Anti-inflammatory activity and Analgesic of methanolic extract of *Jasminum multiflorum* leaves

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**Abstract**

*Jasminum multiflorum* belonging to the family Oleaceae is an important medicinal plant which is used for various ailments. In the present study, an attempt was made to study the anti-inflammatory and analgesic effects of the methanolic extract of *Jasminum multiflorum* in different animal models. Acute toxicity studies were performed, and extract was found to be safe upto 2000 mg/kg. Two doses were selected for this study i.e., 200 mg/kg and 400 mg/kg, b. w. Anti-inflammatory activity was evaluated by using carrageenan induced paw edema in albino rats and analgesic activity by hot plate method and tail clip method at doses of 200 and 400 mg/kg, b.w. in healthy albino rats. In vitro anti-inflammatory activity was performed using protein denaturation technique. MEJM at the dose of 200 mg/kg b.w and 400 mg/kg b.w produced significant(p<0.01) anti-inflammatory activity in the animal models, which was comparable with the standard drug indomethacin. The present findings exhibited a concentration dependent inhibition of protein (albumin) denaturation by the extract. The effect was plausibly due to the flavonoid’s contents of 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 activity which may be due to the presence of the chemical constituents such as flavonoids, triterpenoids, and sterols. From the above results it is clear that methanolic extract of *Jasminum multiflorum* possess anti-inflammatory, analgesic activity.

**Keywords**: *Jasminum multiflorum*, anti-inflammatory, 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 is a warning signal that influences an organism to withdraw from harmful stimuli, protecting it from harm. It is typically related to injury or the threat of injury. Acute pain, which usually occurs in response to tissue injury, results from activation of peripheral pain receptors and their specific A delta and C sensory nerve fibers (nociceptors). Chronic pain related to ongoing tissue injury is presumably caused by persistent activation of these fibers. However, the severity of tissue injury does not always predict the severity of chronic or acute pain. Chronic pain may also result from ongoing damage to or dysfunction of the peripheral or central nervous system (which causes neuropathic pain). Nociceptive pain (pain caused by tissue injury) may be somatic or visceral. Somatic pain receptors are located in skin, subcutaneous tissues, fascia, other connective tissues, periosteum, endosteum, and joint capsules. Stimulation of these receptors usually produces sharp or dull localized pain, but burning is not uncommon if the skin or subcutaneous tissues are involved.[3] [4] [5].

*Jasminum multiflorum* is an evergreen, cultivated, ornamental shrub known as winter jasmine, Indian jasmine, downy jasmine and/ or star jasmine. In India, a poultice made from dried leaves soaked in water is placed on indolent ulcers to promote healing and the flower is used as an emetic. The plant is known to have an astringent effect on the the bowels; and is used to treat fever, dysentery, stomach-ache, stomach ulcers, and kidney stones. *Jasminum multiflorum* (Sanskrit: kunda, Bengali: kundaphul, Hindi: chameli, English: star jasmine) is a large scandent, tomentose shrub with velvety pubescence. The plant is native to India and distributed throughout China, Malaysia, Taiwan, Europe and Africa. The present research is aimed to study the analgesic and anti-inflammatory activity of the plant.

**Ⅱ. MATERIALS AND METHODS**

**A. Collection of plant materials:**

Leaves of *Jasminum multiflorum* were collected from Jangaon, Telangana state in the month of December and was identified and authenticated by the botanist G. Vijaya Basker Reddy assistant professor, ABV government degree college, Jangaon. The leaves were cleaned and dried under shade for about six days and coarsely powdered in a mixer grinder. The powdered material was stored and taken up for extraction process.

**B. 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 method enables continuous extraction, which is nothing more than a succession of brief macerations, to achieve efficient extraction. The resulting organic extracts were dried by evaporation while remaining at room temperature [7].

**C. 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].

**D. Animals:**

Swiss albino mice (weighing 20 to 25 gm) and albino rats (weighing 200 to 250 g) were purchased from Albino research in Hyderabad. The current investigation was completed in the Gokaraju Rangaraju College of Pharmacy's CPCSEA-approved animal facility in Bachupally, Hyderabad, India. 1175/PO/ERe/S/08/CPCSEA, Reg. No. With a cycle of 12 hours of light and 12 hours of darkness, the animals were kept in poly acrylic cages with a maximum of six animals per cage. 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.

**E. 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.

**F. Acute toxicological Evaluation:**

To examine the hazardous effects of the methanolic leaf extract of *Jasminum multiflorum*, an acute toxicity research was conducted. Organization for Economic Cooperation and Development (OECD) guidelines were followed in conducting the study. The up-and-down procedure is the method used to assess acute oral toxicity (OECD guideline 425). Acute toxicity tests using the up-and-down method (OECD guideline-425) were conducted in accordance with this guide.

**G. Invitro method Anti- inflammatory activity:**

**1. Protein denaturation:**

The standard medication is combined with 1 ml of various extract concentrations ranging 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 measure the turbidity at 660 nm. Percentage inhibition of denaturation is calculated by using the following formula[9].

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

**H. In *vivo* anti-inflammatory activity:**

To elicit paw edema, 0.1 ml of 1% w/v carrageenan suspended in 1% CMC was injected into the sub-plantar tissues of the left hind paw of each rat. One of the four groups of rats was made up of each group of six rats.

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)

The paw thickness was measured using a vernier caliper before the carrageenan injection and 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 carrageenan control group.

The formula is used to compute the percentage (%) inhibition of edema.

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

Where To represents the paw thickness of rats in the control group at the same time as T t represents the paw thickness of rats administered the test extract at the corresponding period.

**I. In *vivo* analgesic method:**

**1.Hot plate method:**

Using the hot plate method, 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 min), 15 min, 30 min, 45 min, and 60 min 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 = $\frac{Reaction time for treatment -Reaction time for saline}{45\sec(- Reaction tome for saline ×100)}$

**2.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 about 5 cm from the distal end of the tail. The amount of time it took the rat to flick its tail in response to pain was the reaction time (in seconds). Reaction time was calculated as the average of the next two readings after the first reading was deleted. The reaction time was measured prior to (0 min), 15 min, 30 min, 45 min, and 60 min 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 = $\frac{Reaction time for treatmentˇ-Reaction time for saline}{15sec-reaction time for saline ×100}$

**Ⅲ. RESULTS**

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

**A. Preparation of methanolic extract of 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 the following formula.

 **% of yield obtained =** $\frac{Amount of yield obtained }{Total amount of powder used}×100$

Percentage yield of extract = 84/500×100= 16.8% w/w

**B. 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.

 **Table 1: Preliminary phytochemical screening**

|  |  |
| --- | --- |
|  **Phytochemical constituents** | **Results** |
|  Carbohydrates | ++ |
|  Alkaloids | + |
|  Flavonoids | + |
|  Terpenoids | +++ |
|  Tannins | + |
|  Saponins | ++ |
|  Steroids | ++ |

 Note: + indicates presence

**C. Acute toxicity studies:**

*Jasminum multiflorum* leaf methanolic extract was administered to Swiss albino mice at a dose of 2000 mg/kg body weight. Up to 2000 mg/kg bd. wt., the animal showed no symptoms of toxicity or fatality. Throughout the investigation, a variety of physical and behavioral characteristics were seen. Other factors, such as food and water intake, were also tracked. Even after 14 days of observation, it was discovered that all the animals were secure. The extract was therefore determined to be safe up to 2000 mg/kg bd. wt.

**Dose selection:** From the above toxicity studies, 2000 mg/kg bd. wt. was identified to be safe, and the working dose was considered as 1/10th i.e., 200 mg/kg. bd. wt. In the present study two doses selected are 200 mg/kg. bd. wt and 400 mg/kg. bd. wt.

**D. Anti-inflammatory activity**

**E. In *vitro* anti-inflammatory activity**

Protein denaturation method was performed for the *in vitro* anti-inflammatory activity. The results were expressed in table 2.

**Table 2: Protein denaturation of methanolic leaf extract of *Jasminum multiflorum***

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **S.NO** | **COMPOUNDS** | **CONCENTRATION** | **% INHIBITION** | **IC50 VALUE** |
|  **1.** | MEJM |  50  100  200 400 | 22.23±1.06 42.22±1.04 43.84±0.95 48.24±2.06 | 425 |
|  **2.** | Indomethacin | 50 100 200 400 | 4.58±1.09 8.79±1.02 23.97±1.07 48.82±1.59 | 405 |

In protein denaturation assay, the MEJML was tested at different concentrations of 50, 100, 200, 400 μg/mL. The lowest concentration of 50 μg/mL showed a percentage inhibition of 22.23whereas the highest concentration of 400 μg/mL showed a percentage inhibition of 48.24. The IC50 value for the MEJM was found to be 425 μg/mL which is compared with standard ascorbic acid having IC50 value of 405 μg/mL.

**F. 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.

**G. Carrageenan induced paw edema in rats:**

 **Table 3: Effect of MEJM on paw edema induced by carrageenan in albino rats**

|  |  |  |
| --- | --- | --- |
| **Compound** | **Change in paw volume (mL) at different hours** | **% inhibition at 3h** |
| **1h** |  **2h** |  **3h** |  **4h** |
| Normal control | 1.09±0.01 | 1.08±0.02 | 1.105±0.01 | 1.08±0.02 | - |
| Disease control | 0.21±0.03 | 0.21 ± 0.02 0 | 0.19 ± 0.04 | 0.22 ± 0.02 | - |
| MEJM (200mg/kg bd.wt) | 0.19±0.03 | 0.71 ± 0.04\*\*A | 0.83 ± 0.02\*\*A | 0.93 ± 0.03\*\*A | 31.8 |
| MEJM (400mg/kg bd. wt) | 0.23±0.04 | 0.50 ± 0.03\*\*A | 0.53 ± 0.03\*\*Aa | 0.48 ± 0.03\*\*Aa | 39.6 |
| Indomethacin (10mg/kg bd. wt) | 0.21±0.02 | 0.45 ± 0.02\*\*Ba | 0.49 ± 0.03\*\*Ba | 0.43 ± 0.02\*\*Ba | 46.5 |

Values were expressed as mean ± SEM (n=6). Statistical analysis was performed by using ANOVA followed by Dunnett’s test by comparing with control, negative control & standard. Significant values are expressed as control group (\*\*p<0.01), disease control (a=p<0.01, b=p<0.05) & standard (A=p<0.01, B = p<0.05), ns=non-significant.

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

**Figure 1: Effect of MEJM on paw edema induced by carrageenan in albino rats**

**H. Analgesic activity**

The hot plate method was used to examine the methanolic extract of Jasminum multiflorum for its impact on analgesic activity.The results obtained in this model is given below in table 4.

**I. Hot plate method**

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

|  |  |
| --- | --- |
| **Compound** | **Reaction time in seconds (Mean SEM) 3 h** |
| **0 min**  | **15 min**  | **30 min**  | **45 min**  | **60 min** |
| Control group | 32.67 ± 0.15 | 28.25 ± 0.87 | 31.50 ± 0.57 | 28.08 ± 0.37 | 24.83 ± 0.02 |
| MEJM (200 mg/kg bd. wt) | 32.75 ± 0.04\* | 38.54± 0.55\*\* | 41.58± 0.22\*\* | 41.99 ± 0\*\* | 41.46± 0.55\*\* |
| MEJM (400 mg/kg bd. wt) | 33.04 ± 0.09\* | 27.92± 0.04\*\* | 31.42± 0.61\*\* | 34.38 ± 0.08\* | 31.73± 0.46\*\* |
| Indomethacin (10 mg/kg bd. wt) | 31.78 ± 0.08\* | 26.67± 0.17\*\* | 24.50± 0.08\*\* | 27.17 ± 0.92\* | 27.71 ± 0.06\* |

Values were expressed as mean ± SEM (n=6). Statistical analysis was performed by using ANOVA followed by Dunnett’s test by comparing with control, negative control & standard. Significant values are expressed as control group (\*p<0.01, \*p<0.05) & standard (A=p<0.01, B = p<0.05), ns=non-significant.

**J. 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.

**Table 5: Effect of MEJM in analgesic activity by tail clip method**

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

Values were expressed as mean ± SEM (n=6). Statistical analysis was performed by using ANOVA followed by Dunnett’s test by comparing with control, negative control & standard. Significant values are expressed as control group (\*\*p<0.01, \*p<0.05), & standard (A=p<0.01, B = p<0.05), ns=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.

**A*. In vitro* anti-inflammatory activity**

Proteins can get denaturized by the application of an external stressor or chemical, such as a potent acid or base, a concentrated inorganic salt, an organic solvent, or heat. When 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 inactivating 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 via reducing oxidative damage brought on by H2O2. By obstructing the cyclooxygenase and lipoxygenase metabolic pathways of arachidonic acid metabolism, alkaloids may also reduce inflammation. Tannins are strong antioxidants that work by chelating metal ions like Fe (II) and interfering with one of the steps in the Fenton reaction to delay oxidation. 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-weight 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.

**B.** ***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 prevents 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 (p0.01) decreased carrageenan-induced paw edema in rats as compared to the disease control group (at dosages of 200 mg/kg, bd. wt., and 400 mg/kg, bd. wt.). Steroids, triterpenoids, and flavonoids have been shown to 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.

**C. Analgesic activity**

The hot plate and tail flick techniques are used in animal models of pain that are based on polysynaptic reflexes that are triggered at the spinal level and controlled from supraspinal locations. 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. The behavior and type of pain are frequently determined by the damaged tissue, such as the skin, muscle, joint, viscera, etc. The tail clip model of acute pain involves spinal and bulbospinal pathways, but the hot plate method uses additional supraspinal modulation [16]. Despite having low face and construct validity, the models can be used to predict the efficacy of both opioid and nonopioid (at higher dose) analgesics in humans.

**D. Hot plate method**

In this investigation, MEJM was administered in two separate doses of 200 and 400 mg/kg, both of which significantly increased the threshold of pain at all time intervals in both models. Compared to the usual saline (vehicle) group, the hot plate and tail clip caused more severe discomfort. The maximal effects of MEJM (both at 200 mg/kg and 400 mg/kg) 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.

**E. 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. There was no statistically significant difference in mean tail clip latency between the three drug groups at the predefined intervals of 0, 30, and 60 minutes. 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 anti-inflammatory 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. Rats' paw edema caused by carrageenan was used to test the anti-inflammatory effects. The paw edema in the animal model was greatly reduced by MEJM at 200 mg/kg body weight and 400 mg/kg body weight. 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, namely the hot plate approach and the tail clip method, were 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.

**Ⅵ. REFERENCES**

1. Suffredini AF, Fantuzzi G, Badolato R, Oppenheim JJ, O'grady NP. New insights into the biology of the acute phase response. *J. Clin. Immunol.*1999;19:203–214.
2. Kumar KH, Elavarasi P. Definition of pain and classification of pain disorders. J Advan Clin Res Insights. 2016;3(3):87–90. doi: 10.15713/ins.jcri.112
3. Stanton-Hicks M, Jänig W, Hassenbusch S, Haddox J, Boas R, Wilson P. Reflex sympathetic dystrophy: changing concepts and taxonomy. Pain. 1995;63(1):127–133.
4. Tadele A, Asres K, Melaku D, Mekonnen W. *In vivo* anti-inflammatory and antinociceptive activities of the leaf extracts of Clematis simensis Fresen. Ethiop Pharm J. 2009;**27**:33–41.
5. Salzano S. Redox Regulation of Inflammation and Immunity*.* University of Brighton; 2013.
6. Ankur Jyoti Saikia, Vipin Parkash, Prodip Kumar Hazarika, Muhibul Hussain, Kalyan Das, Pallwabee Duarah. Jasminum multiflorum (Burm. f.) Andrews (Oleaceae) - a new host plant record for Saissetia coffeae (Walker) (Coccidae: Hemiptera) from Assam, India. Emer Life Sci Res. 2019;5(1): 18-22.
7. Suvarchala Reddy NVL, Kusuma Priyanka AY and Raghavendra NM. Isolation and Characterization of triterpenoids from bark of *Syzygium alternifolium* (Wight) Walp. Annals of Phytomedicine. 2012; 1(2): 45-51.
8. Swetha T, Suvarchala Reddy NVL, Rajashekar B, Ushasri S. Isolation and identification of compounds from ethyl acetate extract of *Ficus tinctoria* Stem. International Journal of Pharmacy. 2017; 7(2): 55-58.
9. Ganga Raju M, Kumara Swamy K. Anti-inflammatory and antiradical potential of methanolic extract of *Cajanus cajan*. Asian Journal of Pharmacy and Pharmacology. 2018; 4(6):860-864.
10. Ganga Raju M, Srilakshmi S, Suvarchala Reddy NVL. Anti-inflammatory, *In silico* docking and ADME analysis of some selected isolated compounds of *Tagetes erecta* flower heads. IJPSR. 2020, 11(3): 1358-66.
11. Ezeja M, Omeh Y, Ezeigbo I, Ekechukwu A. Evaluation of the analgesic activity of the methanolic stem bark extract of *Dialium guineense* (wild). Ann Med Health Sci Res. 2011 Jan;1(1):55-62. PMID: 23209955; PMCID: PMC3507093.
12. Leelaprakash G, Mohan Dass S. *In vitro* Anti Inflammatory activity of Methanol extract of Enicostemma Axillare. Int. J. Drug Dev. & Res. 2011, 3(3): 189-196.
13. Sakat S, Juvekar AR, Gambhire MN. *In vitro* antioxidant and anti-inflammatory activity of methanol extract of *Oxalis corniculata* Linn. International Journal of Pharma and Pharmacological Sciences 2010; 2(1):146-155.
14. Kibiti CM, Afolayan AJ. Preliminary Phytochemical Screening and Biological Activities of Bulbine abyssinica Used in the Folk Medicine in the Eastern Cape Province, South Africa. Evidence Based Complement Alternative Medicine. 2015;2015:617607. doi: 10.1155/2015/617607.
15. Hirschelmann, R., Bekemeier, H. Effects of catalase, peroxidase, superoxide dismutase and 10 scavengers of oxygen radicals in carrageenin edema and in adjuvant arthritis of rats. Experientia 37, 1313–1314 (1981).
16. Gulecha, V.; Sivakumar, T.; Upaganlawar, A.; Mahajan, M.; Upasani, C. Screening of Ficus Religiosa Leaves Fractions for Analgesic and Anti-Inflammatory Activities. Indian J Pharmacol. 2011, 43, 662.
17. Koster R, Anderson M, De Beer EJ. Acetic acid for analgesic screening**.**Fed Proc. 1959:18**:**418-420.
18. Chapman CR, Casey KL, Dubner R, Foley KM, Gracely RH, Reading AE. Pain measurement: an overview.Pain. 1985;22:1-31.