Comparative Analysis of the Effects of 17-Beta Estradiol on Proliferation, and Apoptosis in Hepatocellular Carcinoma Hep G2 and LCL-PI 11 Cell Lines

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Abstract

BACKGROUND:

Phytoestrogens are a group of natural compounds with estrogen-like activity and similar structure to estradiol that structurally mimic the mammalian estrogen 17-β estradiol (E2). They have a biphasic effect and exert pleiotropic effects which induce or inhibit estrogen action by activation/inhibition of the estrogen receptors (ERs). These compounds can induce apoptosis at high concentrations. The previous finding indicated that E2 inhibited cell growth and induced apoptosis in hepatocellular carcinoma (HCC) PLC/PRF/5 cell line. The aim of the present study was to investigate the apoptotic and proliferative effects of E2 on hepatocellular carcinoma HepG 2 and LCL-PI 11 cells.

METHODS:

The Hep G2 and LCL-PI 11 cells were cultured and treated with E2 for different time periods and then MTT [3-(4, 5-dimethyl-2-thiazolyl) -2, 5-diphenyl -2H- tetrazolium bromide] assay and flow cytometry assay were done to determine cell viability and cell apoptosis respectively.

RESULTS:

E2 had inhibitory and apoptotic effects on Hep G2 cell line, whereas it indicated a biphasic effect on LCL-PI 11 cell line. The half-maximum inhibitory concentration (IC50) value was 3 μM. The inhibitory effect of E2 on Hep G2 cells was observed with all concentrations of E2 ($P < 0.087$), whereas E2 showed a biphasic effect on LCL-PI 11. This compound induced significant apoptosis in Hep G2 cell line at the all treatment times versus control groups, whereas, in the LCL-PI 11 cell, significant apoptotic cells were observed after 72 and 96h ($P < 0.001$).

CONCLUSION:

E2 can inhibit cell growth and induce apoptosis in hepatocellular carcinoma HepG 2 and LCL-PI 11 cell lines.

KEYWORDS:

Estradiol; viability; apoptosis; hepatocellular carcinoma