Patient-Reported Outcomes Through 1 Year With Guselkumab in Participants With Active PsA and Inadequate Response to 1 Prior TNFi in SOLSTICE

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Key Takeaways

- **At W24 in SOLSTICE, GUS-treated participants with active PsA had greater improvements vs PBO in overall HRQoL as assessed by PROMIS-29 and PsAID-12**
- **Participants receiving GUS also had greater response rates vs PBO at W24 for achieving PsAID-12 meaningful improvement and PASS**
- **Mean improvements and response rates were maintained through W52 among GUS-randomized participants**

Background

- Psoriatic arthritis (PsA) is a chronic inflammatory disease with significant impacts on health-related quality of life (HRQoL)[1]
- Many patients treated with tumor necrosis factor inhibitors (TNFi) experience lack or loss of efficacy[1,2]
 - There are no clear guidelines for subsequent therapy options[1,2]
- Guselkumab (GUS) is a fully human, dual-acting, selective interleukin (IL)-23p19-subunit inhibitor[3]
 - GUS demonstrated efficacy in participants with active PsA and inadequate response (inadequate efficacy and/or intolerance) to 1 prior TNFi (TNFi-IR) through Week (W) 24 in the phase 3b double-blind, randomized, placebo (PBO)-controlled SOLSTICE study[4]

Objective

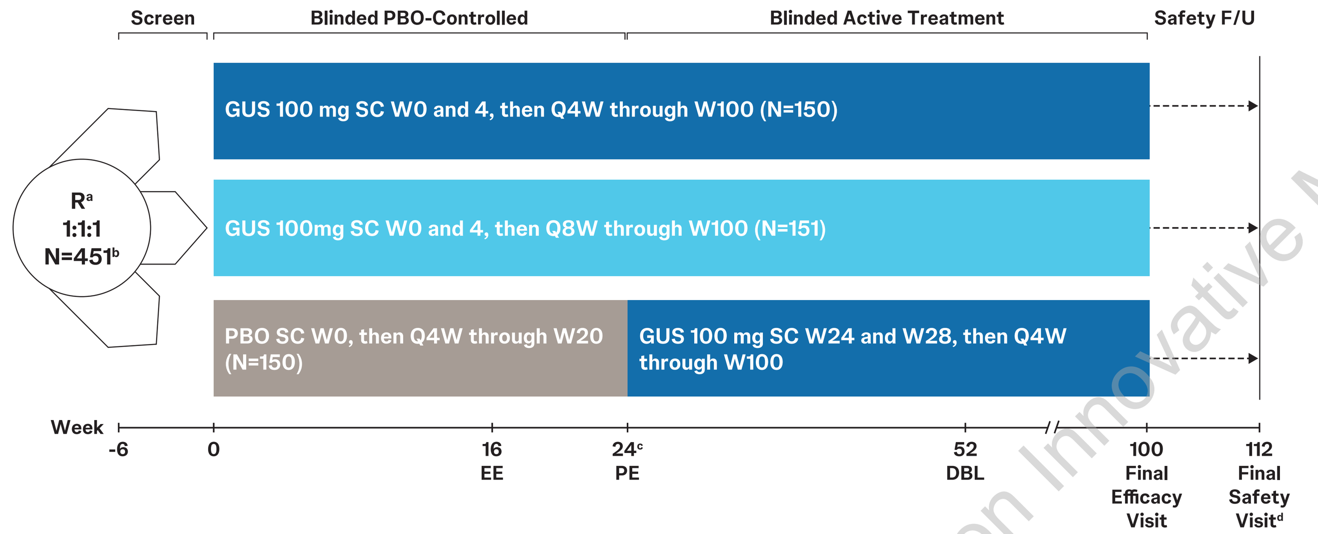
To evaluate the effect of GUS on patient-reported outcomes (PROs) in participants with TNFi-IR in SOLSTICE

Methods

SOLSTICE – Study Design

Key Inclusion Criteria

- Age ≥18 years
- Active PsA (≥3 SJC; ≥3 TJC; CRP ≥0.3 mg/dL); CASPAR criteria met
- **TNFi-IR to 1 prior TNFi**
- History of or active PsO (≥1 plaque ≥2 cm and/or nail PsO)



Screen
Blinded PBO-Controlled
Blinded Active Treatment
Safety F/U
R[a]
1:1:1
N=451[b]
GUS 100 mg SC W0 and 4, then Q4W through W100 (N=150)
GUS 100mg SC W0 and 4, then Q8W through W100 (N=151)
PBO SC W0, then Q4W through W20 (N=150)
GUS 100 mg SC W24 and W28, then Q4W through W100
Week
-6
0
16
EE
24[c]
PE
52
DBL
100
Final Efficacy Visit
112
Final Safety Visit[d]

PRO Assessments:

- PROMIS-29: 7 domains (physical function, social participation, depression, anxiety, pain interference, fatigue, and sleep disturbance) and a pain intensity VAS; domain scores are converted to standardized T-scores
- PsAID-12: 12 domains examining physical and psychological perspectives (final score range, 0 [no difficulty] to 10 [extreme difficulty])

Statistical Analyses:

- LSM changes from baseline in PROMIS-29 domains and total PsAID-12 score, and proportions of participants achieving PsAID-12 MCID (improvement ≥3) and PASS (score ≤4) were determined at W24 and W52

*[a]Randomization was stratified by baseline use of csDMARDs. [b]Total number randomized=453, the full analysis set of 451 excludes 1 participant who was double randomized. [c]Crossover. [d]Final safety F/U at W112 is 12W after final study agent administration. **CASPAR**=ClASsification criteria for Psoriatic ARthritis, **CRP**=C-reactive protein, **csDMARDs**=conventional synthetic disease-modifying antirheumatic drugs, **DBL**=database lock, **EE**=early escape, **F/U**=follow-up, **LSM**=least-squares mean, **MCID**=minimal clinically important difference, **PASS**=Patient Acceptable Symptom State, **PE**=primary endpoint, **PROMIS-29**=Patient-Reported Outcomes Measurement Information System-29 profile instrument, **PsAID-12**=12-item PsA Impact of Disease, **PsO**=psoriasis, **Q4/8W**=every 4/8 weeks, **SC**=subcutaneous, **SJC**=swollen joint count, **TJC**=tender joint count, **VAS**=visual analog scale.*

Results

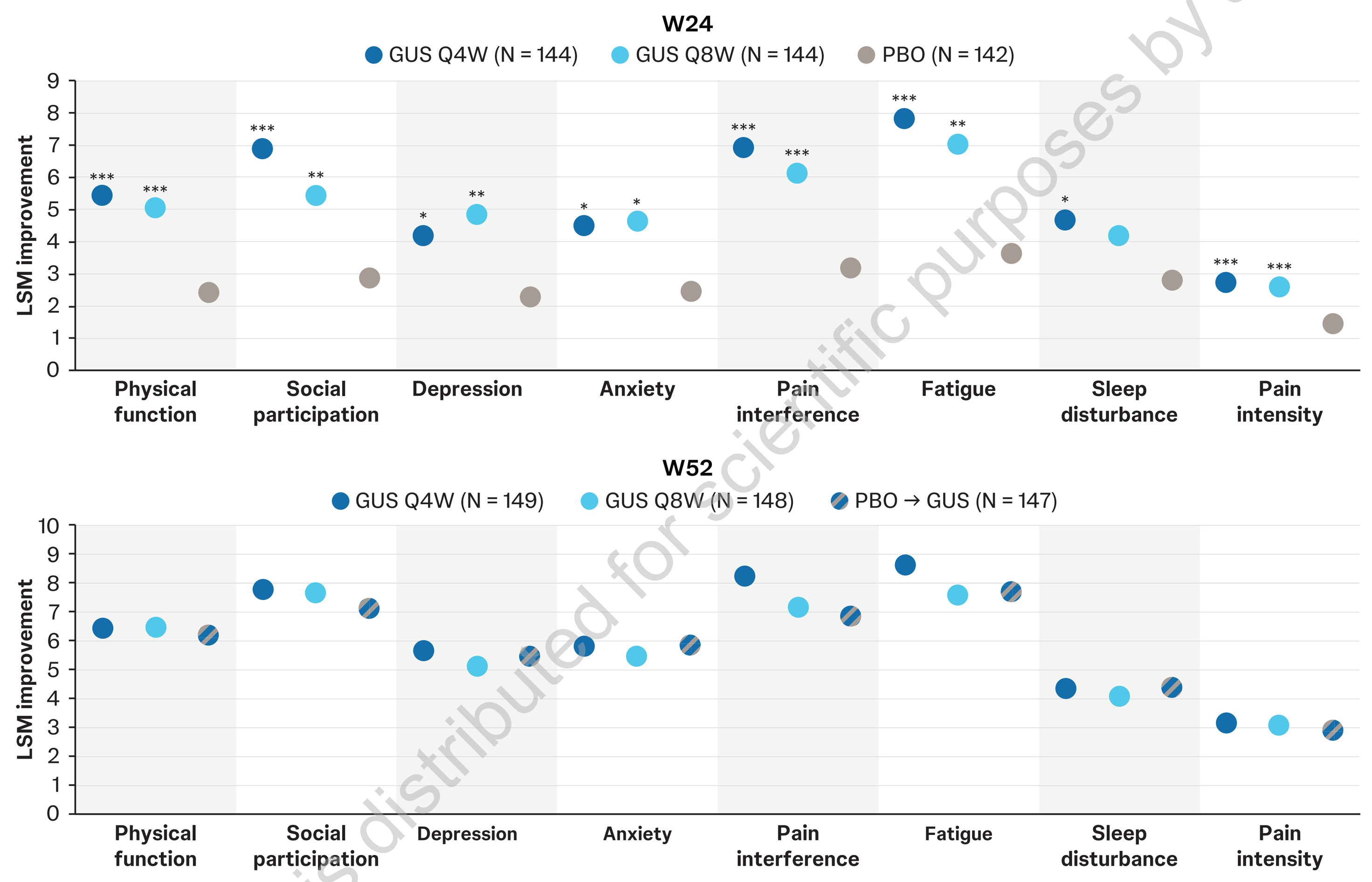
Baseline characteristics were well balanced across treatment groups

	GUS 100 mg Q4W (N = 150)	GUS 100 mg Q8W (N = 151)	PBO (N = 150)
Demographics			
Age, yrs	50.6 (13.3)	51.9 (12.9)	49.2 ± 12.6
Female	50%	51%	57%
Race, White	94%	95%	94%
BMI, kg/m²	30.0 (6.5)	30.9 (6.6)	30.0 (7.3)
Disease Characteristics			
SJC (0-66)	10.7 (7.8)	10.3 (6.6)	10.2 (6.4)
TJC (0-68)	18.1 (12.6)	17.1 (11.2)	16.8 (11.6)
PROMIS-29			
Physical function T-score[a]	38.5 (6.1)	38.0 (5.9)	38.2 (5.4)
Social participation T-score[a]	45.3 (8.1)	45.8 (8.7)	45.0 (7.2)
Depression T-score[b]	51.7 (9.2)	51.9 (9.4)	52.8 (9.7)
Anxiety T-score[b]	53.9 (10.2)	52.5 (9.4)	54.4 (10.4)
Pain interference T-score[b]	62.1 (6.0)	61.9 (6.4)	62.6 (5.9)
Fatigue T-score[b]	55.5 (8.8)	55.2 (9.7)	55.9 (9.4)
Sleep disturbance T-score[b]	53.5 (6.9)	52.7 (7.9)	53.5 (8.3)
Pain intensity VAS (0-10)[b]	6.6 (1.9)	6.5 (1.9)	6.6 (1.8)
PsAID-12 (0-10)	5.6 (1.9)	5.5 (2.0)	5.7 (1.9)
Concomitant methotrexate use	50%	46%	48%

*Data shown are mean (SD) unless otherwise noted. [a]Higher scores indicate better health outcomes. [b]Higher scores indicate more severe symptoms. **BMI**=body mass index, **SD**=standard deviation.*

LSM improvements from baseline in PROMIS-29 T-scores and pain intensity VAS were greater for GUS-treated participants vs PBO at W24 across all domains

LSM improvement from baseline at W24 and W52 in PROMIS-29 domain T-scores and pain intensity VAS



W24
GUS Q4W (N = 144)
GUS Q8W (N = 144)
PBO (N = 142)
LSM improvement
Physical function
Social participation
Depression
Anxiety
Pain interference
Fatigue
Sleep disturbance
Pain intensity
W52
GUS Q4W (N = 149)
GUS Q8W (N = 148)
PBO → GUS (N = 147)

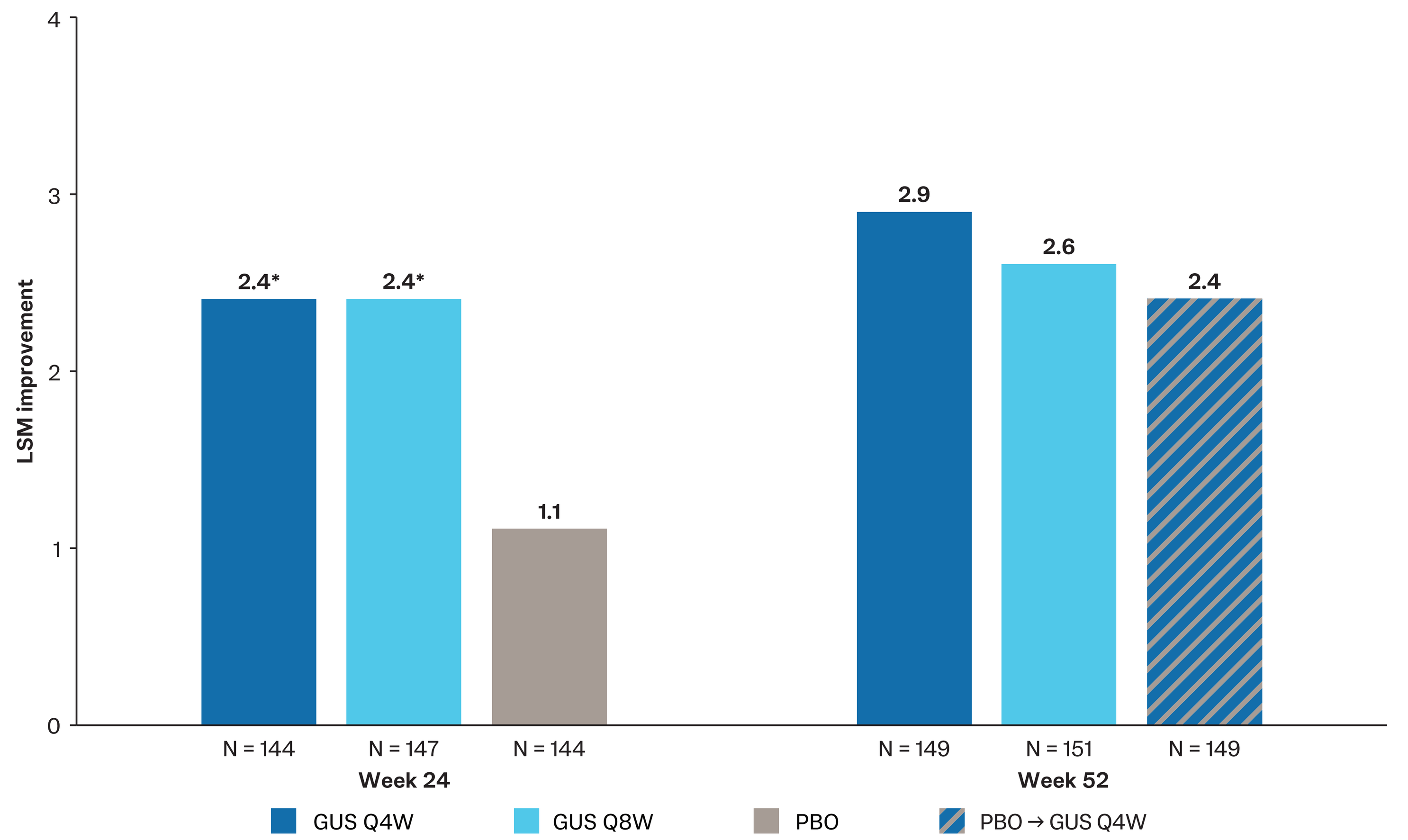
Nominal *p<0.05, **p<0.01, *p<0.001**

LSM and nominal p values were based on Mixed-Effect Model for Repeated Measures. Participants were considered nonresponders through W24 if they increased dose/initiated csDMARDs or oral corticosteroids or initiated protocol-prohibited PsA therapies and through W52 if they discontinued study agent for any reason other than natural disaster (COVID-19 pandemic)/major disruption (Ukraine-Russia crisis). Data impacted by natural disaster/major disruption were not used; other missing data were accounted for in the analysis model.

LSM changes in PsAID-12 total scores showed greater improvement from baseline with both GUS dosing regimens vs PBO at W24

- Improvements with GUS were maintained or numerically increased through W52

LSM improvements from baseline in PsAID-12 total scores at W24 and W52

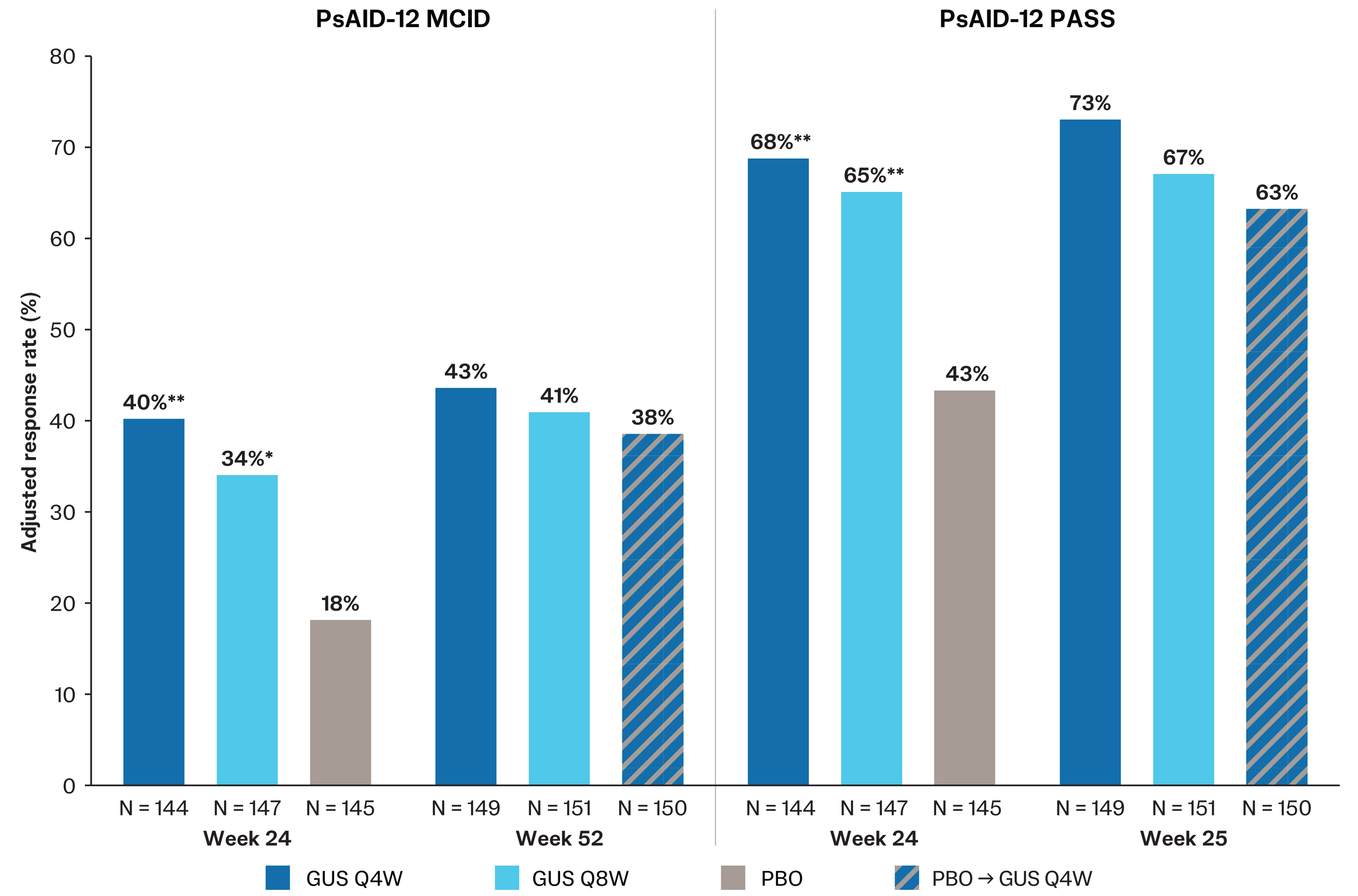


LSM improvement
2.4*
2.4*
1.1
2.9
2.6
2.4
N = 144
N = 147
N = 144
Week 24
N = 149
N = 151
N = 149
Week 52
GUS Q4W
GUS Q8W
PBO
PBO → GUS Q4W

Nominal *p<0.001

LSM and nominal p values for LSM improvements from baseline in PsAID-12 total scores were based on Mixed-Effect Model for Repeated Measures. Participants were considered nonresponders through W24 if they increased dose/initiated csDMARDs or oral corticosteroids or initiated protocol-prohibited PsA therapies and through W52 if they discontinued study agent for any reason other than natural disaster (COVID-19 pandemic)/major disruption (Ukraine-Russia crisis). Data for LSM improvements from baseline in PsAID-12 total scores that were impacted by natural disaster/major disruption were not used; other missing data were accounted for in the analysis model.

Response rates for achieving PsAID-12 MCID and PASS were higher with GUS vs PBO at W24 and numerically increased at W52 with GUS



PsAID-12 MCID
PsAID-12 PASS
Adjusted response rate (%)
40%**
34%*
18%
43%
41%
38%
68%**
65%**
43%
73%
67%
63%
N = 144
N = 147
N = 145
Week 24
N = 149
N = 151
N = 150
Week 52
N = 144
N = 147
N = 145
Week 24
N = 149
N = 151
N = 150
Week 25
GUS Q4W
GUS Q8W
PBO
PBO → GUS Q4W

Nominal *p<0.01, **p<0.001

Response rates and nominal p values for proportions of participants achieving PsAID-12 MCID (improvement ≥3) and PASS (score ≤4) were based on a Generalized Linear Mixed Model. Participants were considered nonresponders through W24 if they increased dose/initiated csDMARDs or oral corticosteroids or initiated protocol-prohibited PsA therapies and through W52 if they discontinued study agent for any reason other than natural disaster (COVID-19 pandemic)/major disruption (Ukraine-Russia crisis). Data for proportions of participants achieving PsAID-12 MCID and PASS that were impacted by natural disaster/major disruption were accounted for in the analysis model; other missing data were imputed using nonresponder imputation.

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REFERENCES: 1. Singh JA. *Arthritis Rheumatol.* 2019;71:5-32. **2.** Coates LC. *Nat Rev Rheumatol.* 2022;18:465-479. **3.** Sachen KL. *Front Immunol.* 2025;16:1532852. **4.** Ogdie A. *Arthritis Rheumatol.* 2026;78:1674-1686.

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