Risankizumab-rzaa (SKYRIZI) in Psoriatic Arthritis National Drug Monograph Addendum August 2022

VA Pharmacy Benefits Management Services, Medical Advisory Panel, and VISN Pharmacist Executives

The purpose of VA PBM Services drug monographs is to provide a focused drug review for making formulary decisions. Updates will be made if new clinical data warrant additional formulary discussion. The Product Information or other resources should be consulted for detailed and most current drug information.

FDA Approval Information

Description / Mechanism of Action

- Risankizumab-rzaa is the second interleukin (IL)-23 inhibitor approved for the treatment of active psoriatic arthritis (PsA).
- It is the first IL-23 inhibitor approved for the treatment of moderately to severely active Crohn's disease in adults.

Indication Under Review in This Document

- Treatment of active PsA in adults.[1]

Dosage Regimen and Dosage Form(s) Under Review

- 150 mg subcutaneously at Weeks 0 and 4, then every 12 weeks, alone or in combination with nonbiologic immunomodulators.
- 150 mg/mL single-dose pen or prefilled single (each in a pack of 1); 75 mg/0.83 mL single-dose prefilled syringe (pack of 2).

Clinical Evidence Summary

Efficacy Considerations

- There are no active-controlled RCTs to inform the place in therapy of risankizumab-rzaa in the treatment of active PsA.
- The FDA approval of risankizumab-rzaa for the treatment of active PsA was mainly based on two phase 3, placebo-controlled randomized clinical trials (RCTs). The KEEPsAKE 1 trial involved biologic-naïve patients who had a previous inadequate response or intolerance to conventional synthetic immunomodulators (csIMMs),[2,3] and KEEPsAKE 2 involved a mixed population of patients who had previous inadequate

response or intolerance to ≥ 1 conventional synthetic immunomodulators (csIMMs) and / or ≤ 2 biologics.[4]

- Data for up to 24 weeks of therapy for each trial have been published.

Randomized Clinical Trials

- The design of the KEEPsAKE 1 and 2 trials are summarized in Table 1.

Table 1 Methods of Phase 3 RCTs

Topic	KEEPsAKE 1	KEEPsAKE 2
Study Design	24-week MN DB PC RCT Randomization was stratified by baseline psoriasis involvement (≥ 3% / < 3% BSA), presence of dactylitis, presence of enthesitis, and current csIMM use. 11 ranked secondary outcomes were controlled for multiplicity.	24-week MN DB PC RCT Randomization was stratified by baseline psoriasis involvement (≥ 3% / < 3% BSA), current csIMM use, and number of prior biologics 6 ranked secondary outcomes were controlled for multiplicity
Major Entry Criteria	Adult (≥ 18 years) Active PsA (symptom onset ≥ 6 months, met classification criteria for PsA, TJC ≥ 5 of 68, SJC ≥ 5 of 66, ≥ 1 erosion in hands and/or feet or high-sensitivity C reactive protein (hsCRP) ≥ 3.0 mg/L and active PsO (≥ 1 plaque ≥ 2 cm diameter or nail psoriasis). Inadequate response, intolerance or contraindication to ≥ 1 csIMM.	Same as KEEPsAKE 1 except patients had to have csIMM and/or biologic inadequate response after ≥ 12 weeks of therapy, intolerance or contraindication.
Interventions	RIS 150 mg SC vs PBO at Weeks 0, 4 and 16 Concomitant therapies (≤ 2 csIMMs at protocolled doses) could be added or modified in patients who had not achieved ≥ 20% improvement in SJC and/or TJC at both Weeks 12 and 16.	RIS 150 mg SC vs PBO at Weeks 0, 4 and 16 Concomitant therapies could include continuation of ≤ 2 csIMMs (stable doses), NSAIDs, oral CS (≤ 10 mg/d prednisone equivalent), and other analgesics.
Maintenance Phase or Long-term Extension	204-week open-label period	Open-label RIS Q12W through Week 208
Primary Efficacy Measure	ACR20 response at Week 24	ACR20 response at Week 24
Baseline Patient Characteristics	Median age 52 years (range, 20–85), White 94%, male 50.4% Mean PGA-disease activity 58 (100-mm VAS) Enthesitis 61%, Dactylitis 31%, Nail psoriasis 67% Prior csIMM: One agent 67%; two 25%, three or more 7%, mainly MTX 89.9%, sulfasalazine 21.5% and leflunomide 12.8%. (Inadequate response 85.2%, intolerance 14.4%, contraindication 0.4%.) Concomitant medications: Mainly MTX 65% and NSAIDs 63%	Median age 53 years (range, 23–84), White 96%, male 45% Mean PGA disease activity 62 Enthesitis 69%, Dactylitis 22%, Nail psoriasis not reported Prior csIMM: One agent 38%, two 27%, three or more 29% Prior biologic: None 60%, one 31%, two or more 9%; TNFI 46% Concomitant medications: Mainly MTX 47% and NSAIDs 64%

SJC, Swollen joint count; **TJC**, Tender joint count

Results

- Efficacy data are summarized in Table 2.

Table 2 Efficacy results at Week 24 from phase 3 trials

Outcome	KEEP-sAKE Trial	RIS	PBO	Relative Risk (95% CI)	Difference (95% CI)	Q
Primary and Ranked Secondary Outcomes						
ACR20, n/N (%)	1	277/483 (57.3)	161/481 (33.5)	**1.7 (1.5, 2.0)**	**24.0 (18.0, 30.0)***	H
	2	115/224 (51.3)	58/219 (26.5)	**1.9 (1.5, 2.5)**	**24.5 (15.9, 33.0)***	H
MDA, n/N (%)	1	121/483 (25.0)	49/481 (10.2)	**2.5 (1.8, 3.3)**	**14.8 (10.2, 19.4)***	H
	2	57/224 (25.6)	25/219 (25.3)	**2.2 (1.4, 3.4)**	**22.6 (13.9, 31.2)***	H
No radiographic progression	1	423/458 (92.4)	401/457 (87.7)	1.1 (1.0, 1.1)	**4.6 (0.9, 8.4)**	L α β
CFB in PsA-mTSS, mean (95% CI)	1	0.23 (0.02, 0.44)	0.32 (0.11, 0.53)	—	–0.09 (–0.4, 0.2)	M α
CFB in mNAPSI, mean (95% CI) / N	1	–9.8 (–11.0, –8.6) / 309	–5.6 (–6.7, –4.4) / 338	—	**–4.2 (–5.7, –2.7)***	M γ
CFB in HAQ-DI, mean (95% CI)	1	–0.31 (–0.36, –0.27)	–0.11 (–0.16, –0.06)	—	–0.20 (–0.26, 0.14)*	H
	2	–0.22 (–28, –0.15)	–0.05 (–0.12, 0.02)	—	–0.16 (–0.26, 0.07)*	H
CFB in SF-36 PCS, mean (95% CI)	1	6.5 (5.8, 7.2)	3.2 (2.5, 3.9)	—	**3.3 (2.4, 4.2)**	H
	2	5.9 (4.9, 6.9)	2.0 (0.9, 3.1)	—	**3.9 (2.4, 5.3)***	H
Prespecified Pooled Analyses						
LEI 0, n/N (%)	1 + 2	215/444 (48.4)	156/448 (34.8)	**1.4 (1.2, 1.6)**	**13.9 (7.6, 20.2)***	M γ
LDI 0, n/N (%)	1 + 2	128/188 (68.1)	104/204 (51.0)	**1.3 (1.1, 1.6)**	**16.9 (7.5, 26.4)***	L β γ
Non-ranked Secondary Outcomes						
ACR70, n/N (%)	1	74/483 (15.3)	23/481 (4.7)	**3.2 (2.0, 5.0)**	**10.5 (6.9, 14.2)**	M δ
	2	27/224 (12.0)	13/219 (5.9)	**2.0 (1.1, 3.8)**	**6.0 (0.8, 11.3)**	M δ

Sources: AMCP Dossier[5]; FDA Multi-discipline Review[6]

Bold blue text indicates differences for which the confidence intervals exclude the value 0 or relative risks for which the confidence intervals exclude the value 1.0.

CFB, Change from baseline; **HAQ-DI**, Health Assessment Questionnaire-Disability Index (MCIC ≥ 0.35); **LDI**, Leeds Dactylitis Index; **LEI**, Leeds Enthesitis Index; **MCIC**, Minimal clinically important change; **MDA**, Minimal disease activity; **mNAPSI**, Modified Nail Psoriasis Severity Index; **PBO**, Placebo; **Q**, GRADE quality of evidence (H = High, M = Moderate, L = Low, VL = Very low); **PsA-mTSS**, Psoriatic arthritis-modified Total Sharp Score; **RIS**, Risankizumab-rzaa 150 mg; **SF-36 PCS**, 36-item Short-Form Health Survey Physical Component Summary (MCIC ≥ 3)[7]

* Statistically significant (p <0.001) under overall type I error control

α Downgraded for inconsistency

β Downgraded for imprecision (optimal information size not met)

γ Downgraded for risk of bias due to subgroup analysis without stratified randomization

δ Downgraded for risk of bias due to multiplicity

- The absolute effects for achieving ACR20 are presented in Table 3.

Table 3 Absolute Effects for Achieving ACR20 at Week 24, Risankizumab-rzaa vs Placebo

KEEPsAKE Trial	AAE, per 1000 pts (95% CI)	NNT (95% CI)
1	234 (167, 335) more	5 (4, 6)
2	238 (132, 397) more	5 (3, 7)

AAE, Anticipated absolute effect for achieving the outcome; **NNT**, Number needed to treat for one additional patient to benefit

- Secondary efficacy results
    - Risankizumab-rzaa showed significant benefit in all other secondary efficacy measures in both phase 3 trials, including PASI90 and ACR50 responses and percentage of patients who achieved minimal clinically important changes in HAQ-DI (CFB ≥ 0.35).
- Subgroup Analyses
    - In KEEPsAKE 1, Week-24 ACR20 response rates were higher with risankizumab-rzaa than placebo regardless of baseline patient or disease characteristics and regardless of whether risankizumab-rzaa was given with concomitant csIMM therapy (57.9% vs 35.9%) or as monotherapy (55.5% vs 26.2%).[2]
    - Similar subgroup results were seen In KEEPsAKE 2. Better ACR20 responses were seen in patients treated with risankizumab-rzaa vs placebo when analyzed by concomitant csIMM or risankizumab-rzaa monotherapy as well as by csIMM inadequate responders (56.3% vs 36.6%, respectively) or biologic inadequate responders (45.7% vs 14.9%, respectively), where inadequate responder referred to patients who had an inadequate response, intolerance or contraindication to therapy.[4] However, ACR20 response rates with risankizumab-rzaa seemed to be lower in the biologic inadequate responders than in the csIMM inadequate responders (45.7% and 56.3%, respectively).

Onset of Treatment Benefit and Duration of an Adequate Therapeutic Trial

- Onset of effects (earliest significant treatment difference) and duration of an adequate therapeutic trial are summarized by outcome measure in Table 4.

Table 4 Onset of Benefit and Adequate Therapeutic Trial

Outcome Measure	Trial	Onset of Significant Treatment Benefit (Wks)	Duration of an Adequate Therapeutic Trial (Wks)
ACR20 / ACR50 / ACR70	KEEPsAKE 1	4 / 4 / 8	16 / ≥ 24 / ≥ 24
	KEEPsAKE 2	4 / 8 / 8	16 / ≥ 24 / 24

Durability of Response

- No published data.

Evidence Gaps

- Patient Satisfaction
- Efficacy in magnetic resonance imaging (MRI)-identified axial PsA

Network Meta-analyses

- No network meta-analyses reviewing risankizumab-rzaa trials in PsA were found.

Safety Considerations

- The safety profile of risankizumab-rzaa in PsA was generally consistent with that in plaque psoriasis.[1]

Other Therapeutic Options

- The FDA approved indication for risankizumab-rzaa in active PsA does not require trials of any prior therapies.
- The general steps in systemic drug therapy of moderate to severe AD are shown in Table 5.

Table 5 Systemic Pharmacotherapies for PsA

Step in Therapy	Class	Treatment Alternatives	For Peripheral Synovitis / Dactylitis	For Joint Erosion	For Enthesitis		For Axial PsA
NSAIDs	NSAIDs	Various	Yes / No†	No	Yes†		Yes†
Glucocorticoids (local or systemic)	Glucocorticoids	Various	Yes	No	Yes		Yes‡
csIMM, typically methotrexate	Folate inhibitor	Methotrexate	Yes	No	Yes∞		No
	Pyrimidine inhibitor	Leflunomide					
	5-ASA	Sulfasalazine					
Non-JAKI tsIMM	PDE4I	Apremilast	Yes	No	Yes		No
First bDMARD, typically TNFI	TNFIs	Adalimumab Certolizumab Etanercept Golimumab Infliximab	Yes	Yes	Yes		Yes
	IL-17AI	Ixekizumab Secukinumab	Yes	Yes	Yes		Yes
	IL-12/23I	Ustekinumab	Yes	Yes	Yes		No
	IL-23I	Guselkumab[8,9,10]		Yes	Yes	Yes	No§
		Risankizumab-rzaa		Yes	No‖	Yes	No
Subsequent bDMARD or JAKI or PDE4I	TNFIs IL-17AI IL-12/23I IL-23I	As above			As above		
	PDE4I						
	JAKI	Tofacitinib Upadacitinib		Yes	Yes	Yes	Yes
	T-cell costimulation inhibitor	Abatacept[11,12,]		Yes	Uncertain	Uncertain∩	No

Sources: 13,14,15

5-ASA, 5-aminosalicylic acid; **csIMM**, Conventional synthetic immunomodulator; **IL-17AI**, Interleukin 17A inhibitor; **IL-12/23I**, Interleukin 12/23 inhibitor; **IL-23I**, Interleukin-12 inhibitor; **JAKI**, Janus kinase inhibitor; **PDE4I**, Phosphodiesterase-4 inhibitor; **tsIMM**, Targeted synthetic immunomodulator

† NSAIDs have not been shown to be effective for dactylitis, and are used for symptomatic relief (mild inflammation, pain, stiffness).

‡ Systemic glucocorticoids are not recommended for axial disease.

§ Guselkumab was ineffective for axial spondyloarthritis. Uncertain effects on MRI-verified axial PsA.

‖ Risankizumab-rzaa was not effective for radiographic progression in short term and not evaluated long-term.

∞ The 2021 GRAPPA guidelines on the management of psoriatic arthritis changed from *does not recommend* (insufficient evidence) to *conditionally recommends* methotrexate for enthesitis in NSAID inadequate responders based on the SEAM-PsA trial results, which showed that methotrexate was not significantly different from etanercept but there was no placebo control to determine effect size (low quality evidence).[15]

∩ The 2021 GRAPPA guidelines conditionally recommends abatacept for enthesitis in NSAIDs / cIMM inadequate responders based on an RCT showing numerical but not statistically significant improvement in enthesitis resolution rates vs placebo (low quality evidence).[15]

- Alternative treatments for active PsA are summarized in Table 6.

Table 6 Alternative Targeted Therapies FDA Approved for Active PsA

Drug (Formulary Status)	CFU Place in Therapy in PsA	2022 GRAPPA Place in Therapy	2019 EULAR Place in Therapy[16]	2018 ACR Place in Therapy[17]	Safety Considerations	Other Considerations
Interleukin-23 Inhibitor						
Risankizumab-rzaa NonF, CFU	TBD	IL-23I strongly recommended for P E D N S (not for axial PsA) and conditionally recommended for IBD.	Not included	Not included	Infections, TB Lacks IBD warning of IL-17AIs.	Approved for Crohn's disease. Effective for nail psoriasis in PsA.[18] Ineffective for axial spondyloarthritis.[19] No data for axial PsA.
Guselkumab NonF, CFU	After one TNFI, one IL-17AI, and ustekinumab.	See risankizumab	Not included	Not included	Infections, TB Lacks IBD warning of IL-17AIs.	A network meta-analysis showed that secukinumab 300 and 150 mg are nonsignificantly better than guselkumab 100 mg Q4W and Q8W in achieving ACR20.[20] Effective for nail psoriasis.
TNF Inhibitors						
Adalimumab Certolizumab Etanercept Infliximab-abda PA-F	—	TNFI strongly recommended for P E D A N S TNFI (not ETA) strongly recommended for IBD and conditionally recommended for uveitis.	Typically the initial bDMARD class selected. Should be started in patients with peripheral arthritis and an inadequate response to ≥ 1 csIMM. Should be considered in patients with enthesitis and inadequate response to NSAIDs or LGCIs.	Treatment-naïve active PsA; TNFIs are conditionally recommended over oral csIMMs or PDE4I, IL-17I, or IL-12/23I. Active PsA despite csIMM or PDE4I; switching to a TNFI is conditionally recommended over a different oral csIMM or PDE4I, switching to IL-17AI, IL-12/23I, abatacept, or tofacitinib.	Serious infections, HBV reactivation, TB, HIV, malignancy, demyelinating disease, autoimmune disorder (lupus-like), heart failure, hematocytopenias. Infliximab infusion reactions, hepatic reactions. Antidrug antibody-associated loss of efficacy. Long-term safety experience; clinician familiarity with use of TNFIs.	When switching within TNFI class, may consider switching from the TNFI mAbs to etanercept or vice versa. Infliximab / biosimilars are given IV; golimumab IV or SC; other TNFIs SC. Effective for nail psoriasis.
Golimumab Infliximab / Other Biosimilars NonF	—					

Drug (Formulary Status)	CFU Place in Therapy in PsA	2022 GRAPPA Place in Therapy	2019 EULAR Place in Therapy[16]	2018 ACR Place in Therapy[17]	Safety Considerations	Other Considerations
Interleukin-17A Inhibitors						
Ixekizumab NonF, CFU Secukinumab NonF, CFU	After TNFI. After TNFI.	IL-17AIs strongly recommended for P E D A N S See ixekizumab	Consider for TNFI inadequate responders with peripheral arthritis ± enthesitis without predominantly axial disease (same level as switching to another TNFI, IL-12/23I, JAKI, or PDE4I). Consider for TNFI or IL-17AI inadequate responders with predominantly axial disease (same level as switching within or between TNFI or IL-17AI class(es)).	Active PsA despite TNFI (conditionally recommended; same level as IL-12/23I, abatacept, or tofacitinib).	Infections (including mucocutaneous candidiasis), TB, onset or worsening of IBD.	Relative to adalimumab, ixekizumab was similar in ACR50 response and better in PASI100 response at Wk 52.[21] No direct comparisons of secukinumab with TNFI. For patients with major skin involvement (and peripheral or predominantly axial disease), an IL-17AI may be preferred over a TNFI. Effective for nail psoriasis.
JAK Inhibitors						
Tofacitinib NonF, CFU	After TNFI	JAKI strongly recommended for P E D A S (not for nail disease) JAKI conditionally recommended for IBD	Consider a JAKI for patients with peripheral arthritis ± enthesitis without predominant axial disease after inadequate response to bDMARD or when a bDMARD is inadvisable (same level as switching to another bDMARD or PDE4I).	For active PsA despite TNFI, IL-17AI, and IL-12/23I (conditionally recommended; same level as abatacept). May consider tofacitinib over TNFI, IL-17AI, or IL-12/23I if patient prefers oral medication.	Higher risk of mortality, malignancy, MACE, and thrombosis than TNFIs in patients ≥ 50 yrs with rheumatoid arthritis and ≥ 1 CV risk factor.[22] Not recommended in severe (Child-Pugh class C) liver disease.	PI Place in Therapy in PsA: Inadequate response or intolerance to ≥ 1 TNFI. Administered orally. Avoid co-use with strong CYP3A4 inducers. Hematologic, renal, and hepatic dosage adjustments. Effective for nail psoriasis. FDA approved for ulcerative colitis.
Upadacitinib NonF, CFU	After TNFI	See tofacitinib	Not specifically reviewed. See tofacitinib.	Not specifically reviewed. See tofacitinib.	Mortality, malignancy, MACE, thrombosis, hematocytopenias, infection, TB, HBV, HZ, hepatotoxicity, increased lipids, bowel perforation.	PI Place in Therapy in PsA: Inadequate response or intolerance to ≥ 1 TNFI.

Drug (Formulary Status)	CFU Place in Therapy in PsA	2022 GRAPPA Place in Therapy	2019 EULAR Place in Therapy[16]	2018 ACR Place in Therapy[17]	Safety Considerations	Other Considerations
					Not recommended in severe (Child-Pugh class C) liver disease.	FDA approved for ankylosing spondylitis and ulcerative colitis. Administered orally. Not recommended for co-use with strong CYP3A4 inhibitors or inducers. Hematologic and renal dosage adjustments. Unknown effects on nail psoriasis.
Interleukin-12/23 Inhibitor						
Ustekinumab NonF, CFU	After one TNFI and one IL-17AI.	Strongly recommended for P E D N S (not for axial disease) Strongly recommended for IBD	Consider for inadequate responders to TNFI with peripheral arthritis ± enthesitis without predominant axial disease (same level as switching to another TNFI, IL-17AI, abatacept, JAKI, or PDE4I).	Active PsA despite TNFI and IL-17AI (conditionally recommended; same level as abatacept or tofacitinib).	Infections, malignancy, noninfectious pneumonia, posterior reversible encephalopathy syndrome, TB.	For patients with major skin involvement and peripheral arthritis, an IL-12/23I may be preferred over a TNFI. Patients > 100 kg may require higher doses. Effective for nail psoriasis.

Drug (Formulary Status)	CFU Place in Therapy in PsA	2022 GRAPPA Place in Therapy	2019 EULAR Place in Therapy[16]	2018 ACR Place in Therapy[17]	Safety Considerations	Other Considerations
Phosphodiesterase-4 Inhibitor						
Apremilast VANF, CFU	After a csIMM and TNFI is medically inadvisable.	PDE4I strongly recommended for P E D N S (not for axial disease)	Consider for mild, nonerosive, nonaxial disease in csIMM inadequate responders when neither a bDMARD nor JAKI is advisable, or for peripheral arthritis ± enthesitis without predominant axial disease in bDMARD inadequate responders (same level as switching to another bDMARD or JAKI).	Not recommended in TNFI inadequate responders.	Slow up-titration of dosage in first week is recommended to reduce GI adverse effects. Neuropsychiatric effects; weight loss. Renal dosage adjustment.	Generally favorable safety profile. Administered orally. Effective for nail psoriasis.
T-cell Costimulation Inhibitor						
Abatacept NonF	—	Conditionally recommended for P E D (not for axial, nail, or skin disease)	Should be limited to inadequate responders to other bDMARDs.	Active PsA despite TNFI, IL-17AI, and IL-12/23I (conditionally recommended; same level as tofacitinib).	Infections, malignancy, COPD exacerbation; TB and HBV screening	Low efficacy. Administered IV or SC. Unknown effects on nail psoriasis.

Sources: 10,13,17,23,24,25

CFU, Criteria for Use; **IBD**, Inflammatory bowel disease; **LGCI**, Local glucocorticoid injection; **NonF**, Nonformulary; **PA-F**, Formulary with Prior Authorization-Facility; **PASI100**, At least 100% improvement in Psoriasis Area and Severity Index; P E D A N S, Peripheral arthritis, enthesitis, dactylitis, axial disease, nail disease, skin disease / psoriasis; **TB**, Tuberculosis; **TBD**, To be decided.

Projected Place in Therapy

- **Potential Place in Therapy Based on the Evidence**. Although no head-to-head trials were available to inform place in therapy, moderate- to high-quality evidence from placebo-controlled trials supports the use of risankizumab-rzaa in patients with active PsA who have had an inadequate response to conventional immunomodulators and/or biologics or for whom these therapies are medically inadvisable. There was low certainty of the effect size for improvement in dactylitis with short-term (24-week) risankizumab-rzaa therapy. Short-term risankizumab-rzaa therapy was ineffective for preventing radiographic progression of peripheral erosive disease; long-term studies are needed to further assess anti-erosive effects. There was no available data to support its efficacy for symptomatic, MRI-verified axial PsA. Overall, short-term risankizumab-rzaa therapy was effective for achievement of ACR20 response (small to moderate effects) and minimal disease activity and for improvement of joint, entheseal,

dactylitis, and nail manifestations, function, and quality of life. Benefits were clinically meaningful. Further studies are required to evaluate the long-term safety of risankizumab-rzaa and its efficacy and safety in preventing radiographic progression and treating axial PsA.

- **Potential Place in Therapy in VHA**. Risankizumab-rzaa may be an alternative to (at the same level as) ustekinumab and the other IL-23I guselkumab in patients with active nonaxial, nonerosive PsA who have an inadequate response to at least one TNFI and at least one IL-17AI (ixekizumab preferred in new starts), unless they are medically inadvisable. IL-23Is may be preferred over ustekinumab for severe PsA-associated plaque psoriasis, and ustekinumab may be preferred over the IL-23Is in patients with ulcerative colitis. There is no comparative clinical evidence available to inform whether one IL-23I can be preferred over the other in the treatment of active PsA; however, guselkumab has been shown in longer term studies to keep radiographic progression low[10] while risankizumab-rzaa has not shown benefit for joint erosions in the short term.

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