

ICCN Poster Presentations

Food, inflammation and the anti-inflammatory aspects of food

In vitro and ex vivo cyclooxygenase inhibition by a hops extract

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While there has been much research on botanical materials as potential pain-relieving Cox inhibitors, it has not yet been demonstrated that oral consumption of botanical agents can inhibit Cox-2 activity in humans. In particular it would be of interest to determine whether any botanical anti-inflammatory has Cox-1-sparing activity, in order to reduce the risk of gastrointestinal side effects. This two-stage study was designed to first screen a variety of botanicals *in vitro*, and then to select one or more promising agents to test in human volunteers.

Method: Seventeen botanical agents, putative anti-inflammatories or pain-relievers all, were evaluated *in vitro* for Cox-1 and -2 inhibitory potency and selectivity using a caco-2 cell line with ibuprofen as an active control. A promising compound, a hops extract high in alpha acids, showed a Cox-2/Cox-1 IC₅₀ selectivity ratio of 0.06, compared to 4.2 for ibuprofen. Two different formulations of a standardized hops extract (resin and powder) were compared with ibuprofen in a double-blind, randomized, *ex vivo* study. Subjects consumed hops powder extract, hops resin extract, or ibuprofen, and provided blood samples before and at intervals for 9 h following the first dose. Plasma was extracted and analyzed in a validated Cox-1 and -2 inhibition assay.

Results: There were no differences between active treatments or ibuprofen control in Cox-2 inhibitory action, as indicated by 9-hour Cox-2 Area over the Inhibition Curve (AOC); however, hops powder or hops resin extract produced a 9-hour Cox-1 / Cox-2 AOC ratio of about 0.4 (i.e., some degree of Cox-1 sparing), compared to 1.5 for ibuprofen (i.e. no Cox-1 sparing).

Conclusion: Hops exhibited Cox-2 inhibition over 9 hours equivalent to ibuprofen 400 mg but had significant Cox-1 sparing activity relative to ibuprofen. Hops extracts may represent a safe alternative to ibuprofen for non-prescription anti-inflammation.

Assessment of micronutrient antioxidants, total antioxidant capacity and lipid peroxidation levels in liver cirrhosis.

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Background: The profile of micronutrient antioxidants in serum may be helpful in assessing lipid peroxidation and inflammation in cirrhotic. Acute phase proteins (APRs) are anti infective, anti-inflammatory, procoagulant and scavengers. Saturation of transferrin (Trf) level in the liver is more susceptible to peroxidative damage of functional integrity of the cells and affects the antioxidant activity. Therefore, we hypothesized decreased intake of micronutrient antioxidant may influence total antioxidant capacity, lipid peroxidation and acute phase reactants.

Objective: To determine the vitamin E, vitamin C, total antioxidant capacity (TAC), lipid peroxidation (MDA), and acute phase reactants in patients with alcoholic and non-alcoholic cirrhosis and compared them with those of healthy controls.

Method: Forty patients with alcoholic cirrhosis as well as non alcoholic cirrhosis and 50 healthy controls were enrolled in the department of Gastroenterology and Human Nutrition, AIIMS, New Delhi. Serum vitamin E and C, APRs and MDA levels were measured and compared with those of controls.

Results: MDA and C-reactive protein (CRP) levels were significantly higher ($p < 0.000$) in alcoholic liver cirrhotics whereas CRP levels were not significant in non alcoholic cirrhotics. MDA levels were significantly higher ($p < 0.05$) in patients with non alcoholic cirrhosis. Total antioxidant capacity was increased in alcoholic cirrhosis while in non-alcoholic cirrhosis were decreased. Vitamin E and vitamin C levels were significantly lower ($p < 0.001$) in both alcoholic and non-alcoholic cirrhotics. Furthermore, transferrin (Trf) levels were significantly higher ($p < 0.000$) in both the groups as compared to healthy controls (Table).

Group (n=40)	Vit-E (μ M/L)	Vit-C (mg/dl)	MDA (nM/ml)	CRP (mg/dl)	Trf (mg/dl)	TAC (μ M/L)
Alcoholic cirrhosis	17.5 \pm 3.62	0.26 \pm 0.12	3.92 \pm 1.64	1.23 \pm 0.41	540 \pm 65.12	1.98 \pm 0.60
Nonalcoholic cirrhosis	16.8 \pm 4.2	0.39 \pm 0.17	3.16 \pm 1.48	0.45 \pm 0.13	375 \pm 36.45	1.69 \pm 0.18
Healthy control (n=50)	24.7 \pm 4.2	0.66 \pm 0.15	2.65 \pm 1.45	0.42 \pm 0.16	285.2 \pm 20.45	2.46 \pm 0.334

Conclusion: Decreased level of vitamin E and C and increased level of total antioxidant capacity indicated that breakdown of antioxidant defence could be the cause of development and progression of liver cirrhosis.