Overview of Gout and Uncontrolled Gout



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Background and Epidemiology

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Gout is Caused by Elevated sUA Leading to MSU Crystal Deposition in Joints[1]



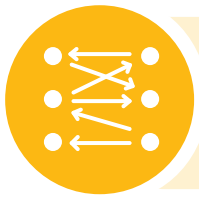
Gout is the most common form of inflammatory arthritis, with a global prevalence of 1–4%[2]



Symptoms include redness, swelling, and severe pain, typically developing rapidly[1]



Gout is caused by hyperuricemia (sUA levels ≥6.8 mg/dL), which can lead to deposition of MSU crystals in joints and tissues, with ~2–6% of patients with gout in the US classified as having uncontrolled gout[3,4]



Gout is associated with a range of both modifiable (e.g., diet and lifestyle) and non-modifiable (e.g., genetics and age) risk factors[5–8]

MSU, monosodium urate; sUA, serum uric acid.

1. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 2. Singh JA, Gaffo A. *Semin Arthritis Rheum* 2020;50(3S):S11–S16; 3. Girardet JL, Miner JN. *Annu Rep Med Chem* 2014;49:151–164;
4. Edwards NL. *Arthritis Rheum* 2008;58:2587–2590; 5. Asghari KM, et al. *BMC Musculoskelet Disord* 2024;25:1047; 6. Zgaga L, et al. *PLoS One* 2012;7:e38123;
7. Qin H, Hua Y. *BMC Public Health* 2024;24:3428; 8. Pascart T. *Joint Bone Spine* 2024;91:105704.

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Gout is the Most Common Form of Inflammatory Arthritis[1]

Gout is caused by hyperuricemia (sUA ≥6.8 mg/dL), which can lead to MSU crystal deposition in joints and tissues[2]

Symptoms[3]

- Redness
- Joint swelling
- Severe pain
- Tophi
- Joint damage

Treatment Goals[4–6]

- Preserve/improve QoL
- Reduce inflammation/flare rates
- Reduce pain
- Maintain sUA levels
- Prevent joint damage/reduce tophi size

Estimated 9.2 M adults in the US living with gout in 2015–2016[7]

3–4× more common than RA[1]



Uncontrolled gout

~2–6% US patients are refractory to treatment[8]

MSU, monosodium urate; QoL, quality of life; RA, rheumatoid arthritis; sUA, serum uric acid.

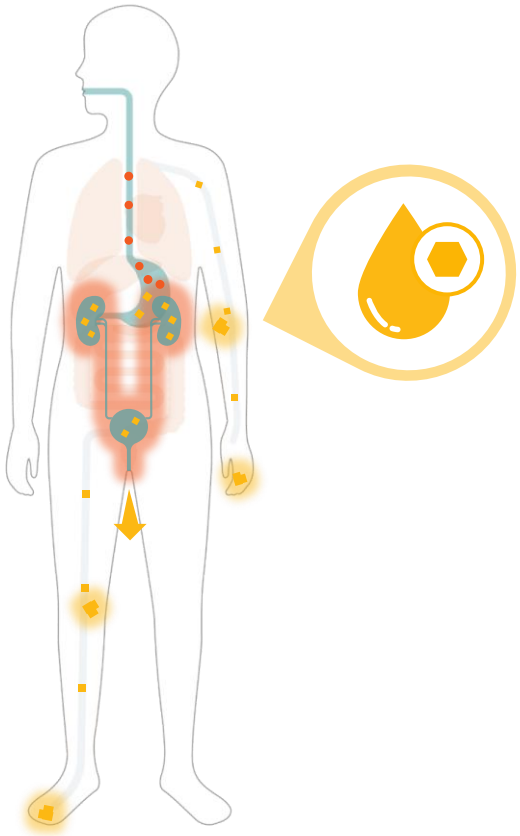
1. Singh JA, Gaffo A. *Semin Arthritis Rheum* 2020;50(3S):S11–S16; 2. Girardet JL, Miner JN. *Annu Rep Med Chem* 2014;49:151–164; 3. Dalbeth N, et al. *Lancet* 2021;397:1843–1855;
4. Arthritis Foundation. Treating arthritis with a total wellness plan. Available at: https://www.arthritis.org/health-wellness/treatment/treatment-plan/disease-management/treating-arthritis-with-a-total-wellness-plan. Accessed November 2025; 5. Hershfield MS. *Curr Opin Rheumatol* 2009;21:138–142; 6. NIH. Gout: Diagnosis, Treatment, and Steps to Take. Available at: https://www.niams.nih.gov/health-topics/gout/diagnosis-treatment-and-steps-to-take. Accessed December 2025; 7. Chen-Xu M, et al. *Arthritis Rheumatol* 2019;71:991–999; 8. Edwards NL. *Arthritis Rheum* 2008;58:2587–2590.

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Gout is a Systemic Inflammatory Disorder Characterized by MSU Crystal Formation and Painful Flares[1,2]

Increased levels of sUA[1]

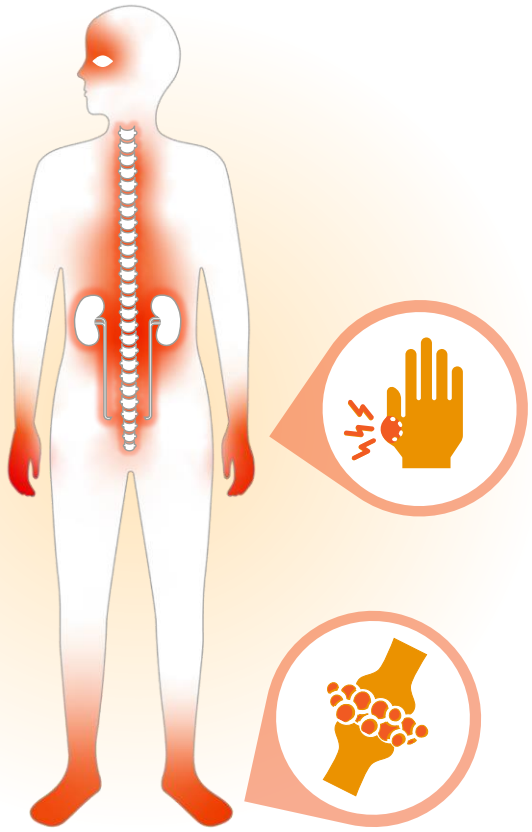
Over-production or insufficient removal of uric acid causes MSU crystals to form throughout the body[1,3]



Pain and inflammation[1]

Increases in pain and inflammation can signal disease worsening – this is known as a flare, which typically has a rapid onset[1]

Large build ups of MSU crystals can lead to tophi, which are not always palpable[1]



ULT can usually lower sUA levels and effectively reduce gout symptoms.
However, ULT may not always be effective, leading to high sUA levels and symptoms – this is known as uncontrolled gout[4,5]

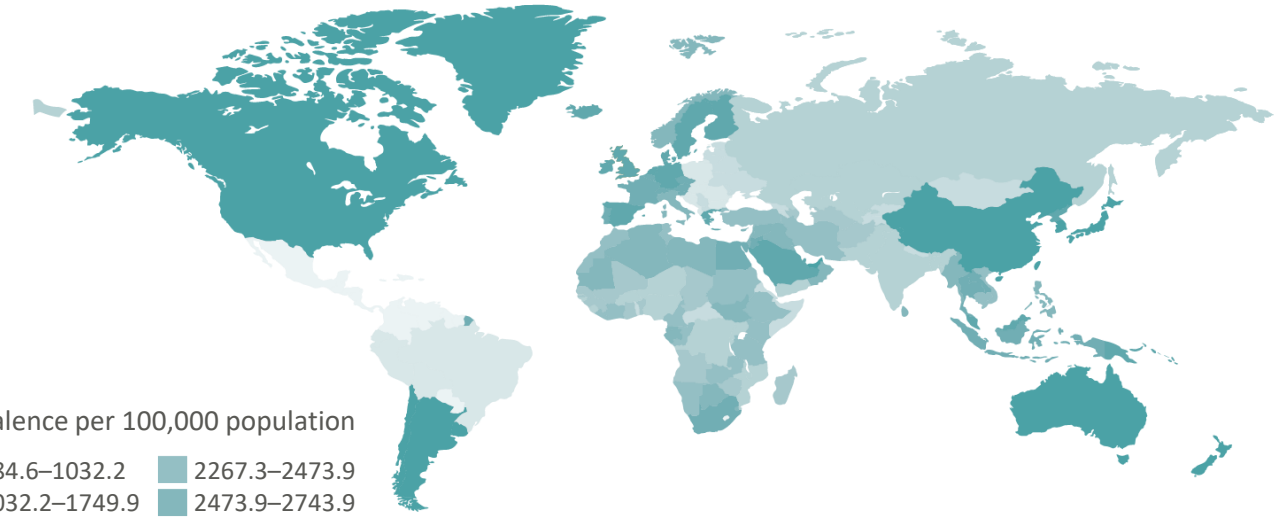
MSU, monosodium urate; sUA, serum uric acid; ULT, urate-lowering therapy.

1. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 2. Srivastava A, et al. *Am J Kidney Dis* 2018;71:362–370; 3. Khanna P, et al. *J Clin Med* 2020;9:3204; 4. FitzGerald JD, et al. *Arthritis Care Res (Hoboken)* 2020;72:744–760; 5. Francis-Sedlak M, et al. *Rheumatol Ther* 2021;8:183–197.

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Global Prevalence and Incidence of Gout is Increasing[1]

Global age-standardized prevalence of gout in 2021[1]



0.1–0.3% Incidence[2]

1–4% Prevalence[2]

11–13% Prevalence in adults aged >80 years[2]

~55 million cases worldwide in 2020, and rising[3]

Figure adapted from Tang X, et al. *Front Public Health* 2025;13:1540190 (CC BY 4.0; https://creativecommons.org/licenses/by/4.0/).

1. Tang X, et al. *Front Public Health* 2025;13:1540190; 2. Singh JA, Gaffo A. *Semin Arthritis Rheum* 2020;50(3S):S11–S16; 3. Punzi L, et al. *Clin Exp Rheumatol* 2025;43:799–808.

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Sex and Ethnicity Influence Gout Prevalence[1]

Gout is ~3× more common in men than in women[1]



Certain ethnic populations in the US have a higher prevalence of gout[1–3]

African American men

26% higher odds of developing gout versus White men
OR 1.26 (95% CI 1.02–1.55)[2]

Hmong men and women

7% vs 3% prevalence of self-reported gout versus general population[3]

~2–6% of patients with gout in the US have uncontrolled gout[4]

Figure adapted from Singh JA, Gaffo A. *Semin Arthritis Rheum* 2020;50(3S):S11–S16.

1. Singh JA, Gaffo A. *Semin Arthritis Rheum* 2020;50(3S):S11–S16; 2. McCormick N, et al. *JAMA Netw Open* 2022;5:e2226804; 3. Portis AJ, et al. *Arthritis Care Res (Hoboken)* 2010;62:1386–1391;
4. Edwards NL. *Arthritis Rheum* 2008;58:2587–2590.

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Modifiable and Non-Modifiable Risk Factors Can Increase the Risk of Developing Gout[1]



Genetics[2,3]

- Family history (~2× risk)[a]
- Variants in genes encoding uric acid transporters



Lifestyle[1,4–6]

- Purine-rich foods
- Sugar-sweetened beverages
- Alcohol
- Sedentary behavior (↑17% odds with ≥11 hours/day)



Medical conditions[1,4,7,8]

- Hypertension
- CKD
- BMI ≥30 kg/m^2



Medications[1,4]

- Diuretics
- Low-dose aspirin
- Cyclosporine



Increased risk of developing gout[1]

[a]Risk of gout in individuals with affected first-degree relatives versus the general population: men, RR 1.91 (95% CI 1.90–1.93); women, RR 1.97 (95% CI 1.94–1.99). Nationwide, population-based study using the Taiwan national health insurance database.[2]

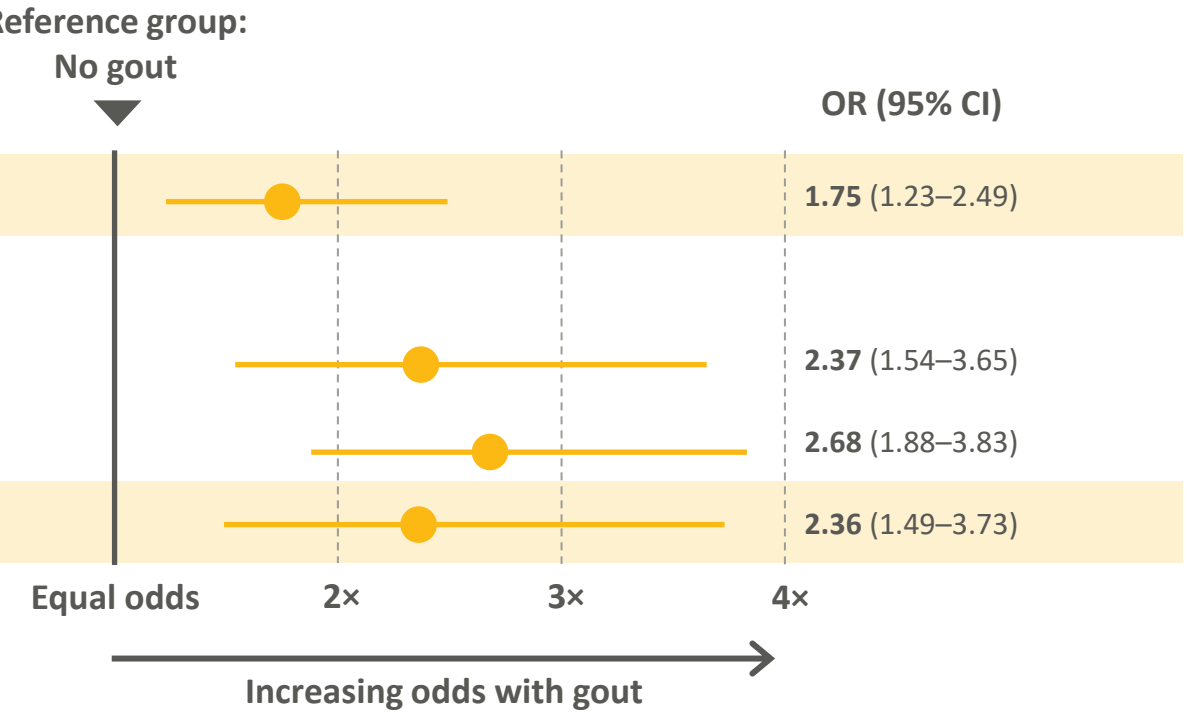
BMI, body mass index; CKD, chronic kidney disease; RR, relative risk.

1. NIH. Gout. Available at: https://www.niams.nih.gov/health-topics/gout. Accessed November 2025; 2. Kuo CF, et al. *Ann Rheum Dis* 2015;74:369–374;
3. Dalbeth N, et al. *BMC Med* 2017:15:108; 4. Asghari KM, et al. *BMC Musculoskelet Disord* 2024;25:1047; 5. Zgaga L, et al. *PLoS One* 2012;7:e38123; 6. Qin H, Hua Y. *BMC Public Health* 2024;24:3428;
7. Evans PL, et al. *Arthritis Res Ther* 2018;20:136; 8. Teramura S, et al. *J Atheroscler Thromb* 2023;30:1483–1491.

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Gout is Associated with Metabolic Comorbidities[1]

Comorbidity prevalence, % (95% CI)[1]	No gout	Gout[a]	OR (95% CI)
CKD stage ≥2[b]	42.1 (38.0–46.1)	71.1 (65.4–76.8)	**1.75** (1.23–2.49)
CV disease			
Myocardial infarction	2.9 (2.2–3.5)	14.4 (10.0–18.7)	**2.37** (1.54–3.65)
Heart failure	2.0 (1.5–2.6)	11.2 (6.9–15.5)	**2.68** (1.88–3.83)
Diabetes	7.8 (6.3–9.3)	25.7 (15.8–35.6)	**2.36** (1.49–3.73)



Treatment

People with gout often have additional comorbidities, and are on average prescribed 5.4–7.5 total medications[c,2]

[a]Data from 5707 participants (≥20 years) from the US NHANES 2007–2008 were analyzed. Prevalence and population estimates of comorbidities in individuals with gout and various levels of hyperuricemia were calculated and compared with those without these conditions. Overall, 3.9% of US adults reported a physician or health professional diagnosis of gout.[1] [b]GFR <60 mL/min/1.73 m^2.[1] [c]Cohort study of 36,431 adult patients with gout treated with allopurinol, febuxostat, or colchicine, using commercial health insurance claims data from January 1, 2009–December 31, 2011.[2]

CI, confidence interval; CKD, chronic kidney disease; CV, cardiovascular; GFR, glomerular filtration rate; NHANES, National Health and Nutrition Examination Survey; OR, odds ratio.

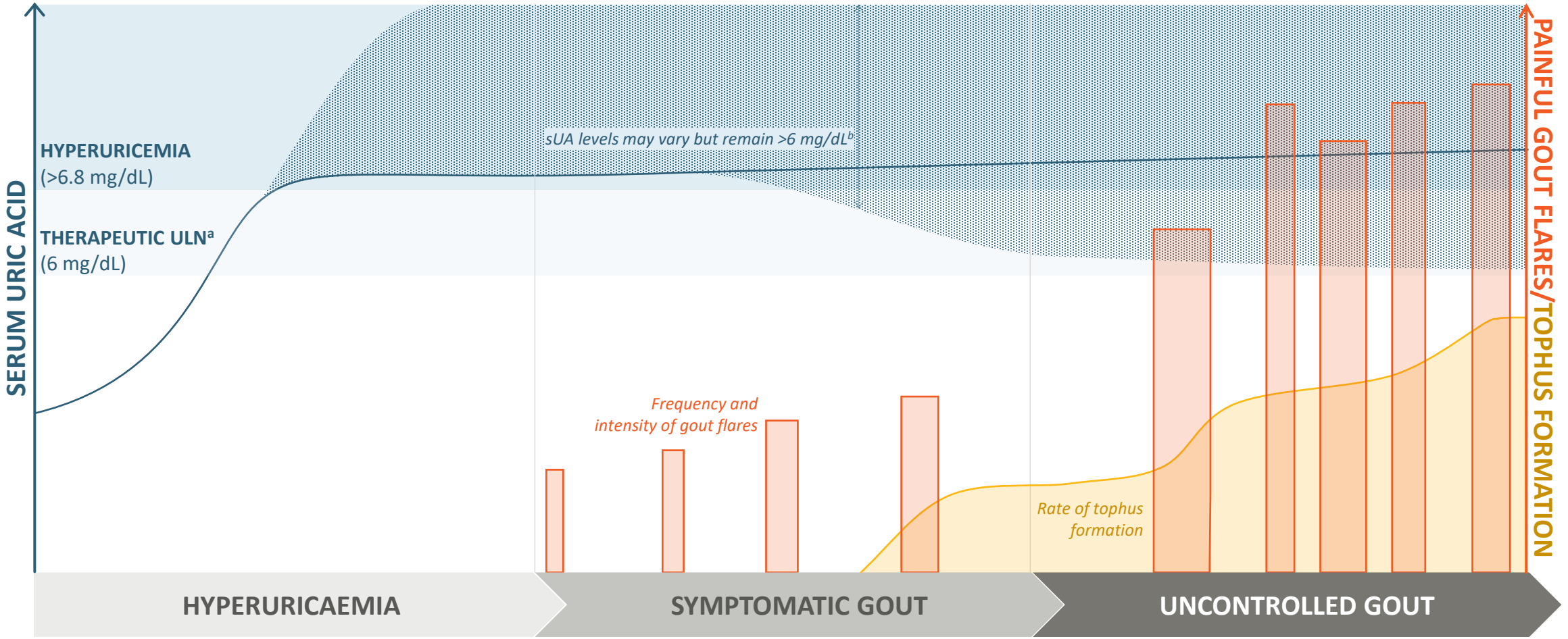
1. Zhu Y, et al. *Am J Med* 2012;125:679–687; 2. Kim SC, et al. *Arthritis Care Res (Hoboken)* 2013;65:2008–2014.

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Gout Progression

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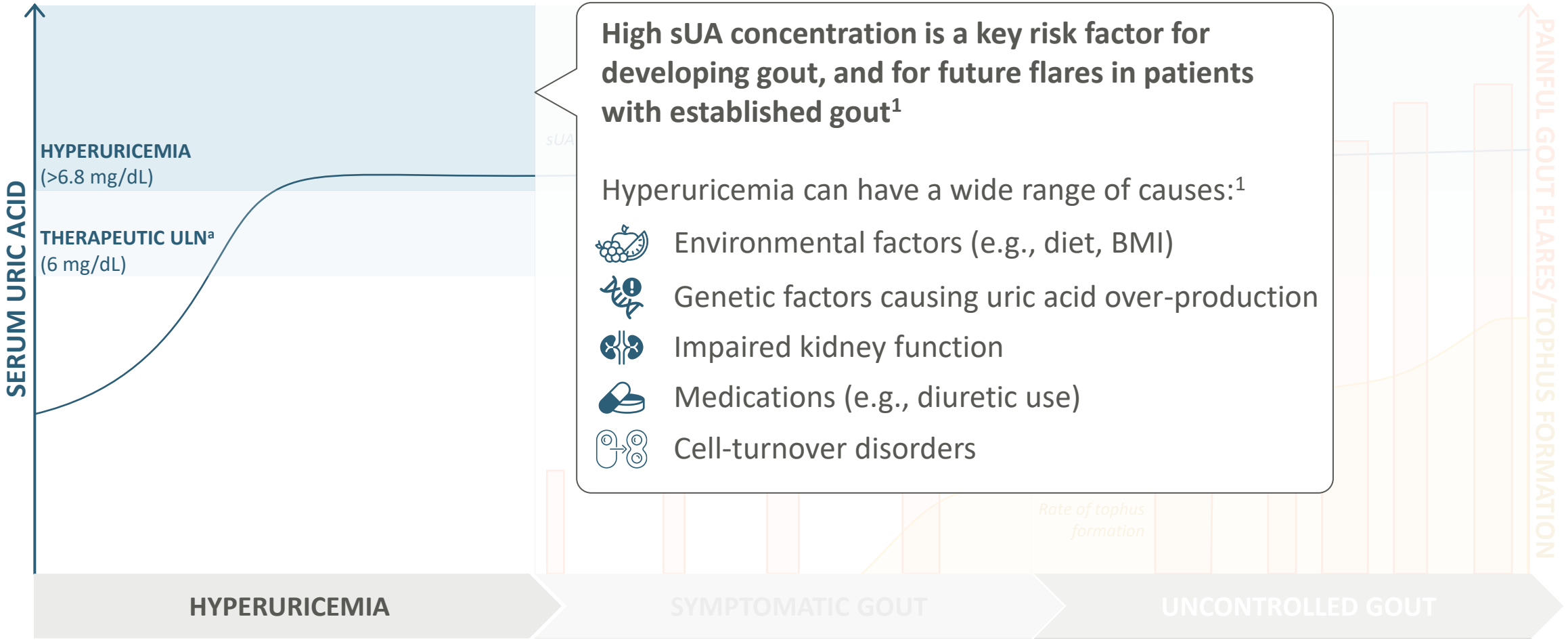
Typical Progression of Untreated Gout[1,2]



Schematic representation of sUA, gout flare frequency and intensity, and MSU crystal deposition with tophus formation over the course of disease progression to uncontrolled gout.
[a]Therapeutic ULN defined as the recommended sUA target for ULTs;[3] [b]Transient normalization of sUA may occur during a gout flare.[1]
MSU, monosodium urate; sUA, serum uric acid; ULN, upper limit of normal; ULT, urate-lowering therapy.
1. Dalbeth N, et al. *Nat Rev Dis Primers* 2019;5:69; 2. Edwards NL. *Arthritis Rheum* 2008;58:2587–2590; 3. FitzGerald JD, et al. *Arthritis Care Res (Hoboken)* 2020;72:744–760.
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Hyperuricemia[1]

Asymptomatic Elevation of sUA



High sUA concentration is a key risk factor for developing gout, and for future flares in patients with established gout[1]

Hyperuricemia can have a wide range of causes:[1]

- Environmental factors (e.g., diet, BMI)
- Genetic factors causing uric acid over-production
- Impaired kidney function
- Medications (e.g., diuretic use)
- Cell-turnover disorders

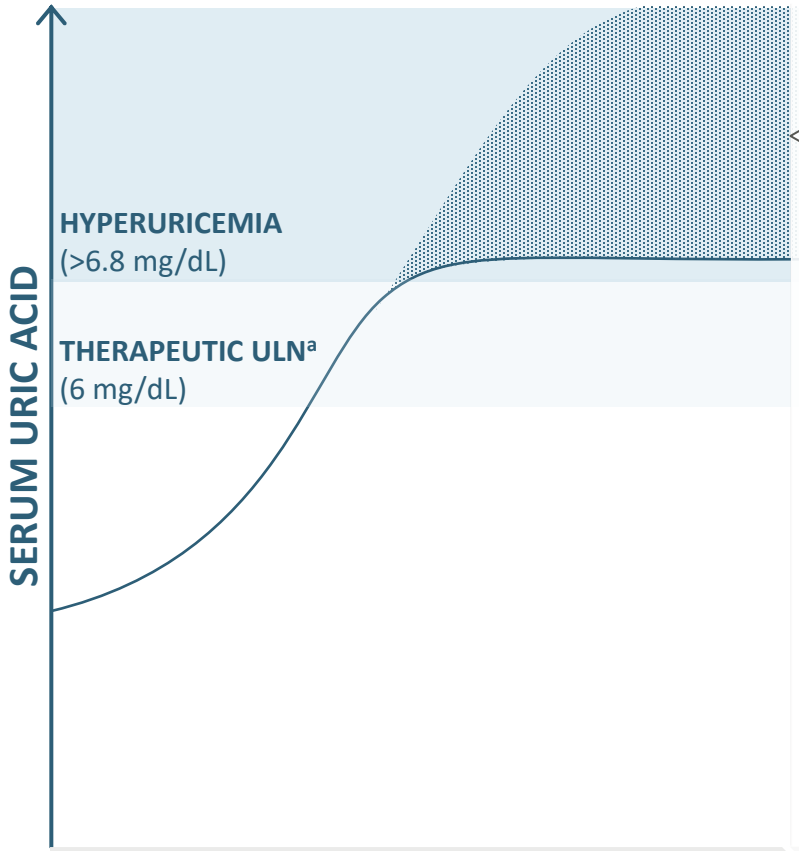
Schematic representation of sUA, gout flare frequency and intensity, and MSU crystal deposition with tophus formation over the course of disease progression to uncontrolled gout.
[a]Therapeutic ULN defined as the recommended sUA target for ULTs.[3]
BMI, body mass index; MSU, monosodium urate; sUA, serum uric acid; ULN, upper limit of normal; ULT, urate-lowering therapy.

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Hyperuricemia[1]

Asymptomatic MSU Crystal Deposition



sUA has a solubility limit of 6.8 mg/dL, at concentrations above this, MSU crystals begin to form[1]

MSU crystals preferentially deposit within joints and surrounding tissues, but also in the:[1,2]

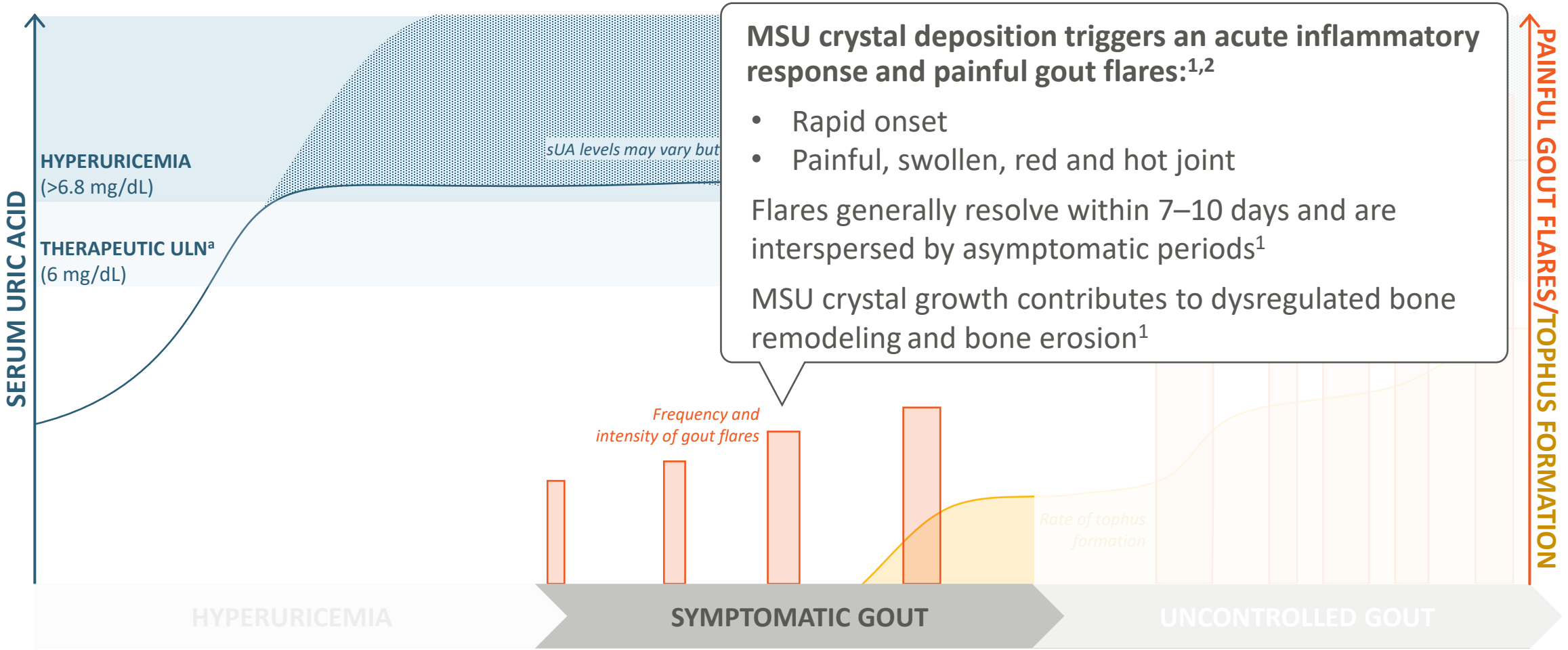
- CV system
- Kidneys
- Spine
- Skin
- Eyes
- GI system

Systemic MSU deposition causes chronic inflammation that may contribute to comorbidities associated with gout[2]

Schematic representation of sUA, gout flare frequency and intensity, and MSU crystal deposition with tophus formation over the course of disease progression to uncontrolled gout.
[a]Therapeutic ULN defined as the recommended sUA target for ULTs.[3]
CV, cardiovascular; GI, gastrointestinal; MSU, monosodium urate; sUA, serum uric acid; ULN, upper limit of normal; ULT, urate-lowering therapy.
1. Dalbeth N, et al. *Nat Rev Dis Primers* 2019;5:69; 2. Khanna P, et al. *J Clin Med* 2020;9:3204; 3. FitzGerald JD, et al. *Arthritis Care Res (Hoboken)* 2020;72:744–760.
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Symptomatic Gout[1]

Painful Gout Flares with Increasing Duration, Frequency, and Intensity



MSU crystal deposition triggers an acute inflammatory response and painful gout flares:[1,2]

- Rapid onset
- Painful, swollen, red and hot joint

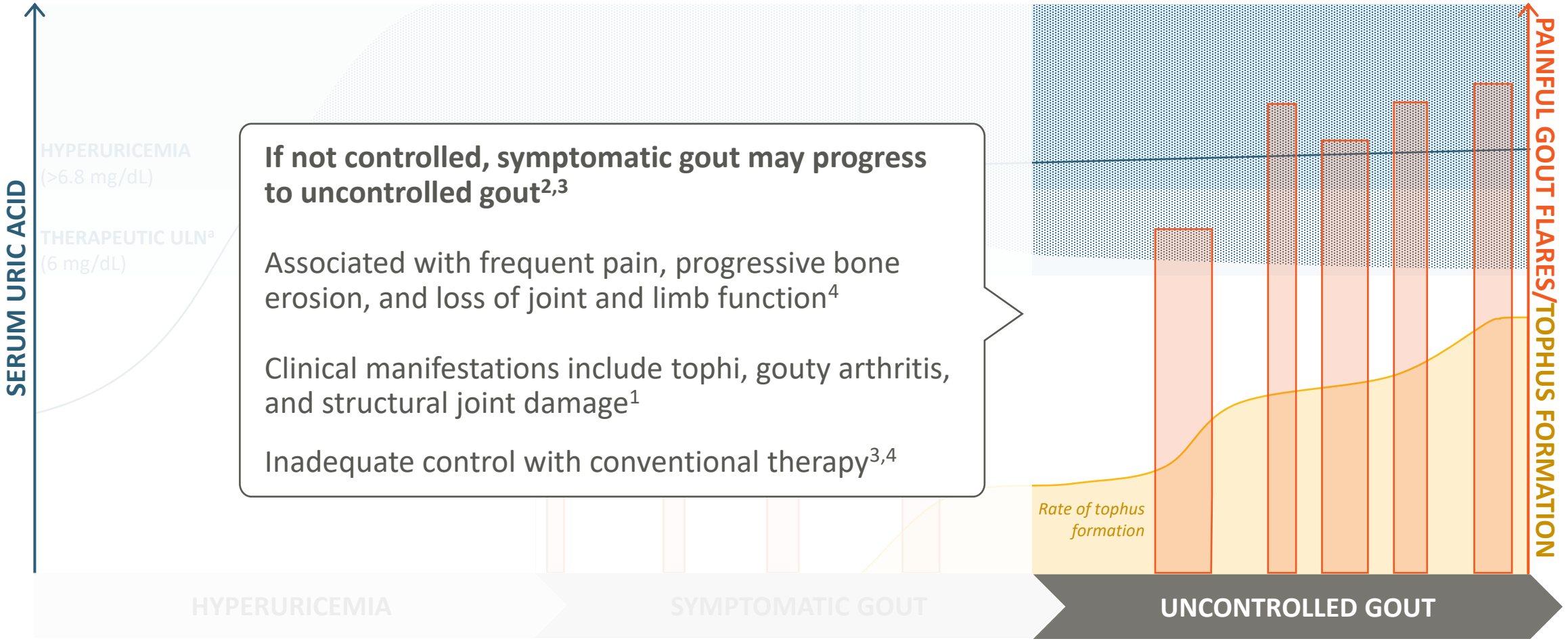
Flares generally resolve within 7–10 days and are interspersed by asymptomatic periods[1]

MSU crystal growth contributes to dysregulated bone remodeling and bone erosion[1]

Schematic representation of sUA, gout flare frequency and intensity, and MSU crystal deposition with tophus formation over the course of disease progression to uncontrolled gout.
[a]Therapeutic ULN defined as the recommended sUA target for ULTs.[3]
MSU, monosodium urate; sUA, serum uric acid; ULN, upper limit of normal; ULT, urate-lowering therapy.
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Uncontrolled Gout[1]

Tophus Formation, Progressive Joint Damage, and Disability



If not controlled, symptomatic gout may progress to uncontrolled gout[2,3]

Associated with frequent pain, progressive bone erosion, and loss of joint and limb function[4]

Clinical manifestations include tophi, gouty arthritis, and structural joint damage[1]

Inadequate control with conventional therapy[3,4]

Schematic representation of sUA, gout flare frequency and intensity, and MSU crystal deposition with tophus formation over the course of disease progression to uncontrolled gout.
MSU, monosodium urate; sUA, serum uric acid; ULN, upper limit of normal.

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Uncontrolled Gout

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Uncontrolled Gout is Associated with High Patient Burden and Metabolic Comorbidities

Uncontrolled gout is associated with unpredictable flares and a high patient burden affecting physical, social, emotional, and financial QoL[1,2]

Uncontrolled gout is associated with tophus formation, progressive joint damage, and disability[1,3]

Factors contributing to uncontrolled gout include delayed prescribing, inadequate treatment adjustments, poor adherence, and failure to adjust lifestyle/diet[3–5]

Patients with uncontrolled gout have a higher prevalence of metabolic disorders (CKD, T2D, and CVD) compared with controlled gout[1]

CKD, chronic kidney disease; CVD, cardiovascular disease; QoL, quality of life; sUA, serum uric acid; T2D, type 2 diabetes.

1. Francis-Sedlak M, et al. *Rheumatol Ther* 2021;8:183–197; 2. Strand V, et al. *Rheumatol Ther* 2024;11:1271–1290; 3. Fels E, Sundy JS. *Curr Opin Rheumatol* 2008;20:198–202;
4. FitzGerald JD, et al. *Arthritis Care Res (Hoboken)* 2020;72:744–760; 5. Edwards NL, Sundy JS. *Arth Rheum* 2008;58:2587–90.

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Uncontrolled Gout is Associated with Progressive Joint Damage and Disability[1,2]

ACR guideline definition[3]

sUA ≥6 mg/dL despite ULT[a] + ≥2 flares per year **or** subcutaneous tophi

Characteristics[1,2,4–7]

- Persistent hyperuricemia with recurrent flares
- Tophi
- Joint deformities
- Bone erosions
- Chronic pain
- Loss of function and disability

Contributing factors[2,3,7,8]

- Delayed prescribing, inadequate titration, ineffective therapy
- Poor adherence
- Failure to modify lifestyle/diet

[a]While the definition does not explicitly refer to uncontrolled gout, it can be inferred to describe patients with gout for whom xanthine oxidase inhibitor treatment, uricosurics, and other interventions have failed to achieve the sUA target.[3]
ACR, American College of Rheumatology; sUA, serum uric acid; ULT, urate-lowering therapy.

1. Francis-Sedlak M, et al. *Rheumatol Ther* 2021;8:183–197; 2. Fels E, Sundy JS. *Curr Opin Rheumatol* 2008;20:198–202; 3. FitzGerald JD, et al. *Arthritis Care Res (Hoboken)* 2020;72:744–760;
4. Schlesinger N, Lisky P. *Semin Arthritis Rheum* 2020;50:S31–S38; 5. Botson JK, et al. *Curr Rheumatol Rep* 2022;24:12–19; 6. Stamp LK, Gaffo A. *Expert Opin Biol Ther* 2023;23:1151–1154;
7. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 8. Edwards NL, Sundy JS. *Arth Rheum* 2008;58:2587–2590.

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Uncontrolled Gout Can Have Many Impacts on Patients[1]



Physical[1,3]

- Chronic body pain including joint tenderness and redness/heat
- Tophi and frequent flares can reduce HRQoL
- Limited range of motion – impact on daily activities, e.g., running errands
- Difficulty gripping or twisting objects and moving when climbing stairs
- Tiredness because of sleep disruption



Social[1,3]

- Difficulty carrying out social activities and hobbies
- Impact on social and romantic relationships



Emotional[1]

- Feeling worn out
- Anxiety, depression, and feelings of sadness



Financial[1–3]

- Flares can impact ability to work
- Greater indirect economic burden
- Potentially higher healthcare costs and more ED visits

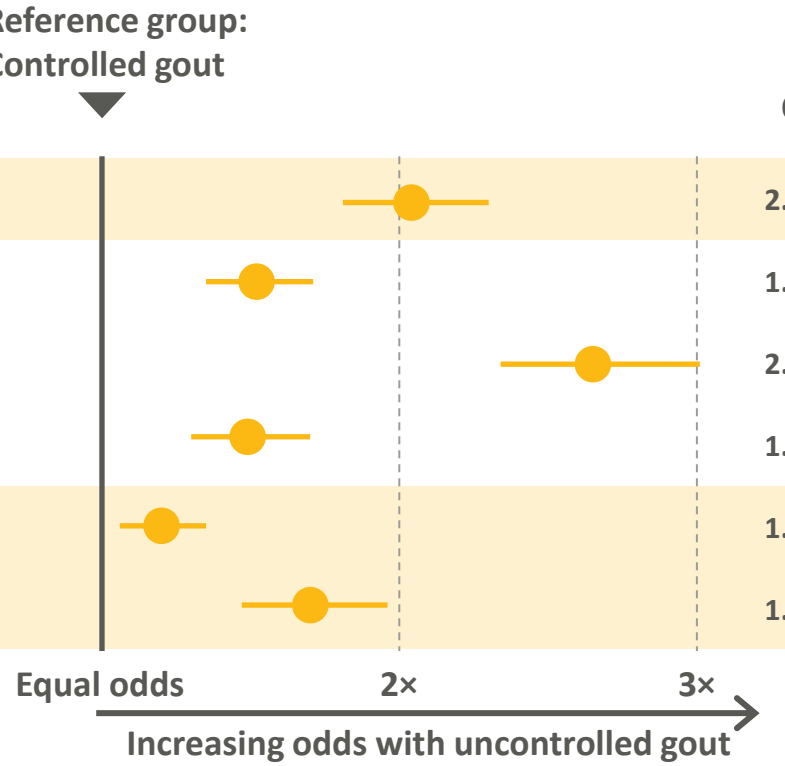
ED, emergency department; HRQoL, health-related quality of life.

1. Strand V, et al. *Rheumatol Ther* 2024;11:1271–1290; 2. Morlock RJ, et al. *Rheumatol Ther* 2024;12:37–51; 3. Edwards NL, et al. *J Med Econ* 2011;14:10–15.

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Patients with Uncontrolled Gout Have a Higher Prevalence of Metabolic Disorders Compared with Controlled Gout[a,1]

Comorbidity prevalence, n (%)[1]	Controlled gout (n=5473)	Uncontrolled gout[a] (n=1358)	OR (95% CI)	*P* value
CKD	1772 (32.4)	671 (49.4)	**2.04** (1.81–2.30)	<0.001
CV disease	2500 (45.7)	761 (56.0)	**1.52** (1.35–1.71)	<0.001
Heart failure	1039 (19.0)	520 (38.3)	**2.65** (2.33–3.01)	<0.001
Atrial fibrillation	1098 (20.1)	369 (27.2)	**1.49** (1.30–1.70)	<0.001
Diabetes	2730 (49.9)	738 (54.3)	**1.20** (1.06–1.35)	0.003
With renal manifestations	842 (15.4)	320 (23.6)	**1.70** (1.47–1.96)	<0.001



Comorbidities may reflect local, chronic inflammation from systemic uric acid deposition[2]

Uric acid deposition has been observed in:[2]

- Blood vessels
- Heart
- Kidney
- Spine
- Eye
- Skin
- GI system

[a]Data from adult patients with gout and who had at least 90 days of continuous ULT was collected from the Humana Research Database from 2007–2016. A total of 6831 patients were identified that met the inclusion criteria (5473 patients had controlled gout and 1358 patients had uncontrolled gout). Uncontrolled gout was defined as sUA ≥8.0 mg/dL.[1]

CI, confidence interval; CKD, chronic kidney disease; CV, cardiovascular; GI, gastrointestinal; OR, odds ratio; sUA, serum uric acid; ULT, urate-lowering therapy.

1. Francis-Sedlak M, et al. *Rheumatol Ther* 2021;8:183–197; 2. Khanna P, et al. *J Clin Med* 2020;9:3204.

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Pathophysiology

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Gout Progression is Driven by Hyperuricemia and Can Lead to Poor Health Outcomes[1,2]

Uric acid underexcretion[1]

- Genetic variants
- Comorbidities
- Certain medications

Uric acid over-production[1]

- Diet[1]
- Medical conditions causing high cell turnover[3]

Hyperuricemia

Super-saturation: sUA level ≥6.8 mg/dL[4]

MSU crystal deposition and growth[4]

Acute gout flare

- Innate inflammatory responses to MSU crystals lead to severe pain and swelling[3]
- Self-resolving[3]
- NLRP3 inflammasome activation[3]

Advanced, tophaceous gout

- Tophi formed by foreign-body reaction of immune cells to MSU crystals[3]
- Chronic inflammation[1]
- Joint damage[1]
- Recurrent flares[5]
- Poor health outcomes[2]

Uncontrolled gout[6,7]

Ongoing symptoms and sUA ≥6 mg/dL despite ULT

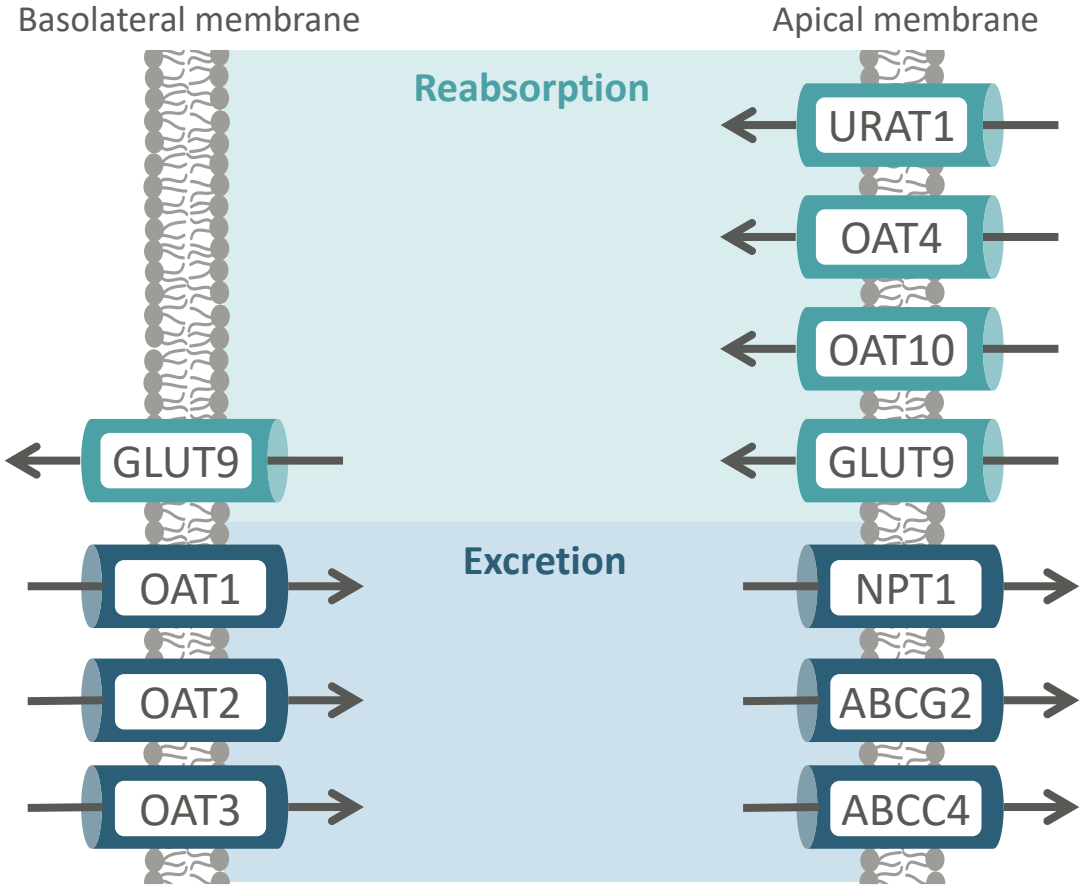
MSU, monosodium urate; NLRP3, NLR family pyrin domain containing 3; sUA, serum uric acid; ULT, urate-lowering therapy.

1. Ahn EY, et al. *J Rheum Dis* 2025;32:8–16; 2. Vincent ZL, et al. *J Rheumatol* 2017;44:368–373; 3. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 4. Oliviero F, et al. *Reumatismo* 2011;63:221–229; 5. Khanna PP, et al. *Health Qual Life Outcomes* 2012;10:117; 6. Francis-Sedlak M, et al. *Rheumatol Ther* 2021;8:183–197; 7. FitzGerald JD, et al. *Arthritis Care Res (Hoboken)* 2020;72:744–760.

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Uric Acid Excretion and Reuptake is Regulated by Transporters in the Kidney and Gut[1]

Kidney uric acid transporters[1,2]



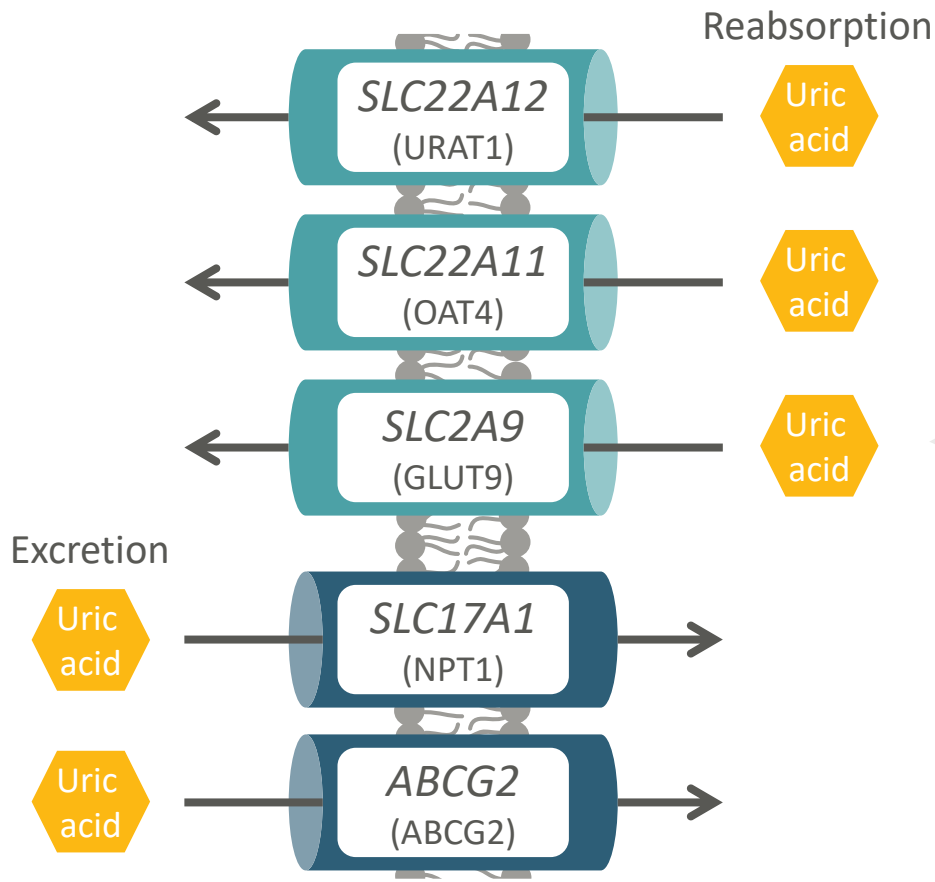
Factors affecting renal excretion of uric acid include elevated circulating insulin and certain medications, such as diuretics[1]

GLUT9 and ABCG2 have also been implicated in gut transport[1,3]

Figure adapted from Dalbeth N, et al. *BMC Med* 2017:15:108 (CC BY 4.0; http://creativecommons.org/licenses/by/4.0/).
ABCC4, ATP binding cassette sub-family C member 4; ABCG2, ATP binding cassette sub-family G member 2; GLUT9, glucose transporter 9; NPT1, sodium phosphate cotransporter 1; OAT, organic anion transporter; URAT, urate transporter.
1. Dalbeth N, et al. *Lancet* 2021:397:1843–1855; 2. Dalbeth N, et al. *BMC Med* 2017:15:108; 3. DeBosch BJ, et al. *Nat Commun* 2014;5:4642.
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Variants in Genes Encoding Uric Acid Transporters are Associated with sUA Levels[1]

Uric acid transporters are located in the kidneys and gut[1]



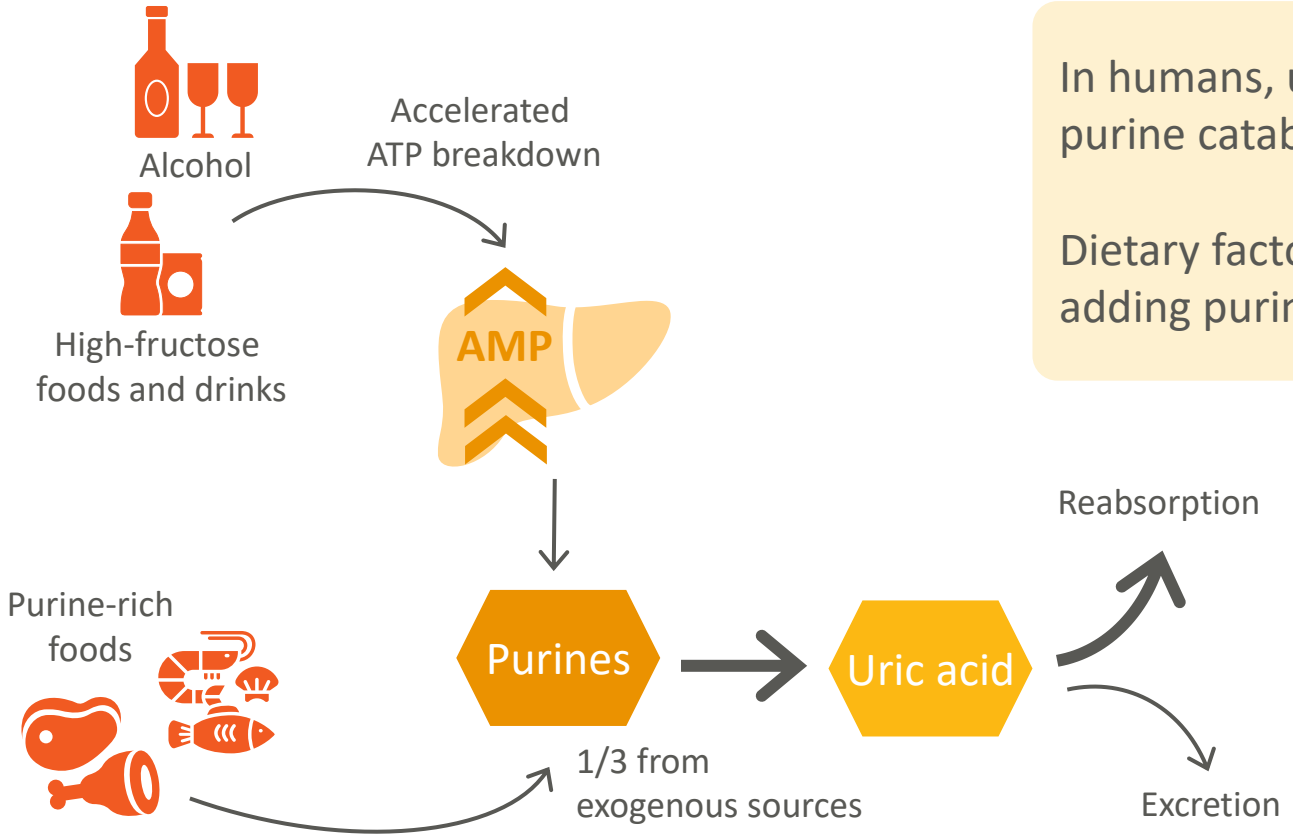
The variant *rs12498742* in *SLC2A9* has a stronger sUA-elevating effect in women[1,2]

SLC2A9 variants are important for uric acid control across ancestral groups, including European, African-American, Indian, Japanese, and East Asian[1]

The variant *rs2231142* in *ABCG2* has a stronger sUA-elevating effect in men[1,2]

Figure adapted from Dalbeth N, et al. *BMC Med* 2017:15:108 (CC BY 4.0; http://creativecommons.org/licenses/by/4.0/).
ABCG2, ATP binding cassette sub-family G member 2; GLUT9, glucose transporter 9; NPT1, sodium phosphate cotransporter 1; OAT, organic anion transporter; sUA, serum uric acid; URAT, urate transporter.
1. Dalbeth N, et al. *BMC Med* 2017:15:108; 2. Köttgen A, et al. *Nat Genet* 2013;45:145–154 (supplementary Information).
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Purine Overload Contributes to Hyperuricemia[1]



In humans, uric acid is the final breakdown product in purine catabolism[1]

Dietary factors can lead to elevations in sUA by directly adding purines, or by altering metabolic processes[1]

Associated with higher sUA levels[2,3]	Associated with lower sUA levels[2,3]
Beer	Eggs
Liquor	Peanuts
Wine	Cold cereal
Potatoes	Skim milk
Poultry	Cheese
Soft drinks	Brown bread
Beef, pork, or lamb	Margarine
Seafood	Non-citrus fruit

AMP, adenosine monophosphate; ATP, adenosine triphosphate; sUA, serum uric acid.

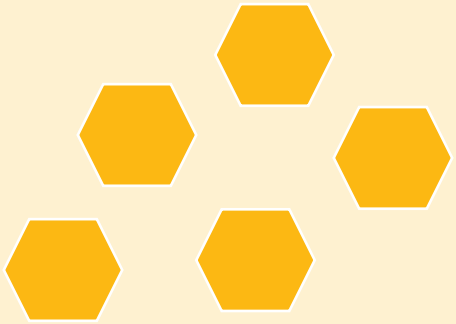
1. Zhang Y, et al. *Nutrients* 2022;14:3525; 2. Major TJ, et al. *BMJ* 2018;363:k3951; 3. Choi HK, et al. *Arthritis Rheum* 2005;52:283–289.

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Uric Acid Super-Saturation is the Key Factor for MSU Crystal Formation[1,2]

Uric acid super-saturation[1,2]

Uric acid levels exceed the solubility limit of 6.8 mg/dL[1,2]



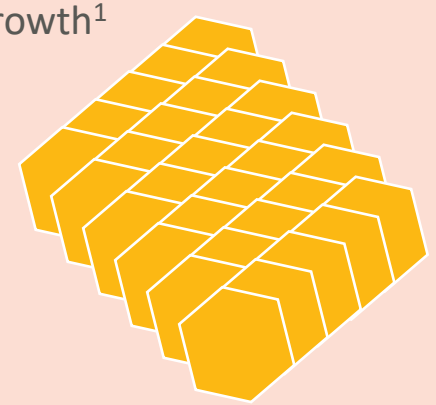
Nucleation[1,2]

Uric acid molecules start to cluster together, acting as centers of crystallization[1,2]



MSU crystal growth[1,2]

Continued sUA super-saturation leads to crystal growth[1]



Factors promoting MSU crystal formation[2]

- High uric acid concentration[a]
- High ion concentrations
- Low temperature
- pH

- High uric acid concentration[a]
- High sodium concentrations
- Uric acid binding antibodies
- Serum factors
- Type I collagen

- High uric acid concentration[a]

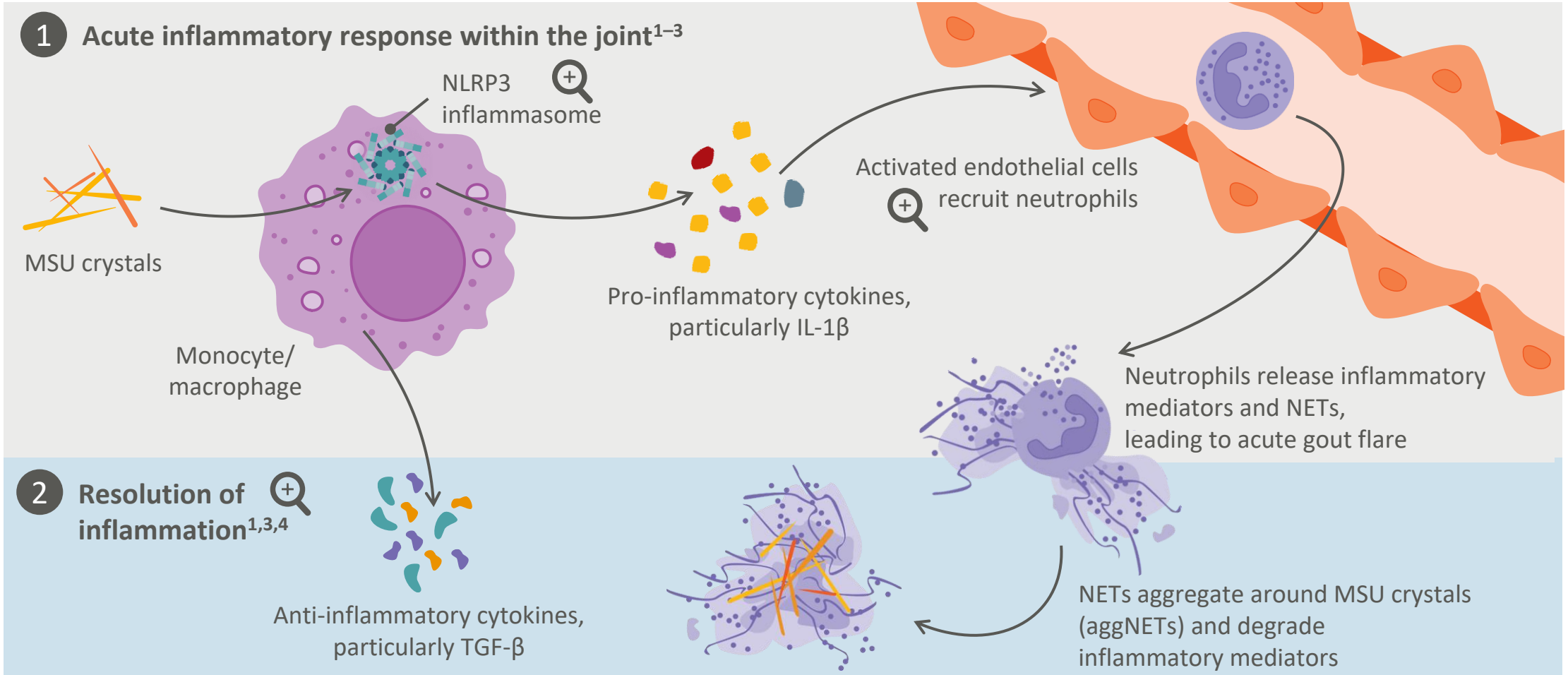
[a]High urate concentration is the main factor required for each stage of MSU crystallization.[2]

MSU, monosodium urate; sUA, serum uric acid.

1. Oliviero F, et al. *Reumatismo* 2011;63:221–229; 2. Chhana A, et al. *BMC Musculoskelet Disord* 2015;16:296.

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Gout Flares are Caused by an Acute Inflammatory Response to MSU Crystals[1,2]



aggNET, aggregated neutrophil extracellular trap; IL-1β, interleukin-1 beta; MSU, monosodium urate; NET, neutrophil extracellular trap; NLRP3, NLR family pyrin domain containing 3; TGF-β, transforming growth factor beta.

1. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 2. Busso N, So A. *Arthritis Res Ther* 2010;12:206; 3. Li C, et al. *J Inflamm Res* 2024;17:1735–1763; 4. Yagnik DR, et al. *Arthritis Rheumatol* 2004;50:2273–2280.

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NLRP3 Inflammasome Activation Requires Two Signals[1,2]

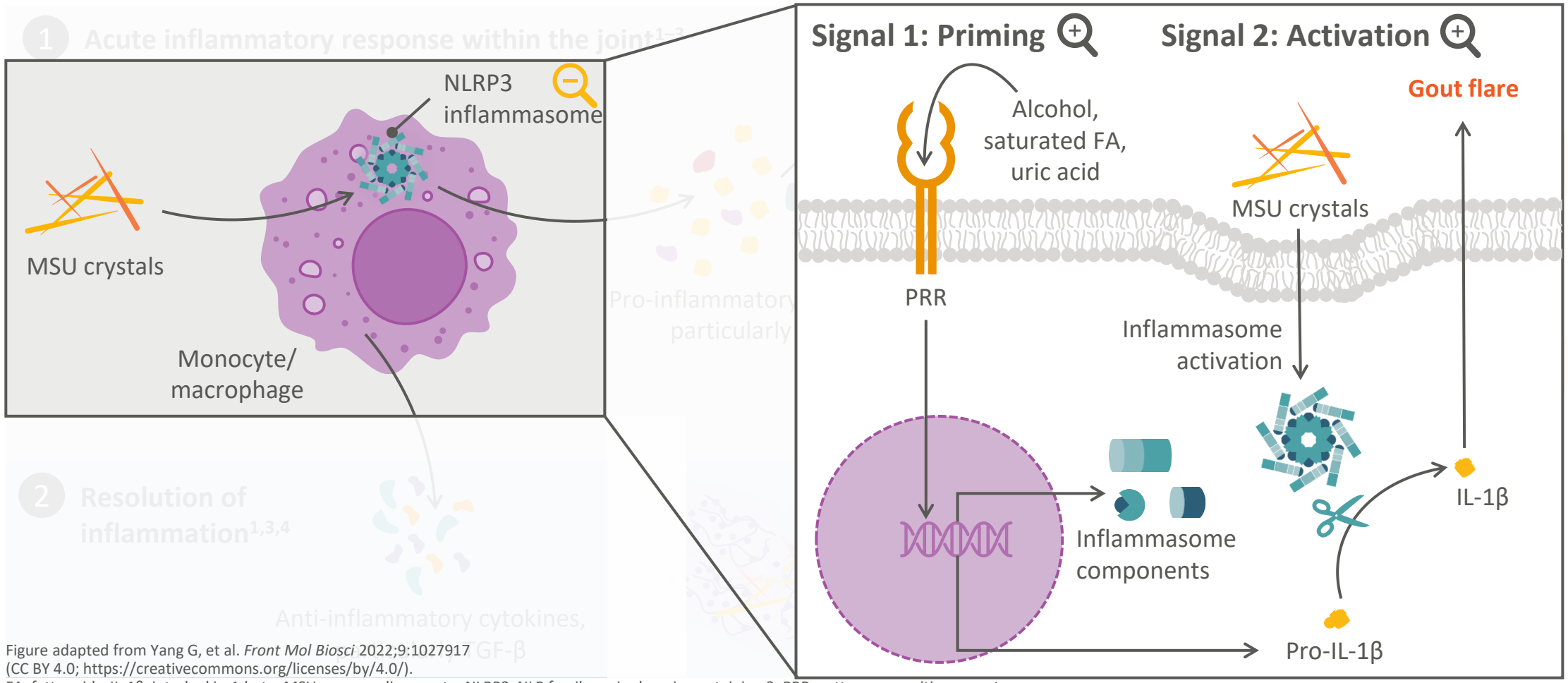


Figure adapted from Yang G, et al. *Front Mol Biosci* 2022;9:1027917 (CC BY 4.0; https://creativecommons.org/licenses/by/4.0/).

FA, fatty acids; IL-1β, interleukin-1 beta; MSU, monosodium urate; NLRP3, NLR family pyrin domain containing 3; PRR, pattern recognition receptor.

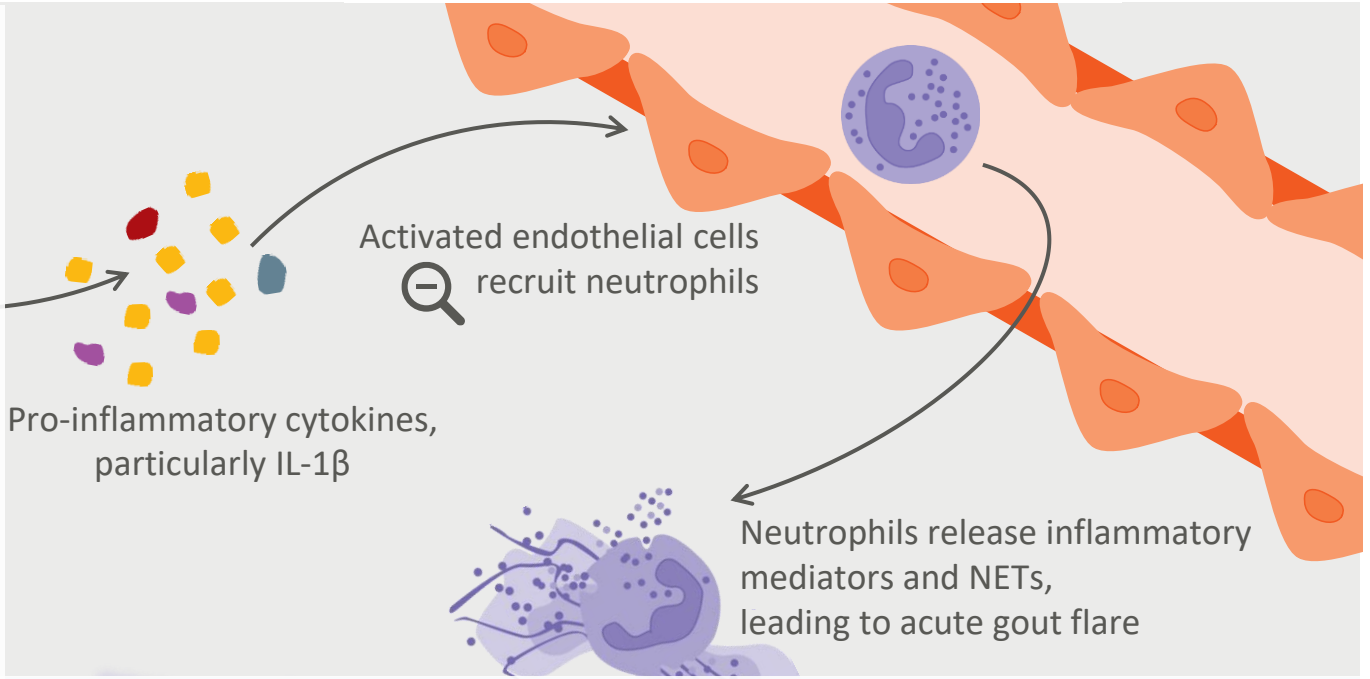
1. Yang G, et al. *Front Mol Biosci* 2022;9:1027917; 2. Poulsen R, Dalbeth N. *Arthritis Rheumatol* 2025;77:1317–1326.

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IL-1β Leads to Neutrophil Recruitment and Gout Flare[1]

Following macrophage activation, neutrophils and other immune cells are recruited to the joint, leading to synovial membrane hyperplasia[2]

- Additional inflammatory mediators, including more IL-1β and ROS, are released[1]
- IL-1β and ROS cause NET release associated with neutrophil death (NETosis), leading to further inflammation[1]



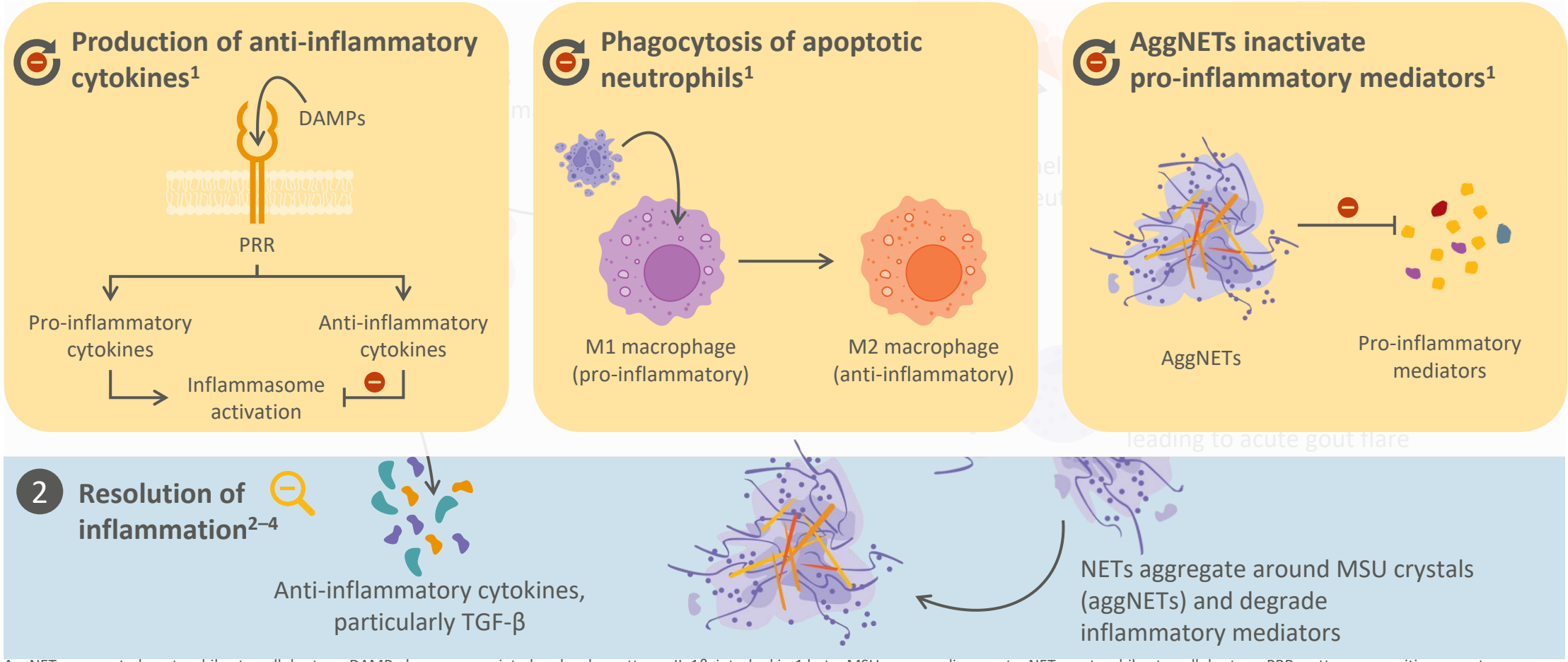
NETs are fibrous, extracellular structures composed of DNA, histones, and enzymes[1,3]

IL-1β, interleukin-1 beta; NET, neutrophil extracellular trap; ROS, reactive oxygen species.

1. Poulsen R, Dalbeth N. *Arthritis Rheumatol* 2025;77:1317–1326; 2. Narang RK, Dalbeth N. *Semin Nephrol* 2020;40:550–563; 3. Li C, et al. *J Inflamm Res* 2024;17:1735–1763.

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Negative Feedback Loops Lead to Self-Resolution of Gout Flare[1]



AggNET, aggregated neutrophil extracellular trap; DAMP, damage-associated molecular pattern; IL-1β, interleukin 1 beta; MSU, monosodium urate; NET, neutrophil extracellular trap; PRR, pattern recognition receptor; ROS, reactive oxygen species; TGF-β, transforming growth factor beta.

1. Poulsen R, Dalbeth N. *Arthritis Rheumatol* 2025;77:1317–1326; 2. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 3. Li C, et al. *J Inflamm Res* 2024;17:1735–1763; 4. Yagnik DR, et al. *Arthritis Rheumatol* 2004;50:2273–2280.

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Genetic Variants Related to NLRP3 Inflammasome Signaling are Associated with Increased Odds of Gout[1]

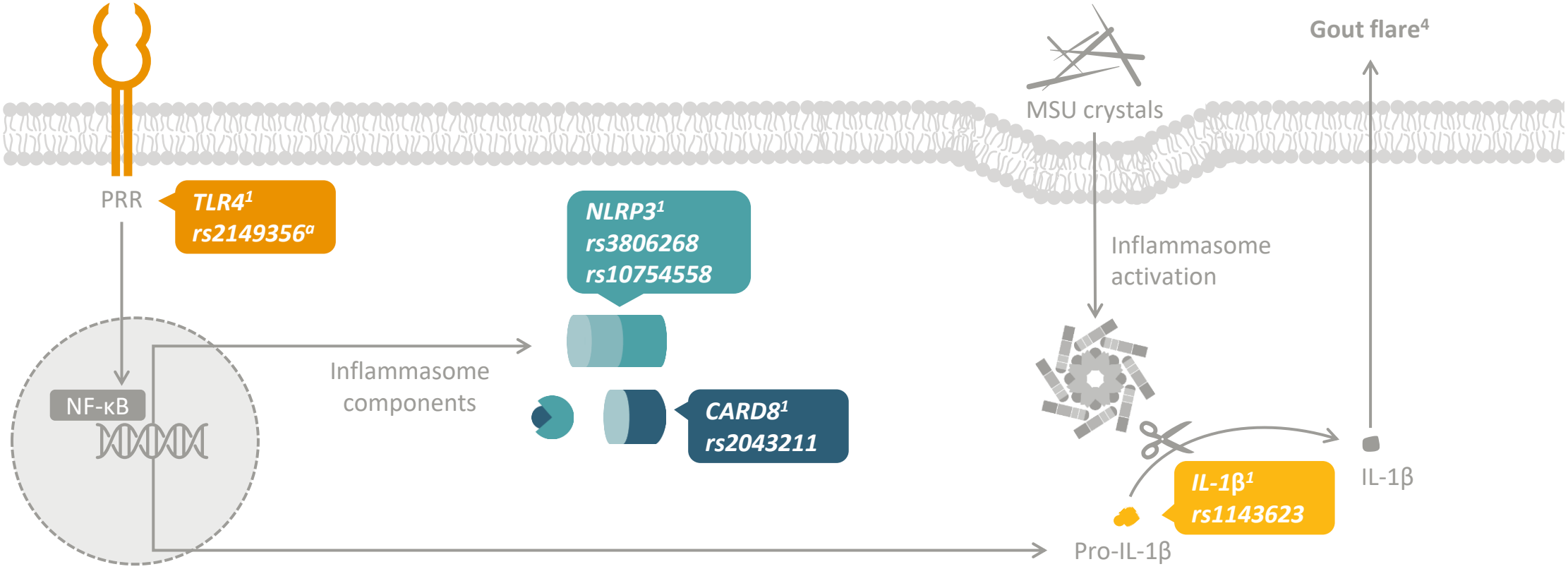


Figure adapted from Yang G, et al. *Front Mol Biosci* 2022;9:1027917 and Urwanisch L, et al. *Int J Mol Sci* 2021;22:1271 (CC BY 4.0; https://creativecommons.org/licenses/by/4.0/).

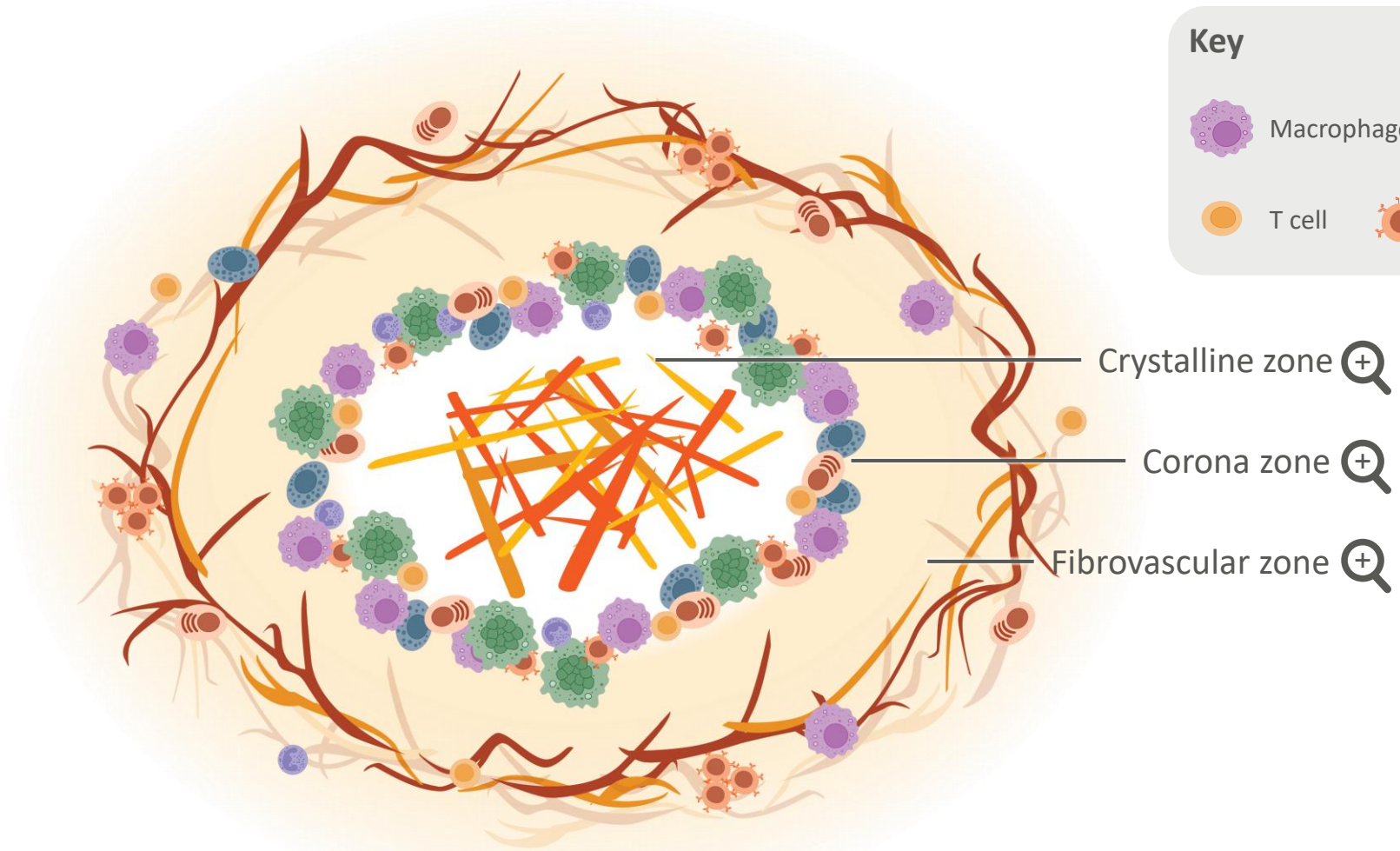
[a]Associated with an increased odds of gout in Chinese Han (OR 1.96; 95% CI 1.40–2.74) and European populations (OR 1.32; 95% CI 1.08–1.61), but a decreased odds of gout in Polynesian populations (OR 0.63; 95% CI 0.45–0.90).[1,5]

CARD, caspase-associated recruitment domain; CI, confidence interval; IL-1β, interleukin-1 beta; MSU, monosodium urate; NF-κB, nuclear factor kappa-light-chain-enhancer of activated B cells; NLRP3, NLR family pyrin domain containing 3; OR, odds ratio; PRR, pattern recognition receptor.

1. Clavijo-Cornejo D, et al. *Int J Rheum Dis* 2021;24:1257–1265; 2. Yang G, et al. *Front Mol Biosci* 2022;9:1027917; 3. Urwanisch L, et al. *Int J Mol Sci* 2021;22:1271; 4. Poulsen R, Dalbeth N. *Arthritis Rheumatol* 2025;77:1317–1326; 5. Qing Y-F, et al. *PLoS One* 2013;8:e64845.

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Tophi are Granuloma-Like Structures Consisting of Three Zones[1]



Key

- Macrophage/monocyte
- Plasma cell
- Mast cell
- T cell
- B cell
- Osteoclast
- Neutrophil

Tophi may be formed by NET aggregation[2]

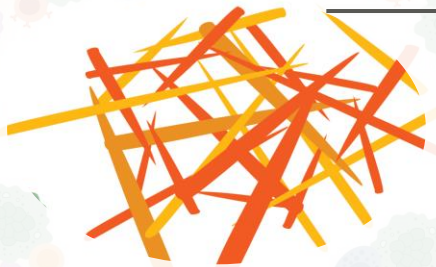
Tophi can occur without manifestations of acute inflammation, such as pain and swelling, but lead to joint destruction over time[2]

NET, neutrophil extracellular trap.

1. Towiwat P, et al. *BMC Musculoskelet Disord* 2019;20:140; 2. Desai J, et al. *Trends Mol Med* 2017;23:756–768.

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The Tophus Crystalline Zone is a Central Core of MSU Crystals



Crystalline zone

Corona zone

ovascular zone

The crystalline zone is acellular

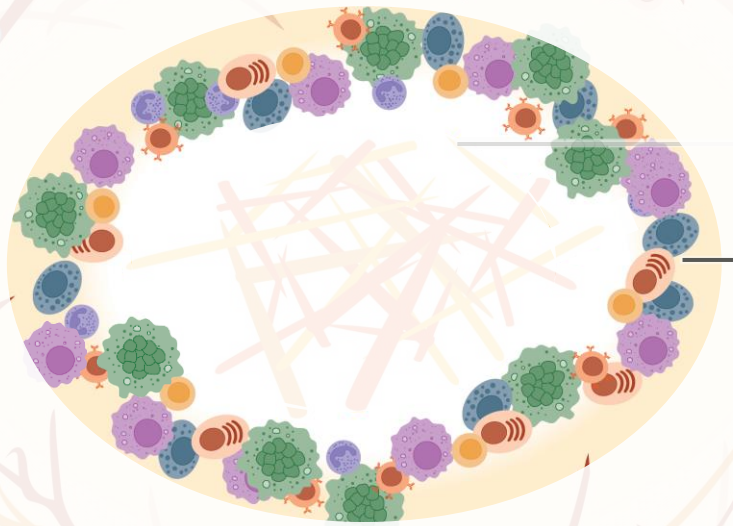
MSU crystals, antibodies, and monocyte-derived debris are present

MSU, monosodium urate.

Towiwat P, et al. *BMC Musculoskelet Disord* 2019;20:140.

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The Tophus Corona Zone Contains Innate and Adaptive Immune Cells[1]



Key

- Macrophage/monocyte
- Plasma cell
- Mast cell
- T cell
- B cell
- Osteoclast
- Neutrophil

Contains mono- and multi-nucleated macrophages and high numbers of plasma cells[2]

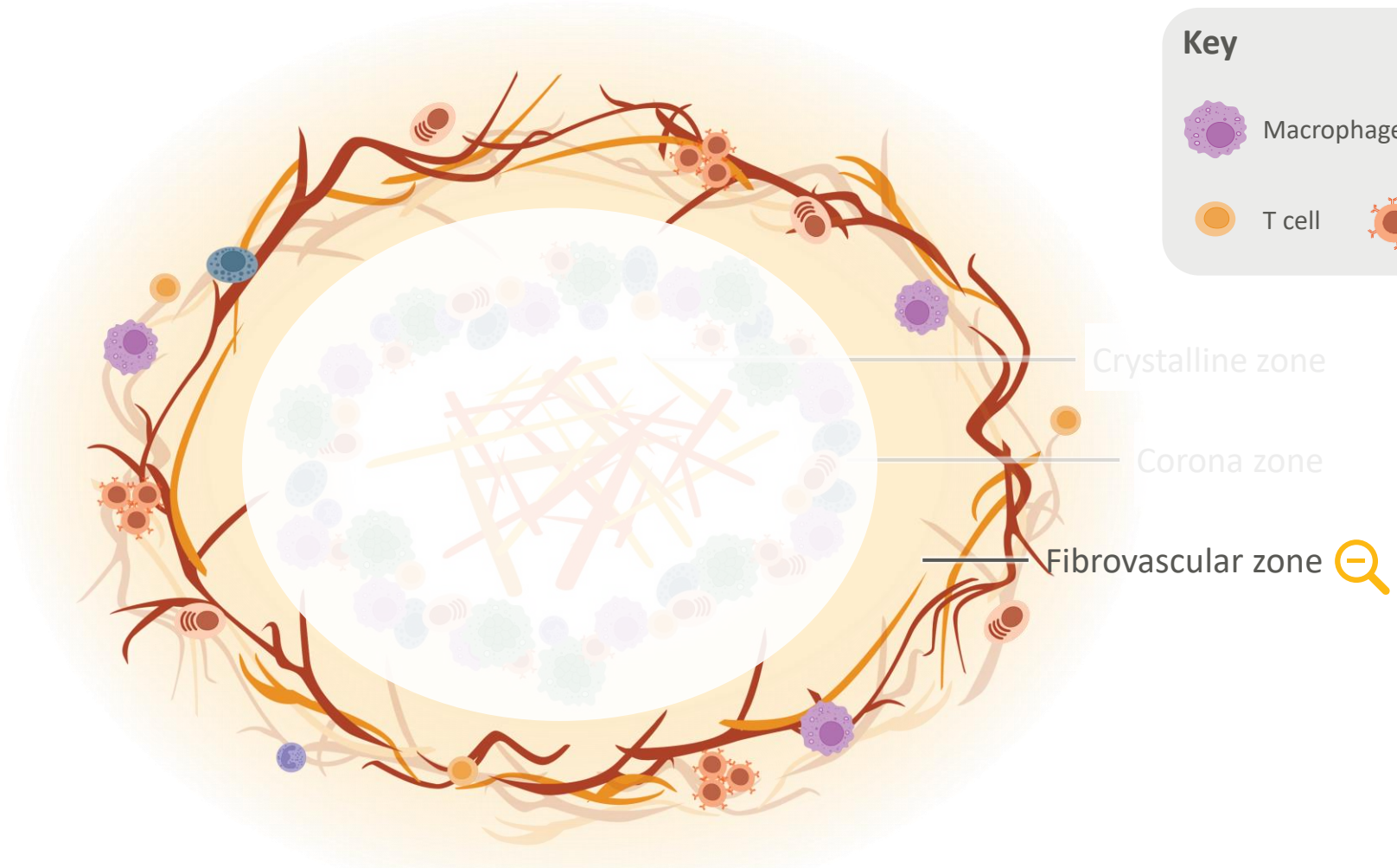
- Cells express both pro-inflammatory IL-1β and anti-inflammatory TGF-β, suggesting a cycle of chronic inflammation, attempted resolution, and tissue remodeling[2]

IL-1β, interleukin-1 beta; TGF-β, transforming growth factor beta.

1. Towiwat P, et al. *BMC Musculoskelet Disord* 2019;20:140; 2. Dalbeth N, et al. *Arthritis Rheum* 2010;62:1549–1556.

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The Tophus Fibrovascular Zone Contains Fibrous Tissue and Sparse Cellular Infiltrate[1]



Key

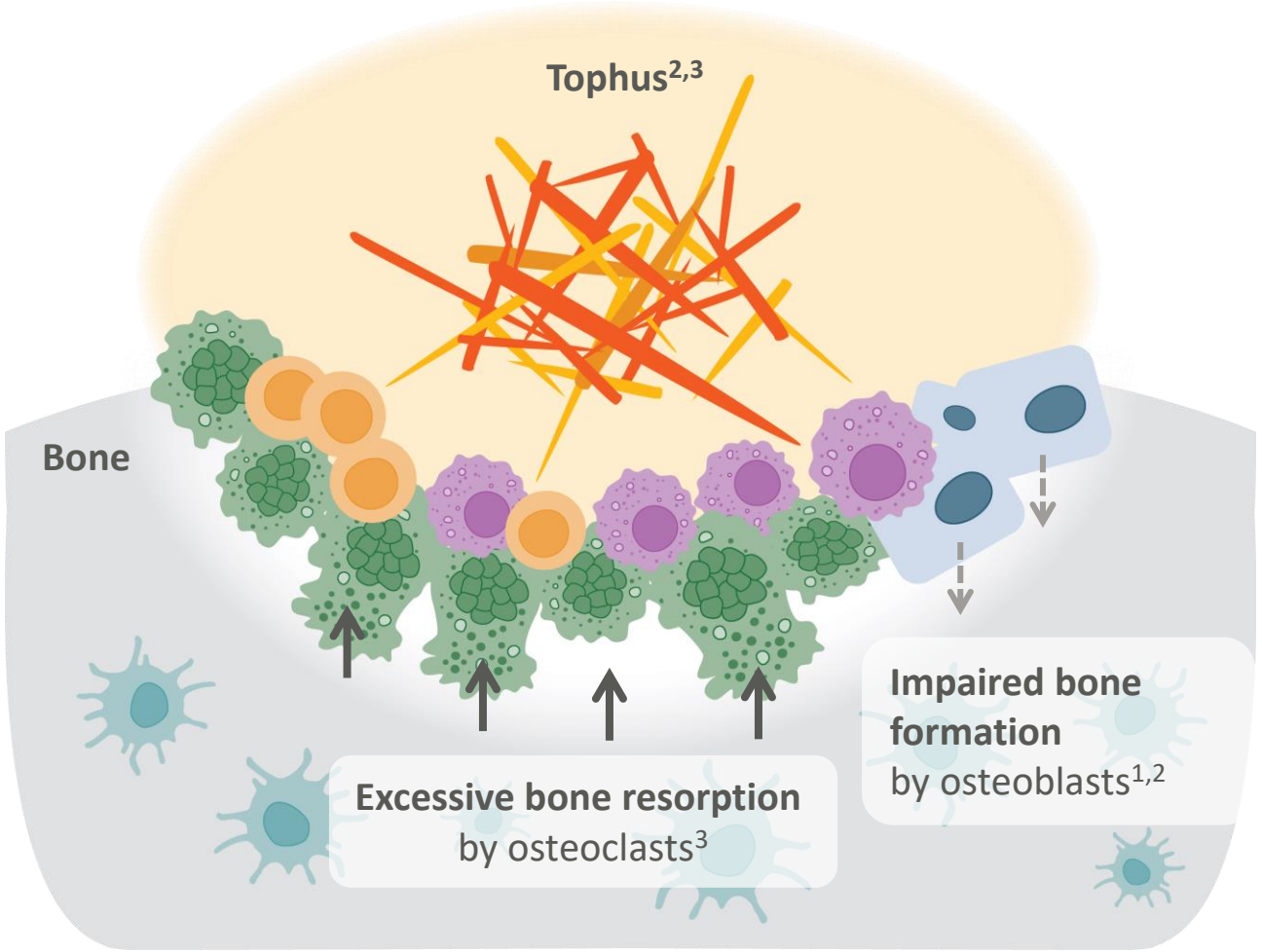
Macrophage/monocyte
Plasma cell
Mast cell
T cell
B cell
Osteoclast
Neutrophil

Consists of connective tissue with scattered immune cells[2]

- Contains low numbers of mononucleated macrophages, mast cells, T cells, and B cells[3]
- B cells may exist in aggregates[3]

1. Towiwat P, et al. *BMC Musculoskelet Disord* 2019;20:140; 2. Narang RK, Dalbeth N. *Semin Nephrol* 2020;40:550–563; 3. Dalbeth N, et al. *Arthritis Rheum* 2010;62:1549–1556.
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Tophus-Induced Joint Damage May be Related to Imbalanced Bone Turnover[1,2]



Key

Activated osteoclast

Osteoclast

Macrophage/ monocyte

Osteocyte

Osteoblast

T cell

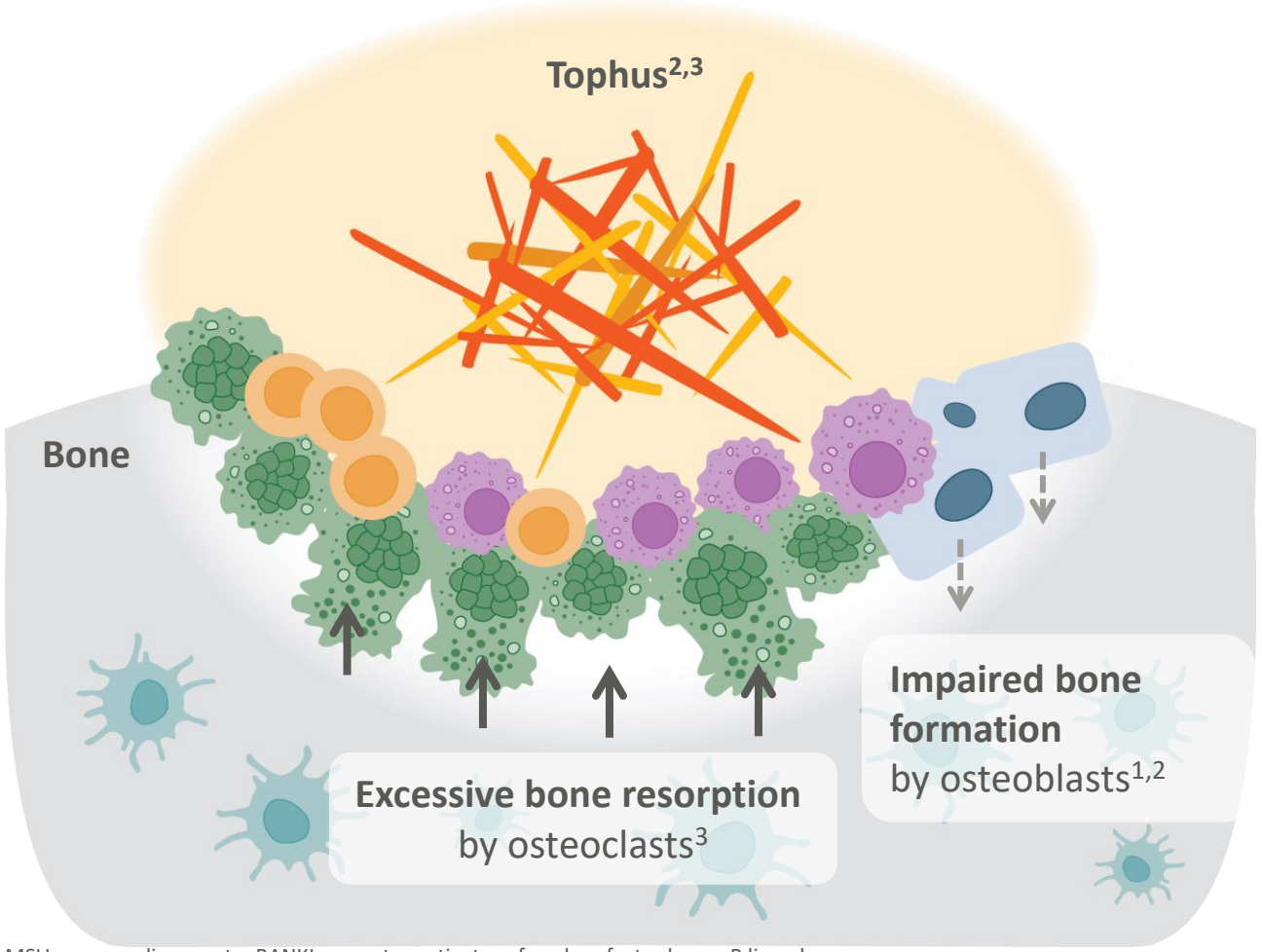
Cartilage may also be damaged by the effects of MSU crystals on chondrocytes (reduced viability and increased catabolism)[4]

MSU, monosodium urate.

1. Chhana A, et al. *Ann Rheum Dis* 2011;1684–1691; 2. Naot D, et al. *Arthritis Res Ther* 2022;24:212; 3. Lee S-J, et al. *Arthritis Res Ther* 2011;13:R164; 4. Chhana A, et al. *J Rheumatol* 2013;40:2067–2074.

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Altered Bone Turnover May be Related to the Direct and Indirect Effects of MSU Crystals on Bone Cells[1,2]



Key

Activated osteoclast

Osteoclast

Macrophage/ monocyte

Osteocyte

Osteoblast

T cell

The tophus microenvironment may promote excessive osteoclast formation and reduce osteoblast differentiation[1]

- RANKL+ T cells can be induced by MSU crystals and promote osteoclast formation[3]
- MSU crystal-stimulated macrophages attenuate osteoblast differentiation by secreting soluble mediators[2]
- MSU crystals reduce osteoblast and osteocyte viability[1,4]

MSU, monosodium urate; RANKL, receptor activator of nuclear factor kappa B ligand.

1. Chhana A, et al. *Ann Rheum Dis* 2011;1684–1691; 2. Naot D, et al. *Arthritis Res Ther* 2022;24:212; 3. Lee S-J, et al. *Arthritis Res Ther* 2011;13:R164; 4. Chhana A, et al. *Arthritis Res Ther* 2018;20:208.

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Tophi are Associated with Increased Mortality and Declining Renal Function[1,2]

Tophi are a sign of advanced disease, are associated with higher flare rates, and may indicate uncontrolled gout[3–5]

2.85× RISK

of all-cause mortality in patients with tophi[a,1]

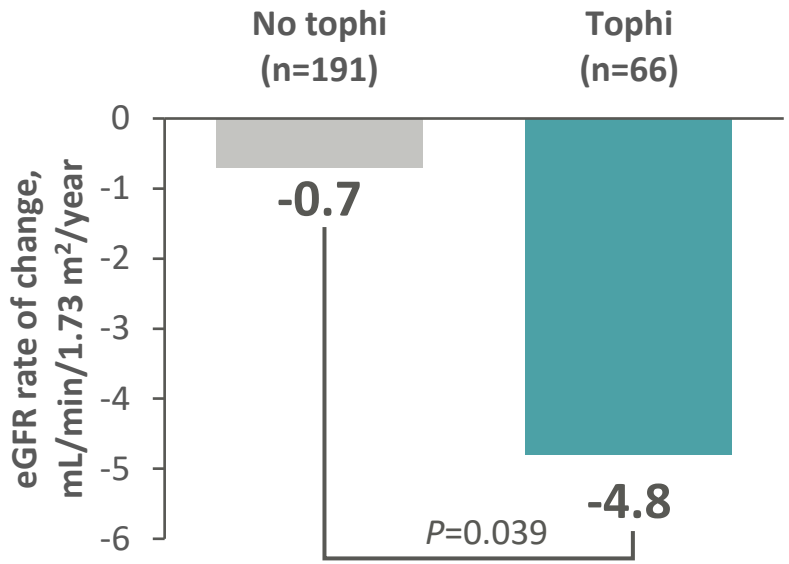
HR 2.85 (95% CI 1.49–5.44)[b]

Increased risk of mortality from CV and non-CV causes in patients with tophi[a,1]

CV: HR 3.13 (95% CI 1.38–7.10)[b]

Non-CV: HR 3.48 (95% CI 1.25–9.63)[b]

Change in eGFR in patients with or without tophi[c,2]



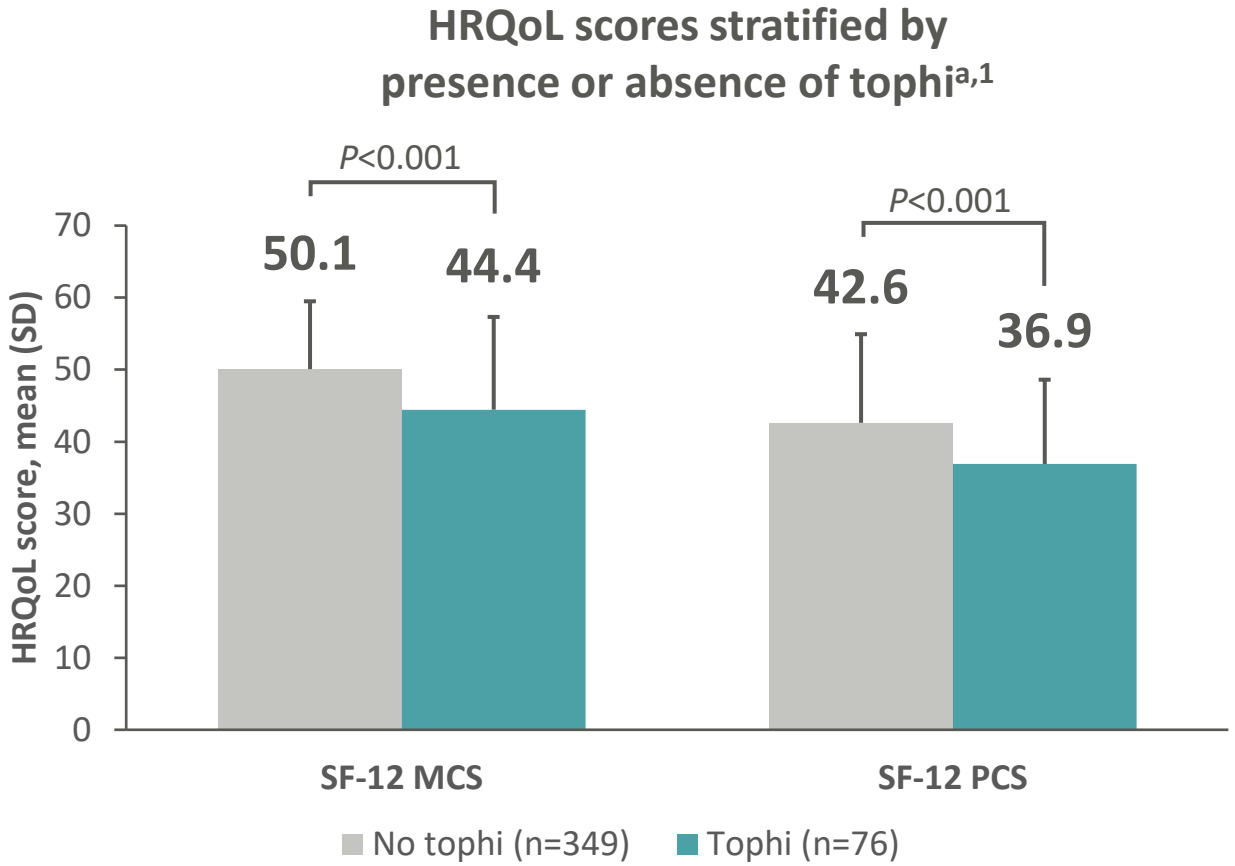
[a]Prospective, observational study of 295 patients with gout disease duration <10 years, from multiple centers in New Zealand. Patients were recruited from December 2006 until January 2014. Mean (SD) follow-up duration was 5.1 (1.6) years.[1] [b]HR calculated using a multivariate-adjusted Cox proportional hazards model for baseline predictors of all-cause mortality, including all variables with p<0.15 in the fully saturated model.[1] [c]Retrospective study of 257 adult patients with gout treated with febuxostat or allopurinol for ≥3 months at a single center in South Korea (January 2012 to December 2018). Median follow-up duration was 61.9 and 34.9 months in the tophi and no tophi groups, respectively.[2] Figure adapted from Oh Y-J, Moon KW. *Sci Rep* 2021;11:5684 (CC BY 4.0; https://creativecommons.org/licenses/by/4.0/).

eGFR, estimated glomerular filtration rate; SD, standard deviation.

1. Vincent ZL, et al. *J Rheumatol* 2017;44:368–373; 2. Oh Y-J, Moon KW. *Sci Rep* 2021;11:5684; 3. Ahn EY, So MW. *J Rheum Dis* 2025;32:8–16; 4. Khanna PP, et al. *Health Qual Life Outcomes* 2012;10:117; 5. Fitzgerald JD, et al. *Arthritis Care Res* 2020;72:744–760.

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Tophi are Associated with Lower HRQoL, Activity Impairment, and Functional Disability[1,2]



1.92× ODDS of functional disability in patients with versus without tophi[b,2]
aOR 1.92 (95% CI 1.07–3.43)[c]

48.7% activity impairment in patients with tophi, versus 33.4% in patients without tophi (*P*<0.001)[1]

[a]Cross-sectional survey of 620 adult patients with gout. Patients were identified through the US and EU versions of the 2010 National Health and Wellness Survey and invited to participate in a web-based questionnaire. HRQoL was assessed using the SF-12v2 questionnaire.[1] [b]Cross-sectional analysis of a multicenter cohort study of 446 patients with gout in Italy, recruited between June 2011 and January 2012. Functional disability was assessed using the HAQ-DI.[2] [c]Adjusted for age, gender, comorbidities, BMI, smoking, alcohol consumption, education, and employment.[2]

aOR, adjusted odds ratio; BMI, body mass index; CI, confidence interval; HAQ-DI, Health Assessment Questionnaire Disability Index; HRQoL, health-related quality of life; MCS, mental component score; PCS, physical component score; SD, standard deviation; SF-12, Medical Outcomes Short Form 12 questionnaire.

1. Khanna PP, et al. *Health Qual Life Outcomes* 2012;10:117; 2. Scirè CA, et al. *Arthritis Res Ther* 2013;15:R101.

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Diagnosis

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MSU Crystal Detection is the Gold Standard for Gout Diagnosis[1,2]



MSU crystal deposition may be asymptomatic initially, but can later lead to gout flares, tophi formation, and progressive joint damage with worsening pain between increasingly prolonged flares[1]



Gout treatment focus on:[1]

- Relieving acute flares with NSAIDs, colchicine, or corticosteroids
- Dissolving MSU crystals through long-term ULT (target sUA: 6 mg/dL)



MSU crystal detection remains the gold standard for gout classification[2]

- ACR/EULAR classification criteria (score ≥8) are used in research



- DECT and ultrasound can detect MSU crystal deposits[2]
- Conventional radiography identifies gout-related joint damage[2]

ACR, American College of Rheumatology; DECT, dual-energy computed tomography; EULAR, European Alliance of Associations for Rheumatology; MSU, monosodium urate; NSAID, non-steroidal anti-inflammatory drug; sUA, serum uric acid; ULT, urate-lowering therapy.

1. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 2. Neogi T, et al. *Ann Rheum Dis* 2015;74:1789–1798.

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Timely Diagnosis and Treatment are Important for Gout Management[1]

Early diagnosis and treatment
Reduced risk of flares and decreased severity of symptoms[1]

	Asymptomatic hyperuricemia[2]	Gout[2]	Advanced, tophaceous gout[2]
Features	• No symptoms/flares • MSU crystal deposition in joints and tissues	• Acute flares of pain, swelling, and redness • Symptom-free periods	• Chronic synovitis • Visible tophi • Progressive joint damage
Treatment focus	• Lifestyle modifications • Monitor sUA levels	• Relieve acute flares (NSAIDs, colchicine, corticosteroids) • Long-term ULT • Manage ongoing flares • Continuous ULT with target sUA of 6 mg/dL • Switch to pegloticase (uricase therapy) when XOI treatment, uricosurics, and other ULT have failed to achieve the sUA target (uncontrolled gout)[3]	
Treatment guide	**ACR/EULAR guidelines for sUA >6.8 mg/dL without flares or tophi:[3]** • Conditionally recommend against starting pharmacologic ULT (allopurinol, febuxostat, probenecid)	**ACR/EULAR guidelines for first-line and second-line long-term gout treatment:[3]** • Conditionally recommend initiating ULT for >1 flare with infrequent flares (<2 flares/year) • Strongly recommend initiating ULT for ≤1 subcutaneous tophi, radiographic damage, or frequent flares (>2 flares/year)	

ACR, American College of Rheumatology; EULAR, European Alliance of Associations for Rheumatology; MSU, monosodium urate; NSAID, non-steroidal anti-inflammatory drug; sUA, serum uric acid; ULT, urate-lowering therapy; XOI, xanthine oxidase inhibitor.

1. NIH. Gout. Available at: https://www.niams.nih.gov/health-topics/gout. Accessed November 2025; 2. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 3. FitzGerald JD, et al. *Arthritis Care Res (Hoboken)* 2020;72:744–760.

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ACR/EULAR Classification Criteria for Gout[1]

MSU crystal detection via microscopy of aspirated joint fluid remains the gold standard for gout classification[2]

Step 1
Entry criterion

≥1 episode of peripheral joint/bursal swelling, pain and/or tenderness

Step 2
Sufficient criterion

MSU crystals in symptomatic joint/bursa (synovial fluid) and/or tophus

If met, can classify as gout without applying additional criteria

Scoring criteria to be used if sufficient criterion not met →

Step 3
Scoring criteria

- Clinical
- Laboratory
- Imaging

Classification criteria are for research purposes and are not intended for making a diagnosis in a clinical setting[a,2]

[a]A web-based calculator can be accessed via the ACR and EULAR websites.[1]

ACR, American College of Rheumatology; EULAR, European Alliance of Associations for Rheumatology; MSU, monosodium urate.

1. Neogi T, et al. *Ann Rheum Dis* 2015;74:1789–1798; 2. Aggarwal R, et al. *Arthritis Care Res (Hoboken)* 2015;67:891–897.

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ACR/EULAR Scoring Criteria

Clinical

Score	Pattern of joint/bursal involvement
1	Ankle/mid-foot
2	First MTP joint
1	Erythema over joint
2	Extreme pain
3	Inability to use joint
4	Evidence of tophus

Time course of episode ≥2

1	Maximal pain <24 h
2	Resolve in ≤14 days

Laboratory

Score	sUA (mg/dL)
−4	<4[a]
2	6 to <8
3	8 to <10
4	≥10

Synovial fluid analysis

−2	MSU negative

Imaging

Score	Evidence of sUA deposition in joint/bursa
4	DECT showing sUA deposition
4	Ultrasound double-contour sign

Evidence of joint damage

4	Conventional radiography of the hands and/or feet showing ≤1 erosions

SCORE ≥8

classified as gout

[a]sUA levels measured by the uricase method (>4 weeks from the start of an episode and no ULT); if sUA is ≥4 mg/dL to >6 mg/dL, score as 0.

ACR, American College of Rheumatology; DECT, dual-energy computed tomography; EULAR, European Alliance of Associations for Rheumatology; MTP, metatarsophalangeal; sUA, serum uric acid; ULT, urate-lowering therapy.

Neogi T, et al. *Ann Rheum Dis* 2015;74:1789–1798.

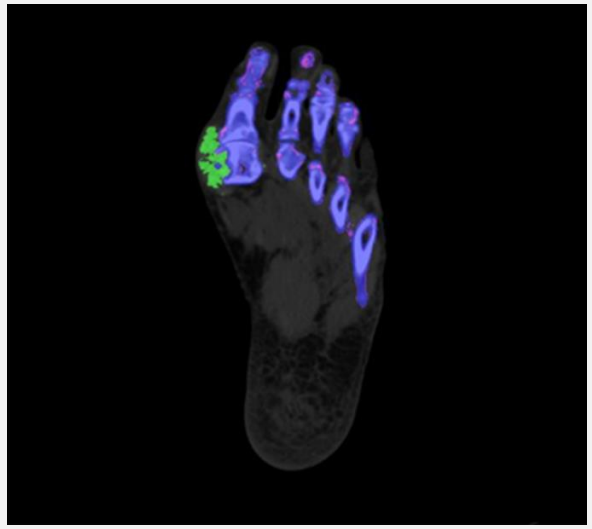
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DECT is Recommended to Identify MSU Crystal Deposits[1]

ACR/EULAR guideline: Imaging evidence of MSU crystals in any symptomatic joint or bursa[1]

DECT[a] | Ultrasound | Conventional radiography

MSU crystal deposition in first MTP joint[2]



Evidence of MSU crystal deposition on DECT:
Presence of color-coded MSU crystals at articular or peri-articular sites[1]

MRI and conventional CT are not recommended for imaging MSU crystal deposition[1]

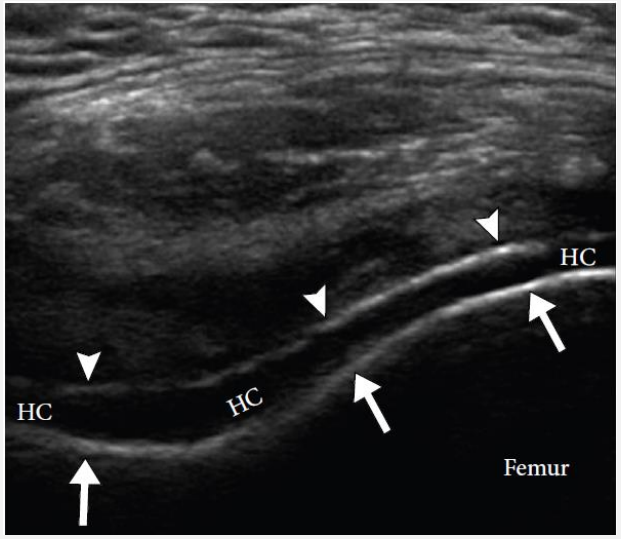
[a]Data were acquired at 80 kV and 140 kV and analyzed using the gout algorithm of commercial CT imaging software.[2]
Image adapted from Lee JS, Seo WJ. *Adv Rheumatol* 2021;61:40 (CC BY 4.0; https://creativecommons.org/licenses/by/4.0/).
ACR, American College of Rheumatology; CT, computed tomography; DECT, dual-energy computed tomography; EULAR, European Alliance of Associations for Rheumatology; MRI, magnetic resonance imaging; MSU, monosodium urate; MTP, metatarsophalangeal.
1. Neogi T, et al. *Ann Rheum Dis* 2015;74:1789–1798; 2. Lee JS, Seo WJ. *Adv Rheumatol* 2021;61:40.
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Ultrasound is Recommended to Identify MSU Crystal Deposits[1]

ACR/EULAR guideline: Imaging evidence of MSU crystals in any symptomatic joint or bursa[1]

DECT | **Ultrasound[a]** | Conventional radiography

Double-contour sign[b,2]



Evidence of MSU crystal deposition on ultrasound: Hyperechoic irregular enhancement on the hyaline cartilage surface, independent of the insonation angle of the ultrasound beam (double-contour sign)[1]

[a]A false-positive double-contour sign may appear on cartilage but will disappear when the probe angle changes.[1] [b]Long arrows indicate the femoral cortex and arrowheads indicate MSU crystals accumulating on the surface of the HC.[2]

Image adapted from Girish G, et al. *Arthritis* 2013;2013:673401 (CC BY 3.0; https://creativecommons.org/licenses/by/3.0/).

ACR, American College of Rheumatology; DECT, dual-energy computed tomography; EULAR, European Alliance of Associations for Rheumatology; HC, hyperechoic hyaline cartilage; MSU, monosodium urate.

1. Neogi T, et al. *Ann Rheum Dis* 2015;74:1789–1798; 2. Girish G, et al. *Arthritis* 2013;2013:673401.

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Conventional Radiography Can be Used to Identify Gout-Related Joint Damage[1]

ACR/EULAR guideline: Imaging evidence of gout-related joint damage of the hands and/or feet (≥1 erosion)[1]

DECT | Ultrasound | **Conventional radiography**

Erosion of the first MTP joint[a,2]



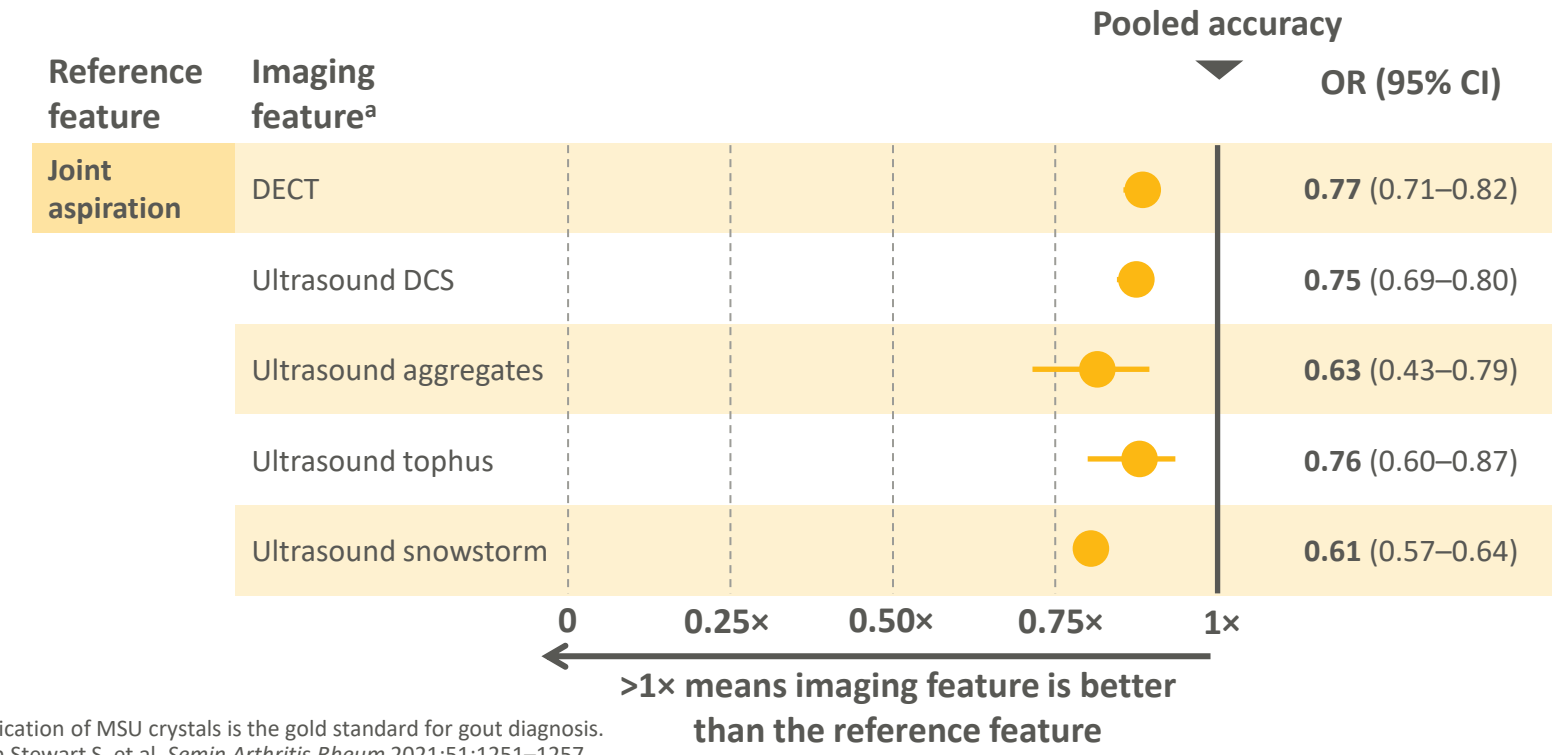
Imaging evidence of gout-related joint damage: Demonstrate ≥1 gout-related erosion, defined as a cortical break with sclerotic margin and overhanging edge[1]

Distal interphalangeal joints and gull-wing appearance are excluded from this evaluation to avoid misdiagnosis with osteoarthritis[1]

[a]Long arrows indicate erosions. Arrowhead indicates preserved joint space. White squares indicate preserved subchondral bone density. Asterisks indicate calcified soft tissue tophi.[2]
Images adapted from Girish G, et al. *Arthritis* 2013;2013:673401 (CC BY 3.0; https://creativecommons.org/licenses/by/3.0/)..
ACR, American College of Rheumatology; DECT, dual-energy computed tomography; EULAR, European Alliance of Associations for Rheumatology; MT, first metatarsal head; MTP, metatarsophalangeal; PP, proximal phalanx.
1. Neogi T, et al. *Ann Rheum Dis* 2015;74:1789–1798; 2. Girish G, et al. *Arthritis* 2013;2013:673401.
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Imaging is Increasingly Used in Clinical Practice for Gout Diagnosis

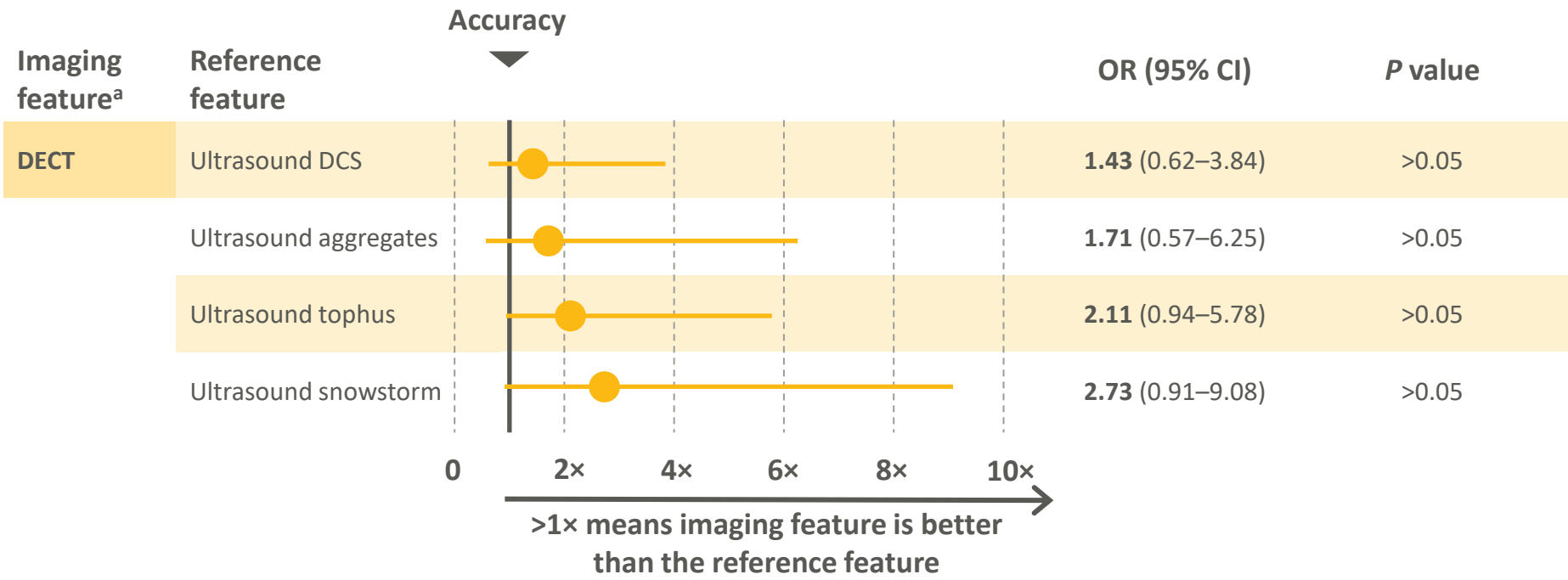
Although the gold standard of joint aspiration is more accurate, it is not always practical



[a]Microscopic identification of MSU crystals is the gold standard for gout diagnosis.
Figure adapted from Stewart S, et al. *Semin Arthritis Rheum* 2021;51:1251–1257.
CI, confidence interval; DCS, double-contour sign; DECT, dual-energy computed tomography; MSU, monosodium urate; OR, odds ratio.
Stewart S, et al. *Semin Arthritis Rheum* 2021;51:1251–1257.
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Both DECT and Ultrasound DCS are Useful Options in the Diagnosis of Gout

There was no significant differences in accuracy between imaging modalities



[a]Traditional direct pairwise meta-analyses were performed to compare different imaging features (≥2 imaging features).
Figure adapted from Stewart S, et al. *Semin Arthritis Rheum* 2021;51:1251–1257.
CI, confidence interval; DCS, double-contour sign; DECT, dual-energy computed tomography; OR, odds ratio.
Stewart S, et al. *Semin Arthritis Rheum* 2021;51:1251–1257.

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Summary

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Summary

Gout is the most common form of inflammatory arthritis, with ~2–6% of patients with gout in the US classified as having uncontrolled gout[1,2]

Gout is caused by hyperuricemia (sUA levels ≥6.8 mg/dL), which can lead to deposition of MSU crystals in joints and tissues[3]

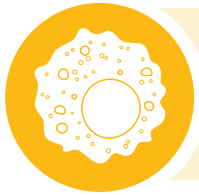
Persistently high sUA levels are associated with unpredictable flares and impact patient HRQoL[4,5]

HRQol, health-related quality of life; MSU, monosodium urate; NLRP3, NLR family pyrin domain containing 3; sUA, serum uric acid; ULT, urate-lowering therapy.

1. Singh JA, Gaffo A. *Semin Arthritis Rheum* 2020;50(3S):S11–S16; 2. Edwards NL. *Arthritis Rheum* 2008;58:2587–2590; 3. Girardet JL, Miner JN. *Annu Rep Med Chem* 2014;49:151–164;
4. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 5. Strand V, et al. *Rheumatol Ther* 2024;11:1271–1290.

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Summary



Gout flares are triggered by macrophage recognition of MSU crystals, leading to NLRP3 inflammasome activation and an acute inflammatory reaction that is self-resolving[1–3]



If MSU crystals remain, tophi can form and cause joint destruction[1]



Gout treatment focuses on relieving acute flares with NSAIDs, colchicine, or corticosteroids, and dissolving MSU crystals through long-term ULT[1]



Both DECT and ultrasound DCS are useful options in the diagnosis of gout[4]

DCS, double-contour sign; DECT, dual-energy computed tomography; MSU, monosodium urate; NSAID, non-steroidal anti-inflammatory drug; ULT, urate-lowering therapy.

1. Dalbeth N, et al. *Lancet* 2021;397:1843–1855; 2. Yang G, et al. *Front Mol Biosci* 2022;9:1027917; 3. Poulsen R, Dalbeth N. *Arthritis Rheumatol* 2025;77:1317–1326; 4. Stewart S, et al. *Semin Arthritis Rheum* 2021;51:1251–1257.

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