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J Cancer Sci Ther 2015, 7:6
http://dx.doi.org/10.4172/1948-5956.S1.048

5th Asia-Pacific Summit on
Cancer Therapy
July 20-22, 2015 Brisbane, Australia

Epigambogic acid, C-2 epimer of gambogic acid, inhibit cell proliferation mediated ER stress induction in HeLa cells

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Endoplasmic reticulum (ER) plays an important role in the maintain of intracellular calcium homeostasis, protein synthesis, posttranslational modifications and protein folding. Recently, the potential of ER stress in tumor is considered important for regulating the balance between tumor cell death and growth, and for developing the sensitivity of chemotherapeutic agents. Epigambogic acid (EGA) is C-2 epimer of gambogic acid (GA), the main active ingredient in gamboge. The gamboge is a yellowish to orange dry resin secreted from Garcinia hanburyi, a plant that mainly grows in Southeast Asia, India and China. It is traditionally used as a coloring material for painting and has been also used as a folk medicine for an internal purgative and externally infected wound. Most studies demonstrated that GA had potent anti-cancer effects on a broad range of human cancer but no report about the effect of EGA. Therefore, this is the first study of anti-cancer associated ER stress induction effect of EGA on HeLa cells. The results suggest that EGA inhibited the proliferation of HeLa cells by MTT assay. EGA also induced ER stress by the up-regulation of spliced XBP1 mRNA and activated GRP78, CHOP, GADD34 and ERdj4 expression using real-time RT-PCR analysis. Comparing to our previous study, both epimers, GA and EGA, exhibited similar activities against HeLa cells. Overall, these observations might suggest that EGA inhibit cancer cell proliferation via ER stress induction which could be a novel strategy for enhancing chemotherapeutic effect of EGA as an anti-cancer agent.

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Review article diet and the risk of gastric cancer

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There are geographic and ethnic differences in the incidence of gastric cancer around the world as well as with its trends for each population over time. The incidence patterns observed among immigrants change according to where they live. All of these factors serve to indicate the close associations of gastric cancer with modify able factors such as diet. This review presents epidemiological evidence on the association between dietary factors and gastric cancer based on previous systematic reviews and subsequent updates. Infection with Helicobacter pylori is a strong and established risk factor of gastric cancer but is not a sufficient cause for its development. Substantial evidence from ecological, case-control and cohort studies strongly suggests that the risk may be increased with a high intake of various traditional salt-preserved foods and salt per se and decreased with a high intake of fruit and vegetables particularly fruit. However, it remains unclear which constituents in fruit and vegetables play a significant role in gastric cancer prevention. Among them, vitamin C is a plausible candidate supported by a relatively large body of epidemiological evidence. Consumption of green tea is possibly associated with a decreased risk of gastric cancer, although the protective effects have been for the most part identified in Japanese women, most of whom are nonsmokers. In contrast, processed meat and N-nitroso compounds may be positively associated with the risk of gastric cancer. Epidemiologic evidence on the relation between nutrition and stomach cancer is reviewed. Stomach cancer shows a distinct international variation and dramatic worldwide decline. These descriptive features suggest that dietary factors are important in determining the risk of stomach cancer. The authors assessed relevant data regarding specific dietary hypotheses in the etiology of stomach cancer. A negative association with fresh vegetables and fruits is highly consistent in numerous case-control studies in different populations. Both epidemiologic and experimental data suggest that vitamins C and carotenoids lower risk of stomach cancer. Evidence is sparse and inconsistent as to protective effects of vitamin E and selenium. Epidemiologic studies have not lent and will not provide supportive evidence for an etiologic role of nitrate intake. High salt intake has been associated with an increased risk in many case-control studies and limited cohort studies. Taken together with animal data, it is considered that high salt intake is a risk factor for stomach cancer. Both epidemiologic and experimental data are inconclusive as to whether high-starch diets confer an increased risk. Cohort studies using quantitative dietary assessment and biologic measurement of micronutrients are needed for further understanding of etiologic roles of dietary factors in the causation of stomach cancer. In conclusion, dietary modification by reducing salt and salted food intake as well as by increasing intake of fruit and vitamin C represents a practical strategy to prevent gastric cancer.

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