5 - 63 Effect of TSPAN4 Gene on Migration and Invasion of Ability

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Tetraspanin family molecules are cell surface proteins with 4 transmembrane domains that form complexes called tetraspanin webs by associating with various adhesion molecules, receptors and other tetraspanin molecules. It is known that the tetraspanins affect various cell functions such as cell adhesion, signal transduction, cell proliferation, cell movement, and cell differentiation. TSPAN4 is a tetraspanin belonging to the transmembrane 4 superfamily that forms complexes with the integrins alpha3beta1 and alpha6beta1[1], and their overexpression was reported in multiple tumors, including lung, breast, colon, prostate cancer, and hepatocellular carcinoma. TSPAN4 was previously shown to be abundant in the migrasome membrane[2]. To detect the effect of TSPAN4 on radiotherapy,

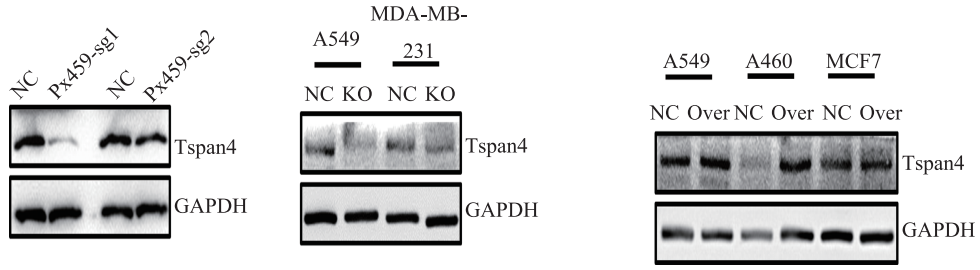

Fig. 1 (color online) The representative western blotting image of Tspan4 in normal, knockout and over expression cells compared to the GAPDH.

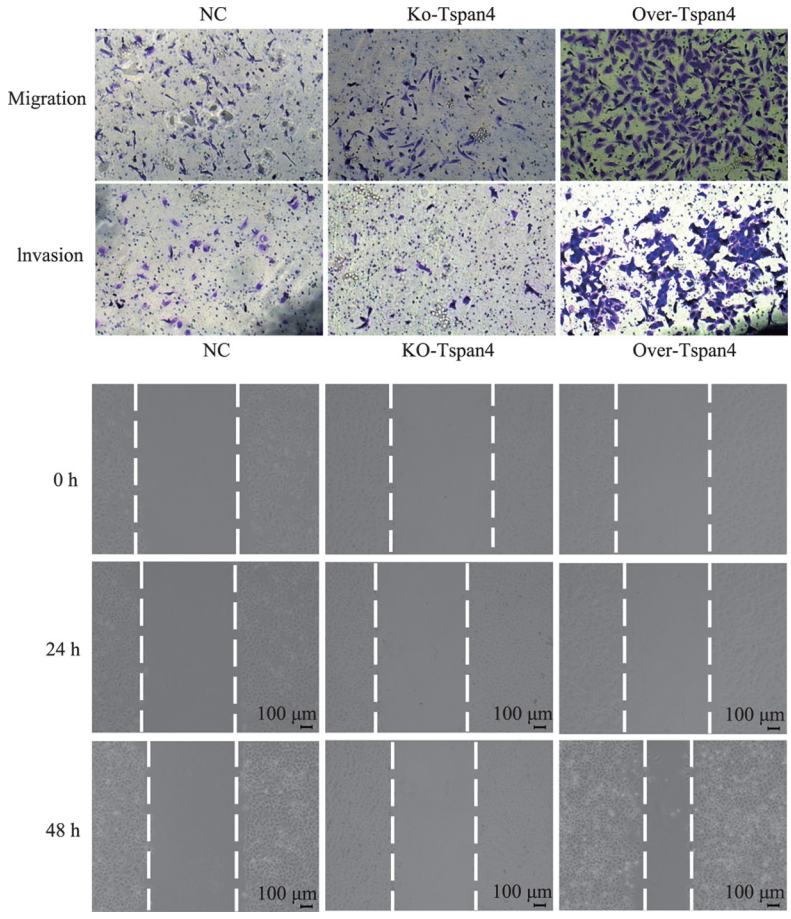

Fig. 2 (color online) Effect of TSPAN4 on invasion and migration of A549 cells by Transwell assay and wound healing assay.

we constructed the knockout vector px459-sgRNA-TSPAN4 and over expression vector pEGFP-N1- TSPAN4 and transfected into cel lines. Monoclonal cells were obtained by puromycin or G418 pressure screening, and the efficiency of TSPAN4 knockout or over expression was identified by Western-blot in the cell lines (Fig. 1). The cell scratch and transwell experiment were used to analysis the effect of the invasion and migration in knockout or over expression TSPAN4 cell lines. Compared to the wild cell lines, there is no statistical difference on the ability of migration in knockout cell line of TSPAN4[3], but the over expression TSPAN4 enhances the ability of the migration and invasion (Fig. 2). It has important scientific value and clinical guiding significance for further understanding the characteristics of radiotherapy of cancer, reducing the risk of tumor cell metastasis, and further improving the efficacy of cancer treatment.

References

[1] I. Tachibana, J. Bodorova, F. Berditchevski, et al., J. Biol. Chem., 272, 46(1997)1.

[2] L. Ma, Y. Li, J. Peng, et al., Cell Res., 25(2015)24.

[3] Y. Liu, Y. R. Du, K. Sun, Chinese Journal of Cell Biology, 45, 2(2023)203. (in Chinese)

5 - 64 IGFBP-3 Participates in Maintaining the Phagocytic Function of MKC Cells After Carbon Ion or X-ray Irradiation

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Ionizing radiation exposure from spaceflights, radiotherapies or nuclear accidents may threaten human health. Our previous study has found that the concentration of serum Insulin Like Growth Factor Binding Protein-3 (IGFBP-3) of mouse significantly increased after total-body irradiation by protons, carbon ions and X-rays, presenting great potential as an effective and minimally invasive biomarker [1−3]. Further study revealed ionizing radiation sensitive IGFBP-3 in blood is mainly derived from Kupffer cells (MKC cells) in the liver. In this study, we found carbon ion irradiation can damage the phagocytic function of MKC cells (Fig. 1(a) and (b)) and another macrophage

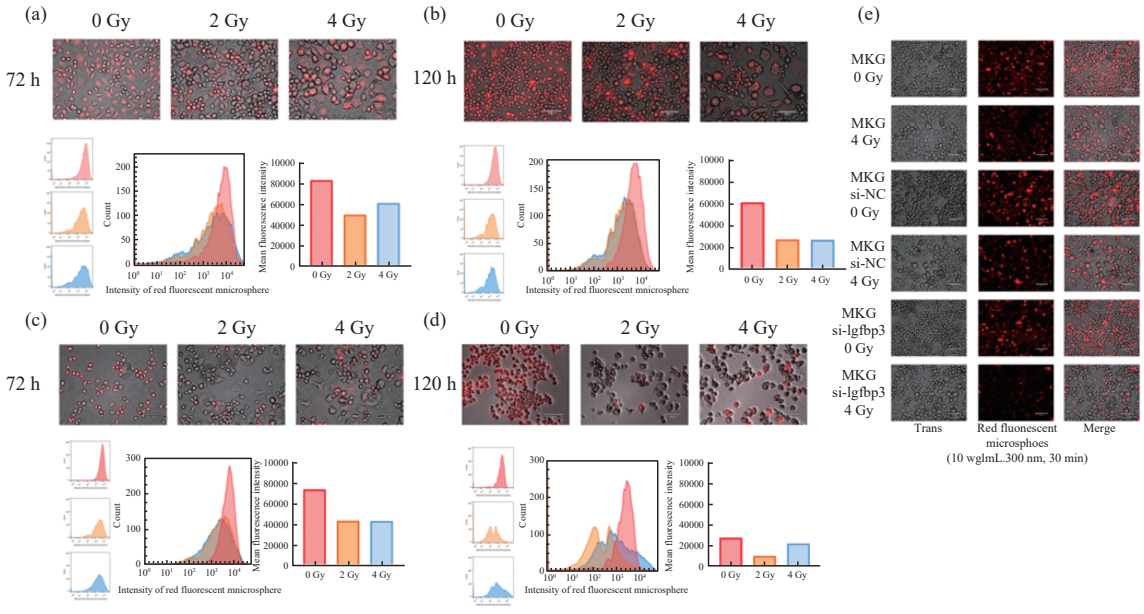

Fig. 1 (color online) IGFBP-3 participates in maintaining the phagocytic function of MKC cells after carbon ion or X-ray irradiation: (a) Impaired phagocytic function of MKC cells 72 h after carbon ion irradiation, (b) Impaired phagocytic function of MKC cells 120 h after carbon ion irradiation, (c) Impaired phagocytic function of RAW 264.7 cells 72 h after carbon ion irradiation, (d) Impaired phagocytic function of RAW 264.7 cells 120 h after carbon ion irradiation, (e) Knockdown of IGFBP-3 protein levels further decreased the phagocytic function of MKC cells after irradiation.