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WHEN MEIS IS UP IN PROSTATE CANCER, THEN MEISi?

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ABSTRACT

Prostate cancer (PCa) is the second most diagnosed cancer in males. Understanding the molecular mechanism and investigation of novel ways to block PCa growth or metastasis are vital and a medical necessity. In this study, we examined differential expression of MEIS1/2/3 and its associated factors in PCa cell lines. MEIS1/2/3 content, reactive oxygen species, cell cycle status, metastatic activities were analyzed in PCa cells pre and post MEIS inhibitor (MEISi) treatments, which is developed in our laboratory as a first-in-class small molecule inhibitor. A correlation was detected between MEIS content and MEISi IC50 values of PCa cells. MEISi decreased the viability of PC-3, DU145, 22Rv-1 and LNCaP cells, and significantly increased apoptosis in parallel with the increased cellular ROS content. The efficacy of MEISi was shown to positively correlate with the levels of MEIS1/2/3 proteins and the long-term exposure to MEISi elevated MEIS1/2/3 protein content in PCa cells. Administration of MEISi was also tested in PC-3 and 22Rv1 xenograft models of SCID mice to block in vivo tumor growth and metastasis. Our findings suggest that MEISi could be used to target PCa with high MEIS expression to lower PCa viability and growth, however, further studies are needed to achieve in vivo efficacy.
Keywords: Prostate cancer, MEIS, Small Molecules