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Evaluation of role of *Jasminum sambac* against ulcerative colitis in rats

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Current investigation is designed to find out the role of *Jasminum sambac* (JS) against Acetic Acid (AA)-induced ulcerative colitis in rats. 30 male albino Wistar rats were randomly separated into 5 batches (n=6): normal: admitted with distilled water for 7 days; negative: received the distilled water for 1- 3 days and from 4-7 days received the 2 mL of AA (4% v/v) rectally; test 1, 2 and standard group: orally received the ethanolic extract of leaves of *Jasminum sambac* (EEJS) at doses 250, 500 mg/kg and prednisolone 2 mg/kg from 1-3 days without AA and from 4-7 days received the 2 mL of 4% v/v AA rectally after 2 h administration of EEJS and prednisolone. On 8th day rats were sacrificed and colon tissues were examined. Rats with acetic acid-induced colitis exhibited significant decrease in body weight, colon length, superoxide dismutase, catalase, reduced glutathione, significant rise in colon weight, oedema, disease activity index, macroscopic damage score, occult blood in stool, stool consistency and lipid peroxidation. Pre-treatment with EEJS at doses 250, 500 mg/kg orally significantly regularized the above-mentioned parameters in dose dependent manner. Ethanolic extract of *Jasminum sambac* could protect the AA-produced ulcerative colitis by attenuation of macroscopic damage, microscopic damage, and oxidative biomarkers.

Keywords: Acetic acid, Antioxidants, Disease activity index, *Jasminum sambac*, Ulcerative colitis

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Ulcerative colitis (UC) is a chronic inflammatory bowel disease in addition to ulcers of the colon. The manifestations of UC are abdominal discomfort and incontinent diarrhoea blended with blood. Though it is not lethal, affects the life quality of the patient due to long-standing abdominal discomfort[1]. The exact cause of UC is not absolutely understood. Recently, its pathogenesis has been measured as ecological stimuluses, extreme microorganisms, genetic disparity, and disruption in the native and acquired defense responses[2]. To withstand remission, reduce complications and get better eminence of life are the therapy goals of UC. Anti-tumour necrosis factor alpha (anti-TNF-α) and monoclonal antibodies are the biological therapies for UC. The price, expiration of patent limits their use and therapy with immunosuppressives is unsafe. Hence, here is a demand for efficient, safe drugs to cure the UC. Alternative treatment for various illnesses relies on natural products[3]. *Jasminum sambac* Linn (Oleaceae) is well-known as Jasmine. *J. sambac* is traditionally used to treat cardiovascular problems. However, there is no scientific data available on this plant against ulcerative colitis. Hence, present study was carried out to assess the effectiveness of *J. sambac* ethanolic extract against acetic acid (AA) - induced ulcerative colitis in rats[4].

Materials and Methods

Collection and authentication of *Jasminum Sambac*

Fresh leaves of *J. sambac* (family *oleaceae*) were collected from Ananthapuramu. The authentication of plant was done by Dr. J. Raveendra Reddy, Raghavendra Institute of Pharmaceutical Education and Research. Specimen was deposited with voucher number 87 and dated 25. 01. 2018.

Drugs and chemicals

Prednisolone was obtained from Bafna Pharmaceuticals Ltd, Chennai and all other chemicals were obtained locally.

Extraction

Fresh leaves of *J. sambac* were collected and shade dried for about 7 days. The dried material was made into powder using grinder. About 2600 g of the

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powdered material was taken in extraction vessel and subjected to ethanol under continuous shaking for about 48 h. Then finally the ethanol-soluble constituents were filtered using muslin cloth. Using vacuum evaporator, the filtrate was dried under reduced pressure at 50°C. Finally, the ethanol extract was quantified (10.4 g) and stored in 4°C until use.

Docking studies

The library was submitted to *in silico* molecular docking investigations using Schrodinger Glide (version 12.8) to find the best-suited molecule with the highest docking scores and interactions. To determine anti ulcerative colitis effect, Quercetin, Linalool were docked with the Cyclooxygenase -2 (code: 3MDL) (Fig. 1).

Animals and grouping

We procured male Albino Wistar rats of 150-180 g from Ragahvendra enterprises, Bengaluru, India. Then, we housed the rats in the institutional animal house under standard parameters. Protocol was accepted by institutional animal ethical committee (IAEC/XI/08/RIPER/2018).

After 10 days of acclimatization period of animals, they were segregated into groups of 5 (N=6). The first group served as normal: received distilled water for 7 days. Second group served as disease control: received distilled water for 1-3 days and from 4 -7 days received the 2 mL of 4% v/v AA rectally. The third and fourth groups served as test: received the EEJS at doses 250, 500 mg/kg orally respectively for 1-3 days without acetic acid and from 4 -7 days received the 2 mL of 4% v/v AA rectally after 2 h administration of EEJS at doses 250, 500 mg/kg orally respectively. The fifth group served as standard: received the rednisolone at dose 2 mg/kg orally for 1-3 days without acetic acid and from 4-7 days received the 2 mL of 4% AA rectally after 2 h administration of Prednisolone. Ulcerative colitis was diagnosed by recording of clinical score, colon length and weight, morphological changes in colon and biochemical parameters.

Assessment parameters

Body weight

Body weight was noted before and after respective treatment.

Colon oedema

The anti-ulcerogenic activity was evaluated after completion of the treatment period. The animals were sacrificed and 9 cm of distal colon was excised. It was flushed with saline to remove faecal matter. Later, the colon was dried to reach stable weight. The difference between before and after drying was considered as colon oedema (Fig. 2).

Clinical scoring

Severity of inflammation was measured with scale of 0 to 4 based on characteristics of stool consistency and blood stained (Table 1).

Determination of oxidative parameters in colonic tissue homogenate

Quantification of oxidative stress parameters were estimated in the homogenate of colon tissue using a spectrophotometer as per the standard protocol[5].

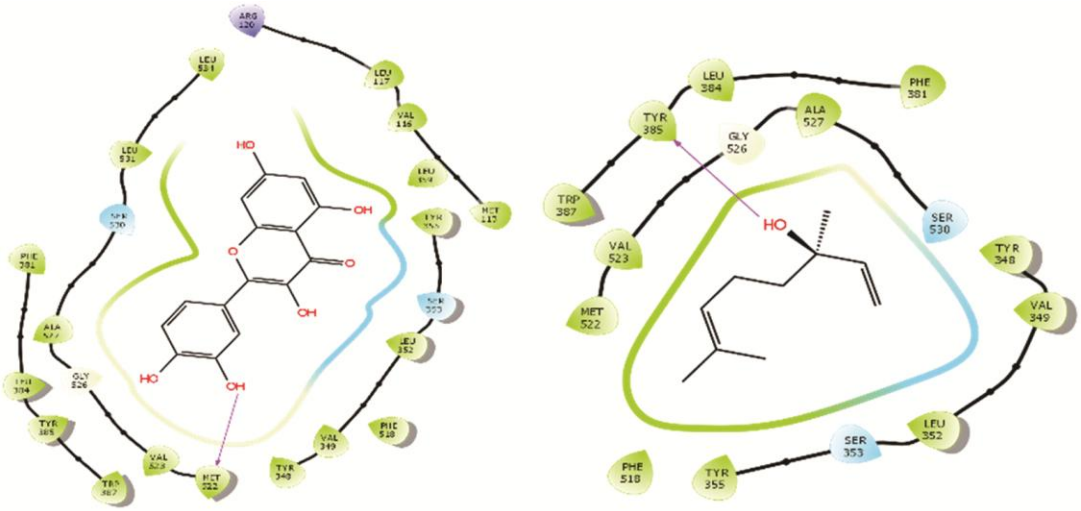

Fig. 1 — Docking studies

Statistical data

Results were stated as mean ±S.E.M. Significant difference in groups were confirmed using one way ANOVA and by using the post hoc test, tukey. $p<0.05$ was considered as the statistical difference of mean.

Results

Outcome of EEJS on body weight

Decrease in body weight of AA received rats from day 4 to 7 was observed related to control rats ($p<0.05$). Pre- treatment with EEJS at 250 mg/kg body weight dose showed considerable decrease in body weight at day 4 ($p<0.01$), day 5 ($p<0.05$) and considerable increase in body weight at day 6 ($p<0.05$), day 7 ($p<0.01$) related to AA- induced colitis rats. Pre-treatment with EEJS at 500 mg/ Kg body weight dose, showed no noteworthy change at day 4, significant rise in body weight at day 5, 6 and 7 ($p<0.05$) related to AA-induced colitis rats. Prednisolone pre-treated rats showed significant rise in body weight at day 4, 5, 6 and 7 correspondingly ($p<0.01$, $p<0.01$, $p<0.05$, $p<0.05$) related to AA induced colitis rats (Table 2).

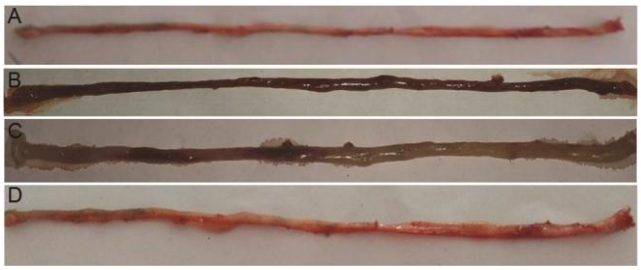

Fig. 2 — Representative colon photos from each group a) Normal group b) Acetic acid induced group c) EEJS at dose 250 mg/kg d) EEJS at dose 500 mg/kg e) Prednisolone group (2 mg/kg)

Table 1 — Disease Activity Index

Score	Body weight score (%reduction)	stool consistency	Occult blood in stool
0	<5%	Solid pellets	No blood in stool
2	5-15%	Semisolid	Blood present in stool
4	>15%	Diarrhea	Bleeding from rectum

To measure the extent of inflammation, the Disease Activity Index (DAI) was calculated based on the formula, DAI = (Body weight score + Stool consistency Score + Occult blood Score)/3

Outcome of EEJS on occult blood in stool

Significant rise in occult blood in stool in AA administered rats at day 4, 5, 6 and 7 respectively ($p<0.001$) was observed in relation to normal rats. There was no significant change in occult blood in stool at day 4, 5 and significant decrease in occult blood in stool at day 6, 7 respectively ($p<0.001$) in pre-treated rats with EEJS at dose of 250 mg/kg body weight in relation to AA-induced colitis rats was observed. There was no significant change at day 4, significant decrease in occult blood in stool at day 5, 6, 7, respectively ($p<0.001$) in pre-treated rats with EEJS at dose of 500 mg/kg body weight compared to AA-induced colitis rats was observed. Prednisolone-pre-treated rats exhibited that no noteworthy change at day 4, 5 and significant decrease in occult blood in stool at day 6, 7, respectively ($p<0.001$) in relation to AA-produced colitis rats (Table 3).

Table 2 — Effect of EEJS on body weight

Group	1 day	2 day	3 day	4 day	5 day	6 day	7 day
Normal	190±4.4	190±4.4	190.4.4	190±4.4	190±4.4	188±5.8	190±4.4
Disease control	192±3.4^{ns}	192±3.7^{ns}	192±3.7^{ns}	182±3.7*	178±2.0*	172±3.7**	160±3.1**
EEJS (250 mg/kg)	196±2.4^{ns}	196±2.4^{ns}	196±2.4^{ns}	172±2.0**	176±2.4*	180.0.0*	184±2.4*
EEJS (500 mg/kg)	200±3.1^{ns}	200±3.1^{ns}	200±3.1^{ns}	182±3.4*	186±2.4*	190±3.6*	196±2.4*
Prednisolone (2 mg/kg)	198±2.4^{ns}	196±2.0^{ns}	196±2.0^{ns}	172±3.7**	178±3.4**	184±2.4*	190±3.1*

Ns - Non significant with contrast to normal, $^{*}p<0.05$, ** $p<0.01$ with contrast to normal, ns- Non significant with contrast to disease control, $^{**}p<0.01$, $^{*}p<0.05$ with contrast to disease control.

Table 3 — Effect of EEJS on occult blood in stool

Group	1 day	2 day	3 day	4 day	5 day	6 day	7 day
Normal	0±00	0±00	0±00	0±00	0±00	0±00	0±00
Disease control	0±00^{ns}	0±00^{ns}	0.00^{ns}	4±0.0***	4±0.0***	4±0.0***	4±0.0***
EEJS (250 mg/kg)	0.00^{ns}	0.00^{ns}	0.00^{ns}	4±0.0^{ns}	3.2±0.4^{ns}	2.4±0.4**	0.8±0.4***
EEJS (500 mg/kg)	0.00^{ns}	0.00^{ns}	0.00^{n}	4.0±0.0^{ns}	2.8±0.4***	2.0±0.0***	0±0.0***
Prednisolone (2 mg/kg)	0.00^{ns}	0.00^{ns}	0.00^{ns}	4.0±0.0^{ns}	3.2±0.49^{ns}	2.4±0.40***	0.8±0.4***

Ns- Non significant with contrast to normal, $^{***}p<0.001$ with contrast to normal, ns- non significant with contrast to disease control, $^{***}p<0.01$, ** $p<0.01$ with contrast disease control.

Table 4 — Effect of EEJS on stool consistency

Group	1 day	2 day	3 day	4 day	5 day	6 day	7 day
Normal	0±0.0	0±0.0	0±0.0	0±0.0	0±0.0	0±0.0	0±0.0
Disease control	0±0.0^{ns}	0±0.0^{ns}	0±0.0^{ns}	3.2±0.4**	4±0.0***	4±0.0***	4±0.0^{c***}
EEJS (250 mg/kg)	0±0.0^{ns}	0±0.0^{ns}	0±0.0^{ns}	3.2±0.4^{ns}	3.6±0.4^{ns}	2±0.0***	2.0±0.0***
EEJS (500 mg/kg)	0±0.0^{ns}	0±0.0^{ns}	0±0.0^{ns}	2.8±0.4^{ns}	2.4±0.4***	2.0±0.0***	0±0.0***
Prednisolone (2 mg/kg)	0±0.0^{ns}	0±0.0^{ns}	0±0.0^{ns}	3.4±0.4^{ns}	3.6±0.4^{ns}	2.0±0.0***	2.0±0.0***

Ns- Non significant with contrast to normal, **p<0.01, *** p<0.001 with contrast to normal, ns- Non significant with contrast to disease control, *** p<0.001 with contrast to disease control.

Table 5 — Effect of EEJS on Disease Activity Index (DAI)

Group	1 day	2 day	3 day	4 day	5 day	6 day	7 day
Normal	0±0.0	0±0.0	0±0.0	0±0.0	0±0.0	0±0.0	0±0.0
Disease control	0±0.0^{ns}	0±0.0^{ns}	0±0.0^{ns}	2.3±0.1*	2.7±0.0*	3.7±0.0**	4.0±0.0**
EEJS (250 mg/kg)	0±0.0^{ns}	0±0.0^{ns}	0±0.0^{ns}	2.6±0.0^{ns}	2.2±0.0^{ns}	1.3±0.0***	0.3±0.0***
EEJS (500 mg/kg)	0±0.0^{ns}	0±0.0^{ns}	0±0.0^{ns}	2.4±0.0^{ns}	2.0±0.0^{ns}	0.8±0.0***	0.2±0.0***
Prednisolone (2 mg/kg)	0±0.0^{ns}	0±0.0^{ns}	0±0.0^{ns}	2.5±0.0^{ns}	2.1±0.0^{ns}	1.5±0.1***	1.7±0.1***

Ns - Non significant with contrast to normal, *p<0.05, **p<0.01, *** p<0.001 with contrast to normal, ns- Non significant with contrast to disease control, *** p<0.001 with contrast to disease control.

Outcome of EEJS on stool consistency

Significant increase in stool consistency at day 4, 5, 6 and 7, respectively (p<0.01) in AA received rats was observed in relation to normal rats. There was no significant change at day 4, 5 and significant decrease in stool consistency at day 6, 7, respectively (p<0.01) in pre-treated rats with EEJS at dose of 250 mg/kg body weight in relation to AA-induced colitis rats. There was no significant change at 4th day and considerable decrease in faecal consistency at day 5, 6 and 7, respectively (p<0.001) in pre-treated rats with EEJS at dose of 500 mg/kg body weight in relation to AA-induced colitis rats. Prednisolone-treated rats showed no significant change at day 4, 5 and significant decrease in faecal consistency at day 6, 7, respectively (p<0.001) in relation to AA-induced colitis rats (Table 4).

Outcome of EEJS on disease activity index (DAI)

Observed that considerable increase in DAI at day 4, 5, 6 and 7, correspondingly (p<0.05, p<0.05, p<0.01, p<0.01) in AA received rats with contrast to normal rats. Pre-treatment with EEJS at doses 250 mg/kg, 500 mg/kg body weight and standard drug prednisolone exhibited no considerable change at day 4, 5 and significant decrease in DAI at day 6, 7, respectively (p<0.001) in relation to AA induced colitis rats (Table 5).

Outcome of EEJS on colon length, weight and oedema

AA administered rats exhibited that considerable reduction in colon length (p<0.001), rise in colon weight (p<0.001) and increase in colon oedema (p<0.001) compared to normal rats. Pre-treatment with EEJS at 250 mg/kg body weight dose exhibited significant increase in colon length, decrease in colon weight and oedema correspondingly (p<0.05, p<0.001) in relation to AA-induced colitis rats. Pre-treatment with EEJS at 500 mg/kg body weight dose and prednisolone significantly increased the colon length, decreased the colon weight and oedema respectively (p<0.01) compared to AA induced colitis rats (Tables 6 & 7).

Table 6 — Effect of EEJS on colon length and weight

Group	Colon length (cm)	Colon weight (mg)
Normal	17.8±0.037	699±2.77
Disease control	11.2±0.37***	916±18***
EEJS (250 mg/kg)	13.2±0.24*	856±11.86^{ns}
EEJS (500 mg/kg)	13.42±0.28^{ns}	845.4±4.04*
Prednisolone (2 mg/kg)	14.5±0.288**	875±11.86**

***p<0.001 with contrast to normal, ns-Non significant with contrast to disease control, *p<0.05, **p<0.01 with contrast to disease control.

Table 7 — Effect of EEJS on colon edema

Group	Colon Edema
Normal	47.48±2.7
Disease control	76.69±4.606***
EEJS (250 mg/kg)	60.64±1.484**
EEJS (500 mg/kg)	65.26±1.272*
Prednisolone (2 mg/kg)	63.09±1.225**

***p<0.001 with contrast to normal; **p<0.01, *p<0.05 with contrast disease control.

Outcome of EEJS on macroscopic damage score

An increase in macroscopic damage score (p<0.001) in AA received rats in relation to normal

Table 8 — Effect of EEJS on macroscopic damage score

Group	MDS
Normal	0±0.0
Disease control	3.6±0.2***
EEJS (250 mg/kg)	2.2±0.3**
EEJS (500 mg/kg)	1.8±0.3***
Prednisolone (2 mg/kg)	2.2±0.3**

***p<0.001 with contrast to normal, **p<0.01, ***p<0.001 with contrast to disease control.

Table 9 — Effect of EEJS on oxidative parameters in colon tissue homogenate

Group	SOD (U/mg protein)	CAT (µmol H_2O_2 decomposed/mg protein)	GSH (nmol GSH/mg protein)	LPO (nmol MDA/mg protein)
Normal	94.5±1.2	30.29±0.881	37.88±1.45	26.78±0.92
Disease control	46±3.60***	21.32±1.276***	28.38±0.06***	45.73±1.12***
EEJS (250 mg/kg)	56.67±1.8*	26.84±1.12*	24.73±0.514*	38.77±1.18*
EEJS (500 mg/kg)	59±2.51**	28.05±0.862*	26.95±0.71ns	38.29±0.71*
Prednisolone (2 mg/kg)	85.35±1.4***	29.72±0.74***	33.26±1.09***	31.06±0.34***

***p<0.001 with contrast to normal, *p<0.05 with contrast to disease control, ns- non significant with contrast to disease control, **p<0.01, ***p<0.001 with contrast to disease control.

group was seen. Pre-treatment with EEJS at doses of 250 mg/kg, 500 mg/kg body weight and prednisolone considerably decreased the macroscopic damage score correspondingly (p<0.01, p<0.001) in relation to AA-induced colitis rats (Table 8).

Outcome of EEJS on anti-oxidants of colonic tissue

AA-induced colitis rats produced considerable reduction in SOD, CAT, GSH (p<0.001) and increase in LPO (p<0.001) in relation to normal rats. Pre-treatment with EEJS at doses 250 mg/kg, 500 mg/kg body weight dose and prednisolone considerably increased the SOD (p<0.05), CAT (p<0.01), GSH (p<0.01) and considerably decreased the LPO (p<0.001) in relation to AA-induced colitis rats (Table 9).

Discussion

UC is one of the major inflammatory bowel syndrome (IBS) distressing mostly the colon and rectum. Rising in prevalence and incidence of IBD show its development as a worldwide disease[6]. Neutrophil infiltration, cytokines and reactive oxygen species (ROS) are the contributors of pathogenesis of UC[7]. The drugs of choice for treatment of UC are 5-aminosalicylic acid and salazosulphapyridine. Corticosteroids, Mercaptopurines, Azathioprine and Cyclosporine are added in severe forms of the UC but these are not deprived of side effects[8]. These drawbacks of current drugs resulted in investigation towards to traditional medicine[9]. Traditionally, many countries relied on folk medicine to control the symptoms of IBD plus UC. Numerous scientific studies have established the effectiveness of several natural compounds to palliate UC[10]. Development of colitis in rats with AA has a resemblance to human colitis[11]. AA-induced colitis triggered the body weight loss, decrease colon length, enhancement in colon weight and oedema in relation to normal group. The current study aimed to find out the effect of *J. sambac* in AA-induced ulcerative colitis in rats. Energy docking scores of Quercetin and Linalool were -9.57, 4.65 kcal mol^{-1}. Pre-treatment with EEJS at doses 250, 500 mg/kg exhibited the increase in body weight, colon length and decrease in colon oedema compared to colitis rats. The clinical manifestations of UC include diarrhoea, blood in stool, abdominal pain[12]. AA-induced colitis rats exhibited noticeable increase in occult blood in stool, stool consistency, macroscopic damage score compared to normal rats. Pre-treatment with EEJS at doses 250, 500 mg/kg normalized the occult blood in stool, stool consistency and macroscopic damage dose dependently. The free radicals may damage cells if their degree of formation surpasses the capacity of endogenous antioxidant defend systems[13]. To scavenge free radicals, gastric cells have enzymatic and non-enzymatic anti-oxidants that are SOD, CAT, and GSH, however excessive generation of free radicals enhances LPO and should decline these anti – oxidants[14]. In this study AA-induced colitis rats revealed considerable depletion of SOD, CAT, GSH and enhancement of lipid peroxidation in relation to normal rats. Pre-treatment with EEJS at doses of 250, 500 mg/kg exhibited

marked enhancement of SOD, CAT, GSH and declining of lipid peroxidation dose dependently compared to AA-induced colitis in rats.

Conclusion

The present study concludes that ethanolic extract of *Jasminum sambac* protects against ulcerative colitis in rats through ameliorating the macroscopic damage as well as oxidative markers.

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Conflict of Interest

The authors declare that they do not have any conflit of interest.

Author's Contributions

KSR: Investigation, methodology, validation Conceptualization; AS: Methodology, resources reviewing, and editing; BP: Supervision, formal analysis, visualization, and writing-review & editing; PLS: Investigation, Docking studies; MSRC: Supervision, project administration, funding acquisition, writing reviewing, and editing.

References

1 Yajuan Ni, Mengyang Liu & Haiyang Yu, Desmethyl bellidifolin from *Gentianella acuta* ameliorate TNBS – induced ulcerative colitis through antispasmodic effect and anti inflammation, *Front Pharmacol*, 10 (1104) (2019) 1-12.

2 Jignesh Patel I & Suresh Sanja D, Protective effect of *Abies pindrow* on dextran sulphate sodium induced ulcerative colitis in rats, *Indian J Nat Prod Resour*, 12 (1) (2021) 43-51.

3 Attia Atta H, Sarmarmounier M & Soad Nasr M, Phytochemical studies and anti-ulcerative colitis effect of *Moringa oleifera* seeds and Egyptian propolis meth and extracts in a rat model, *Asian Pac J Trip Biomed*, 9 (3) (2019) 98-108.

4 Khan I A, Hussain M & Munawar S H, *Jasminum sambac*: A Potential candidate for drug development to cure cardiovascular ailments, *Molecules*, 26 (2021) 5664.

5 Shanmugam S, Thangaraj P & dos Santos Lima B, Protective effects of flavonoid composition rich *P. subpeltata Ortega* on indomethacin induced experimental ulcerative colitis in rat models of inflammatory bowel diseases, *J Ethnopharmacol*, (2019) 1-42.

6 Tahmasebi P, Abtahi froushani M & Afzale Ahangaran N, Thymol has beneficial effects on the experimental model of ulcerative colitis, *Avicenna J Phytomed*, 9 (6) (2019) 538-550.

7 Gehan EI-Akabawy & Neveen EI-sherif M, Zeaxanthin exerts protective effects on acetic acid – induced colitis in rats via modulation of pro-inflammatory cytokines and oxidative stress, *Biomed Pharmacother*, 111 (2019) 841-851.

8 Heba Abdallah M I, Naglaa Ammar M & Mohamed F, Protective mechanism of *Acacia saligna* butanol extract and its nano-formulations against ulcerative colitis in Rats as revealed via biochemical and metabolomic Assays, *Biology*, 9 (195) (2020) 1-21.

9 Babitha S, Bindu K, Taj Nageena & Verapur V P, Fresh fruit juice of *Opuntia dillnii* Haw attenuates acetic acid – induced ulcerative colitis in rats, *J Diet Suppl*, (2018) 1-13.

10 Sachan N, Chandra P & Pal D, Effect of Delonix (Boj. Ex Hook.) Raf. stem bark extract against experimentally induced ulcers in rats, *Indian J Exp Biol*, 55 (2017) 49-54.

11 Zaware B, Gilhotra R & Chaudhari S R, Potential of *Mimosa pudica* leaves in the treatment of ulcerative colitis in rat, *Bangladesh J Pharmacol*, 13 (2018) 241-47.

12 Nariyaa M, Nariya P, Ravishankar B & Goswami S, Ameliorative effects of Triphala on mucosal damage in rat model of ulcerative colitis, *Indian J Tradit Know*, 20 (4) (2021) 951-955.

13 Rafeeq Mhttps://pubmed.ncbi.nlm.nih.gov/33441125/ - full-view-affiliation-1, Murad H A S, Abdallah H M, El-Halawany A M, Protective effect of 6-paradol in acetic acid induced ulcerative colitis in rats, *BMC Complement Med Ther*, (2021) 1-10.

14 Badr G, Elsawy H, Malki M A, Alfwuaires M, El-Gerbed M S A, *et al*., Protective effects of myristicin against ulcerative colitis induced by acetic acid in male mice, *Food Agric Immunol*, 31 (1) (2020) 435-46.