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Letter to Editor

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Wnt: A Main Protein in Bladder Cancer

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Abstract

Bladder cancer (BC) is one of the most common cancers in the world. Wnt/b-catenin is one of involved signaling pathways in this cancer. This letter aimed to discuss about the importance of Wnt in BC.

Keywords: Bladder cancer, Wnt.

Dear Editor

Bladder cancer (BC) is the ninth most common cancer in the world and has a long-term treatment course with significant costs for the patients and mortality. Almost 95% of these cases originate from the urothelium and attack the underlying muscle layer before spreading body entire. The lowly consequences related to advanced illness show a crucial need for novel and better therapeutic plans. More and more research is being done on the molecular pathways involved in the pathogenesis of bladder cancer with the aim of discovering potential therapeutic goals. The main signaling types involved in bladder cancer include Wnt, MAPK, NF-kB and PI3K [1].

The Wnt/b-catenin, PI3K/AKT/mTOR, and NF-kB signaling pathways are involved in both MIBC and NMIBC, but EGFR and VEGFR factors are related to danger of metastasis to other parts [2].

Abnormal triggering of the Wnt signaling pathway plays an important character in the pathogenesis of many cancers. Wnt inhibitory factor-1 (Wif 1) has been recognized as one of the key antagonists capable of binding to the Wnt. Wif-1 is an important factor involved in the pathogenesis of bladder cancer. A study showed that hypermethylation of CpG of the Wif-1 factor promoter is a common occurrence in bladder cancer and may be a crucial process related to its pathogenesis via unusual Wnt/β-catenin main pathway (canonical) [3]. Also, today has been recognized several new relations of SNPs in the Wnt/b-catenin signaling with bladder cancer danger. Especially it was proved that CSNK1E exhibited a high expression in bladder cancer matched to that in usual bladder [4].

Many Wnt-associated proteins are strongly related to cancer metastasis and invasion. EFEMP2, a protein of extracellular matrix, or epidermal growth factor-containing fibulin-like extracellular matrix protein 2 is one of them. On the other hand, under the influence of the cancer microenvironment, EFEMP2 has many characters in tumor types. Results have showed that reduction of expression of EFEMP2 could considerably decrease the epithelial factor (E-cadherin), enhance some mesenchymal factors such as Vimentin in addition to the main markers of Wnt/β-catenin pathway specially β-catenin. It is important to note that the associated changes in the expression of epithelial-mesenchymal transition (EMTs) factors and Wnt/β-catenin signaling proteins stimulated by an increase or decrease in the expression level of EFEMP2 are eliminated with LiCl or XAV939 [5].

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