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.

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 because several studies compare pathological characteristics by sex but not by hormonal status. When premenopausal women have been considered in some studies, they were compared 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 ERβ have an important role in lung carcinogenesis [4,5]. Previously we reported that lung adenocarcinomas from premenopausal women exhibited higher signal of estrogen receptor (ERβ), 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 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 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 (ERβ) 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.

References