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CORRELATION BETWEEN C-FOS AND RADIOIODINE EFFECT IN BREAST CANCER CELL LINES

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When ionizing radiation hits water molecules in the cell, water will be degraded to produce free radicals. Free radicals inhibit the switch from *c-Fos* to Fra1 in chromatin. This inhibition will lead to the failure in cells to express cyclin D1, which then followed by cell cycle arrest. The aim of this study is to investigate the correlation between *c-Fos* with radioiodine effect in breast cancer cell lines. Breast cancer cell lines (MCF7 and SKBR3), and keratinocyte cell line (HaCaT) were used in this study. To induce *c-Fos* expression, cells were treated with 50 ng/ml epidermal growth factor (EGF), 100µM adenosine triphosphate (ATP) and a combination of both. Radioiodine effect was measured by reproductive ability of the cells after which they had been treated with 74.10^4 Becquerel/well of NaI-131. A quantitative real-time reverse transcriptase polymerase chain reaction (qRT-PCR) and immunocytofluorescence were used to assess *c-Fos* expressions. *c-Fos* expressions are found only in MCF-7 cells. A combination of ATP and EGF has a potential to induce 22.74±3.67 fold ($p<0.05$) of *c-Fos* mRNA. Adenosine triphosphate or EGF or combination of both increases *c-Fos* protein expression ($p<0.05$). Induction of EGF or a combination of ATP and EGF reduces the reproductive ability of MCF-7 and SKBR3 cells up to 100% after radioiodine treatment ($p<0.05$). We find an inverse correlation between *c-Fos* mRNA and protein expressions with radioiodine effect are $r=-0.90$ and $r=-0.97$ ($p<0.05$) respectively. Based on the above mentioned results, it appears that radioiodine is able to reduce the reproductive ability of breast cancer cells. Therefore, it opens an opportunity for radioiodine to be used for breast cancer treatment. *c-Fos* plays pivotal role in cell death pathways by radioiodine exposure in MCF7 cells, and other genes may correspond to cell death in SKBR3 cells.

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