J Cancer Sci Ther 2014, 6:10
http://dx.doi.org/10.4172/1948-5956.S1.035

OMICS Group Conferences
Accelerating Scientific Discovery

4th World Congress on
Cancer Science & Therapy
October 20-22, 2014 DoubleTree by Hilton Hotel Chicago-North Shore Conference Center, USA

Estrogen receptor β as a potential target for lymphoma therapy

Konstantin Yakimchuk
Karolinska Institutet, Sweden

Cellular signaling by estrogens is mediated by estrogen receptors α (ERα) and β (ERβ). Estrogens stimulate proliferation via ERα, but inhibit proliferation and promote differentiation via ERβ. With regard to the immune system, ERβ was found to be the dominant expressed ER. Estrogens regulate various physiological pathological processes, including cancers. Several epidemiological studies demonstrate that men have a higher incidence of B-cell lymphomas and leukemias in comparison to women, suggesting that lymphomas may be under estrogen influence. Our studies have demonstrated ERβ expression in human B cell lymphoma cell lines derived from patients with Burkitt's (BL) and mantle cell (MCL) lymphomas. Grafting human BL and MCL lymphoma cells to immune-compromised NOD.SCID mice showed an inhibition of tumor growth following treatment with the ERβ selective agonist diarylpropionitrile (DPN). To test whether ERβ activation will inhibit lymphoma dissemination *in vivo*, we engrafted NOD/SCID mice subcutaneously with Raji BL cells previously shown to be able to disseminate. The numberand the sizeof tumor foci of Raji cell dissemination in liver were significantly reduced by DPN treatment of mice with Raji lymphomas. In addition, expression of a chemokine receptor CXCR4, involved in lymphoma dissemination and homing to secondary of lymphoid tissues, was found to be downregulated by DPN. These results show that ERβ agonist treatment significantly inhibits lymphoma dissemination *in vivo* and suggest that specifically targeting ERβ in lymphomas with ERβ agonists will therefore be useful in the treatment of B-cell lymphomas.

Biography

Konstantin Yakimchuk has completed his MD in 1998 and PhD in immunology and genetics in 2001. He obtained his postdoctoral training at Brigham and Women's Hospital and Harvard Medical School, Boston, USA. Currently, he is the Senior Reseacher at KarolinskaInstitutet, Stockholm, Sweden. His research is focused on the functions of estrogen receptors in immune system and hematological malignancies. His studies on estrogen receptors in lymphomas were published in *Leukemia* and *Blood* journals.

Overexpression of type VI collagen in neoplastic lung tissues

Hua-Chen Chang
Indiana University, USA

Type VI collagen (COL6), an extracellular matrix protein (ECM), is important in maintaining the integrity of lung tissue. An increase in COL6 mRNA and protein deposition was found in the lungs of patients with pulmonary fibrosis that has a strong association with lung cancer. In this study we demonstrated overexpression of COL6 in the lungs of non-small cell lung cancers (NSCLC). We hypothesized that excessive COL6 in the lung interstitium may directly exert pro-inflammatory effects on the adjacent cells that may promote tumorigenesis in the lung. In vitro stimulation of monocytes with COL6 resulted in the secretion of IL-23, which may promote tumor development in an environment of IL-23-mediated lung inflammation, where tissue modeling occurs concurrently with excessive COL6 production. In addition, COL6 is capable of stimulating signaling pathways that activate focal adhesion kinase (FAK) and extracellular signal-regulated kinase (ERK)1/2 in lung epithelial cells, which may also facilitate the development of lung neoplasm. Taken together, our data demonstrated the potential role of COL6 in promoting lung neoplasia in diseased lungs where COL6 is overexpressed.

huchang@iupui.edu