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Editorials

Debates in Nephrology

Moderator Commentary

Original Investigation

Acid/Base and Electrolyte Disorders

Acute Kidney Injury and ICU Nephrology

Chronic Kidney Disease

Clinical Images in Nephrology and Dialysis

On the Cover

Gut microbiota dysbiosis and bacterial metabolites in the gut-liver-kidney axis. Gut dysbiosis induces bacterial translocation to the liver, where it promotes the release of proinflammatory cytokines, oxidative species and pro-fibrogenic factors. These molecules can enhance the gut dysbiosis and also induce kidney damage. Renal dysfunction results in accumulation of uremic toxins, which increase the gut dysbiosis and the liver damage. On the other hand, gut dysbiosis promotes an increased release of indoxyl sulfate, p-cresol, LPS and proinflammatory cytokines (due to the activation of the TLR4/Myd88/NF-κB pathway by LPS), which promote kidney damage. Also, gut dysbiosis increases the production of TMAO which can promote further liver damage and trigger kidney dysfunction. TLR4: toll-like receptor 4; Myd88: myeloid differentiation factor 88; NF-κB: nuclear factor kappa B; TMAO: trimethylamine-N-oxide. Figure 1 in "Renal sensing of bacterial metabolites in the gut-kidney axis" by Orestes Foresto-Neto, Bruno Ghirotto, and Niels Olsen Saraiva Câmara. KIDNEY360 2: 1501–1509, 2021. DOI: 10.34067/KID.0000292021.