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In vitro apoptotic and antiproliferative effects of propranolol on human breast cancer cells

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Breast cancer is an issue of concern with increasing incidence among women worldwide. Propranolol, as an antihypertensive drug, exerts anticancer effects too. We conducted this study to analyze the *in vitro* apoptotic and anti-proliferative effects of propranolol in human MCF-7 breast cancer cells. MCF-7 cells were seeded into 6-well plates and treated with 50 µL propranolol for 24 hours. After cell homogenization, the levels of pro-apoptotic proteins BCL2 associated X (BAX), apoptosis inducing factor (AIF), C/-EBP homologous protein (GADD153), and glucose-regulated protein 78 (GRP78), anti-apoptotic protein BCL2 apoptosis regulator (BCL-2), and cycle-regulator WEE1 G2 checkpoint kinase (WEE1) were measured with ELISA. Propranolol significantly upregulated pro-apoptotic proteins AIF, BAX, GADD153, and GRP78 while downregulated anti-apoptotic protein BCL2. The level of WEE1, as the main regulatory cell cycle protein at the G2/M checkpoint, significantly increased after propranolol treatment. Propranolol inhibited the proliferation of MCF-7 human breast cancer cells by upregulating pro-apoptotic factors AIF, BAX, GADD153 and GRP78 and by downregulating antiapoptotic BCL2. Elevated WEE1 levels after propranolol treatment might lead the tumor cells into a sustained cell-cycle arrest which eventually resulted in caspase-dependent or -independent mitochondrial or endoplasmic-reticulum stress-induced apoptosis. So, propranolol can be utilized as a potential therapeutic agent in breast cancer therapy.

Keywords: Anitcancer, Antitumorigenic activity, MCF7