REVIEW

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The association between body mass index and serum uric acid levels: a narrative review

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ABSTRACT

Hyperuricemia represents an escalating global public health challenge, acting as the fundamental precursor to gout and a significant contributor to systemic inflammation, hypertension, and cardiovascular disease. In Indonesia, the prevalence of physician-diagnosed gout is particularly pronounced in regions such as Bali and Buleleng, coinciding with a steady nationwide increase in overweight and obesity rates. Given this parallel epidemiological trend, understanding the precise biological interactions between adiposity and urate metabolism is essential for developing effective preventive strategies. This narrative review aims to synthesize current evidence regarding the epidemiological association and the underlying pathophysiological mechanisms linking Body Mass Index (BMI) to serum uric acid levels. Findings from diverse international cohorts and Indonesian-specific studies consistently demonstrate a significant positive correlation between elevated BMI and serum uric acid across various demographic groups. This association is underpinned by four interrelated biological pathways: adipose-driven insulin resistance that enhances renal urate reabsorption; leptin resistance that impairs urinary urate excretion; upregulated xanthine oxidase activity in adipose tissue that augments endogenous production; and obesity-associated oxidative stress that accelerates cellular purine degradation. These synergistic mechanisms create a metabolic environment conducive to chronic urate accumulation. Consequently, BMI is a critical, modifiable risk factor; integrated weight management strategies, encompassing regular physical activity and targeted dietary modifications that restrict purine-rich foods, fructose, and alcohol, are foundational to the clinical prevention and management of hyperuricemia and its associated metabolic sequelae.

Keywords: body mass index, hyperuricemia, uric acid.
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INTRODUCTION

Uric acid is the final product of purine catabolism in humans, generated from both dietary and endogenously synthesised purines via the xanthine oxidase pathway in the liver, before being excreted renally.[1] Under physiological conditions, circulating uric acid serves as an endogenous antioxidant, scavenging reactive oxygen species.[2] When purine load exceeds renal excretory capacity, serum uric acid accumulates a condition termed hyperuricaemia, defined as serum uric acid exceeding 7.0 mg/dL in males and 6.0 mg/dL in females.[3] Chronic hyperuricaemia precipitates monosodium urate crystal deposition in joints and soft tissues, manifesting as gout, nephrolithiasis, and through systemic inflammation contributing to hypertension and cardiovascular disease.[4] Globally, the WHO estimated that uric acid–related conditions affected 34.2% of the world population in 2017,[5] with the United States reporting approximately 13.[6] cases per 100,000 population.6 In Indonesia, the national prevalence of physician-diagnosed gout among individuals aged ≥15 years was 7.30%, with Bali Province at 10.46% and Buleleng Regency at 12.93% among the highest nationally.[7,8] These figures underscore the need for identifying and addressing modifiable determinants of hyperuricaemia.

BMI calculated as body weight divided by height squared (kg/m^2) is endorsed by the WHO as the most practical epidemiological measure of nutritional status and adiposity.[9] Excess BMI is associated with dyslipidaemia, hypertension, type 2 diabetes mellitus, cardiovascular disease, and elevated serum uric acid.[10,11] National data show a steady rise in overweight prevalence in Indonesia from 8.6% in 2007 to 13.6% in 2018,[12] occurring in parallel with rising hyperuricaemia rates an epidemiological pattern that has intensified interest in the biological link between adiposity and uric acid dysregulation.[13] This review aims to synthesise the evidence on hyperuricaemia epidemiology and pathophysiology, BMI classification and determinants, the epidemiological association between BMI and serum uric acid, and the underlying biological mechanisms.

RESULTS

Uric Acid: Physiology, Pathophysiology, and Etiology of Hyperuricaemia

Uric acid is generated from purine catabolism via sequential oxidation of hypoxanthine and xanthine by xanthine oxidase, ultimately yielding uric acid as the terminal product.[1] In the kidney, approximately 90% of the filtered uric acid load is reabsorbed in the proximal tubule, with only 6–10% excreted in urine.[3] Hyperuricaemia arises from overproduction, underexcretion, or a combination of both.14 When serum uric acid supersaturates, monosodium urate crystals precipitate in joints, triggering NLRP3 inflammasome activation, IL-1β release, and the intense synovitis of acute gouty arthritis.4 Beyond gout, persistent hyperuricaemia is linked to renal stone formation, tubulointerstitial nephropathy, endothelial dysfunction, and accelerated atherosclerosis.[3,4]

Deficiency of the salvage enzyme hypoxanthine-guanine phosphoribosyltransferase (HGPRT) or overactivity of PRPP synthetase leads to excess de novo purine synthesis and uric acid overproduction.[14] Clinically, however, underexcretion driven by impaired renal tubular urate transport — accounts for approximately 90% of hyperuricaemia cases, making renal handling the dominant therapeutic and preventive target.[3,14]

Factors Influencing Serum Uric Acid Levels

Serum uric acid is modulated by both non-modifiable and modifiable factors.[14] Non-modifiable determinants include sex, age, and genetic predisposition. Males have higher baseline uric acid than females because oestrogen promotes renal urate excretion; this protective effect is lost after menopause, explaining why gout predominantly affects men and post-menopausal women.[15,16] With ageing, declining renal function and reduced enzymatic urate clearance progressively raise serum uric acid independently of BMI.[17] Genetic polymorphisms in renal urate transporter genes (URAT1/SLC22A12, GLUT9/SLC2A9, ABCG2) are among the strongest genetic determinants of interindividual variability in serum uric acid.[4]

Modifiable factors include dietary purine intake, alcohol consumption, physical activity, and medications. High-purine foods organ meats, shellfish, red meat, and processed meat extracts directly increase hepatic uric acid synthesis.[15] Alcohol, particularly beer, raises uric acid both by accelerating purine catabolism and by promoting lactic acidosis, which competitively inhibits renal urate secretion.[15] Physical inactivity compounds adiposity-related metabolic dysfunction, while strenuous acute exercise transiently raises uric acid via lactate accumulation and dehydration.[17,18] Medications such as thiazide diuretics, low-dose aspirin, pyrazinamide, and ethambutol reduce renal urate excretion and are significant pharmacological contributors to hyperuricaemia.[3,15]

Body Mass Index: Definition, Classification, and Determinants

BMI is derived by dividing body weight in kilograms by the square of height in metres, and is endorsed by the WHO as the standard epidemiological index for classifying nutritional status.[9,19] The WHO Asia-Pacific classification which applies lower thresholds appropriate for Asian body composition categorises BMI as: underweight (<18.5 kg/m²), normal (18.5–22.9 kg/m²), overweight at risk (23.0–24.9 kg/m²), obese class I (25.0–29.9 kg/m²), and obese class II (≥30.0 kg/m²). 20 While BMI does not directly quantify body fat proportion, it correlates reasonably with adiposity and cardiometabolic risk at the population level.[19]

BMI is shaped by multiple determinants. Age reduces physical activity and increases fat-to-muscle ratio, progressively elevating BMI over the life course.[21] Sex influences fat distribution patterns, with male-pattern central adiposity conferring greater metabolic risk per BMI unit.[21] Dietary habits particularly diets high in refined carbohydrates, saturated fats, and ultra-processed foods and physical inactivity are the principal modifiable drivers of excess BMI, and are therefore primary targets for population-level prevention strategies.[21,22]

Epidemiological Evidence for the Association Between BMI and Serum Uric Acid

The positive association between BMI and serum uric acid has been demonstrated consistently across diverse populations. Wang et al. (2022), analysing NHANES 2013–2018 data (n = 18,473), confirmed a significant positive correlation between BMI and serum uric acid in both males and females across multiple racial and ethnic groups, persisting after adjustment for age and dietary factors.23 Liu et al. (2019), in a large Chinese health check-up cohort (n = 144,856), similarly reported a weak but significant positive correlation in both males and females, with the correlation attenuating with increasing age in men a finding attributed to declining renal function and accumulating comorbidities.[24]

The twin study of Tanaka et al. (2015) provided particularly robust evidence by controlling for shared genetic and environmental confounders among 422 adult twin pairs. Within-pair difference analyses which eliminate shared genetic and environmental variance demonstrated significant positive associations between BMI and serum uric acid in both male and female pairs, confirming that adiposity exerts a genuine biological effect on uric acid metabolism independent of genetic background.[25]

Indonesian studies are consistent with these international findings. Leokuna and Malinti (2020) demonstrated a significant positive correlation between BMI and serum uric acid among Indonesian adults, with males more frequently presenting with elevated uric acid.[26] Kusumawati et al. (2024) and Ningrum et al. (2024) similarly found significant positive correlations in housewives and pre-elderly adults, respectively.[27,28] Sari et al. (2019) reported a significant association among post-menopausal Balinese women, a population in whom loss of oestrogen-mediated uricosuric protection amplifies adiposity-driven hyperuricaemia.[29] Deng et al. (2024) further demonstrated that liver enzyme markers partially mediate the BMI–uric acid relationship through adiposity-driven hepatic dysfunction,[30] while Wang et al. (2025) showed that elevated BMI and hypertension synergistically predict new-onset hyperuricaemia.[31]

Across these studies, correlation coefficients consistently fall in the weak-to-moderate range, reflecting the multifactorial nature of hyperuricaemia. BMI is one of several concurrent determinants, and residual variance attributable to diet, medications, renal function, genetics, and sex attenuates any single predictor's explanatory power. Nonetheless, the universally positive direction of the association replicated across all study populations is the central and clinically actionable epidemiological finding.[23,24,26–28]

Pathophysiological Mechanisms Linking Elevated BMI to Hyperuricaemia

The BMI and uric acid relationship is underpinned by at least four interrelated biological mechanisms (Figure 1). First, excess adipose tissue drives insulin resistance and compensatory hyperinsulinaemia, which directly stimulates renal tubular urate reabsorption via the URAT1 transporter, reducing urinary uric acid excretion.[3,11,32] Studies have confirmed an inverse relationship between insulin sensitivity and 24-hour urinary uric acid clearance, establishing insulin resistance as the principal mechanistic link between obesity and impaired renal urate handling.[11]

Second, obesity elevates circulating leptin an adipokine released by adipose tissue which in lean individuals stimulates renal tubular urate secretion and thereby promotes uricosuria.[33] In overweight individuals, chronic hyperleptinemia leads to leptin resistance at the renal tubule, impairing this uricosuric effect and causing urate accumulation in the bloodstream.[33] Third, xanthine oxidase activity is upregulated in adipose tissue in proportion to fat mass, augmenting uric acid production beyond the liver a mechanism that explains why serum uric acid rises with adiposity even when dietary purine intake is controlled.[34] Fourth, obesity-associated chronic inflammation and oxidative stress driven by excess production of TNF-α, IL-6, and reactive oxygen species from adipose macrophages accelerate cellular nucleic acid degradation, providing additional purine substrate for conversion to uric acid, while also impairing the antioxidant function of physiological uric acid.[4,33]

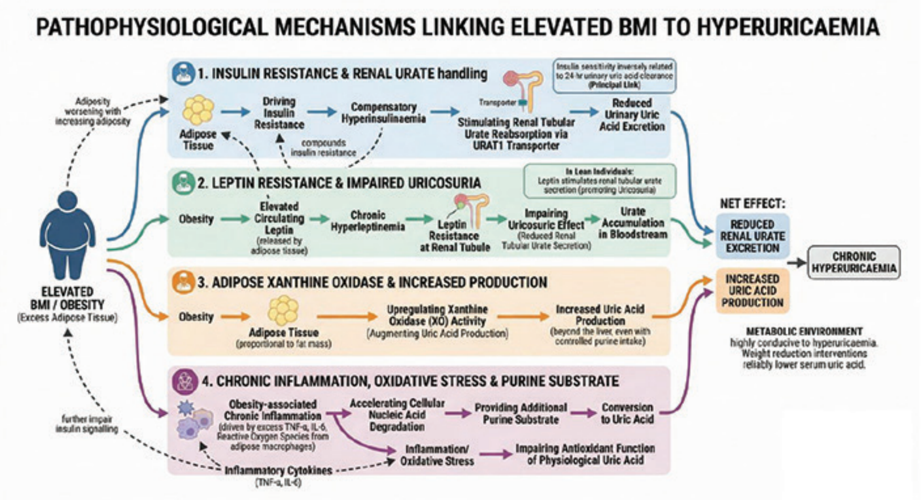

Figure 1. Interrelated pathophysiological mechanism linking elevated BMI to hyperuricemia

These four mechanisms are mutually reinforcing: insulin resistance worsens with increasing adiposity, which elevates leptin, which compounds insulin resistance; adipose xanthine oxidase activity rises with fat mass; and inflammatory cytokines further impair insulin signalling. The net effect reduced renal urate excretion combined with increased uric acid production creates a metabolic environment highly conducive to chronic hyperuricaemia and explains why weight reduction interventions reliably lower serum uric acid.[3,4,33]

CONCLUSION

This narrative review confirms that body mass index is a significant and modifiable risk factor for elevated serum uric acid, with a positive association consistently reported across diverse populations and demographic groups. The relationship is driven by interrelated mechanisms insulin resistance, leptin dysregulation, enhanced xanthine oxidase activity in adipose tissue, and obesity-associated oxidative stress all of which are exacerbated by excess adiposity. To prevent hyperuricaemia and its complications, weight management through dietary modification with restriction of purine-rich foods, fructose, and alcohol, alongside regular physical activity and sustained health education, should be incorporated as foundational strategies in both clinical practice and community health promotion programmes.

CONFLICT OF INTEREST

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AUTHOR'S CONTRIBUTION

All authors contributed equally in the writing process of this article.

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