

Anti-inflammatory activity of Indian black tea (Sikkim variety)[☆]

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Abstract

In this study, the anti-inflammatory (in reference to the cardinal signs of inflammation) and other related pharmacological activities of the hot water extract of black tea (*Camellia sinensis*, Sikkim variety) were evaluated along with certain standard drugs. The extract showed significant inhibitory activity against carrageenin, histamine, serotonin and prostaglandin-induced pedal inflammation. The extract inhibited exudative inflammation. The tea extract also inhibited cotton pellet-induced granuloma formation and adjuvant-induced polyarthritis. Black tea extract showed significant inhibition against glucose oxidase-mediated inflammation. The present observations establish the efficacy of this particular variety of black tea, both in the exudative and proliferative forms and as well in the chronic phase of inflammation.

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1. Introduction

Tea (*Camellia sinensis*) has been utilised, since time immemorial, as a beverage possessing encouraging health benefits. However, with respect to modern scientific point of view, only by the end 1980s, green tea became even more popular for its definite medicinal values, globally. By this time, sufficient data were generated, for claiming that consumption of green tea has preventive effects especially on occurrence of cancer and cardiovascular disorders.

Today, tea is the most widely used beverage, aside water with a per capita worldwide consumption of approximately 0.12 L per year [1]. Among the three varieties of tea based on the manufacturing procedures (e.g. green tea, oolong tea and black tea), black tea alone accounts for about 72% of the total tea manufactured globally [2]. Claims and counterclaims have been made regarding the beneficial as well as deleterious effect of tea drinking on human health. Recent reports on

tea and human health claim a plethora of therapeutic properties including antilipidemic [3], antineoplastic [4], antidiabetic [5], antioxidant [6] and many others. Whereas important charges against this beverage are that it causes hypertension, insomnia, peptic ulceration and even heart disease [7].

While quite a lot of work has been carried out with green tea and its constituents, not much work was done till 1990, regarding evaluation of the pharmacological properties of black tea (which accounts for the majority of the tea consumed in India and also globally). However, during the 1990s, Food and Agricultural Organisation (FAO) recommended the evaluation of the protective effects of green and black tea. This led to a systemic research on black tea and its constituents, thereby unfolding several beneficial effects of black tea.

It may also be pertinent to mention that there exist some differences in the chemical constituents of green tea from its black variety. It is well established that during the process of manufacture, green tea catechins (namely epicatechin, epicatechin gallates (ECG), epigallocatechin (EGC) and epigallocatechin gallates (EGCG)) are oxidized and converted into orange or brown products known as theaflavins (TF) and thearubigins (TR) [8]. These compounds retain the basic C₆–C₃–C₆ structure and are thus still classified as flavonoids. Theaflavins consist of two catechin molecules joined together, and account for about 10% of the converted

[☆] This paper is dedicated to the loving memory of our mentor Prof. A.K. Nag Chaudhuri. He stimulated our scientific curiosity and nurtured our development as researcher and we still admire and respect him as our teacher, scientist and a great scholar.

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catechins, whereas the thearubigins are more complex flavonoid molecules, whose structural chemistry is still unknown, and may account for up to 70% of flavonoids in black tea [9]. Black tea is generally assumed to be much less beneficial than the green variety due to its lower content of unpolymerised polyphenols; however, no specific data is available in this respect. [10] Even though on survey of literature, it is revealed that black tea does possess antihypertensive [11], antiulcer [12], antihyperglycaemic [13] properties.

Despite the large number of pharmacological studies on tea, carried out worldwide, search of published articles reveal that there is a need to investigate its anti-inflammatory activity. The present study is, therefore, aimed to evaluate the possible anti-inflammatory and related properties of Indian black tea (Temi tea; Sikkim variety). Keeping in view the cardinal signs of inflammation (i.e. oedema, exudation, pain, pyrexia), the models has been chosen in order to ascertain the effects of tea on acute inflammation (using oedema, exudation, change in capillary permeability) using various inflammogens' pain response and chronic inflammatory condition (formaldehyde-induced arthritis).

2. Materials and methods

2.1. Animals used

The studies were performed on male Albino rats of the Charles Forster strain (120–150 g) and Swiss Albino mice (18–25 g). The animals were housed in standard laboratory conditions ($24 \pm 2^\circ\text{C}$) and were supplied with food pellets (M/s Lipton, India) and water ad libitum.

2.2. Preparation of hot water extract of the black tea

Experiments were done with a particular variety of tea, branded as Temi tea. Grown in the Temi tea gardens in the state of Sikkim, India. This brand of tea was collected from Sikkim (India). Tea (*C. sinensis*) weighing 400 mg was soaked in 10 ml of boiling water (fraction T_1) and 4 g of tea was soaked in 10 ml of boiling water (fraction T_2). After 5 min, it was filtered and the filtrate was administered orally in a dose of 5 ml kg^{-1} (T_1) and 2.5 ml kg^{-1} (T_2) to the different groups of rats and mice. The freshly prepared filtrate was considered as black tea extract.

2.3. Carrageenin-induced rat paw oedema

Carrageenin (type I; 0.1 ml of 1% solution) was injected into the plantar aponeurosis of the right hind paw of the rats [14], while the control vehicle or the test material or the standard drug was fed orally 30 min prior to the injection of the carrageenin. The paw volume was measured just before and on hourly basis (up to 6 h) following carrageenin administration using the volume displacement method [15].

2.4. Arachidonic acid-induced rat paw oedema

Arachidonic acid (0.1 ml of 0.5% solution in 0.2 M carbonate buffer; pH 8.43–8.56) was injected into the subplantar tissue of the right hind paw of male rats. The animals were pretreated 2 h earlier with standard drug (orally) and 30 min earlier with black tea extract (orally). The oedema was measured using the volume displacement method 1 h after arachidonic acid injection [16].

2.5. Mediator-induced rat paw oedema

The method of Parmar and Ghosh [17] was essentially followed for this experiment. A solution (0.1 ml) of histamine base ($10^{-3} \text{ g ml}^{-1}$; 1 h), serotonin ($10^{-3} \text{ g ml}^{-1}$; 30 min) and prostaglandin E_2 ($10^{-6} \text{ g ml}^{-1}$; 30 min) was injected into the hind paw after 30 min following administration of black tea extract (200 and 1000 mg kg^{-1} ; oral) to groups of rats. The oedema volume was determined following the method of Bhatt et al. [15]. The respective dosage of the mediators and the time of determination of oedema volumes are indicated in parenthesis against each.

2.6. Exudative inflammation

In order to evaluate the effect on peritoneal inflammation in mice, the control vehicle or black tea extract and aspirin (100 mg kg^{-1} ; i.p.) was administered to each animal. One hour later, 4 ml of 0.05N acetic acid in normal saline (i.p.) was administered to each animal. Three hours later, the animals were sacrificed by decapitation, and the protein content in the peritoneal exudates was determined following the method of Bradford [18].

2.7. Acetic acid-induced writhing

Control vehicle or test material or the standard drug were administered (orally) to group of animals and 30 min later, 3% acetic acid (0.1 ml/10 g of mice; i.p.) was injected into each animal and the number of writhing responses were recorded for a period of 20 min [19].

2.8. Tail clip-induced algesia

Black tea extract (1000 mg kg^{-1} ; oral) pethidine (10 mg kg^{-1} , i.p.) and control vehicle (oral) was administered to different groups of mice (pre-screened). The tail clip was applied to the mice, 15 and 30 min following the administration of the test substances and the response of each animal was determined [20].

2.9. Yeast-induced pyrexia

Groups of animals ($n = 6$) were injected with 15% Brewer's yeast suspension in normal saline (1 ml/100 g of body weight) and 5 h later the rectal temperature was recorded

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