Extracellular acid-sensitive two-pore domain K channels are involved in epithelial-mesenchymal transition in bladder cancer cell lines

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Bladder cancer is a common malignant tumor of the urinary tract in humans, and although treatment methods for bladder cancer have been improved, recurrence occurs frequently after surgery and treatment. TASK-3 (KCNK9) is a member of the two-pore domain K channel (K2P) family that is inhibited by extracellular acidic pH and is known to have oncogenic potential. TASK3 has been reported to be frequently overexpressed in breast cancer, lung cancer, colon cancer, and prostate cancer, but the expression and role of TASK3 in bladder cancer are not well known. In the present study, we investigated whether small interfering RNA (siRNA) targeting the TASK3 channel regulates cell proliferation, migration, invasion, and epithelial-mesenchymal transition (EMT) in a bladder cancer cell line (5637). TASK3 knockdown by TASK3 siRNA reduced cell proliferation and also inhibited cell migration and invasion. In the 5637 cell line, knockdown of TASK3 increased the expression of E-cadherin, an epithelial marker, and decreased mesenchymal cell markers (vimentin, slug). Whereas, in 5637 cell line, TASK3 overexpression increased mesenchymal cell markers (vimentin, slug). These results suggest that TASK3 may be a novel therapeutic target for bladder cancer because TASK3 channels contribute to the regulation of EMT in bladder cancer and EMT is associated with drug resistance to various cancer therapies.