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Assessment of Crocus Sativus Antiinflammatory Properties in Male Sprague Dawley Rats

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Abstract
The present study was conducted to evaluate the anti-inflammatory activity of *Crocus sativus* extract in various experimental models of inflammation in rats. The extract was administered orally at doses of 400 mg/kg and 600 mg/kg, and its effects were compared with a standard anti-inflammatory drug, diclofenac (10 mg/kg). Inflammatory responses were induced using models such as carrageenan, histamine, dextran, serotonin, formaldehyde, and cotton pellet granuloma.
Biochemical parameters including total protein, albumin, acid phosphatase (ACP), and alkaline phosphatase (ALP) were also assessed to understand the systemic effects of the extract. The results showed that the 600 mg/kg dose significantly increased total protein levels without altering albumin concentrations, while ACP levels remained stable, suggesting no harmful cellular stress. A significant reduction in ALP levels was observed in the 400 mg/kg group, indicating potential liver-protective effects.
Overall, the *Crocus sativus* extract demonstrated considerable anti-inflammatory activity with minimal adverse effects, in contrast to diclofenac, which showed elevated liver enzyme levels. These findings suggest that the extract, particularly at 600 mg/kg, may serve as a safer and effective alternative for managing inflammation.

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Keywords: Crocus Sativus, Anti-Inflammatory, Adverse Effects

1. Introduction

Inflammation usually occurs when infectious microorganisms such as bacteria, viruses or fungi invade the body, reside in particular tissues and/or circulate in the blood [1, 2, 3]. Inflammation may also happen in response to processes such as tissue injury, cell death, cancer, ischemia and degeneration [1, 4, 5, 6, 7, 8, 9]. Mostly, both the innate immune response as well as the adaptive immune response are involved in the formation of inflammation [1, 5, 9]. The innate immune system is the foremost defense mechanism against invading microorganisms and cancer cells, involving the activity of various cells including macrophages, mast cells and dendritic cells. The adaptive immune system involves the activity of more specialized cells such as B and T cells who are responsible for eradicating invading pathogens and cancer cells by producing specific receptors and antibodies.
Numerous inflammatory mediators are synthetized and secreted during inflammatory responses of different types. Inflammatory substances are usually divided to two main categories: pro- and anti-inflammatory mediators. Nevertheless, some mediators such as interleukin (IL)-12 possess both pro- and anti-inflammatory properties [10]. Among the inflammatory mediators and cellular pathways that have been extensively studied in association with human pathological conditions are cytokines (e.g., interferons, interleukins and tumor necrosis factor α), chemokines (e.g., monocyte chemoattractant protein 1), eicosanoids (e.g., prostaglandins and leukotrienes) and the potent inflammation-modulating transcription factor nuclear factor κ B.
Tumor necrosis factor (TNF)-α is an important pro-inflammatory cytokine that is secreted from various cells and exerts many cellular effects [11,12]. TNF-α has been associated with multiple illness states in humans, including immune and inflammatory diseases, cancer, psychiatric disorders, among others.

Another cytokine which mostly exerts a pro-inflammatory activity is IL-1α [13, 14]. It stimulates the secretion of pro-inflammatory cytokines such as IL-1β and TNF-α [13, 14]. However, IL-1α has also been associated with anti-inflammatory activity. Similar to IL-1α, IL-6 usually acts as a pro-inflammatory cytokine but it also has some anti-inflammatory effects. As mentioned above, the IL-12 family of cytokines (including IL-12, IL-23, IL-27 and IL-35) possess both pro- and anti-inflammatory functions [10,15,16]. On the other hand, IL-10 is a potent anti-inflammatory cytokine the activity of which impedes the action of many pro-inflammatory mediators [17, 18, and 19]. By weakening and controlling the inflammatory response IL-10 helps to maintain tissue homeostasis and attenuates the damage that may result from an exaggerated inflammatory response [17, 18, 19].

Prostaglandin (PG) E2 is probably the most studied PG in association with human physiological and pathological conditions [20]. It has various physiological roles including regulation of normal body temperature, gastric mucosal integrity, renal blood flow and the function of female reproductive system. On the other hand, alterations in PGE2 activity are associated with pathological conditions such as inflammatory diseases, abnormal changes in body temperature, colorectal cancer, among others. The pathway of PGs synthesis starts with generation of arachidonic acid from cell membrane phospholipids by phospholipase A2 (PLA2). Then, arachidonic acid is converted to PGs by the enzyme cycloogygenase (COX) [20]. Among the three known COX isoforms (COX-1, COX-2 and COX-3), the inducible enzyme COX-2 is recognized as the most active during inflammatory processes. Leukotrienes (LTs) such as LTB4 were also linked to human illness states including inflammation, asthma and depression [21,22,23]. LTs are produced by the enzyme 5-lipooxygenase (5-LOX) [22]. Another enzyme that is highly associated with inflammatory conditions is nitric oxide synthase (NOS) which produces nitric oxide (NO) [24]. Similar to COX-2, inducible NOS (iNOS) is the most pro-inflammatory NOS isoform.

The transcription factor nuclear factor κ B (NFκB) is a prominent regulator of immune and inflammatory responses and is highly involved in the pathophysiology of cancer [25,26,27]. In mammals, the NFκB machinery comprises several members (e.g., p50 and p65) which regulate both physiological and pathological processes [25,26]. At resting (un-stimulated) conditions NFκB resides in the cytoplasm [26]. Following activation by various infectious/inflammatory/mitogenic stimuli, NFκB proteins translocate to the nucleus and induce transcription of inflammatory-associated genes [26, 27].

The practice of using plants, their parts or extracts as anti-inflammatory compounds is known since antiquity. For example, concentrated, viscous aqueous extract of ripe carob (Ceratonia siliqua L.) has been used for decades in Arab folk medicine, especially for treating mouth inflammations [28]. The use of plants or plant products for medicinal purposes was mostly documented in books and, lately, in an enormous number of websites (where the reliability of some of these websites must be examined carefully). In the last decades, hundreds of research and review articles were published regarding the anti-inflammatory activities of plants. In this review we introduce some highlights of the literature published mainly during the last three decades, with a few references to earlier reports.

Material and Methods

Collection and Identification of Plant Material

The fresh Crocus sativus are collected and identified was purchased from an herbal Market of Hyderabad, Telangana, India, and authenticated by Dr. K. Madhava Chetty, Assistant Professor, Department of Botany, S.V University, and Tirupati.

Extraction procedure

Coarsely powdered Crocus sativus were used for extraction with methanol by using Soxhlet method for 6-8 hours. 50g of dried powder was weighed and 250mL of solvent methanol is used for the extraction. The extracts were evaporated by using a rotary evaporator and dried at room temperature. The obtained crude extracts were weighed and stored at 4°C for further analysis [29, 30].

Pharmacological Study

Animals

Wistar albino rats of both sexes (180-220 g) were used for the study. The animals were obtained from Jeeva life sciences. All the rats were kept in standard plastic rat cages with stainless steel coverlids and wheat straw was used as bedding material. The animals were kept at the animal house of Department of Biosciences. The animals were facilitated with standard environmental condition of photoperiod (12:12 h dark: light cycle) and temperature (25±2°C). They were provided with commercial rat feed and water given ad libitum. The use of these animals and the study protocols were approved by CCSEA recognized by ethical committee.

Animal grouping for anti-inflammatory studies

Four groups of animals (six animals in each group) will selected for anti-inflammatory studies for all animal model. Total of 96 numbers of animals required.

Group I: Vehicle treated control (distilled water)

Group II: Methanol extract of *Crocus sativus* - 400 mg/kg body weight (CSM-400)

Group III: Methanol extract of *Crocus sativus* - 600 mg/kg body weight (CSM-600)

Group IV: Diclofenac sodium- 10 mg/kg body weight (diclofenac- 10)

The anti-inflammatory activity of selected plant extract will plan to carried out by following animal model

Carrageenan induced rat paw edema by the method of Winter *et al*. (1962) [31].

Carrageenan induced rat paw edema was done by the method of Winter *et al*. (1962) [32]. Inflammation was induced by injection of 0.1 ml of freshly prepared carrageenan (1%) aqueous suspension in normal saline underneath the plantar tissue of the right hind paw of rats. The different groups of rats were administered with CSM (400 and 600 mg/kg, p.o.) and diclofenac (10 mg/kg, p.o.). The control group received vehicle (distilled water, 10 ml/kg, p.o.). 1 h after drug treatment, paw edema was induced by the injection of carrageenan (an edematogenic agent). The paw volume was measured by a Plethysmometer. The measures were determined at 0 h (V_0: before edematogenic agent injection) and 1,2,3,4 and 5h intervals later (V_t). The difference between V_t and V_0 was taken as the edema value. The percentage of inhibition was calculated according to the following formula:

$$\%\ of\ inhibition = \frac{(V_t - V_0)_{control} - (V_t - V_0)_{treated}}{(V_t - V_0)_{control}}$$

Histamine induced rat paw edema

Inflammation was induced by injection of 0.1 ml of freshly prepared histamine (1%) aqueous suspension in normal saline underneath the plantar tissue of the right hind paw of rats. The drug treatment and paw volume were measured in a similar manner to that of carrageenan induced paw edema model.

Dextran induced rat paw edema

Inflammation was induced by injection of 0.1 ml of freshly prepared dextran (1%) aqueous suspension in normal saline underneath the plantar tissue of the right hind paw of rats [33]. The drug treatment and paw volume were measured in a similar manner to that of carrageenan induced paw edema model.

Serotonin induced rat paw edema

Inflammation was induced by injection of 0.1 ml of freshly prepared Serotonin (1%) aqueous suspension in normal saline underneath the plantar tissue of the right hind paw of rats [34, 35]. The drug treatment and paw volume was measured in a similar manner to that of carrageenan induced paw edema model.

Cotton pellet induced granuloma in rats

The effect of methanol extract of *Crocus sativus* stigmas on the chronic phases of inflammation was assessed in the cotton pellet induced granuloma rat model, as described by Swingle and Shideman (1972) [36]. Autoclaved cotton pellets weighing 100 mg each were implanted subcutaneously. One on each side of the abdomen of the animal, through a small ventral incision of rats anesthetized with ether. The different groups of rats were administered with CSM (400 and 600 mg/kg, p.o.) and diclofenac (10 mg/kg. p.o.) once daily for 7 consecutive days from the day of cotton pellet insertion. The control group received vehicle (distilled water, 10 ml/kg, p.o.). On the eighth day the animals were sacrificed and the cotton pellets were removed, dried at 60°C for 24 h and their mass was determined. The results are expressed as mg granulation tissue formed per 100 g body weight.

Biochemical Analysis

On the eighth day, the animals were sacrificed under mild ether anesthesia and blood was collected in clean centrifuge tubes. The serum was obtained by centrifugation and used for the estimation of various biochemical parameters. The absorbance of all the biochemical parameters was measured in a UV-VIS Spectrophotometer -1601 (Shimadzu, Tokyo, Japan).

Estimation of total protein content

The serum total protein was estimated by modified Biuret method[37] (Yatzidis, 1977) using the total protein test kit (Span Diagnostics Ltd.).

Procedure

3.0 ml of Reagent I was added to all the test tubes. Thereafter, 0.03 ml serum was added for the test and 0.03 ml Reagent II was added for the standard, while in blank 0.03 ml of purified water was added. They were then mixed well and incubated at 37ºC for 5 minutes. The absorbance was read at 578 nm.

Estimation of albumin content

The serum albumin was estimated by the method given by Corcoran and Durnan (1977) [37] using albumin test kit (Span Diagnostics Ltd.).

Procedure

3.0 ml of albumin reagent (Reagent I) was added to all the test tubes. Thereafter, 0.03 ml serum was added for the test and 0.03 ml Reagent II was added for the standard, while in blank 0.03 ml of purified water was added. They were then mixed well and incubated at room temperature for 1 min. The absorbance was read at 630 nm.

Estimation of acid phosphatase (ACP) activity

The serum acid phosphatase activity was estimated by the method of King and Jagatheesan (1959) [39] using ACP test kit (Span Diagnostics Ltd.).

Procedure

All the test tubes were marked properly as blank (B), standard (S), control (C) and test (T). 0.5 ml of solution I was added in control (C) and test (T). 0.5 ml of purified water was added in control (C) and test (T). 0.6 ml of purified water was added in standard (S) and 1.1 ml of purified water was added in blank (B). All the tubes were mixed well and incubated at 37ºC for 3 min. 0.1 ml of serum was added in test (T), 0.5 ml of working standard was added in standard (S). All the tubes were mixed well and incubated at 37ºC for 60 min. 0.5 ml of reagent II was added in all the tubes. 0.1 ml of serum was added in control (C). 0.5 ml of reagent III, 0.5 ml of solution II and 0.5 ml of solution III was added in all the tubes. All the tubes were mixed well and absorbance was read at 510 nm. Serum acid phosphatase activity is expressed as KA units.

Estimation of alkaline phosphatase (ALP) activity

Alkaline phosphatase activity was estimated by the method of Kind and King (1954) using ALP test kit (Span Diagnostics Ltd.).

Procedure

All the test tubes were marked properly as blank (B), standard (S), control (C), and test (T). 0.5 ml of working buffered substrate was added in clean tubes. 1.5 ml of purified water was added in all the tubes. They were mixed well and incubated at 37°C for 3 min. 0.05 ml of serum was added in test (T), 0.05 ml of reagent III (Phenol standard) was added in standard (S) and 0.05 ml of purified water was added in blank (B) tubes. All the tubes were mixed well and incubated at 37°C for 15 min. 1 ml of reagent II was added in all the tubes. 0.05 ml of serum was added in control (C). All the tubes were mixed well and absorbance was read at 510 nm. Serum alkaline phosphatase activity is expressed as KA units.

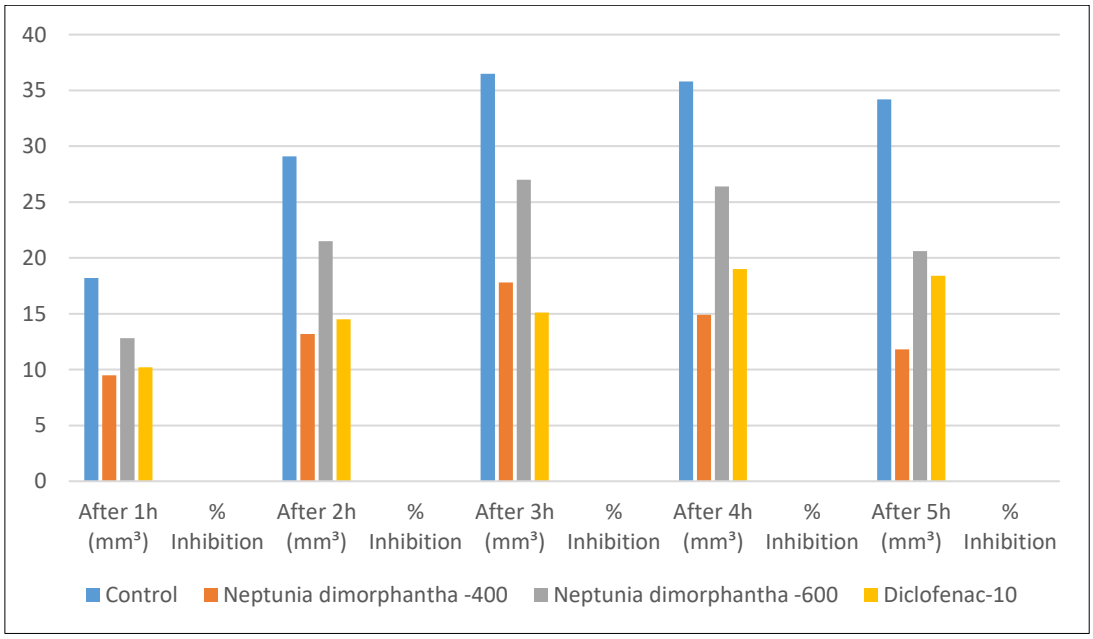
Statistical Analysis

The data obtained from animal experiments are expressed as mean±SEM (standard error of mean). For statistical analysis data were subjected to analysis of variance (ANOVA) followed by Student's t-test. Values are considered statistically significant at $F < 0.05$ for ANOVA and $P < 0.05$ for t-test.

Results

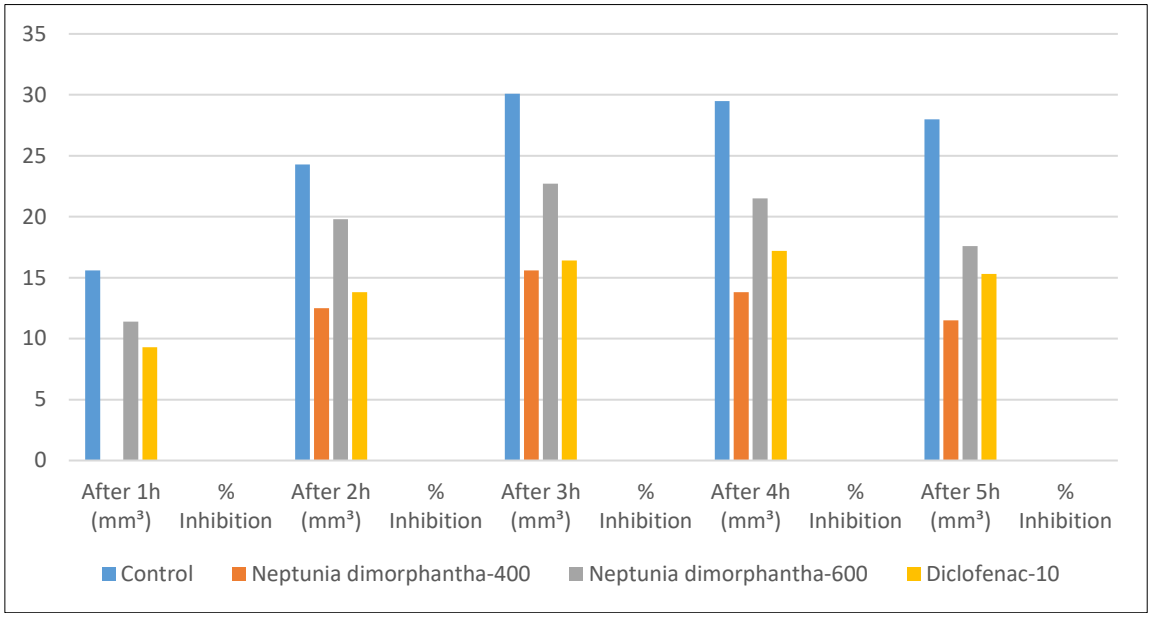
Sl. No.	Phytoconstituents	Test result
1	Alkaloid	-ve
2	Glycosides	-ve
3	Carbohydrate	-ve
4	Protein	-ve
5	Amino acid	-ve
6	Steroids	-ve
7	Flavonoids	+ve
8	Terpenoids	+ve
9	Phenols	+ve
10	Saponins	-ve
11	Tannin	+ve

Group	After 1h (mm^3)	% Inhibition	After 2h (mm^3)	% Inhibition	After 3h (mm^3)	% Inhibition	After 4h (mm^3)	% Inhibition	After 5h (mm^3)	% Inhibition
Control	18.20±2.5	–	29.10±3.1	–	36.50±4.2	–	35.80±4.7	–	34.20±3.9	–
Crocus sativus-400	9.50±1.4 *	47.80 ↓	13.20±2.3 **	54.68 ↓	17.80±2.5 **	51.23 ↓	14.90±2.1 **	58.40 ↓	11.80±2.4 **	65.45 ↓
Crocus sativus-600	12.80±2.0	29.67 ↓	21.50±3.4	26.12 ↓	27.00±3.8	26.03 ↓	26.40±4.0	26.26 ↓	20.60±3.7	39.77 ↓
Diclofenac-10	10.20±1.8	43.96 ↓	14.50±2.6 *	50.17 ↓	15.10±2.7 **	58.63 ↓	19.00±3.2 *	46.93 ↓	18.40±3.1 *	46.96 ↓



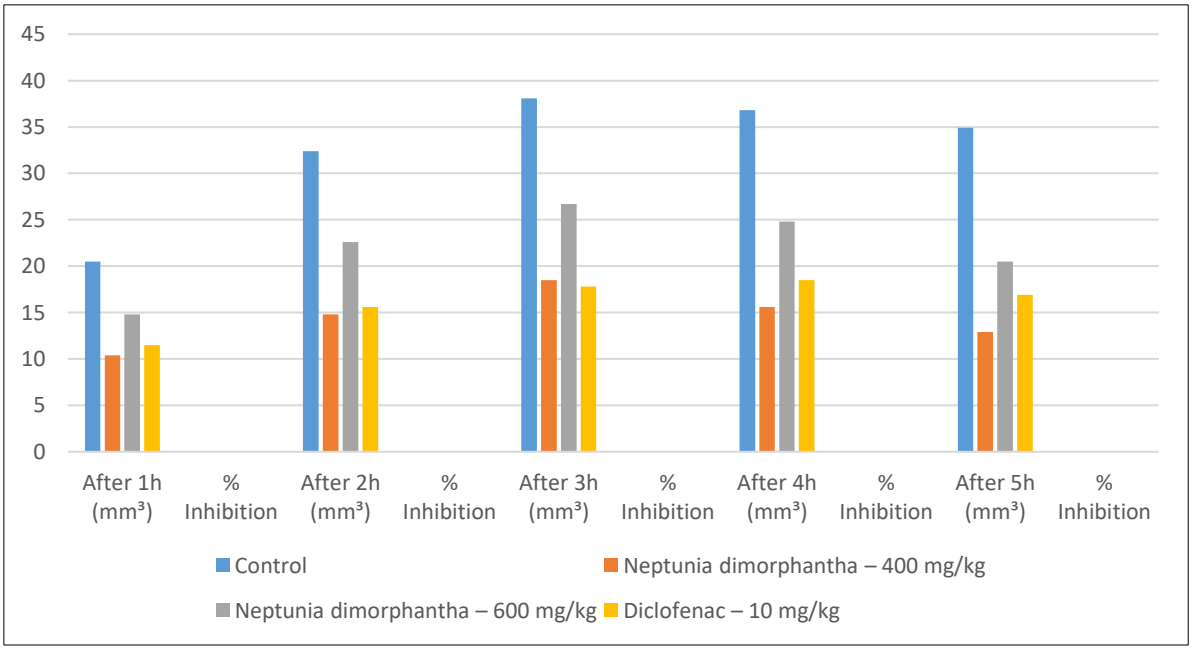
Histamine-Induced Paw Edema

Group	After 1h (mm^3)	% Inhibition	After 2h (mm^3)	% Inhibition	After 3h (mm^3)	% Inhibition	After 4h (mm^3)	% Inhibition	After 5h (mm^3)	% Inhibition
Control	15.60±1.8	–	24.30±2.2	–	30.10±3.1	–	29.50±2.9	–	28.00±3.2	–
Crocus sativus - 400	8.20±1.2 **	47.44 ↓	12.50±2.0 **	48.55 ↓	15.60±2.3 **	48.17 ↓	13.80±1.8 **	53.22 ↓	11.50±2.1 **	58.93 ↓
Crocus sativus - 600	11.40±1.5	26.92 ↓	19.80±2.5	18.52 ↓	22.70±3.0	24.58 ↓	21.50±2.6	27.12 ↓	17.60±2.8	37.14 ↓
Diclofenac-10	9.30±1.3 *	40.38 ↓	13.80±2.1 **	43.21 ↓	16.40±2.2 **	45.51 ↓	17.20±2.3 *	41.69 ↓	15.30±2.5 **	45.36 ↓



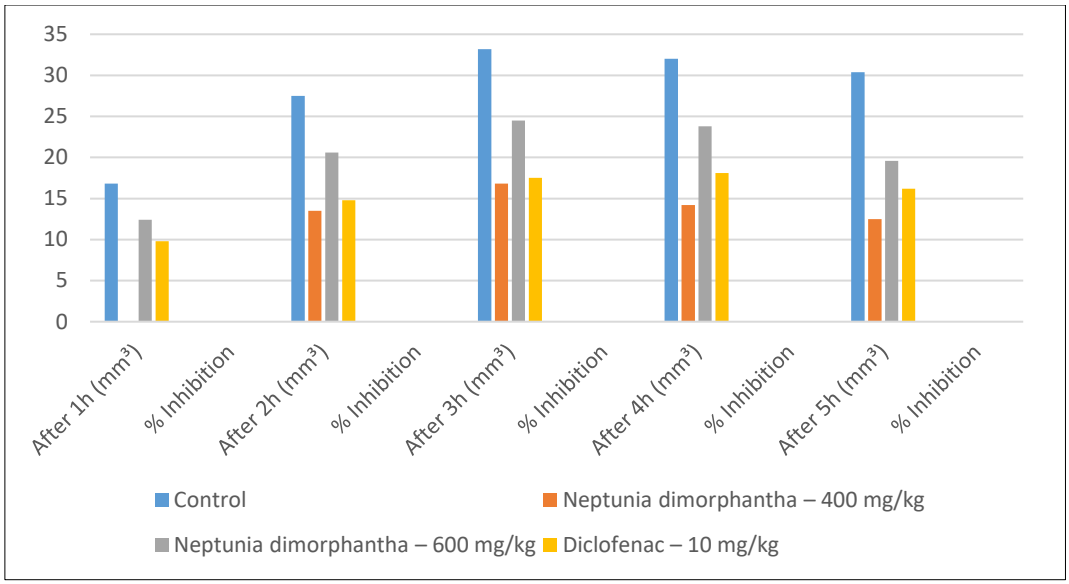
Dextran-Induced Paw Edema

Group	After 1h (mm³)	% Inhibition	After 2h (mm³)	% Inhibition	After 3h (mm³)	% Inhibition	After 4h (mm³)	% Inhibition	After 5h (mm³)	% Inhibition
Control	20.50±2.3	–	32.40±3.1	–	38.10±3.7	–	36.80±4.0	–	34.90±3.5	–
Crocus sativus–400 mg/kg	10.40±1.5 **	49.27 ↓	14.80±2.3 **	54.32 ↓	18.50±2.8 **	51.35 ↓	15.60±2.2 **	57.61 ↓	12.90±2.0 **	63.04 ↓
Crocus sativus–600 mg/kg	14.80±2.0	27.80 ↓	22.60±3.1	30.25 ↓	26.70±3.5	29.92 ↓	24.80±3.3	32.61 ↓	20.50±2.7	41.26 ↓
Diclofenac – 10 mg/kg	11.50±1.6 *	43.90 ↓	15.60±2.4 **	51.85 ↓	17.80±2.6 **	53.26 ↓	18.50±2.8 **	49.68 ↓	16.90±2.5 **	51.58 ↓



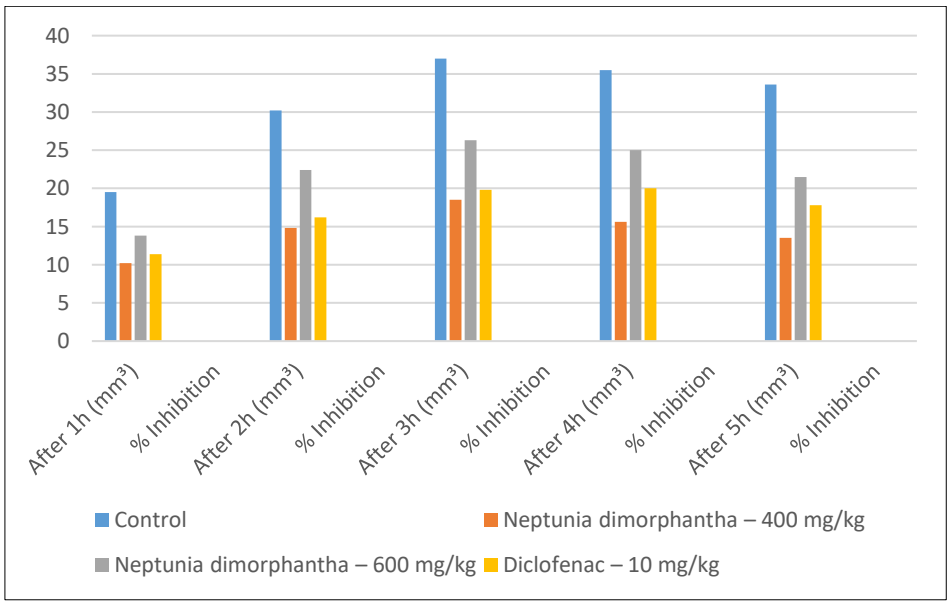
Serotonin induced rat paw edema

Group	After 1h (mm^3)	% Inhibition	After 2h (mm^3)	% Inhibition	After 3h (mm^3)	% Inhibition	After 4h (mm^3)	% Inhibition	After 5h (mm^3)	% Inhibition
Control	16.80±2.1	–	27.50±3.2	–	33.20±3.5	–	32.00±3.8	–	30.40±3.6	–
Crocus sativus–400 mg/kg	8.60±1.3 **	48.81 ↓	13.50±2.1 **	50.91 ↓	16.80±2.4 **	49.36 ↓	14.20±2.2 **	55.63 ↓	12.50±2.0 **	58.88 ↓
Crocus sativus–600 mg/kg	12.40±1.8	26.19 ↓	20.60±3.0	25.09 ↓	24.50±3.3	26.20 ↓	23.80±3.2	25.63 ↓	19.60±2.8	35.53 ↓
Diclofenac – 10 mg/kg	9.80±1.4 *	41.67 ↓	14.80±2.2 **	46.18 ↓	17.50±2.5 **	47.34 ↓	18.10±2.7 *	43.44 ↓	16.20±2.3 **	46.71 ↓



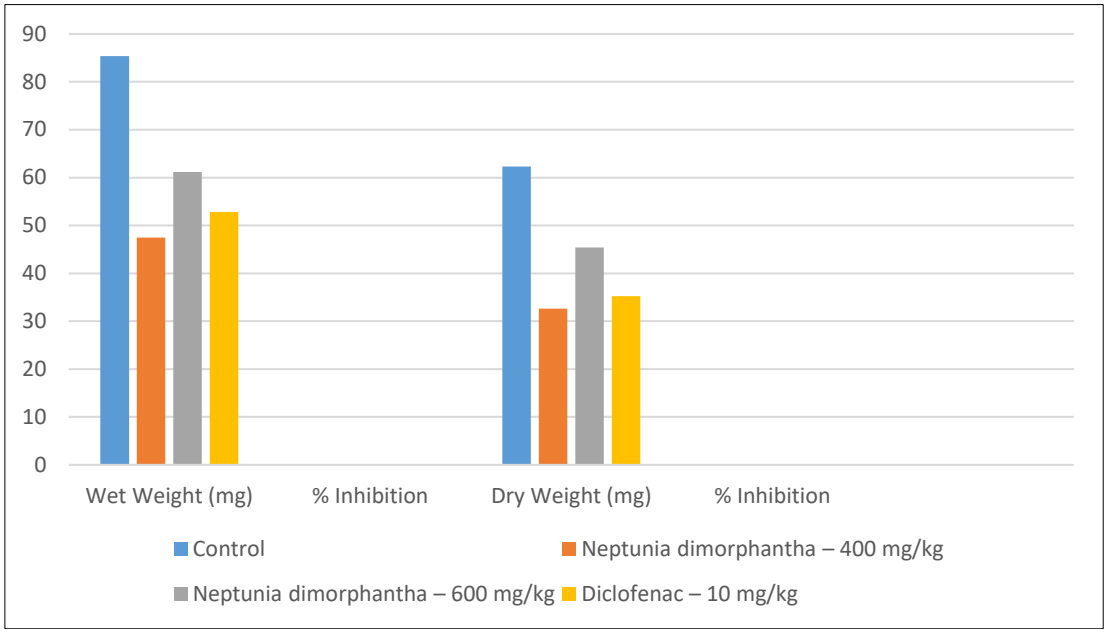
Formaldehyde induced rat paw edema

Group	After 1h (mm^3)	% Inhibition	After 2h (mm^3)	% Inhibition	After 3h (mm^3)	% Inhibition	After 4h (mm^3)	% Inhibition	After 5h (mm^3)	% Inhibition
Control	19.50±2.5	–	30.20±3.4	–	37.00±3.8	–	35.50±4.1	–	33.60±3.7	–
Crocus sativus–400 mg/kg	10.20±1.6 **	47.69 ↓	14.80±2.5 **	51.00 ↓	18.50±2.9 **	50.00 ↓	15.60±2.4 **	56.06 ↓	13.50±2.1 **	59.82 ↓
Crocus sativus–600 mg/kg	13.80±2.2	29.23 ↓	22.40±3.2	25.83 ↓	26.30±3.5	28.92 ↓	25.00±3.3	29.58 ↓	21.50±3.0	36.01 ↓
Diclofenac – 10 mg/kg	11.40±1.8 *	41.54 ↓	16.20±2.6 **	46.36 ↓	19.80±2.8 **	46.49 ↓	20.00±3.0 **	43.66 ↓	17.80±2.5 **	47.02 ↓



Cotton Pellet-Induced Granuloma in Rats

Group	Wet Weight (mg)	% Inhibition	Dry Weight (mg)	% Inhibition
Control	85.40±5.3	–	62.30±4.1	–
Crocus sativus– 400 mg/kg	47.50±3.8 **	44.39 ↓	32.60±2.5 **	47.68 ↓
Crocus sativus– 600 mg/kg	61.20±4.6	28.30 ↓	45.40±3.7	27.11 ↓
Diclofenac – 10 mg/kg	52.80±3.9 **	38.15 ↓	35.20±2.8 **	43.47 ↓



Effect of extract Crocus sativusof on serum biochemical parameters

Treatment	Total Protein (g/dL)	Albumin (g/dL)	ACP (KA/unit)	ALP (KA/unit)
Control (distilled water)	5.21±0.06	3.46±0.04	12.04±1.0	51.09±7.12
Crocus sativus– 400 mg/kg	5.16±0.07	3.27±0.03	12.25±1.2	35.95±6.26
Crocus sativus– 600 mg/kg	5.52±0.06 *	3.45±0.08	12.11±0.61	42.67±5.04
Diclofenac – 10 mg/kg	5.37±0.13	3.21±0.36	17.42±5.4	59.24±13.08

Discussion

The biochemical analysis in this study revealed that *Crocus sativus* extract exerts a protective effect on protein metabolism and liver function in rats with induced inflammation. The total protein level was significantly increased in the group treated with 600 mg/kg of the extract, suggesting that it may enhance protein synthesis or reduce protein loss during inflammatory stress. At

The same time, albumin levels remained stable across all groups, indicating that the extract did not adversely affect albumin production or renal function.

The acid phosphatase (ACP) levels did not show significant differences among the groups. This suggests that the extract does not cause cellular damage or excessive immune activation, which are often linked to inflammation. The absence of any major changes in ACP supports the safety profile of the extract.

A significant observation was the reduction of alkaline phosphatase (ALP) in the 400 mg/kg group, indicating that the extract may help protect the liver or stabilize metabolic processes during inflammation. In contrast, the diclofenac-treated group exhibited elevated ALP levels, suggesting possible liver stress or altered bone metabolism, which are known side effects of prolonged use of non-steroidal anti-inflammatory drugs.

In conclusion, the results suggest that *Crocus sativus* extract, particularly at 600 mg/kg, can support protein metabolism and reduce inflammation without causing adverse effects on liver enzymes. These findings highlight its potential as a safer alternative to conventional anti-inflammatory drugs like diclofenac. Further studies are recommended to explore its mechanisms of action and confirm its therapeutic benefits.

Conclusion

The present study demonstrates that *Crocus sativus* extract possesses significant anti-inflammatory activity, as evidenced by improvements in various biochemical parameters in rats. The extract, especially at a dose of 600 mg/kg, was effective in enhancing total protein levels without disturbing albumin concentrations, indicating its supportive role in protein metabolism during inflammation.

The stability of acid phosphatase (ACP) levels across all groups further confirms that the extract does not induce cellular stress or immune overactivation, suggesting its safety even at higher doses. Additionally, the reduction in alkaline phosphatase (ALP) levels, particularly in the 400 mg/kg group, indicates a protective effect on liver function and metabolic balance during inflammation.

Compared to the standard drug diclofenac, which showed

signs of liver stress, *Crocus sativus*extract demonstrated a safer profile while still exerting considerable anti-inflammatory effects. These findings support its potential as a natural alternative to conventional anti-inflammatory therapies.

In summary, *Crocus sativus* extract shows promise as an effective and safer anti-inflammatory agent. Future studies should focus on its long-term safety, underlying mechanisms, and clinical applicability to further validate its therapeutic potential.

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