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2nd Global Summit on

ONCOLOGY & CANCER

March 12-14, 2018 Singapore

Regulating PI3K/AKT and ERK1/2 MAPK signal pathway by Naringenin induces mitochondria related apoptosis and ROS production in choriocarcinoma cells

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Choriocarcinoma is the trophoblastic tumor with high rates of metastasis and reoccurrence. The pathogenesis of choriocarcinoma is not clear and only few of chemotherapeutic chemicals partially work on choriocarcinoma. However, in some patient does not show sufficient reaction to those chemotherapies and chemotherapies have lots of side-effects. Thus, it is important to find a novel phytochemical to treat choriocarcinoma. Naringenin is known as a phytoestrogen and anti-estrogenic effects at the same time per cell type. Even though Naringenin was studied in various cancer with signal molecules regulation, there is few research on choriocarcinoma. Therefore, we investigated the Naringenin on choriocarcinoma (JAR and JEG3) in this study. Naringenin reduced cell viability and migratory function of both cells whereas increased mitochondria related apoptosis in a dose-dependent manner. We also studied intracellular signaling pathway by western blot assay. Naringenin suppressed phosphorylation of AKT, S6 and ERK1/2 whereas increased phosphorylation of P90RSK, P70S6K, P38 and JNK in both cell lines. In addition, we investigated the mechanism of signal molecules through Naringenin and pharmacological inhibitor (LY294002 and U0126) combination treatment on JAR and JEG3 cell lines. Collectively, these results supported that Naringenin is a crucial therapeutic molecule that has anti-cancer effect on choriocarcinoma cells by apoptosis pathway and regulating PI3K and ERK1/2 MAPK signal transduction mechanism.

Biography

Sunwoo Park has completed her under graduation from Korea University and pursuing Master's in Biotechnology at Korea University, South Korea. She has published 3 papers in reputed journals.

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