ABSTRACT

KEYWORDS: *Commiphora berryi*, Pharmacognosy, Antiulcer, Antitumor, Hepatoprotective, Antioxidant.

The present thesis deals with the exploration of Pharmacognostical and Pharmacological Evaluation of Stem Bark of *Commiphora berryi* (Arn) Engl, which is traditionally used by the local people in South India for the treatment of ulcer.

The *Commiphora berryi*, a small tree, was collected from Namakkal district of Tamil Nadu state, India and authenticated at Botanical Survey of India, Southern Circle, Coimbatore District, Tamil Nadu, India. For the present study the stem bark of the plant was used. The macroscopical, microscopical evaluation, analytical parameters, microchemical studies were carried out for its identification. Heavy metal analysis of the methanol extract of *Commiphora berryi* was carried out to find out whether the heavy metals are within the permissible limit, as per WHO guidelines.

The total methanol extract was used instead of isolated compounds, since in Ayurvedic and Herbal medicine practice, the total extract is used as therapeutic agent instead of isolated compounds on the scientific approach that certain components in the extract nullify the side effects of other components.

In preliminary phytochemical analysis, the methanol extract of *Commiphora berryi* (MECB) showed the presence of phytoconstituents such as carbohydrates, gum, mucilage, phytosterols, tannins, phenolic compounds and triterpenoids.

The MECB was subjected to column chromatography for the separation of its phytoconstituents. The fractions obtained from column chromatography were subjected to gas chromatography coupled with mass spectrometry (GC-MS).
GC-MS study confirmed that MECB is having 38 prominent compounds among them with their biological activities reported in Dr. Duke's Phytochemical and Ethnobotanical Databases. They are phenol 2, 4-\textit{bis} (1, 1-dimethylethyl) - (analgesic, antioxidant and cancer preventive), dodecanoic acid- (antioxidant, COX-1 and COX-2 inhibitor), n-hexadecanoic acid- (antioxidant), 9, 12-octadecadienoic acid- (anti-inflammatory, cancer preventive, hepatoprotective, antiarthritic and antihistaminic activities), and 1, 2-benzenedicarboxylic acid, mono (2-ethylhexyl) ester- (anti-inflammatory activity). Hence the antiulcer, analgesic, anti-inflammatory, anti-tumor, hepatoprotective and antioxidant activities of \textit{Commiphora berryi} stem bark have been investigated.

Acute oral toxicity study of MECB was conducted as per OECD-423 guidelines and it showed no mortality or acute toxicity up to 3 g/kg, b. wt. by oral dose.

Antiulcer activity of MECB was studied by aspirin plus pylorus ligation and stress induced ulcer models in rats. In aspirin plus pylorus ligation model, when compared with ranitidine treated group, the group treated with 500 mg/kg extract showed marginal activity and the group treated with 750 mg/kg extract showed significant activity, which was higher than that of the ranitidine treated group. In stress induced ulcer model, when compared with ranitidine treated group, the groups treated with MECB 500 mg/kg and 750 mg/kg showed significant activity which was equal to that of ranitidine treated group. The above result confirms the antiulcer activity of \textit{Commiphora berryi} stem bark.

MECB was screened for the anti-inflammatory, analgesic and antipyretic activity by the methods like carrageenan induced hind paw edema (acute), formaldehyde induced hind paw inflammation (sub-acute), cotton pellet induced granuloma (sub-chronic), acetic acid induced writhing response, formalin test, tail immersion test and
Brewer’s yeast induced hyperthermia. The effect of MECB in edema reduction was compared with indomethacin (10 mg/kg). In acute model of inflammation, oral administration of MECB at 750 mg/kg suppressed the edematous response 2 h after carrageenan injection and this effect continued up to 6 h. In formaldehyde induced hind paw inflammation model, oral administration of MECB considerably reduced the paw inflammation by dose-dependent manner. In cotton pellet induced granuloma model, MECB and indomethacin treated orally showed marked inhibition in granuloma weight. In acetic acid induced writhing response model, an increase in the doses of MECB resulted in a greater inhibition (dose-dependent fashion). In formalin test, MECB (750 mg/kg) was able to block both phases of formalin response but the effect was more pronounced in the second phase. In the tail immersion test method, the MECB in a dose of 750 mg/kg showed lesser anti-nociceptive activity than pentazocine at 2 hour. In antipyretic study, MECB showed considerable reduction in pyrexia, induced by yeast in rats.

The antitumor activity of MECB was investigated at the dose levels of 500 mg/kg and 750 mg/kg against Ehrlich ascites carcinoma (EAC) cells for nine days. On tenth day, six mice from each group were sacrificed for the study of antitumor activity. The haematological parameters and antioxidant activities in lipid peroxidation (LPO), reduced glutathione (GSH), superoxide dismutase (SOD) and catalase (CAT) were measured. The remaining animals in each of the groups were kept to check the life span of the tumor bearing mice. From the results, it is seen that MECB dramatically inhibits the tumor growth induced by EAC cell line by altering the LPO and antioxidant system in EAC treated mice. This antitumor activity of MECB might be due to the antioxidant potential of the plant.
MECB was tested for the hepatoprotective and antioxidant activity induced by carbon tetrachloride in rats at the dose of 100 mg/kg and 200 mg/kg and standard drug, silymarin at 25 mg/kg. The potential of MECB on liver markers and antioxidant liver enzymes was measured. The MECB and silymarin exhibited significant hepatoprotective effect by reducing the amount of serum enzymes, bilirubin. In antioxidant system, the liver enzyme level of SOD, CAT and glutathione peroxidase increased in a dose dependent manner. The above results reveal that the hepatoprotective effect of MECB may be due to its antioxidant and free radical scavenging properties.