SYNERGY EFFECT OF B-ESTRADIOL IN HUMAN JURKAT T-CELLS

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Abstract: β-estradiol is the most potent estrogen of a group of endogenous estrogen steroids which includes estrone and estriol. This steroid hormone is the most potent natural estrogen, produced mainly by the ovary, placenta, and in smaller amounts by the adrenal cortex, and the male testes. Although β-estradiol is well known to protect the renal and cardiovascular systems, the molecular mechanisms of action involved in this protection remain unclear. Therefore, the purpose of the present investigation was to determine whether oxidative stress plays important role in β-estradiol induced toxicity in human Jurkat T-cells beyond the physiological doses. To reach this goal, we performed the MTT and the trypan blue exclusion test for cell viability, and lipid hydroperoxide assay for assessing the levels of the degradation products of polyunsaturated fatty acid (PUFA) hydroperoxide in Jurkat T-cells subjected to β-estradiol. Human Jurkat T-cells were treated with various doses (0-16 µM) of β-estradiol for 24, 48, and 72 hrs respectively. Data obtained from the MTT assay indicated a somewhat biphasic response that encompasses a slight increase in cell viability between (0-2 µM) of β-estradiol, and a gradual decrease in cell viability above 2 µM of β-estradiol, indicating the stimulatory effect of this compound a low doses and inhibitory effect at doses of exposure in a dose and time-dependent manner. Similar result was obtained with the trypan blue exclusion test. Data generated from lipid hydroperoxide assay resulted in a significant increase (p < 0.05) in the production of hydroperoxide (degradation product of lipid peroxidation) with increasing doses of β-estradiol. In summary the results of the present study demonstrate that physiological levels of β-estradiol induce cell growth and cellular proliferation of human Jurkat T-cells whereas higher doses inhibit cell growth and induce cell death. The lipid hydroperoxide assay demonstrated that β-estradiol toxicity is associated with oxidative stress, a biomarker of cellular injury.

Keywords: β-estradiol, lipid hydroperoxide, toxicity, human Jurkat T-cells

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