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Uric Acid and the Kidney: What Hyperuricemia and Hypouricemia Tell Us

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Uric acid, the final product metabolism in humans, has emerged as more than a simple metabolic byproduct, offering important insights into kidney physiology and pathology. Both hyperuricemia and hypouricemia reflect distinct alterations in renal handling of urate and are increasingly recognized as clinically meaningful conditions rather than incidental laboratory findings. Hyperuricemia has long been associated with gout and nephrolithiasis, but accumulating evidence suggests a broader role in the progression of chronic kidney disease (CKD), hypertension, and cardiovascular morbidity. Experimental and epidemiologic studies indicate that elevated uric acid levels may contribute to endothelial dysfunction, oxidative stress, and intrarenal inflammation, although causality remains debated. Conversely, hypouricemia, often underrecognized, provides unique insights into renal tubular function. Genetic defects affecting urate transporters, such as URAT1 and GLUT9, can lead to renal hypouricemia and are associated with an increased risk of exercise-induced acute kidney injury and nephrolithiasis. These conditions underscore the importance of uric acid as a marker of proximal tubular integrity and systemic oxidative balance.

Keywords: Uric acid, Hyperuricemia, Hypouricemia, CKD, Urate transporter