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THE ROLE OF EPITHELIAL-MESENCHYMAL TRANSITION IN THE PATHOGENESIS OF CHRONIC KIDNEY DISEASE AND RENAL CELL CARCINOMA (PROBLEMS AND PROSPECTS)

Epithelial-mesenchymal transition is a process in which epithelial cells lose their intrinsic properties (cell-cell adhesion and apical-basal polarity) and acquire properties of mesenchymal cells (spindle-shape, motility and synthesis of extracellular matrix components). Tubular EMT is proposed as an orchestrated, highly regulated process that consists of four key steps: 1) loss of epithelial cell adhesion; 2) de novo α-smooth muscle actin expression and actin reorganization; 3) disruption of tubular basement membrane; 4) enhanced cell migration and invasion. The key molecular features of the EMT is the loss of E-cadherin expression associated with the activation of transcription factors Snail1, Snail2 (Slug), ZEB1, Sip1/ZEB2, E2A, and the reorganization of actin fibrils and formation of stress fibers as a consequence of the activation of transcription factors Twist1 and MRTF. Over the last years it has been established that EMT may underlie the development and progression of many pathological processes in the kidney and reflects the adaptive changes in the cells in response to an injury or change of the gene activity in tumor growth. The study of the EMT molecular mechanisms opens up opportunities for fibrosis development risk individual assessment and may lead to the design of specific and effective target therapy the interstitial fibrosis and renal cell carcinoma.