🔀 Close Window



Submitted on February 14, 03:05 AM for wcio2012

Proof

CONTROL ID: 1355683

TITLE: Isoliquiritigenin Inhibited Malignant Phenotype of Oral Squamous Cell Carcinoma

AUTHORS (FIRST NAME INITIAL LAST NAME): T. Shieh¹, S. Hsia², Y. Shih³, Y. Huang¹

INSTITUTIONS (ALL): 1. Department of Dental Hygiene, China medical university, Taichung, Taiwan. 2. Department of Nutrition and Health Sciences, Kainan University, Taoyuan, Taiwan.

3. Institute of Oral Biology, National Yang-Ming University, Taipei, Taiwan.

PRESENTATION TYPE: Poster Only

CURRENT CATEGORY: Basic Science

ABSTRACT BODY:

Objectives: Oral cancer is the sixth in numbers of deaths and crude death rates from leading cancer. There is almost 90% oral squamous cell carcinoma (OSCC) in oral cancer population. It is helpful to realize the tumorigenesis mechanism of OSCC and to exploit new anti-cancer drugs, which decrease the death rate of oral cancer. Isoliquiritigenin (ISL) was found in licorice. It has various biological actions, including anti-virus, anti-oxidation, anti-inflammation and anti-ulcer activity. It is also utilized in cancer research, such as prostate and breast cancer. However there are a few research papers about oral cancer.

Methods: We set up normal human oral keratinocyte (OK), oral fibroblast (OF) and 5 OSCC cell lines (Ca9-22, HSC3, OECM-1, SAS and SCC4) culture for anti-oral cancer drugs screening platform. Cell viability is detected by MTT assay after variant ISL dosage 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 malignant phenotypes including cell colony formation, migration, invasion, were using transwell, and anchorage independent ability was using soft agar assay.

Results: The IC50 of ISL in OK, OF and 5 OSCC cell lines were list in table 1. OK, HSC3, and SCC4 were sensitive to ISL, but OF, Ca9-22, OECM-1, and SAS were not. Treat OK, OF, HSC3, OECM-1, and SAS with 25 μ M and 50 μ M ISL, induce cell cycle G2/M arrest and HSC3 apoptosis. ISL induced cell cycle arrest and apoptosis might by ataxia-telangiectasia mutated (ATM) pathway. Furthermore, we verified the malignancy phenotypes of OSCC cells. HSC3 and OECM-1 migration, HSC3, OECM-1 and SAS colony formation, OECM-1 and SAS anchorage independent growth, SAS invasion abilities were inhibited after low dosage ISL 3.125 μ M and 6.25 μ M treated.

Conclusions: The cell cycle arrested at G2/M phase even the cell lines sensitive or insensitive to ISL. The apoptotic cells were dose dependent increased. All cell lines express upper than 95% viability after 3.125 μ M and 6.25 μ M ISL treated, but the cell malignant phenotypes, such as migration, colony formation, anchorage independent growth, and invasion, were significant inhibited. These results indicate ISL is a high potential new anti-cancer drug.

TABLE TITLE:

Cytotoxic activity of ILG (IC50)

Cell lines	IC50 (µM)
ОК	56
OF	>400
Ca9-22	135
HSC3	47
OECM-1	105
SAS	103
SCC4	47

Cytotoxic activity of ILG (IC50)

Cytotoxic activity was determined by MTT assay.

TABLE FOOTER:

Cytotoxic activity was determined by MTT assay.

(No Image Selected)

ScholarOne Abstracts® (patent #7,257,767 and #7,263,655). © <u>ScholarOne</u>, Inc., 2012. All Rights Reserved. ScholarOne Abstracts and ScholarOne are registered trademarks of ScholarOne, Inc.



Terms and Conditions of Use

Product version number 3.14.1 (Build 16) Build date Feb 09, 2012 14:55:03. Server tss1be0015