5.2)	5 (2.2)	14 (6.3)

† Occurring in >5% of patients in the povorcitinib group in either study.
‡ Grade ≥3 TEAEs considered related to povorcitinib treatment were blood CPK increased (n=3), neutropenia (n=2), GGT increased (n=1), pneumonia (n=1), and UTI (n=1) in STOP-V1 and neutropenia (n=2), deep vein thrombosis (n=1), pulmonary embolism (n=1), and thrombosed varicose vein (n=1) in STOP-V2.
§ Serious TEAEs considered related to povorcitinib treatment were pneumonia (n=1) and UTI (n=1) in STOP-V1 and basal cell carcinoma (n=1) and pulmonary embolism (n=1) in STOP-V2.
¶ Only 1 TEAE led to discontinuation of povorcitinib in ≥1 patient in either study (blood CPK increased; n=4 in STOP-V1). Discontinuations due to increased CPK events were based on prespecified stopping criteria.