Protein arginine methyltransferase 5 promotes bladder cancer growth through inhibiting NF-kB dependent apoptosis

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Published Nov 20, 2018

DOI https://doi.org/10.17179/excli2018-1719

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Abstract

Protein arginine methyltransferase 5 (PRMT5) has emerged as a key regulator of tumorigenesis. However, how PRMT5 functions in bladder cancer, the most common malignancy of the urological system, is unknown. We described here that PRMT5 is highly expressed in bladder cancer cell lines and primary human bladder cancer tissues. PRMT5 enhances the proliferation and colony formation of bladder cancer cells. PRMT5 knockdown induces bladder cancer cell apoptosis. Mechanistically, PRMT5 enhances NF-kB activation by targeting crucial anti-apoptotic genes such as *BCLXL* and *c-IAP1*, thereby inhibiting tumor cell apoptosis and sustaining proliferation. Importantly, PRMT5 inhibitor opposed tumor growth and *BCLXL* and *c-IAP1* transcription in the bladder cancer xenograft model. Collectively, the current suggests the crucial role of PRMT5 as a promising therapeutic target in bladder cancers.

How to Cite

Hu, G., Wang, X., Han, Y., & Wang, P. (2018). Protein arginine methyltransferase 5 promotes bladder cancer growth through inhibiting NF-kB dependent apoptosis. *EXCLI Journal*, *17*, 1157-1166. https://doi.org/10.17179/excli2018-1719

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