Poster

[P25-11] P25-11: Clinical toxicology (1)

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[P25-11-2] Mitigation of lung toxicity induced by B(a)P via modulation of oxidative stress and inflammation by carvacrol in mice

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Keywords: Carvacrol, benzo(a)Pyrene, lung toxicity

Background

Benzo(a)pyrene [B(a)P] is a ubiquitous environmental carcinogen present in coal tar, tobacco smoke, food like grilled/barbecued steaks, hamburger, chicken, etc. The commercial promotion and use of foods supplemented with carvacrol to protect against environmental carcinogens is growing high globally; although the scientific basis behind such practice is obscure. The present study was conducted to evaluate the mechanism, if any, of the protective effects of carvacrol against B(a)P induced lung toxicity in experimental mice.

Methods

Five groups of Swiss albino mice (n=6 in each group) were treated with: Group 1: corn oil; Group 2: B(a)P (125mg/kg, single dose oral on 7th day); Group 3: carvacrol (25 mg/kg oral) for 7 days + B(a)P (same dose); Group 4: carvacrol (50 mg/kg oral) for 7 days + B(a)P (same dose), and Group 5: carvacrol (50 mg/kg oral) for 7 days. Different antioxidant enzyme activities, histopathological changes and inflammatory (iNOS, NFkB, COX-2) markers were assessed. Routine toxicity study was performed in Group 5 animals. Tukey-Kramer multiple comparisons test was used for analysis.

Results

Carvacrol treatment at low dose (Group 3) significantly increased the activities of glutathione peroxidase ($p<0.01$), glutathione reductase ($p<.01$), glutathione-S-transferase ($p<.001$) and catalase ($p<.01$) and significantly decreased the activities of xanthine oxidase ($p<.05$) and malondialdehyde enzymes ($p<.05$) as compared to the animals of Group 2. Higher dose of carvacrol was found more effective. Protein expressions (iNOS, NFkB and COX-2) in lung were found to be upregulated by B(a)P, which were reversed by carvacrol. Carvacrol also restored the histopathological changes against B(a)P-induced lung toxicities. Carvacrol was also found to be safe in routine toxicity study.

Conclusions

Carvacrol in both the doses was protective against B(a)P-induced lung toxicity. Prevention of oxidative stress and inflammation were the probable mechanisms involved. Clinical trials are warranted to further explore its protective role against environmental toxins. Dietary supplementation with carvacrol might have a scientific basis as well.