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Expression of estrogen receptor alpha66 and its truncated isoform 36 in thyroid cancer cells

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Abstract

The full-length estrogen receptor (ER)-a66 (ERa66) and its truncated isofrom ERa36 have been reported to play roles in the growth of certain human cancers including breast cancer and prostate cancer. However their role in the thyroid cancer is poorly understood. We therefore examined ER-α66 and its isoform ER-α36 in human thyroid cells. We found that ER-α66 was not expressed in normal thyroid cells. In thyroid cancer cells, ER-α66 was weakly expressed or not expressed. However, thyroid cancer cells expressed a significant amount of ER-α36, a novel isoform of ER-α66, at both mRNA and protein levels while this isoform was absent in normal thyroid cells. Over expression of ER-α66 by ER-α66 DNA transfection enhanced the proliferation and autophagy in thyroid cancer cells. Inhibition of ER-α36 attenuated the proliferation and autophagy in thyroid cancer cells. The increase of phosphorylated ERK but the decrease of Bcl-xL contributed to ER-α-related proliferation and autophagy in cancer cells. Collectively, human normal thyroid cells do not express ER-α66 but cancer cells express ER-α66. A significant amount of ER-α36 is present in thyroid cancer cells but not in normal thyroid cells. ER-α66 promotes the proliferation and autophagy and the inhibition of ER-α36 attenuates both in thyroid cancer cells. The increase of phosphorylated ERK but the decrease of Bcl-xL contributes to ER-α-related proliferation and autophagy. These findings may help to design some target therapies towards ER-α36 in thyroid cancer cells.

Biography

George G Chen had medical training in China and PhD and postdoctoral training in United Kingdom. Currently, he is a Professor at Department of Surgery, Faculty of Medicine, The Chinese University of Hong Kong, and he is also the Director of Surgical Laboratories at the same institute. He has published more than 150 papers in peer-reviewed journals and served several journals as a board member.