UNCONTROLLED GOUT

- Symptomatic gout with serum uric acid (sUA) ≥6 mg/dL despite conventional therapy[1]
- Associated with progressive joint damage and disability[1]
- Management goal: Durable reduction of sUA to <6 mg/dL to reduce gout flares and tophi[1]

Genetics and diet may contribute to gout and comorbidities[2–4]

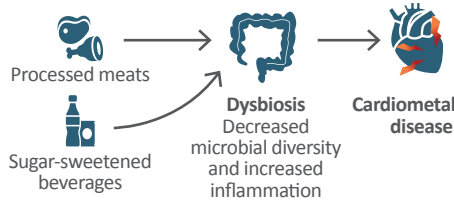
Two separate gene clusters have been associated with sUA and different comorbid traits[3]



Metabolic cluster
sUA + BMI, systolic blood pressure, and blood glucose

CKD cluster
sUA + serum creatinine

Diet can alter the composition of the gut microbiome (dysbiosis), which may contribute to cardiometabolic disease in gout[4,5]

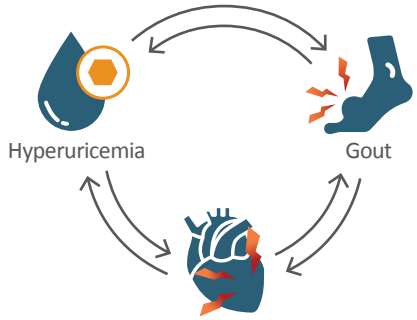


3.13
HAZARD RATIO

A high genetic risk score combined with an unfavorable lifestyle increased risk of developing gout beyond genetics or lifestyle alone[a,2]

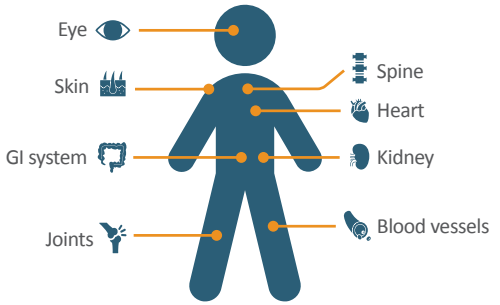
- **High-risk + unfavorable lifestyle**
  HR 3.13 (95% CI 2.79–3.52)
- **High-risk + favorable lifestyle**
  HR 1.98 (95% CI 1.75–2.24)
- **Low-risk + unfavorable lifestyle**
  HR 1.55 (95% CI 1.36–1.77)

Persistent hyperuricemia in uncontrolled gout can be a driver of cardiometabolic disease[6–8]



Comorbidities may reflect systemic, chronic inflammation from urate crystal deposits[9]

Urate crystal deposits have been observed in:[9]



Diabetes

1.20
ODDS RATIO

Patients with uncontrolled gout were 20% more likely to develop diabetes than those with controlled gout[b,8]

OR 1.20 (95% CI 1.06–1.35)

Chronic kidney disease

2.04
ODDS RATIO

Patients with uncontrolled gout were twice as likely to develop chronic kidney disease as those with controlled gout[b,8]

OR 2.04 (95% CI 1.81–2.30)

Cardiovascular disease

1.52
ODDS RATIO

Patients with uncontrolled gout were 52% more likely to develop cardiovascular disease than those with controlled gout[b,8]

OR 1.52 (95% CI 1.35–1.71)

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Footnotes
[a]Population-based cohort study using 416,481 participants of White British descent from the UK Biobank (2006–2021). Lifestyle factors included alcohol consumption, smoking status, physical activity, and diet. Weighted polygenetic risk score was calculated using 33 independent SNPs previously associated with gout.[2]
[b]Retrospective database study (2007–2016) of patients with controlled (sUA <6.0 mg/dL; n=5473; median follow-up 2.7 years) versus uncontrolled gout (sUA ≥8 mg/dL ≥90 days after starting urate-lowering therapy; n=1358; median follow-up 1.9 years). Limitations included inability of claims data to determine causality, no matching of study groups for age or sex, and that successful sUA lowering therapy may be proxy for medical care access.[8]

Abbreviations
BMI, body mass index; CI, confidence interval; CKD, chronic kidney disease; GI, gastrointestinal; HR, hazard ratio; OR, odds ratio; SNP, single nucleotide polymorphism; sUA, serum uric acid.

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