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Addressing Unmet Needs in Psoriatic Arthritis: Diagnosis, Pathogenesis, and Newer/Emerging Oral Therapies

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Faculty Disclosures

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Learning Objectives

- Summarize unmet needs in psoriatic arthritis (PsA) care, including delayed diagnosis and undertreatment
- Describe the pathophysiology of PsA and the therapeutic implications of targeting the JAK/STAT signaling pathway for PsA treatment
- Evaluate the safety, efficacy, and indications of available and emerging oral treatments for PsA
- Generate individualized treatment strategies for PsA that consider eligibility for oral treatment, comorbidities, and patient preferences

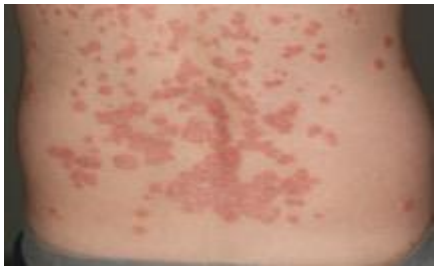
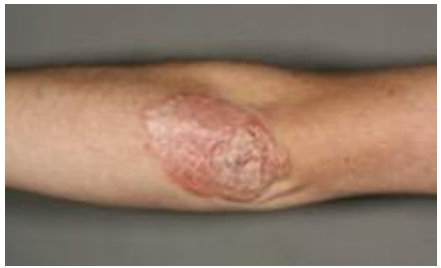
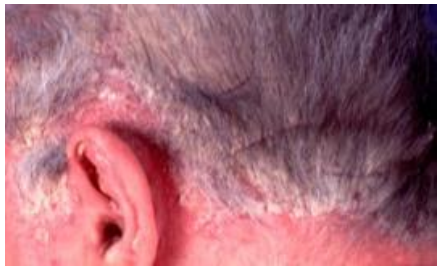
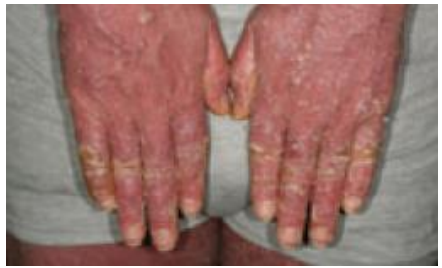
Psoriasis Affects ~7.5 Million Adults in the United States[1]

Plaque psoriasis
(psoriasis vulgaris)
affects
≈85%-90%
of all patients with psoriasis[2]

Characterized by **raised, red, scaly patches** primarily on **elbows, knees,** and **lower back**[2]

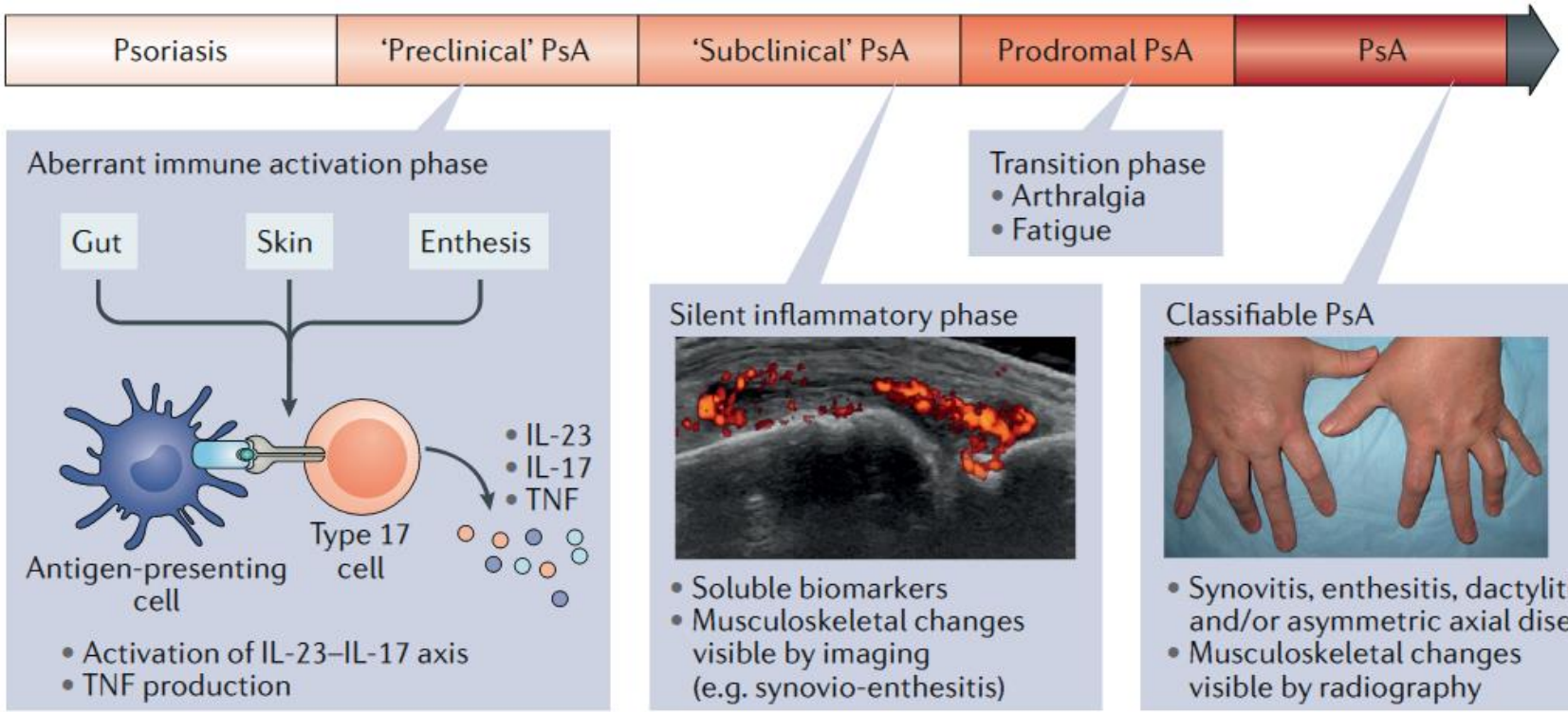
Up to
30%
of psoriasis patients develop psoriatic arthritis[3]

Patients with characteristic psoriasis plaques on trunk, elbow, scalp, and hands[4,5]

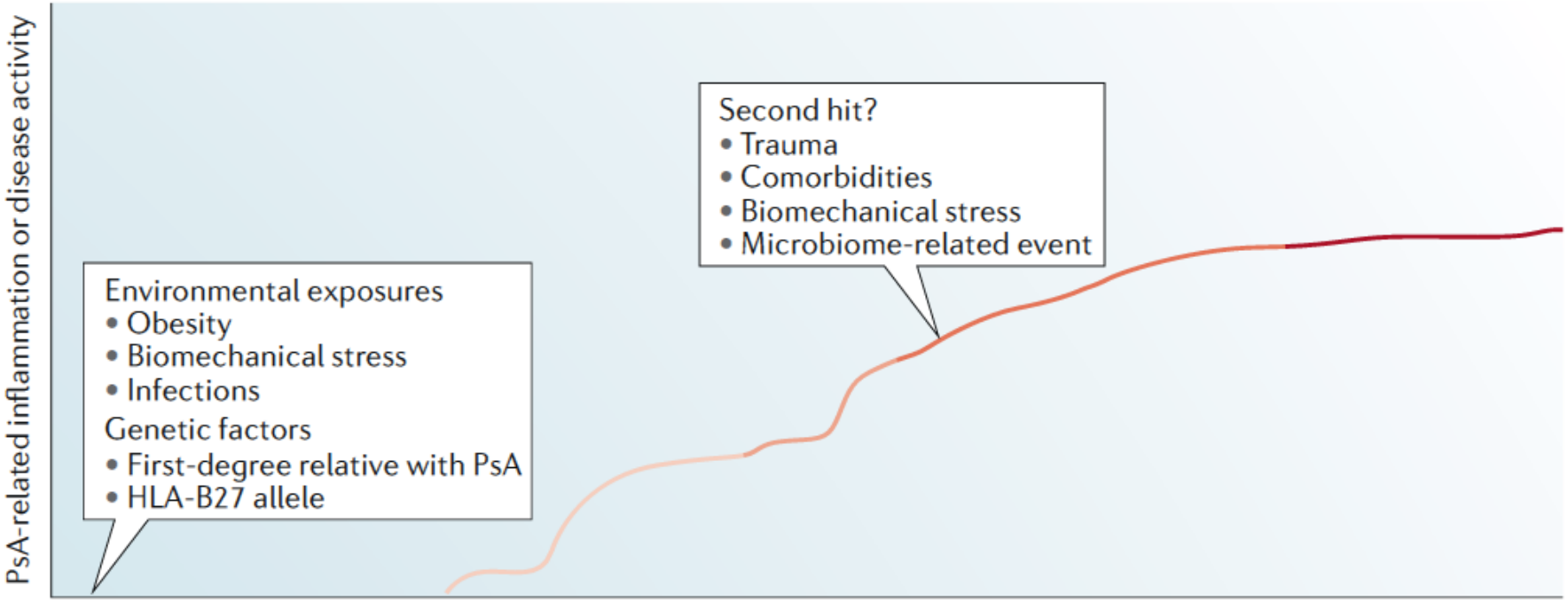
1. National Psoriasis Foundation [www.psoriasis.org]. Accessed August 15, 2018. Last updated June 24, 2025. https://www.psoriasis.org/about-psoriasis. 2. Nestle FO, et al. *N Engl J Med*. 2009;361(5):496-509. 3. Boehncke WH, Menter A. *Am J Clin Dermatol*. 2013;14(5):377-388. 4. Augustin M, et al. *J Eur Acad Dermatol Venereol*. 2012;26(Suppl 4):1-16. 5. Wozel G. *Clin Dermatol*. 2008;26(5):448-459.

Psoriasis → Psoriatic Arthritis



IL = interleukin; TNF = tumor necrosis factor.
Scher JU, et al. *Nat Rev Rheumatol.* 2019;15(3):153-166.

Who Is at Risk for Developing Psoriatic Arthritis?



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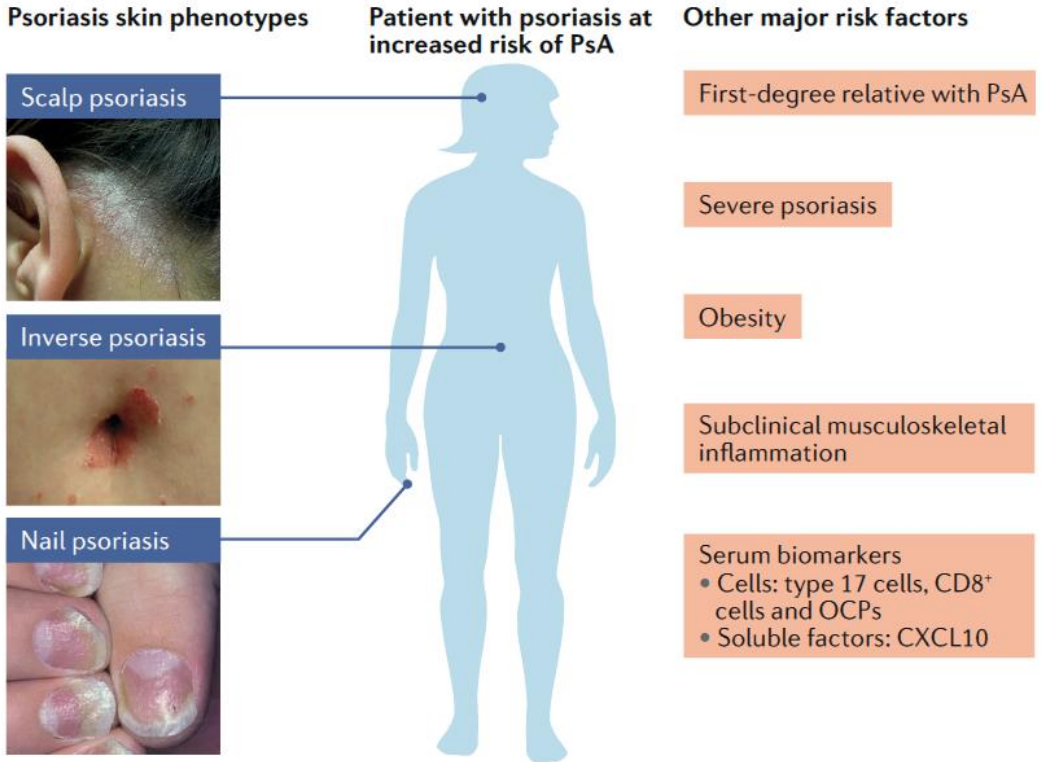
Who Is at Risk for Developing Psoriatic Arthritis?



OCP = osteoclast precursor.

Scher JU, et al. *Nat Rev Rheumatol*. 2019;15(3):153-166.

More Than 1 Million People in the United States Have PsA[1]

Peak onset between

30 and **50**

years of age[2]

Affects

men and women equally[2]

Approximately

85%

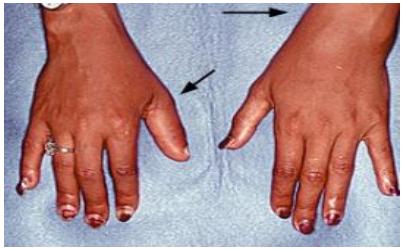
of psoriatic arthritis patients develop psoriasis before experiencing joint symptoms[3]

1. PR Newswire [www.prnewswire.com]. Accessed August 15, 2018. Last updated June 26, 2014. https://www.prnewswire.com/news-releases/national-psoriasis-foundation-prioritizes-psoriatic-arthritis-with-new-program-264714991.html. 2. Gottlieb A, et al. *J Am Acad Dermatol*. 2008;58(5):851-864. 3. Spondylitis Association of America [www.spondylitis.org]. Accessed February 4, 2026. https://spondylitis.org/spondylitis-plus/a-comprehensive-review-of-psoriatic-arthritis-symptoms-diagnosis-and-treatment.

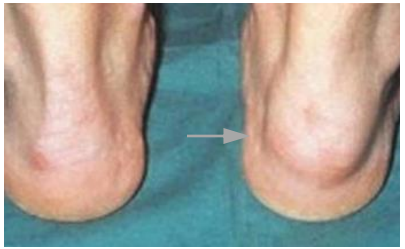
PsA Is a Heterogeneous Disease That Manifests in Multiple Ways across Different Domains[1,2]

- Clinical presentations can range from a few affected joints asymmetrically distributed on either side of the body to many joints on both sides of the body[3]
- Each patient experiences a unique burden of disease with an individual combination of symptoms and severity[4]
- Limited use of screening tools, absence of definitive biomarkers, and variable clinical presentations in PsA contribute to delayed diagnosis and undertreatment and represent a continuing unmet need in psoriatic arthritis care

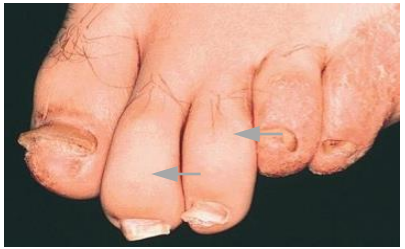
Swollen joints[5]



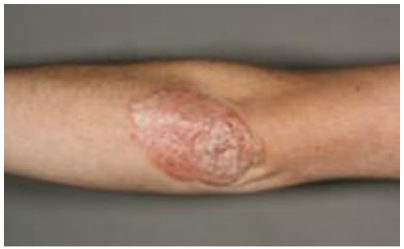
Enthesitis[6]



Dactylitis[7]

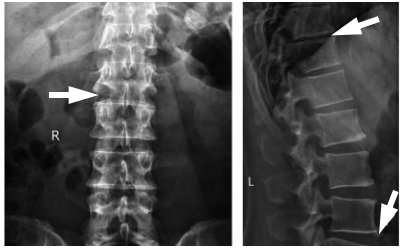


Psoriasis[8]



Nail disease[9]

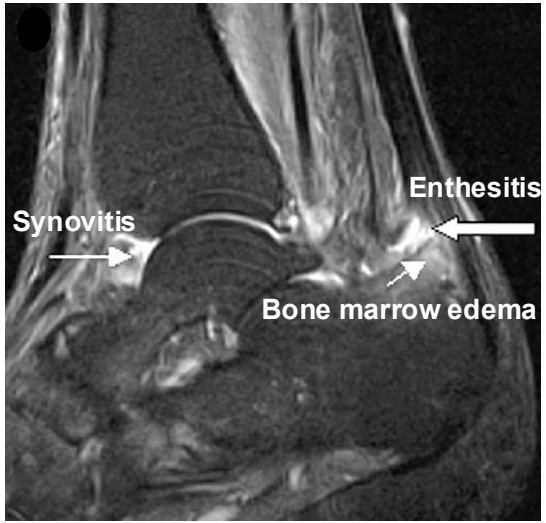
Axial disease[10]



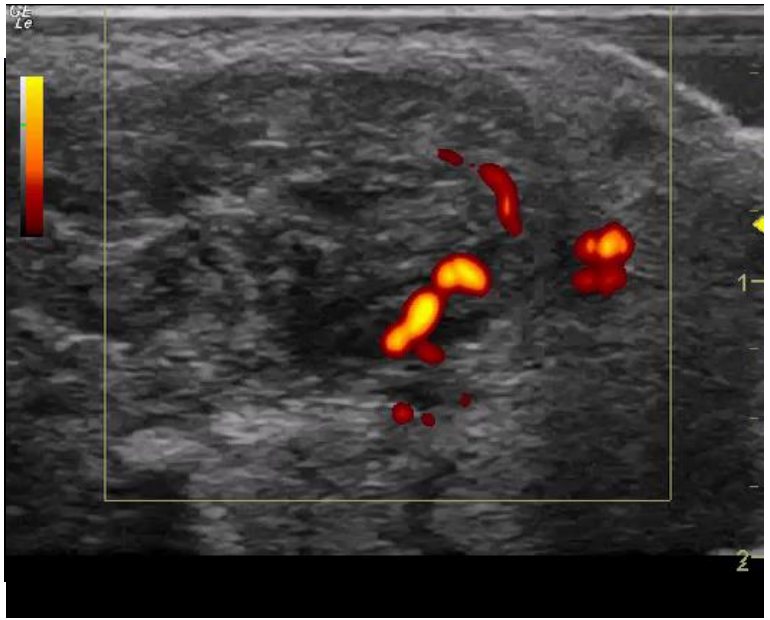
1. Sritheran D, Leung YY. *Ther Adv Musculoskel Dis*. 2015;7(5):173-186. 2. Coates LC, Helliwell PS. *Clin Med (Lond)*. 2017;17(1):65-70. 3. Moll JM, Wright V. *Semin Arthritis Rheum*. 1973;3(1):55-78. 4. Kavanaugh A, et al. *Rheumatol Ther*. 2016;3(1):91-102. 5. *Kataria R, Brent LH. Am Fam Physician. 2004;69(12):2853-2860*. 6. Gottlieb A, et al. *J Am Acad Dermatol*. 2008;58(5):851-864. 7. American College of Rheumatology (ACR) [images.rheumatology.org]. Accessed August 15, 2018. http://images.rheumatology.org/viewphoto.php?imageId=2897706&albumId=77030. 8. Augustin M, et al. *J Eur Acad Dermatol Venereol*. 2012;26(Suppl 4):1-16. 9. Sobolewski P, et al. *Reumatologia*. 2017;55(3):131-135. 10. Baraliakos X, et al. *Clin Exp Rheumatol*. 2015;33(5 Suppl 93):S31-S35.

Imaging Changes in PsA Are Thought to Be the Result of Chronic Aberrant Immune Responses[1-3]

The primary site of disease in psoriatic arthritis is believed to be the enthesis[4]



Sagittal MRI of the ankle region of a patient with psoriatic arthritis[3]



IFN = interferon; LT = lymphotoxin; MRI = magnetic resonance imaging.

1. Schafer P. *Biochem Pharmacol*. 2012;83(12):1583-1590. 2. Juvenile Arthritis Information. Accessed July 29, 2013. http://juvenilearthritisinfo.wordpress.com/jia/era. 3. McQueen F, et al. *Arthritis Res Ther*. 2006;8(2):207. 4. McGonagle D, et al. *Dermatology*. 2012;225(2):100-109. 5. Chimenti MS, et al. *Autoimmun Rev*. 2013;12(5):599-606. 6. ACR Psoriatic Arthritis [www.rheumatology.org]. Accessed July 29, 2013. https://rheumatology.org/patients/psoriatic-arthritis. 7. Conigliaro P, et al. *Autoimmun Rev*. 2011;10(10):577-581. 8. Gottlieb A, et al. *J Am Acad Dermatol*. 2008;58(5):851-864.

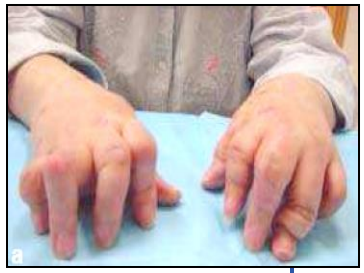
CASPAR Criteria for Classification of PsA[1-4]

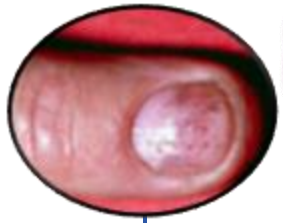
Established inflammatory articular disease with at least 3 points from the following

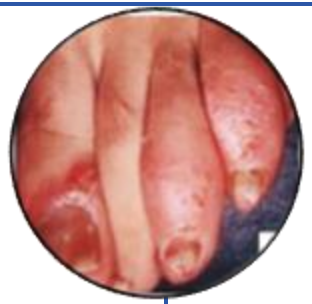
- Current psoriasis (2 points)
- Personal or family history of psoriasis (1 point)

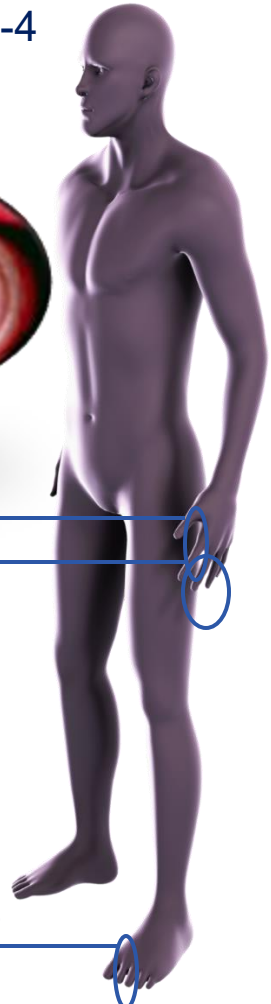
All of the remaining (1 point each)

- Dactylitis
- RF negativity
- Psoriasis nail dystrophy
- Periarticular osteitis









Psoriasis nail dystrophy[3]

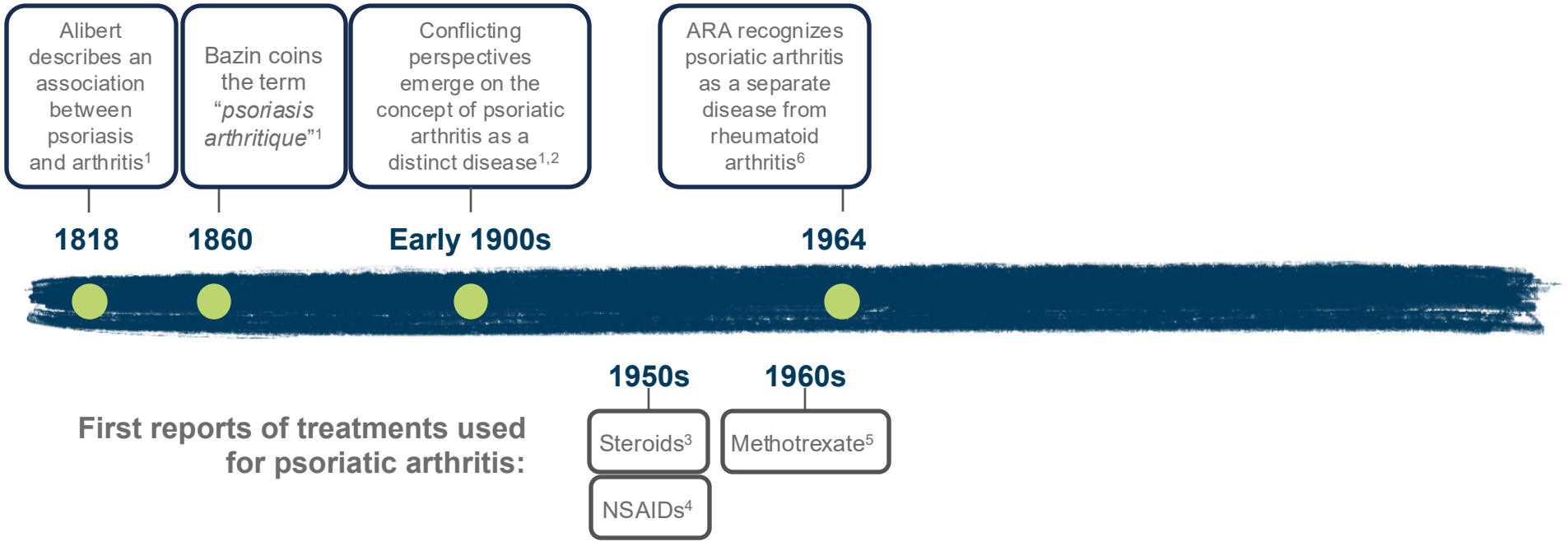
Inflamed joint[2]

Dactylitis[4]

CASPAR = ClASsification for Psoriatic ARthritis; RF = rheumatoid factor.

1.Taylor W, et al; and the CASPAR Study Group. *Arthritis Rheum.* 2006;54(8):2665-2673. 2. Leung YY, et al. *J Postgrad Med.* 2007;53(1):63-71. 3. Menter A, et al. *J Am Acad Dermatol.* 2008;58(5):826-850. 4. Gottlieb A, et al. *J Am Acad Dermatol.* 2008;58(5):851-864.

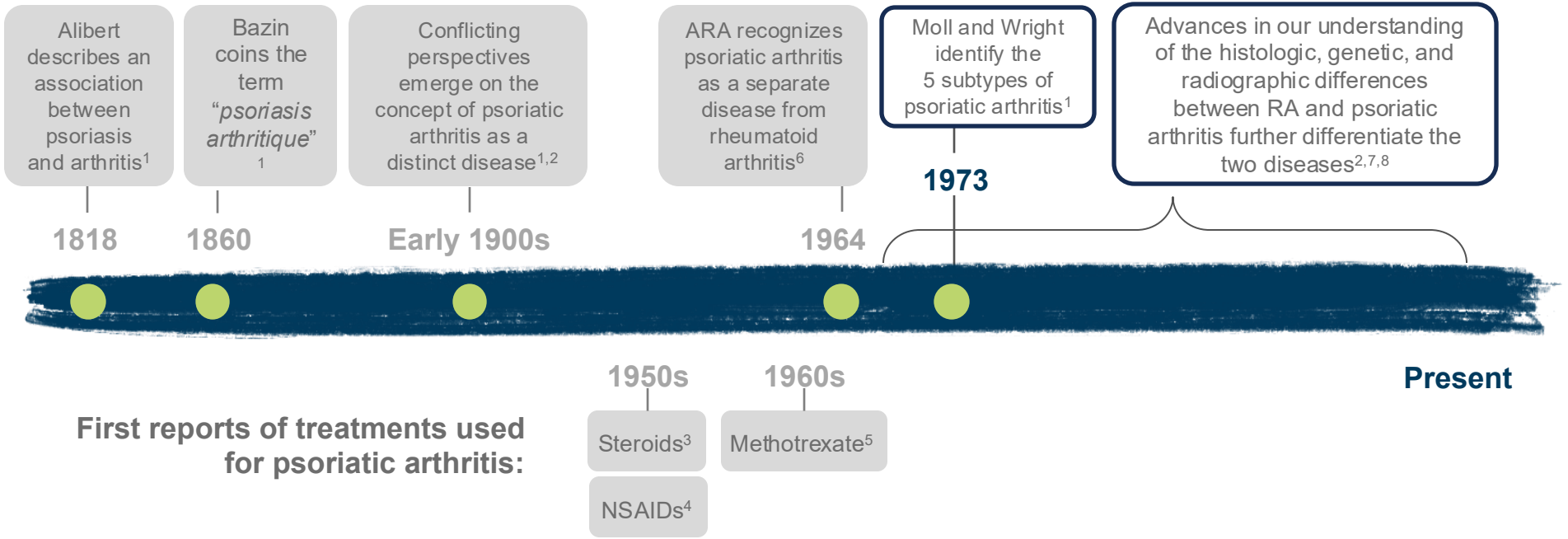
Evolution of the Understanding of Psoriatic Arthritis



ARA = American Rheumatism Association (now the American College of Rheumatology [ACR]); NSAID = nonsteroidal antiinflammatory drug.

1. Moll JM, Wright V. *Semin Arthritis Rheum*. 1973;3(1):55-78. 2. Espinoza LR. *Curr Rheumatol Rep*. 2018;20(10):58. 3. Boland EW. *Calif Med*. 1950;72(6):405-414. 4. Ulbricht H. *Dermatol Wochenschr*. 1952;126(50):1189-1191. 5. Weinblatt ME. Trans Am Clin Climatol Assoc. 2013;124:16-25. 6. Blumberg BS, et al. *Arthritis Rheum*. 1964:7:93-97.

Evolution of the Understanding of Psoriatic Arthritis



RA = rheumatoid arthritis.

1. Moll JM, Wright V. *Semin Arthritis Rheum*. 1973;3(1):55-78. 2. Espinoza LR. *Curr Rheumatol Rep*. 2018;20(10):58. 3. Boland EW. *Calif Med*. 1950;72(6):405-414. 4. Ulbricht H. *Dermatol Wochenschr*. 1952;126(50):1189-1191. 5. Weinblatt ME. Trans Am Clin Climatol Assoc. 2013;124:16-25. 6. Blumberg BS, et al. *Arthritis Rheum*. 1964:7:93-97. 7. Espinoza LR. Curr Rheum Rep. 2018;20(10):58. 8. McGonagle D. *Ann Rheum Dis*. 2005;64(Suppl 2):ii58-ii60.

Since the Turn of the Century, the Therapeutic Landscape Has Evolved to Include Many New FDA-Approved Therapies

Treatments Prior to Indicated Therapies[1-4]

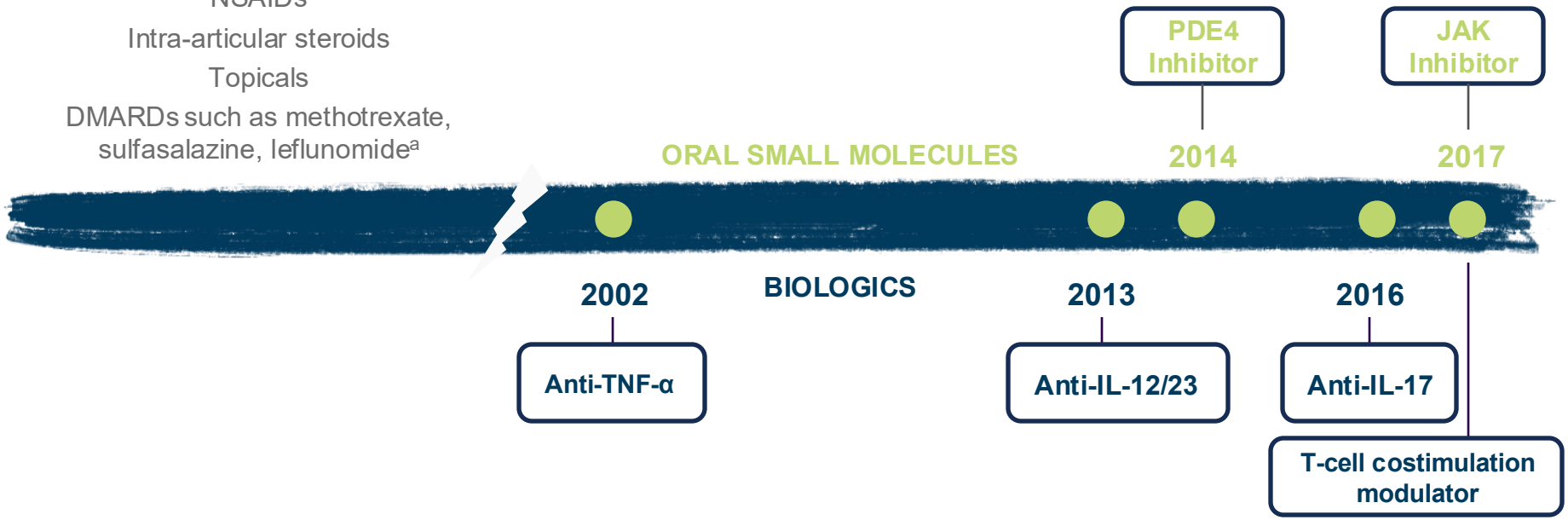
NSAIDs

Intra-articular steroids

Topicals

DMARDs such as methotrexate, sulfasalazine, leflunomide[a]

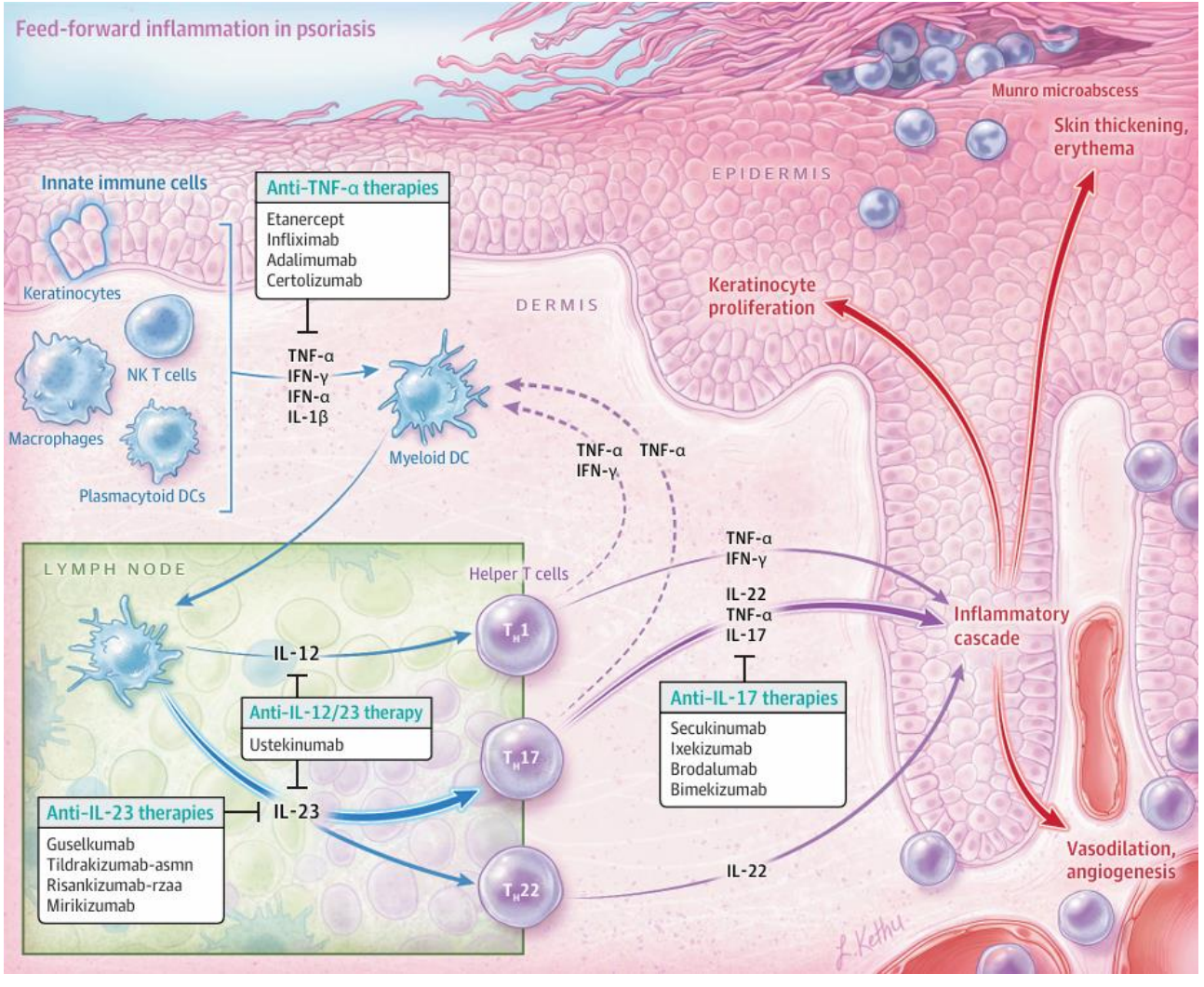
Timeline of FDA-Approved Treatments Indicated for Psoriatic Arthritis[4-5]



[a]Methotrexate, sulfasalazine, and leflunomide are not US Food and Drug Administration (FDA)-approved for the treatment of psoriatic arthritis.
DMARD = disease-modifying anti-rheumatic drug; PDE4 = phosphodiesterase 4; JAK = Janus kinase.

1. Gottlieb A, et al. *J Am Acad Dermatol.* 2008;58(5):851-864. 2. Coates LC, et al. *Arthritis Rheumatol.* 2016;68(5):1060-1071. 3. Mease PJ. *Clin Exp Rheumatol.* 2015;33(5 Suppl 93):S104-S108. 4. Merola JF, et al. *RMD Open.* 2018;4(2):e000656. 5. National Psoriasis Foundation [www.psoriasis.org]. Accessed August 15, 2018. https://www.psoriasis.org/treatments-for-psoriatic-disease/?page=1.

Pathogenesis of Psoriasis



Armstrong AW, Read C. *JAMA*. 2020;323(19):1945-1960.

JAK/STAT Signaling in PsA

- OSM induces pSTAT3 expression in PsAFLS
- OSM-induced secretion of MCP-1 and IL-6 was inhibited by all JAK inhibitors (JAKi)
- In contrast, JAKi had no significant impact on IL-8 expression in response to OSM
- PsAFLS cell invasion; migratory capacity; and MMP1, 3, and 9 were suppressed following JAKi treatment
- These functional effects were accompanied by a change in the cellular bioenergetic profile of PsAFLS, where JAKi significantly decreased glycolysis and the ECAR/OCR, resulting in a shift to a more quiescent phenotype
- JAK/STAT signaling mediates the complex interplay between inflammation and cellular metabolism in PsA pathogenesis
- This inhibition shows effective suppression of inflammatory mechanisms that drive pathogenic functions of PsAFLS, further supporting the role of JAKi as a therapeutic target for the treatment of PsA

OSM = oncostatin M; pSTAT = phosphorylated STAT; PsAFLS = primary PsA synovial fibroblasts; ECAR/OCR = extracellular acidification rate/oxygen consumption rate.

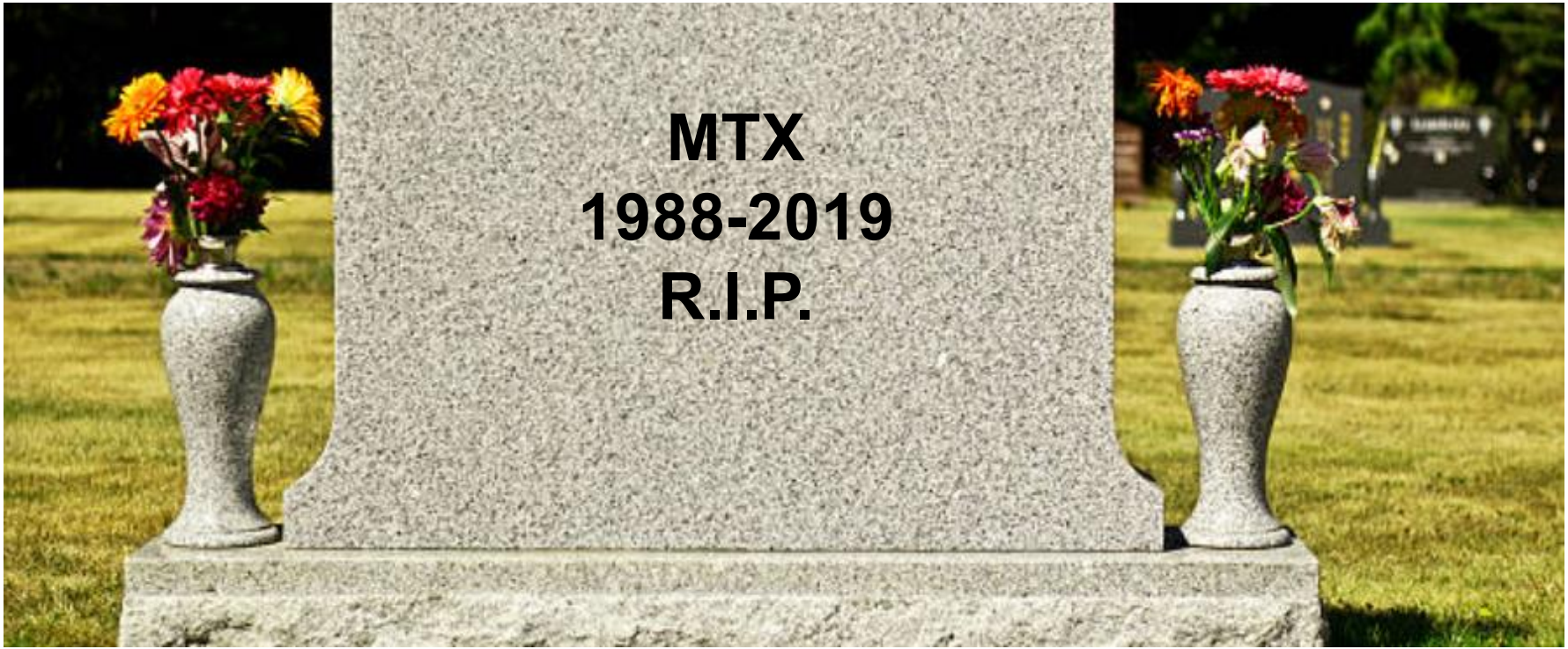
O'Brien A, et al. *Front Immunol.* 2021;12:672461.

Currently Approved Biologics and Small Molecules for PsA

- Abatacept
- Adalimumab
- Apremilast
- Bimekizumab
- Brodalumab (PsO only)
- Certolizumab
- Deucravacitinib (PsO only)
- Etanercept
- Golimumab
- Guselkumab
- Infliximab
- Ixekizumab
- Risankizumab
- Secukinumab
- Tildrakizumab (PsO only)
- Tofacitinib
- Upadacitinib
- Ustekinumab

PsO = psoriasis.

Is It Time to Stop Using MTX?



MTX Update

Methotrexate can cause serious side effects that can lead to death, including:

Organ system toxicity. People who use methotrexate for the treatment of cancer, psoriasis, or rheumatoid arthritis have an increased risk of death from organ toxicity. Types of organ toxicity can include: gastrointestinal, nerve, bone marrow, lung, liver, kidneys, immune system, and skin.

Your doctor will do blood tests and other types of tests before you take and while you are taking methotrexate to check for signs and symptoms of organ toxicity. Call your doctor right away if you have any of the following symptoms of organ toxicity: vomiting, neck stiffness, diarrhea, paralysis, mouth sores, irritability, fever, sleepiness, confusion, problems with coordination, weakness, dry cough, temporary blindness, trouble breathing, seizures, severe skin rash, headache, and back pain.

Women who are pregnant are at increased risk for death of the baby and for birth defects in the baby. Women who are pregnant or who plan to become pregnant must not take methotrexate. A pregnancy test should be performed before starting methotrexate. Contraception should be used by both females and males while taking methotrexate. Pregnancy should be avoided if either partner is receiving methotrexate

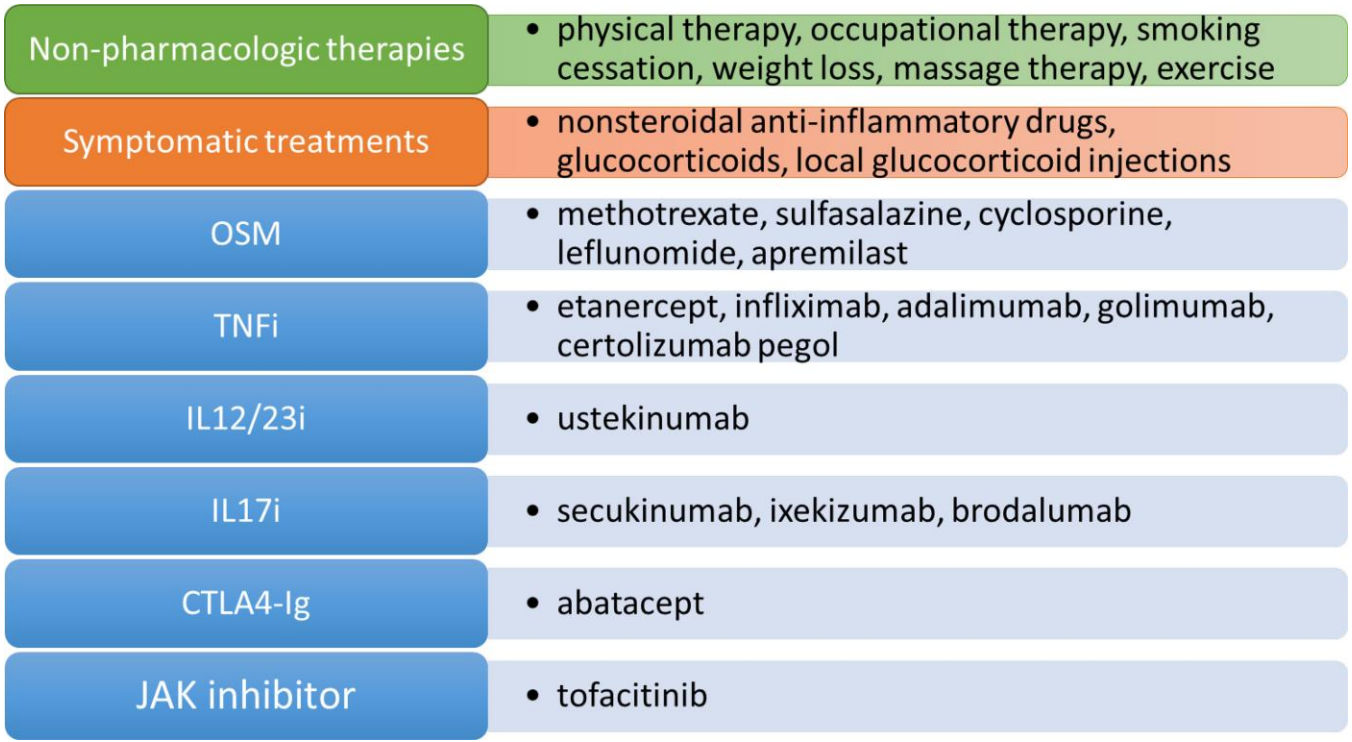
- For a minimum of 3 months after treatment with methotrexate for males
- During and for at least 1 menstrual cycle after treatment with methotrexate for females

What are the possible side effects of methotrexate?

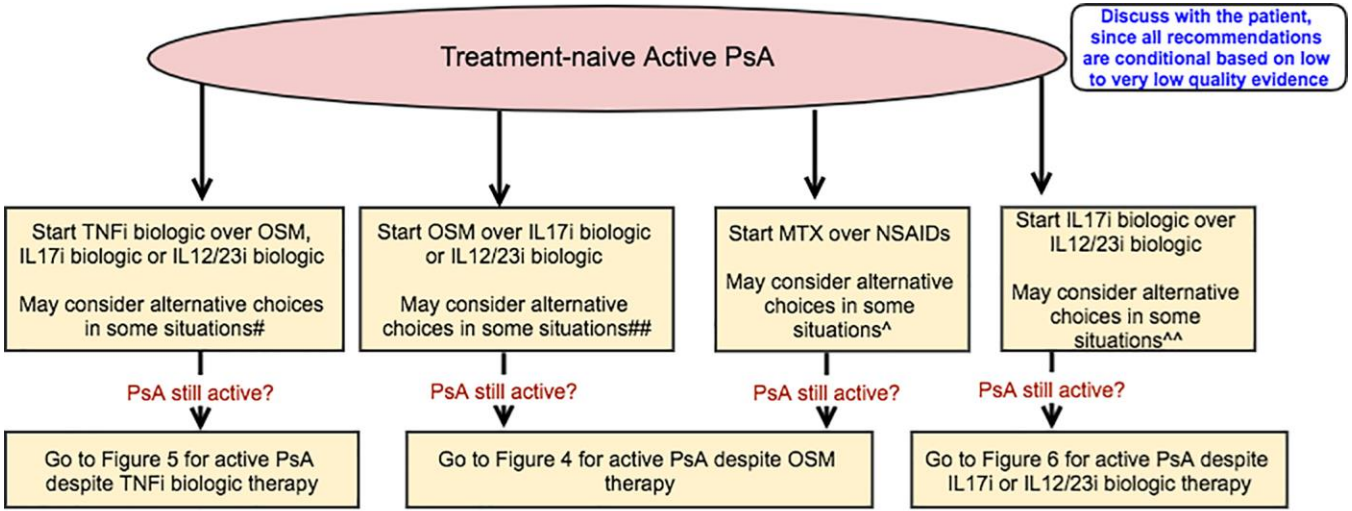
Methotrexate may cause serious side effects, including

- **Fertility problems.** Methotrexate may affect your ability to have a baby. Males may have a decreased sperm count, and females may have changes to their menstrual cycle. This can happen while taking methotrexate and for a short period of time after you stop.
- **Certain cancers.** Some people who have taken methotrexate have had a certain type of cancer called non-Hodgkin lymphoma and other tumors. Your doctor may tell you to stop taking methotrexate if this happens.
- **Tissue and bone problems.** Taking methotrexate while having radiation therapy may increase the risk of your tissue or bone not receiving enough blood. This may lead to death of the tissue or bone.

Drugs@FDA: FDA-Approved Drugs. Last updated March 11, 2020. Accessed February 17, 2026. https://www.accessdata.fda.gov/drugsatfda_docs/label/2020/205776s004lbl.pdf.



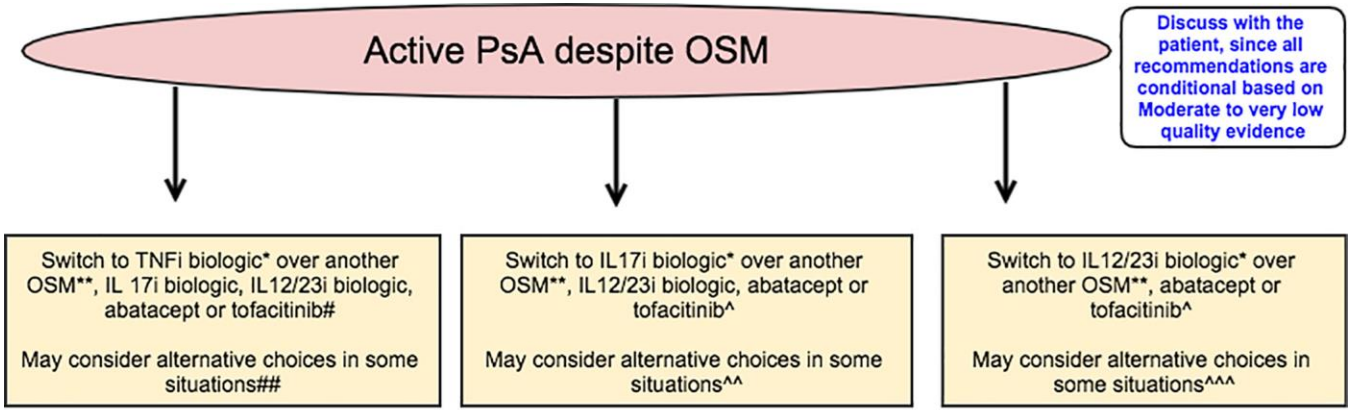
OSM = oral small molecule disease-modifying antirheumatic drug; TNFi = TNF inhibitor.
Singh JA, et al. *Arthritis Care Res (Hoboken).* 2019;71(1):2-29.



May consider alternatives (indicated in parentheses), if patient has severe psoriasis (IL17i or IL12/23i biologic); has contraindications to TNFi biologic including recurrent infections, congestive heart failure, or demyelinating disease (OSM, IL17i biologic, or IL12/23i biologic); prefers oral medications (OSM) or less frequent administrations (IL12/23i biologic); has concern over starting biologic as the first therapy (OSM); or does not have severe psoriasis or severe PsA (OSM).
May consider alternatives (indicated in parentheses), if patient has severe psoriasis or severe PsA (IL12/23i biologic or IL17i biologic); has concomitant active IBD (IL12/23i biologic); or prefers less frequent administrations (IL12/23i biologic).
^ May consider NSAIDs in patients with less active disease, after careful consideration of cardiovascular risks and renal risks of NSAIDs.
^^ May consider IL12/23i biologic if patient has concomitant IBD or desires less frequent drug administration.
The order of listing of various conditional recommendations or of different treatment choices within a conditional statement does not indicate any sequence in which treatment options would be chosen; each conditional statement stands on its own.

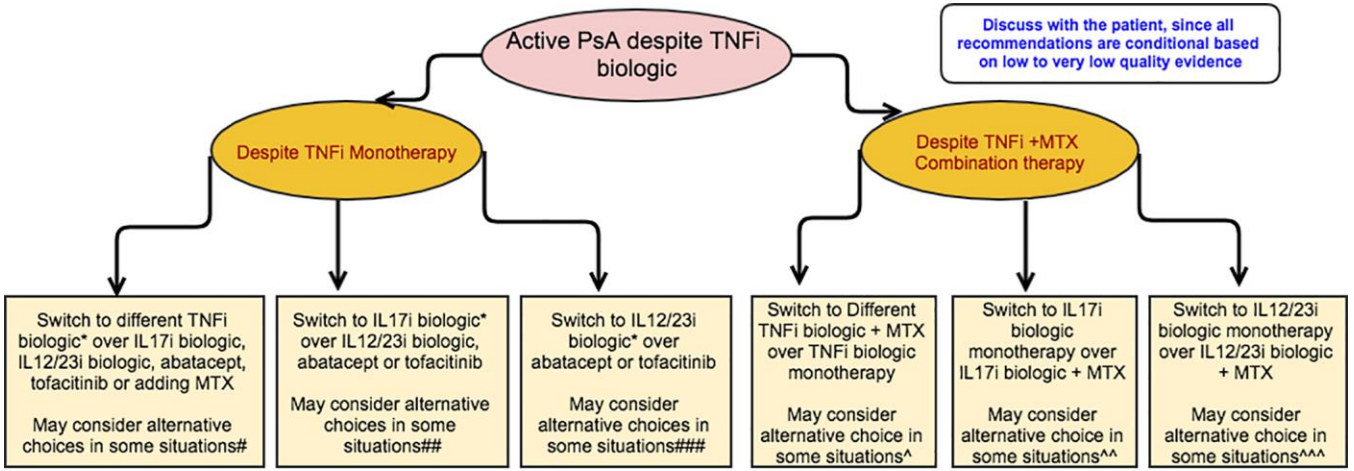
IBD = inflammatory bowel disease.

Singh JA, et al. *Arthritis Care Res (Hoboken)*. 2019;71(1):2-29.



* For each biologic, biologic monotherapy is conditionally recommended over biologic + MTX combination therapy.
** Add apremilast over switching to apremilast; Switch to another OSM (except apremilast) over adding another OSM
# Please see Figure 5 for details and treatment options if patient has active PsA despite TNFi biologic.
^ please see Figure 6 for details and treatment options if patient has active PsA despite IL17i or IL12/23i biologic
May consider alternatives (indicated in parentheses), if patient has severe psoriasis (IL17i or IL12/23i biologic); has contraindications to TNFi including recurrent infections, congestive heart failure, or demyelinating disease (OSM, IL17i biologic, IL12/23i biologic, abatacept or tofacitinib); prefers oral medications (OSM, tofacitinib) or less frequent administrations (IL12/23i biologic).
^^ May consider alternatives (indicated in parentheses), if patient has concomitant active IBD (IL12/23i biologic); absence of severe psoriasis or PsA (OSM); has recurrent serious infections (abatacept); has recurrent candida infections (tofacitinib); prefers oral medications (OSM, tofacitinib) or less frequent administrations (IL12/23i biologic).
^^^ May consider alternatives (indicated in parentheses), if patient has absence of severe psoriasis or severe PsA (OSM); has recurrent or serious infections (abatacept); prefers oral medications (OSM, tofacitinib).
The order of listing of various conditional recommendations or of different treatment choices within a conditional statement does not indicate any sequence in which treatment options would be chosen; each conditional statement stands on its own.

Singh JA, et al. *Arthritis Care Res (Hoboken).* 2019;71(1):2-29.



* For each biologic, biologic monotherapy is conditionally recommended over biologic + MTX combination therapy.

May consider alternatives, if patient has primary TNFi biologic efficacy failure (IL17i biologic, IL12/23i biologic, abatacept, tofacitinib); has TNFi biologic-associated serious adverse event (IL17i biologic, IL12/23i biologic, abatacept, tofacitinib); patients have demonstrated partial response to the current TNFi biologic therapy, especially if the TNFi biologic is a monoclonal antibody (adding MTX); prefers an oral therapy (tofacitinib); has severe psoriasis (IL17i); or prefers patient prefers less frequent drug administration (IL12/23i).

May consider alternatives (indicated in parentheses), if the patient has inflammatory bowel disease (IL12/23i biologic, tofacitinib); prefers IV dosing (abatacept); has recurrent or serious infections (abatacept); prefers an oral therapy (tofacitinib); a history of recurrent candida infections (tofacitinib); or prefers patient prefers less frequent drug administration (IL12/23i).

May consider alternatives (indicated in parentheses), if patient prefers IV dosing (abatacept); has had recurrent or serious infections (abatacept); or prefers oral therapy (tofacitinib).

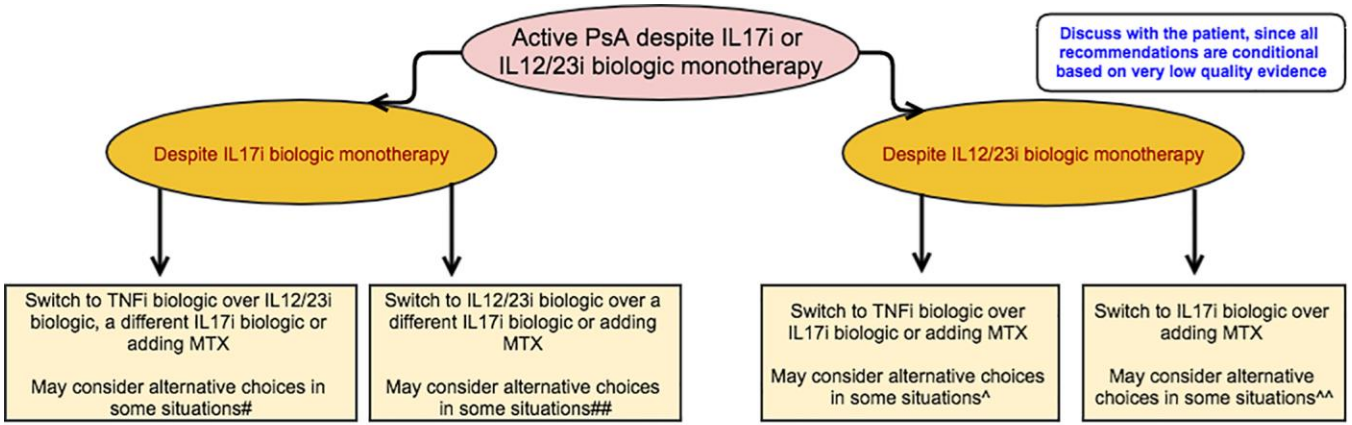
^ May consider the alternative, TNFi biologic monotherapy, if patient has demonstrated MTX-associated adverse events, prefers fewer medications or perceives MTX as a burden.

^^ May consider the alternative, IL17i biologic + MTX, if patient had had a partial response to the existing regimen or in patients with concomitant uveitis, as uveitis may respond to MTX therapy. Continuing MTX during the transition to an IL17i biologic was discussed as potentially beneficial to allow the new therapy time to work.

^^^ May consider the alternative, IL12/23i biologic + MTX, if patient had had a partial response to the existing regimen or in patients with concomitant uveitis, as uveitis may respond to MTX therapy. Continuing MTX during the transition to an IL12/23i biologic was discussed as potentially beneficial to allow the new therapy time to work.

The order of listing of various conditional recommendations or of different treatment choices within a conditional statement does not indicate any sequence in which treatment options would be chosen; each conditional statement stands on its own.

Singh JA, et al. *Arthritis Care Res (Hoboken).* 2019;71(1):2-29.



May consider alternatives (indicated in parentheses), if patient has contraindications to TNFi biologic including recurrent infections, congestive heart failure, or demyelinating disease (switching to IL12/23i biologic, or switching to a different IL17i biologic or adding MTX to the current regimen); if the patient had had a secondary efficacy failure (initial response, but lack of response/efficacy with continued use) to the current IL17i (different IL17i biologic); severe psoriasis (different IL17i biologic); if the patient had had a partial response to the existing regimen (adding MTX to the current regimen); or prefers less frequent administrations (IL12/23i biologic).

May consider alternatives (indicated in parentheses), if the patient had had a secondary efficacy failure to current IL17i (different IL17i biologic); severe psoriasis (different IL17i biologic); or if the patient had had a partial response to the existing regimen (adding MTX to the current regimen).

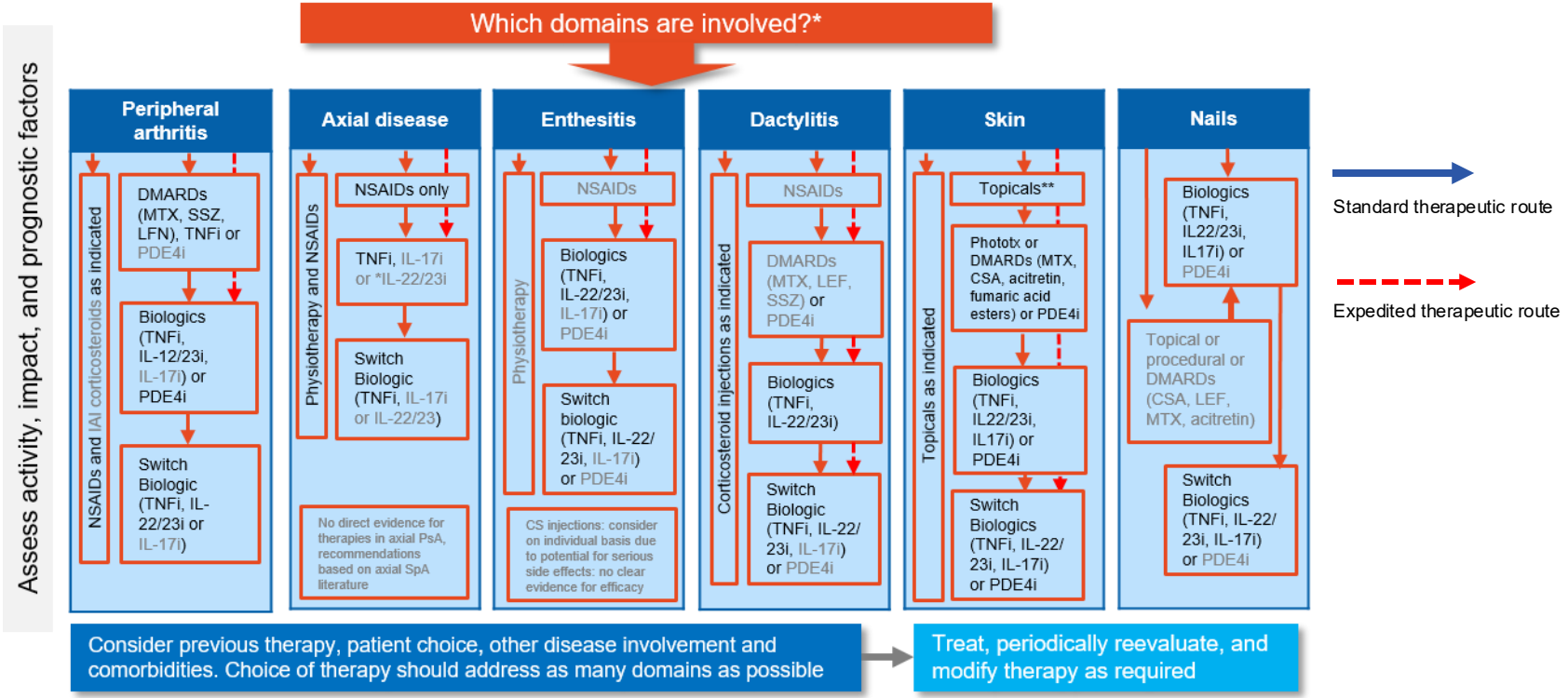
^ May consider alternatives (indicated in parentheses), if the patient had had contraindications to TNFi biologic including recurrent infections, congestive heart failure, or demyelinating disease (switching to IL17i biologic or adding MTX to the current regimen); severe psoriasis not responding to the current therapy (switching to IL17i biologic or adding MTX to the current regimen);

^^ May consider adding MTX in patients with only partial response to the current therapy or in those who potentially have not had enough time to adequately respond.

The order of listing of various conditional recommendations or of different treatment choices within a conditional statement does not indicate any sequence in which treatment options would be chosen; each conditional statement stands on its own.

Singh JA, et al. *Arthritis Care Res (Hoboken).* 2019;71(1):2-29.

Treatment Recommendations for Clinical Manifestations of PsA (GRAPPA)



***Gray text identifies conditional recommendations for drugs without current regulatory approvals or where recommendations arebased on abstract data only. This diagram was created in 2015 prior to the approval of secukinumab. **Topicals: keratolytics, steroids, vitamin D analogs, emollients, calcineurin inhibitors.**
CSA = cyclosporine A; GRAPPA = Group for Research and Assessment of Psoriasis and Psoriatic Arthritis; IAI = intra-articular injection; LEF = leflunomide; phototx = phototherapy; SSZ = sulfasalazine.
Coates LC, et al. *Arthritis Rheumatol.* 2016;68(5):1060-1071.

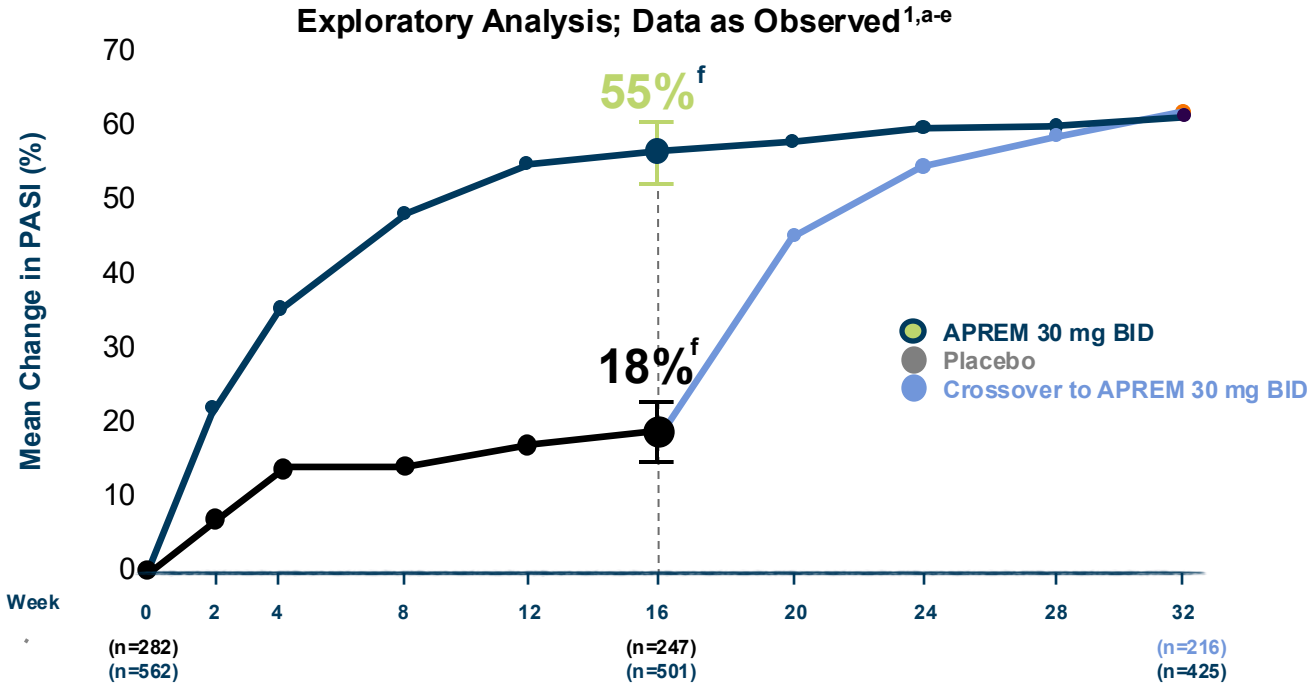
Medications Currently Used in the Treatment of PsA and RA: Small Molecules

Agent	Time to Benefit	Potential for Toxicity	Dose	Toxicities to Monitor
MTX	1-2 mo	Moderate	7.5-25 mg/week (+ folic acid)	Myelosuppression, hepatic fibrosis and cirrhosis, pulmonary infiltrates, infection, stomatitis, alopecia, fatigue, malaise
HCQ	2-6 mo	Low	200-400 mg/day	Macular damage, rash, anemia, GI
LEF	4-12 wk	Moderate	10-20 mg/day	Hepatotoxicity, diarrhea, alopecia, rash, headache, pulmonary infiltrates, infection
SSZ	1-3 mo	Low	500-1500 mg BID	Myelosuppression, infection
Cyclosporine	4-8 wk	High	1-3 mg/kg	Renal insufficiency, anemia, hypertension
Gold, parenteral	3-6 mo	Moderate	50 mg q week to 1000 mg then 50 mg q month	Myelosuppression, proteinuria, rash, stomatitis
Azathioprine	2-3 mo	Moderate	2 mg/kg/day	Myelosuppression, hepatotoxicity, lymphoproliferative disorders, infection

HCQ = hydroxychloroquine; GI = gastrointestinal.
ACR Subcommittee on Rheumatoid Arthritis Guidelines. *Arthritis Rheum*. 2002;46(2):328-346.

Apremilast in PsO: Mean Change in PASI Scores

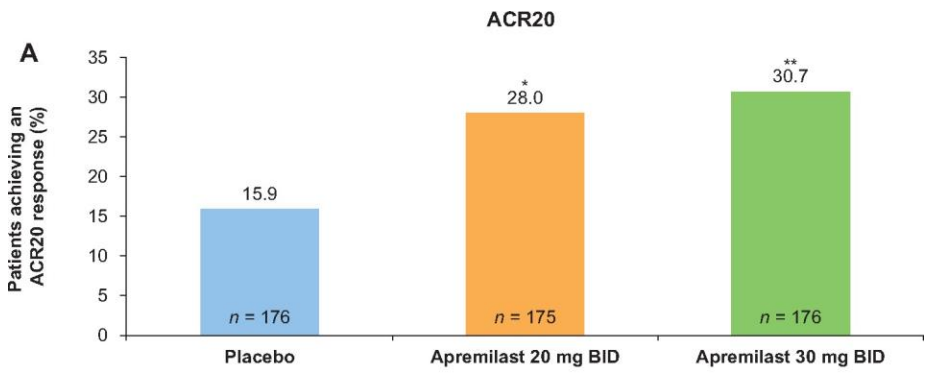
ESTEEM 1: Change in PASI Score



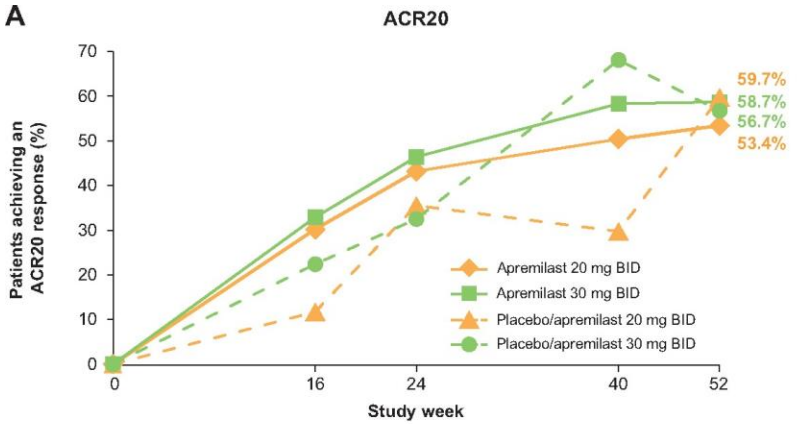
[a]Results were consistent between ESTEEM 1 and ESTEEM 2[1-3]. [b]Week 16: secondary endpoint; all other time points: exploratory endpoints. [c]Baseline mean PASI scores: placebo, 19; apremilast, 19; total, 19. [d]At week 16, patients receiving placebo were switched to apremilast. [e]Causes of patient dropout include adverse reactions, lack of efficacy, and patient withdrawal. [f]95% confidence interval.
PASI = Psoriasis Area and Severity Index.
1. Adapted with permission from Papp K, et al. *J Am Acad Dermatol*. 2015;73(1):37-49. 2. Paul C, et al. *Br J Dermatol*. 2015;173(6):1387-1399.

Apremilast Joint Response

PALACE 4: ACR20 Response (Primary Endpoint) at 16 Weeks
NRI, FAS Population



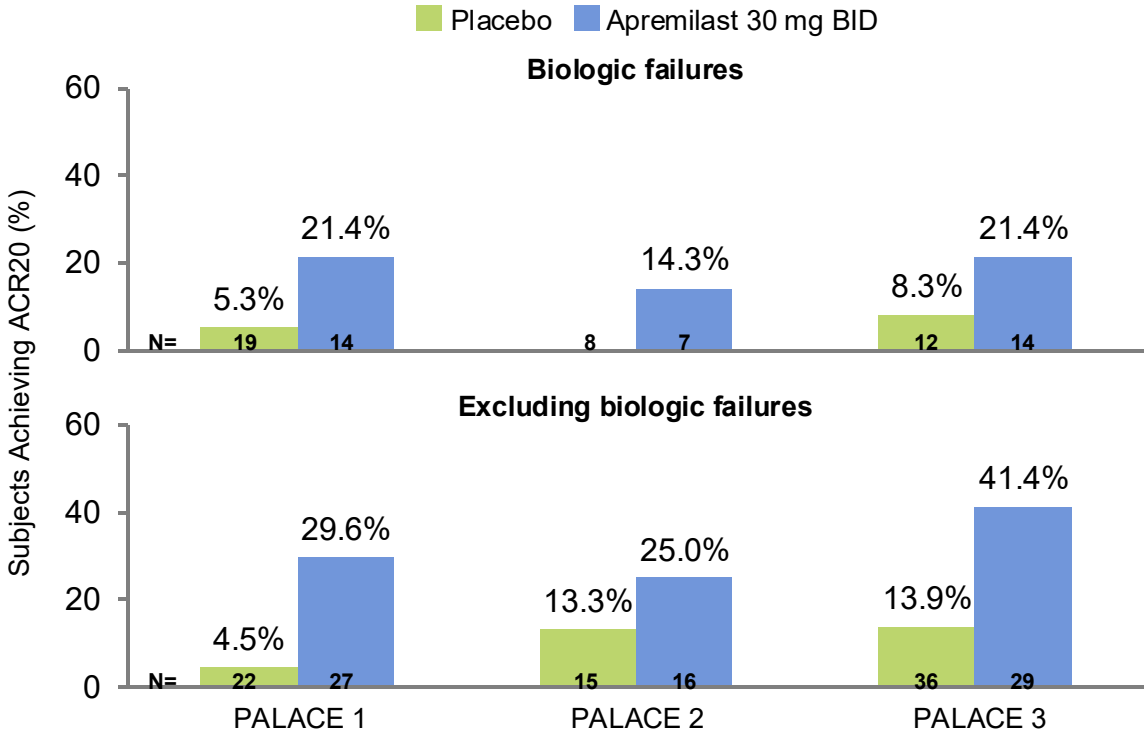
PALACE 4: ACR20 Response Rates
over 52 Weeks (Data as Observed)



Apremilast 20 mg BID, *n/N*	49/162	67/155	66/131	70/131
Apremilast 30 mg BID, *n/N*	54/164	71/153	84/144	81/138
Placebo/apremilast 20 mg BID, *n/N*	9/77	27/76	19/64	37/62
Placebo/apremilast 30 mg BID, *n/N*	17/76	25/77	47/69	38/67

[a]Includes all patients exposed to apremilast from baseline who completed treatment through week 156; [b]Data are presented "as observed" with no imputation for missing values in order to describe outcomes among those patients who continued to receive treatment over 156 weeks;[c]Patients discontinued treatment during the study due to adverse events, lack of efficacy, and other (withdrawal by patient, loss of follow-up, protocol violation, non-compliance, and other); [d]n/N, number of responders/number of subjects who had sufficient data for a definitive determination of response status at the time point, which includes subjects who discontinued early between the preceding time point and the specific time point.
NRI = nonresponder imputation; FAS = full analysis set.
1. Wells AF, et al. *Rheumatology (Oxford)*. 2018;57(7):1253-1263.

PALACE 1-3: ACR20 Response at Week 16 by Prior Biologic Use



NRI, FAS population.

Nash P, et al. *Ann Rheum Dis*. 2018;77(5):690-698. Wells AF, et al. *Rheumatology (Oxford)*. 2018;57(7):1253-1263. Kavanaugh A, et al. *Arthritis Res Ther.* 2019;21(1):118.

POETYK PsA-1: Deucravacitinib

Table 1. Efficacy endpoints at W16

Endpoint at W16		Placebo (n = 334)	Deucravacitinib 6 mg QD (n = 336)	Δ (95% CI) *P* value
Composite clinical efficacy *(prespecified efficacy analyses)*	ACR 20 response, %	34.1	54.2	20.0 (12.7, 27.4) < 0.0001
	ACR 50 response, %	13.5	24.7	11.2 (5.3, 17.1) 0.0002
	ACR 70 response, %	5.4	11.6	6.2 (2.0, 10.4) 0.0039
	PASI 75 response, %[a]	7.1	51.9	44.1 (35.4, 52.7) < 0.0001
	MDA response, %	10.2	19.0	8.9 (3.6, 14.2) 0.0012
	DAS28-CRP score, mean CfB	−0.83	−1.33	−0.51 (−0.67, −0.34) < 0.0001
Patient-reported outcomes *(prespecified efficacy analyses)*	HAQ-DI score, mean CfB	−0.22	−0.39	−0.17 (−0.24, −0.09) < 0.0001
	SF-36 PCS score, mean CfB	3.71	6.06	2.34 (1.28, 3.41) < 0.0001
	FACIT-Fatigue score, mean CfB	2.0	4.6	2.6 (1.4, 3.9) < 0.0001
Extra-articular manifestations of PsA (pooled POETYK PsA-1 and PsA-2 analyses) *(prespecified efficacy analyses)*	Pooled LEI enthesitis resolution, %[b]	45.1	50.3	5.3 (2.4, 12.9) 0.1781
	Pooled SPARCC enthesitis resolution, %[c]	36.1	47.1	10.8 (4.1, 17.6) 0.0018
	Pooled dactylitis resolution, %[d]	44.1	57.6	12.8 (3.2, 22.4) 0.0100
Radiographic analyses	mSvdH score, *(prespecified efficacy analysis)*[e] n, mean CfB	251, 0.64	245, 0.78	0.14 (0.456) 0.7597
	mSvdH score (with windowing)[e] n, mean CfB	251, 0.50	245, 0.41	NE 0.0187
	mSvdH score (without windowing)[f] n, mean CfB	305, 0.60	306, 0.37	NE 0.0090
	Nonprogressors (with windowing), n/N (%)[e,g]	183/251 (72.9)	201/245 (82.0)	8.8 (1.7, 16.0) 0.0166
	Nonprogressors (without windowing), n/N (%)[f,g]	218/305 (71.5)	251/306 (82.0)	10.2 (3.6, 16.7) 0.0025

All efficacy analyses were conducted in the randomized population unless otherwise stated. If a patient discontinued treatment prior to W16, the composite outcome was set to nonresponder. The multiplicity-controlled hierarchy was as follows: ACR 20, HAQ-DI, PASI 75, SF-36 PCS, MDA, LEI (pooled), mSvdH, FACIT-Fatigue, dactylitis (pooled), and DAS28-CRP. For multiplicity-controlled endpoints, a Cochran-Mantel-Haenszel test stratified by screening hsCRP level (< 10 mg/mL or ≥ 10 mg/mL) and csDMARD use at baseline (yes/no) was used for binary variables to compare the response rates with deucravacitinib 6 mg QD to those with placebo. The Clopper-Pearson estimation method was used to estimate CI. Nonresponder imputation was used to handle missing data for binary endpoints. ANCOVA was used to analyze continuous variables, with treatment, randomization stratification variables, and baseline value of the endpoint as the independent variable and CfB as the dependent variable. The (1) adjusted mean CfB (LS means) with SE and 95% CI per treatment group and (2) the difference between deucravacitinib 6 mg QD to placebo in adjusted mean CfB with SE and 95% CI were provided from each analysis model. *P* values were based on the Wald test. Control-based pattern imputation was used to handle missing data for continuous endpoints. Italics indicate nominal *P* values. Additional prespecified efficacy analyses were descriptive.

[a]Assessed in all randomized patients with ≥ 3% BSA and a sPGA score of ≥ 2 at baseline (deucravacitinib, n = 162; placebo, n = 170); [b]Assessed in pooled patients from POETYK PsA-1 and POETYK PsA-2 in patients with an LEI score of ≥ 1 at baseline (deucravacitinib, n = 318; placebo, n = 317); [c]Assessed in pooled patients from POETYK PsA-1 and POETYK PsA-2 in patients with a SPARCC score of ≥ 1 at baseline (deucravacitinib, n = 393; placebo, n = 407); [d]Assessed in pooled patients from POETYK PsA-1 and POETYK PsA-2 in patients with a tender dactylitis count of ≥ 1 at baseline (deucravacitinib, n = 210; placebo, n = 188); [e]Assessed in all randomized patients with W16 scans performed during the analysis window between days 100 to 127 and before the first dose in the active treatment period; data were imputed for patients with missing W16 scans or with out-of-window baseline (prior to day −38 or after day −1) or W16 scans; [f]Assessed in all randomized patients with available radiographic assessments (except 4 early terminations), with windowing rules removed; [g]Nonprogressors are defined as patients with a CfB in mSvdH total score of ≤ 0 from baseline.

ACR 20, American College of Rheumatology 20% improvement in response; ACR 50, American College of Rheumatology 50% improvement in response; ACR 70, American College of Rheumatology 70% improvement in response; ANCOVA, analysis of covariance; BSA, body surface area; CfB, change from baseline; conventional synthetic disease-modifying antirheumatic drug; DAS28-CRP, 28-joint Disease Activity Score–C-reactive protein; FACIT-Fatigue, Functional Assessment of Chronic Illness Therapy - Fatigue Scale; HAQ-DI, Health Assessment Questionnaire Disability Index; hsCRP, high-sensitivity C-reactive protein; LEI, Leeds Enthesitis Index; LS, least squares; MDA, minimal disease activity; mSvdH, modified Sharp-van der Heijde; NE, not estimable; PASI 75, ≥ 75% improvement in the Psoriasis Area and Severity Index; PCS, physical component summary; QD, once daily; SE, standard error; SF-36, 36-Item Short Form Survey; SPARCC, Spondyloarthritis Research Consortium of Canada; sPGA, static Physician Global Assessment; W, week.

Table 2. Safety summary at W16

Patients with an AE, n (%)	Placebo (n = 333)	Deucravacitinib 6 mg QD (n = 332)
Any AEs	160 (48.0)	200 (60.2)
Serious AEs	8 (2.4)	6 (1.8)
AEs leading to discontinuation	6 (1.8)	8 (2.4)
Deaths	0	0
Most frequent AEs (≥ 5% in any arm) by PT		
Upper respiratory tract infection	10 (3.0)	17 (5.1)

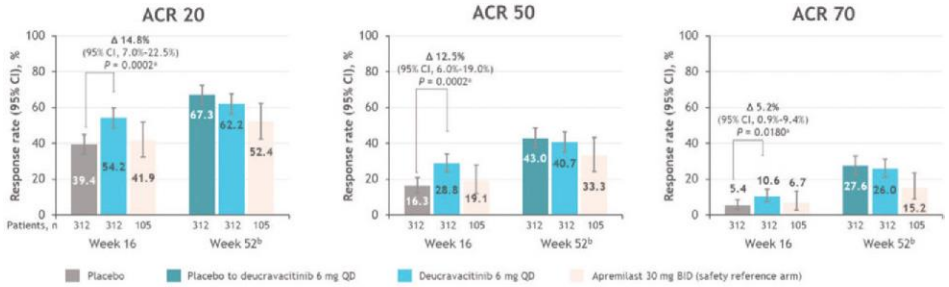
AEs are defined as treatment-emergent AEs with an onset date on or after the first dose of study treatment up to 30 days after the last dose date of treatment in the study. All infections not shown above, GI disorders, and skin disorders were reported in < 5% of patients in either arm.
AE, adverse event; GI, gastrointestinal; PT, preferred term; QD, once daily.

"Treatment with deucravacitinib, the first oral TYK2 inhibitor evaluated in phase 3 studies of active PsA, resulted in superior efficacy vs placebo at W16 across multiple endpoints"

Van der Heijde D, et al. *Ann Rheum Dis*. 2025;84(Suppl 1):313-314.

POETYK PsA-2

Figure. ACR 20/50/70 at weeks 16 and 52



No prespecified statistical comparisons were performed for the apremilast arm.

All randomized patients were assessed. Nonresponder imputation was used to handle missing data. Treatment discontinuations prior to week 16 were considered treatment failures (composite variable strategy). All rescue medication–related intercurrent events were treated with a treatment policy–estimand strategy. The Clopper-Pearson estimation method was used to estimate CI. [a]A Cochran-Mantel-Haenszel test stratified by TNF inhibitor (yes/no), screening hsCRP concentration (< 10 mg/L vs ≥ 10 mg/L), and csDMARD use at baseline (yes/no) was used to compare the response rates with deucravacitinib 6 mg QD to placebo; [b]Exploratory endpoint.
ACR 20, American College of Rheumatology 20% improvement in response; ACR 50, American College of Rheumatology 50% improvement in response; ACR 70, American College of Rheumatology 70% improvement in response; BID, twice daily; csDMARD, conventional synthetic disease-modifying antirheumatic drug; hsCRP, high-sensitivity C-reactive protein; QD, once daily; TNF, tumor necrosis factor.

Table. Most frequent AEs (≥ 5% in any arm) by preferred term: cumulative treatment period weeks 0-52[a]

Most frequent AEs (≥ 5% in any arm) by preferred term	Total deucravacitinib 6 mg QD[b] (n = 604)		Apremilast 30 mg BID (n = 105)	
	n (%)	IR/100 PYs	n (%)	IR/100 PYs
Total patients with an event	451 (74.7)	220.8	92 (87.6)	296.8
Nasopharyngitis	71 (11.8)	16.0	8 (7.6)	9.7
COVID-19	70 (11.6)	15.8	13 (12.4)	16.4
Upper respiratory tract infection	66 (10.9)	15.0	11 (10.5)	13.3
Diarrhea	27 (4.5)	5.8	20 (19.0)	25.6
Hypertension	23 (3.8)	5.0	7 (6.7)	8.8
Psoriatic arthropathy	23 (3.8)	4.9	7 (6.7)	8.2
Nausea	20 (3.3)	4.3	9 (8.6)	10.9
Headache	20 (3.3)	4.3	13 (12.4)	16.3
Urinary tract infection	16 (2.6)	3.4	7 (6.7)	8.3

PY is defined as exposure based on time to first onset. Incidence rate per 100 PYs of exposure (IR/100 PYs) is calculated as event count×100/PYs of exposure. MedDRA v27.1 was used to define preferred terms for AEs.
[a]AEs in the treated population are shown. AEs are treatment-emergent AEs with an onset on or after the first dose date of study treatment up to 30 days after the last treatment dose; [b]The total deucravacitinib group includes patients who received deucravacitinib from baseline through week 52 as well as patients who switched from placebo to deucravacitinib at week 16 and continued through week 52.
AE, adverse event; BID, twice daily; IR, incidence rate; MedDRA, Medical Dictionary for Regulatory Activities; PY, person-year; QD, once daily.

"Deucravacitinib, the first oral TYK2 inhibitor evaluated in a phase 3 PsA study, showed superior efficacy vs placebo across multiple endpoints at week 16, including musculoskeletal and dermatologic manifestations, overall disease activity measures, and quality of life in adults with active PsA."

Mease PJ, et al. *Ann Rheum Dis*. 2025;84(Suppl 1):84-85.

POETYK PsA-1 and PsA-2

Table 1. Efficacy endpoints at weeks 16 and 52

Endpoint	Week 16[a]		Week 52	
	Placebo (n = 334)	Deucravacitinib 6 mg QD (n = 336)	Placebo to deucravacitinib 6 mg QD (n = 334)	Deucravacitinib to deucravacitinib 6 mg QD (n = 336)
Disease activity measures (NRI)				
PASI 75 response (95% CI), %[b]	7.1 (3.7 to 12.0)	51.9 (43.9 to 59.8)	51.8 (44.0 to 59.5)	66.0 (58.2 to 73.3)
MDA response (95% CI), %	10.2 (7.2 to 13.9)	19.0 (15.0 to 23.7)	34.4 (29.3 to 39.8)	33.9 (28.9 to 39.3)
DAS28-CRP score, adjusted mean CfB (95% CI)	−0.83 (−0.95 to −0.70)	−1.33 (−1.46 to −1.21)	−2.05 (−2.18 to −1.92)	−2.11 (−2.24 to −1.98)
Patient-reported outcomes (NRI)				
HAQ-DI score, adjusted mean CfB (95% CI)	−0.22 (−0.27 to −0.16)	−0.39 (−0.44 to −0.33)	−0.49 (−0.56 to −0.42)	−0.51 (−0.58 to −0.44)
SF-36 PCS score, adjusted mean CfB (95% CI)	3.71 (2.91 to 4.51)	6.05 (5.24 to 6.85)	8.32 (7.39 to 9.26)	8.57 (7.64 to 9.50)
FACIT-Fatigue score, adjusted mean CfB (95% CI)	2.0 (1.1 to 2.9)	4.6 (3.7 to 5.6)	6.3 (5.3 to 7.3)	6.4 (5.4 to 7.5)
Extraarticular manifestations of PsA (pooled POETYK PsA-1 and POETYK PsA-2 analysis; NRI)				
Pooled LEI enthesitis resolution (95% CI), %[c]	45.1 (38.4 to 54.0)	50.3 (40.8 to 55.9)	59.9 (54.3 to 65.4)	58.8 (53.2 to 64.3)
Pooled SPARCC enthesitis resolution (95% CI),%[d]	36.1 (31.4 to 41.0)	47.1 (42.0 to 52.1)	56.5 (51.5 to 61.4)	54.2 (49.1 to 59.2)
Pooled dactylitis resolution (95% CI), %[e]	44.1 (34.0 to 53.4)	57.6 (50.2 to 67.6)	63.3 (56.0 to 70.2)	71.0 (64.3 to 77.0)
Radiographic progression (as observed, post hoc analysis)				
Nonprogressors (95% CI), %[f]	71.5 (66.1 to 76.5)[g]	82.0 (77.3 to 86.2)[g]	66.5 (60.5 to 72.2)[h]	73.3 (67.6 to 78. 4)[h]

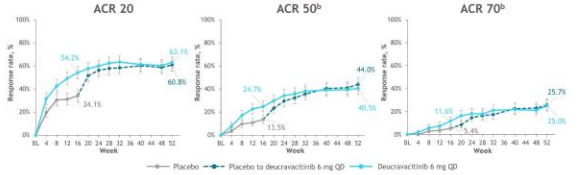
A Cochran-Mantel-Haenszel test stratified by screening hsCRP concentration (< 10 mg/L vs ≥ 10 mg/L) and csDMARD use at baseline (yes/no) was used to compare the response rates of deucravacitinib 6 mg QD vs placebo. Nonresponder imputation was used for missing data for binary variables. CfB was analyzed using ANCOVA with treatment, randomization stratification variables, and baseline value as independent variables. Control-based pattern multiple imputation was used for missing data for continuous variables.

[a]Patients were randomized 1:1 to receive placebo or deucravacitinib 6 mg QD through W16. At W16, patients initially randomized to receive placebo were switched to deucravacitinib 6 mg QD through W52. Patients initially randomized to receive deucravacitinib 6 mg QD continued to receive deucravacitinib 6 mg QD through W52; [b]Assessed in patients with ≥ 3% BSA and an sPGA score of ≥ 2 at baseline (placebo, n = 170; deucravacitinib, n = 162); [c]Assessed in randomized patients with enthesitis by LEI at baseline (placebo to deucravacitinib, n = 317; deucravacitinib to deucravacitinib, n = 318); [d]Assessed in randomized patients with enthesitis by SPARCC at baseline (placebo to deucravacitinib, n = 407; deucravacitinib to deucravacitinib, n = 393); [e]Assessed in randomized patients with dactylitis at baseline (placebo to deucravacitinib, n = 188; deucravacitinib to deucravacitinib, n = 210); [f]Nonprogressors were patients who had a CfB to W16 or W52 in modified SvdH total score ≤ 0. Response rate 95% CI was based on the Clopper-Pearson exact method; [g]Assessed in randomized patients with available, observed SvdH total scores collected at baseline and W16 nominal visits (placebo, n = 305; deucravacitinib, n = 306); [h]Assessed in randomized patients with available, observed SvdH data collected at baseline and W52 nominal visits (placebo to deucravacitinib, n = 266; deucravacitinib to deucravacitinib, n = 273). The total number of patients is randomized patients with nonmissing baseline and W52 SvdH total scores based on purely observed data.

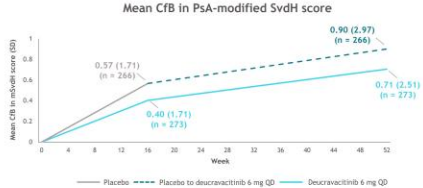
ANCOVA, analysis of covariance; BSA, body surface area; CfB, change from baseline; csDMARD, conventional disease-modifying antirheumatic drug; FACIT-Fatigue, Functional Assessment of Chronic Illness Therapy–Fatigue Scale; HAQ-DI, Health Assessment Questionnaire Disability Index; hsCRP, high sensitivity C-reactive profile; LEI, Leeds Enthesitis Index; MDA, minimal disease activity; NRI, nonresponder imputation; PASI 75, ≥ 75% improvement in the Psoriasis Area and Severity Index; PCS, physical component score; PsA, psoriatic arthritis; QD, once daily; SF-36, 36-Item Short Form Survey; SPARCC, Spondylarthritis Research Consortium of Canada; sPGA, static Physician Global Assessment; SvdH, Sharp-van der Heijde; W, week.

Figure 1. ACR 20/50/70 and radiographic progression analyses through week 52

A. ACR 20/50/70 kinetics (NRI)[a]



B. Radiographic progression: sustained inhibition of progression of structural damage (as observed, post hoc analysis)[c]



[a]All randomized patients were assessed. Treatment discontinuations prior to week 16 were considered treatment failures (composite variable strategy). All rescue medication–related intercurrent events were treated with a treatment policy–estimand strategy. The Clopper-Pearson exact method was used to estimate CI. Nonresponder imputation was used to handle missing data; [b]Additional secondary endpoint up to week 16 and exploratory endpoint up to week 52; [c]The population was based on randomized patients with available, observed SvdH total score, which was collected only at screening, W16, and W52 nominal visits. ACR 20, American College of Rheumatology 20% improvement in response; ACR 50, American College of Rheumatology 50% improvement in response; ACR 70, American College of Rheumatology 70% improvement in response; BL, baseline; CfB, change from baseline; mSvdH, modified Sharp-van der Heijde; NRI, nonresponder imputation; QD, once daily; SD, standard deviation; SvdH, Sharp-van der Heijde.

Table 2. Overall safety summary: cumulative treatment period (weeks 0-52)

Safety parameter	Placebo to deucravacitinib 6 mg QD (n = 306)		Deucravacitinib to deucravacitinib 6 mg QD (n = 322)	
	n (%)	IR/100 PY	n (%)	IR/100 PY
Any AEs	206 (67.3)	196.0	262 (78.9)	218.7
Treatment-related AEs	54 (17.6)	31.2	92 (27.7)	38.0
Serious AEs	17 (5.6)	9.0	26 (7.8)	8.9
AEs leading to discontinuation	10 (3.3)	5.2	18 (5.4)	6.0
Deaths	0	0	0	0
Most frequent AEs (≥ 5%) in any arm by PT				
Upper respiratory tract infection	33 (10.8)	18.0	46 (13.9)	16.6
Nasopharyngitis	26 (8.5)	14.2	30 (9.0)	10.6
Hypertension	14 (4.6)	7.4	26 (7.8)	9.1
COVID-19	9 (2.9)	4.7	24 (7.2)	8.4
Urinary tract infection	7 (2.3)	3.7	19 (5.7)	6.5

AEs are defined as treatment-emergent AEs with an onset date on or after the first dose date of study treatment up to 30 days after the last dose date of treatment in the study. MedDRA: 28.0.

AE, adverse event; COVID-19, coronavirus disease 2019; IR, incidence rate; MedDRA, Medical Dictionary for Regulatory Activities; PsA, psoriatic arthritis. PT, preferred term; PY, patient-year; QD, once daily.

"Deucravacitinib was efficacious in patients with PsA based on clinical responses, PROs, and inhibition of radiographic progression, which continued to improve after W16 and were durable through W52."

Van der Heijde D, et al. Presented at: ACR Convergence; 2025. Abstract LB20.

Comparison of Drugs in PsA

Comparative Efficacy of Small Molecules in PsA: Joint Response

	ACR20	ACR50	ACR70
Apremilast	38%	17%	4.5%
Methotrexate (not FDA approved)	19%	ND	ND
Tofacitinib	50%	28%	17%
Upadacitinib	57%	33%	9%

Phase 3 published trials. Drugs@FDA: FDA-Approved Drugs. Last updated December 23, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/205437s016lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated March 11, 2020. https://www.accessdata.fda.gov/drugsatfda_docs/label/2020/205776s004lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated October 16, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/203214s039,213082s011lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated October 10, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/211675Orig1s028,218347Orig1s005lbl.pdf.

Comparative Efficacy of Small Molecules in PsA: Skin Response

	PASI75	PASI90	PASI100
Apremilast	33%	ND	ND
Methotrexate	20%	ND	ND
Tofacitinib	43% (44%)	27% (30%)	ND
Upadacitinib	52%	35%	25%

Phase 3 published trials. Drugs@FDA: FDA-Approved Drugs. Last updated December 23, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/205437s016lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated March 11, 2020. https://www.accessdata.fda.gov/drugsatfda_docs/label/2020/205776s004lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated October 16, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/203214s039,213082s011lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated October 10, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/211675Orig1s028,218347Orig1s005lbl.pdf.

Comparative Efficacy of Biologic Agents in PsA: Joint Response (Week 12)

	ACR20	ACR50	ACR70
Anti-TNF			
Adalimumab	58%	36%	20%
Certolizumab	58%	36%	24%
Etanercept	59%	39%	20%
Golimumab	49% (74%)	25% (33%)	17% (33%)
Infliximab	51%	40%	32%
Anti-IL-12/23			
Ustekinumab	49%	27%	14%
Anti-IL-17			
Bimekizumab	65%	46%	25%
Secukinumab	60% (57%)	37% (35%)	17% (15%)
Ixekizumab	58%	40%	23%
Anti-CD80/CD86			
Abatacept	32%	19%	10%

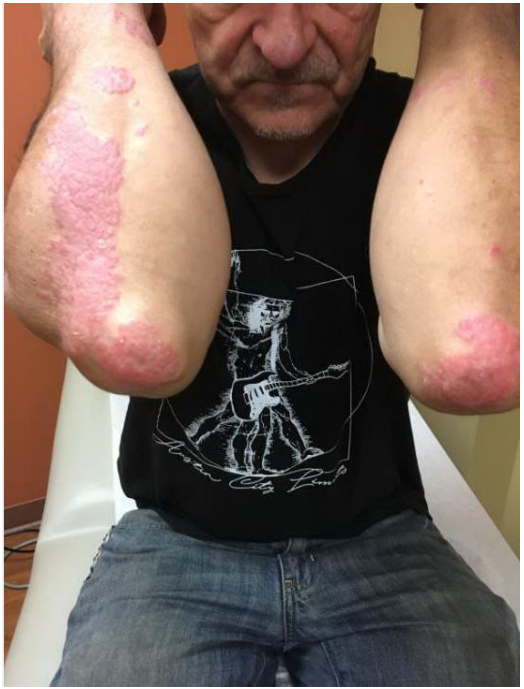
Phase 3 published trials. Drugs@FDA: FDA-Approved Drugs. Last updated December 23, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/205437s016lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated March 11, 2020. https://www.accessdata.fda.gov/drugsatfda_docs/label/2020/205776s004lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated October 16, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/203214s039,213082s011lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated October 10, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/211675Orig1s028,218347Orig1s005lbl.pdf.

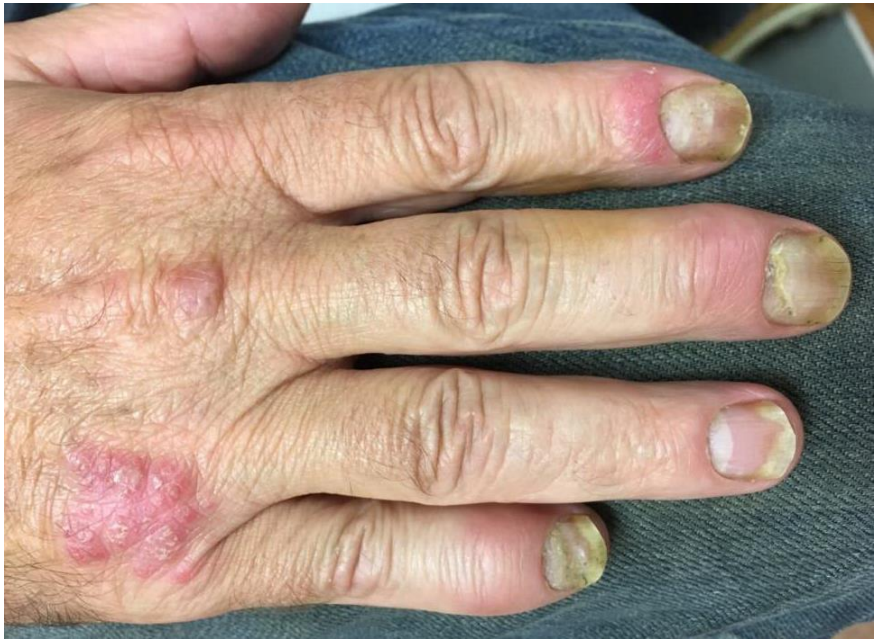
Comparative Efficacy of Biologic Agents in PsA: Skin Response (Week 12)

	PASI75	PASI90	PASI100
Anti-TNF			
Adalimumab	59%	42%	ND
Certolizumab	46%	22%	ND
Etanercept	23%	20%	ND
Golimumab	56% (63%)	30% (37%)	ND (15%)
Infliximab	54%	ND	ND
Anti-IL-12/23			
Ustekinumab	67%	48%	26%
Anti-IL-17			
Bimekizumab	82%	68%	58%
Secukinumab	66%	48%	33%*
Ixekizumab	75%	52%	32%
Anti-CD80/CD86			
Abatacept	20%	ND	ND

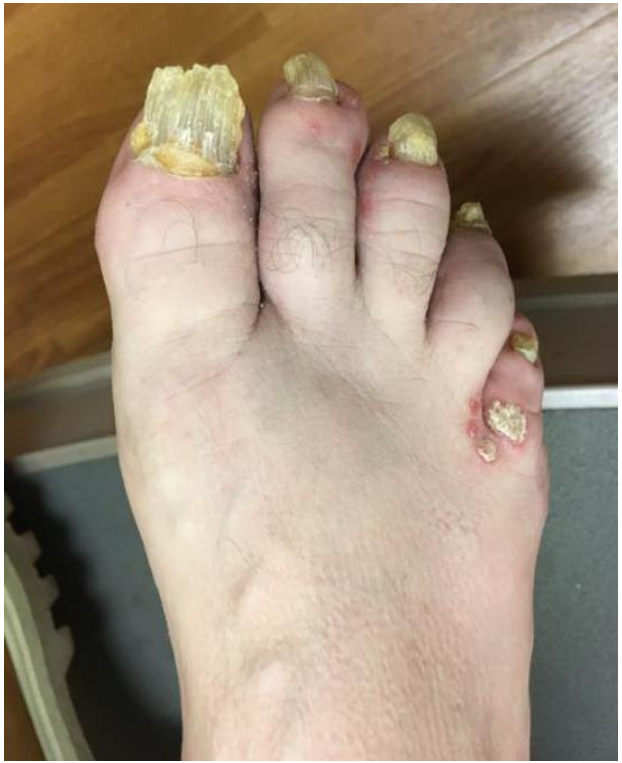
Phase 3 published trials. Drugs@FDA: FDA-Approved Drugs. Last updated December 23, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/205437s016lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated March 11, 2020. https://www.accessdata.fda.gov/drugsatfda_docs/label/2020/205776s004lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated October 16, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/203214s039,213082s011lbl.pdf. Drugs@FDA: FDA-Approved Drugs. Last updated October 10, 2025. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/211675Orig1s028,218347Orig1s005lbl.pdf.

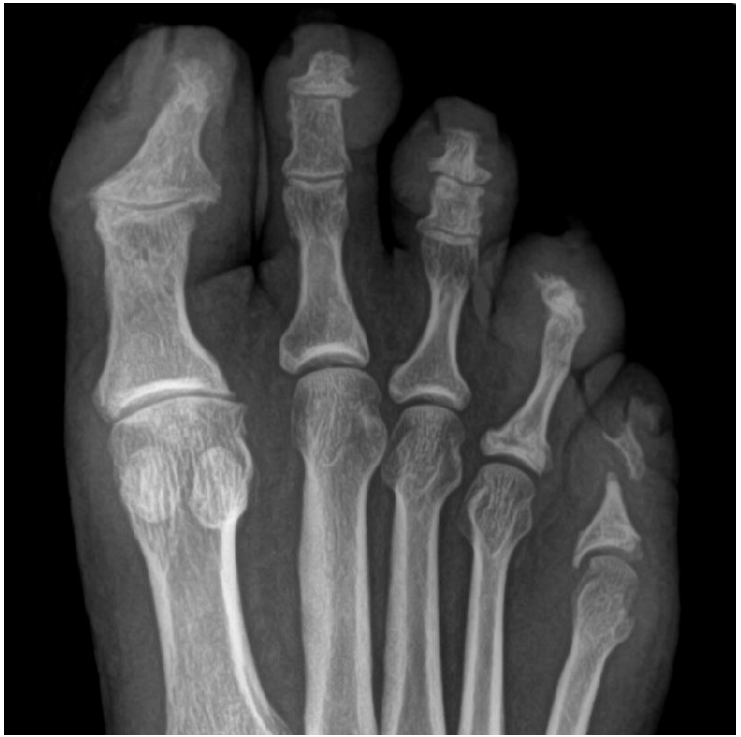
Features of PsA



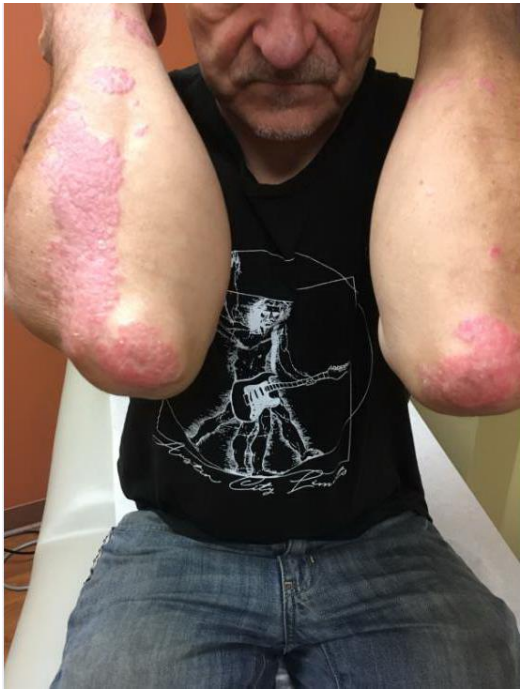


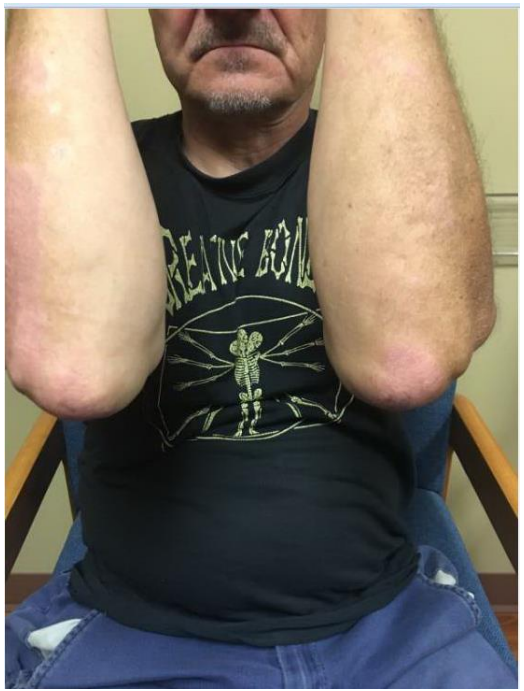
Features of PsA



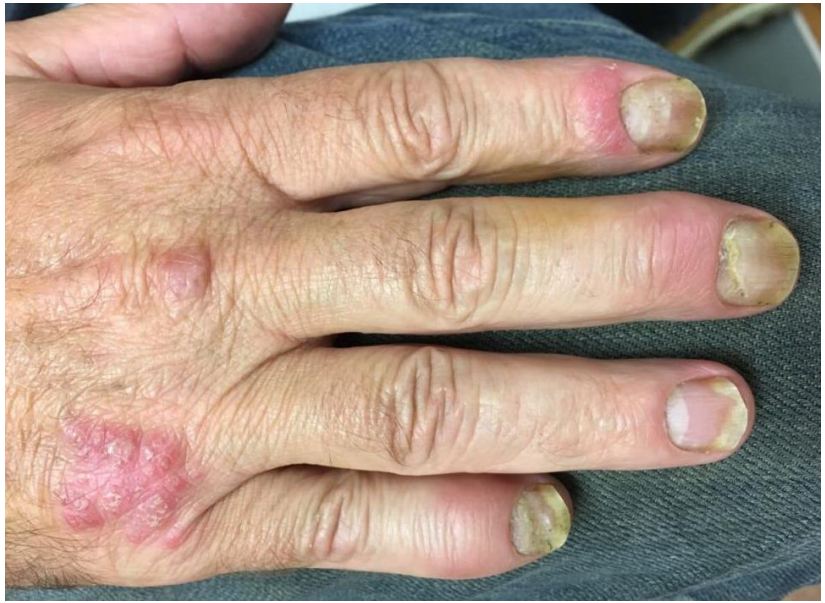


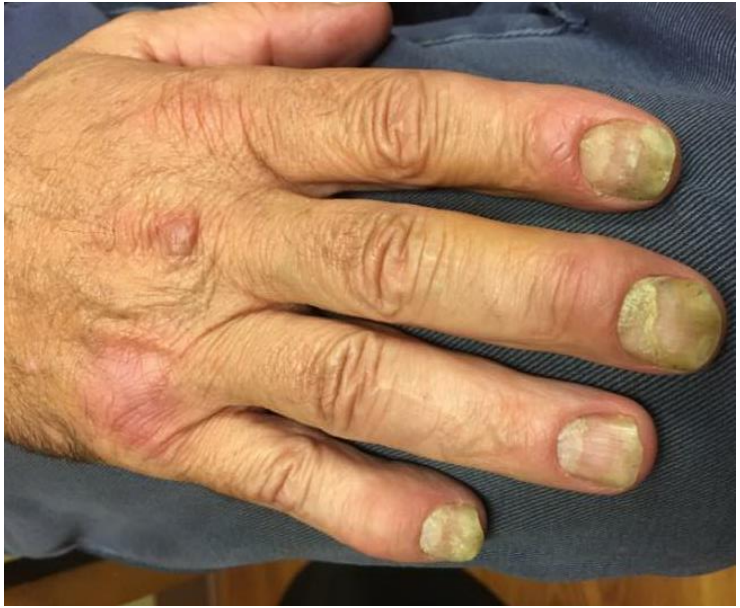
Features of PsA





Features of PsA





Key Learning Points

- PsA is becoming more common in rheumatology practices
- Limited use of screening tools, absence of definitive biomarkers, and variable clinical presentations in PsA contribute to delayed diagnosis and undertreatment
- JAK/STAT signaling mediates the complex interplay between inflammation and cellular metabolism in PsA pathogenesis
- Newer treatment options have allowed better clinical responses in most patients, including new oral therapies
- More data are needed on the impact of therapies on extra-articular manifestations of the diseases

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