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Methanolic bark extract of *Acacia catechu* (L.f.) willd attenuates Benzo(a)pyrene induced oxidative stress, inflammation and apoptosis in the lung of mice. Possible role of TNF-α, p53, Bax

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Background: Benzo(a)pyrene (B(a)P) is a well-known carcinogen present in an environment. Its main source is tobacco smoke, forest fire and automobile exhaust. Extract of *Acacia catechu* (L.f.) willd bark has shown various pharmacological properties including anti-inflammatory, antioxidant, anti-lipid peroxidative, anti-hyperglycemic and protect against DNA strand breaks.

Purpose: The purpose of this study was to investigate the protective effects of methanolic extract of the bark of *Acacia catechu* (L.f.) willd (MEBA) against B(a)P induced oxidative stress, inflammation, and apoptosis.

Study design: Thirty male Swiss albino mice were randomly allocated into five groups having six animals in each group. MEBA was given orally once daily for seven consecutive days at the doses of 200 and 400 mg/kg body weight in the distilled water. On day 7, a single dose of B(a)P was given orally in the corn oil at the dose of 125 mg/kg body weight.

Methods: After 24 hours of last treatment, mice were sacrificed then toxicity markers (lipid peroxidation, lactate dehydrogenase and xanthine oxidase activity), antioxidant enzymes activities (catalase, superoxide dismutase, glutathione reductase, and glutathione peroxidase glutathione-S-transferase and quinone reductase) and reduced glutathione content were measured by different methods. Expression of NF-kB, COX-2, TNF-α, p53, Bax, Caspase-3 and bcl-2 were measured by Immunohistochemistry and histopathology also was done.

Results: Pretreatment with MEBA significantly attenuated B(a)P-induced induced lipid peroxidation, lactate dehydrogenase activity, xanthine oxidase activity, glutathione depletion, decrease in antioxidant enzymes (catalase, superoxide dismutase, glutathione reductase, and glutathione peroxidase glutathione-S-transferase and quinone reductase) activities. MEBA also attenuated expression of NF-kB, COX-2, TNF-α, p53, Bax, Caspase-3, and bcl-2. Histological results further confirmed the protective role of MEBA against B(a)P-induced lung toxicity.

Conclusion: The data of the present study showed that MEBA successfully suppresses B(a)P induced lungs damage by ameliorating oxidative stress, inflammation, and apoptotic responses.

Biography:

Sarwat Sultana is an Associate Professor in the Department of Medical Elementology & Toxicology at Jamia Hamdard (Hamdard University), India. She has 25 years of research experience in Cancer Biology. Her research specialization is Metabolic basis of chemical carcinogenesis, correlation of alteration in DNA with cancer induction. Targeting the molecular mechanism and elucidation of their plausible role in induction of carcinogenesis. She is a member and life member of several societies of Toxicology. She has 189 papers published in International Journals of high repute.