Deucravacitinib (SOTYKTU) in Psoriatic Arthritis
National Drug Mini-Monograph

June 2026

VA Pharmacy Benefits Management Services and National Formulary Committee

The purpose of VA PBM Services drug monographs is to provide a focused drug review for making formulary decisions. The Product Information or other resources should be consulted for detailed and most current drug information.

Abbreviations: AAU, acute anterior uveitis; AC, active-controlled; ACR20, ≥ 20% improvement from baseline on the American College of Rheumatology criteria; ARD, absolute risk difference; AS, ankylosing spondylitis; bDMARD, biologic disease-modifying antirheumatic drug; BIO, biologic; BMS, Bristol Myers Squibb; CO, crossover; csIMM, conventional synthetic immunomodulator/immunosuppressant such as methotrexate and azathioprine, etc.; CTLA4-Ig, cytotoxic T-lymphocyte antigen-4 immunoglobulin; DB, double-blind; DC, dose-controlled; FAERS, FDA Adverse Event Reporting System; hs-CRP, high-sensitivity C-reactive protein; GRADE, Grading of Recommendations, Assessment, Development, and Evaluation; HI, hepatic impairment; IBD, inflammatory bowel disease; -IR, inadequate response/responder; LD, loading dose; LEF, leflunomide; mAb, monoclonal antibody; MN, multinational; MTX, methotrexate; nbIMM, nonbiologic immunomodulator/immunosuppressant; OLE, open-label extension; OSM, oral small molecule (methotrexate, sulfasalazine, cyclosporine, leflunomide, apremilast – excludes tofacitinib); PC, placebo-controlled; pp, percentage points; PsA, psoriatic arthritis; PsO, psoriasis; RCT, randomized clinical trial; ROR, reporting odds ratio; SJC, swollen joint count; SSZ, sulfasalazine; TG, triglycerides; TJC, tender joint count

SUMMARY OF PRESCRIBING INFORMATION[1]	
Description / MOA	Tyrosine kinase 2 (TYK2) inhibitor previously approved for treatment of moderate-to-severe plaque psoriasis. It binds to the regulatory (JH2) domain of TYK2, stabilizing it in an inactive conformation—blocking IL-23, IL-12, and type I interferon signaling—while sparing JAK1, JAK2, and JAK3 pathways. It is the first TYK2 inhibitor and fourth oral agent FDA-approved for psoriatic arthritis (PsA), following apremilast, tofacitinib, and upadacitinib.
Indication Under Review	Treatment of active PsA in adults
Dosage Regimen	6 mg PO QD with or without food
Dosage Forms Under Review	6-mg tablet
Boxed Warnings	None
Contraindications	History of hypersensitivity reaction to deucravacitinib or product excipients
Other Warnings	□ Infections: Avoid in patients with active or serious infection. □ Tuberculosis (TB): Evaluate for latent or active TB before initiating therapy. □ Viral Reactivation. Consider viral hepatitis screening before initiating therapy. Not recommended in patients with active hepatitis B or hepatitis C. □ Malignancies, including lymphomas, were observed in clinical trials. Consider risk-benefits before initiating or continuing therapy in patients with malignancy. □ Rhabdomyolysis and Elevated CPK: Discontinue therapy if there is significant elevation in CPK levels or a diagnosis or suspicion of myopathy. □ Triglyceride Elevations: Periodically evaluate serum triglycerides. □ Liver Enzyme Elevations: Evaluate liver enzymes at baseline and during therapy in patients with known or suspected liver disease. □ Immunizations. Avoid use with live vaccines. Before initiating therapy, complete all age-appropriate immunizations per current guidelines including prophylactic herpes zoster vaccination. □ Potential Risks Related to JAK Inhibition: It is unknown whether TYK2 inhibition may be associated with the observed or potential adverse reactions of JAK inhibition. (Increased rates of mortality, malignancy other than nonmelanoma skin cancer, major adverse cardiovascular events, and thrombosis have been observed with a JAKi compared with TNFi therapy in patients ≥ 50 years with rheumatoid arthritis who had ≥ 1 cardiovascular risk factor.)
Most Common AEs (≥ 1%)	Upper respiratory infections, elevated blood CPK, herpes simplex, mouth ulcers, folliculitis, acne
Drug Interactions	**For Psoriasis**: Not recommended with other potent immunosuppressants. Avoid with live vaccines. **For PsA**: Avoid with live vaccines.
Pregnancy	Insufficient human data. No effects on embryofetal development were observed in animal studies.
Lactation	Insufficient human data. Likely to be present in human milk based on presence of drug in rat milk.

Hepatic Impairment	Not recommended in patients with severe hepatic impairment (Child-Pugh C)

CLINICAL EVIDENCE SUMMARY

Phase 3 Trials	Data on the POETYK PsA-1 and POETYK PsA-2 trials are available only from the prescribing information.[1]
Design	Two MC DB PC RCTs At Week 16, placebo patients were switched to deucravacitinib.
Primary Endpoint	ACR20 response at Week 16
Population	Adults with active PsA (≥ 3 SJC, ≥ 3 TJC, CRP ≥ 3 mg/L), and active or history of plaque psoriasis. Diagnosis of PsA for ≥ 3 months. • PsA-1: Presence of ≥ 1 bone erosion on X-ray of hands and/or feet. • PsA-2: Apremilast as active safety reference treatment. *Baseline characteristics*: Mean age 51 years; mean weight 84 kg; 49% male; 78% White, 11% Asian; 76% had polyarthritis; 14% oligoarthritis; 16% had peripheral plus psoriatic spondyloarthritis; 49% enthesitis; 31% dactylitis; **majority were BIO-naïve and had an inadequate response, intolerance, or loss of response to NSAIDs, csIMMs, or apremilast**. • PsA-2: Included **TNFi-experienced patients**
Intervention	Deucravacitinib 6 mg QD Placebo
Co-therapy	None required by protocol. 56% concomitant methotrexate; 10% other concomitant csIMM; 34% not on csIMM.
Efficacy Results (Nonresponder Imputation Analyses)	**POETYK PsA-1 (Patients with Hand/Foot Bone Erosion[s]): Peripheral Arthritis Results at Week 16**

Endpoint	Deucravacitinib	Placebo	RR (95% CI)	ARD (95% CI)	GRADE
ACR20, n/N (%)	181/336 (54)	114/334 (34)	1.58 (1.32, 1.89)	20 (13, 27)	NA
ACR70, n/N (%)*	40/336 (12)	17/334 (5)	2.34 (1.36, 4.03)	6 (2, 10)	NA

NA, not assessable

POETYK PsA-2 (Apremilast Results Not Shown): Peripheral Arthritis Results at Week 16

Endpoint	Deucravacitinib	Placebo	RR (95% CI)	ARD (95% CI)	GRADE
ACR20, n/N (%)	168/312 (54)	122/312 (39)	1.38 (1.16, 1.63)	15 (7, 23)	NA
ACR70, n/N (%)*	34/312 (11)	16/312 (5)	2.13 (1.20, 3.76)	5 (1, 9)	NA

Limitations	Unpublished, non–peer-reviewed data. Demographically mismatched with typical VHA patient population, especially in the percentage of males (49% in trials vs >90% in VHA[2]), the low representation of Black patients (0.4% vs 15–21% non-White VHA patients), younger age (51 years vs 64 years in VHA inflammatory arthritis patients), and fewer comorbidities than VHA patients (significant CV disease, uncontrolled diabetes, and major psychiatric illnesses were excluded from trials). Real-world PsA patients have been shown to have higher CRP levels (1.4 vs 1.1 mg/dL) despite lower joint counts, suggesting they have greater systemic inflammation.[3] These factors introduce some uncertainty in whether the trial results can be extrapolated to the VHA population; however, JAK/TYK2 inhibitors have been shown to have sex-neutral efficacy[4] and consistent efficacy regardless of concomitant csIMM use and prior treatment.

Phase 2 Trial	**Efficacy and safety of selective TYK2 inhibitor, deucravacitinib, in a phase II trial in psoriatic arthritis**[5,6,7,8,9,10]
Design	Initial 16-week PC period (Part A) of a phase 2 MN DB DC PC RCT conducted in CA, DE, HU, PL, ES, RU, and US. Randomization was stratified by previous TNFi use (experienced/naïve) and body weight (≥ 90/< 90 kg). Multiplicity-controlled, hierarchical testing.
Primary Endpoint	ACR20 response at Week 16

Population

Diagnosis of PsA for ≥ 6 months; active joint disease (≥ 3 tender and ≥ 3 swollen joints); hs-CRP ≥ 3 mg/L (ULN, 5 mg/L); ≥ 1 plaque PsO lesion (≥ 2 cm); **failed to respond or intolerant to ≥ 1 prior therapy** (could include NSAIDs, corticosteroids, csIMM, and/or TNFi).

Baseline Characteristics (N = 203): Mean age 49.8 years; 48.8% male; 98.0% White; mean body weight 88.6 kg (51.2% < 90 kg / 48.8% ≥ 90 kg); **65.0% prior/concomitant csIMM**; 15.3% one prior/concomitant TNFi; 0.5% two prior/concomitant TNFis; 12.3% prior/concomitant oral steroid (mean 4.0 mg/day); median disease duration 4.5 years (range, 0.1–42.8 years); 81.3% PsO with ≥ 3% BSA; 47.3% enthesitis with Leeds Index ≥ 1; 38.9% dactylitis. Uveitis at baseline was not mentioned.

Interventions

Deucravacitinib 12 mg PO QD

Deucravacitinib 6 mg PO QD

Placebo PO QD

Duration: 16 weeks

Co-therapy

csIMMs (e.g., methotrexate, leflunomide, sulfasalazine, or hydroxychloroquine) were permitted if used for ≥ 3 months at a stable dose for ≥ 28 days.

Phase 2 Efficacy Results

Selected Efficacy Measures at Week 16

Endpoint	Deucravacitinib 6 mg QD	Deucravacitinib 12 mg QD	Placebo	RR, 6 mg (95% CI)	ARD, 6 mg (95% CI)	GRADE
ACR20, n/N (%)	37/70 (52.9)	42/67 (62.7)	21/66 (31.8)	1.7 (1.10, 2.52)	21 (5, 37)	Mod (D, P, PB)‡
ACR70, n/N (%)*	10/70 (14.3)	13/67 (19.4)	1/66 (1.5)	9.4 (1.25, 71.6)	13 (4, 22)	Mod (D, P, PB)‡
MDA, n/N (%)	16/70 (22.9)	10/67 (23.9)	5/66 (7.6)	3.0 (1.17, 7.77)	15 (4, 27)	Mod (D, P, PB)‡
Enthesitis resolution (LEI = 0), n/N (%)*	20/39 (51.3)	13/26 (50.0)	7/31 (22.6)	2.3 (1.11, 4.67)	29 (7, 50)	Low (D, P, PB)‡
Dactylitis resolution (LDI = 0), n/N (%)	23/30 (76.7)	19/24 (79.2)	15/25 (60.0)	1.3 (0.88, 1.86)	17 (8, 41)	Low (D, P, PB)‡
HAQ-DI response*†	27/70 (38.6)	27/67 (40.3)	10/66 (15.2)	2.5 (1.34, 4.84)	23 (9, 38)	Mod (P, PB)‡

The FDA-approved dosage is 6 mg QD.

* Not controlled for multiplicity

† ≥ 0.35 improvement from baseline (minimum clinically important difference in PsA) on the Health Assessment Questionnaire–Disability Index

‡ Not downgraded for serious risk of bias, although the rates of early withdrawals and reasons such as adverse events or lost to follow-up were not accessible

D Downgraded for serious indirectness (ACR20 does not include functional ability, which was considered the key clinical outcome)

P Downgraded for serious imprecision (optimal information size not met)

PB Downgraded for potential serious publication bias

Abbreviations: HAQ-DI, Health Assessment Questionnaire Disability Index; LDI, Leeds Dactylitis Index; LEI, Leeds Enthesitis Index; MDA, minimal disease activity (defined as achieving ≥ 5 of the following: tender joint count ≤ 1; swollen joint count ≤ 1; PASI ≤ 1 or body surface area ≤ 3%; tender entheseal points ≤ 1; patient global assessment of pain ≤ 15; patient global assessment of disease activity ≤ 20; and HAQ-DI ≤ 0.5)

- The number needed to treat (NNT) was 5 (95% CI 3, 21) to achieve one additional ACR20 responder and 16 (4, 27) to achieve one additional MDA responder compared with placebo.
- PASI-75 response rate was 42.4% vs 20.4% for deucravacitinib 6 mg QD vs placebo (RR [95% CI] 2.0 [1.18, 3.46]; ARD [95% CI] 22 [7, 37]).
- Quality of life, as measured with the Short Form-36 Health Survey Physical Component Summary (SF-36 PCS) score, significantly improved on deucravacitinib vs placebo, with an adjusted mean change from baseline of 5.6 vs 2.3, respectively. Changes from baseline of ≥ 2.5 and ≥ 5 points were used as the minimal clinically important differences (MCIDs) for SF-36 summary and domain scores.[10] However, based on a small anchor-based analysis in Chinese patients with PsA who were treated with TNFis (which may not be generalizable to other populations), an MCID of about 4 points has been validated to correspond with "slightly better" current health status.[11]

Analyses by Planned Subgroups

- Deucravacitinib was superior to placebo regardless of prior TNFi exposure, body weight (< 90 kg/≥ 90 kg), and sex (male/female).

Safety Results

- Most adverse events in the deucravacitinib-treated patients were mild to moderate in intensity, and there were no serious adverse events (including serious infections), no thrombotic events, and no herpes zoster, tuberculosis, opportunistic infections, or malignancy.
- Adverse events that occurred numerically more often on deucravacitinib 6 mg (the approved dose) than placebo included discontinuations due to adverse event, any adverse event, and treatment-related adverse events.
- Specific adverse events that occurred on deucravacitinib 6 mg and not on placebo included upper respiratory tract infection, sinusitis, rash, and diarrhea.

Summary of Safety in Phase 2 RCT

Adverse Event	Deucravacitinib 6 mg QD (N = 70)	Deucravacitinib 12 mg QD (N = 67)	Placebo (N = 66)
Deaths	0	0	0
Serious Adverse Event	0	0	1 (1.5)
Discontinuation Due to Adverse Event	3 (4.3)	4 (6.0)	1 (1.5)
Any Adverse Event	46 (65.7)	44 (65.7)	28 (42.4)
Treatment-related Adverse Events	22 (31.4)	17 (25.4)	6 (9.1)
Adverse Events in ≥ 5% of Patients			
Nasopharyngitis	4 (5.7)	12 (17.9)	5 (7.6)
Upper respiratory tract infection	4 (5.7)	1 (1.5)	0
Sinusitis	0	5 (7.5)	0
Bronchitis	4 (5.7)	0	1 (1.5)
Headache	5 (7.1)	1 (1.5)	3 (4.5)
Rash	3 (4.3)	4 (6.0)	0
Diarrhea	4 (5.7)	0	0

- No substantial differences between deucravacitinib and placebo were observed in hematology, serum lipids, or chemistry laboratory values.

Authors' Conclusions

In patients with PsA, TYK2i therapy with deucravacitinib produced greater improvements than placebo in ACR20, multiplicity-controlled secondary endpoints and other exploratory outcome measures. Larger and longer trials are needed to confirm its safety and efficacy.

Trial Limitations

Single, sponsor-funded phase 2 trial (results have not been confirmed by a second trial; potential publication bias); details of study withdrawals were not accessible; modest sample size and short study duration are insufficient to fully assess risks of rare or latent adverse events and durability of effects; exploratory endpoints were not adjusted for multiplicity (potential false-positive results)

Duration of an adequate therapeutic trial

At least 16 weeks. Based on ACR20 response rates over time, a plateau was reached around Weeks 12–16 in the two POETYK trials.[1] In MDA post hoc analyses of the phase 2 trial, individual components of MDA continued to increase through Week 16.[7]

Long-term Extension

No study publication found for PsA.

Deucravacitinib has been shown to be safe and effective in *plaque psoriasis* for up to 3 years.[12]

Articles/Trials of Potential Interest

Phase 3 Trial Results of Deucravacitinib in Psoriatic Arthritis [online news article].[13]

Efficacy and Safety of Deucravacitinib up to Week 52: A Multicenter, Randomized, Double-Blind, Placebo-Controlled, Phase 3 Study in Patients With Active Psoriatic Arthritis Who Are Naive to Biologic Disease-Modifying Antirheumatic Drugs [abstract].[14]

Deucravacitinib in Patients with Plaque Psoriasis Who Screened Positive for Psoriatic Arthritis: Improvements in Joint Pain and the Impact of Musculoskeletal Symptoms[15]

META-ANALYTIC INDIRECT COMPARISONS

JAK Inhibitors

A meta-analysis of 8 RCTs evaluated 10 JAK inhibitors vs placebo in PsA.[16]

ACR Responses. Deucravacitinib was effective in ACR20, ACR70 and, but not ACR50 response. Upadacitinib 30 mg was likely better than tofacitinib (5 and 10 mg) and upadacitinib 15 mg, and upadacitinib 15 mg was likely better than tofacitinib 10 mg.

Dactylitis. Deucravacitinib was ineffective for resolution of dactylitis. Upadacitinib 30 mg was better than deucravacitinib 6 mg for this endpoint (RR 1.52; 95% CI 1.0002, 2.3228).

Enthesitis. Deucravacitinib 6 mg, but not 12 mg, was more effective than placebo. There were no apparent differences between deucravacitinib and the other drugs (upadacitinib 15 and 30 mg, tofacitinib 5 and 10 mg, and brepocitinib 10, 30, and 60 mg).

Disability. Deucravacitinib was significantly better than placebo in the change from baseline in Health Assessment Questionnaire Disability Index (HAQ-DI).

SAEs. Only upadacitinib 30 mg increased the risk of SAEs over placebo.

CROSS-TRIAL INDIRECT COMPARISONS

ACR20 ARDs for Drugs vs. Placebo at or near Week 16 in predominantly biologic-naïve PsA populations

Agent (Dose)	Trial	Population	Time (Wk)	ACR20 Drug (%)	ACR20 PBO (%)	ARD (95% CI), pp
Ixekizumab 80 mg Q2W	SPIRIT-P1	BIO-naïve	24*	64/103 (62%)	32/106 (30%)	**+32** (+19, +44)
Ixekizumab 80 mg Q4W	SPIRIT-P1	BIO-naïve	24*	62/107 (58%)	32/106 (30%)	**+28** (+15, +40)
Bimekizumab 160 mg Q4W	BE OPTIMAL	bDMARD-naïve	16	268/431 (62%)	67/281 (24%)	**+38** (+32, +45)
Secukinumab 300 mg (LD)	FUTURE 5	Mixed (~70% TNFi-naïve)	16	139/222 (63%)	61/222 (27%)	**+35** (+27, +43)
Secukinumab 150 mg (LD)	FUTURE 5	Mixed (~70% TNFi-naïve)	16	123/222 (56%)	61/222 (27%)	**+28** (+20, +36)
Apremilast 30 mg BID	PALACE 3	csIMM/BIO-IR (~78% BIO-naïve)†	16	69/169 (41%)	30/169 (18%)	**+23** (+14, +33)
Apremilast 30 mg BID	PALACE 1	csIMM/BIO-IR (~78% BIO-naïve)†	16	66/168 (40%)	32/168 (19%)	**+21** (+11, +30)
Deucravacitinib 6 mg QD	POETYK PsA-1	Mostly BIO-naïve	16	181/336 (54%)	114/334 (34%)	**+20** (+13, +27)
Apremilast 30 mg BID	ACTIVE	BIO-naïve	16	42/110 (38%)	22/109 (20%)	**+18** (+7, +30)
Deucravacitinib 6 mg QD	POETYK PsA-2	Mostly BIO-naïve (~20% anti-TNF-exp)	16	168/312 (54%)	122/312 (39%)	**+15** (+7, +23)
Apremilast 30 mg BID	PALACE 4	DMARD-naïve	16	54/176 (31%)	28/176 (16%)	**+15** (+6, +23)
Adalimumab 40 mg Q2W	ADEPT (PsA-I)	BIO-naïve	12*	88/151 (58%)	23/162 (14%)	**+44** (+35, +53)
Tofacitinib 5 mg BID	OPAL Broaden	csIMM-IR	12*	54/107 (50%)	35/105 (33%)	**+17** (+5, +30)
Upadacitinib 15 mg QD	SELECT-PsA 1	nbDMARD-IR	12*	303/429 (71%)	153/423 (36%)	**+35** (+28, +41)

† ~78% of patients were biologic-naïve across the pooled PALACE 1–3 population[21]

Blue shading indicates drugs with higher ARDs with non-overlapping CIs vs deucravacitinib. Drugs assessed at the 12-week timepoint (adalimumab and upadacitinib) already had higher ARDs before the 16-week timepoint at which deucravacitinib was assessed.

CROSS-TRIAL INDIRECT COMPARISONS

Other Treatment Contrasts

Oral PsA Agents

Factor	Deucravacitinib	Apremilast	Tofacitinib	Upadacitinib	References
PASI 75 (except as noted; ARDs vs placebo were based on patient subsets with ≥3% affected BSA)	Data not reported for PsA trials. **PsO-1:** 58%; **+46 pp** (39, 53) at Wk 16. Also significantly better than **apremilast** (35%): +23 pp [14, 32]) at Wk 16. ARD vs apremilast +31 pp at Wk 24. **PsO-2:** 53%; **+44 pp** (38, 49); apremilast 40%; ARD vs **apremilast**: +13 pp (6, 21) at Wk 16, and 58% vs 38%, respectively; +20 pp at Wk 24.	PASI 50 (primary): ~41% at Wk 16 (PALACE 3, csIMM-/bDMARD-exposed; BSA ≥3%); **ARD +17 pp (~4.6, 29.5)** **PASI 75 in noncomparative long-term PsA trials**: 43.6% at Wk 260 (PALACE 1–3, pooled data) 47.6% at Wk 260 (PALACE 4) **PASI 75 in PsO trials**: 33.1% at Wk 16 (ESTEEM 1); **ARD +27.8** pp (23.1, 32.5) 28.8% at Wk 16 (ESTEEM 2); **+23.0 pp** (16.4, 29.6) 39.8% at Wk 16 (LIBERATE); ARD +28 pp (~16, 40) **Meta-analytic Data** (18 PsO or PsA PC RCTs; N = 6,036)[17]: RR 4.08 (3.12, 5.33)	OPAL Broaden: **~43%** at Wk 12 (5 mg); **ARD ~+28 (~13, 43)** OPAL Beyond: 5 mg – ARD +**8 pp** at Wk 12**; NSD** vs placebo in TNFi-IR)	**15 mg: 62.6%** at Wk 16 (SELECT-PsA 1, csIMM-IR); **ARD +41.3 pp** (32.8, 49.8)) **30 mg: 62.4%** at Wk 16 (SELECT-PsA 1, csIMM-IR; ARD **+41.1 pp** (32.5, 49.6) **15 mg**: 52.3% at Wk 24 (SELECT-PsA 2, bDMARD-IR); **ARD +33 pp** (20.6, 46.2)	[18,19,20]
Laboratory monitoring	LFTs, CPK, TG	Not required	CBC, CMP, lipid panel at baseline; repeat at 4–8 weeks, then every 12 weeks; consider CPK monitoring (not labeled but potential JAKi-class effect)	CBC, hepatic function, lipid panel at baseline; repeat at ~12 weeks, then per clinical judgment; also monitor CPK	[1,21]
Boxed Warning	None	None	Serious infections, MMMT (based on ORAL Surveillance in RA ≥50 y with ≥1 CV risk factor)	Serious infections, MMMT (JAKi-class boxed warning)	[1,21,22,23]
TB screening before initiation	Required	Not required	Required	Required	[1,21,22,23]
Chronic infections (HIV, HBV, HCV)	Limited data	Safely used	Screen for TB, hepatitis, and HIV before initiation; **not recommended** with active HBV/HCV; HBV reactivation reported postmarketing	Screen for viral hepatitis; **not recommended** with active HBV or HCV; HBV reactivation risk applies	[1,21,22,23,24]
Recurrent infections	Limited long-term data	Guideline-preferred	Increased risk of serious infections (SI rate ~2.0/100 PY); herpes zoster risk elevated (RR ~1.4 vs placebo); GI perforation signal unique to tofacitinib	Increased risk of serious infections (SI rate ~3.0/100 PY at 15 mg); herpes zoster risk elevated (OR 1.55, 95% CI: 1.06–2.27 vs placebo)	40,[25],[26]

Factor	Deucravacitinib	Apremilast	Tofacitinib	Upadacitinib	References
TNFi-experienced	Effective (PsA-2)	Limited data	**Effective** — OPAL Beyond: ACR20 50% (5 mg) vs 24% placebo in TNFi-IR patients	**Effective** — SELECT-PsA 2: ACR20 56.9% (15 mg) vs 24.1% placebo in bDMARD-IR patients; FDA-approved indication is specifically after TNFi failure	[1,27,28]
Long-term safety data	Indirect evidence: Up to 3 years (psoriasis OLE)	Up to 5 years (PALACE 1–3 pooled; PALACE 4)	**~8+ years** (RA); **48 months** (PsA, OPAL Balance); ORAL Surveillance safety signal (RA) is the primary concern	**~2 years** (PsA, SELECT-PsA 1 extension); **5+ years** (RA); no PsA-specific long-term safety trial comparable to ORAL Surveillance	[12,29,30,31,32]
Depression risk	No specific warning	Warning for depression/suicidality	Preclinical data suggest potential **anti-depressant** properties via JAK-STAT/microglial pathway; no clinical signal for increased depression; may be beneficial in comorbid depression/anxiety	No specific signal for increased depression risk in clinical trials; insufficient data to claim antidepressant benefit	[1,33,34]
Weight loss concern	Not reported	Monitor for >5% weight loss	**Weight-neutral** — No significant weight change observed in PsA trials; JAKi class is generally weight-neutral	**Weight-neutral** — No significant weight change observed; JAKi class is generally weight-neutral	[35]
GI tolerability	Generally well tolerated	Diarrhea, nausea common (especially early)	Common GI AEs: diarrhea, nausea, abdominal distension; unique signal for **GI perforation**; caution with diverticular disease	Common GI AEs: nausea, abdominal pain; elevated GI event reporting in FAERS (ROR 2.04 for respiratory, also GI signals); **no specific GI perforation signal**	[1,36,37,38]
Dosing	Once daily	Twice daily (with 5-day titration)	5 mg **twice daily** (or XR 11 mg once daily)	15 mg **once daily**	[1,21,22,23]
Dosing in severe renal impairment	No dosage adjustment needed	Dose reduction to 30 mg QD	**Dose reduction required**: 5 mg once daily (halved) for moderate-to-severe renal impairment	**No dose adjustment** needed for mild, moderate, or severe renal impairment (eGFR 15–30); not recommended in ESRD (eGFR 15)	[1,21,22,23]
Use in hepatic impairment (HI)	Not recommended in severe HI (Child-Pugh C)	No dosage adjustment	Reduce to 5 mg QD in moderate HI (Child-Pugh B) Not recommended in severe HI (Child-Pugh C)	Not recommended in severe HI (Child-Pugh C)	[1,21,22,23]

OTHER CONSIDERATIONS	
FDA Review	Not available
ICER Review	None
NICE Review	None

GUIDELINE-BASED TREATMENT SEQUENCING

Place in Therapy	Peripheral Arthritis	Enthesitis	Dactylitis	Axial Disease	Uveitis	References
1st line	NSAIDs ± csIMMs (MTX, SSZ, LEF)	NSAIDs (mild) TNFi mAb (moderate-severe)	NSAIDs ± local GC injection (mild) TNFi or IL-17i (moderate-severe)	NSAIDs (≥ 2 agents, max dose, ≥ 2–4 wk each)	Topical corticosteroids ([AAU])	[39,40,41,42]
2nd line (csIMM-IR or NSAID-IR)	TNFi (preferred) or IL-17i, IL-23i, IL-12/23i, JAKi	TNFi mAb (preferred) IL-17i or IL-12/23i if TNFi contraindicated or severe psoriasis	TNFi, IL-17i, IL-23i, or IL-12/23i (all strongly recommended)	TNFi mAb (preferred) IL-17i if TNFi contraindicated or severe psoriasis	TNFi mAb — adalimumab or infliximab preferred (recurrent AAU)	[39,40,41,42,43,44,45]
3rd line (BIO-IR)	Switch TNFi, or IL-17i, IL-23i, JAKi	IL-17i, IL-23i, or JAKi	IL-17i, IL-23i, or JAKi	IL-17i (if TNFi-IR) JAKi (upadacitinib, tofacitinib)	Certolizumab or golimumab (alternative TNFi mAbs) Adalimumab if not yet tried	[39,40,42,46]
Oral alternatives	Apremilast (mild) Tofacitinib or upadacitinib (moderate–severe, TNFi-IR) Deucravacitinib (emerging)	Apremilast (mild, oral preference) JAKi (moderate-severe)	Apremilast (limited data)	csIMMs NOT effective JAKi (tofacitinib, upadacitinib) effective Apremilast NOT effective	JAKi — limited data; not established for uveitis	[39,40,41,47]

Domain-specific Considerations

- **Peripheral arthritis** — ACR/NPF 2018 conditionally recommends a TNFi over an oral small molecule (OSM; methotrexate, sulfasalazine, cyclosporine, leflunomide, apremilast – excludes tofacitinib), IL-17i, and IL-12/23i in OSM- and other treatment-naïve patients with active PsA, with choice depending on such factors as disease severity, comorbidity risks, severity of concomitant psoriasis, oral preference, concerns about using a biologic as the first treatment, and frequency of administration.[40] GRAPPA 2021 strongly recommends csIMMs (methotrexate, sulfasalazine, leflunomide) and PDE4 inhibitors for csIMM-naïve patients (excluding cyclosporine), with early escalation to biologics or JAKi if targets are not met. TNFi, IL-17i, IL-12/23i, IL-23i, and JAKi are all strongly recommended for csIMM-IRs, with no clear hierarchy among them.[39,42]
- **Enthesitis** — The ACR/NPF 2018 guideline conditionally recommends TNFi over an OSM and other biologics as first-line for moderate-to-severe enthesitis. However, network meta-analyses suggest IL-17i may have the highest probability of enthesitis resolution (RR vs TNFi: 1.22, 95% CI 1.02–1.47), and IL-23i also demonstrate significant resolution (RR vs placebo: 1.46, 95% CI 1.29–1.64). The csIMMs (other than possibly methotrexate) lack evidence for enthesitis.[40,43,48] GRAPPA did not recommend a preferential treatment sequence because none of the drug classes (TNFi, IL-17i, IL-12/23i, IL-23i, JAKi and PDE4i) have been shown to be clearly and consistently superior to the others.[39]
- **Dactylitis** — The ACR/NPF 2018 guideline made no specific recommendations. GRAPPA 2021 strongly recommended all biologic classes (TNFi, IL-17i, IL-12/23i, IL-23i). Meta-analyses showed comparable efficacy between TNFi and IL-17/IL-23 inhibitors for dactylitis resolution.[44,49] Local glucocorticoid injection may be considered for isolated dactylitis.
- **Axial disease** — csIMMs are **not effective** for axial PsA. TNFi monoclonal antibodies (mAbs) are preferred first-line, with IL-17i as an alternative (secukinumab has the only dedicated axial PsA RCT data). IL-12/23i (ustekinumab) failed in ankylosing spondylitis (AS) trials and is not recommended. IL-23p19 inhibitors (guselkumab, risankizumab) failed in primary AS but post hoc PsA data are mixed — their role in axial PsA remains uncertain.[40,47]
- **Uveitis** — TNFi mAbs (adalimumab, infliximab preferred; certolizumab, golimumab also effective) are the treatment of choice for recurrent uveitis. **Etanercept should be avoided** (does not prevent uveitis). Secukinumab was not efficacious for posterior uveitis or panuveitis. IL-12/23i therapy has insufficient evidence to support use. IL-23 inhibitors lack uveitis data. Adalimumab is the only biologic FDA-approved for noninfectious uveitis and was recently shown to be superior to conventional immunosuppressants (antimetabolites, calcineurin inhibitors, or both) in achieving successful corticosteroid sparing at 6 months and successful corticosteroid discontinuation at 12 months in the ADVISE trial.[41,45,46] The ACR/NPF 2018 stated that a biologic in combination with methotrexate could be used instead of biologic monotherapy in patients who have concomitant uveitis since methotrexate may be effective for uveitis.[40]

Other Considerations

- NSAIDs provide **only symptomatic relief** – they do not modify disease progression, suppress immune-mediated inflammation, or prevent structural damage.
- When multiple domains are active, the therapy should ideally address all active domains simultaneously, and the domain with the highest activity level should generally drive the treatment choice.[39]
- **Inflammatory bowel disease (IBD) comorbidity** shifts preference toward TNFi mAbs, IL-12/23i, or IL-23i. IL-17i treatment should be avoided (risk of new-onset or exacerbation of IBD).[39]
- **Cardiovascular risk** — JAKi carry a boxed warning that includes mortality, malignancy, MACE, and thromboembolism based on the ORAL Surveillance trial in rheumatoid arthritis patients ≥ 50 years with ≥ 1 cardiovascular risk factor. The FDA restricts the use of JAKi to TNFi-IR patients.

Summary of Evidence	1. Deucravacitinib is FDA-approved for "active psoriatic arthritis in adults" without a line-of-therapy restriction. However, the trial population had mostly experienced inadequate response to NSAIDs, csIMMs, or apremilast, and 56% were on concomitant methotrexate. There is no evidence that deucravacitinib is superior to or should replace csIMMs as initial therapy. 2. There were no active-comparator trials of deucravacitinib to inform its place in therapy in the treatment of active psoriatic arthritis. Indirect comparisons suggest that, in predominantly biologic-naïve patients, deucravacitinib may be comparable in Week-16 ACR20/peripheral arthritis response to apremilast and potentially inferior (based on non-overlapping 95% CIs) to bimekizumab, upadacitinib, and secukinumab. ACR20 responses were numerically higher with deucravacitinib than apremilast (54% vs. 32%–41%, respectively); however, placebo response rates were also higher with deucravacitinib (34%–39% vs. 18%–20%, respectively), resulting in comparable absolute risk differences of 15–20 pp and 13–23 pp, respectively, with overlapping 95% CIs. Deucravacitinib does not carry the JAKi-class boxed warnings and in short-term (16-week) studies was not associated with cytopenias. Therefore, it has the potential to have safety advantages over JAK inhibitors (tofacitinib, upadacitinib). However, long-term safety data in PsA are not yet available. Like JAK inhibitors and, to a lesser extent, TNF inhibitors, but unlike IL-17 inhibitors, IL-23 inhibitors, and apremilast, deucravacitinib is associated with elevated CPK. Like methotrexate, deucravacitinib is associated with mouth ulcers; however, the incidence may be lower (1.9% vs. 5.7%–8%, indirect comparison).[1, 50] Unlike IL-17 and IL-23 inhibitors and apremilast, deucravacitinib is associated with acne (a JAKi-class adverse effect) and folliculitis. 3. Evidence of uncertain quality from two non–peer-reviewed, phase 3, placebo-controlled RCTs (POETYK PsA-1 and POETYK PsA-2) showed that, in patients who had an inadequate response or intolerance to ≥1 prior therapy (mainly csDMARDs/NSAIDs, with PsA-2 also including anti-TNF-experienced patients), deucravacitinib 6 mg daily significantly improved peripheral joint symptoms. Phase 2 data suggested significant improvements in enthesitis and dactylitis (although resolution of these symptoms was not reported), and moderate-quality evidence from the phase 2 trial suggested significant improvement in patient-reported physical function (HAQ-DI) and quality of life (SF-36 PCS). Deucravacitinib had a medium effect for ACR20 response, a small effect for ACR70 response, and a negligible effect for MDA response; however, 95% confidence intervals for NNTs were wide. Skin manifestations (plaque psoriasis) as measured by PASI 75 also significantly improved. 4. The FDA label does not report enthesitis or dactylitis resolution rates from the phase 3 trials. A meta-analysis showed that deucravacitinib was ineffective for resolution of dactylitis and did not support the positive phase-2 dactylitis findings.[16] In the phase 2 trial, deucravacitinib showed inconsistent resolution of enthesitis, with the 6-mg but not the 12-mg dose showing significantly better effects than placebo. 5. Doubling the dose of deucravacitinib resulted in minimal, if any, additional improvement in the measured endpoints. 6. The FDA-approved dose is 6 mg once daily with no recommended dose modifications for adverse events. Except for treatment discontinuation due to adverse events, there was no description of whether adverse events were manageable with dosage modifications. As mentioned above, deucravacitinib does not have the Boxed Warnings of JAK inhibitors. 7. Full peer-reviewed publications of these trials are desirable to provide detailed domain-specific data (enthesitis resolution, dactylitis resolution, axial outcomes) not included in the FDA label. Further studies are needed to determine the effects of deucravacitinib on resolution of enthesitis and dactylitis. Long-term studies are needed to evaluate the longer term efficacy and safety of deucravacitinib in active PsA. There are no data on the efficacy of deucravacitinib for axial involvement.

Potential Treatment Sequencing	In general, deucravacitinib may have a role in patients with active, nonsevere peripheral PsA and significant psoriasis who prefer oral therapy over injectable biologics, who have had an inadequate response or intolerance to a csIMM (or for whom a csIMM is inadvisable), or who are at higher risk of JAKi–class adverse effects (e.g., serious infection, MACE, thromboembolism)—though the comparative safety advantage of TYK2 inhibition over JAK inhibition has not been established in head-to-head trials. 1. **csIMM** (for predominantly peripheral arthritis, no axial disease, no severe skin, no IBD/uveitis) or **TNFi** 2. **IL-17i** (ixekizumab preferred; may prefer over TNFi if concomitant psoriasis is severe) or **JAKi** 3. **IL-12/23i** (not for axial disease) or **IL-23i** (not for axial disease; may prefer over IL-12/23i if concomitant psoriasis is severe) 4. **Abatacept** (not for axial disease) **Other**: **Apremilast** (after a csIMM + TNFi is medically inadvisable); **Deucravacitinib** (after csIMM or TNFi, IL-17i or JAKi, IL-12/23i or IL-23i, and apremilast; not for axial disease)

Original: June 2026.

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