Environmental Toxicology / Early View

RESEARCH ARTICLE

Nerolidol induced apoptosis via PI3K/JNK regulation through cell cycle arrest in MG-63 osteosarcoma cells

Yang Yu, Periyannan Velu, Yulong Ma, Annamalai Vijayalakshmi

First published: 31 March 2022
https://doi.org/10.1002/tox.23522

Abstract

The aim of the present study was to determine the cell proliferation, apoptotic pathway analysis through protein, mRNA and cell cycle arrest mechanism in nerolidol induced osteosarcoma MG-63 cells. The osteosarcoma MG-63 cells were treated with various doses of nerolidol (15 and 20 μM/ml) for 24 h. Cell proliferation was examined using assist method of MTT assay, fixed the IC50 value of nerolidol 15 μM/ml. Reactive oxygen species (ROS) generation was analyzed by DCFH-DA dye, mitochondrial potential detected by Rh-123 dye, apoptotic morphological changes identified by AO/EtBr, PI, DAPI staining, and cell adhesion were detected by using fluorescence microscope. Cell proliferation, and apoptotic molecular protein and mRNA expressions such as ERK, P38, p-PI3K, p-JNK, Bcl-2, JNK, p-P38, cyclin-D1, and Bax were analyzed in osteosarcoma MG-63 cells. Nerolidol significantly suppressed the osteosarcoma cells progression in a dose dependent manner ($p < .05$) evident in the oxidative stress induction and apoptotic morphological changes. Nerolidol also regulated the protein PI3K/AKT mechanistically via induction of apoptosis Nerolidol suppresses osteosarcoma MG-63 cells by PI3K/AKT by cell cycle arrest at early phase of G0/G1. To sum up, nerolidol suppressed the growth of bone cancer cells and can be finally targeted as a potent drug for analyzing its chemotherapeutic effects in future.

Open Research

DATA AVAILABILITY STATEMENT

The data sets used and/or analyzed during the current study are available from the corresponding author on reasonable request.