P37 HINOKITIOL SUPPRESSES ORAL SQUAMOUS CELL CARCINOMA CELLS GROWTH

Y.H. Shih[1], T.M. Shieh[2], S.M. Hsia[3], K.W. Chang[4]

[1]Institute of Oral Biology, National Yang-Ming University, Taipei, [2]Department of Dental Hygiene, China Medical University, Taichung, [3]School of Nutrition and Health Science, Taipei Medical University, [4]School of Dentistry, National Yang-Ming University, Taipei, Taiwan

Background/Aims: Hinokitiol is a compound contained in the wood of Cupressaceae. It is well known to possess the bactericidal activity and practically used in antiseptic agents of foods and cosmetics.In previous studies, hinokitiol shows more toxic effect on human prostate carcinoma and melanoma than the human normal fibroblast cells. In Taiwan, the oral cancer rank five in the top ten leading cancer causes of death and the oral squamous cell carcinoma (OSCC) account for approximately 90% of all cases of oral cancer.The aim of the study is to evaluate the toxic effect of hinokitiol on OSCC cell line and the normal human oral keratinocyte (NHOK) cells.

Methods: The toxic effect of hinokitiol to OSCC cell line, HSC3, and the NHOK cells was detected by MTT assay. The cell cycle and cell apoptosis was detected by flow cytometry. The cancer cell malignant phenotypes were detected by colony formation assay, migration assay and invasion assay.

Results and Discussion: The cell viability of HSC3 and NHOK cells were 40% and 80% after treated with 25 μM hinokitol for 24 h. Hinokitiol induced HSC3 cell cycle arrest at G1 phase and increased cell apoptosis by a dose dependent manner. The cell migration, colony formation, and invasion ability were inhibited effectively in low dose (0.75 μM,1.5 μM) hinokitiol that didn't cause HSC3 and NHOK cells death.It seems that hinokitiol would be a potent anti-cancer drug in the future.

Conclusions: The cell viability of HSC3 cells was lower than NHOK cells after Hinokitiol treatment for 24hr and the HSC3 cells apoptosis increased by a dose dependent manner. The malignant phenotypes of the HSC3 cells were inhibited in a low dosage (0.75μM,1.5μM) hinokitiol treatment that did not cause the HSC3 and NHOK cells death.

P38 BILBERRY ANTHOCYANINS AS ANTI-GLYCATION AGENTS: POSSIBLE THERAPEUTIC POTENTIAL FOR DIABETIC COMPLICATIONS

W.U. Chi-Hao, H.S.I.A. Hsih-Min

School of Nutrition and Health Sciences, Taipei Medical University, Taipei, Taiwan

Diabetes is a life-long disease marked by elevated blood sugar levels. A significant factor associated with hyperglycemia is the resultant nonenzymatic glycation of biological proteins, with the irreversible formation of advanced glycation endproducts (AGEs). AGEs, also known as glycotoxins, are a diverse group of highly reactive compounds with pathogenic significance in diabetic complications and in a number of chronic diseases. The current study was designed to evaluate the effects of bilberry anthocyanins (BA) on AGEs formation and monocyte activation in response to AGEs. The in vivo verification of antiglycation, antioxidant and anti-inflammatory capacities was examined by six-month of bilberry extracts administration in AGEs-induced diabetic rats. In vitro glycation assays demonstrated that BA exerted marked inhibition during the late stages of glycation and subsequent crosslinking. Dual action mechanisms, namely antioxidant and reactive carbonyl trapping activities, may contribute to its antiglycation effect. BAM produced a significant decrease in monocytic IL-1β and COX-2 levels and prevented oxidant formation caused by AGEs, which appeared to be mediated by inhibition of p47phox membrane translocation. Chromatin immunoprecipitation demonstrated that AGEs increased the recruitment of NF-κB transcription factor as well as CBP and CARM1 cofactors to the IL-1β promoter, whereas these changes were inhibited with BA treatment. In vivo, BA reduced tissue AGEs accumulation, tail collagen crosslinking and concentrations of plasma glycated albumin. Levels of oxidative and inflammatory biomarkers were also significantly decreased in BA-treated groups when compared with the diabetic group. These data suggest that BA supplementation may reduce the burden of AGEs in diabetics and may prevent resulting complications.