ICCN Poster Presentations

Nutrition and cancer

Vitamin E and its effect on aspirin induce gastric lesion in rats

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This study examined the effects of vitamin E on aspirin induced gastric lesions. The study was divided into two phases: phase 1 determined the effects of various doses of palm vitamin E on the factors affecting mucosal integrity. There was a significant decrease in gastric MDA and gastric acid in all the palm vitamin E supplemented groups compared to control. However, these doses of palm vitamin E had no significant effect on gastric mucus. Phase 2 study determined the effect of multiple doses of palm vitamin E and tocopherol on the prevention of aspirin induced gastric lesions. Fifty rats were randomized into seven groups. Group I was fed a normal diet, Groups II to GroupVII were fed with palm vitamin E/tocopherol enriched diet in a dose of 60mg/20, 100mg/30mg and 150mg/50mg /kg food respectively. After four weeks of feeding, the rats were challenged with a single intragastric dose of aspirin (400 mg/kg body weight). The rats were killed 6 hours post-aspirin exposure for the determination of gastric lesion index and gastric parameters as mentioned in phase I study. The gastric lesions index was significantly lower in all the vitamin E groups compared to control. The lowest ulcer index was observed in the groups that received 100mg of palm vitamin E and 30mg tocopherol in the diet. However, there was no significant difference in ulcer indices between palm vitamin E and tocopherol treated groups. The lower ulcer index was only accompanied by lower gastric MDA content. We conclude that both palm vitamin E in a dose of 60mg, 100mg and 150 mg/kg food as well as tocopherol in a dose of 20mg, 30mg and 50mg/kg food are equally effective in preventing aspirin-induced gastric lesions. The most probable mechanism is through their ability in limiting lipid peroxidation that is involved in aspirin–induced gastric lesions.

The effect of Azadirachta indica on distribution of antioxidant elements and glutathione S-transferase activity in the liver of rats during hepatocarcinogenesis.

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The liver is often the first organ to be infected by metastasizing cancer. Hepatocarcinogenesis is one of the most prevalent and deadly cancers worldwide, which ranks seventh among cancers in order of frequency of occurrence. Numbers of natural and synthetic antioxidants are known to treat initiation and promotion of chemical carcinogenesis in experimental animal models. The effect of 5% w/v of *Azadirachta indica* extract in diethylnitrosamine and acetyl-aminofluorene induced hepatocellular carcinoma, which is a vital mechanism in cancer treatment, was studied in male *Sprague dawly* rats. The result of microscopic observation of the lesion score during hepatocarcinogenesis revealed that cells of cancer group without treatment were severely necrotic at week 12. However, cells of cancer group with *Azadirachta indica* treatment appeared nearly normal. The tracking of the elements during hepatocarcinogenesis was done using energy filtering transmission electron microscope (EFTEM). According to EFTEM results, some of anti-oxidant elements such Na, Ca, and P is highly distributed in *Azadirachta indica* treated normal and cancer group. However, the distribution is too low in normal control and cancer control group without *Azadirachta indica* treatment. The obtained results have shown a significant, decrease (P=0.05) of liver cytosol Glutathione S-transferase in cancer control group rats. Meanwhile, treatment with *Azadirachta indica* caused overall increase in liver GST activity nearly to control group. Distinct evidence from this study contribute that oral administration of 5% *Azadirachta indica* extract demonstrated anticancer activity by increasing the distribution of antioxidant elements and GST activity may to protect cells in preneoplastic nodules in cancer treated groups. However, there was no evidence of side effects of Azadirachta *indica* towards normal cells indicating *Azadirachta indica* as a potential preventive agent for cancer.