

عنوان مقاله:

Investigating the effects of rubiadin on apoptosis induction and autophagy inhibition in HT29 cells

محل انتشار:

اولین کنگره ملی تازه های همگرایی علوم پایه و علوم پزشکی (سال: 1401)

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خلاصه مقاله:

Background: Investigating the effect of rubiadin on cytotoxicity, apoptosis induction, ROS level and cell cycle arrest as well as its effects on the autophagy pathway by examining changes in the expression levels of P62, BECLIN1, LC3BI/II, ATG5 and mTOR proteins in HT29 cells. **Materials and Methods:** The effect of rubiadin toxicity on HT29 cells in the concentration range (0.01-50 µg/ml) and 48h was investigated by MTT Assay. Then, the amount of ROS, cell cycle arrest, apoptosis through flow cytometry and changes in the expression of the desired proteins were checked by western blot. **Results:** IC50 was reported at a concentration of 18 µg/ml. Significant increase of intracellular ROS (p-value for concentrations of 10, 18, 25 µg/ml was <0.0001) and apoptosis (10, 18, 25 µg/ml) as well as significant increase of P62, BECLIN1, LC3BI/II, ATG5 and mTOR proteins (18 µg/ml) were observed in the group treated with rubiadin compared to the control group. **Conclusion:** Rubiadin is able to induce cytotoxicity, apoptosis and intracellular ROS on HT29 cells. The increase of P62 means the inhibition of autophagy, and its increase has the ability to activate the mTOR pathway, which also inhibits autophagy. Although autophagy and apoptosis are distinct processes, but there are pathways that can regulate both of them, the increase of P62, BECLIN1, LC3BI/II and ATG5 can help to promote apoptosis.

کلمات کلیدی:

Cancer, Rubiadin, Cytotoxicity, Apoptosis, ROS, Autophagy, HT29

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