ANTIOXIDANT AND POTENTIAL ANTI-ARTHRITIC EFFECTS OF *Senna tora* (L.) Roxb. FRUIT EXTRACT IN CFA-INDUCED ARTHRITIC MICE

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Abstract
Objectives: To evaluate an ethanol extract of *Senna tora* (L.) Roxb. fruit (STEE) in CFA-induced arthritic mice and characterize its phytochemical and antioxidant properties. Materials and Methods: Female Swiss albino mice were assigned to normal, CFA, CFA+meloxicam, and CFA+STEE (100, 200, or 300 mg/kg/day) groups. Arthritis was induced on Day 0; treatments were administered orally on Days 8–28. The hind-paw response was normalized to each group's Day 0 mean and analyzed descriptively because individual repeated-measures records were unavailable. Joint ROS signal and catalase (CAT), superoxide dismutase (SOD), and glutathione peroxidase (GPx) activities were measured. Phytochemicals and in vitro DPPH, ABTS, and FRAP responses were assessed. Results: STEE produced concentration-related antioxidant responses in vitro. At 300 mg/kg/day, joint ROS was 1.72 ± 0.03 AU/mg protein versus 6.33 ± 0.04 in CFA controls, with higher CAT, SOD, and GPx activities ($p < 0.05$). Normalized hind-paw trajectories declined after treatment with meloxicam or STEE. Conclusion: STEE attenuated CFA-associated joint oxidative stress and showed a descriptive reduction in the hind-paw response. Anti-arthritic efficacy requires confirmation using verified individual longitudinal data, joint histopathology, inflammatory biomarkers, and longer-term safety assessment.
Keywords: *Senna tora*, rheumatoid arthritis, antioxidant activity, oxidative stress, anti-inflammatory effect, CFA-induced arthritis model

1. INTRODUCTION

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disorder characterized by persistent synovial inflammation, progressive cartilage degradation, and joint deformity, often leading to severe disability and impaired quality of life [1,2]. Despite the availability of various synthetic disease-modifying antirheumatic drugs (DMARDs) and biological agents, current therapeutic strategies are frequently associated with adverse effects, high cost, and suboptimal long-term efficacy [1]. These limitations have spurred growing interest in the development of alternative or complementary approaches, particularly plant-based therapies that offer immunomodulatory, anti-inflammatory, and antioxidant properties with lower toxicity profiles [2].

Senna tora (L.) Roxb., a leguminous plant widely distributed across tropical and subtropical regions, has traditionally been used in Asian medicine to treat skin disorders, constipation, and inflammation [3]. Phytochemical investigations have shown that *S. tora* fruits contain bioactive compounds such as anthraquinones, flavonoids, glycosides, and phenolic acids, many of which possess anti-inflammatory and antioxidant activities [4]. Additional screening studies have documented diverse constituents in S. tora extracts, although direct in vivo evidence in rheumatoid arthritis remains limited [5].

CFA-induced rodent models are commonly used to examine joint responses and oxidative-stress outcomes [6–9]. This study therefore evaluated orally administered *S. tora* fruit extract in a CFA-induced murine model, focusing on a normalized hind-paw response and joint oxidative stress while also characterizing the extract's phytochemical composition and antioxidant capacity.

2. MATERIALS AND METHODS

2.1. Collection, extraction, and phytochemical characterization of *Senna tora* fruit

Fruits of *Senna tora* (L.) Roxb. were collected in February 2025 from Di An District, Binh Duong Province, Vietnam. A voucher specimen (code ST130225VST) was authenticated and deposited at the Laboratory of Biotechnology, Institute of Biotechnology and Food Technology, Industrial University of Ho Chi Minh City, for future reference. The fruits were thoroughly washed with distilled water, shade-dried at ambient temperature, and ground into a fine powder using a mechanical grinder.

A total of 100 g of the powdered material was macerated in 1000 mL of 70% ethanol (v/v) at a solid-to-

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solvent ratio of 1:10 (w/v) for 72 hours at room temperature with intermittent shaking. The extract was filtered through Whatman No. 1 filter paper, and the solvent was removed under reduced pressure using a rotary evaporator. The resulting crude ethanolic extract of *Senna tora* (STEE) was stored at 4 °C in airtight containers until further analysis. The final dry mass of the crude extract was not retained in the archived laboratory record; therefore, the extraction yield could not be calculated retrospectively.

Preliminary phytochemical screening of STEE was performed to identify alkaloids, flavonoids, phenolics, steroids, terpenoids, and saponins using standard qualitative protocols. Quantitative estimation of key phytoconstituents was carried out using spectrophotometric techniques. The total polyphenol content was expressed as milligrams of gallic acid equivalents per gram of extract (mg GAE/g), total flavonoid content as milligrams of quercetin equivalents per gram (mg QE/g), total alkaloid content as milligrams of atropine equivalents per gram (mg AE/g), and total terpenoid content as milligrams of taraxerol equivalents per gram (mg TAE/g) [3,10]. The qualitative results were recorded as present or not detected according to the cited color- and precipitation-based screening procedures. The archived record did not retain assay-specific reagent recipes, calibration equations, coefficients of determination, or LOD/LOQ values; consequently, the quantitative values should be checked against the source laboratory worksheets before publication.

2.2. Experimental animals

Healthy female Swiss albino mice (6–7 weeks old; 28–30 g) were obtained from the Pasteur Institute, Ho Chi Minh City, Vietnam. Before the experiment, the animals were acclimatized for 7 days under controlled environmental conditions (26–28 °C, 55–60% relative humidity, and a 12 h light/12 h dark cycle). Mice were housed in sanitized glass cages containing biologically treated rice-husk bedding and were provided with a nutritionally balanced rodent pellet diet and filtered drinking water ad libitum. Animal procedures followed institutional animal-care requirements; the archived study record did not contain the protocol approval identifier. Humane endpoints included severe distress, a moribund condition, and inability to access food or water. Body weight was monitored as an additional welfare indicator.

2.3. Acute oral toxicity test

Acute oral toxicity of the ethanolic extract of *Senna tora* (STEE) was evaluated using the sequential fixed-dose approach of OECD Guideline 423 [11,12]. Female Swiss albino mice (8–10 weeks old; 30–32 g) were acclimatized for 7 days under controlled conditions (24 ± 2 °C, 55–60% relative humidity, and a 12 h light/12 h dark cycle) with free access to standard diet and filtered water. Animals were fasted overnight (approximately 12 h) before dosing, with water available ad libitum.

STEE was administered by oral gavage at 10 mL/kg using 0.5% (w/v) carboxymethylcellulose sodium (CMC-Na) as the vehicle. In the first step, three females received 300 mg/kg and were observed individually during the first 4 h after dosing and at least once daily thereafter for 14 days for clinical signs, morbidity, mortality, and behavioral alterations. Body weight was recorded on Days 0, 7, and 14.

Because no mortality or severe toxicity occurred at 300 mg/kg, a second group of three females received 2000 mg/kg under the same observation schedule, giving a total of six animals in the acute-toxicity experiment. Humane endpoints were predefined, and a gross necropsy was conducted on all animals at death or at study termination. No additional confirmatory cohort was included in the archived study record. Results were summarized according to the LD_{50} cut-off categories specified by OECD 423. Because the experiment comprised two sequential groups without an additional confirmatory cohort, the resulting LD_{50} cut-off is reported as a preliminary estimate limited to the highest dose tested.

2.4. Antioxidant capacity assays

Antioxidant capacity of STEE was assessed in vitro using DPPH, ABTS, and FRAP assays. A methanolic STEE stock solution of 1.0 mg/mL was prepared by dissolving 10 mg of dried extract in 10 mL of methanol. The stock solution was freshly diluted with the corresponding assay solvent to working concentrations of 50, 100, 200, and 300 µg/mL. Matched solvent without STEE served as the 0 µg/mL control. Each condition was measured once in triplicate (n = 3 technical readings per condition); no independent replicate experiment was available. Technical replicates were used only to describe assay precision and were not treated as independent biological observations [13].

2.4.1. DPPH assay: A 0.1 mM DPPH solution was prepared in methanol and protected from light. For each

reaction, 1.0 mL of DPPH solution was mixed with 1.0 mL of sample or solvent blank and incubated for 30 min at 25 °C in the dark. Absorbance was measured at 517 nm. Radical-scavenging activity was calculated as: scavenging (%) = [(Acontrol − Asample)/Acontrol] × 100. No positive reference antioxidant was included in the archived assay. Results were expressed as mean ± SD [14].

2.4.2. ABTS assay*:* The ABTS•$^{+}$ working solution was generated by reacting 7 mM ABTS with 2.45 mM potassium persulfate for 12–16 h in the dark and then diluting the solution to an absorbance of 0.70 ± 0.02 at 734 nm. A sample (200 µL) was mixed with 2.0 mL of ABTS•$^{+}$ working solution, incubated for 6 min at 25 °C, and measured at 734 nm. Scavenging activity was calculated using the DPPH formula [14].

2.4.3. FRAP assay: FRAP reagent (acetate buffer, pH 3.6; 10 mM TPTZ in 40 mM HCl; and 20 mM $FeCl_3$ at a ratio of 10:1:1, v/v/v) was prewarmed to 37 °C. Sample (100 µL) was added to 3.0 mL of FRAP reagent, and absorbance at 593 nm was recorded after 4 min. Results were expressed as apparent µmol Fe^{2+}/L from a $FeSO_4$ calibration curve, including the solvent-control assay baseline [14]. The solvent-control absorbance was not subtracted because paired raw absorbance values were unavailable in the archived dataset. Accordingly, FRAP results are reported as unblanked apparent Fe^{2+}-equivalent concentrations and should be interpreted as within-assay concentration-response values rather than blank-corrected absolute antioxidant capacity.

2.5. *In vivo* arthritis model assessment

***2.5.1. Arthritis induction and treatment groups*:** Following acclimatization, mice were randomly allocated to six groups (n = 6): normal control, CFA control, CFA+meloxicam positive control, and CFA+STEE at 100, 200, or 300 mg/kg/day. On Day 0, arthritis was induced by a single subcutaneous injection into the plantar surface of the right hind paw of 100 µL of complete Freund's adjuvant (CFA; 1 mg/mL heat-killed *Mycobacterium tuberculosis* H37Ra, ATCC 25177; Sigma-Aldrich, St. Louis, MO, USA; catalog no. F5881) in all groups except the normal control. Arthritis was allowed to develop for 7 days. Treatment began on Day 8 after CFA injection and continued once daily by oral gavage for 21 consecutive days, ending on Day 28 [7]. Meloxicam (Mobic; Boehringer Ingelheim, Vietnam) was administered at 0.2 mg/kg/day in 0.5% (w/v) CMC-Na. STEE was administered at 100, 200, or 300 mg/kg/day in the same vehicle. Normal and CFA control animals received an equivalent volume of 0.5% CMC-Na (10 mL/kg/day).

2.5.2. Body weight monitoring: Body weight was measured every 7 days using a Sartorius Entris 3202i-1S electronic scale (Sartorius, Germany) and was used for welfare surveillance because systemic inflammation can influence body composition and weight trajectories [15,16]. Group-level summaries showed separation among the future treatment groups at Day 7, before dosing began on Day 8. Because individual records were unavailable to verify randomization balance or perform a repeated-measures analysis, body weight was not used as an efficacy outcome and is not presented in the Results.

2.5.3. Hind-paw response: The right hind-paw response was recorded weekly using the study's digital-caliper procedure, with the assessor blinded to group allocation. The absolute scale in the archived dataset could not be independently verified; therefore, only normalized changes from the Day 0 group mean are presented. Relative change was calculated descriptively as $[(M_t - M_0)/M_0] \times 100$, where M_t is the group mean at each time point, and M_0 is the corresponding Day 0 group mean. These normalized group-level values were not used for inferential testing.

2.6. Evaluation of oxidative stress markers in vivo

At the end of Day 28, mice were deeply anesthetized with ketamine (100 mg/kg) and xylazine (10 mg/kg), administered intraperitoneally. After loss of the pedal-withdrawal and corneal reflexes, euthanasia was completed by cervical dislocation performed by trained personnel. Death was confirmed by the permanent cessation of spontaneous respiration and heartbeat, together with the absence of corneal and pedal-withdrawal reflexes. Right hind-paw joint-region tissue (the articular/periarticular tissue dissected from the CFA-injected limb) was rapidly excised, rinsed in ice-cold PBS, snap-frozen in liquid nitrogen, and stored at −80 °C until analysis. For each assay, approximately 50 mg of tissue was homogenized in 500 µL of the assay-specific ice-cold buffer, corresponding to a tissue-to-buffer ratio of 1:10 (w/v). Total protein concentration was determined using the Pierce BCA Protein Assay Kit (Thermo Fisher Scientific, Rockford, IL, USA; catalog no. 23225) according to the manufacturer's microplate protocol, with bovine serum albumin as the standard.

***2.6.1. ROS levels*:** Reactive oxygen species in joint tissue were assessed using the DCFH-DA assay. Tissue supernatant was incubated with 10 µM DCFH-DA at 37 °C for 30 min in the dark, and fluorescence was measured at 488 nm excitation and 525 nm emission [17]. Because the source method did not document a DCF calibration curve, the results are reported as arbitrary fluorescence units per milligram of protein (AU/mg protein), not nmol/mg protein.

2.6.2. Catalase (CAT) activity: CAT activity was measured by monitoring the decrease in absorbance of 30 mM H_2O_2 at 240 nm. Tissue supernatant (50 µL) was mixed with 950 µL H_2O_2 solution, and absorbance was recorded every 15 s for 1 min in a 1-cm optical path. Activity was calculated as $(-\Delta A_{240}/\text{min} \times V\text{total})/(\varepsilon_{240} \times l \times V\text{sample} \times \text{protein concentration})$, using $\varepsilon_{240} = 43.6\ M^{-1}\ cm^{-1}$ and $l = 1$ cm, and was expressed as µmol H_2O_2 decomposed/min/mg protein [9].

***2.6.3. Superoxide dismutase (SOD) activity*:** SOD activity was measured using the SOD Assay Kit–WST (Dojindo Laboratories, Kumamoto, Japan; catalog no. S311) according to the manufacturer's instructions. Briefly, 20 µL of appropriately diluted tissue supernatant was mixed with 200 µL of WST working solution and 20 µL of enzyme working solution. Corresponding sample and reagent blanks were prepared using dilution buffer or double-distilled water. After incubation at 37 °C for 20 min, absorbance was measured at 450 nm. SOD inhibition was calculated from the corrected absorbance values, and one unit of SOD was defined as the amount of enzyme producing 50% inhibition of WST-1 reduction. Activity was corrected for dilution and normalized to total protein, with results expressed as U/mg protein [9].

2.6.4. Glutathione peroxidase (GPx) activity: GPx activity was measured by coupling glutathione oxidation to NADPH consumption at 340 nm. Tissue supernatant (20 µL) was mixed with 100 µL reaction buffer containing 5 mM GSH, 1 U/mL glutathione reductase, and 0.2 mM NADPH; 20 µL of 1.5 mM H_2O_2 initiated the reaction. Absorbance was recorded every 30 s for 5 min. Activity was calculated from $-\Delta A340/\text{min}$ using $\varepsilon 340(\text{NADPH}) = 6.22\ mM^{-1}\ cm^{-1}$ and the verified optical path length. One unit was defined as oxidation of 1 µmol NADPH/min under the assay conditions, and results were expressed as U/mg protein [9].

2.7. Statistical analysis

Data are presented as mean ± SD. Endpoint biochemical outcomes (joint ROS signal, CAT, SOD, and GPx) were analyzed by one-way ANOVA followed by Tukey's honestly significant difference test (two-sided $\alpha = 0.05$). Assumption checks based on the original animal-level data could not be independently verified because the animal-level worksheet was unavailable. In vitro antioxidant results were summarized descriptively because the reported n = 3 values were technical replicates. The normalized hind-paw series was also presented descriptively because individual repeated-measures data were unavailable for a mixed-effects or repeated-measures analysis. Sample sizes were n = 6 mice per in vivo group and n = 3 technical replicates per in vitro condition. Omnibus ANOVA results are reported as F statistics with numerator and denominator degrees of freedom, exact p values, and eta-squared (η^2) effect sizes. Because the supplied manuscript did not include the animal-level endpoint worksheet, these omnibus statistics were recalculated from the archived group means, SDs, and n values and must be cross-checked against the original analysis output before publication.

3. RESULTS AND DISCUSSION

3.1. Phytochemical composition of the ethanol extract of *Senna tora*

STEE contained flavonoids, terpenoids, polyphenols, and alkaloids, whereas saponins and steroids were not detected by the qualitative screening procedure used (Table 1).

Table 1. Qualitative and quantitative analysis of phytochemicals in the ethanol extract of *Senna tora* (STEE).

Phytochemical	Presence in STEE	Content
Flavonoids	+	40.89 ± 1.37 mg QE/g
Terpenoids	+	66.95 ± 1.76 mg TAE/g
Polyphenols	+	69.98 ± 1.49 mg GAE/g
Alkaloids	+	4.28 ± 0.21 mg AE/g
Saponins	–	ND
Steroids	–	ND

Note: "+" = detected; "–" = not detected by the qualitative screening procedure used. Quantitative values are mean ± SD (n = 3). ND = not detected by the qualitative screening procedure.

The quantified constituents were present at appreciable levels, indicating that STEE is rich in phenolic and terpenoid compounds.

Flavonoids, polyphenols, terpenoids, and alkaloids have reported anti-inflammatory, antioxidant, and immunomodulatory activities [18–20]. The phytochemical profile observed here is consistent with previous reports on Senna tora and related Senna species [3,10]. These constituents may contribute collectively to the biological effects observed in the CFA-induced arthritis model; however, their individual contributions were not determined in the present study.

3.2. Acute oral toxicity

In the two sequential OECD 423 dose groups (three mice at 300 mg/kg and three mice at 2000 mg/kg), no mortality or severe treatment-related toxicity was observed during the 14-day observation period. Animals remained clinically normal without signs of distress or gross behavioral abnormalities, and gross necropsy revealed no reported treatment-related abnormality. Under the conditions of this limited two-step experiment, the LD_{50} cut-off was preliminarily estimated to be greater than 2000 mg/kg; no conclusion beyond the highest dose tested is warranted [12].

These findings are consistent with reports of low acute oral toxicity for S. tora preparations in rodents [21,22]. Together with the absence of overt toxicity in the present study, the results support the selection of 100–300 mg/kg/day for subsequent in vivo testing and provide a basis for subsequent subchronic and chronic toxicity studies.

3.3. Antioxidant capacity assessed by DPPH, ABTS, and FRAP assays

STEE showed concentration-related increases in DPPH and ABTS radical-scavenging activity and in apparent FRAP values (Table 2).

Table 2. In vitro antioxidant capacity of STEE at 0–300 µg/mL, assessed by DPPH, ABTS, and FRAP assays.

STEE concentration	% DPPH scavenging	% ABTS scavenging	FRAP assay (apparent µmol Fe^{2+}/L)
STEE 0 µg/mL (solvent control)	0.00 ± 0.00	0.00 ± 0.00	278.66 ± 42.75
STEE 50 µg/mL	14.15 ± 0.41	29.35 ± 1.19	402.57 ± 37.66
STEE 100 µg/mL	39.85 ± 1.13	52.56 ± 1.65	594.24 ± 39.79
STEE 200 µg/mL	58.37 ± 0.90	63.47 ± 1.38	777.95 ± 45.28
STEE 300 µg/mL	75.48 ± 0.98	74.81 ± 1.31	986.43 ± 51.25

Values are mean ± SD of triplicate measurements (n = 3 technical replicates per condition). DPPH and ABTS are reported as percentage scavenging. FRAP values are unblanked apparent Fe^{2+}-equivalent concentrations derived from the $FeSO_4$ calibration curve. The 0 µg/mL result (278.66 ± 42.75 µmol Fe^{2+}/L) is the retained solvent/reagent baseline and was not subtracted because paired raw absorbance data were unavailable. Thus, the FRAP series should be interpreted only as a within-assay concentration-response pattern. No inferential comparisons were made across technical replicates.

The solvent control showed negligible DPPH and ABTS scavenging, whereas progressively higher responses were observed at 50–300 µg/mL. Because the triplicate readings were technical replicates, these results are interpreted as descriptive concentration-response patterns rather than independent inferential comparisons.

The DPPH, ABTS, and FRAP findings are consistent with the high polyphenol, flavonoid, and terpenoid contents of STEE. These compound classes can donate electrons or hydrogen atoms and thereby neutralize reactive species [23]. Oxidative stress contributes to inflammatory signaling and tissue injury in rheumatoid arthritis [9]. Proposed effects on NF-κB, Nrf2, and cytokine signaling remain mechanistic hypotheses because these pathways were not directly measured [24–26].

3.4. Descriptive normalized hind-paw response

All CFA-induced groups showed a comparable marked increase in the normalized hind-paw response by Day 7, before treatment began. From Day 14 through Day 28, the response continued to increase in the untreated CFA group but declined in the meloxicam and STEE groups. At Day 28, the relative change from the Day 0 group mean was approximately 731.3% in the CFA group, 309.7% in the CFA+meloxicam group, and 395.1%, 381.0%, and 323.9% in the CFA+STEE100, CFA+STEE200, and CFA+STEE300 groups, respectively (Figure 1).

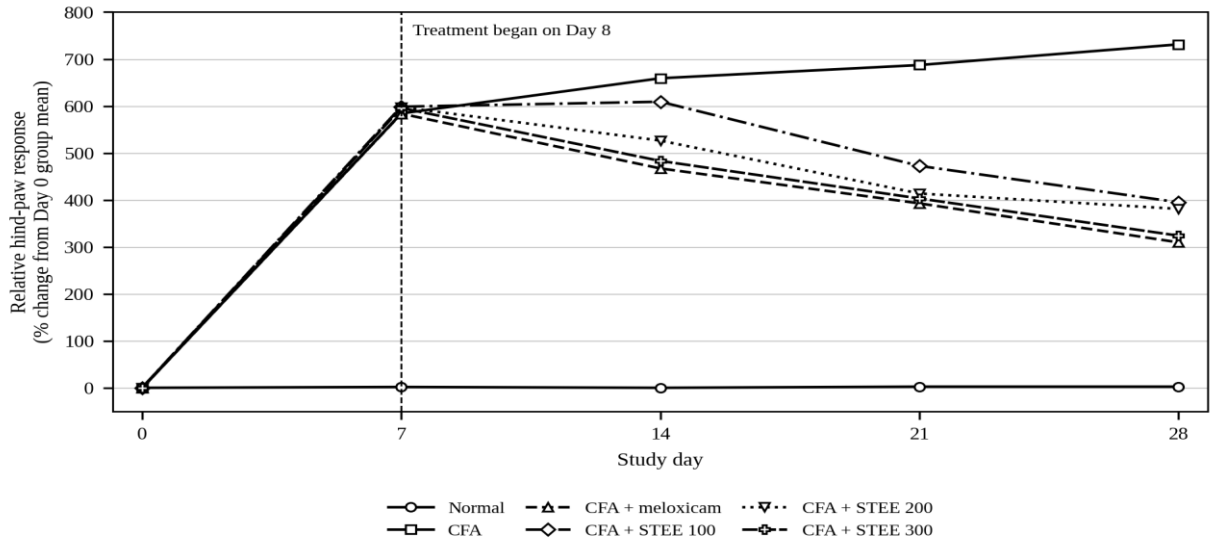


Figure 1. Descriptive relative change in the recorded right hind-paw response during the 28-day study. Values were calculated from group means after normalization to each group's Day 0 mean. Treatment began on Day 8. Individual repeated-measures data and the absolute measurement scale were unavailable; therefore, error bars and inferential comparisons are not shown.

These trajectories are compatible with a lower post-treatment hind-paw response in the STEE groups, particularly at 300 mg/kg/day. However, because the absolute scale and individual longitudinal records could not be verified, the normalized series is descriptive and does not by itself establish treatment efficacy. Plant-derived extracts can influence cytokine, prostaglandin, and other inflammatory pathways in experimental models [27–30]. However, cytokines, COX-2, iNOS, matrix metalloproteinases, and joint histopathology were not measured here; proposed anti-inflammatory mechanisms therefore remain hypotheses.

3.5. Effects of STEE on oxidative stress markers in CFA-induced arthritic mice

CFA-induced arthritis markedly increased the joint-tissue ROS signal and decreased CAT, SOD, and GPx activities (Figure 2 and Table 3). The ROS signal was 6.33 ± 0.04 AU/mg protein in the CFA group compared with 1.22 ± 0.03 AU/mg protein in the normal group. STEE reduced the ROS signal in a dose-related manner, reaching 1.72 ± 0.03 AU/mg protein at 300 mg/kg/day.

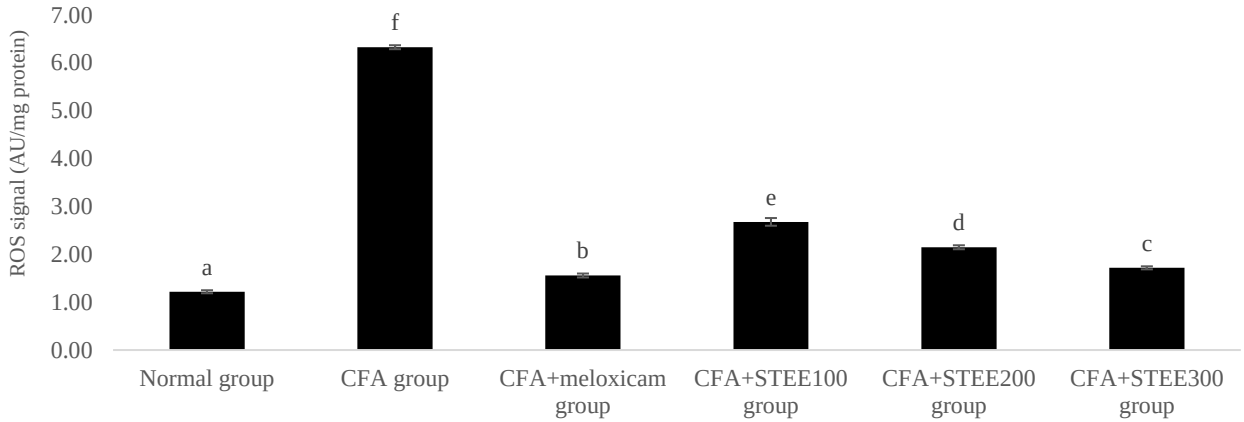


Figure 2. Joint-tissue ROS signal in CFA-induced arthritic mice treated with STEE, expressed as AU/mg protein (mean ± SD, n = 6). Bars with different superscript letters (a–f) differ significantly ($p < 0.05$, one-way ANOVA followed by Tukey's test). Omnibus ANOVA: $F(5, 30) = 9383.93$, $p = 5.95 \times 10^{-47}$, $\eta^2 = 0.999$.

Table 3. Effect of STEE on CAT, SOD, and GPx activities in joint tissues of CFA-induced arthritic mice.

Experimental group	Catalase (CAT) (μmol H_2O_2/min/mg protein)	Superoxide dismutase (SOD) (U/mg protein)	Glutathione peroxidase (GPx) (U/mg protein)
Normal group	3.82 ± 0.06^{f}	28.46 ± 1.44^{e}	47.64 ± 1.94^{e}
CFA group	1.53 ± 0.03^{a}	11.38 ± 1.22^{a}	19.06 ± 1.35^{a}
CFA+meloxicam group	3.18 ± 0.08^{e}	23.72 ± 1.58^{d}	39.71 ± 1.89^{d}
CFA+STEE100 group	2.01 ± 0.05^{b}	14.98 ± 1.34^{b}	25.07 ± 1.56^{b}
CFA+STEE200 group	2.25 ± 0.04^{c}	16.74 ± 1.64^{b}	28.02 ± 1.87^{b}
CFA+STEE300 group	2.73 ± 0.07^{d}	20.33 ± 1.92^{c}	34.03 ± 1.95^{c}

Note: Data are expressed as mean ± SD (n = 6). Within each column, groups with different superscript letters differ significantly ($p < 0.05$, one-way ANOVA followed by Tukey's test). Omnibus ANOVA results were: CAT, $F(5, 30) = 1250.46$, $p = 7.49 \times 10^{-34}$, $\eta^2 = 0.995$; SOD, $F(5, 30) = 97.37$, $p = 1.30 \times 10^{-17}$, $\eta^2 = 0.942$; and GPx, $F(5, 30) = 205.46$, $p = 2.94 \times 10^{-22}$, $\eta^2 = 0.972$.

CAT activity increased from 1.53 ± 0.03 μmol H_2O_2/min/mg protein in the CFA group to 2.73 ± 0.07 μmol H_2O_2/min/mg protein in the CFA+STEE300 group. SOD and GPx activities showed similar partial recovery. Each biochemical n represented one joint-region tissue homogenate from one mouse. The very small ROS and CAT SDs were retained as archived; because the animal-level worksheet was not supplied with the manuscript, these dispersion estimates could not be independently audited and require verification against the original raw data before publication.

The in vivo results indicate that STEE counteracted the oxidative imbalance associated with CFA-induced arthritis. Excess ROS can damage joint tissues and amplify inflammatory signaling through redox-sensitive pathways [31]. Polyphenols and flavonoids may reduce oxidative stress directly through radical scavenging and indirectly by supporting endogenous antioxidant systems [32–34]. The recovery of CAT, SOD, and GPx activities is compatible with improved cellular redox regulation. Although Nrf2 activation is a plausible mechanism [35], Nrf2 and related signaling proteins were not directly assessed and should be investigated in future work.

4. CONCLUSIONS

Oral administration of *Senna tora* (L.) Roxb. fruit extract at 100–300 mg/kg/day for 21 consecutive days was associated with reduced joint-tissue ROS signal and partial restoration of CAT, SOD, and GPx activities in CFA-induced arthritic mice. A normalized group-level hind-paw series also decreased after treatment, but it was interpreted descriptively because the absolute scale and individual repeated-measures records were unavailable. The results support further investigation of STEE as a source of antioxidant and

potentially anti-arthritic compounds, but they do not establish clinical efficacy. Future studies should include a documented animal-ethics approval identifier, verified individual longitudinal data, inflammatory cytokines, joint histopathology, active-compound identification, pharmacokinetics, and long-term safety.

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TÁC DỤNG CHỐNG OXY HÓA VÀ TIỀM NĂNG CHỐNG VIÊM KHỚP CỦA DỊCH CHIẾT ETHANOL TỪ QUẢ THẢO QUYẾT MINH (*Senna tora* (L.) Roxb.) TRÊN MÔ HÌNH CHUỘT GÂY VIÊM KHỚP BẰNG CFA

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Tóm tắt.
Mục tiêu: Đánh giá dịch chiết ethanol từ quả *Senna tora* (L.) Roxb. (STEE) trên mô hình chuột gây viêm khớp bằng CFA, đồng thời khảo sát thành phần hóa thực vật và khả năng chống oxy hóa. Vật liệu và phương pháp: Chuột cái bạch tạng Thụy Sĩ được chia vào các nhóm bình thường, CFA, CFA+meloxicam và CFA+STEE (100, 200 hoặc 300 mg/kg/ngày). Viêm khớp được gây vào ngày 0; thuốc và dịch chiết được dùng đường uống từ ngày 8 đến ngày 28. Đáp ứng bàn chân sau được chuẩn hóa theo trung bình ngày 0 của từng nhóm và phân tích mô tả vì không có dữ liệu lặp lại theo từng cá thể. Tín hiệu ROS trong mô khớp và hoạt độ CAT, SOD, GPx được đo; các đáp ứng DPPH, ABTS và FRAP in vitro cũng được khảo sát. Kết quả: STEE cho đáp ứng chống oxy hóa tăng theo nồng độ. Ở liều 300 mg/kg/ngày, tín hiệu ROS là 1,72 ± 0,03 AU/mg protein so với 6,33 ± 0,04 ở nhóm CFA, đồng thời hoạt độ CAT, SOD và GPx cao hơn ($p < 0,05$). Quỹ đạo đáp ứng bàn chân sau đã chuẩn hóa giảm sau điều trị bằng meloxicam hoặc STEE. Kết luận: STEE làm giảm stress oxy hóa ở mô khớp liên quan đến CFA và cho thấy xu hướng giảm đáp ứng bàn chân sau ở mức mô tả. Hiệu quả chống viêm khớp cần được xác nhận bằng dữ liệu dọc theo từng cá thể, mô bệnh học khớp, các dấu ấn viêm và đánh giá an toàn dài hạn.

Từ khóa: *Senna tora*, thảo quyết minh, viêm khớp dạng thấp, hoạt tính chống oxy hóa, stress oxy hóa, tác dụng chống viêm, mô hình chuột gây viêm khớp bằng CFA.

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