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BIOCHEMICAL STUDIES ON THE ANTI-ULCEROGENIC POTENTIAL OF *CARDIOSPERMUM HALICACABUM* L. SEEDS

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ABSTRACT

Cardiospermum halicacabum L. var. *microcarpum* (Kunth) Blume and var. *luridum* (Blume) Adelb. (Sapindaceae) are used for various ailments in Indian traditional medicines. The present study evaluated the antiulcerogenic property of seed oils (Petroleum ether extracts) in animal model. Ethanol induced gastric ulcers was used for this study and analysed for gastric volume, ulcer score pH, free and total acidity and sodium and potassium ions. Bio-chemical estimations like total proteins, total hexoses, hexosamine, fucose, sialic acid and protein were also made. Ulcer score was calculated for the same model. Oils were found to be more effective and exhibit concentration dependent anti-ulcer property.

INTRODUCTION: Gastric ulcer is a common disorder where discontinuity in the gastric mucosa is observed. The conventional treatment of ulcer comprises of regular feeds and adequate rest, antacid, various drugs and avoidance of ulcerogenic agents such as coffee, alcohol and tobacco. The drugs used in the treatment of ulcer include receptor blockers, proton pump inhibitors, drugs affecting the mucosal barrier and act on the central nervous system [1]. Eventhough a range of drugs and available for the treatment of ulcer, many of these do not fulfill all the requirements and side effects such as arrhythmias, impotence and hematopoietic changes are noted [2 & 3].

Cardiospermum halicacabum L. is an herbaceous climber found throughout the plains of India. This plant commonly known as "Kanphuti" is used in Ayurveda and folk medicine for the treatment of rheumatism, lumbago, earache and fever [4]. In Unani literature, the seeds are used as anticancerous [5].

The seed oil has been analysed for its fatty acid composition [6]. The antiinflammatory activity of ethanol extract has been shown against carrageenin induced rat paw oedema [7]. Previous studies the remarkable antiulcerogenic activity of ethanol extract 200-600mg/kg of this plant against ethanol induced ulcers in rats [8]. The reported chemicals of the plant contains tannins, saponins and traces of alkaloids [9].

It is known the ethanol is among many factors increasing risk of gastric ulcer formation due to stress. Ethanol is widely used to induce experimental gastric ulcer in animals [10].

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The present study was undertaken to establish the antiulcer activity of seed oils of two varieties of *C. halicacabum* on animal model for ulcer studies.

MATERIALS AND METHODS:

Test Materials: The plant materials were collected from Thirumalairayan Pattinam, Karaikal, Puducherry state, The Union Territory of India. Voucher specimen was kept at the Herbarium of Tamil University, Thanjavur, Tamil Nadu, South India.Voucher specimen No.TUH51A and TUH51B. The plant material was identified with the help of Floras [11, 12 & 13]. The seeds were shade dried, powdered and subjected to hot extraction method using petroleum ether through Soxhlet apparatus [14]. The oil was stored in sealed vials in a refrigerator (5-8) and subjected for screening of antinuclear study.

Animals: Wistar strain albino rats of either sex weighing between 130-150 g were taken from the inbreed group maintained at Tamil University animal house, Thanjavur. The animals were fed with standard pellet diet supplied by Lipton & Co. Ltd., Bangalore. Water was made available to animal's ad-libitum. The animal experiments were carried out in accordance with animal ethical committee norms.

Statistical Analysis: Data were expressed as mean ± S.D and statistical analysis was carried out using student's t-test.

Ethanol – induced Gastric Ulcer: The gastric ulcers were induced in rats of either sex weighing between 130-150 by administrating absolute ethanol (8ml/kg). They were kept in specially constructed cages to prevent coprophagia during and after the experiment. The rats were divided into nine groups each containing six animals and fasted for 24h and allowed free access to water. The first group received distilled water and second group received ethanol only. The third group received ethanol and standard anti-ulcer drugs Ranitidine (150mg/kg). The 4-6th groups were given absolute ethanol and oil of *C. halicacabum* var. *microcarpum* at *a* dose of 3.3, 6.6, and 9.9ml/kg respectively. The 7-9th group received absolute ethanol and oil of *C. halicacabum* var. luridum at a doses of 3.3, 6.6 and 9.9 ml/kg.

The drugs were administered orally 30 min. prior to the oral administration at absolute ethanol. The animals were anaesthetized 6th h later with ether and stomach were incised along the greater curvature, collected the gastric juice and ulceration was scored [15].

1. **Biochemical parameters:** The stomach was excised carefully keeping the esophagus closed, opened along the greater curvature and luminal contents were removed. The gastric contents were collected in a beaker and centrifuged at 1000rpm for 10min. The samples were analysed for gastric volume, pH, free and total acidity and sodium and potassium output. Biochemical estimations like total proteins, total hexoses, hexosamine, fucose, sialic acid and pepsin were also made. The mucosa was flushed with saline and stomach pinned on a frog board and scored.

2. **Collection of Gastric Juice:** Gastric juice was collected from the ethanol induced ulcer rats. The gastric juice thus collected was centrifuged and the volume of gastric juice as well as pH of gastric juice was measured [16]. Then the gastric juice was subjected to bio-chemical estimation as follows.

3. **Determination of Free and Total Acidity in Gastric Juice [17]:** 1ml of gastric juice was pipetted into a 100 ml conical flask, added 2 to 3 drops of Topfer's reagent and triturated with 0.01 N NaOH (which was previously standardized with 0.01 N of oxalic acid) until all traces of the red colour disappears and the colour of solution was yellowish orange. The volume of alkali added was noted. The volume corresponds to free acidity. Then 2 to 3 drops of phenolphthalein solution were added and tituration was continued until a definite red tinge reappears. Again the total volume of alkali added was noted. The volume corresponds to total acidity.

Acidity=

$$\frac{\text{Volume of NaCl} \times \text{Normality of NaOH} \times 100}{0.1 \text{ meq}/1/100 \text{ g}}$$

4. **Sodium and Potassium ion concentration in Gastric Juice [18]:** This was carried out using Systronics Mediflame 127-Flame Photometer. Stock solution was prepared by dissolving 2.542 g

NaCl in 1 litre of distilled water. It contains 1mg Na per ml (i.e., 1000 ppm). Stock solution was diluted to give four solutions containing 10, 5, 2.5 and 1 ppm of sodium ions. Then Potassium stock solution was prepared by dissolving 1.909 g KCl in 1 litre of distilled water. It contains 1 mg potassium per ml (i.e., 1000 ppm). Stock solution was diluted to give four solutions containing 20, 10, g and 2 ppm of potassium ions. For sodium and potassium, the flame intensity corresponding to the concentration of stock solution was noted by using appropriate filters. The results were plotted in a graph. The flame intensity of the gastric juice was noted. The concentration of sodium and potassium ions was calculated from the graph. The results are expressed in terms of mg/l.

5. **Total Proteins [19]:** The dissolved protein in gastric juice was estimated in the alcoholic precipitate obtained by adding 90% alcohol with gastric juice in 9:1 ratio. Then 0.1 ml of alcoholic precipitate of gastric juice was dissolved in 1 ml of 0.1 N NaOH and from this 0.05 ml was taken in another test tube, to this 4 ml of alkaline mixture was added and kept for 10 min. Then 0.4 ml of phenol reagent was added and again 10 min. was allowed for colour development. Reading was taken against blank prepared with distilled water at 610 nm in Hitachi 15-20 spectrophotometer. The protein content was calculated from standard curve prepared with bovine albumin and was expressed in terms of μg/ml of gastric juice.

6. **Estimation of Total Carbohydrates [20]:** The dissolved mucosubstances in gastric juice were estimated in the alcoholic precipitate obtained by adding 90% alcohol with gastric juice in 9:1 ratio. Briefly the method consists of taking two aliquots of gastric juice and treated as described. To 1 ml of gastric juice, 9 ml of 90% alcohol was added. The mixture was kept for 10 min. before it was centrifuged. The supernatant was discarded. The precipitate was dissolved in 0.5 ml of 0.1 N NaOH. To this 1.8 ml of 6 N HCl was added. The mixture was hydrolysed in water bath at 100°C for 2h. The hydrolysate was neutralized by 5 N NaOH using phenol phthalein as indicator and the volume was made upto 4.5 ml with distilled water and used for the estimation of total hexoses, hexosamine and fucose as described to the other aliquot of 0.5 ml of gastric juice, 4.5 ml of alcohol was added. The mixture was shaken for 10 min. and centrifuged to obtain precipitate. The precipitate was dissolved in 0.5 ml of 0.1 N H_2SO_4. This reconstituted solution was transferd to glass-stoppered tubes and then hydrolysed in a water bath at 100°C for 1 h. The hydrolysis, the volume restored to 0.5 ml; 0.2 ml of this hydrolysate was used for the estimation of sialic acid. After obtaining the concentration (μg/ml) of individual carbohydrates namely, hexoses, hexosamine, fucose. Sialic acid and the total carbohydrate content were calculated by adding the concentration of individual carbohydrates. Mucosubstances activity has been expressed as ratio of total carbohydrates to total protein.

a. **Hexoses [21]:** To 0.4 ml of hydrolysate, 3.4 ml of orcinol reagent was added. The mixture was then heated in the boiling water bath at 60°C for 15 minutes. This was then cooled under running tap water and intensity of the colour was read in Hitachi 15-20 spectrophotometer at 540 nm against the blank by using distilled water instead of hydrolysate. Total hexoses content was determined from the standard curve of D(+)-galactose-mannose and has been expressed in μg/ml of gastric juice.

b. **Hexosamine [22]:** About 0.5 ml of the hydrolysate fraction was taken. To this 0.5 ml of acetyl-acetone reagent was added. The mixture was heated in boiling water bath at 60°C for 20 minutes, then cooled under running tap water. 1.5 ml of 90% alcohol was added and allowed for 30 minutes. The colour intensity was measured in Hitachi 15-20 spectrophotometer at 530 nm against blank prepared by using distilled water instead of hydrolysate. Hexosamine content was determined from the standard curve prepared by using D (+)-glucosamine hydrochloride and concentration has been expressed in μg/ml of gastric juice.

c. **Fucose [23]:** In this method, three test tubes were taken. In one tube 0.4 ml of distilled water was taken to serve as control and in each of the other two tubes 0.4 ml of hydrolysates were taken. To

all three tubes 1.8 ml of H_2SO_4: water (6:1) was added by keeping the test tubes in ice-cold water bath to prevent breakage due to strong exothermic reaction. The mixture was then heated in boiling water bath for exactly 3 minutes. The tubes were taken out and cooled. To the blank and to one of the hydrolysate containing tube (unknown), 0.1 ml of cysteine reagent was added while cysteine reagent was not added to the last test tube containing the hydrolysate (unknown blank). It is then allowed for 90 min. to complete the reaction. The reading was taken in Hitachi 15-20 spectrophotometer at 396 and 430 nm setting zero with the distilled water. The optical density for the fucose in the hydrolysate was calculated from the differences in the reading obtained at 396 and 430 nm and subtracting the values without cysteine. This as read against standard curve prepared with D(+)-fucose. The fucose content was expressed in terms of μg/ml of gastric juice.

True optical density =

$$\frac{(OD_{396} - OD_{430})^{\text{Unknown}} - (OD_{396} - OD_{430})^{\text{Unknown blank}}}{(OD_{396} - OD_{430})^{\text{water blank}}}$$

d. **Sialic acid [24]:** To 0.5 ml of the hydrolysate in 0.1 N H_2SO_4, 0.2 ml of sodium periodate was added and mixed thoroughly by shaking. A time of 20 min. was allowed to elapse before addition of 1 ml of sodium arsenite solution to this mixture. The brown colour produced disappeared after shaking. Then 3 ml of thiobarbituric acid was added and the mixture was heated in boiling water bath for 15 min. After cooling the tubes, 4.5 ml of cyclohexanone was added and thorough shaking was done for 15 seconds till at the colour was taken up by the cyclohexanone supernatant.

The mixture was centrifuged to get a clear pink layer of cyclohexanone. This supernatant was pipetted out and intensity of colour was measured in Hitachi 15-20 spectrophotometer at 550nm. The sialic acid content of the sample was determined from the standard curve of sialic acid and has been expressed in terms of μg/ml of gastric juice.

e. **Pepsin [25]:** Four tubes (1) and (2) containing 5 ml of substrate, (3) and (4) containing 10 ml of TCA was placed in the water bath at 37°C. The gastric juice was mixed with an equal volume of HCl at pH 2.1, warmed to 37°C and added 1 ml of mixture to each tubes (1) and (4), incubated for 15 min. and at the end mixed the contents of tube (1) with tube (3) and allowed to stand in the bath for about 4 minutes. Contents of tube (1) and tube (3) give test and contents of tube (2) and tube (4) give blank. Both the contents were filtered after 25-30 min. 2 ml of filtrate was pipetted into 10 ml of NaOH, mixed by gentle rotation, then 1 ml of phenol was added and again mixed by gentle rotation. After 30 min., the intensity of colour was measured at 680 nm in Hitachi 15-20 spectrophotometer.

The difference between test and blank gives a measure of peptic activity. As standard, mixed 2 ml of freshly prepared phenol solution containing 50 μg/ml with 10 ml of NaOH and 1 ml of phenol reagent was added. After 5-10 min. the colour intensity was measured at 680 nm.

Zero corresponds to normal rugal pattern, 1 for alteration in normal rugal pattern, 2 for ten scattered hemorrhage lesions, 3 for hemorrhage lesion and ulcers and 4 for penetration and perforating ulcers.

RESULTS: The seed oils of two varieties of *Cardiospermum halicacabum* showed anti-ulcer activities in all parameters studied comparable with that of reference and control groups. The oil of *C. halicacabum* var. *microcarpum* on ulcer index, gastric volume and other biochemical parameters are shown in **Table 1**. The oil significantly reduce the gastric volume, ulcer score, total acidity and protein and increased protective parameters like hexose, hexosamine etc. were compared to untreated ethanol administrated control. The reference drug Ranitidine (30mg/kg) was found to be better than the oil reducing the ulcer as observed through all the above parameters. The oil of var. *luridum* also exhibited more are less similar anitulcer activity as observed by all the parameters (**Table 2**). However, the activity is less when compared to var. *microcarpum.*

TABLE 1: EFFECT OF ANTIULCER PROPERTY OF *C. HALICACABUM* VAR.*MICROCARPUM* ON ETHANOL-INDUCED GASTRIC ULCERS IN RATS

Group	Animal weight	Gastric volume (ml)	Ulcer score	pH	Na^+ mg/l	K^+ mg/l	Free acidity (mg/l)	Total acidity (mg/l)	Pepsin (µg/ml)	Total protein (µg/ml)	Total carbohydrates (µg/ml)				Carbohydrate and protein ratio
											Total hexose	Hexo samine	Fucose	Salic acid	
Control	152.166 ± 3.37	2.073 ± 0.1474	3.333 ± 0.082	2.233 ± 0.034	2.108 ± 0.082	0.87 ± 0.033	42.33 ± 1.505	70.33 ± 1.033	8.075 ± 0.076	617.5 ± 14.404	188.83 ± 3.970	281.33 ± 3.72	51.66 ± 1.032	34.33 ± 0.82	0.92 ± 0.04
Standard	153.5 ± 5.5***	5.092 ± 0.128***	1.033 ± 0.137***	5.0833 ± 0.194***	1.71 ± 0.054***	0.7116 ± 0.017***	32 ± 0.632***	49.66 ± 1.032***	5.055 ± 0.166***	317.66 ± 4.131***	312 ± 3.162***	361.83 ± 1.17***	30.33 ± 1.86***	46 ± 0.0632***	2.32 ± 0.07***
3.8 ml/kg	153.81 ± 6.533***	4.12 ± 0.175***	2.1 ± 0.063***	2.8716 ± 0.109*	1.805 ± 0.221*	0.85 ± 0.0098^{NS}	41.833 ± 1.602^{NS}	61.33 ± 0.816***	7.38 ± 0.257***	473.5 ± 2.664***	212 ± 4.33***	287.3 ± 3.66^{NS}	46 ± 1.79***	42.66 ± 0.780***	1.24 ± 0.03***
6.6 ml/kg	151.631 ± 4.261***	3.428 ± 0.150***	2.85 ± 0.083***	3.4 ± 0.4***	1.922 ± 0.004**	0.83 ± 0.022^{NS}	38.5 ± 0.836**	54.66 ± 1.032***	6.24 ± 0.38***	384.83 ± 3.060***	2.67 ± 3.544***	317.16 ± 4.53***	37.5 ± 0.837***	38.033 ± 0.62***	1.03 ± 0.02***
9.9 ml/kg	152.111 ± 4.912	2.732 ± 0.519*	2.533 ± 0.051*	4.166 ± 0.382***	1.845 ± 0.03***	0.812 ± 0.0132*	37.5 ± 0.836***	48 ± 0.632***	6.395 ± 0.248***	345 ± 7.071***	273 ± 3.656***	323.16 ± 3.763***	36.5 ± 1.870***	37.6 ± 0.521***	1.94 ± 0.06***

Values are expressed as mean ± SD, n = 6 compared to control; *** $p \leq 0.001$; ** $p \leq 0.01$; * $p \leq 0.05$; NS – Not significant

TABLE 2: EFFECT OF ANTIULCER PROPERTY OF *C. HALICACABUM* VAR. *LURIDUM* ON ETHANOL-INDUCED GASTRIC ULCERS IN RATS

Group	Animal weight	Gastric volume (ml)	Ulcer score	pH	Na^+ mg/l	K^+ mg/l	Free acidity (mg/l)	Total acidity (mg/l)	Pepsin (µg/ml)	Total protein (µg/ml)	Total carbohydrates (µg/ml)				Carbohydrate and protein ratio
											Total hexose	Hexo samine	Fucose	Salic acid	
Control	152.166 ± 3.37	2.073 ± 0.1474	3.333 ± 0.082	2.233 ± 0.034	2.108 ± 0.082	0.87 ± 0.033	42.33 ± 1.505	70.33 ± 1.033	8.075 ± 0.076	617.5 ± 14.404	188.83 ± 3.970	281.33 ± 3.72	51.66 ± 1.032	34.33 ± 0.82	0.92 ± 0.04
Standard	153.5 ± 5.5***	5.092 ± 0.128***	1.033 ± 0.137***	5.0833 ± 0.194***	1.71 ± 0.054***	0.7116 ± 0.017***	32 ± 0.632***	49.66 ± 1.032***	5.055 ± 0.166***	317.66 ± 4.131***	312 ± 3.162***	361.83 ± 1.17***	30.33 ± 1.86***	46 ± 0.0632***	2.32 ± 0.07***
3.3 ml/kg	154.167 ± 8.166***	3.675 ± 0.319***	3.033 ± 0.051***	2.05 ± 0.137^{NS}	1.928 ± 0.033**	0.845 ± 0.013^{NS}	36 ± 0.632***	64 ± 1.414***	7.34 ± 0.177***	500.83 ± 435***	202.5 ± 4.415***	282.166 ± 3.311^{Ns}	49.833 ± 1.471^{ns}	43.833 ± 0.983***	1.15 ± 0.04***
6.6 ml/kg	153.131 ± 5.522***	3.063 ± 0.076***	2.816 ± 0.075***	3.15 ± 0.137***	1.84 ± 0.037***	0.805 ± 0.008**	32.83 ± 0.752***	60.166 ± 0.752***	8.791 ± 0.329***	457.33 ± 6.377***	251.166 ± 1.329***	307.5 ± 6.745**	44.33 ± 1.505***	41.833 ± 0.752***	1.40 ± 0.16***
9.9 ml/kg	151.831 ± 8.74***	2.125 ± 0.128^{ns}	2.566 ± 0.051***	3.9 ± 0.089***	1.858 ± 0.044***	0.795 ± 0.010**	29.66 ± 1.032***	55.166 ± 1.722***	6.733 ± 0.062***	420.83 ± 2.483***	262.833 ± 3.763***	319.83 ± 1.940***	42.33 ± 2.065***	38.666 ± 0.516***	1.57 ± 0.20

Values are expressed as mean ± SD, n = 6 compared to control; *** $p \leq 0.001$; ** $p \leq 0.01$; * $p \leq 0.05$; NS – Not significant

DISCUSSION: Seed oils of *C. halicacabum* varieties significantly reduced the formation of gastric ulcer in ethanol induced ulcer rat model. A dose dependent response on the intensity of gastric ulceration was noted. However, statistically more effect was noted in var. *microcarpum.* The increase in potassium content is significant because it alters the hydrogen ion content there by increasing the mucosal protective action. The mucosal defense mechanism may be due to the epithelial cells of the gastric mucosa which are impermeable to hydrogen ions thereby forming a physical barrier [26]. Carbohydrate and protein ratio also supports the above observation. Difference between the two seed oils may be due to the quantitative variation of the biological compounds.

The antiulcer effect is also supported by the decrease in the aggressive factors like pepsin and proteins and an increase in the resistance factors like pH, hexosamine, fucose and sialic acid. Protection against experimental ulcers may be due to the effect of prostaglandin in the parietal cells [27], as prostaglandin enhance the mucosal resistance, perhaps by increasing the secretion of mucous and bicarbonates [28] strengthening the mucosal barrer, decreasing the gastric motility [29], increasing the release of endogenous mediators [30], decreasing the release of endogenous amines [31], and stimulation of cellular growth and repair [32].

The antiulcer agent may protect the mucosa from acid effects by selectively increasing prostoglandin PGF [33]. It may be due to the presence of saponins, terpenoids and amino acids [34].

The increase in the potassium ion in-turn reflects in the increase in hydrogen ion concentration and bicarbonate ion concentration. The increase in bicarbonate ion concentration plays an important role in protecting the gastric and duodenal mucosa against hydrochloric acid [35].

Both the seed oils exhibited antiulcer activity by increasing hexosamine and carbohydrate protein ratio and decreasing pepsin content. This results in the increase in mucous secretion. The importance of mucousal trauma has long been recognized [36] (the more production of mucous, the less was the degree of ulceration).

Mucous also protects the mucosa and sub-mucosa from inflammatory reaction. The higher the mucin contents the lower is the free acidity [37] suggested the significance of mucosal barriers.

The increase in carbohydrate protein ratio is the direct reflection of mucin activity [38]. This suggests the increase in glycoprotein content of the gastric mucosa. The same results have been reported in ethanol extract of *C. halicacabum* whole plant against the ethanol induced ulcer in rats [39] and also similar action have been reported for anti-ulcer drug, carbonoxolone sodium. Antiulcer properties of *C. halicacabum* seed oils have merit to further investigations like mechanism of action and further phytochemical, pharmacological and clinical studies to confirm the present results.

CONCLUSION: The seed oils of both the varieties of *Cardiospermum halicacabum* showed significant antiulcer activity in ethanol induced ulcer in animal model. It has mucoprotective activity by selectively increasing prostoglandin. Further studies are being carried out to characterise and explore the biologically active substances present in seed oils of *C. halicacabum*.

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