ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Evaluation of Analgesic Activities of Black Tea Decoction on Experimental Animal Model.

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ABSTRACT

Tea is most commonly consummated beverage in the world. Black tea is made from the dried leaves of Camellia sinensis, a shrub formerly known as Thea sinensis. We had set forward this study to examine the analgesic activities of black tea in experimental animal model. Evaluation of analgesic effects of black tea on mice. Black tea leaves were commercially obtained from P and A Arse, Rajdhani Apt. BIK‐ I Rg Barua Road,Ganeshguri, Guwahati , tea leaves (100 gm) were extracted with ethyl acetate using soxhlet assembly. The extract was concentrated in a rotary flash evaporator under reduced pressure to semisolid mass. black tea decoction (10% and 20%) was prepared by soaking 10 and 20 g of black tea in 100 ml boiled water separately, soaked for 2 min and thereafter filtered. This filtrate was designated as black tea decoction. Central and peripheral analgesic activities were evaluated by tail flick, tail immersion and acetic acid-induced writhing test respectively. This is an experimental study. In central analgesic model black tea decoction (10% and 20%) had shown no analgesic action at different hours as the reaction time was less than 10 seconds at all time interval. But at peripheral analgesic model black tea decoction (10% and 20%) had shown 20% and 39.81% inhibition, respectively, as compared to control group whereas Aspirin had shown around 52.81% ($p<0.002$) of inhibition. Taken together, our data indicate that black tea (20%) has a potential peripheral analgesic activity. Further studies involving isolation of active principles will help to pinpoint the mechanisms contributing to the analgesic activity of tea.

Keywords: analgesic, black tea, decoction

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ISSN: 0975-8585

INTRODUCTION

Tea is most commonly consummated beverage in the world. [1] . Black tea, green tea, and oolong tea are all derived from the same plant. Black tea is produced by oxidation of *Camellia sinensis* leaves and is a traditional beverage in India. The quality and activity of tea depends on the age of the tea leaves.

During tea oxidation, simple polyphenols undergo polymerization, which leads to more complex molecules, such as theaflavins and thearubigins. These molecules are more specific to black tea and may have health benefits, as discussed below.

Black tea is a source of caffeine, a methylxanthine that stimulates the central nervous system, relaxes smooth muscle in the bronchioles, stimulates the heart, and acts on the kidney as a diuretic. Tea also contains polyphenols (catechins, anthocyanins, phenolic acids),vitamins, tannin, and trace elements.

The active components of tea are known to be catechins (known as polyphenols), which constitute seven forms including epigalocatechin-gallate (EGCg).[1] EGCg is a major catechin compound present in tea extracts and is also the most active form in various biological activities. Studies on experimental animal model have revealed its anti-inflammatory activities in different situation. Tea drinking provide many health benefits: promotion of antiaging, antioxidant, oral health, anticancer, cardiovascular health and anti-inflammatory activities.Therefore as pain is an important component of inflammation, this study was undertaken to examine the analgesic effect of black tea decoction in mice.

Objectives

Evaluation of analgesic effects of black tea on mice

MATERIALS AND METHODS

Animals

The entire study was carried out in a government medical college as a part of post-graduate dissertation using Swiss mice of average weight 15-25 g. of either sex. The animals were maintained under standard laboratory conditions with free access to commercial pellet feed and water *ad libitum*. The animals were housed for a period of seven days for acclimatization prior to the commencement of experimental work under fixed 12-hour alternate light and darkness cycle.

Preparation of Plant Product

Black tea leaves were commercially obtained from P and A Arse, Rajdhani Apt. BIK‐ I Rg Barua Road,Ganeshguri, Guwahati , tea leaves (100 gm) were extracted with ethyl acetate using soxhlet assembly. The extract was concentrated in a rotary flash evaporator under reduced pressure to semisolid mass. Black tea decoction (10% and 20%) was prepared by soaking 10 and 20 g of black tea in 100 ml boiled water separately, soaked for 2 min and thereafter filtered. This filtrate was designated as black tea decoction. The dose of this decoction orally administered to each rat was 0.1 ml/10 g of body weight. An initial pilot study suggested that 20% of this preparation gave best results. Therefore, we had decided to set forward our study with 10% and 20% black tea decoction.[1] It was authenticated by Prof Sukanta Banerjee Choudhury , dept of Botany and voucher no was submitted to appropriate authority.

Evalution of analgesic profile of Tea decoction

In experimental animal models, the analgesic profile of black tea was screened in the following models.

ISSN: 0975-8585

Models for evaluation of Central analgesic activity

- Tail flick method
- Tail immersion method

Models for evaluation of Peripheral analgesic activity

- Acetic acid induced writhing test

Method for evaluation of central analgesic activity

The method as described by publications by D' Armour et al., (1941) was followed[2,3]. Forty eight rats were taken and they were divided into four groups of twelve rats each. The groups were treated as follows:

Group (n=12)	Treatment
Group I	Control
Group II	Standard drug, Pethidine (12 mg/kg)
Group III	10% black tea decoction
Group IV	20% black tea decoction

Tail Flick Test

Each animal was placed in a mice restraint cage, which has an opening for the tail at the rear end. The tail of each mouse was placed 2 mm above the nichrome wire of the analgesiometer (Model Mark I Techno Electronic, Lucknow). A current strength of 5 amp was passed and the radiant heat thus generated was directed to the proximal third of the tail. The time taken by each animal to withdraw (flick) the tail away from the hot wire was recorded as the reaction time. Any animal that failed to withdraw its tail within 15 seconds was not used for the experiment. The escape reaction (i. e., pulling away of the tail and turning the head), which is the end point of the test, is regarded as a complex phenomenon mediated by the brain. In contrast, the simple tail flick as an end point of this test may be mediated as a spinal reflex. Therefore, the observation of the escape reaction can be regarded as a true assessment of the influence of the drug on the brain. Before administration of the test compound or the standard drug, the normal reaction time was determined. The reaction time was measured at 30, 60, 120 and 150 min after administration of test and standard drugs. The analgesic activity was classified as positive if the mice failed to withdraw its tail within 15 sec of exposure.

Tail immersion method

This test was based on the method as adopted by Ramabadran *et al*. (1989). [4] The albino mice were divided into four groups of twelve animals each. The lower 5.0 cm portion of the tail was then dipped in a beaker of water maintained at 55 ± 0.5ºC. The time in seconds required to withdraw the tail clearly out of water was taken as the reaction time. The animals that could not lift its tail out of the water within 10 seconds were discarded. The basal reaction time for all the animals were recorded as the 0 minute observation. The test drug tea decoction was administered orally in doses of 10% and 20%, while the standard drug, pethidine (12 mg/kg) was administered intraperitoneally to the respective three groups as was maintained for the previous tests. A normal control, without any treatment was also maintained. The reaction time was noted 30, 60, 90, 120 and 150 min after drug administration.

Model for evaluation of Peripheral analgesic activity

Acetic acid induced writhing test

This was based on the method described by Koster *et al.* (1959).[5] Albino mice of either sex were divided into four groups of six animals each. Tea (10% and 20%) at doses of 0.1ml/10gm, aspirin (30 mg/kg; standard) were administered respectively to the three groups orally before i.p. injection of 0.6 % v/v acetic acid solution in water at a dose of 10 ml/kg. A control group without any drug treatment was maintained,

ISSN: 0975-8585

which was also treated with acetic acid. Immediately after administration of acetic acid, the number of writhes or stretches (a syndrome, characterized by a wave of contraction of the abdominal musculature followed by extension of hind limbs) were counted for 15 min. A reduction in the number of writhes as compared to the control group was considered as evidence for the presence of analgesia, expressed as percent inhibition of writhing, which is calculated according to the following formula:

$$\% \text{ Inhibition} = \frac{\text{Mean number of writhes in control group} - \text{Mean number of writhes in test group}}{\text{Mean number of writhes in control group}}$$

RESULTS

In central analgesic model black tea decoction (10% and 20%) had shown no analgesic action at different hours as the reaction time was less than 10 seconds at all-time interval. (Fig 1, Fig 2) Remarkable analgesic effect was observed with pethidine. (Fig 1, Fig 2) But at peripheral analgesic models black tea decoction (10% and 20%) had shown significant inhibition as compared to control group. The number of writhes in untreated controls was 55.25 ± 1.26; in comparison to 25.25 ± 4.72 in aspirin pretreated animals.10% black tea showed about 20% ($p<0.005$) inhibition of number of writhes whereas percentage inhibition of writhing produced by 20% black tea was 39.81% ($p<0.001$). That produced by 30 mg/kg of aspirin, the standard drug was 52.81% ($p<0.002$).(Fig 3)

Figure 1: Evaluation of analgesic activity of Black tea by tail immersion method

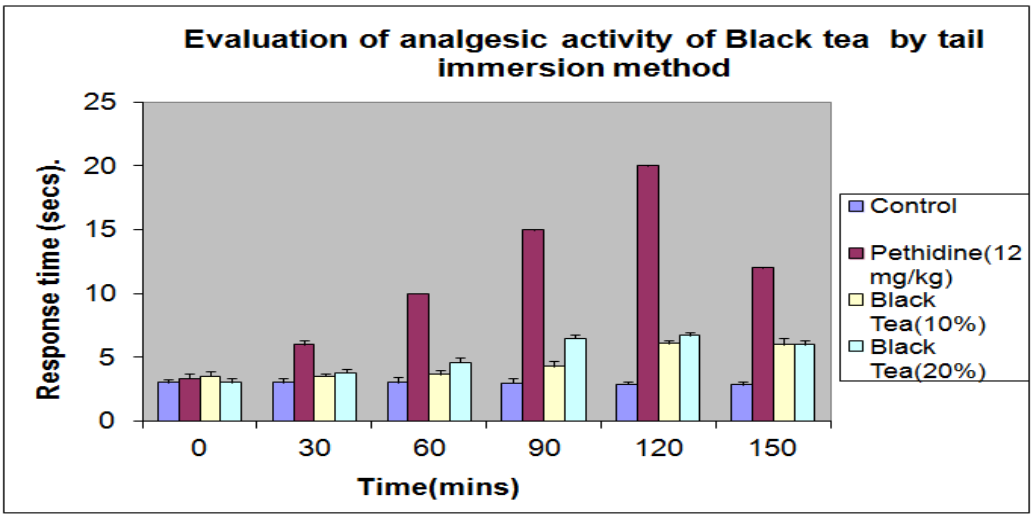
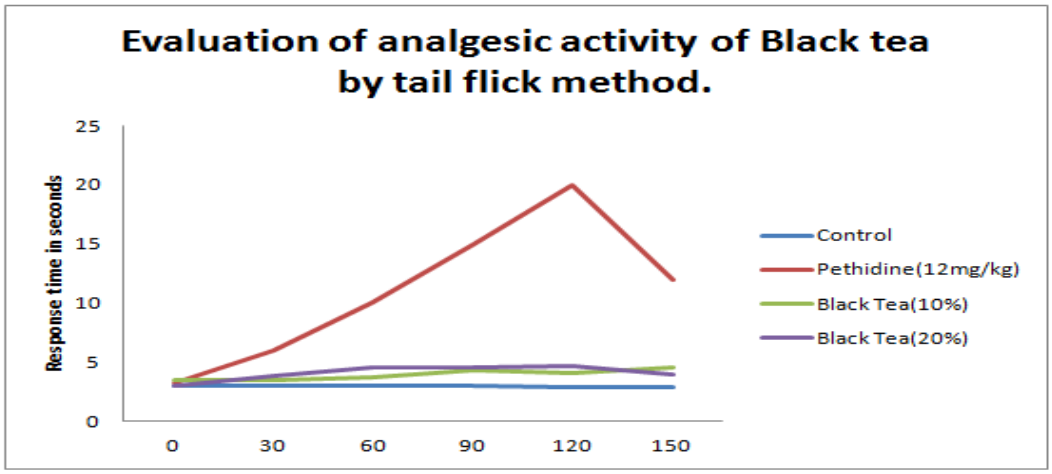
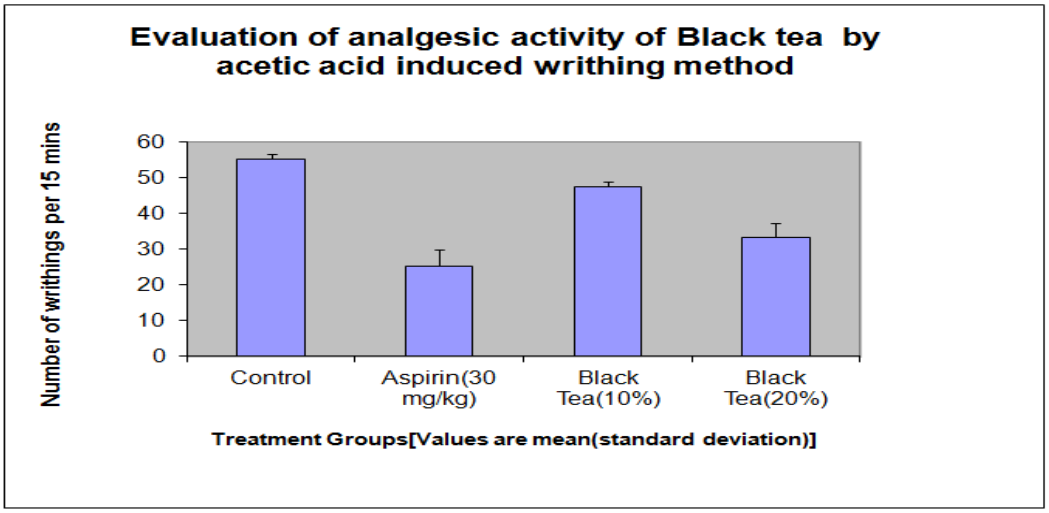


Figure 2: Evaluation of analgesic activity of Black tea by tail flick method



ISSN: 0975-8585

Figure 3: Evaluation of analgesic activity of Black tea by acetic acid induced writhing test.



DISCUSSION

Experimental animal models have been taken for assessing both the central and peripheral analgesic activity. Although today the classification of central and peripheral analgesics is definitely too simplified, it provides a guide for differentiation by pharmacological methods.[6] In the tail immersion and tail flick method of assessing the central analgesic activity tea did not show any significant activity.

In order to distinguish between central and peripheral analgesic action of tea decoction, acetic acid induced writhing response in mice was used to examine the effect. This method reliably affords rapid evaluation of analgesic action. It was found that tea significantly inhibited acetic acid induced responses in a dose dependent manner. It effectively reduced the wave of constriction and elongation passing caudally along the abdominal wall with twisting of the trunk and extension of the hind limb in mice in response to the nociceptive property of acetic acid. The number of writhes in untreated controls was 55.25 $\pm$ 1.26; in comparison to 33.25 $\pm$ 4.72 in Aspirin pretreated animals. Black tea showed about 19% ($p<0.005$) inhibition of number of writhes whereas percentage inhibition of writhing produced by 20% Black tea was 35.74% ($p<0.001$). it was similar to that produced by 30 mg/kg of aspirin, the standard drug that inhibited 52.81% ($p<0.002$). This suggests that the tea possesses analgesic property that is peripherally mediated.

Sensitization of peripheral nociceptors causes hyperalgesia. It occurs when chemical products, such as bradykinin, 5-HT, histamine and eicosanoids (prostaglandins and leukotrienes) are released near nociceptor terminals after or during tissue inflammation. For, example, a noxious stimulus applied to the skin or produced during inflammation may destroy cells near nociceptor. Dying cells release proteolytic enzymes that react with circulating globulins to from bradykinin. Bradykinin binds to a receptor on the membrane of the nociceptor and activates a second messenger system, sensitizing the nerve ending [7]. Bradykinin also provokes the release of neuropeptides such as substance P, neurokinin A, and CGRP [8].

Histamine and 5-HTcan elicit pain but they are 50 times less potent than bradykinin. Prostaglandins sensitize the nerve endings to the effects of bradykinin and other algogens [9]. Underlying mechanism of nerve ending sensitization is either opening up of the ion channels or by activating second messenger. Therefore, it is possible that the peripheral analgesic activity of tea observed was due to its concomitant anti inflammatory activity.

CONCLUSION

Pain is a common symptom of most inflammatory conditions and most anti-inflammatory drugs also possess analgesic activity. Therefore, the analgesic activity of tea decoction was screened in experimental animal models. Tea showed significant peripheral analgesic activity, as elucidated in the 'acetic acid writhing'

model, whereas no effect was observed using models for screening central analgesic activity namely tail immersion and tail flick models.

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