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Antihistaminic activity of *Clitoria ternatea* L. roots

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ABSTRACT: Clonidine, a α_2 adrenoreceptor agonist induces dose dependent catalepsy in mice, which was inhibited by histamine H1 receptor antagonists but not by H2 receptor antagonist. Clonidine releases histamine from mast cells which is responsible for different asthmatic conditions. *Clitoria ternatea* L. (Family: Fabaceae) is a perennial twining herb. The roots have anti-inflammatory properties and are useful in severe bronchitis, asthma. In present study ethanol extract of *Clitoria ternatea* root (ECTR) at doses 100, 125 and 150 mg/kg i.p were evaluated for antihistaminic activity using clonidine and haloperidol induced catalepsy in mice. Finding of investigation showed that chlorpheniramine maleate (CPM) and ECTR inhibit clonidine induced catalepsy significantly $P < 0.001$ when compare to control group, while CPM and ECTR fail to inhibit haloperidol induced catalepsy. Present study concludes that ECTR possesses antihistaminic activity.

KEYWORDS: *Clitoria ternatea*, Clonidine, antihistamine, Chlorpheniramine maleate

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INTRODUCTION

C*litoria ternatea* L. (Family: Fabaceae) a perennial twining herb, stems are terete, more or less pubescent. The roots have a sharp bitter taste and cooling, laxative, diuretic, anthelmintic, anti-inflammatory properties; they are useful in severe bronchitis, asthma and hectic fever [1,2]. The fatty acid content of *C.ternatea* seeds includes palmitic, stearic, oleic, linoleic, and linolenic acids [3-5]. The seeds also contain a water-soluble mucilage, delphinidin 3, 3', 5'-triglucoside useful as a food dye [6]; beta-sitosterol [7]. *C. ternatea* possesses number of pharmacological activities such as nootropic, anxiolytic, antidepressant, anticonvulsant [8], sedative [9], antipyretic, anti-inflammatory and analgesic activities [10]. It enhance the memory, and increase acetylcholine content and acetylcholinesterase activity in rats [11-13]. Objective of present study was to evaluate ethanol extract of *Clitoria ternatea* root (ECTR) on Clonidine and haloperidol induced catalepsy in mice.

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MATERIALS AND METHODS

Plant material

Roots of *C. ternatea* were collected in December 2008, from Baramati localities, Pune district (Maharashtra), and dried in the shade at room temperature. Dried roots were coarsely powdered in grinder and powder material was kept in air tight container for further study. The plant was identified and authenticated by Prof. R. B. Deshmukh Head Dept. of Botany, Shardabai Pawar Mahila Mahavidyalaya, Baramati; Specimens are deposited, with plant specimen PASR-113.

Extraction

Dried and coarsely powder of *C. ternatea* roots 500 gm extracted successively with ethanol (95 %) in soxhlet extractor. Extract was concentrated to dryness in rotary evaporator under reduced pressure and dried on water bath to yield ethanol extract of *Clitoria ternatea* roots (ECTR) 11.27 % w/w.

Animals

Swiss albino mice of either sex weighing 25-30g were housed under standard laboratory conditions,

in groups of five. The animals had free access to food and water. The animal ethical committee of the institute approved all the protocols of the study.

Drugs and Chemicals

Clonidine (Unichem, Ltd.); Chlorpheniramine maleate (Alkem, Mumbai), Haloperidol.

Statistical Analysis

The results were reported as mean \pm SEM and analyzed for statistical significance using One way ANOVA followed by Dunnett' test $P < 0.05$ was considered significant.

Clonidine-induced catalepsy in mice

Bar test was used to study effect of extracts on clonidine-induced catalepsy, to determine indirect antihistaminic activity. Mice were divided into five groups, five animals in each group. Groups of animal pretreated with (Tween-80 1%, 5 ml/kg, i.p.), ECTR at doses (100, 125 and 150 mg/kg i.p) and chlorpheniramine maleate (10 mg/kg, i.p.); received Clonidine (1mg/kg, s.c.) 30 min after treatment. The forepaws of mice were placed on a horizontal bar (1 cm in diameter, 3 cm above the table) and the time required to remove the paws from bar was noted for each animal. The duration of catalepsy was measured at 30, 60, 90, 120, 150 and 180 minute interval after administration of clonidine [14, 15].

Haloperidol-Induced Catalepsy

The same bar test was used using haloperidol. Groups of animal pretreated with (Tween-80 1%, 5 ml/kg, i.p.), ECTR at doses (100, 125 and 150 mg/kg i.p) and chlorpheniramine maleate (10 mg/kg, i.p.) received Haloperidol (1 mg/kg, i.p.) 30 min after treatment. The forepaws of mice were placed on a horizontal bar (1 cm in diameter, 3 cm above the table) and the time required to remove the paws from bar was noted for each animal. The duration of catalepsy was measured at 30, 60, 90, 120, 150 and 180 minute interval after administration of clonidine [4, 15].

RESULTS

Clonidine-induced catalepsy in mice

Clonidine releases histamine from mast cells which is responsible for different asthmatic conditions. Catalepsy produced by clonidine is mediated by histamine via H_1 receptors. The maximum catalepsy was observed after 90 minute of clonidine administration (1 mg/kg, i.p.) in vehicle treated (control) group. Prior treatment with ECTR at doses (100, 125, 150 mg/kg i.p) and Chlorpheniramine maleate (10 mg/kg, i.p.) showed significant ($P < 0.001$) inhibition of clonidine induced catalepsy in dose dependent as shown in (Figure 1).

Haloperidol-Induced Catalepsy

The maximum catalepsy was observed 90 minute after administration of haloperidol (1 mg/kg, i.p.).

Figure 1: Effect of ECTR on clonidine induced catalepsy in mice. One way ANOVA followed by Dunnett's test * $P < 0.001$, as compared to control group.

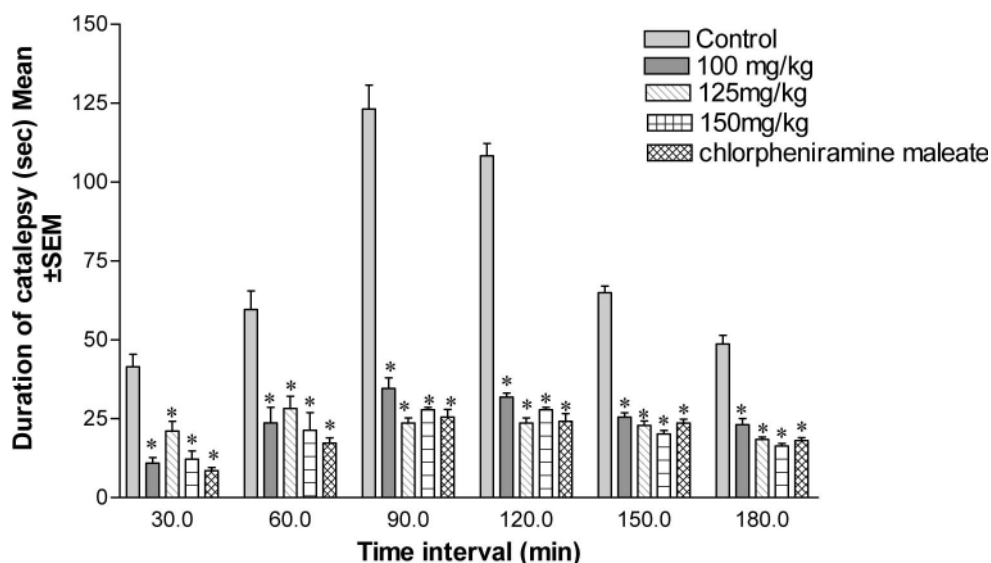
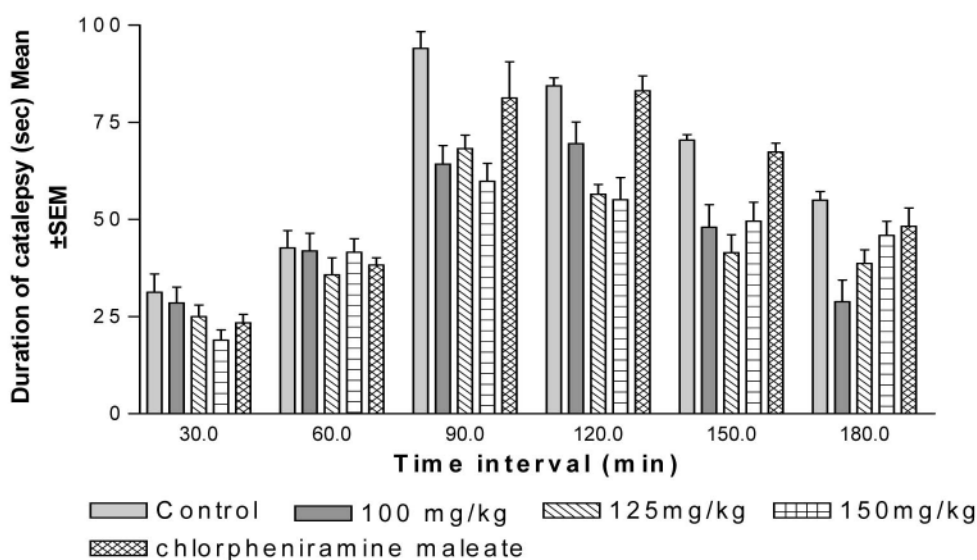


Figure 2: Effect of ECTR on haloperidol induced catalepsy in mice.

None of the groups prior treated with ECTR at doses (100, 125, 150 mg/kg i.p) and Chlorpheniramine maleate (10 mg/kg, i.p.) inhibits haloperidol induced catalepsy as shown in (Figure 2).

DISCUSSION

Clonidine, a α_2 adrenoreceptor agonist induces dose dependent catalepsy in mice, which was inhibited by histamine H1 receptor antagonists but not by H2 receptor antagonist [16]. Clonidine releases histamine from mast cells which is responsible for different asthmatic conditions [17]. Catalepsy produced by clonidine is mediated by histamine via H1 receptors. In present study it was found that Chlorpheniramine maleate (10 mg/kg, i.p.) and ECTR at doses (100, 125 and 150 mg/kg i.p) inhibit catalepsy in dose dependent manner. Dhanalakshmi et al., (2004) showed that extracts having antihistaminic or mast cell stabilizing effect inhibit clonidine-induced catalepsy [15]. Haloperidol, a non-selective D2 dopamine antagonist induces catalepsy is primarily due to blockade of dopamine receptors in the striatum. The agents increasing dopamine transmission inhibits haloperidol-induced catalepsy [18]. An antihistaminic drug chlorpheniramine maleate and ECTR fail to inhibit haloperidol induced catalepsy. It indicate that haloperidol induced catalepsy is not mediated by histamine release as antihistaminic drug does not inhibit catalepsy but clonidine induces catalepsy by the release of histamine as it is inhibited by

antihistaminic drug. Some plants were investigated for antihistaminic activity inhibit clonidine induced catalepsy in mice *Allium sativum* and *Terminalia bellerica* [19], *Clerodendrum serratum* [20], *Tamarindus Indica* [21], *Clitoria ternatea* [22], *Ficus bengalensis* [23], etc.

Present study concluded that the drugs having antihistaminic potential inhibit clonidine induced catalepsy, so ethanol extract of *Clitoria ternatea* roots possesses antihistaminic activity. Future scope of present investigation is isolate active phytoconstituents which is responsible for antihistaminic activity.

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