

P35 INHIBITORY EFFECTS OF 6'-DEOXYCHALCONE ON THE MIGRATION AND INVASION OF HUMAN ORAL SQUAMOUS CELL CARCINOMA IN VITRO

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Background: Oral cancer is the 5th in numbers of deaths and crude death rates from leading cancer of males in Taiwan. Almost 90% oral cancer was oral squamous cell carcinoma (OSCC). These OSCC patients usually contact to high risk factors, such as betel chewing, smoking, and alcohol drinking. 6'-deoxychalcone was purified from licorice. It has various biological actions, including antioxidant, anti-inflammatory, anticancer activity. 6'-deoxychalcone had been study in ovarian, prostate and breast cancers. However, there were a few research papers about oral cancer.

Methods: The OSCC cell line HSC3 and SAS were used for 6'-deoxychalcone anti-oral cancer study in vitro, and SG cell was used for normal control. Cell viability is detected by MTT assay after variant 6'-deoxychalcone dosage 24 h treatment. Cell cycle and apoptosis was analyzed by flow cytometry. Messenger RNA and protein expression were detected by RT-PCR and western blotting, respectively. The cell colony formation, migration, and anchorage independent ability were test for malignancy.

Results: The IC50 of 6'-deoxychalcone in SG, HSC3, and SAS were 300 μ M, 50 μ M, and 100 μ M for each. **Treat HSC3 and SAS** with 25 μ M and 50 μ M 6'-deoxychalcone, induce G2/M arrest and HSC3 apoptosis. The cyclin A and ATM were down-regulated, and caspase 3 and PARP were up regulated by 6'-deoxychalcone. Furthermore, we verified migration, colony formation, and anchorage independent growth, were inhibited after low dosage 6'-deoxychalcone 3.125 μ M and 6.25 μ M treated.

Conclusion: The G2/M arrest and apoptosis were induced by 6'-deoxychalcone even the OSCC cell lines sensitive or insensitive, but not in normal control. All cell lines express upper than 95% viability after 3.125 μ M and 6.25 μ M 6'-deoxychalcone treated, but the cell malignant phenotypes were significant inhibited. These results indicate that 6'-deoxychalcone could be used in cancer preventive medicine.

P36 COMBINATION EFFECTS OF ADLAY TESTA EXTRACTS AND DOXORUBICIN ON THE GROWTH INHIBITION OF HUMAN UTERINE SARCOMA CANCER CELLS

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Cancer has been the top ten causes of death in Taiwan from 1982. Chemotherapy is one of the cancer treatments. However, chemotherapy will cause many side effects and multiple drug resistance (MDR). These are the main poor effects of chemotherapy. MDR can be the result of a variety of mechanisms that are not completely understood, but many studies have shown P-glycoprotein expression was higher in tumor cells, and it induced MDR. On the other hand, there are evidences that P-gp positive cells are resistant to apoptosis. In addition, it's also closely related between the poor effects of chemotherapy and tumor metastasis. Previous studies revealed that adlay seed extracts could have anti-cancer activity. Thus, this study will investigate that combination of adlay testa ethanolic extracts (ATE) and doxorubicin has on the growth inhibition of human uterine sarcoma cells. Cell viability is detected by MTT assay after variant ATE 24 h~72 h treatment. Cell cycle and apoptosis was analyzed by flow cytometry. Messenger RNA and protein expression were detected by RT-PCR and western blotting, respectively. Results demonstrated that (1) ATE-Hex had the best effects of inhibition on MES-SA and MES-SA/Dx5 cells. Co-treatment of ATE-Hex and sub-toxic doxorubicin could synergistically or additively inhibit cancer cells proliferation. (2) ATE-Hex reduced the rhodamine efflux in MES-SA/Dx5 cells, indicated that ATE-Hex could reduce P-gp expression. ATE-Hex also could inhibit migration of MES-SA and MES-SA/Dx5 cancer cells. (3) Combination of ATE-Hex and doxorubicin induced apoptosis by increasing sub G1 phase and PARP being cleaved. (4) Analysis of anti-cancer activity, phytosterols had better inhibition on cancer cells growth than fatty acids, especially campesterol and β -sitosterol. These present findings showed that ATE could inhibit on the growth of human uterine sarcoma cancer cells. Furthermore, the combination of ATE and doxorubicin could decrease drug resistance and increase synergistic effect.