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Chronic kidney disease (CKD) is the progressive kidney damage and loss of renal function over time. Its global prevalence in 2022 was more than 10% of the world's population and this is steadily increasing. TMAO is a uremic toxin that physiologically originates from gut microbiota metabolism. TMAO is known to be associated with CKD but its role is not clearly described. The aim of this thesis is to investigate the role of TMAO in the renal tubulointerstitium and CKD. In vitro, TMAO promotes proliferation and collagen production of renal fibroblasts, enhances the effect of TNF α and suppresses albumin uptake by proximal tubular cells. In patients with CKD, we identified significant concentration differences and correlations between TMAO or its precursors and urine megalin, urine lysine, urine albumin and markers of tubular damage compared to healthy controls.

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