Non-small cell lung cancer and estrogen

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Fastest growing area of lung cancer caused by oestrogen, not smoking

Non-small cell lung cancer has risen from 12 per cent of all lung cancer to 40 per cent in the last 20 or so years in America. And it is controlled by oestrogen (estrogen) receptors, in much the same way most breast cancers are.

The first clues came in a Canadian study covered in Cancer Watch about 5 years ago, where the growing area of lung cancer was amongst non-smokers and particularly younger women. An inconvenient truth for the cancer charities and health authorities who want to tell you it’s your fault because you smoke!

There are differences between lung cancer in women, and lung cancer in men. And, it is now known that lung cancer cells have oestrogen receptors. An evaluation of research to date in America has shown that women with more children have less risk of lung cancer, women who have an early menopause have greater risk of lung cancer, and women who have taken HRT have a 60% increased risk of dying from lung cancer whether or not they smoke. Conversely, women who have a diet high in phytoestrogens (plant oestrogens) have a lowered risk of lung cancer. And men may be protected by their oestrogen receptor sites.

Fat is broken down by Aromatase enzymes that convert it to oestrogen. And aromatase inhibitors have been studies for both breast cancer and lung cancer. Oestrogen is known, not just to be causal, but to encourage metastases too. Treatment with cisplatin, and exemestane has been shown to reduce progression. The oestrogen receptor pathway seems linked to developments connected with the epidermal growth factor receptor pathway. Erlotinib (Tarceva) is a drug used to treat this pathway.

Under normal conditions in women, there is a feedback system between the hormones progesterone and oestrogen. Each limits the other. But increasingly it is being found that chemical oestrogens (synthetic xenoestrogens) bypass this system and act unchecked. Just small levels, in parts per trillion, have been shown to cause osestrogenic effects.

Only recently the World Health Organisation recommended that Governments banned these ‘gender bender chemicals, like BPA and phthalates. But there are many such chemicals of concern and they have been linked to cancer and non-cancer issues (such as earlier periods, lowered sperm counts, enlarged prostates and endometriosis for example).

Read more on this subject; and on what you can do naturally to limit oestrogen in your life read ‘Oestrogen – the killer in our midst’ by Chris Woollams. Click here to read more.

Read more on lung cancer by clicking here.


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Estrogen-induced cell proliferation results mainly from activation of cAMP, MAPK and AKT signaling pathways through the non-genomic pathway. Estradiol also promotes LC cell proliferation by inducing rapid phosphorylation of ERK and EGFR. Through the genomic pathway, estradiol promotes the expression of c-myc, Cyclin D, and Id proteins, resulting in cell cycle progression and proliferation (52,53). Estrogens may be involved in lung carcinogenesis, and estrogen receptors, mainly estrogen receptor-ß (ER-ß), are present and functional in normal lung and tumor cell lines and tissues. In addition, estrogens and growth factors may promote the progression of human non-small cell lung cancer (NSCLC). Previously, we have immunohistochemically demonstrated that MK and ER-ß proteins were overexpressed in NSCLC and their expression levels were both significantly negatively correlated with the pathological classification. The purpose of this study was to further verify their expression and its correl.

Lung cancer is histologically classified into small-cell lung carcinoma and non-small-cell lung carcinoma (NSCLC). NSCLC accounts for âˆ’80% of all the lung cancer cases, and represents heterogeneous groups which include the squamous cell carcinoma, adenocarcinoma, and large cell carcinoma. Â T47D cells were used as positive controls for progesterone receptor and estrogen receptors (21, 22). Equal loading of protein in each lane was confirmed by probing the membrane with anti-human ß-actin monoclonal antibody (Sigma). Luciferase assay. Â Expression of progesterone receptor and estrogen receptors in non-small-cell lung cancer cell lines.