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**Pharmacodynamic effects of *Cedrus deodara* wood essential oil**

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The pharmacodynamic effects of *Cedrus deodara* wood essential oil were investigated in mice and rats. The oil was found to possess significant anti-inflammatory activity in carrageenin-induced oedema in rats. It was devoid of analgesic, sedative and motor incoordinating activities in mice. However, it caused a significant increase in pentobarbitone-induced hypnosis which may be due to inhibition of drug metabolising enzymes.

**C**EDRUS *deodara* wood essential oil has been indicated in the treatment of ulcers and skin diseases<sup>1-3</sup> including mange (a skin infection caused by mited viz. *Sarcoptic*, *Psoroptic* and *Demodectic* types) in horses and sorefeet in cattle<sup>4</sup>. Initial work carried out in this Laboratory revealed that 15% *Cedrus deodara* wood essential oil in castor oil is effective in the treatment of mange in different species of domestic and pet animals<sup>5</sup>. Further, safety evaluation studies revealed that the formulation is non-irritant to the skin of rabbit and sheep<sup>6</sup> and is devoid of any, adverse effect on vital organs on long-term exposure<sup>7</sup>. Apparently, no published report is available on the pharmacodynamic effects of the essential oil of *Cedrus deodara* wood. Hence, the present study has been conducted to investigate the pharmacodynamic effects of *Cedrus deodara* wood essential oil.

**MATERIALS AND METHODS**

Steam distilled essential oil of *Cedrus deodara* was procured from M/S Essential oil and Company, Jammu, India. Ten per cent emulsion of essential oil was prepared utilising Tween-80 and homogenised. It was given orally in doses of 100, 300 and 100 mg/kg. Control animals received an equal amount of vehicle.

**Pharmacological studies:** Various pharmacological tests were conducted on albino mice (18-22 g) of either sex, procured from the Laboratory Animal Resource Section of this Institute. Unless otherwise indicated, six animals were used in each group. The following experiments were performed.

**a. Anti-inflammatory effect:** Acute inflammation was produced by injecting 0.1 ml of 1% carrageenin in saline into planter aponeurosis of right hind paw of rats<sup>8</sup>. Oil was given 1 h before carrageenin. Aspirin (300 mg/kg, p.o.) was used as a reference drug. The paw volume was measured before and 3 h after drug administration.

**b. Analgesic effect**

(i) **Acetic acid-induced writhing syndrome:** It was induced in female mice by i.p injection of 3% acetic acid (300 mg/kg)<sup>9</sup>. The test drug and acetyl salicylic acid were administered 1 h before acetic acid.

(ii) **Tail clip method:** Male albino mice in groups of six were administered with graded doses of test drug. The reaction time of each mouse to pain due to bull dog clamp application at the base of the tail was determined<sup>10</sup>.

**C. Test for hypnotic activity** (Pentobarbitone sleeping time<sup>11</sup>) Animals were divided into five groups of six animals

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**Table 1: Effect of *Cedrus deodara* wood essential oil on carrageenin-induced paw oedema in rats**

Treatment <sup>n</sup>	Dose (mg/kg)	Oedema Vol. in ml (Mean ± S.E.)	% Inhibition
Control	—	0.46±0.04	—
<i>C. deodara</i> Essential oil	100	0.22±0.01*	51.00
	300	0.20±0.04*	55.00
	1000	0.16±0.03*	65.00
Acetyl salicylic acid	300	0.15±0.04*	66.00

*n*=Six animals in each group; \* *P*<0.05 as compared to control.

**Table 2: Effect of *Cedrus deodara* wood essential oil on pentobarbitone-induced sleeping time**

Treatment	Dose (mg/kg)	No. of animals (mean±S.E.)	Sleeping time in min
Control	-	6	48.33±2.61
<i>C. deodara</i> Essential oil	100	7	100.28±22.15*
	300	6	171.40±24.11*
	1000	6	272.33±12.88*
Meprobamate	200	8	228.12±21.48*

\* *P* < 0.05 as compared to control.

each. Test groups received 100, 300 and 1000 mg/kg of the emulsion of *C. deodara* essential oil while control animals received equal volume of vehicle. All the animals received 40 mg/kg of pentobarbitone sodium, i.p., 1 h after administration of test drug. The duration of loss of righting reflex was observed. Meprobamate (200 mg/kg, oral) was used as a positive control.

#### d. Motor coordination:

(i) **Rota rod test**<sup>12</sup>: The test consists of putting a mouse on a rough rotating rod at 16 rpm. The animals were trained to remain on the rod for 300 s. Satisfactorily trained group of mice were administered *C. deodara* essential oil while control group received vehicle. The animals from each group were tested at an interval of 1, 2 and 3 h after drug administration. The animals that failed to remain on the rod for 300 s were recorded.

(ii) **Traction test**<sup>13</sup>: Mice were placed in such a way that their fore arms gripped a wire that was stretched horizontally. Control mouse would take less than 5 s on the fore and hind paw gripping. One h following administration of *C. deodara* wood oil and chlorpromazine, 15 mg/kg, orally, each mouse was suspended by its fore and hind paws on the wire and time taken by the mouse to put its four paws on the wire was noted. Thirty s was the maximum time taken for calculation of the data if animals failed to grip wire with its fore and hind paws.

e. **Anticonvulsant activity**<sup>14</sup>: It was studied using the chemoshock test. Chemoshock was induced by pentylenetetrazole (90 mg/kg, s.c). Pentylenetetrazole was administered 1 h after administration of test or reference drug. The duration of onset of clonic convulsions was recorded in control and treated groups.

Statistical analysis was carried out by Student's unpaired 't' test and the level of significance was set at  $P < 0.05$ .

## RESULTS

Essential oil of *C. deodara* wood significantly inhibited hind paw oedema volume in rats at 3 h after carrageenin in 100, 300 and 1000 mg/kg doses. The percent inhibition in oedema volume was 52, 55 and 65, respectively (Table-1).

There was no significant change in the reaction time of mice treated with vehicle or essential oil at doses of 100, 300 and 1000 mg/kg when tested by tail clip test. The mean number of writhing movements in vehicle-treated mice was  $61.5 \pm 6$  in acetic acid-induced writhing test. Treatment with essential oil failed to reduce the response significantly at any of the dose level used in this study.

Table-2 reveals the duration of pentobarbitone-induced sleep after administration of *C. deodara* essential oil in comparison with those obtained following vehicle and meprobamate administration. *C. deodara* essential oil potentiated the duration of pentobarbitone-induced sleep.

The essential oil did not produce any incoordination in mice at 1 and 3 h after its administration. The mice remained on the rotating rod for 300 s, a time for which they were trained. Chlorpromazine (10 mg/kg, oral), a reference drug, produced incoordination in 37.5 and 50% of animals at a similar time interval. Essential oil (300 and 1000 mg/kg) prolonged the time taken by the animal to stand on its four paws on a horizontal wire from mean control value of  $1.16 \pm 0.33$  to  $2.11 \pm 0.84$  and  $11.6 \pm 4.62$  s, respectively. The effect was significant ( $P < 0.05$ ) with 1000 mg/kg dose. Chlorpromazine also significantly ( $P < 0.05$ ) increased the reaction time ( $12.45 \pm 4.86$  s). The oil failed to alter the onset of duration of clonic convulsions and 24 h mortality in mice following pentylenetetrazole administration.

## DISCUSSION

The data obtained in this study suggested that *C. deodara* wood essential oil possessed good anti-inflammatory activity in acute inflammation induced by carrageenin in rats. The effects were statistically significant at all the doses employed in the study. The anti-inflamma-

tory effect shown by 1000 mg/kg of *C. deodara* wood essential oil was comparable to 300 mg/kg acetylsalicylic acid. Anti-inflammatory activity has been reported from polar fraction of the plant extract<sup>15</sup>. The essential oil of *C. deodara* wood oil did not show significant analgesia in tail clip test and acetic acid-induced writhing test in mice. Hence, it is evident from these results that the oil is devoid of central as well as peripheral analgesic effect. The oil failed to demonstrate anticonvulsant activity in chemoshock test.

The *C. deodara* wood oil did not show any sedation and motor incoordination. However, it caused significant increase in the time taken by the mouse to stand on four paws on horizontal wire. Further, the oil potentiated the pentobarbitone-induced hypnosis. Increase in pentobarbitone hypnosis is observed when a chemical has CNS depressant effect or when it inhibits liver microsomal enzymes, thereby increasing the time of biotransformation of pentobarbitone<sup>16</sup>. In the present study, it appears that the *C. deodara* wood essential oil has inhibitory effect on drug metabolising enzymes since it was devoid of any CNS depressant effect. However, this needs further confirmation.

In conclusion, the *C. deodara* wood essential oil does not appear to possess any appreciable activity on CNS. Although, further work is needed, the present results suggest that the oil possesses some anti-inflammatory activity which may be of therapeutic advantage in the treatment of mange.

## REFERENCES

1. Kirtikar K. R. and Basu B. D. In; Blatter, E., Cains, J. F., Mahashakar, K. S., Eds Indian Medicinal Plants, Vol. II, Allahabad, India, 1933, 2390.
2. Chopra, R. N., Nayar S. L. and Chopra I. C. In; Glossary of Indian Medicinal Plants, CSIR, New Delhi, 1956, 56.
3. Maheshwari, P. and Singh, U. In; Dictionary of Economic Plants of India, Indian Council of Agricultural Research, New Delhi, 1965, 36.
4. Nadkarni A. K. In; Dr. Nadkarni's Indian Materia Medica, III Ed, Dhooteshwar Prakashn Ltd., Bombay, 1955, 295.
5. Lal, J., Sambasivarao, K., Chandra, S., Naithani, R. C., Chattopadhyaya S. K. and Sabir M., Indian Vet. J., 1976, 53, 543.

6. Chandra S., Prasad M. C., Tandan S. K., Gupta S. and Lal J., *Indian Vet. J.*, 1989, 66, 30.
  7. Tandan S. K., Singh R., Gupta S., Chandra S. and Lal J., *Indian Vet. J.*, 1989, 66, 1088.
  8. Winter C. A., Risley E. A. and Nuss C. W., *Proc. Soc. Exp. Biol. Med.*, 1962, 111, 544.
  9. Witkin L. B., Heuner C. F., Galdi F., Kees E. D. and Spitalleta P., *J. Pharmacol. Exp. Therap.*, 1961, 133, 400.
  10. Bianchi C. and Franceschini J., *Brit. J. Pharmacol.*, 1954, 9, 280.
  11. Bilbey D. L. J., Salen H. and Grossman M. H., *Brit. J. Pharmacol.*, 1960, 15, 540.
  12. Janssen P. A. J., Jageneau A. H. and Niemegeers C. J. E., *J. Pharmacol. Exp. Therap.*, 1960, 129, 471.
  13. Turner R. A., In; *Screening Methods in Pharmacology*, Academic Press, New York, London, 1965, 89.
  14. Swinyard E. A., Brown W. C. and Goodman L. S., *J. Pharmacol. Exp. Therap.*, 1952, 106, 319.
  15. Agrawal P. K., Agrawal S. K., Rastogi R. P. and Osterdahal B. G., *Planta Medica*, 1981, 43, 82.
  16. Coney A. H., *Pharmacological Rev.*, 1967, 19, 115.
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