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Might Estrogen Promote Lung Cancer Progression?

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Abstract

In order to understand the differences between sexes in lung cancer presentation and due to the observation that young women have worse prognosis, we studied the participation of estrogens in lung carcinogenesis. We found that estradiol increases the expression of CXCR₄ and promotes the activation of the CXCL₁₂/CXCR₄ axis, an important chemokine pathway involved in lung carcinogenesis and tumor progression.

these patients. Estradiol promotes lung carcinogenesis in several forms; we found other mechanism by which this hormone might stimulate lung cancer progression. Due to the role of estrogen in lung cancer and the effect that antiestrogen drugs have on lung cancer cells, E₂/ER pathway can be seen a new therapeutic target. Although further research is needed on the role of estrogen in lung cancer, it seems to identify Estrogen receptor (ER β) and aromatase enzyme expression in lung tumours, might be important in designing new therapeutic strategies in lung cancer based in antiestrogen drugs, since by inhibiting estrogenic pathway, other important signalling pathways involved in lung carcinogenesis are also affected.

Letter to Editor

Lung cancer remains the leading cause of cancer death in men and has increased worldwide in women in the last decades [1]. Lung cancer behavior appears to be different between women and men [2]. However information about these differences is still controversial maybe because several studies compare pathological characteristics by sex but not by hormonal status. When premenopausal women have been considered in some studies, they coursed with more advanced stages of cancer at diagnosis, poorly differentiated tumors and worse prognosis compared to postmenopausal women and men [3]. Some studies have showed that sex hormones, mainly estradiol, through its receptor ER β have an important role in lung carcinogenesis [4,5]. Previously we reported that lung adenocarcinomas from premenopausal women exhibited higher signal of estrogen receptor (ER β), as well as CXCL₁₂ and CXCR₄ compared to adenocarcinomas from postmenopausal women and men, suggesting that expression of this proteins in tumors could be related with estradiol levels [6]. Recently we also reported that estradiol stimulates CXCR₄ expression in a dose- and a time-dependent manner and favors CXCL₁₂/CXCR₄ activation pathway by promoting lung cancer cell migration *in vitro*. In the same study, Tamoxifen treatment reduced CXCR₄ expression and suppressed lung cancer cell migration [7]. The CXCL₁₂/CXCR₄ pathway is an important Chemokine/Receptor axis, involved in proliferation, migration and metastasis in lung cancer. Expression of CXCR₄ is related with advanced stages, metastasis and poor survival in patients with lung cancer. High expression of CXCR₄ in lung adenocarcinoma from premenopausal women explains partially the worst prognosis of

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