Genetics alterations of TNFAIP3 and BCL2 mediate NFkB pathway in cancers

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Abbreviations:FISH, fluorescence in situ hybridization

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Abstract

Background:This TNFAIP3 gene was identified as a tumor suppressor gene whose expression is rapidly induced by the tumor necrosis factor (TNF). Inhibit NF-kappa B activation as well as TNF-mediated apoptosis. BCL2 gene in mitochondria membrane protein suppresses apoptosis. **Motivation:** TNF, lipopolysaccharide, interlukin-1,

CD40, hepatitis C virus core protein, influenza virus infection and latent membrane protein 1 (LMP1) and then activate the expression of TNFAIP3 and BCL2, P53 inhibited the activation of apoptosis by TNFAIP3. So we can confirm that tumor-specific gene expression in different ways. Hypothesis: Tumors induced by the LMP1 oncogene and anti-apoptotic gene Bcl TNFAIP3 activation, interference with anti-p53 cells do, so we assume that TNFAIP3 and BCL2 in the tumor in the abnormal expression or genetic structure of the distortion. Aim: TNFAIP3 and BCL2 inhibition of P53 apoptosis resistance, also associated NF κ B act as circle role we use fluorescence in situ hybridization confirmed that gene tumor marker. Materials and Methods: Twenty-three cell lines included nine renal cell tumor cell lines, we used two-color fluorescence in situ hybridization compared gene expression levels. Self-made probe positioning of the metaphase chromosomes in the business slide, confirmed the exact location of genes in other cells after the hybridization of the samples. Results: We observed that in addition to TNFAIP3, BCL2 amplification and break-apart of the renal cell carcinoma, hepatocellular carcinoma, non-small cell lung cancer, BCL2 is also in the transitional cell carcinoma of gene copy number loss. **Conclusions:**Gene produces an abnormal derivative chromosome and gene copy number variation, mutation methods may include chromosomal translocation. In current study, our data present more consist of expression level of TNFAIP3 and BCL2 in tumor cells, except the colon cancer SW620 cells. The anti-apoptosis mechanism of TNFAIP3 so far is unclear, the further investigations were necessary to demonstrate the relation between BCL2 and TNFAIP3 in cancer, while TNFAIP3 and BCL2 can also be a tumor markers.