

## ANALGESIC ACTIVITY OF THE ETHANOLIC EXTRACT OF FLOWER PARTS OF *CELOSIA ARGENTEA* LINN

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Article Received on  
26 Feb. 2017,

Revised on 18 March 2017,  
Accepted on 06 April 2017

DOI: 10.20959/wjpr20175-8332

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### ABSTRACT

*Celosia argentea* has been widely used for its reported biological activity in indigenous system of medicines. The present investigation was carried out to found the analgesic effect of ethanolic extract of flowers of *Celosia argentea* in experimental animal model of pain. The analgesic activity was evaluated by acetic acid induced writhing method and by tail flick latency method in albino mice respectively. The percentage protection against writhing method showed by ethanolic extract of *Celosia argentea* in doses of 200 & 400mg/kg, p.o. was 24.12 & 29.60 respectively. In the tail flick model, the ethanolic extract of *Celosia argentea* in the above doses increased the pain threshold significantly and showed the basal reaction time of 10 & 14.25 sec respectively at third hour. After the administration of the ethanolic extract of *Celosia argentea* in different doses such as 200 & 400mg/kg exhibited dose dependent and significant analgesic activity in both models of pain.

**KEYWORDS:** Analgesic, *Celosia argentea*, acetic acid induced writhing and tail flick methods.

### INTRODUCTION

Pain is a multidimensional experience that is essential for the maintenance and preservation of an individual. It warns of the danger of bodily harm and alerts to trauma and injury. Pain is a specific enteroceptive sensation; it can be perceived as arising from a particular portion of the body, its temporal properties can be detailed, it can be differentiated qualitatively (for

example, as stinging, pricking, burning, throbbing, dull or aching) and it involves dedicated subsets of peripheral and central neurons. The experience of pain has a distinctly unpleasant character, that is, an affective or motivational aspect that can be distinguished from its discriminative sensory aspects and from the long-term emotional experience of 'suffering'. The unpleasantness ranges in intensity from the discomfort of a cold room, fatigued muscles or colonic tension to the excruciating agony of a severe burn, toothache, gallstone or migraine. Under normal circumstances, primary afferent pain fibres activate particular central pathways that engage protective mechanisms at several functional levels: autonomic, homeostatic, motoric, behavioural and mnemonic. However, injury or disease can alter the balance of this system and result in persistent, pathological pain. Analgesic substances, such as aspirin and morphine, that interact with the transmitters and modulators of the pain system are helpful for many people with pain, but there is a great need for the development of better methods for the alleviation and control of both acute (immediate) and chronic (long-term, pathological) pain.

#### **TYPES OF PAIN**

- ❖ Acute pain
- ❖ Chronic pain
- ❖ Survived pain
- ❖ Physiological pain
- ❖ Pathological pain
- ❖ Vascular pain
- ❖ Bone & Joints pain
- ❖ Myalgia<sup>[1]</sup>

#### **MATERIALS AND METHODS**

**Chemicals and drugs:** Diclofenac sodium, Tramadol and acetic acid were used in the study.

**Collection of Plants:** The flowers of *Celosia argentea* were collected from rural area of Nalgonda. They were authenticated by pharmacognosist Dr.KNV. Rao, HOD of department Pharmacognosy at Nalanda college of pharmacy Nalgonda, Telangana State, India.

**Preparation of the extract:** The powdered plant material was extracted with ethanol in a Soxhlet apparatus for 48 hrs. The extracts were filtered through Wattsman filter paper and

concentrated by vacuum evaporation. The yield of extract as per solvent used was 4.25% w/w. The dried extract was used for experiments.

**Phytochemical studies:** *Celosia argentea L. var. cristata (L.)* showed the presence of flavonoids, mucilages, phenolic compounds & tannins, saponins, triterpenoids, alkaloids, carbohydrates, proteins, amino acids, gums and steroids. The flowers were also used as astringent, styptic, depurative, uterine sedative, constipating, antibacterial and corrective of urinary pigments, febrifuge and alexeteric.<sup>[4]</sup>

**Test animals:** Albinomice were obtained from the animal house, department of pharmacology, Nalanda college of pharmacy, Nalgonda. They were housed in microlon boxes in a controlled environment (temperature  $25\pm 2^{\circ}\text{C}$  and 12hrs dark/light cycle) with standard laboratory diet and water *adlibitum*. All experimental procedures and protocol used in this study were reviewed and approved by institutional animal ethical committee, Nalanda College of pharmacy, Nalgonda.

### **Analgesic activity**

**Acetic acid-induced writhing method:** The prescreened animals were divided into four groups with four albino mice in each group. Each group was treated with vehicle, standard drug (Diclofenac sodium, 50 & 100mg/kg, p.o.) test drug (EECA 200 and 400mg/kg, p.o.) respectively. Writhing was induced 30 min later by intraperitoneal injection of 10 ml/kg of 0.6% acetic acid in water for injection.<sup>[3]</sup> The number of writh was counted for 20 min immediately after the acetic acid injection. The percentage protection was calculated. Percentage protection against writhing was taken as an index of analgesia.<sup>[4]</sup>

It is calculated as.

$$\frac{X1-X2}{X1} \times 100$$

Where, X1=No. of writhing in control group

X2= No. of writhing in treated group

### **Tail flick method**

The prescreened animals (reaction time:1-5 sec) were divided into four groups with four albino mice in each group. All groups were treated with vehicle, standard drug(tramadol, 5 & 10mg/kg,i. p) test drug (EECA200 and 400mg/kg, p. o.) respectively. After administration of drugs, tail flick latency was observed at 1hr, 2hrs and 3hrs. The distance between the heat

source and the tail skin was 1.5 cm. The site of application of the radiant heat in the tail was maintained at 2.5 cm, measured from the root of the tail. The cut-off reaction time was fixed at 15 sec to avoid tissue damage.<sup>[5,6]</sup>

**Statistical Analysis:** All values were shown as mean±SEM. Statistical analysis was performed using one-way analysis of variance (ANOVA) followed by Dun net's t test. P<0.001, P<0.05 was considered statistically significant.<sup>[10]</sup>

## RESULTS

**Table 1: Analgesic activity (Acetic induced writhing method).**

Groups	Treatment (mg/kg,p.o)	Number of writhing	% protection
A	Control	85±1.29	-
B	Diclofenac sodium	27±0.853*	68%
C	EECA(200mg/kg)	41±1.29*	51%
D	EECA(400mg/kg)	31±1.29*	63%

Students *t*-test. The readings are expressed as mean+SEM. \*P<0.001 as compared to control was considered significant.

The results obtained as percentage protection against writhing are shown in table -1. The results showed that standard drug diclofenac sodium, ethanolic extract of CA (200 and 400 mg/kg, p.o) suppressed the acetic acid-induced writhing response significantly in a dose-dependent manner. The results were found to be highly significant in comparison to the control.

**Table 2: Analgesic activity (Tail flick latency method).**

Group	Treatment (mg/kg)	Predrug reaction time in sec (mean±SEM)	Reaction time in sec (mean± SEM)		
			1hr	2hrs	3hrs
A	Control	3.41±0.08	2.25±0.25	2.5±0.2885	3.25±0.25
B	Tramadol	3.62±0.04	9.75±0.47**	11.5±0.645ns	13.5±0.645**
C	CA(200mg/kg)	3.85±0.04	6.25±0.62*	8±0.81635ns	10±0.8164**
D	CA(400 mg/kg)	3.65 ±0.05	9.75±0.25**	12.25±0.47ns	14.25±0.7872**
One way ANOVA		F	26.9	1.88	81
		P	<0.01*	<0.001**	n.s
		N=6 in each group	df=3,12		(non significant)

The results for tail flick model are shown in table-2. It shows that there was no significant difference in the mean predrug reaction time between the different groups. After drug administration, reaction time increased significantly for the standard and test groups when

compared to the predrug reaction time. Standard drug (Tramadol 5mg/kg,i.p), test drug (EECA200& 400mg/kg p.o) produced a dose-dependent increase in the reaction time at various time intervals of observation. The results were found to be highly significant in comparison to the control.<sup>[10]</sup>

## DISCUSSION

The abdominal constriction response induced by acetic acid is a sensitive procedure to establish peripherally acting analgesics. This response is thought to involve local peritoneal receptors. In the tail flick model, the test drug in different doses increased the pain threshold significantly during the period of observation and this indicates the involvement of a higher centre. The results of the present study suggest that the ethanolic extract of CA in doses of 200 and 400 mg/kg demonstrated significant analgesic activity in acetic acid-induced writhing and tail flick models. However, the analgesic activity of EECA was found to be more significant on the acetic acid-induced model ( $P<0.001$ ) than the tail flick model ( $P<0.01$ ) and thus it appears that the test drug inhibits predominantly the peripheral pain mechanism.<sup>[6,7]</sup>

## CONCLUSION

The present experimental study protocol showed that ethanolic extract of *Celosia argentea* elicited significant analgesic activity in acetic induced writhing model and tail flick latency model. In both model they exhibited analgesic effect in a dose dependent manner which can be comparable with that of diclofenac and tramadol respectively. On preliminary phytochemical screening the ethanolic extract of EECA was found to contain flavonoids, phenolic compounds & tannins, saponins, triterpenoids, alkaloids, and steroids compounds. Flavonoids are known to target prostaglandins which are involved in the late phase of acute inflammation and pain perception. Hence, the presence of flavonoids may be contributory to the analgesic activity of EECA. Further studies may reveal the exact mechanisms of action responsible for the analgesic activity of EECA.<sup>[8]</sup>

It was also concluded that the extract showed analgesic activity peripherally and centrally. The plant may have the phytoconstituents which inhibit cyclooxygenase enzyme for reducing analgesia peripherally or act on central opioid receptors ( $\mu$  receptors) for reducing analgesia centrally. Standard drug diclofenac sodium act on cyclooxygenase pathway of prostaglandins synthesis and tramadol act on central opioid receptors mechanism.<sup>[9]</sup>

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