Lu Tao, Virol-mycol 2015, 4:2
http://dx.doi.org/10.4172/2161-0517.S1.002

OMICS International
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International Conference on
Retroviruses & Novel Drugs

June 08-09, 2015 Chicago, USA

VBIM technology identifies adenomatous polyposis coli like protein (ALP) as a novel negative regulator of NF-κB

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Colon cancer is the second leading cause of cancer related deaths in the United States. The nuclear factor κB (NF-κB) is an important family of transcription factors whose aberrant activation has been found in many types of cancer, including colon cancer. Therefore, understanding the regulation of NF-κB is of ultimate importance for cancer therapy. The purpose of this study is to use a novel validation-based insertional mutagenesis (VBIM) strategy to identify novel regulators of NF-κB, and further evaluate their roles in the regulation of NF-κB signaling in colon cancer cells. We infected Z cells (293 derived cells with hyper active NF-κB activity) with VBIM virus to cause the over expression of negative regulators of NF-κB, and then further selected the mutant cells with low NF-κB activity under ganciclovir (GCV) treatment. Targeted gene was then identified by using VBIM specific primers. In a preliminary screen, we identified the novel adenomatous polyposis coli like protein (ALP) gene as a negative regulator of NF-κB. Over expression of *ALP* led to decreased NF-κB activity by κB reporter assay, while knocking it down had the opposite effect. Furthermore, we found that over expression of *ALP* in HT29 colon cancer cells greatly reduced both the number and the size of colonies that were formed in a soft agar assay, while using shRNA resulted in an opposite effect, confirming that *ALP* is a tumor suppressor in HT29 cells. Future experiments aim to further assess the role of *ALP* in colon tumor formation in a mouse xenograft model. In summary, by using the novel VBIM technique, we identified *ALP* as a novel negative regulator of NF-κB. This discovery could lead to the establishment of *ALP* as a potential biomarker and therapeutic target in colon cancer.

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