# ANTI-INFLAMMATORY AND ANALGESIC ACTIVITY OF METHANOLIC EXTRACT OF JASMINUM MULTIFLORUM LEAVES

#### Abstract

Jasminum multiflorum, belonging to the Oleaceae family, is a significant medicinal plant used to treat a variety of illnesses. In the current investigation, an effort was made to examine the antiinflammatory and analgesic results of methanolic extract of the Jasminum multiflorum in various animal models. Acute toxicity studies revealed that the extract was safe in doses up to 2000 mg/kg.For this study, two doses were selected:200 mg/kg and 400 mg/kg bodyweight. Utilizing carrageenan-induced paw edema in albino rats and other animal models, anti-inflammatory effectiveness was assessed, as was analgesic effect with the hot plate technique and tail clip method at doses of 200 and 400 mg/kg bd.wt. in The healthy albino rats. protein denaturation approach was used to achieve in vitro anti-inflammatory effects. MEJM at two doses such as 200 mg/kg bd.wt. and 400 mg/kg bd.wt, produced significant (p <0.01) anti-inflammatory activity in the animal models, which was contrast with the standard drug indomethacin. The present findings exhibited concentration а dependent inhibition of protein (albumin) denaturation by the extract. The effect was plausibly due to the flavonoid's contents in the extract. Methanolic extract of Jasminum multiflorum significantly increased the latency of paw licking in hot plate method and increased the latency of tail licking in tail flick method. The results suggest that methanolic extract of Jasminum multiflorum leaves possessed potent anti-inflammatory and analgesic properties, presumably as a result of chemical constituents like sterols.

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triterpenoids, and flavonoids. The methanolic extract of *Jasminum multiflorum* exhibits anti-inflammatory and analgesic properties, as demonstrated by the aforementioned findings.

**Keywords:** *Jasminum multiflorum*, antiinflammatory, analgesic, Oleaceae.

#### I. INTRODUCTION

A primary host defense mechanism in the presence of injury, tissue ischemia, autoimmune reactions, or pathogenic pathogens is inflammation. Localized inflammation appears as the traditional symptoms of swelling, redness, heat, and frequently pain in tissues outside the brain. Inflammation has now been defined in more mechanistic terms, such as the invasion of circulating immune cells (lymphocytes and macrophages) and the induction or activation of inflammatory mediators like proteins, peptides, glycoproteins, arachidonic acid metabolites (prostaglandins and leukotrienes), nitric oxide, oxygen-free radicals, kinins, cyclooxygenase products, and cytokines. Numerous of these molecules have been shown to be involved in tissue inflammation, are produced locally, and are thus important candidates for therapeutic intervention in a wide variety of illnesses [1].

According to the definition of pain given by [2], it is "an unpleasant sensory and emotional experience related to actual or potential tissue damage or described in terms of such damage. Pain serves as a warning signal that prompts an organism to retreat from dangerous stimuli, thereby preventing damage. Usually, it has to do with an accident or the possibility of one. Acute pain, which usually occurs in response to tissue injury, results from the activation of peripheral pain receptors and their specific A delta and C sensory nerve fibers (nocicept+++ors). Persistent activation of these fibers is likely the cause of chronic pain associated with ongoing tissue injury. However, the severity of a tissue injury does not always predict the severity of chronic or acute pain. Neuropathic pain, which is a kind of chronic pain, can also be brought on by persistent injury to or dysfunction of the central or peripheral nervous systems. Visceral or somatic pain can result from tissue damage is known as nociceptive pain. Somatic pain receptors are located in the skin, subcutaneous tissues, fascia, other connective tissues, periosteum, endosteum, and joint capsules.When these receptors are stimulated, the typical response is either dull or sharp localized pain; however, burning is frequently experienced if the skin or subcutaneous tissues are affected.. [3] [4] [5].

The evergreen ornamental shrub *Jasminum multiflorum* is also referred to as winter jasmine, Indian jasmine, downy jasmine, and/or star jasmine. In India, the flower is used as an emetic, and a poultice composed of dried leaves soaked in water is applied to indolent sores to encourage healing. The plant is used to cure fever, diarrhea, stomach aches, stomach ulcers, and kidney stones. It is also believed to have an astringent effect on the intestines. *Jasminum multiflorum* is a large hirsute shrub with velvety pubescence. It is also known as star jasmine, or kunda in Sanskrit, kundaphul in Bengali, chameli in Hindi, and kunda in English. The plant is regional to India and can be found in China, Malaysia, Taiwan, Europe, and Africa. The present research is aimed at studying the analgesic and anti-inflammatory activities of the plant.

#### **II. MATERIALS AND METHODS**

1. Collection of Plant Materials: Leaves of *Jasminum multiflorum* were collected from Jangaon, Telangana state, in the month of December and and identified and authenticated by the botanist G. Vijaya Basker Reddy, assistant professor, ABV government degree college, Jangaon. The leaves were washed, allowed to dry in the shade for about six days, and then ground into a coarse powder in a mixer grinder. The powdered material was stored and taken up for the extraction process.

- 2. Plant Extraction Process: The Soxhlet extraction equipment was used to prepare the plant extract in a variety of solvents. 500 g of powdered leaves were extracted using the Soxhlet method in 2,000 mL of methanol over the appropriate time at various temperatures. A continuous hot percolation process is used in the Soxhlet equipment to obtain the crude plant extract. This technique allows for continuous extraction, which is simply a series of quick macerations to achieve efficient extraction. The resultant organic extracts were kept at room temperature and then dried by evaporation [7].
- **3. Preliminary Qualitative Phytochemical Screening:** To create a stock solution, the *J.multiflorum* leaf methanol crude plant extract was dissolved in a 1:10 ml solvent. The resulting extracts were then put through a preliminary, qualitative phytochemical screening [8].
- **4. Animals:** Swiss albino mice (weighing 20 to 25 g) and albino rats (weighing 200 to 250 g) were purchased from Albino research in Hyderabad. The Gokaraju Rangaraju College of Pharmacy's CPCSEA-approved animal facility in Bachupally, Hyderabad, India, served as the site for the current inquiry. 1175/PO/ERe/S/08/CPCSEA, Reg. No. With a cycle of 12 hours of light and 12 hours of darkness, a maximum of six animals per cage were used to house the animals in polyacrylic cages. Rats have limitless access to a conventional diet and unlimited water. Before the trial began, the mice were given a week to get used to the laboratory setting.
- **5. Drugs and Chemicals:** Sigma Chemical Co. (St. Louis, Missouri, USA) provided the carrageenan; Novartis India Ltd., Bombay provided the diclofenac injection (Voveran); and Ranbaxy (Rankem) provided the formalin. The study made use of a Vernier calibration instrument was supplied by Percision India Ltd. for the study, whilst Ashirwad Industries (Punjab), Ropar provided a regular chow diet.
- 6. Acute Toxicological Evaluation: To examine the hazardous effects of the methanolic leaf extract of *Jasminum multiflorum*, an acute toxicity research was conducted. Guidelines set forth by the Organization for Economic Cooperation and Development (OECD) were followed in the study's execution. According to OECD Guideline 425, the up-and-down approach is used to evaluate acute oral toxicity. Up-and-down acute toxicity testing (OECD guideline 425) was performed in accordance with this guide.

# 7. In Vitro Method Anti-inflammatory activity

• **Protein Denaturation:** The standard medication is combined with 1 ml of different extract concentrations varying from 50 to 100 g/ml, in the reaction mixture, along with 3 ml of phosphate buffer saline (pH 6.4) and 1 ml of egg albumin solution (1%). The control reaction mixture is used, and it is incubated at 37°C for 20 minutes without any plant extracts. By holding the mixture in a water bath for two minutes at 90°C, denaturation is induced. After cooling, a spectrophotometer is used to determine the turbidity at the wavelength of 660 nm. Using the formula below, denaturation rate inhibition can be measured [9].

Inhibition% = 
$$\frac{(At - Ac)}{Ac} \times 100$$

• *In Vivo* Anti-inflammatory Activity: To elicit paw edema, each rat received an injection into the sub-plantar tissues of its left hind paw containing 0.1 ml of 1% w/v carrageenan suspended in 1% CMC. One of the four groups of rats was made up of six rats in each group.

Group 1: Carrageenan control Group 2: Aqueous extract (200 mg/kg) Group 3: Aqueous extract (400 mg/kg) Group 4: Diclofenac sodium as a standard reference (20 mg/kg)

A vernier caliper was used to measure the thickness of the paws prior to the carrageenan injection, then again after 60, 120, 180, and 240 minutes [10]. The test extract's anti-inflammatory activity was calculated by comparing the proportion of oedema that was inhibited in the animals given it compared to the control group using carrageenan.

To calculate the percentage inhibition of edema, apply the formula.

% inhibition =  $\frac{To - Tt}{To} \times 100$ 

Where  $_{To}$  represents the paw width of rats in the untreated group and  $_{T t}$  represents the paw width of rats that received the sample extract at the same time.

# 8. In Vivo Analgesic Method

• Hot Plate Method: Using the hot plate approach, the extract's analgesic efficacy was also evaluated. The heated plate within the restrainer was kept at 55°C while the rats were there. The amount of time it took for the rats to lick their paws or leap in response to the thermal discomfort was used to calculate the reaction time (measured in seconds), also known as the latency period. The reaction time was measured prior to 0 minutes, 15 minutes, 30 minutes, 45 minutes, and 60 minutes following the introduction of the drugs. To avoid any harm to the paw tissues, the maximum reaction time was set at 45 seconds. Maximum analgesia would be deemed to have occurred if the reading was longer than 45 seconds [11]. The maximum possible analgesia (MPA) was calculated as follows:

$$MPA = \frac{\text{Reaction time for treatment } -\text{Reaction time for saline}}{45 \text{ sec} - \text{Reaction tome for saline } \times 100}$$

• **Tail Flick method:** The tail-flick method was used to assess the extract's antinociceptive (analgesic) activity. Each rat was submerged in warm water that was kept at a constant 50°C for around 5 cm from the tail's distal end. The amount of time it took the rat to flick its tail in response to pain was the reaction time (in seconds). Response time was calculated as the mean of the following two readings after the first reading was deleted. The reaction time was measured prior (0 minutes), 15 minutes, 30 minutes, 45 minutes, and 60 minutes after the administration of the drugs. For the purpose of protecting the tail tissue, the maximum reaction time was set at 15

seconds. Maximum analgesia would be deemed to have occurred if the reading lasted more than 15 seconds [11]. The maximum possible analgesia (MPA) was calculated as follows:

 $MPA = \frac{Reaction \text{ time for treatment `-Reaction time for saline}}{15 \text{ sec} - \text{reaction time for saline } \times 100}$ 

#### **III.RESULTS**

In the current study, relevant rodent models were used to examine the *in vivo* antiinflammatory and analgesic effects of a methanolic extract of *Jasminum multiflorum* leaf extract. The following lists all of the study's findings.

**1. Preparation of Methanolic Extract of the Leaves of** *Jasminum multiflorum***:** By using the soxhlation method, a methanolic extract of Jasminum multiflorum leaves was created. The percentage yield of methanolic extract was calculated by using following formula:

Percentage of yield obtained = 
$$\frac{\text{Amount of yield obtained}}{\text{Total amount of powder used}} \times 100$$

Percentage yield of extract =  $84/500 \times 100 = 16.8\%$  w/w

2. Preliminary Phytochemical Analysis: The presence and absence of carbohydrates, alkaloids, flavonoids, terpenoids, saponins, steroids, and tannins were shown by methanol extracts of *J. multiflorum* leaves. The result obtained in Table 1.

Phytochemical constituents	Results
Carbohydrates	++
Alkaloids	+
Flavonoids	+
Terpenoids	+++
Tannins	+
Saponins	++
Steroids	++

# Table 1: Preliminary Phytochemical screening

Note: + indicates presence

**3.** Acute Toxicity Studies: *Jasminum multiflorum* leaf methanolic extract was administered to Swiss albino mice at a dose of 2000 mg/kg body weight. The animal displayed no signs of toxicity/death up to 2000 mg/kg bd.wt. A range of morphological and behavioral traits were observed throughout the inquiry. Additionally, the consumption of food and water was monitored. All the animals were found to be secure, even after 14 days of observation. The extract was therefore determined to be safe up to 2000 mg/kg bd. wt.

**4. Dose Selection:** From the above toxicity studies, 2000 mg/kg bd.wt. was identified as safe, and the working dose was considered as 1/10th i.e., 200 mg/kg bd.wt. In the present, the two doses chosen for the trial are 200 mg/kg bd.wt. and 400 mg/kg bd.wt.

# 5. Anti-inflammatory Activity

**6.** *In vitro* **anti-inflammatory activity:** A Protein denaturation approach has been performed for its *in vitro* anti-inflammatory property. Results were expressed in Table 2.

S.NO	COMPOUNDS	CONCENTRATION	%	IC <sub>50</sub>
			INHIBITION	VALUE
1.	MEJM	50	22.23±1.06	425
		100	42.22±1.04	
		200	43.84±0.95	
		400	$48.24 \pm 2.06$	
2.	Indomethacin	50	4.58±1.09	405
		100	8.79±1.02	
		200	23.97±1.07	
		400	$48.82 \pm 1.59$	

Table 2: Protein Denaturation of Methanolic Leaf Extract of Jasminum multiflorum

In the protein denaturation assay, the MEJML was tested at different concentrations of 50, 100, 200, and 400  $\mu$ g/mL. The lowest concentration of 50  $\mu$ g/mL showed a percentage inhibition of 22.23, whereas the highest concentration of 400  $\mu$ g/mL showed a percentage inhibition of 48.24. The IC<sub>50</sub> value for the MEJM was found to be 425  $\mu$ g/mL, which is compared with standard ascorbic acid, which has an IC<sub>50</sub> value of 405  $\mu$ g/mL.

7. *In vivo* anti-inflammatory activity: The paw edema brought on by carrageenan was utilized to test the methanolic extract of *Jasminum multiflorum's* anti-inflammatory properties. The results obtained in this model are given below.

# 8. Carrageenan-induced paw edema in rats

Compound	Change in	%			
	1h	2h	3h	4h	inhibition
					at 3h
Normal control	1.09 ±	$1.08 \pm 0.02$	$1.105 \pm 0.01$	$1.08 \pm 0.02$	-
	0.01				
Disease control	0.21 ±	$0.21 \pm 0.02$	$0.19\pm0.04$	$0.22\pm0.02$	-
	0.03	0			
MEJM	0.19 ±	0.71 ±	0.83 ±	$0.93 \pm 0.03^{**A}$	31.8
(200mg/kg bd.	0.03	$0.04^{**A}$	$0.02^{**A}$		
wt.)					

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MEJM (400mg/kg bd. wt.)	0.23 ± 0.04	$\begin{array}{c} 0.50 & \pm \\ 0.03^{**A} & \end{array}$	0.03 <sup>**Aa</sup>	$0.48 \pm 0.03^{**Aa}$	39.6
Indomethacin (10mg/kg bd. wt.)	$\begin{array}{ccc} 0.21 & \pm \\ 0.02 & \end{array}$	$\begin{array}{c} 0.45 & \pm \\ 0.02^{**Ba} \end{array}$	$\begin{array}{c} 0.49 & \pm \\ 0.03^{**Ba} \end{array}$	$0.43 \pm 0.02^{**Ba}$	46.5

The mean SEM of the values was used (n = 6). ANOVA was used in the statistical analysis, and Dunnett's test was used to compare the results to the standard, the negative control, and the control. The terms "monitor" group" (\*\*p0.01), "disease group" (a=p 0.01, b=p 0.05), and "standard" (A= p 0.01, B = p 0.05) are used to represent significant results, whereas "ns" stands for "non-significant."

The Anti-inflammatory activity is expressed as Percentage Inhibition. The Percentage Inhibition in the Paw edema model produced by carrageenan had been tabulated in Table 3. Percentage inhibition was found to be 0%, 31.8%, 39.6%, and 46.5% in the disease control group, the MEJM (200 mg/kg bd. wt.) treated group, the MEJM (400 mg/kg bd. wt.) treated group, and indomethacin (10 mg/kg bd. wt.) treated group.

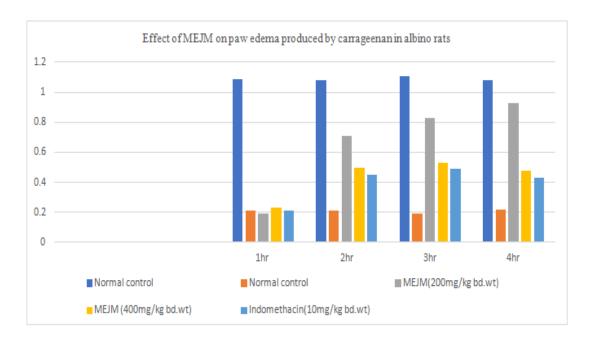


Figure 1: MEJM's impact on Paw edema produced by Carrageenan in Albino Rats

**9. Analgesic activity:** The hot plate approach was used to examine the methanolic extract of *Jasminum multiflorum* for its impact on analgesic activity. The results obtained in this model are given below in Table 4.

### **10. Hot Plate Method**

Compound	Reaction time in seconds (Mean SEM) 3 h					
	0 minutes	15 minutes	30 minutes	45 minutes	60 minutes	
Control group	32.67 ±	$28.25\pm0.87$	31.50 ±	28.08 ±	24.83 ±	
	0.15		0.57	0.37	0.02	
MEJM	32.75 ±	$\begin{array}{rrr} 38.54 & \pm \\ 0.55^{**} & \end{array}$	41.58 ±	$41.99 \pm 0^{**}$	41.46± 0.55 <sup>**</sup>	
(200 mg/kg bd.	$0.04^{*}$	$0.55^{**}$	$0.22^{**}$		$0.55^{**}$	
wt.)						
MEJM	33.04 ±	27.92 ±	31.42 ±	34.38 ±	31.73±	
(400 mg/kg bd.	$0.09^{*}$	$0.04^{**}$	0.61**	$0.08^{*}$	$0.46^{**}$	
wt.)						
Indomethacin	31.78 ±	26.67 ±	24.50 ±	27.17 ±	27.71 ±	
(10 mg/kg bd.	$0.08^{*}$	$0.17^{**}$	$0.08^{**}$	$0.92^{*}$	$0.06^{*}$	
wt.)						

#### Table 4: Effect of MEJM in analgesic activity by Hot plate method

The mean SEM of the values was used (n = 6) ANOVA was used in the statistical analysis, and Dunnett's test was used to compare the results to the standard, the negative control, and the control. Control group (\*p0.01, \*p0.05) and standard (A=p 0.01, B = p 0.05) are used to indicate significant data, whereas ns stands for non-significant.

**11. Tail Clip Method:** The tail clip method was used to assess the methanolic extract of *Jasminum multiflorum* for its impact on analgesic activity. The results obtained in this model is given below in table 5.

Compound	Reaction time in seconds (mean SEM) 3 h				
	0 minutes	15 minutes	30 minutes	45 minutes	60 minutes
Control	4.25 ± 0.57	$4.50 \pm 0.34$	$4.42 \pm 0.45$	$4.58 \pm 0.44$	$5.17 \pm 0.80$
MEJML	4.13±0.54**,ª	5.29±0.57*, <sup>b</sup>	6.75±0.62*a	6.85±0.56*,	6.98±1.24*,
(200 mg/kg				a	a
bd. wt.)					
MEJML	6.67±0.85*, <sup>b</sup>	7.04±0.67**,ª	9.04±0.73**,	9.5±0.92**,	9.67±0.86**,
(400 mg/kg			b	b	b
bd. wt.)					
Indomethac	6.40 ±0.32*	10.04 ±0.73*	$10.82 \pm 0.84*$	11.83±	12.33 ±
in				0.35*	0.83*
(10 mg/kg					
bd. wt.)					

The mean SEM of the values was used (n = 6) ANOVA was used in the statistical analysis, and Dunnett's test was used to compare the results to the standard, the negative control, and the control. Control group (\*p0.01, \*p0.05) and standard (A=p 0.01, B = p 0.05) are used to indicate significant data, whereas ns stands for non-significant.

#### **IV. DISCUSSION**

*Jasminum multiflorum* leaf methanolic extract underwent phytochemical screening, which revealed the presence of a number of phytoconstituents, including alkaloids, flavonoids, carbohydrates, terpenoids, tannins, and saponins. These ingredients are recognized to have anti-inflammatory and analgesic action, according to early investigations.

1. In vitro anti-inflammatory activity: An external stressor or chemical, such as Strong acids or bases, heat, an organic solvent, concentrated inorganic salt, can denaturize proteins. Once denatured, the majority of biological proteins cease to function biologically. Inflammation has been linked to protein denaturation repeatedly [12]. The release of cellular components into the intracellular space as a result of ROS interaction with membrane lipids leads to cellular membrane instability, which in turn leads to inflammation. Proteins frequently undergo denaturation and renaturation; during denaturation, functional qualities may be lost; however, if denaturing agents are removed, the protein renatures and regains its full functional structure. Permanent protein denaturation occurs when the removal of the denaturing agents does not always restore the protein's original structure. In these circumstances, the denatured proteins eventually precipitate and are cleared from the targeted region by macrophages. Another reason for inflammation at this location is the excessive deposition of denatured protein and the accumulation of macrophages [13]. Phenolic chemicals have the ability to mop up free radicals by either deactivating lipid free radicals or by stopping hydroperoxides from breaking down into free radicals. By chelating metal ions and quenching or protecting antioxidant defenses, flavonoids with hydroxyl groups mediate their antioxidant actions. Alkaloids have also been demonstrated to have antioxidant effects by reducing oxidative damage brought on by H2O2. By obstructing the cyclooxygenase, and lipoxygenase metabolic pathways of arachidonic acid metabolism, alkaloids may reduce inflammation. Tannins are strong antioxidants that work by delaying oxidation by chelating metal ions such as Fe (II) and obstructing a step in the Fenton process. Through the suppression of the cyclooxygenase enzyme, tannins also prevent lipid peroxidation [14].

NSAIDs work by inhibiting the formation of endogenous prostaglandins by inhibiting the cyclooxygenase enzyme, but they also prevent protein denaturation. The inflammatory response to carrageenan injection has been described as a biphasic, age-dependent event in which a number of mediators act sequentially to cause it. Inflammation is mediated by a number of different mediators. In the early stages of carrageenan-induced inflammation, histamine, serotonin, and bradykinin are the first mediators that can be identified; prostaglandins (PGs), which are involved in enhanced vascular permeability, are only visible in the late stages of inflammation. TNF-, IL-1, and IL-6 levels are increased in conditions where there is local or systemic inflammation. Nitric oxide (NO), which is created in pathological situations by three different isoforms of nitric oxide synthase (NOS), including endothelial NOS (eNOS), neuronal NOS (nNOS), and inducible NOS (iNOS), is another crucial mediator in acute inflammation.

- 2. In vivo anti-inflammatory activity: Inflammation is an organism's protective attempt to get rid of harmful stimuli and start the healing process. NSAIDs and other anti-inflammatory medications work by blocking the enzymes cyclooxygenase-1 and cyclooxygenase-2, which prevent the production of prostaglandins from arachidonic acid. A frequently used initial test for the evaluation of novel anti-inflammatory drugs is carrageenan-induced paw edema, which is thought to be biphasic. Histamine or serotonin release causes the first phase of edema (1–2 hours), whereas prostaglandin release causes the second phase [15]. According to the findings of the current investigation, MEJM significantly (p<0.01) decreased Paw edema in Rats caused by carrageenan as compared to the disease control group (at dosages of 200 mg/kg, bd. wt., and 400 mg/kg, bd. wt.). It has been demonstrated that steroids, triterpenoids, and flavonoids have anti-inflammatory properties in earlier studies. These chemical components may be the cause of the anti-inflammatory effect because they are also found in MEJM.
- **3. Analgesic Activity:** The tail flick and hot plate techniques are applied in animal pain models based on supraspinal locations that initiate polysynaptic reflexes that are regulated at the spinal level. Training and acclimation improve these reflexes' cortical and brainstem control. These reactions take place in response to the introduction of heat, cold, mechanical, and electrical stimuli. Hot plate and tail clip types use thermal heat and radiant heat, respectively. Jumping and licking of the paws are signs of pain reflex behavior when using a hot plate, but pulling the tail back or biting the clip are signs of pain reflex behavior when using a tail clip. Skin, muscle, joint, viscera, and other injured tissue can dictate the form and behavior of pain. Hot plate method adds supraspinal modulation, while the tail clip model of acute pain uses spinal and bulbospinal pathways [16]. Although the models have low face and construct validity, they can be used to predict the effectiveness of analgesics that are nonopioid (at higher doses) and opioid in humans.
- **4. Hot Plate Method:** In this investigation, Two different dosages of MEJM (200 and 400 mg/kg) were given to the models, and both considerably raised the threshold of pain at all time intervals. Compared to the usual saline (vehicle) group, the hot plate and tail clip caused more severe discomfort. The maximal effects of MEJM (200 mg/kg and 400 mg/kg, respectively) were seen in the hot plate model of acute pain within 30 minutes, as opposed to the maximum effects of normal diclofenac (10 mg/kg), which were seen after 60 minutes. The greatest effects of the two drugs did not differ statistically significantly at 30 or 60 minutes, though.
- **5.** Tail Clip Method: Both of the drug groups—MJEM 200 and MJEM 400—exhibited a noticeably larger pain threshold than the control (vehicle) group at all time intervals in a dose-dependent manner in the tail clip model of acute pain. While MEJM 200 began to lose efficacy after 30 minutes, the mean tail clip latency increased for diclofenac and MEJM 400 up until 90 minutes. At the specified intervals of 0, 30, and 60 minutes, there was no statistically significant change in the mean tail clip latency between the three medication groups. Therefore, during the 90-minute testing period, it was shown that the three medication dosages used in the tail clip procedure had comparable analgesic efficacy. Comparing the outcomes of the two analgesiometric assays that we employed in our research—the hot plate and the tail clip—made it evident that the drug groups showed various time courses for drug effects based on the analgesiometer type. While diclofenac

and MEJM 400 showed different peak responses in the two trials, MEJM (200 mg/kg) showed its highest response at the same time (30 minutes into the testing). Previous research has suggested that the peripheral analgesia brought on by COX inhibition may not be the only neuronal mechanisms at play in the anti-nociceptive action of NSAIDs [17,18]. It is also known that the hot plate method considerably modulates supraspinal pain perception more than the tail clip method.

## V. CONCLUSION

The following results were reached after Jasminum multiflorum's potential for antiinflammatory and analgesic action was assessed in the current study. Jasminum multiflorum's methanolic leaf extract underwent a preliminary phytochemical analysis, which identified flavonoids, steroids, saponins, alkaloids, and tannins as the most abundant compounds. Sesquiterpene hydrocarbons, nerolidol, and lupeol are examples of essential oils. The extract has demonstrated a decrease in protein denaturation, which may be related to the tannins, alkaloids, and flavonoids that are present. Carrageenan-induced paw edema in rats was utilized to investigate the anti-inflammatory properties. In the animal model, MEJM at 200 mg/kg bd. wt. and 400 mg/kg bd. wt. considerably decreased the edema in paw. Since MEJM also contains various chemical components, such as flavonoids, triterpenoids, and steroids, they may be to blame for the anti-inflammatory effects. Two models, such as hot plate approach another one is the tail clip approach, had been used to perform analgesic activity. Previous research has suggested that the peripheral analgesia brought on by COX inhibition may not be the only neuronal mechanisms at play in the anti-nociceptive action of NSAIDs. The current study consequently validates the claims of the plant's historic use as an analgesic, anti-inflammatory, and wound-healing agent. Additional research using purified fractions of the bioactive molecule is required to determine the precise mechanism of action of MEJM.

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