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Research Article

IN-VITRO EVALUATION OF THE ANTI-INFLAMMATORY ACTIVITY OF *BHARANGI* (ROTHECA SERRATA) USING PRO-INFLAMMATORY CYTOKINE MARKERS

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ABSTRACT

Inflammation is a protective biological response to harmful stimuli; however, excessive production of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α contributes to chronic inflammatory disorders and immune dysregulation. Targeted cytokine inhibitors are effective but often costly and associated with adverse effects, highlighting the need for safer, affordable alternatives. In Ayurvedic medicine several had described with anti-inflammatory properties*, Bharangi* (*Rotheca serrata)*, commonly used in inflammatory and respiratory conditions. Despite its classical indications, scientific validation of its cytokine-modulating activity remains limited, necessitating systematic in-vitro evaluation to substantiate its therapeutic potential. **Objectives**: To evaluate the anti-inflammatory activity of *Bharangi* (*Rotheca serrata*) through an in-vitro method using interleukin-1β, interleukin-6, and TNF-α as inflammatory markers. **Material and Methods**: *Bharangi* (*Rotheca serrata*) root powder was extracted with ethanol, centrifuged, and the supernatant was used for analysis. Anti-inflammatory activity was evaluated in THP-1 human monocytic cells using TNF-α, IL-6, and IL-1β inhibition assays, with diclofenac as the standard. Cytotoxicity was assessed by the MTT assay, and cytokine levels were measured using ELISA kits. The data were analyzed using one-way ANOVA followed by Tukey's post-hoc test at a 5% significance level. **Results**: *Bharangi* (*Rotheca serrata*) root powder showed notable inhibition of pro-inflammatory cytokines in vitro. TNF-α inhibition ranged from 46.03% to 39.71%, IL-6 from 70.33% to 66.56%, and IL-1β from 57.92% to 47.52% across the tested concentrations, indicating moderate anti-inflammatory activity. **Conclusions**: *Bharangi* root demonstrated in-vitro anti-inflammatory activity by inhibiting TNF-α, IL-6, and IL-1β, supporting its traditional use in inflammatory conditions

INTRODUCTION

Inflammation is a regulated biological response that protects the host against infections, tissue injury, and toxic insults. It is initiated through recognition of pathogen-associated and damage-associated molecular patterns by innate immune receptors, leading to activation of intracellular signaling cascades and transcriptional programs that coordinate immune defence and tissue repair.[1] Although acute inflammation is essential for maintaining homeostasis, its persistence or dysregulation contributes to the pathogenesis of numerous chronic and immune mediated disorders.

A hallmark of the inflammatory response is the production of pro-inflammatory cytokines, which act as critical regulators of immune cell recruitment, activation, and effector function.[2] Among these, interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor-alpha (TNF-α) constitute a central cytokine axis driving both local and systemic inflammation. IL-1 functions as a master upstream mediator, amplifying inflammatory cascades through induction of adhesion molecules, chemokines, and secondary cytokines, thereby sustaining leukocyte recruitment and febrile responses.[3] TNF-α promotes endothelial activation, vascular permeability, and propagation of inflammatory signalling networks,

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reinforcing chronic inflammatory states. IL-6 exerts pleiotropic effects, regulating acute-phase protein synthesis, B-cell differentiation, and T-cell responses. Notably, IL-6 signalling through both classical and trans-signalling pathways has been implicated in the transition from acute to chronic inflammation.[4,5]

Mounting evidence implicates sustained overproduction of IL-1, IL-6, and TNF-α in the pathophysiology of autoimmune and inflammatory diseases, including rheumatoid arthritis, inflammatory bowel disease, and atherosclerosis.[6] Furthermore, uncontrolled cytokine release can precipitate hyperinflammatory syndromes, commonly referred to as cytokine storms, characterized by systemic immune dysregulation and multiorgan dysfunction, as observed in severe viral infections such as COVID-19.[6,7,8] These insights have led to the development of targeted biologic therapies that inhibit IL-1, IL-6, or TNF-α signalling, demonstrating the clinical relevance of cytokine modulation.[9,10] However, despite their therapeutic efficacy, biologics are associated with limitations including high cost, risk of opportunistic infections, and incomplete or variable patient response, underscoring the need for alternative or adjunctive anti-inflammatory strategies.

Traditional medicinal systems, particularly Ayurveda, describe several botanicals with anti-inflammatory and immunomodulatory potential. *Bharangi*, botanically identified as *Rotheca serrata* (syn. *Clerodendrum serratum*), is a well-documented medicinal plant cited in classical Ayurvedic texts for its *Shothaghna* (anti-inflammatory) and *Kasahara* (anti-tussive) properties.[11] It has traditionally been prescribed for inflammatory conditions, respiratory disorders, and swelling, suggesting a possible role in modulating immune-mediated pathways. Preliminary pharmacological investigations have indicated the presence of bioactive phytoconstituents such as flavonoids, phenolics, and diterpenoids in *Bharangi*, which are known to possess anti-inflammatory and antioxidant activities. However, the precise molecular mechanisms underlying its anti-inflammatory effects, particularly its influence on key pro-inflammatory cytokines central to immune dysregulation, remain inadequately characterized.

In light of the critical role of IL-1β, IL-6, and TNF-α in inflammatory pathogenesis and therapeutic targeting, systematic evaluation of *Bharangi's* modulatory effects on these cytokines is warranted. Therefore, the present study was designed to investigate the anti-inflammatory potential of *Bharangi* through in vitro assessment of IL-1β, IL-6, and TNF-α, aiming to provide mechanistic insight and scientific validation for its traditional use.

MATERIALS & METHODS

Materials

Plant material: collection and identification

The roots of *Bharangi* (*Rotheca serrata* (L.) Steane & Mabb., syn. *Clerodendrum serratum* (L.) Moon) were collected in May 2024 from Sai Village, Panvel, Raigad, Maharashtra. The plant material was identified based on morphology & macroscopic characteristics as per API.

Plant authentication

Authentication of the collected plant material was carried out as per the standards described in the Ayurvedic Pharmacopoeia of India (API).[12] The authentication was performed at the Y.M.T Ayurveda Medical College, Navi Mumbai, and further verified at the Alarsin Pharmaceutical Laboratory, Mumbai. The in-vitro study was conducted at the Scientific Research Centre of Kelkar Education Trust's, Mulund, Mumbai.

Preparation of Plant Extract

The collected roots were thoroughly washed with running tap water to remove adhering soil and foreign matter and shade-dried at room temperature (25–30°C) until complete drying. The dried material was coarsely powdered using a mechanical grinder and stored in airtight containers.

Bharangi root powder (10mg) was accurately weighed and dissolved in 1ml of ethanol to obtain a stock solution (10mg/ml). The mixture was vortexed thoroughly to ensure proper dissolution and subsequently centrifuged at 3000 rpm for 10 minutes to remove insoluble particulate matter. The clear supernatant was carefully collected and used for further experimental procedures.[13]

Working concentrations were prepared by diluting the supernatant with RPMI-1640 culture medium to obtain final concentrations of 0.125, 0.25, and 0.5mg/ml. The final ethanol concentration in the culture medium was maintained at a minimal level to avoid solvent-induced cytotoxicity.

Preparation of standard drug solution

Diclofenac was used as the standard anti-inflammatory drug. A stock solution was prepared at a concentration of 10mg/ml in RPMI-1640 medium. Serial dilutions were performed using culture medium to obtain working concentrations of 0.06, 0.125, 0.25, and 0.5mg/ml, corresponding to the test sample concentrations.[14,15]

All prepared solutions were freshly prepared prior to each experiment.

Cell line and culture conditions

The human monocytic cell line THP-1 was procured from a certified cell repository and maintained in RPMI-1640 medium supplemented with 10% heat-inactivated foetal bovine serum and 1%

penicillin–streptomycin solution. Cells were cultured at 37°C in a humidified incubator containing 5% CO_2.

For experimental assays, cells were seeded into sterile 96-well culture plates at a density of 0.5×10^4 cells per well in 100µl of complete medium and allowed to stabilize for 48hr under standard culture conditions.[16]

Experimental design and treatment protocol

To induce an inflammatory response, cells were stimulated with lipopolysaccharide (LPS) at an optimized concentration of 1µg/ml. After stabilization, culture medium was replaced with fresh medium containing different concentrations of the test sample or the standard drug (diclofenac), in the presence of LPS.[17]

The experimental groups were as follows:

- LPS control
- Test sample + LPS
- Diclofenac + LPS

Following treatment, plates were incubated for an additional 24 h at 37°C in 5% CO_2.

Cell viability assessment (MTT assay)

Cell viability was evaluated using the MTT assay to determine the cytotoxicity of the test sample and diclofenac. After completion of treatment, 20µl of MTT reagent (5mg/mL in PBS) was added to each well and incubated for 3–4 h at 37°C to allow formazan crystal formation by viable cells.

The supernatant was carefully removed, and the crystals were dissolved in dimethyl sulfoxide (DMSO). Absorbance was measured at 570nm using a microplate reader. Cell viability was expressed as a percentage relative to the normal control group.[18]

Estimation of pro-inflammatory cytokines

After treatment, culture supernatants were collected and centrifuged at 3000 rpm for 10 min at 4°C to remove cellular debris. The concentrations of tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β) were quantified using commercially available human ELISA kits, according to the manufacturer's instructions.[19]

Briefly, standards and samples were added to pre-coated ELISA plates and incubated as specified in the kit protocol. After washing steps, biotinylated detection antibody followed by streptavidin–HRP conjugate was added. Colour development was achieved using TMB substrate, and the reaction was terminated using stop solution. Optical density was measured at 450nm using a microplate reader. Cytokine concentrations were calculated from the standard curve and expressed in pg/mL.[20]

Statistical Analysis

All experiments were performed in triplicate. Data were expressed as mean±standard deviation (SD). Statistical comparisons were performed using one-way analysis of variance (ANOVA) followed by an appropriate post hoc test. A p-value <0.05 was considered statistically significant.[21,22]

RESULT

Macroscopy & Microscopy



Fig 1. *Bharangi* (*Rotheca serrata*) plant



Fig 2. Inflorescence of *Bharangi* (*Rotheca serreta*)



Fig 3. Root of *Bharangi* (*Rotheca serrata*)



Fig 4. Powder Root of *Bharangi* (*Rotheca serrata*)

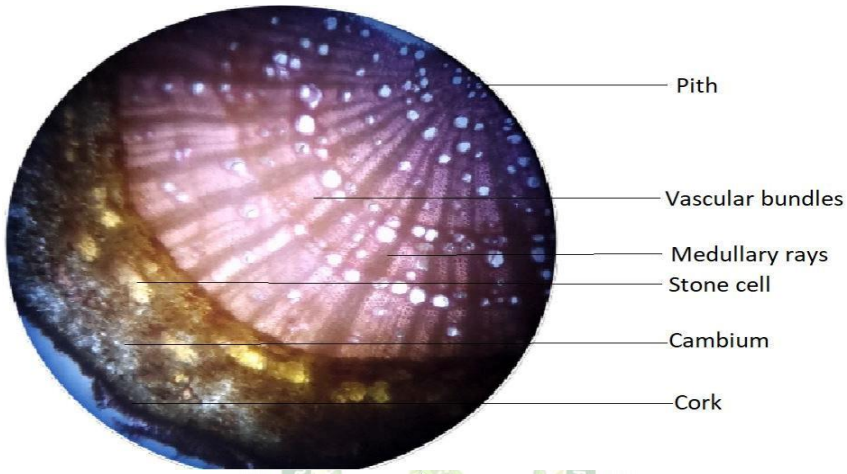


Fig. 5 T.S of *Bharangi* (Rotheca serrata) Root

Physico- Chemical Property

Ash analysis

Table 1: Ash Analysis of *Bharangi* root powder

S.No	Ash value %	Obtained	As per API (Part- I volume-3)
1	Total Ash % (W/W not > than)	7.65%	Not more than 11%
2	Acid Insoluble Ash % (W/W not > than)	0.83%	Not more than 1%

Extractive value

Table 2: Extractive value of *Bharangi* root powder

S.No		Obtained	As per API (Part- I volume-3)
1	Water extractive % (w/w Not < than)	13.34%	Not less than 12%
2	Alcohol extractive % (w/w not < than)	8.16%	Not less than 6 %

Phytochemical analysis

Table 3: Phytochemical analysis of *Bharangi* root powder

No.	Plant constituents Test	Water extract	Ethanol Extract
i)	Test for Starch	+	+
ii)	Test for Reducing sugars		
	a) Fehling test	+	+
	b) Benedict's test	+	+
iii)	Test for Mucilage	+	-
iv)	Test for amino acid	-	-
v)	Test for Proteins		

	a) Biuret test	+	+
	b) Xanthoproteic test	+	+
	c) Millon's test	+	+
vi)	Test for Alkaloids		
	a) Mayer's test	+	+
	b) Wagner's test	+	+
	c) Dragendorff's test	+	+
vii)	Test for Tannins		
	a) Ferric chloride test	+	+
	b) Lead acetate test	+	+
	c) Potassium dichromate test	+	+
viii)	Test for Cardiac Glycosides	+	+
ix)	Test for Anthraquinone Glycosides	+	+
x)	Test for Cyanogenetic Glycosides	-	-
xi)	Test for Steroids		
	a) Salkowski test	+	-
	b) Liebermann-Burchard test	-	-
xii)	Test for Terpenoids	+	+
xiii)	Test for Flavonoids	+	+
xiv)	Test for saponin		
	a) Foam test	+	+
	b) Haemolysis test	+	+

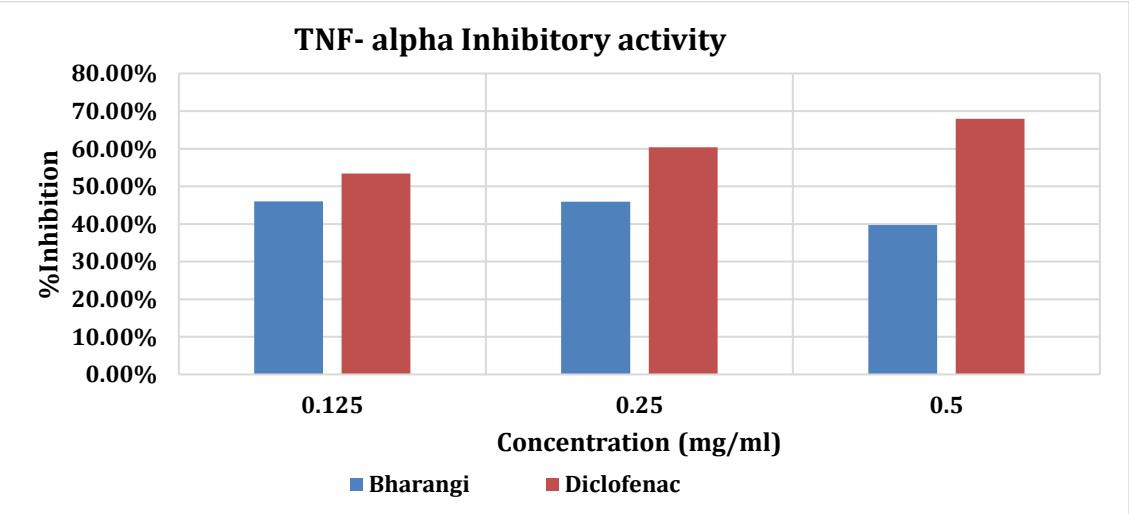
Pharmacological Study

TNF- alpha Inhibitory activity

Table 4: TNF- alpha Inhibitory activity of *Bharangi*, Diclofenac & Control

Concentration (mg/ml)	Bharangi root powder Percent Inhibition (%)	Diclofenac Percent Inhibition (%)	LPS Control Percent Inhibition (%)
0.125	46.03%	53.45%	0%
0.25	45.91%	60.41%	0%
0.5	39.71%	67.94%	0%

Table 5: Graphical representation of TNF- alpha Inhibitory activity

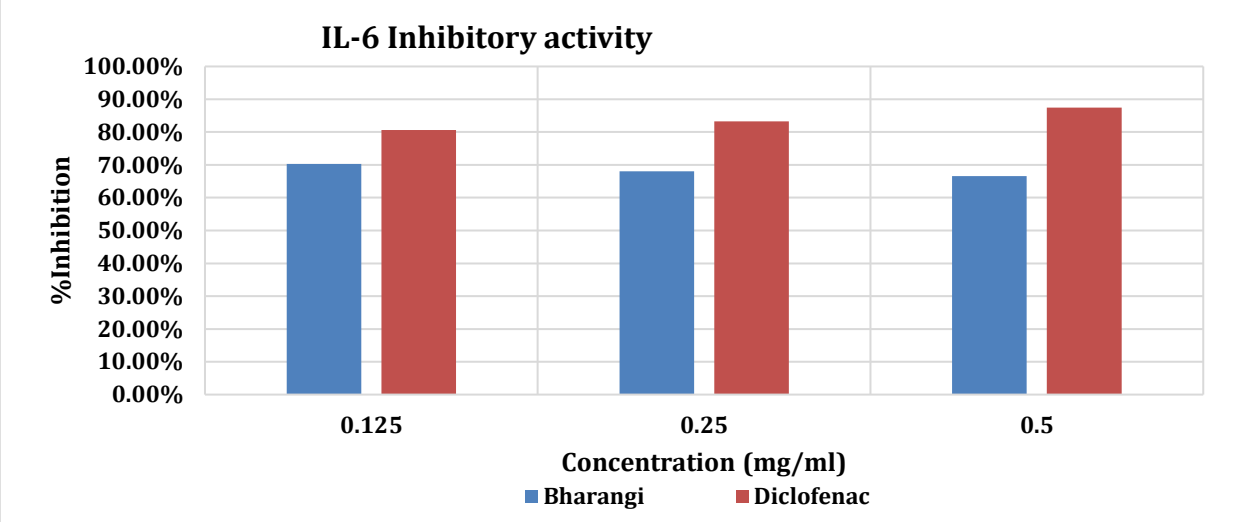


IL-6 Inhibitory activity

Table 6: IL-6 Inhibitory activity of *Bharangi*, Diclofenac & Control

Concentration (mg/ml)	*Bharangi* root powder Percent Inhibition (%)	Diclofenac Percent Inhibition (%)	LPS Control Percent Inhibition (%)
0.125	70.33%	80.59%	0%
0.25	68.07%	83.26%	0%
0.5	66.56%	87.49%	0%

Table 7: Graphical representation of IL-6 Inhibitory activity

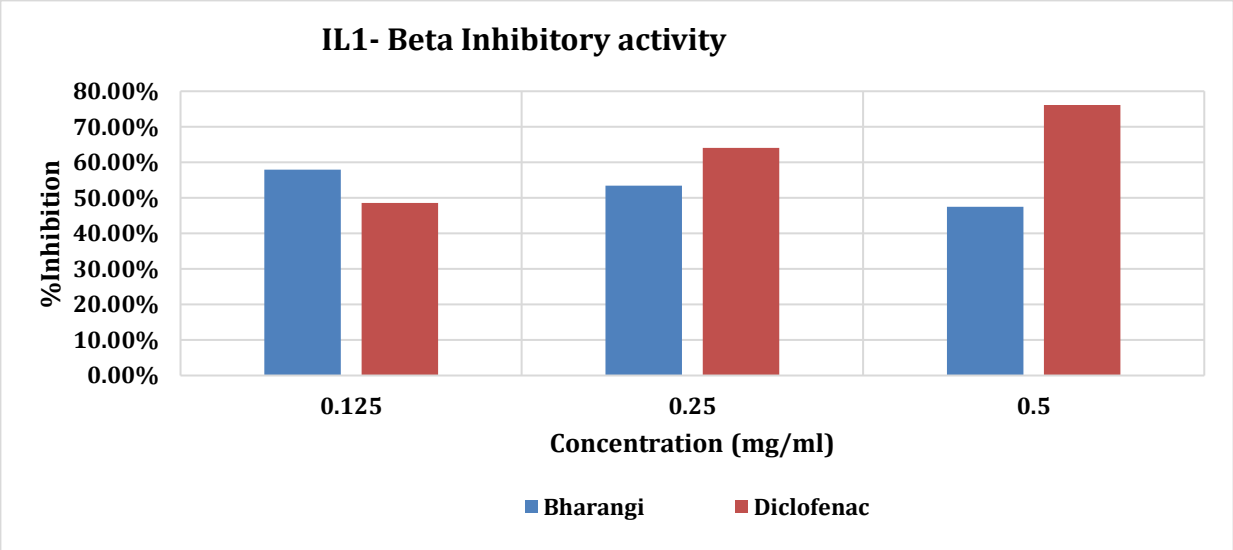


IL1- Beta Inhibitory activity

Table 8: IL1- Beta Inhibitory activity of *Bharangi*, Diclofenac & Control

Concentration (mg/ml)	Bharangi root powder Percent Inhibition (%)	Diclofenac Percent Inhibition (%)	LPS Control Percent Inhibition (%)
0.125	57.92%	48.55%	0%
0.25	53.46%	64.05%	0%
0.5	47.52%	76.13%	0%

Table 9: Graphical representation of IL1- Beta Inhibitory activity



Statistical Analysis

For each assay, we test the following hypotheses:

Null Hypothesis (H_0): There is no significant difference in the inhibition levels among the sample types (*Bharangi* root powder, diclofenac, and control).

Alternative Hypothesis (H_1): There is significant difference in the inhibition levels among the sample types (*Bharangi* root powder, diclofenac, and control).

Results for TNF-alpha Assay

Table 10: Descriptive statistic for TNF- alpha Inhibitory activity

Sample Type	N (Number of Cases)	Mean Inhibition (%)	Std. Deviation
Bharangi root powder	3	43.88	2.086
Diclofenac	3	60.60	4.183
Control	3	0	0

Table 11: ANOVA Table for TNF-alpha

Source	Sum of Squares	df	Mean Square	F	p-value
Between groups	5877.55	2	2938.78	134.43	<0.001
Within groups	131.17	6	21.86		
Total	6008.72	8			

Table 12: Post Hoc Tukey Test for TNF-alpha

Sample Type	Sample Type	Mean Difference	Std. Error	Sig.
Bharangi root powder	Diclofenac	-16.72	3.58	<0.001
Bharangi root powder	Control	43.88	2.58	<0.001
Diclofenac	Control	60.60	2.58	<0.001

Results for IL-6 Assay

Table 13: Descriptive statistic for IL-6 Inhibitory activity

Sample Type	N (Number of Cases)	Mean Inhibition (%)	Std. Deviation
Bharangi root powder	3	68.32	1.095
Diclofenac	3	83.78	2.0087
Control	3	0	0

Table 14: ANOVA Table for IL-6

Source	Sum of Squares	df	Mean Square	F	p-value
Between groups	11925.72	2	5962.86	1139.01	<0.001
Within groups	31.41	6	5.24		
Total	11957.13	8			

Table 15: Post Hoc Tukey Test for IL-6

Sample Type	Sample Type	Mean Difference	Std. Error	Sig.
Bharangi root powder	Diclofenac	-15.46	1.5	<0.001
Bharangi root powder	Control	68.32	1.5	<0.001
Diclofenac	Control	83.78	1.5	<0.001

Results for IL-1 Beta Assay

Table 16: Descriptive statistic for IL-1 Beta Inhibitory activity

Sample Type	N (Number of Cases)	Mean Inhibition (%)	Std. Deviation
Bharangi root powder	3	52.96	3.012
Diclofenac	3	62.91	7.982
Control	3	0	0

Table 17: ANOVA Table for IL-1 Beta

Source	Sum of Squares	df	Mean Square	F	p-value
Between groups	6862.01	2	3431.00	47.14	<0.001
Within groups	436.72	6	72.79		
Total	7298.73	8			

Table 18: Post Hoc Tukey Test for IL-1 Beta

Sample Type	Sample Type	Mean Difference	Std. Error	Sig.
Bharangi root powder	Diclofenac	-9.95	4.93	0.18
Bharangi root powder	Control	52.96	4.93	<0.001
Diclofenac	Control	62.91	4.93	<0.001

DISCUSSION

The present study evaluated the anti-inflammatory potential of *Rotheca serrata (Bharangi)* root powder using an in-vitro model assessing key pro-inflammatory cytokines: TNF-α, IL-6, and IL-1β. The findings demonstrated significant cytokine inhibition compared to the control group, confirming the anti-inflammatory activity of *Bharangi* root extract. One-way ANOVA revealed highly significant differences among groups for TNF-α ($F=134.43$, $p<0.001$), IL-6 ($F=1139.01$, $p<0.001$), and IL-1β ($F=47.14$, $p<0.001$), indicating that the treatments produced statistically meaningful effects.

Post hoc Tukey's HSD analysis provided further clarification of intergroup differences. For TNF-α and IL-6, all pairwise comparisons were statistically significant ($p<0.001$). Diclofenac exhibited significantly higher inhibition than *Bharangi* root powder, and both treatments demonstrated significantly greater inhibition compared to the control group. These results are consistent with the established pharmacological action of Diclofenac as a potent non-steroidal anti-inflammatory drug (NSAID), known to suppress inflammatory mediators through cyclooxygenase (COX) inhibition and modulation of downstream cytokine production.

In contrast, the IL-1β results revealed a different pattern. Although both *Bharangi* and diclofenac showed significant inhibition compared to the control ($p<0.001$), the difference between *Bharangi* and diclofenac was not statistically significant ($p=0.18$). This finding suggests that *Bharangi* root powder exhibited comparable inhibitory activity to diclofenac against IL-1β. Given the central role of IL-1β in initiating and amplifying inflammatory responses, this comparable inhibition is particularly noteworthy and indicates meaningful immunomodulatory potential.

The observed cytokine inhibition may be attributed to the phytochemical constituents present in *Bharangi* root, including flavonoids, alkaloids, tannins, and phenolic compounds. These bioactive compounds are widely reported to exert anti-inflammatory effects through antioxidant activity, inhibition of NF-κB activation, and suppression of pro-inflammatory cytokine gene expression. Flavonoids, in particular, are known to downregulate TNF-α and IL-6 production and interfere with inflammatory signalling pathways, which may explain the substantial inhibition observed in this study.

Although diclofenac demonstrated overall higher inhibition percentages for TNF-α and IL-6, *Bharangi* exhibited considerable cytokine suppression, especially in IL-1β inhibition where its activity was statistically comparable to Diclofenac. This suggests that *Bharangi* may exert selective or balanced cytokine-modulating effects rather than broad-spectrum enzyme inhibition. Such a profile could be advantageous in reducing inflammatory responses while potentially minimizing adverse effects associated with synthetic NSAIDs.

Despite these promising findings, several limitations must be acknowledged. The study was confined to in-vitro assays, which do not fully replicate the complexity of inflammatory processes in vivo. Factors such as absorption, metabolism, pharmacokinetics, tissue distribution, and systemic immune interactions were not evaluated. Additionally, while major phytochemical groups were identified, the specific active compounds responsible for the observed activity were not isolated or quantified.

Overall, the results provide strong preliminary evidence supporting the anti-inflammatory potential of *Rotheca serrata* root powder. The statistically significant inhibition of TNF-α, IL-6, and IL-1β, particularly the comparable IL-1β suppression relative to Diclofenac, highlights its potential as a natural cytokine-modulating agent. Further in-vivo investigations, molecular pathway studies, and clinical trials are required to establish its therapeutic efficacy, safety profile, and dosage standardization for clinical application.

CONCLUSION

Rotheca serrata (L.) Steane & Mabb. (syn. *Clerodendrum serratum*) root powder exhibited significant in-vitro anti-inflammatory activity through effective suppression of the pro-inflammatory cytokines TNF-α, IL-6, and IL-1β. Statistical analysis demonstrated highly significant differences among treatment groups ($p<0.001$), confirming the reliability of the observed effects. While diclofenac showed comparatively greater inhibition of TNF-α and IL-6, the inhibitory activity of *R. serrata* against IL-1β was comparable, highlighting its notable cytokine-modulating potential, particularly at the level of early inflammatory signalling.

From an Ayurvedic standpoint, *Bharangi* is traditionally indicated in conditions characterized by *Shotha* (inflammation) and disorders involving aggravated *Kapha* and *Vata*. The demonstrated cytokine suppression provides preliminary scientific support for these classical indications, suggesting a meaningful correlation between traditional therapeutic use and measurable immunomodulatory activity.

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