

## Anti-Stress and Antiallergic Effect of *Argemone Mexicana* Stems in Asthma

R. D. Bhalke \*, S.A. Gosavi.

Department of Pharmacognosy, Sanjivani College of Pharmaceutical Education and research,  
Kopergaon, M.S. India

Petroleum ether, acetone and methanol and aqueous extracts of *Argemone mexicana* stem (50 mg/kg, i.p.) was screened for its antiallergic and antistress potential in asthma by milk-induced leucocytosis and milk-induced eosinophilia. Aqueous extracts showed significant ( $P < 0.05$ ) decrease in leucocytes and eosinophils, methanol extract also showed comparable results with aqueous extract while petroleum ether and acetone extracts were inactive. This shows polar constituents of *A. mexicana* stem are responsible for antistress and antiallergic activity.

**Keywords:** *Argemone mexicana*; Antistress; Antiallergic; Asthma

### INTRODUCTION

*Argemone mexicana* is common everywhere by road-sides and fields in India. Two aliphatic compounds; mexicanol & mexicanic acid have been isolated from leaves. Three isoquinoline alkaloids have been isolated as dihydropalmitine hydroxide; berberine & protopine, from the seeds. Oil contain up to 40% free glycerides of fatty acids. (Anonymous, 2004; Kirtikar, 1991; Rastogi, 1979) Seeds are useful in cough and asthma. Seeds are laxative, nauseant, emetic, expectorant and demulcent. The root is an anthelmintic. (Nadkarni, 1982)

It was our objective to study effect of plant extracts on milk-induced leucocytosis (antistress) and eosinophilia (antiallergic) as there is no scientific proof of the efficacy of plant extracts for antiasthmatic activity.

### MATERIAL AND METHODS

Plant Material:

*Argemone mexicana* stems and leaves were collected from Ahmednagar district of Maharashtra in June 2007 and authenticated

by Dr. P.G. Diwakar, Botanical Survey of India, Pune, where a sample specimen (voucher number: YMA1) has been deposited.

Extraction:

Dried and coarsely powdered *Argemone mexicana* stems and leaves was subjected to successive solvent extraction in Soxhlet extractor using petroleum ether, acetone and methanol as solvent and the marc left was refluxed with water. All the extracts were vacuum dried to produce PEES (3.00%), ACES (2.29%), MEES (6.13%) and AQES (5.68%), respectively.

Animals:

Healthy wistar albino mice of either sex and of approximately the same age, weighing about 20-25 gm were used for study. They were housed in polypropylene cages maintained under standard condition. The experimental protocol was subjected to the scrutiny of the Institutional Animal Ethical Committee and was cleared by the same before starting.

Milk-induced leucocytosis in mice: (Taur et.al., 2007)

Mice were divided into six groups of six animals in each group. Blood samples were collected from retro-orbital plexus. Total leukocyte count was done in each group before drug administration and 24 h

### Corresponding Author

Bhalke Rasika D.

E-mail : rasikabhalke@yahoo.co.in

after milk injection (boiled and cooled). Blood was sucked in WBC pipette up to mark and

further diluted with WBC diluting fluid. Pipette was shaken for few seconds

Table 1: Effect of various extract of *A. mexicana* stem on milk-induced leucocytosis and eosinophilia in mice.

Treatment	Difference in number of leucocytes before and after treatment(mean $\pm$ SEM)	Difference in number of eosinophil before and after treatment(mean $\pm$ SEM)
Vehicle	92.6	23.5
Control	4459.47 $\pm$ 3.63	119.38 $\pm$ 2.61
PEES	3478.9 $\pm$ 2.02*	94.67 $\pm$ 2.45
ACES	3120.3 $\pm$ 0.86*	92.6 $\pm$ 0.23*
MEES	1208.68 $\pm$ 1.34	48.92 $\pm$ 2.41*
AQES	842.36 $\pm$ 4.82*	34.76 $\pm$ 3.25

Values are mean  $\pm$  S.E.M. \*p<0.05 significant as compared to control group.

and kept aside for 5 min. Neubaur's chamber was charged with above fluid and total leukocyte count was done. Group I received vehicle, 5% polyethylene glycol (5 ml/kg, i.p.). Group II was treated with milk (4 ml /kg, i.p.). Group III– VI were treated with PEES, ACES, MEES and AQES (50 mg/kg, i.p., each), respectively and after 1 h of drug treatment each animal was injected with milk (4 mg/kg, i.p.). Difference in total leucocytes count before and after 24 h of drug administration was calculated.

Milk-induced eosinophilia in mice: (Taur et.al., 2007 )

Mice were divided into six groups of six animals in each group. Blood samples were collected from retro-orbital plexus. Eosinophil count was done in each group before drug administration and 24 h after milk injection (boiled and cooled). Blood was sucked in WBC pipette up to mark and further diluted with eosin solution. The eosin solution facilitates destruction of all corpuscles except eosinophil. Pipette was shaken for few seconds and kept aside for 5 min. Neubaur's chamber was charged with above fluid and eosinophil count was done. Group-I received vehicle, 5% polyethylene glycol (5 ml/kg, i.p.). Group II served as control and treated with milk (4 ml /kg, i.p.). Group III– VI were treated with PEES, ACES, MEES and AQES

(50 mg/kg, i.p., each), respectively and after 1 h of drug treatment each animal was injected with milk (4 mg/kg, i.p.). Difference in eosinophil count before and 24 h after drug administration was calculated.

Statistical significance:

The data is presented as mean  $\pm$  SEM. The data was analyzed by one-way ANOVA followed by Dunnet's test. P<0.05 was considered significant.

## RESULTS

### Milk-induced leucocytosis:

Amongst mice pretreated with various extracts of *A. mexicana* stem, aqueous and methanol extracts showed significant reduction in leucocytes, induced by milk as shown in table 1. Whereas petroleum ether and acetone extracts did not significantly reduce leucocytes count.

### Milk-induced eosinophilia:

Amongst mice pretreated with various extracts of *A. mexicana* stem, aqueous and methanol extracts showed significant reduction in eosinophil count, induced by milk as shown in table 1. Whereas petroleum ether and acetone extracts did not significantly reduced eosinophil count.

## DISCUSSION

### Milk-induced leucocytosis:

Physical and chemical stressors such as trauma, polluted air exposure, radiation etc. has been reported to concurrently produce immunodeficiency and oxidative stress (Elstner, 1991; Bowler, 2004). Suppression of immunity takes place due to exposure to polluted air and leads to respiratory diseases. Reactive nitrogen and oxygen species damages airways and play a role in pathophysiology of asthma. So, a drug having antistress activity induces a state of non-specific increased resistance (SNIR) against a variety of stress (Joharapurkar, 2003). After parental administration of milk, there is increase in total leucocytes count, and this stressful condition can be made normalized by administration of an antistress or adaptogenic drug. Furthermore, leucocytes during asthmatic inflammation releases the inflammatory mediators like cytokines, histamine, and major basic protein, which promote the ongoing inflammation (Brekhman, 1969). Thus aqueous and methanol extract showed protective effect against milk-induced leucocytosis.

### Milk-induced eosinophilia:

The type-I hypersensitivity reaction leads to the development of edema, vascular dilatation and eosinophilic infiltration (Justice, 2003). In late phase, especially in the development of allergic asthma, eosinophil plays a role as an inflammatory cell as it secretes mediators which result in epithelial shedding, bronchoconstriction, and promotion of inflammation in respiratory tract (Brigden,

1999). So abnormal increase in peripheral eosinophil to more than 4% of total leucocyte count (Eosinophilia) is associated with respiratory disorder, often allergic in nature together with pulmonary infiltrates (Ehright,1989). Aqueous and methanol extracts significantly reduced eosinophils, thus these extracts are useful as antiallergic in asthmatic condition.

## REFERENCES

1. Anonymous, In *the Wealth of India*. A Dictionary of Indian Raw Materials and Industrial Products, Council of Scientific and Industrial Research, New Delhi, Vol. I, I<sup>st</sup> reprint, 2004; 86-87.
2. Kirtikar KR and Basu BD..In *Indian Medicinal plants*, Vol. I, II<sup>nd</sup> Edn., Delhi, Sri Satguru Publications, 1991; 129-131.
3. Rastogi RP and Mehotra BN. In: Compendium of Indian Medicinal Plants, CDRI, Lucknow, vol. II, 1970-1979; 446.
4. Nadkarni KM. In *Indian Materia Medica*, Published by: Bombay popular prakashan, Vol.- I, 1982;133.
5. Taur DJ, Nirmal SA, Patil RY, Kharya MD. *Nat.Prod. Res.* 2007; 21:1266–1270.
6. Elstner EF, Ostwald W. *Free. Rad. Res. Commun.* 1991; 12: 789-792.
7. Bowler RP. *Curr. Aller. Asth. Rep.* 2004; 4: 123-127.
8. Joharapurkar AA, Deode NM, Zambad SP, Umathe SN. *Indian Drugs.* 2003; 40: 179-181.
9. Brekhman LI, Dardymov IV. *Ann. Rev. Pharmacol.* 1969; 9: 419-424.
10. Justice JP, McGary MP, Lee NA, Lee JJ. *Am J. Physiol. Lung. Cell. Mol. Physiol.* 2003; 284: 169-174.
11. Brigden ML.. *Postgraduate. Med.* 1999; 3: 105-109.
12. Ehright T, Chua S, Lim DJ. *Ann. Allergy.* 1989; 62: 277-279.