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Gut Microbiota and the Gut-Peritoneal Axis in Peritoneal Dialysis: A Literature Review

(Microbiote Intestinal et l'Axe Intestinal-Péritonéal en Dialyse Péritonéale, Revue de la littérature)

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Summary

The gut microbiota corresponds to the entirety of the microbial flora present in the various sections of the intestine. It is altered in end-stage renal disease on peritoneal dialysis and may play a role in peritoneal physiology and possibly in the prognosis of peritoneal dialysis. Furthermore, until recently, the peritoneal cavity was considered a sterile environment. New studies challenge this dogma and identify a microbiome within this cavity, not only in patients undergoing peritoneal dialysis but also in those with chronic kidney disease, even in the absence of a cavity breach. This review article aims to summarize the existing literature on the potential impact of the gut microbiota and the gut-peritoneal axis on the prognosis of peritoneal dialysis.

Keywords : Gut microbiota, peritoneal microbiota, peritoneal dialysis, peritoneal membrane, peritonitis

Résumé

Le microbiote intestinal correspond à l'ensemble de la flore microbienne présente dans les différentes parties de l'intestin. Celui-ci est altéré dans la maladie rénale terminale en dialyse péritonéale. Il pourrait jouer un rôle dans la physiologie du péritoine et possiblement dans le pronostic de la dialyse péritonéale. Par ailleurs, jusque récemment, la cavité péritonéale était réputée une cavité stérile. De nouvelles études contredisent ce dogme et retrouvent un microbiome en son sein non seulement chez les patients en dialyse péritonéale mais également au stade de la maladie rénale chronique, sans que cette cavité n'ait été effractée. Cet article de revue a pour objectif de décrire la littérature existante sur l'impact potentiel du microbiote intestinal et de l'axe intestinal-péritonéal sur le pronostic de la dialyse péritonéale.

Mots-clés : Microbiote intestinal, microbiote péritonéal, dialyse péritonéale, membrane péritonéale, péritonite

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Introduction

The gut microbiota represents the entirety of the microbial flora present in various sections of the intestine. The microbiome refers to the collective genetic material associated with this microbiota [1]. The gut microbiota plays a critical role in several physiological functions, including metabolic functions, vitamin synthesis (B and K groups), maintenance of the immune system, and modulation of the autonomic nervous system [2–4]. Additionally, through the production of short-chain fatty acids, it contributes to colonocyte survival, regulation of gene expression, intestinal gluconeogenesis (via a cAMP-dependent mechanism), and appetite regulation [5,6]. Healthy gut microbiota also provides defense against pathogenic species through direct mechanisms (competition for resources, modification of partial oxygen pressure, production of toxins or antibiotics, detoxification of antibiotics) and indirect mechanisms (via host immune system stimulation) [5]. The gut microbiota interacts with the host's autonomic nervous system and also plays a role in intestinal motility [7].

Peritoneal dialysis (PD) is an extracorporeal purification technique that uses the peritoneal cavity and peritoneal membrane as a filter to eliminate uremic toxins, regulate ions, and manage fluid balance. This technique requires the placement of a catheter to access the peritoneal cavity for exchange procedures. PD may be complicated by infections at the catheter exit site, subcutaneous tunnel infections, and peritonitis [8–10]. Other non-infectious complications are also encountered, such as peritoneal membrane dysfunction characterized by insufficient ultrafiltration [11]. The pathophysiology and susceptibility factors for these complications remain only partially understood.

The gut microbiota is altered in chronic kidney disease (CKD) and end-stage renal disease, particularly in patients undergoing PD [12–14].

This article aims to review the existing literature on the gut microbiota in patients undergoing peritoneal dialysis and the potential associations between alterations in the gut microbiota and certain infectious and non-infectious complications of PD.

Literature Review

1. General Overview of the Gut Microbiota

The gut microbiota encompasses the entire microbial flora present in the different sections of the intestine. It is composed of 3.8 $x10^{13}$ microorganisms in a 70 kg adult male, corresponding to a total mass of 200 g. The concentration of microorganisms is not uniform throughout the digestive tract, with an anterograde increasing gradient from the stomach to the colon [15,16].

Defining a healthy gut microbiota in adults is challenging due to inter-individual variability. The Human Microbiome Project Consortium studied the composition of the microbiota in 242 disease-free individuals and demonstrated that, unlike highly variable microbial taxa, the metabolic pathways carried by the microbiota are stable over time and across individuals. Therefore, characterizing a healthy microbiota should focus on the functional capacities of the microbiota rather than its taxonomic composition [2,17].

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2. Composition of the Microbiota

Normal Microbiota

The identification of species colonizing the gut microbiota has undergone significant advancements over the past decades with the advent of molecular biology-based technologies [18]. Rajilić-Stojanović et al. described, based on ribosomal RNA sequences, a phylogenetic framework of 1,057 species (92 Eukaryotes, 8 Archaea, and 957 Bacteria) cultured from the human gut microbiota. It was found that the phyla *Actinobacteria*, *Bacteroidetes*, *Firmicutes*, and *Proteobacteria* are the most abundant and diverse. However, there are other bacterial phyla of varying abundance, such as *Verrucomicrobia*, *Lentisphaerae*, *Synergistetes*, *Planctomycetes*, *Tenericutes*, and the *Deinococcus-Thermus* group.

In addition to bacteria, fungi are also present (the mycobiota, primarily composed of *Candida*, *Saccharomyces*, *Malassezia*, and *Cladosporium*), as well as archaea, viruses, and phages [19].

Gut Microbiota in Chronic Kidney Disease

Alterations in gut microbiota composition (dysbiosis) are well documented in CKD and end-stage renal disease, with findings including increased levels of *Alphaproteobacteria*, *Streptococcaceae*, *Streptococcus*, *Blautia*, and *Bacilli*, and decreased levels of *Prevotellaceae*, *Prevotella*, *Firmicutes*, and *Roseburia* [20-22]. With moderate evidence, an increase in *Klebsiella* and *Escherichia-Shigella* has also been observed [23-25].

Gut Microbiota in PD

Dysbiosis is also reported in patients on PD. Stadlbauer V. et al. compared the gut microbiota of 15 hemodialysis (HD) patients, 15 peritoneal dialysis patients, and 21 controls [14]. They observed a decrease in potentially beneficial species and an increase in potentially pathogenic ones in both HD and PD patients compared to controls, with more pronounced changes in HD patients. This taxonomic alteration is associated with changes in metagenomic functionality. For example, the reduction of *Roseburia intestinalis* [14,26], a bacterial genus associated with numerous health benefits [27], may contribute to decreased butyrate synthesis in the intestines [14].

Consistent with findings from CKD studies, PD patients also exhibit reduced levels of *Firmicutes* and *Actinobacteria*, particularly *Bifidobacteriaceae sp*. and *Lactobacillaceae sp*., alongside an overrepresentation of *Enterobacteriaceae sp*. [12-14].

Li J. et al. demonstrated that gut microbiota diversity in patients undergoing continuous ambulatory peritoneal dialysis (CAPD) is reduced compared to non-dialyzed CKD patients and healthy controls [28]. Interestingly, they found that intestinal microbial environments were more favorable in patients on CAPD for a longer duration (60 months vs. 24-36 months), suggesting a capacity for self-regulation and microbiota adaptation over time under stable conditions. The relationship between residual renal function and protection against intestinal dysbiosis may partially be explained by a less aggressive peritoneal dialysis strategy [29].

Several studies indicate that *Helicobacter pylori* infection is less frequent in dialysis patients compared to those with preserved renal function [30,31]. Furthermore, CAPD patients reportedly have lower infection rates compared to HD patients [30]. A meta-analysis showed an inverse correlation between dialysis duration (both HD and CAPD) and *Helicobacter pylori* infection, despite limited data specifically comparing PD to HD [32]. The causes for this reduction remain unclear but may involve the unfavorable uremic conditions for infection development.

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Several factors have been implicated in intestinal dysbiosis in PD patients [33], including a phosphorus- and potassium-restricted diet that reduces fiber and symbiotic intake, uremic toxins such as urea that alter gut flora composition [34], medications like potassium binders, and finally, the PD technique itself [28].

Peritoneal dialysis has several unique features related to gut microbiota that distinguish it from other of end-stage renal disease treatment modalities. These include intraperitoneal hyperglycemia and elevated intra-abdominal pressure, which promote specific microbiota changes such as increased *Enterobacteriaceae*. Additionally, preservation of residual diuresis partially mitigates the deleterious effects of dysbiosis. Finally, the progressive adaptation of gut microbiota in long-term PD patients highlights a unique adjustment mechanism in this technique. These specificities strengthen the hypothesis of a close interaction between gut microbiota and peritoneal physiology in PD.

3. Association with PD Complications

3.1. Non-infectious Complications

Microbiota and Peritoneal Fibrosis

Peritoneal sclerosis is a severe complication associated with ultrafiltration failure and solute transport abnormalities. It is characterized by morphological alterations of the peritoneal membrane, including interstitial fibrosis and hyalinizing vasculopathy [35]. This condition has long been attributed to the lack of biocompatibility of the dialysis solution on the peritoneal membrane, involving factors such as high osmolarity, low pH, high glucose concentrations, advanced glycation end-products (AGEs), and glucose degradation products. These agents trigger degenerative damage to the tissue components of the peritoneal membrane and induce abnormal biological responses [36].

However, recent evidence suggests that factors independent of PD, such as uremia, may play a significant role in the pathogenesis of peritoneal sclerosis even before the initiation of PD [37]. These findings broaden our understanding of peritoneal sclerosis, suggesting that this complication could result from multiple mechanisms involving both PD-related factors and pre-existing conditions.

An emerging area of interest is the role of gut microbiota in the progression of PD-related complications. A prospective cohort study demonstrated that reduced gut microbiota diversity is associated with an increased risk of technique failure in PD patients. This association persists even after adjusting for various risk factors, including age, coronary artery disease history, diabetes, statin use, and levels of albumin, phosphorus, NT-proBNP, and glucose. Patients with lower gut microbiota diversity had significantly poorer PD technique survival compared to those with more diverse microbiota (HR, 2.038; 95% CI, 1.057–3.929; p = 0.034) [38].

Guo et al.'s study revealed a significant enrichment of *Escherichia-Shigella* in PD patients with reduced gut microbiota diversity compared to those with greater microbial diversity. This bacterial genus, known to include opportunistic pro-inflammatory pathogens in the intestinal tract, has been associated with elevated levels of both systemic and local pro-inflammatory cytokines [39,40]. Additionally, *Escherichia-Shigella* increases intestinal permeability by elevating

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luminal lipopolysaccharide (LPS) levels, facilitating the translocation of pathogens and harmful metabolites into systemic circulation, thereby contributing to systemic inflammation [41,42]. In PD patients, this chronic inflammation—both local and systemic—can stimulate peritoneal angiogenesis and fibrosis, exacerbating ultrafiltration failure, increasing cardiovascular event risks, and leading to discontinuation of PD therapy or even death [43,44].

The interplay between reduced microbial diversity and poor PD technique survival may, therefore, be partly explained by alterations in gut microbiota. Chronic inflammatory responses induced by the relative expansion of *Escherichia-Shigella* seem to play a key role in the association between gut dysbiosis and PD technique failure.

Gut dysbiosis, defined as a disruption of the normal gut microbiota composition, is increasingly recognized as a key factor in the pathogenesis of various chronic diseases, including obesity [45], insulin resistance, diabetes [46], and CKD [13]. In the context of PD, low microbial diversity has also been correlated with metabolic imbalances, such as increased triglycerides and decreased HDL-C, both of which are established cardiovascular risk factors [47,48].

Additionally, Enterobacteriaceae have been implicated in the production of uremic toxins, such as indole and p-cresol, whose elevated levels can exacerbate various diseases by inducing oxidative stress and increasing pro-inflammatory cytokine production [49,50]. Residual renal function (RRF) may play a protective role by eliminating these toxins, suggesting a potential interaction between RRF and protection against gut dysbiosis in PD patients.

Gut dysbiosis is thus a potentially important mechanism in the onset and progression of numerous diseases, including renal, metabolic, and inflammatory conditions. In PD patients, enrichment of uremic toxin-producing bacteria and depletion of short-chain fatty acid-producing bacteria can disrupt the intestinal barrier, leading to the translocation of microorganisms and their toxins into the bloodstream, thereby exacerbating peritoneal complications [51,52].

Although studies on PD patients are limited, research on hemodialysis patients has shown that altered gut microbiota is a major source of uremic toxins not cleared by the kidneys [53]. Consequently, it is plausible that the gut microbiota in PD patients is also a significant source of toxic metabolites, thereby contributing to fibrosis in organs, including the heart, kidneys, and peritoneum [35].

The accumulation of bacterial structural components, their metabolites, and uremic toxins significantly impacts fibrosis development, suggesting that uremia is a globally pro-fibrotic condition [54,55]. This condition may activate various signaling pathways promoting epithelial-to-mesenchymal transition and extracellular matrix production by mesenchymal cells [55-57].

In summary, gut dysbiosis associated with CKD and PD creates a vicious cycle where chronic inflammation—both intraperitoneal and systemic—and the adverse effects of glucose-based solutions contribute to peritoneal fibrosis. This complex interaction between gut microbiota and PD underscores the importance of microbiota-targeted strategies to prevent or mitigate PD-related complications [53].

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Effects of Dysbiosis on Cognition

PD is a home-based treatment requiring self-management and operation by patients. Consequently, cognitive function is particularly important in PD patients, as any impairment could lead to adverse events [58]. In elderly patients with cognitive disorders, the loss of executive or memory functions may result in errors in PD management, increasing the risk of PD-associated peritonitis [59]. Additionally, cognitive impairment is an independent predictor of mortality and survival in PD patients [60]. Recently, growing evidence has demonstrated a strong link between gut dysbiosis and the onset and progression of nervous system disorders, with the underlying mechanism potentially involving the «microbiota-gut-brain axis» [61]. This association has been reported in various conditions, including end-stage CKD [62] and in HD patients [63]. Very recently, this link was also confirmed in PD patients [64], suggesting a negative impact of gut dysbiosis on the progression of cognitive impairment in peritoneal dialysis patients.

Nutritional Status and Gut Microbiota

Malnutrition is a common complication among dialysis patients, both in HD and PD. It results from a complex mechanism involving multiple factors, including patient-related ones such as loss of residual renal function, intestinal dysfunction [65], and inadequate nutritional intake [66], as well as PD-related factors, such as the inefficiency of dialysis, protein loss with PD effluent, and chronic microinflammation [67]. Malnutrition is also a key factor affecting the quality of life and prognosis of patients [68]. Malnourished PD patients experience higher hospitalization rates, longer hospital stays, and significantly increased risks of peritonitis and mortality [69]. Therefore, malnutrition in these patients deserves heightened attention in clinical practice. Recent studies point to a possible imbalance in the gut microbiota of malnourished PD patients [70].

3.2. Infectious Complications

Recent studies reveal the presence of microbiomes in body sites previously considered sterile, such as the peritoneum [13]. As previously mentioned, gut dysbiosis can disrupt the intestinal barrier in PD patients, favoring «atopobiosis,» defined as the translocation of enteric organisms or bacterial toxins into the peritoneal cavity through the intestinal epithelial barrier. This mechanism may represent an underestimated cause of peritonitis in PD patients. The detection of bacterial DNA in PD effluent supports the hypothesis of atopobiosis in PD patients [71,72]. Thus, the gut microbiome may represent a novel pathway for PD-associated infections, warranting further research. Nevertheless, to date, no study has definitively attributed PD-associated peritonitis to dysbiosis.

4. Therapeutic Perspectives – The Role of Gut Microbiota Modulation

Numerous factors can potentially influence the gut microbiota of PD patients, including antibiotic use, rural or urban environment, diet, physical activity, alcohol, and tobacco. Genetics appears to play a minor role in gut microbiota composition [73,74].

Physical exercise has been associated with an improvement in gut bacterial profiles [75,76]. In an interventional study of insulin-resistant subjects [77], regular physical activity was associated with reduced endotoxemia via decreased markers of intestinal inflammation, as well as changes in gut microbiota favoring protective bacterial genera (reduced *Firmicutes*/*Bacteroidetes* ratio, reduced *Clostridium* and *Blautia*, and increased *Bacteroidetes* genera).

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In numerous studies, the role of diet in modulating gut microbiota and its metabolic capacities has been extensively documented [76,78–85].

Probiotics represent another promising intervention for positively modulating the gut microbiota of CKD patients. In the specific context of PD, Wang I-K et al. demonstrated in a double-blind, randomized controlled trial that administering probiotics over six months to PD patients (21 in the probiotic group and 18 in the placebo group) significantly reduced blood levels of several inflammatory markers (TNF-α, IL-5, IL-6) and endotoxins, significantly increased IL-10 (anti-inflammatory), and preserved residual renal function [86].

5. Conclusions and Recommendations

Gut dysbiosis is well-documented in PD, but the literature lacks studies evaluating the clinical implications of dysbiosis in this context. Dysbiosis appears to be associated with reduced PD technique survival and may represent a risk factor for infectious complications through the phenomenon of atopobiosis.

The gut microbiota and the gut-peritoneal axis could thus represent a promising research avenue in PD, with potential clinical implications for preventing both infectious and non-infectious complications.

Currently, professional societies do not recommend modulating the gut microbiota in PD patients.. However, the literature suggests a potential beneficial effect of such interventions. The clinical impact of each taxonomic modification of the microbiota remains to be fully investigated.

Authors' Contributions

Maxime Taghavi and Lucas Jacobs designed the project. Lucas Jacobs, Laura Mannie-Corbisier, and Maxime Taghavi jointly drafted the manuscript, while Maxime Taghavi and Joëlle Nortier supervised the writing of the article.

Ethical Considerations

Not applicable.

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Conflicts of Interest

The authors declare no conflicts of interest in relation to this article.

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References

1. Fan Y, Pedersen O. Gut microbiota in human metabolic health and disease. Nat Rev Microbiol. 2021;19:55–71.
2. Huttenhower C, Gevers D, Knight R, Abubucker S, Badger JH, Chinwalla AT, et al. Structure, function and diversity of the healthy human microbiome. Nature. 2012;486:207–14.
3. Magnúsdóttir S, Ravcheev D, de Crécy-Lagard V, Thiele I. Systematic genome assessment of B-vitamin biosynthesis suggests co-operation among gut microbes. Front Genet. 2015;6:148.
4. Kho ZY, Lal SK. The Human Gut Microbiome - A Potential Controller of Wellness and Disease. Front Microbiol. 2018;9:1835.
5. Rowland I, Gibson G, Heinken A, Scott K, Swann J, Thiele I, et al. Gut microbiota functions: metabolism of nutrients and other food components. Eur J Nutr. 2018;57:1–24.
6. Parada Venegas D, De la Fuente MK, Landskron G, González MJ, Quera R, Dijkstra G, et al. Short Chain Fatty Acids (SCFAs)-Mediated Gut Epithelial and Immune Regulation and Its Relevance for Inflammatory Bowel Diseases. Front Immunol. 2019;10:277.
7. Anitha M, Vijay–Kumar M, Sitaraman SV, Gewirtz AT, Srinivasan S. Gut Microbial Products Regulate Murine Gastrointestinal Motility via Toll-Like Receptor 4 Signaling. Gastroenterology. 2012;143:1006-1016.e4.
8. Li PK-T, Chow KM, Cho Y, Fan S, Figueiredo AE, Harris T, et al. ISPD peritonitis guideline recommendations: 2022 update on prevention and treatment. Perit Dial Int. 2022;42:110–53.
9. Taghavi M, Dratwa M. Overview of ISPD 2022 guideline recommendations for peritonitis prevention and treatment. Bulletin de la Dialyse à Domicile. 2022;5:93–103.
10. Chow KM, Li PK-T, Cho Y, Abu-Alfa A, Bavanandan S, Brown EA, et al. ISPD Catheter-related Infection Recommendations: 2023 Update. Perit Dial Int. 2023;43:201–19.
11. Morelle J, Stachowska-Pietka J, Öberg C, Gadola L, La Milia V, Yu Z, et al. ISPD recommendations for the evaluation of peritoneal membrane dysfunction in adults: Classification, measurement, interpretation and rationale for intervention. Perit Dial Int. 2021;41:352–72.
12. Sampaio-Maia B, Simões-Silva L, Pestana M, Araujo R, Soares-Silva IJ. The Role of the Gut Microbiome on Chronic Kidney Disease. Adv Appl Microbiol. 2016;96:65–94.
13. Simões-Silva L, Araujo R, Pestana M, Soares-Silva I, Sampaio-Maia B. The microbiome in chronic kidney disease patients undergoing hemodialysis and peritoneal dialysis. Pharmacol Res. 2018;130:143–51.
14. Stadlbauer V, Horvath A, Ribitsch W, Schmerböck B, Schilcher G, Lemesch S, et al. Structural and functional differences in gut microbiome composition in patients undergoing haemodialysis or peritoneal dialysis. Sci Rep. 2017;7:15601.
15. Sender R, Fuchs S, Milo R. Revised Estimates for the Number of Human and Bacteria Cells in the Body. PLOS Biology. 2016;14:e1002533.
16. Adak A, Khan MR. An insight into gut microbiota and its functionalities. Cell Mol Life Sci. 2019;76:473–93.
17. Greenhalgh K, Meyer KM, Aagaard KM, Wilmes P. The human gut microbiome in health: establishment and resilience of microbiota over a lifetime. Environmental Microbiology. 2016;18:2103–16.
18. Rajilić-Stojanović M, de Vos WM. The first 1000 cultured species of the human gastrointestinal microbiota. FEMS Microbiol Rev. 2014;38:996–1047.
19. Hou K, Wu Z-X, Chen X-Y, Wang J-Q, Zhang D, Xiao C, et al. Microbiota in health and diseases. Sig Transduct Target Ther. 2022;7:1–28.

journal officiel du Registre de Dialyse Péritonéale de Langue Française **RDPLF** www.rdplf.org

https://doi.org/10.25796/bdd.v8i1.86213
ISSN 2607-9917

20. Voroneanu L, Burlacu A, Brinza C, Covic A, Balan GG, Nistor I, et al. Gut Microbiota in Chronic Kidney Disease: From Composition to Modulation towards Better Outcomes-A Systematic Review. J Clin Med. 2023;12:1948.
21. Ren Z, Fan Y, Li A, Shen Q, Wu J, Ren L, et al. Alterations of the Human Gut Microbiome in Chronic Kidney Disease. Adv Sci (Weinh). 2020;7:2001936.
22. Durand P-Y, Nicco C, Serteyn D, Attaf D, Edeas M. Microbiota Quality and Mitochondrial Activity Link with Occurrence of Muscle Cramps in Hemodialysis Patients using Citrate Dialysate: A Pilot Study. Blood Purification. 2018;46:301–8.
23. Huang H-W, Chen M-J. Exploring the Preventive and Therapeutic Mechanisms of Probiotics in Chronic Kidney Disease through the Gut–Kidney Axis. J Agric Food Chem. 2024;72:8347–64.
24. Huang Y, Xin W, Xiong J, Yao M, Zhang B, Zhao J. The Intestinal Microbiota and Metabolites in the Gut-Kidney-Heart Axis of Chronic Kidney Disease. Front Pharmacol. 2022 Mar 18;13:837500.
25. Stanford J, Charlton K, Stefoska-Needham A, Ibrahim R, Lambert K. The gut microbiota profile of adults with kidney disease and kidney stones: a systematic review of the literature. BMC Nephrology. 2020;21:215.
26. Gao Q, Li D, Wang Y, Zhao C, Li M, Xiao J, et al. Analysis of intestinal flora and cognitive function in maintenance hemodialysis patients using combined 16S ribosome DNA and shotgun metagenome sequencing. Aging Clin Exp Res. 2024;36:28.
27. Tamanai-Shacoori Z, Smida I, Bousarghin L, Loreal O, Meuric V, Fong SB, et al. Roseburia spp.: a marker of health? Future Microbiol. 2017;12:157–70.
28. Li J, Xing H, Lin W, Yu H, Yang B, Jiang C, et al. Specific gut microbiome and metabolome changes in patients with continuous ambulatory peritoneal dialysis and comparison between patients with different dialysis vintages. Front Med (Lausanne). 2023;10:1302352.
29. Lee Y, Chung SW, Park S, Ryu H, Lee H, Kim DK, et al. Incremental Peritoneal Dialysis May be Beneficial for Preserving Residual Renal Function Compared to Full-dose Peritoneal Dialysis. Sci Rep. 2019;9:10105.
30. Sugimoto M, Yasuda H, Andoh A. Nutrition status and Helicobacter pylori infection in patients receiving hemodialysis. World J Gastroenterol. 2018;24:1591–600.
31. Korucu B, Helvaci O, Sadioglu R, Ozbas B, Yeter H, Derici U. Can Helicobacter pylori Colonization Affect the Phosphate Binder Pill Burden in Dialysis Patients? Ther Apher Dial. 2020;24:380–6.
32. Li K-J, Chen L. Association between duration of dialysis and Helicobacter pylori infection in dialysis patients: a meta-analysis. Int Urol Nephrol. 2019;51:1361–70.
33. Lambert K, Rinninella E, Biruete A, Sumida K, Stanford J, Raoul P, et al. Targeting the Gut Microbiota in Kidney Disease: The Future in Renal Nutrition and Metabolism. Journal of Renal Nutrition. 2023;33:S30–9.
34. Amini Khiabani S, Asgharzadeh M, Samadi Kafil H. Chronic kidney disease and gut microbiota. Heliyon. 2023;9:e18991.
35. Williams JD, Craig KJ, Topley N, Von Ruhland C, Fallon M, Newman GR, et al. Morphologic changes in the peritoneal membrane of patients with renal disease. J Am Soc Nephrol. 2002;13:470–9.
36. Margetts PJ, Bonniaud P. Basic mechanisms and clinical implications of peritoneal fibrosis. Perit Dial Int. 2003;23:530–41.
37. Sherif AM, Nakayama M, Maruyama Y, Yoshida H, Yamamoto H, Yokoyama K, et al. Quantitative assessment of the peritoneal vessel density and vasculopathy in CAPD patients. Nephrol Dial Transplant. 2006;21:1675–81.
38. Guo S, Wu H, Ji J, Sun Z, Xiang B, Wu W, et al. Association between gut microbial diversity and technique failure in peritoneal dialysis patients. Ren Fail. 2023;45:2195014.
39. Zhu Y, He C, Li X, Cai Y, Hu J, Liao Y, et al. Gut microbiota dysbiosis worsens the severity of acute pancreatitis in patients and mice. J Gastroenterol. 2019;54:347–58.

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https://doi.org/10.25796/bdd.v8i1.86213
ISSN 2607-9917

40. Cattaneo A, Cattane N, Galluzzi S, Provasi S, Lopizzo N, Festari C, et al. Association of brain amyloidosis with pro-inflammatory gut bacterial taxa and peripheral inflammation markers in cognitively impaired elderly. Neurobiol Aging. 2017;49:60–8.
41. Zheng Q, Chen Y, Zhai Y, Meng L, Liu H, Tian H, et al. Gut Dysbiosis Is Associated With the Severity of Cryptogenic Stroke and Enhanced Systemic Inflammatory Response. Front Immunol. 2022;13:836820.
42. Mohr AE, Crawford M, Jasbi P, Fessler S, Sweazea KL. Lipopolysaccharide and the gut microbiota: considering structural variation. FEBS Lett. 2022;596:849–75.
43. de Lima SMA, Otoni A, Sabino A de P, Dusse LMS, Gomes KB, Pinto SWL, et al. Inflammation, neoangiogenesis and fibrosis in peritoneal dialysis. Clin Chim Acta. 2013;421:46–50.
44. Shi J, Yu M, Sheng M. Angiogenesis and Inflammation in Peritoneal Dialysis: The Role of Adipocytes. Kidney Blood Press Res. 2017;42:209–19.
45. Le Chatelier E, Nielsen T, Qin J, Prifti E, Hildebrand F, Falony G, et al. Richness of human gut microbiome correlates with metabolic markers. Nature. 2013;500:541–6.
46. Brown CT, Davis-Richardson AG, Giongo A, Gano KA, Crabb DB, Mukherjee N, et al. Gut microbiome metagenomics analysis suggests a functional model for the development of autoimmunity for type 1 diabetes. PLoS One. 2011;6:e25792.
47. Vojinovic D, Radjabzadeh D, Kurilshikov A, Amin N, Wijmenga C, Franke L, et al. Relationship between gut microbiota and circulating metabolites in population-based cohorts. Nat Commun. 2019;10:5813.
48. Fu J, Bonder MJ, Cenit MC, Tigchelaar EF, Maatman A, Dekens JAM, et al. The Gut Microbiome Contributes to a Substantial Proportion of the Variation in Blood Lipids. Circ Res. 2015;117:817–24.
49. Mishima E, Fukuda S, Mukawa C, Yuri A, Kanemitsu Y, Matsumoto Y, et al. Evaluation of the impact of gut microbiota on uremic solute accumulation by a CE-TOFMS-based metabolomics approach. Kidney Int. 2017;92:634–45.
50. Gryp T, Huys GRB, Joossens M, Van Biesen W, Glorieux G, Vaneechoutte M. Isolation and Quantification of Uremic Toxin Precursor-Generating Gut Bacteria in Chronic Kidney Disease Patients. Int J Mol Sci. 2020;21:1986.
51. Graboski AL, Redinbo MR. Gut-Derived Protein-Bound Uremic Toxins. Toxins. 2020;12:590.
52. Wang X, Yang S, Li S, Zhao L, Hao Y, Qin J, et al. Aberrant gut microbiota alters host metabolome and impacts renal failure in humans and rodents. Gut. 2020;69:2131–42.
53. Stepanova N. The Gut-Peritoneum Axis in Peritoneal Dialysis and Peritoneal Fibrosis. Kidney Med. 2023;5:100645.
54. Mutsaers HAM, Stribos EGD, Glorieux G, Vanholder R, Olinga P. Chronic Kidney Disease and Fibrosis: The Role of Uremic Retention Solutes. Front Med (Lausanne). 2015;2:60.
55. Zhang L, Xie F, Tang H, Zhang X, Hu J, Zhong X, et al. Gut microbial metabolite TMAO increases peritoneal inflammation and peritonitis risk in peritoneal dialysis patients. Transl Res. 2022;240:50–63.
56. Lee G, You HJ, Bajaj JS, Joo SK, Yu J, Park S, et al. Distinct signatures of gut microbiome and metabolites associated with significant fibrosis in non-obese NAFLD. Nat Commun. 2020;11:4982.
57. Tan J-Y, Tang Y-C, Huang J. Gut Microbiota and Lung Injury. Adv Exp Med Biol. 2020;1238:55–72.
58. Jacobs L, Clevenbergh P, Collart F, Brayer I, Mesquita M, Taghavi M, et al. [Negative impact of COVID-19 pandemic on peritonitis rate in peritoneal dialysis patients: Pleading for a continuous educational training]. Nephrol Ther. 2022;18:526–33.
59. Liao J-L, Zhang Y-H, Xiong Z-B, Hao L, Liu G-L, Ren Y-P, et al. The Association of Cognitive Impairment with Peritoneal Dialysis-Related Peritonitis. Perit Dial Int. 2019;39:229–35.
60. Griva K, Stygall J, Hankins M, Davenport A, Harrison M, Newman SP. Cognitive impairment and 7-year mortality in dialysis patients. Am J Kidney Dis. 2010;56:693–703.
61. Yang T, Richards EM, Pepine CJ, Raizada MK. The gut microbiota and the brain-gut-kidney axis in hypertension and chronic kidney disease. Nat Rev Nephrol. 2018;14:442–56.

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ISSN 2607-9917

62. Wang YF, Zheng LJ, Liu Y, Ye YB, Luo S, Lu GM, et al. The gut microbiota-inflammation-brain axis in end-stage renal disease: perspectives from default mode network. Theranostics. 2019;9:8171–81.
63. Zhu B, Shen J, Jiang R, Jin L, Zhan G, Liu J, et al. Abnormalities in gut microbiota and serum metabolites in hemodialysis patients with mild cognitive decline: a single-center observational study. Psychopharmacology (Berl). 2020;237:2739–52.
64. Wang J, Wu S, Zhang J, Li Y, Wu Y, Qi X. Correlation between gut microbiome and cognitive impairment in patients undergoing peritoneal dialysis. BMC Nephrol. 2023;24:360.
65. Lameire N, Van Biesen W. Epidemiology of peritoneal dialysis: a story of believers and nonbelievers. Nat Rev Nephrol. 2010;6:75–82.
66. Zha Y, Qian Q. Protein Nutrition and Malnutrition in CKD and ESRD. Nutrients. 2017;9:208.
67. Martín-del-Campo F, Batis-Ruvalcaba C, González-Espinoza L, Rojas-Campos E, Angel JR, Ruiz N, et al. Dietary micronutrient intake in peritoneal dialysis patients: relationship with nutrition and inflammation status. Perit Dial Int. 2012;32:183–91.
68. Szeto C-C, Kwan BC-H, Chow K-M, Law M-C, Li PK-T. Geriatric Nutritional Risk Index as a Screening Tool for Malnutrition in Patients on Chronic Peritoneal Dialysis. Journal of Renal Nutrition. 2010;20:29–37.
69. Müller M, Dahdal S, Saffarini M, Uehlinger D, Arampatzis S. Evaluation of Nutrition Risk Screening Score 2002 (NRS) assessment in hospitalized chronic kidney disease patient. PLOS ONE. 2019;14:e0211200.
70. Tian N, Yan Y, Chen N, Xu S, Chu R, Wang M, et al. Relationship between gut microbiota and nutritional status in patients on peritoneal dialysis. Sci Rep. 2023;13:1572.
71. Schindler R, Beck W, Deppisch R, Aussieker M, Wilde A, Göhl H, et al. Short bacterial DNA fragments: detection in dialysate and induction of cytokines. J Am Soc Nephrol. 2004;15:3207–14.
72. Szeto C-C, Lai K-B, Kwan BC-H, Chow K-M, Leung C-B, Law M-C, et al. Bacteria-derived DNA fragment in peritoneal dialysis effluent as a predictor of relapsing peritonitis. Clin J Am Soc Nephrol. 2013;8:1935–41.
73. Rothschild D, Weissbrod O, Barkan E, Kurilshikov A, Korem T, Zeevi D, et al. Environment dominates over host genetics in shaping human gut microbiota. Nature. 2018;555:210–5.
74. Ji J, Jin W, Liu S-J, Jiao Z, Li X. Probiotics, prebiotics, and postbiotics in health and disease. MedComm. 2023;4:e420.
75. Wegierska AE, Charitos IA, Topi S, Potenza MA, Montagnani M, Santacroce L. The Connection Between Physical Exercise and Gut Microbiota: Implications for Competitive Sports Athletes. Sports Med. 2022;52:2355–69.
76. Campaniello D, Corbo MR, Sinigaglia M, Speranza B, Racioppo A, Altieri C, et al. How Diet and Physical Activity Modulate Gut Microbiota: Evidence, and Perspectives. Nutrients. 2022;14:2456.
77. Motiani KK, Collado MC, Eskelinen J-J, Virtanen KA, Löyttyniemi E, Salminen S, et al. Exercise Training Modulates Gut Microbiota Profile and Improves Endotoxemia. Med Sci Sports Exerc. 2020;52:94–104.
78. Wu GD, Chen J, Hoffmann C, Bittinger K, Chen Y-Y, Keilbaugh SA, et al. Linking long-term dietary patterns with gut microbial enterotypes. Science. 2011;334:105–8.
79. Muegge BD, Kuczynski J, Knights D, Clemente JC, González A, Fontana L, et al. Diet drives convergence in gut microbiome functions across mammalian phylogeny and within humans. Science. 2011;332:970–4.
80. David LA, Maurice CF, Carmody RN, Gootenberg DB, Button JE, Wolfe BE, et al. Diet rapidly and reproducibly alters the human gut microbiome. Nature. 2014;505:559–63.
81. Singh RK, Chang H-W, Yan D, Lee KM, Ucmak D, Wong K, et al. Influence of diet on the gut microbiome and implications for human health. J Transl Med. 2017;15:73.
82. Travis AC, Katz PO, Kane SV. Mentoring in gastroenterology. Am J Gastroenterol. 2010;105:970–2.
83. Wastyk HC, Fragiadakis GK, Perelman D, Dahan D, Merrill BD, Yu FB, et al. Gut-microbiota-targeted diets modulate human immune status. Cell. 2021;184:4137-4153.e14.

https://doi.org/10.25796/bdd.v8i1.86213
ISSN 2607-9917

84. Suez J, Korem T, Zeevi D, Zilberman-Schapira G, Thaiss CA, Maza O, et al. Artificial sweeteners induce glucose intolerance by altering the gut microbiota. Nature. 2014;514:181–6.
85. Ahmad SY, Friel J, Mackay D. The Effects of Non-Nutritive Artificial Sweeteners, Aspartame and Sucralose, on the Gut Microbiome in Healthy Adults: Secondary Outcomes of a Randomized Double-Blinded Crossover Clinical Trial. Nutrients. 2020;12:3408.
86. Wang I-K, Wu Y-Y, Yang Y-F, Ting I-W, Lin C-C, Yen T-H, et al. The effect of probiotics on serum levels of cytokine and endotoxin in peritoneal dialysis patients: a randomised, double-blind, placebo-controlled trial. Benef Microbes. 2015;6:423–30.

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