Unraveling the Potential Role of Probiotics in Ameliorating Hyperuricemia

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ABSTRACT

Hyperuricemia has gained clinical interest due to emerging evidence of its association with various diseases, such as hypertension, diabetes mellitus, cardiovascular disease, chronic kidney disease, and fatty liver. Gut dysbiosis has been suggested to be linked to hyperuricemia, as the gastrointestinal tract is responsible for approximately one-third of the total human uric acid excretion. Alterations in gut microbiota composition have been observed in individuals with hyperuricemia.. Probiotics have regained popularity due to their beneficial health effects, and several studies have shown their potential as a dietary intervention for hyperuricemia. This review aims to examine the intestinal dysbiosis in individuals with hyperuricemia and explore the role of probiotics in ameliorating uric acid levels.

Keywords: *Gut Dysbiosis, Hyperuricemia, Probiotics, Uric Acid*

ABSTRAK

Hiperurisemia menjadi perhatian klinis berdasarkan data yang menunjukkan hubungannya dengan berbagai macam penyakit, antara lain hipertensi, diabetes mellitus, penyakit jantung pembuluh darah, penyakit ginjal kronis, dan perlemakan hati. Disbiosis saluran cerna telah banyak dikaitkan dengan hiperurisemia, karena saluran cerna terlibat dalam sepertiga dari total ekskresi asam urat dalam tubuh manusia. Terdapat perubahan komposisi mikrobiota usus pada subjek dengan hiperurisemia. Probiotik menjadi popular karena memiliki efek yang menguntungkan bagi kesehatan. Beberapa penelitian menunjukkan potensi manfaat dari probiotik sebagai terapi nutrisi pada hiperurisemia. Tinjauan pustaka ini bertujuan untuk menginvestigasi disbiosis saluran cerna pada subjek hiperurisemia dan peranan probiotik dalam memperbaiki kadar asam urat darah.

Kata kunci: *Disbiosis Usus, Hiperurisemia, Probiotik, Asam Urat*

INTRODUCTION

Hyperuricemia has increasingly been recognized as a global public health issue due to its rising prevalence worldwide, ranging from 2.6% to 36% across different countries and showing an age-related increase. Hyperuricemia is defined as a serum uric acid level ≥ 7.0 mg/dL in men and ≥ 6.0 mg/dL in women.[1] It is an inflammatory condition, and emerging evidence has demonstrated a strong association with various diseases, including cardiovascular disease, cerebrovascular diseases, type 2 diabetes mellitus, hypertension, non-alcoholic fatty liver disease, and chronic kidney disease. Nevertheless, hyperuricemia is often overlooked and inadequately managed, resulting in poor outcomes, reduced quality of life, and substantial economic burden.[2-4] Urate-lowering

agents are essential in the treatment of hyperuricemia. However, existing treatment modalities still have notable shortcomings. [5]

The kidney is the primary organ involved in uric acid metabolism; however, the gastrointestinal tract has increasingly been recognized as playing a crucial role in urate homeostasis.[6] Several studies have reported alterations in the richness and diversity of gut microbiota in patients with hyperuricemia, and intestinal dysbiosis has been implicated in the development of gout. Multiple pathways have been hypothesized to underlie this beneficial effect, including modulation of the host inflammatory response through the action of anti-inflammatory cytokines, regulation of uric acid absorption, catabolism, or secretion.[7-9] This finding may open a new avenue for hyperuricemia treatment by targeting probiotics.

This review aimed to elucidate intestinal dysbiosis in hyperuricemia and to evaluate the potential role of probiotics as a modality in alleviating hyperuricemia. A comprehensive search was conducted in PubMed and Google Scholar to identify relevant English-language studies published up to March 31, 2025. The search strategy combined terms related to probiotics (gut microbiota, gut dysbiosis, probiotics, *Lactobacillus*) and hyperuricemia (serum uric acid, hyperuricemia, urate, gout). Both observational and interventional studies were included if they investigated probiotics and reported outcomes related to uric acid levels.

HYPERURICEMIA

Several notable risk factors for hyperuricemia have been widely known, such as genetics, sex, dietary pattern, alcohol consumption, body mass index, certain medications, and diseases [10, 11] Uric acid levels are maintained through a dynamic equilibrium involving the intake of purine-rich food, endogenous urate synthesis, and urate excretion through various routes. Hyperuricemia develops when uric acid intake or production exceeds its excretion; the majority of cases are attributed to underexcretion of uric acid. This leads to elevated uric acid levels in extracellular fluid and various tissues. Hyperuricemia is considered a metabolic disorder and potentially contributes to the development of multiple complications such as chronic kidney disease, kidney stone formation, cardiovascular and cerebrovascular diseases, fatty liver, hypertension, insulin resistance, and diabetes mellitus [5, 6]

Uric acid is the final catabolic product of purine nucleotides. It is primarily synthesized in the liver, intestine, and vascular endothelium. Approximately 70% of uric acid is excreted by the kidneys, with the remainder eliminated via the gastrointestinal tract.[1, 12] Uric acid is a weak diprotic acid, having one dissociable H+ at physiological pH. It exhibits both antioxidant and pro-oxidant properties. At low concentrations, uric acid may protect against oxidative stress, scavenge free radicals, and enhance nitric oxide–mediated vasodilation. In contrast, high levels of uric acid act as a pro-oxidant, inducing vascular endothelial dysfunction and immune dysregulation, which results in an inflammatory state in hyperuricemia. [1, 13] Interestingly, the intestine, which plays a role in urate homeostasis, has also been implicated in hyperuricemia and the inflammatory processes through gut microbiota dysbiosis. Gut dysbiosis can stimulate excessive production of reactive oxygen species, leading to inflammation, disruption of intestinal permeability, and immune dysregulation.[14, 15]

IMMUNE DYSREGULATION IN HYPERURICEMIA

At physiological levels, soluble uric acid is considered to act as a natural inhibitor of certain immune cell functions. It suppresses macrophage production of interleukin (IL)-1β and tumor necrosis factor (TNF)-α by macrophages. It also promotes macrophage polarization toward the M2 phenotype, which has anti-inflammatory properties. In addition, uric acid diminishes β2 integrin and toll-like receptor (TLR) signaling pathways, thereby inhibiting neutrophil migration [4, 16-18.] In contrast, when uric acid levels exceed the saturation point, leading to the formation of monosodium urate crystals, pro-inflammatory effects become predominant.[19] These crystals can act as damage-associated molecular patterns (DAMPs), interact with TLR, and activate both the innate and adaptive immune system, including macrophages, monocytes, neutrophils, natural killer cells, and T cells. This activation subsequently drives the production of multiple pro-inflammatory cytokines, including IL-1β, TNF-α, transforming growth factor (TGF-β), IL-6, and IL-18, as well as chemotactic cytokines such as monocyte chemoattractant protein (MCP)-1 and IL-8. Moreover, urate crystals induce macrophage M1 polarization, enhance $CD8^+$ responses, and promote TH17 proliferation.[4, 16]

URIC ACID REGULATION AND GUT MICROBIOTA

The human gastrointestinal tract harbors a vast and diverse array of microbial species, forming a complex ecological community known as the gut microbiota. Notably, the gastrointestinal tract plays a crucial role in the excretion of uric acid. Accumulating evidence suggests that the gut microbiota is closely linked to uric acid metabolism. Studies have reported differences in gut microbiota composition between healthy individuals and patients with hyperuricemia, including a decrease in alpha diversity and an increase in the *Bacteroidetes*, *Actinobacteria*, and *Firmicutes*.[14,15] An animal study showed that hyperuricemic rats demonstrated altered diversity, characterized by reduced *Bacteroidetes*, *Ruminococcus*, and *Lactobacillus*, and increased *Bacteroides*, *Proteobacteria*, and *Actinobacteria* compared to normal rats [20-22]. There were higher levels of inflammation-related microbiota, upregulation of TLR 2/4/5, and increased expression of inflammatory cytokines, such as tumor necrosis factor (TNF)-α and interleukin (IL)-1β, in the intestines of hyperuricemic mice.[23] A study in China reported altered composition of gut microbiota in children with hyperuricemia, characterized by a decrease in microbiota producing short-chain fatty acids such as *Alistipes*, *Oscillospira*, *Faecalibacterium*, *Parabacteroides*, and *Phascolarctobacterium*.[9] In line with these findings, studies in adults have shown depletion of *Faecalibacterium prausnitzii* and *Bifidobacterium pseudocatenulatum* in patients with gout, along with increased abundance of *Bacteroides caccae* and *Bacteroides xylanisolvens*.[24] Hyperuricemia subjects had a higher Firmicutes-to-Bacteroidetes ratio.[25] Furthermore, a study identified increased abundance of opportunistic pathogens in the faecal microbiome of men with hyperuricemia, including *Bacteroides*, *Erysipelatoclostridium*, *Porphyromonadaceae*, *Rhodococcus*, and *Anaerolineaceae*. Additional fecal metabolite analyses in hyperuricemic subjects revealed elevated levels of acetate, succinate, and glucose, which are involved in the inflammatory process, including the induction of IL-6, IL-8, TNF-α, and IL-1β production, as well as T cell activation [15, 26.] Interestingly, some studies have shown that uric acid–lowering therapy may help alleviate gut dysbiosis. For instance, allopurinol treatment was shown to increase the abundance of *Bifidobacterium* in hyperuricemic rats.[27] The altered gut microbiota in gout subjects could be restored partly in febuxostat-treated subjects.[28]

It has been proposed that the gut microbiota closely interacts with hyperuricemia. Alterations in gut microbiota composition may influence uric acid catabolism through several mechanisms, including the regulation of uric acid catabolism, modulation of uric acid secretion and absorption, enhancement of uric acid excretion, regulation of intestinal barrier permeability, and modulation of gut inflammatory responses.[15,29] *Lactobacillus*, which is depleted in individuals with gout, plays a vital role in reducing purine absorption and producing uric acid-metabolizing enzymes such as allantoinase, uricase, and allantoicase, which break down uric acid into 5-hydroxyisourate, allantoin, allantoate, and ultimately urea.[29-31] Decreased bacteria-producing butyrate in hyperuricemia subjects results in depleted butyrate levels. Butyrate exerts beneficial effects by repairing the intestinal epithelium and stimulating the expression of uric acid transporters. It also lessens monosodium urate-induced production of IL-6, IL-8, and IL-1β.[31] Conversely, in hyperuricemia subjects, enriched *Shigella* would secrete xanthine deaminase, converting xanthine and hypoxanthine, resulting in a higher level of uric acid.[15]

PROBIOTICS AND HYPERURICEMIA

Probiotics, derived from the Ancient Greek term meaning "for life," are live microorganisms that confer health benefits to the host when administered in adequate amounts. They exert beneficial effects by preventing or treating diseases, influencing the balance of the intestinal microbiota, enhancing gut barrier integrity, and supporting the immune system. Different types of probiotics contribute to different functions. In recent years, probiotics have gained growing interest due to their reported benefits in a variety of diseases, including metabolic, rheumatic, and musculoskeletal disorders [32-38]

Recent studies have revealed a promising role of probiotics in the management of hyperuricemia. An animal study demonstrated that administering *Lactobacillus rhamnosus* Fmb14 to hyperuricemic mice for 12 weeks resulted in a 36.8% reduction in serum uric acid levels, a decrease in serum inflammatory cytokines (IL-1β, IL-18, and TNF-α), and improvements in both fecal short-chain fatty acid levels and gut microbiota diversity.[30] Similarly, 15 days of *Limosilactobacillus fermentum* JL-3 administration in mice led to a 31% reduction in serum uric acid compared with controls, accompanied by enhanced gut microbiota diversity and reduced the inflammatory cytokine IL-1β.[39]

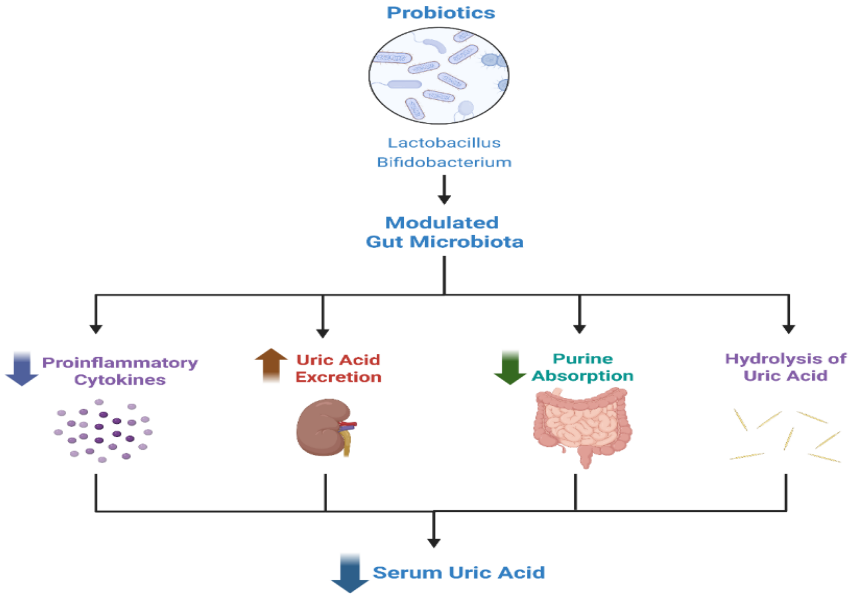

Figure 1. Probiotics and Its' role in Serum Uric Acid Level

A cross-sectional analysis of the National Health and Nutrition Examination Survey (NHANES) from 2011–2018, involving 7,176 participants, demonstrated that probiotic consumption was associated with a protective effect against hyperuricemia, with an adjusted odds ratio (OR) of 0.58 (95% CI: 0.36–0.89); $p = 0.019$).[40] In a randomized, double-blind, controlled trial of 120 volunteers, the effects of consuming probiotic yogurt containing the uric acid–degrading strain *Limosilactobacillus fermentum* GR-3 were compared with conventional yogurt. The study showed that after one month, probiotic yogurt significantly reduced serum uric acid levels (9.65 ± 0.76 vs. 8.35 ± 0.54 μmol/L, $p < 0.0001$), with an even greater reduction observed after two months (9.65 ± 0.76 vs. 7.12 ± 0.22 μmol/L, $p < 0.0001$). In contrast, no significant decrease was observed in the conventional yogurt group. The probiotic intervention also alleviated the production of inflammatory cytokines, including IL-6 and TNF-α, in probiotic yoghurt ($p = 0.0152$ and $p = 0.0020$, respectively). It also significantly increased urinary uric acid excretion compared with conventional yogurt ($p = 0.0009$ at one month and $p = 0.0125$ at two months).[41] Another randomized, double-blind, placebo-controlled trial investigated the effects of 12 weeks of multispecies probiotic supplementation (*Bifidobacterium bifidum* W23, *Bifidobacterium lactis* W51, *Bifidobacterium lactis* W52, *Lactobacillus acidophilus* W37, *Lactobacillus brevis* W63, *Lactobacillus casei* W56, *Lactobacillus salivarius* W24, *Lactococcus lactis* W19, and *Lactococcus lactis* W58) in 81 obese postmenopausal women. High-dose supplementation significantly reduced serum uric acid levels in a dose-dependent manner ($p = 0.0001$).[42] Taken together, the probiotics' strain, dose, and treatment duration varied considerably across studies.

It has been proposed that probiotics regulate the intestinal microbiota and exert their effects through several mechanisms, including downregulation of pro-inflammatory cytokines, facilitation of uric acid hydrolysis, modulation of uric acid gut transporter proteins and intestinal barrier permeability, reduction of purine absorption, and enhancement of uric acid excretion **(Figure 1).**[40, 43-45] However, further clinical trials are required to clarify the specific strains, optimal doses, and underlying mechanisms by which probiotics attenuate serum uric acid levels.

CONCLUSION

Accumulating evidence supports a link between gut microbiota and serum uric acid levels. Various probiotic strains, each with distinct characteristics and potential mechanisms, have demonstrated potential in alleviating hyperuricemia. Hence, probiotics may serve as a promising dietary treatment for individuals with hyperuricemia. Nevertheless, further rigorous, large-scale, and long-term clinical studies are needed to determine the optimal strains, dosages, and treatment regimens.

Conflict of interest

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