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The Effects of Tert-butyl Hydroquinone (TBHQ) on Estrogen Receptor Alpha (ER\(\alpha\)) and Tumor Suppressor Protein p53 in T-47D and MCF-7 Breast Cancer Cell Lines

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The Effects of Tert-butyl Hydroquinone (TBHQ) on Estrogen Receptor Alpha (ERα) and Tumor Suppressor Protein p53 in T-47D and MCF-7 Breast Cancer Cell Lines

Tert-butyl hydroquinone (TBHQ) is an aromatic compound that is commonly used as a preservative in processed food to prevent rancidity and lengthen shelf life. TBHQ is known to act as an antioxidant by protecting cells from radical oxygen species and thus preventing DNA damage. Although previous studies have found TBHQ to cause cancer cell death at high concentrations, they have also contrastingly found TBHQ, when studied in animal models, to enhance carcinogenic effects. However, the effect of TBHQ on breast cancer has not been thoroughly explored. With the prevalence of breast cancer and the wide use of TBHQ in processed food items, it is imperative that we explore their possible relationship. This study examined the effects of TBHQ, alone and in combination with hormones and anti-hormones, on ERα and p53 expression in both MCF-7 and T-47D breast cancer cell lines. To ensure treatment conditions without the presence of endogenous steroids or growth factors, the cells were cultured with a 5% charcoal-stripped fetal bovine serum (FBS) for six days. Western blot analysis revealed alterations in the expression of ERα and p53 protein levels after 24 hours of treatment with varying concentrations of TBHQ (0.005 to 1 mM). A concentration-dependent decrease of ERα protein levels was observed in both cell lines, with a 49% reduction occurring with 100 µM TBHQ as compared to the control. P53 levels portray a continued increase of expression through concentrations of TBHQ (0.005 to 1 mM), found similarly in both cell lines. To gain further insight into possible similarities between BPS and other known effectors of ERα, the optimal concentration of TBHQ (100 µM) was used in combination with hormones and anti-hormones. Down-regulation of ERα protein levels was observed after 24-hour co-treatment of T-47D & MCF-7 cells with a combination of TBHQ and E2. Antiestrogen ICI with TBHQ showed a significant down-regulation as compared to TBHQ alone, and TBHQ with TAM portrayed no significant differences. A similar trend in the effects on p53 expression was depicted in T-47D and MCF-7 cells. Image cytometric analysis with propidium iodide staining was utilized to quantify cell values and viability changes to further portray the effects of TBHQ on T-47D and MCF-7 cellular growth. The viability assay shows a biphasic effect with increasing concentrations of TBHQ, with a maximum decrease in proliferation seen at a concentration of 100 uM TBHQ. TBHQ alone and in combination with E2 and antiestrogens showed a decreased proliferation compared to the control in T-47D cells. However, cytolocalization of ERα upon treatment with estradiol and TBHQ remained unaltered. Our studies offer a unique perspective on the effects of TBHQ on two different breast cancer cell lines and provide valuable insight for further exploration of the mechanism of action of TBHQ on tumor suppressor proteins and steroid receptors.