Gut microbiome and kidney disease: Targeted therapies for diabetic nephropathy.

Monica Suet*

Department of Nephrology and Hypertension, University of Tokyo, Japan

Introduction

The gut microbiome plays a critical role in maintaining overall health, influencing metabolic processes, immune responses, and even organ function. Emerging research has highlighted the connection between gut dysbiosis and chronic kidney disease (CKD), particularly in individuals with diabetic nephropathy (DN). Diabetic nephropathy, a leading cause of CKD, results from prolonged hyperglycemia and inflammation, leading to kidney damage. Understanding the gut-kidney axis has opened new avenues for targeted therapies, aiming to modulate the microbiome for better kidney health [1].

The gut-kidney axis refers to the intricate relationship between the intestinal microbiota and kidney function. Disruptions in gut microbiota composition, known as dysbiosis, can contribute to systemic inflammation and oxidative stress, both of which are key drivers of diabetic nephropathy. Harmful gut-derived metabolites, such as trimethylamine-N-oxide (TMAO) and indoxyl sulfate, have been linked to renal damage by promoting fibrosis and endothelial dysfunction [2].

Short-chain fatty acids (SCFAs), including butyrate, acetate, and propionate, are essential metabolites produced by gut bacteria through the fermentation of dietary fiber. SCFAs have anti-inflammatory properties and help maintain gut barrier integrity. Studies suggest that reduced SCFA levels in diabetic nephropathy patients contribute to increased intestinal permeability, allowing endotoxins to enter the bloodstream and exacerbate kidney inflammation [3].

Probiotics and prebiotics have gained attention as potential therapeutic interventions for diabetic nephropathy. Probiotics, such as Lactobacillus and Bifidobacterium strains, help restore microbial balance, reduce gut-derived toxins, and improve metabolic health. Prebiotics, which are non-digestible fibers that stimulate beneficial bacteria growth, further enhance SCFA production and lower systemic inflammation, potentially slowing the progression of diabetic nephropathy [4].

Diet plays a crucial role in shaping gut microbiota and influencing kidney health. A plant-based diet rich in fiber promotes beneficial microbial populations, leading to increased SCFA production and reduced inflammation. Conversely, a high-fat, high-protein diet, particularly one rich in red meat, can elevate harmful metabolites like TMAO, accelerating kidney damage. Dietary modifications tailored to improve gut microbiota composition are increasingly being recognized as a complementary strategy in managing diabetic nephropathy [5].

Gut dysbiosis is often associated with metabolic endotoxemia, a condition characterized by elevated levels of bacterial lipopolysaccharides (LPS) in circulation. LPS triggers systemic inflammation, which in turn contributes to insulin resistance and renal dysfunction. Reducing gut permeability through dietary interventions and probiotics may help mitigate LPS-induced damage and slow the progression of diabetic nephropathy [6].

Fecal microbiota transplantation (FMT), which involves transferring gut microbiota from a healthy donor to a recipient, has shown promise in restoring microbial diversity and reducing inflammation. While its application in kidney disease is still in early stages, preliminary studies suggest that FMT may improve insulin sensitivity, lower systemic inflammation, and protect renal function in patients with diabetic nephropathy [7].

Beyond probiotics and prebiotics, postbiotics and synbiotics are gaining traction in nephrology research. Postbiotics refer to bioactive compounds produced by beneficial bacteria that exert health benefits without the need for live bacteria. Synbiotics, a combination of probiotics and prebiotics, offer a synergistic approach to optimizing gut microbiota, reducing uremic toxins, and enhancing kidney function [8].

Several pharmaceutical strategies are being explored to manipulate the gut microbiome in favor of kidney health. Drugs targeting TMAO production, such as inhibitors of gut microbial enzymes responsible for TMA synthesis, are under investigation. Additionally, the use of SCFA supplements and selective antibiotics to eliminate harmful bacteria while preserving beneficial strains represents a promising area of research [9].

Advancements in microbiome research offer exciting possibilities for the management of diabetic nephropathy. Personalized medicine approaches, leveraging microbiome profiling and tailored dietary interventions, may soon become integral to nephrology care. Large-scale clinical trials are needed to validate the efficacy of microbiome-targeted therapies and establish guidelines for their implementation in routine clinical practice [10].

***Correspondence to:** Monica Suet**,** Department of Nephrology and Hypertension, University of Tokyo, Japan. E-mail: monica@suet.jp

***Received:** 2-Feb-2025, Manuscript No. AACNT-25-161906; **Editor assigned:** 3-Feb-2025, PreQC No. AACNT-25-161906(PQ); **Reviewed:** 16-Feb-2025, QC No. AACNT-25-161906; **Revised:** 20-Feb-2025, Manuscript No. AACNT-25-161906(R); **Published:** 27-Feb-2025, DOI: 10.35841/aacnt-9.1.243*

***Citation:** Suet M. Gut microbiome and kidney disease: Targeted therapies for diabetic nephropathy. J Clini Nephrol. 2025; 9(1):243.*

Conclusion

The gut microbiome's influence on kidney health is an emerging field with profound implications for diabetic nephropathy management. Targeted therapies, including probiotics, prebiotics, dietary modifications, and pharmacological interventions, hold great promise in slowing disease progression and improving renal outcomes. As research continues to unravel the complexities of the gut-kidney axis, integrating microbiome-based strategies into standard treatment protocols could revolutionize nephrology care and offer new hope for patients with diabetic nephropathy.

References

1. Neilson EG. Pathogenesis and therapy of interstitial nephritis. Kidney Int. 1989;35(5):1257-70.
2. Muriithi AK, Leung N, Valeri AM, et al. Biopsy-proven acute interstitial nephritis, 1993-2011: a case series. Am J Kidney Dis. 2014;64(4):558-66.
3. Dick GF, Dick GH. The etiology of scarlet fever. J Am Med Assoc. 1924;82(4):301-02.
4. Councilman WT. Acute interstitial nephritis. J Exp Med. 1898;3(5):393-420.
5. Michel DM, Kelly CJ. Acute interstitial nephritis. J Ame Soc Nephrol. 1998;9(3):506-15.
6. Baker RJ, Pusey CD. The changing profile of acute tubulointerstitial nephritis. Nephrol Dial Transplant. 2004;19(1):8-11.
7. Joyce E, Glasner P, Ranganathan S, et al. Tubulointerstitial nephritis: diagnosis, treatment, and monitoring. Pediatr Nephrol. 2017;32(4):577-87.
8. Krishnan N, Perazella MA. Drug-induced acute interstitial nephritis: pathology, pathogenesis, and treatment. IJKD. 2015;9(1).
9. Praga M, González E. Acute interstitial nephritis. Kidney Int. 2010;77(11):956-61.
10. Fogo AB, Lusco MA, Najafian B, et al. AJKD atlas of renal pathology: acute interstitial nephritis. Am J Kidney Dis. 2016;67(6):e35-6.

Citation: *Suet M. Gut microbiome and kidney disease: Targeted therapies for diabetic nephropathy. J Clini Nephrol. 2025; 9(1):243.*