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Activation of the Wnt/beta-catenin pathway by the tumor suppressor Pten. PTEN is a dual-specificity phosphatase that antagonizes phosphatidylinositol 3-kinase signaling and plays an important role in the regulation of cell proliferation, cell survival, and the cytoskeletal structure. PTEN is also known to be a negative regulator of the Wnt/beta-catenin pathway. The human PTEN gene is frequently mutated in various cancers, and functional analyses of mutations in PTEN have shown that the protein is likely to be a tumor suppressor. In this study, we found that the downregulation of PTEN by RNA interference in prostate cancer cells (DU145 and PC3 cells) led to the activation of the Wnt/beta-catenin signaling pathway and the upregulation of genes associated with epithelial-mesenchymal transition. The inhibition of the Wnt/beta-catenin signaling pathway by the specific inhibitors of Tcf and GSK3beta in the PTEN-downregulated cells effectively blocked the overexpression of transcription factors associated with epithelial-mesenchymal transition, including Snail, Twist, and Smad4. Moreover, the forced expression of PTEN in DU145 cells inactivated the Wnt/beta-catenin signaling pathway and reduced the expression of genes associated with epithelial-mesenchymal transition. We also found that PTEN is located at the level of cytoplasmic membrane in cells and is translocated to the nucleus after stimulation of the Wnt/beta-catenin pathway. Moreover, the levels of the phosphorylated beta-catenin and its target gene c-Myc were increased after PTEN was downregulated in cells. Our data suggest that the tumor suppressor PTEN plays an important role in regulating the Wnt/beta-catenin pathway by directly binding to and phosphorylating beta-catenin and recruiting it to the cytoplasmic membrane. The PTEN may be involved in the prevention of tumor metastasis.

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