dose of glucoraphanin. In conclusion, broccoli sprout extracts as a source of sulforaphane either topically or in the diet protect against skin tumor formation.

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Antioxidant activity of flavonoids and other polyphenols isolated from Annona squamosa Linn. leaf extracts

G.A.N. Punzalan[1], G.P. Concepcion[2], R.P. Dator[3], A.R. Mazahery[4], S.D. Jacinto[5]. [1]*UP Institute of Biology, College of Science, Developmental Biology Academic Group, Quezon City, Philippines;* [2]*UP Marine Science Institute, College of Science, Marine Natural Products, Quezon City, Philippines;* [3]*UP Institute of Chemistry, Natural Products Laboratory, Quezon City, Philippines;* [4]*UP Institute of Biology, College of Science, Developmental Biology Program, Quezon City, Philippines;* [5]*UP Institute of Biology, College of Science, Developmental Biology Academic Group, Quezon City, Philippines*

Leaf extracts from Annona squamosa Linn., a ubiquitous fruit tree with recognized medicinal uses, were partitioned with chloroform and subjected to column chromatography and preparative thin-layer chromatography yielding two bands that indicate the presence of the flavonoid polyphenolic compounds. These semipure fractions exhibited the highest inhibitory activity when assayed for the ability to scavenge the diphenylpicrylhydrazyl (DPPH) free radical. This sensitive assay useful even for slow reactions, colorimetrically quantifies the removal of the DPPH free radical generated by the reaction, thus serving as a convenient in vitro monitor of potential oxidative assault on normal cells. At the same time, liquid chromatographic peaks and dereplication of mass spectral data suggest the presence of three polyphenolic compounds including a novel xanthone not yet reported for this plant. This affirms the recognition of plant phenolics' antioxidant properties and consequent antitumor role and may also provide a useful beginning for the characterization and elucidation of this class of phenolic compounds.

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Apoptosis induction by green tea compounds in cervical cancer cells

F. Garcia[1], J.M. Feugang[2], J. Wang[3], S.J. Cheng[4], C.P. Zou[5]. [1]*University of Arizona, College of Medicine, Obstetrics and gynecology, Tucson, United States of America;* [2]*University of Arizona, College of Medicine, Obstetrics and gynecology, Tucson, United States of America;* [3]*University of Arizona, College of Medicine, Obstetrics and gynecology, Tucson, United States of America;* [4]*University of Arizona, College of Medicine, Obstetrics and gynecology, Tucson, United States of America;* [5]*University of Arizona, College of Medicine, Obstetrics and gynecology, Tucson, United States of America*

Human Papilomavirus (HPV) infection is closely associated with the development of over 95% of cervical cancer and 50-60% of head and neck cancer and skin cancer. Clinical trials using several chemopreventive agents are underway, but early results are inconclusive. All agents used in the trials were able to inhibit the growth of cancer cells and about half of the patients responded to the treatment. However, relapse occurred after discontinuation of the drugs. Therefore, selection of non-toxic agents especially food, beverage, and natural products that can suppress HPV virus and inhibit malignant cell growth which can be used long time is vitally important in prevention of cervical cancers. We evaluated green component of EGCG and polyphenol E (poly E) on growth inhibition and apoptosis induction of cervical epithelial cells and cervical cancer cells. HPV-immortalized cervical epithelial cells, TCL-1 and HPV-positive cervical cancer cells, Me180 and HeLa were used in the study. Both green tea compound EGCG and poly E were able to inhibit cervical epithelial and cancer cell growth. Apoptosis induction by EGCG was detected in cervical cancer cells. The growth inhibition and apoptosis induction were in dose-dependent manner. Apoptosis-related genes, such as p53 and p21 were induced by EGCG in cervical cancer cells. The green tea compounds in suppression of HPV-E6 and E7 were also tested by immunohistochemistry. The results of this study provided information on potential mechanisms of green tea compounds in prevention of HPV-related cervical cancer. This information will enable us to assess the feasibility of using these agents in clinical trial setting. This study was supported by the Women's Fund for Health, Education, and Research and the National Institutes of Health, National Cancer Institute (NIH/NCI), grant NOI-CN-35158.

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Cyclooxygenase-2 (COX-2) independent tumor-killing effect of chemical COX-2 inhibitors compared to small interfering RNA of COX-2 in head and neck cancer cell lines

S.W. Park[1], M.W. Sung[2]. [1]*Seoul National University College of Medicine, Department of Tumor biology, Cancer Research Institute, Seoul, South Korea;* [2]*Seoul National University College of Medicine, Department of Otolaryngology-Head and Neck Surgery, Seoul, South Korea*

The observed over-expression of cyclooxygenase-2 (COX-2) in many types of cancer has highlighted this molecule as a potential target for therapeutic intervention. Using head and neck squamous cell carcinoma (HNSCC) cell lines, COX-2 was found to be up-regulated by many oncogenic factors and COX-2 inhibitors exhibited a good anti-tumor effect. However, little physiological change in cell viability by increased prostaglandin E2 (PGE2) was detected, contrary to cases using colon cancer cells. COX-2 inhibitors were found to have an anti-tumor effect at much higher concentrations than doses required to block COX-2 activity. From these considerations, the anti-tumor effect of chemical COX-2 inhibitors was thought to result from a COX-2-independent action at high concentrations in HNSCC cell lines. Firstly, the growth-inhibitory effect of several COX-2 inhibitors was compared with small interfering RNA (siRNA) against COX-2. Additionally, to discriminate between the mechanisms of action of inhibitors and siRNA of COX-2, the effects on intracellular signaling were tested by two inhibitory methods. In conclusion, siRNA against COX-2 was not able to inhibit the proliferation of HNSCC cell lines and its action seemed to differ from anti-tumor action of COX-2 inhibitors. On the other hand, the findings that co-inhibition of both COX-2 and COX-1 may decrease VEGF production partially in HNSCC cell lines imply that anti-cancer effect of