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TO STUDY ACTIVITY OF ACACIA FARNESIANA BARK ON INFLAMMATION INDUCED DIARRHOEA

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ABSTRACT

Acacia farnesiana, native to tropical parts of the Indian subcontinent, thrives in dry climates with minimal rainfall. Its bark extract has shown activity against Mycobacterium tuberculosis and dysentery bacteria. Studies reveal its anti-inflammatory properties, attributed to proteins in the seeds and phenolic compounds in the bark extract. These compounds inhibit paw edema and have antioxidant properties. A. farnesiana also demonstrates antidiarrheal activity, inhibiting intestinal motility and secretion. Similarly, Acacia seyal roots exhibit antibacterial properties against various pathogens, suggesting their potential in treating diarrhoea. Therefore, both A. farnesiana and A. seyal are promising candidates for developing anti-inflammatory and antidiarrheal agents.

Keywords- Anti-Inflammatory, Anti-Diabetic, Anti-Diarrhoeal, Mycobacterium Tuberculosis.

INTRODUCTION

Traditional Use of *Acacia Farnesiana*

Traditional herbal medicine plays an important historical role, and this certainly applies to the use of products presented as "traditional herbal medicine". In many developing countries, a large proportion of the population depends on traditional healers and their medicinal plants for their health care needs. Modern medicine exists alongside traditional practice for historical and cultural reasons. Herbal medicines now often kept their popularity. Health care of well-preserved herbal medicines; Although there are many differences between traditional pharmacological and herbal treatments, the effectiveness of herbal medicines must be tested using a standard experimental method, and several specific plant extracts have been shown to be effective for a specific disease. Ayurveda is one of the oldest existing health systems of the world with underlying principles and theoretical practices.

Sanskrit word Ayurveda is science of life. Because, Ayurveda is based on the six darsanas, mainly the logic of the Samkhya and Nyaya-Vaiseikan philosophies.

Acacia Farnesiana L.Wild (synonym *Acacia farnesiana*)cultivated in India, it is native to India. In tropical Burma, Sri Lanka, Saudi Arabia, Egypt and in Africa. In India, it is mostly found in Maharashtra, Gujarat, Andhra Pradesh and Karnataka. Local people use it for various treatments such as cold and cough. Ayurveda has proven relatively effective in the treatment of skin diseases.

Shodhana and Shamana are two important therapies that can be used in these conditions. For local elimination of aggravated doshas, Lepa is considered the best treatment. Therefore, alder was chosen as the treatment method. In, Pitta and Rakta are vitiated doshas and improve

skin color which cannot be considered in treatment. India is widely spread across the country due to its rich flora.

Plant Description

- **Synonyms:** Sweet acacia, Vachellia Farnesiana
- **Biological origin**: Comprising of shrubs and small trees of *Acacia farnesiana* from the Leguminosae family
- **Geographical origin**:*Acacia farnesiana* is native to India, specifically in Tropical Burma, Sri Lanka, Saudi Arabia, Egypt, and Africa. In India, it is predominantly found in Maharashtra, Gujarat, Andhra Pradesh, and Karnataka.
- **Description**:*Acacia farnesiana* is characterized as a spreading shrub typically 1.5-4m tall, with smooth or finely fissured grey-brown bark. The leaves have petioles 0.2-2 cm long, are hairy especially on top, with a circular to elongated gland, occasionally with a sugary gland apex, and lack interjugary glands. The plant bears globose heads with 33-95 flowers, yellow or orange in color, located in 1-3 axils of leaves, with mostly 3-30mm long hairy peduncles. The pods resemble cigars, straight to strongly curved, terete, turgid, 1.5-8.5 cm long, 8-17 mm wide, dark brown to blackish, glabrous, with transverse or oblique seeds separated by pith.
- **Chemical components**: Pods contain a variety of polyphenols such as gallic acid, ellagic acid, m-digallic acid, methyl gallate, kaempferol, aromadendrin, and naringenin. Additionally, the seeds of A. farnesiana are a rich source of amino acids including lysine, arginine, glycine, and histidine.

Medical benefits of *Acacia Farnesiana* Plant

- **Anti-Inflammatory / Cytotoxicity**: A study revealed the presence of four new diterpenes–acasiane B, farnesirana A, farnesirana B along with three known diterpenes and eight flavonoids. Some of these compounds displayed cytotoxicity to human cancer cell lines while others exhibited moderate anti-inflammatory activity.
- ***Vibrio cholera* inhibition**: Research on 32 medicinal plants demonstrated that the ethanolic extracts of A. farnesiana and Artemisia ludoviciana effectively inhibited the growth of Cholera vibrio strains. The effects on enterotoxin production and adhesion were also investigated.
- **Antihyperglycemic Activity**: A study was conducted to assess the anti-hyperglycemic activity of an active fraction derived from an aqueous extract in alloxan induced diabetic rats. The results demonstrated promising anti-diabetic effects, with no noticeable toxic symptoms observed in the active fraction. Another study evaluated the anti-hyperglycemic activity of *Acacia farnesiana* extracts, and it was found that a water extract significantly reduced blood glucose levels. The activity was primarily observed in the soluble fraction, suggesting a direct stimulatory effect on glucose uptake without the involvement of insulin, which could be the main mechanism.
- **Antiulcer / Adsorbent**: The ulcer healing activity of Acacia farnersiana methanol leaf extract was evaluated in a rat model of ulcer induction. The results showed a significant reduction in the ulcer index compared to the control group treated with Ranitidine, indicating the potential of the methanol extract in promoting ulcer healing.
- **Bronchodilator / Anti-Inflammatory**: A study investigated the effects of a glycosidal fraction obtained from unripe pods of *Acacia farnesiana* on smooth muscle relaxation and inflammation. The results revealed a direct relaxant effect on bronchial muscles and inhibition of carrageenan and formaldehyde-induced inflammation, indicating its potential as a bronchodilator and anti-inflammatory agent.
- **Antioxidant / Protection against Oxidative Induced Damage**: The antioxidant properties of Acacia pods extracts, specifically Acacia Schaffer and *Acacia*

farnesiana, suggest that the antioxidant components present in these pods can transfer to animal products such as milk, meal, and by-products when included in their diet. This transfer of antioxidants can provide protective effects against oxidative-induced damage.

- **Antibacterial / Antioxidant / Anti-Inflammatory**: In a study evaluating ethanolic extracts of five plants, including *Acacia farnesiana*, S. alata, S. grand flora, S. cumini, and T. divaricata, all extracts exhibited antioxidant and antibacterial activity. Furthermore, these extracts demonstrated anti-inflammatory effects by reducing interleukin (IL)-6 secretion and/or tumor necrosis factor (TNF)-a production.(6)
- **Study about Inflammation:** Inflammation is the body's biological reaction to harmful stimuli, including pathogens, damaged cells, or irritants. This response entails alterations in blood flow, accumulation of fluids, and migration of white blood cells to the site of injury or infection. The primary objective of inflammation is to eradicate the source of damage and initiate the process of healing. It can manifest as acute, lasting a short period of time, or chronic, persisting for months or even years. The effects of inflammation can be either advantageous or detrimental, contingent upon the cause, duration, and intensity of the response.
    - **Causes:**Various factors can trigger inflammation, including microorganisms, physical agents, chemicals, inappropriate immunological responses, and tissue death. Infectious agents like viruses and bacteria are common culprits of inflammation. Viruses cause inflammation by invading and destroying cells, while bacteria release endotoxins that kick start the inflammatory process. Physical trauma, burns, radiation injury, and frostbite can also lead to tissue damage and subsequent inflammation, as can corrosive chemicals like acids, alkalis, and oxidizing agents. Dysfunctional immunological responses can provoke an improper and harmful inflammatory reaction. Additionally, inflammation can occur when tissues perish due to a lack of oxygen or nutrients, often due to reduced blood flow to the affected area
    - **Signs**: The classic signs of inflammation—redness (rubor), heat (calor), swelling (tumor), and pain (dolor)—were first described by the Roman medical writer Aulus Cornelius Celsus in the 1st century AD. Redness results from the widening of small blood vessels near the injury site. Heat is a consequence of increased blood flow in the area and is typically felt in peripheral body parts like the skin. Fever is induced by inflammatory chemical mediators and contributes to the elevated temperature at the site of injury. Swelling, known as edema, is primarily caused by fluid accumulation outside blood vessels. The pain associated with inflammation is partly due to tissue distortion caused by edema and is also triggered by specific inflammatory chemical mediators like bradykinin, serotonin, and prostaglandins.The initial inflammatory response involves vascular changes. Upon tissue injury, the affected blood vessels undergo a brief constriction known as vasoconstriction. Subsequently, the blood vessels dilate, a process called vasodilation, which leads to an increased blood flow into the injured area. This vasodilation can persist for a duration ranging from 15 minutes to several hours.
- **Cellular alterations**: Inflammation's key characteristic is the gathering of white blood cells at the injury site. The majority of these cells are phagocytes, a specific type of leukocytes that consume bacteria and other foreign particles, as well as clear cellular debris caused by the injury. Neutrophils, a type of white blood cell containing granules of cell-destroying enzymes and proteins, are the primary phagocytes involved in acute inflammation. In cases of minor tissue damage, a sufficient number

of these cells can be obtained from those already present in the bloodstream. However, in cases of extensive damage, stores of neutrophils—some in an immature state—are released from the bone marrow, where they are produced.

For neutrophils to carry out their functions, they must not only exit through the blood vessel wall but also actively move from the blood vessel towards the damaged tissue. This movement is facilitated by chemical substances that diffuse from the damaged area, creating a concentration gradient that the neutrophils follow. These substances, known as chemotactic factors, induce the one-way migration of cells along the gradient, a process called chemotaxis.A large number of neutrophils typically arrive at the injury site first, sometimes within an hour of the injury or infection. Following the neutrophils, another group of white blood cells, the monocytes, appear around 24 to 28 hours after the onset of inflammation. These monocytes eventually develop into macrophages, which are known for their ability to consume cells. Macrophages usually become more prominent at the injury site only after several days or weeks and are a characteristic feature of chronic inflammation.

- **Chemical mediators of inflammation**: While injury initiates the inflammatory response, chemical factors released in response to this stimulation lead to the vascular and cellular changes described above. These chemicals primarily originate from blood plasma, white blood cells (such as basophils, neutrophils, monocytes, and macrophages), platelets, mast cells, endothelial cells lining the blood vessels, and more.

Histamine is a well-known chemical mediator that is released from cells during inflammation. It plays a role in triggering vasodilation and increasing vascular permeability. Histamine is stored in granules found in circulating basophils and mast cells, and it is released immediately when these cells are injured. In addition to histamine, other substances such as lysosomal compounds released from neutrophils, as well as certain small proteins in the complement system like C3a and C5a, also contribute to increasing vascular permeability. Furthermore, many cytokines secreted by cells involved in inflammation possess vasoactive and chemotactic properties.

Prostaglandins, on the other hand, are a group of fatty acids that are produced by various types of cells. Some prostaglandins enhance the effects of other substances that promote vascular permeability, while others impact the aggregation of platelets, which is a crucial part of the clotting process. Prostaglandins are associated with the pain and fever experienced during inflammation. Anti-inflammatory drugs, such as aspirin, are effective in part because they inhibit an enzyme involved in prostaglandin synthesis. It is worth noting that prostaglandins are synthesized from arachidonic acid, just like the vasoactive leukotriene's, which are another group of chemical mediators.

The plasma consists of four interconnected protein systems: complement, kinins, coagulation factors, and the fibrinolytic system. These systems work together to produce various mediators of inflammation. Activated complement proteins act as chemotactic factors for neutrophils, increase the permeability of blood vessels, and trigger the release of histamine from mast cells. They also bind to the surface of bacteria, making them more susceptible to phagocytes. The kinin system, activated by coagulation factor XII, generates substances that further increase vascular permeability. One of the most important kinins is bradykinin, which is responsible for much of the pain and itching associated with inflammation. The coagulation system converts fibrinogen, a plasma protein, into fibrin, a major component of the fluid exudate. The fibrinolytic system contributes to inflammation primarily by producing plasmin, which breaks down fibrin into products that affect vascular permeability.

After the onset of acute inflammation, several outcomes can occur. These include healing and repair, suppuration, and chronic inflammation. The specific outcome depends on the type of

tissue involved and the extent of tissue damage, which are influenced by the underlying cause of the injury.

MATERIALS AND EQUIPMENTS[12-14]

Material for Animal Trial

1. **Collection of Plant Material:**Bark of *Acacia farnesiana* L. Wild locally known as Devbabul were collected from Malegoan Baramati, Pune district, Maharashtra, during Augustan authenticated voucher specimen deposited in the department of botany Sharadabai Pawar Mahila College Baramati.
2. **Preparation ofExtract:***Acacia farnesiana* bark was cleaned properly to remove soil and dirt. The bark was shade dried and then coarsely powdered. The powder was successively extracted with petroleum ether (60- 80 ºC), methanol and distilled water by cold maceration method. These extracts were concentrated at reduced temperature and pressure using rotary evaporator and completely freed of solvents. Yields of petroleum ether, methanol and water extracts were 0.94 and4.38 and 2.54 % w/w, respectively.

1. **Standard Operating Procedure:**

A. Heating Mantle:

- Switch ON the Power button
- Set up flask and condenser as required.
- Connect hose to tap and turn on to give a gentle flow of water
- Switch on heating mantle and set to required temperature setting. Monitor temperature.
- Do not use a mercury thermometer.
- Place HOT warning sign near the heating mantle.
- Monitor system during heating procedure.
- When procedure complete, carefully remove glassware, using heat proof gloves.
- Switch off heating mantle and leave HOT warning sign in place until everything is cool.

B. Sieve Shaker:

- Switch “ON” the mains.
- Set the time interval and amplitude.
- Open the clamping device.
- Place the sieve stack on the sieving platform. Load the sample. Clamp the top cover with the help of a screw.
- Press starts. The shaker starts sieving until the set time.
- Once the time is over, it will stop and make a beep sound 3 times.
- During sieving, the amplitude, time, and interval time can vary.
- Once the sieving is completed, unscrew the clamp to remove it.
- Take out the sieves.
- Turn “OFF” the mains.

C. Rotary Evaporator:

- Preparation:
  - Familiarize yourself with the equipment.
  - Prepare your sample and solvent.
- Setup:
  - Set up the rotary evaporator by connecting glassware and ensuring a water reservoir
  - Fill the round-bottomed flask with the solution to be evaporated.
  - Use a Keck clip to connect the flask to the bump trap securely.
- Initiate Evaporation:

- Lower the flask into the water bath using the joystick knob, ensuring it is not too low.
- Turn on the vacuum source to create a partial vacuum that holds the flask securely.
- Adjust the rotation notch to rotate the flask at a medium rate.

- Monitoring and Completion:
  - Close the stopcock to stop evaporation gradually.
  - Allow the solvent to evaporate, collecting in the flask.
  - Keep the sample at reduced pressure for thorough solvent removal.
  - Reverse the steps to stop evaporation and collect the final residue.

OBSERVATION AND RESULTS

Observations: Animals were observed individually for first 30, 60, 120, 180 and 240 minutes after dosing, with special attention and once daily thereafter, for a total of 14 days. However, the duration of observation was not fixed rigidly and was determined by the toxic reactions and time of onset and length of recovery period. All observations of toxic signs were systematically recorded for each animal in the daily observation record format.

Clinical Signs and Symptoms

All animals were observed for the following signs-

Changes in skin, fur, eyes, mucous membranes, respiratory, circulatory, autonomic and central nervous systems, somatomotor activity, behaviour pattern. Attention should be directed to the observations of tremors, convulsions, salivation, diarrhoea, lethargy, sleep and coma.

Body Weight

The body weights were recorded on test day 0 (pre-administration - fasting weight) and on days 7 and 14 post treatment or at death.

Necropsy and Pathology

All animals were subjected to gross necropsy. In gross necropsy the animals were observed at all the body openings, opened up and observed it with naked eye for any alterations in normal body organs. At this point major organs like liver, lungs, ovaries, kidneys, adrenal gland, spleen, pancreas, heart, brain etc. were observed.

- ✓ **Body weight**

Normal Body weight gain was observed during 14 days observation period and there were no any signs of toxicity considering weight gain.

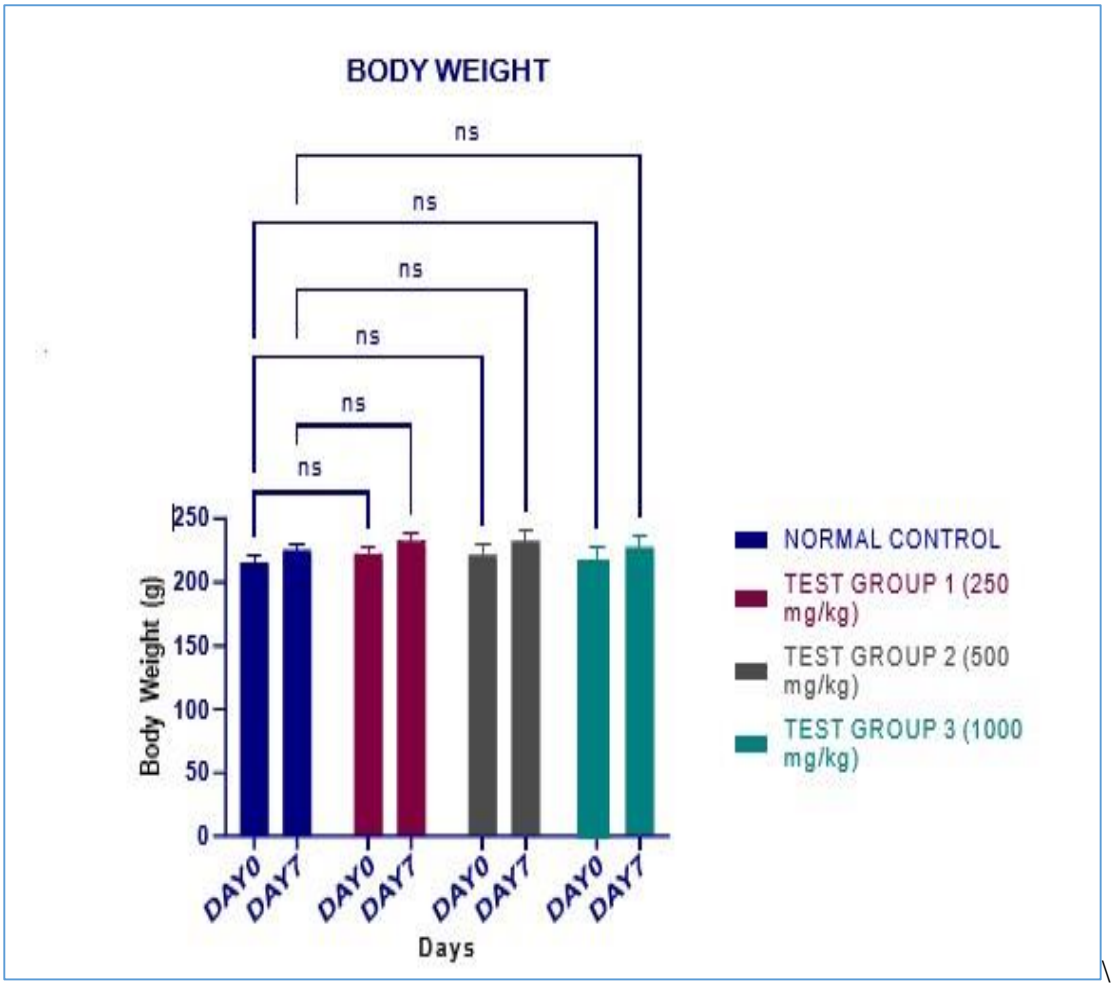

Fig 6. Body Weight

Values are expressed as mean ± SD, n=6, *p<0.05, **p<0.01, *** p<0.001, **** p<0.0001. Values of Groups were compared with Normal Control by t-test. Values of test groups were compared with lowest conc. by Two-way ANOVA by Dunnett's test.

Table 3. Body weight

Groups	**Day 0**	**Day 7**
Normalcontrol	215.667±5.465	225.833±4.708
Testgroup1(250mg/kg)	222.833±5.345	233.333 ±5.680
Testgroup2(500mg/kg)	221.83 ±8.57	233.00 ±7.87
Testgroup3(1000mg/kg)	218.167 ±8.658	228.333 ±7.448

✓ **Clinical signs and symptoms**

1) CRP

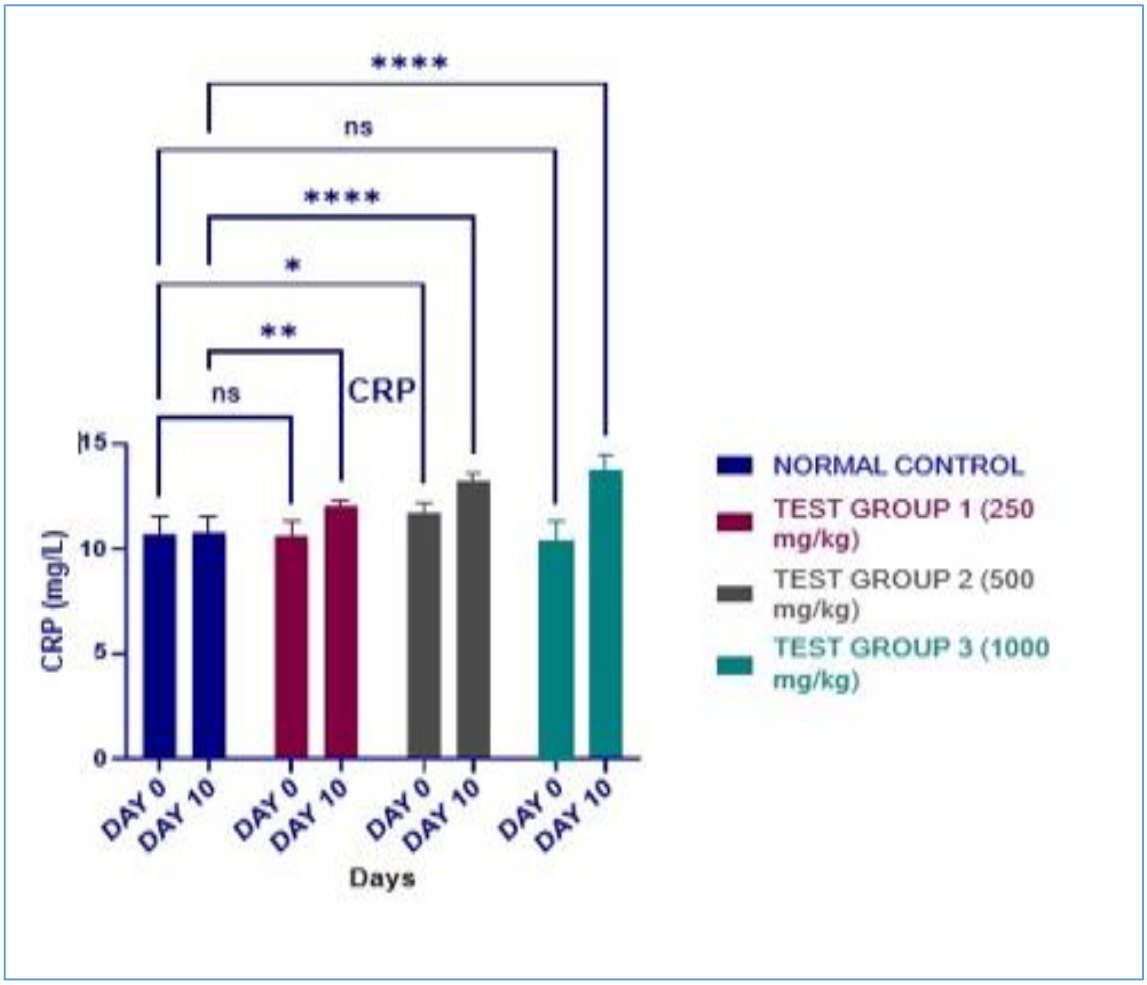

Fig 7. Prism data of CRP

Values are expressed as mean ± SD, n=6, *p<0.05, **p<0.01, *** p<0.001, **** p<0.0001. Groups were compared with Normal Control by t-test. Values of test groups were compared with lowest conc. by Two-way ANOVA by Dunnett's test.

Table 4. CRP

Groups	**Day 0**	**Day10**
Normalcontrol	10.697±0.862	10.785±0.744
Testgroup1(250mg/kg)	10.615±0.662	**12.068 ±0.282
Testgroup2(500mg/kg)	*11.732 ±0.44	****13.268 ±0.36
Testgroup3(1000mg/kg)	10.803±0.492	****13.767 ±0.700

2) IL 6

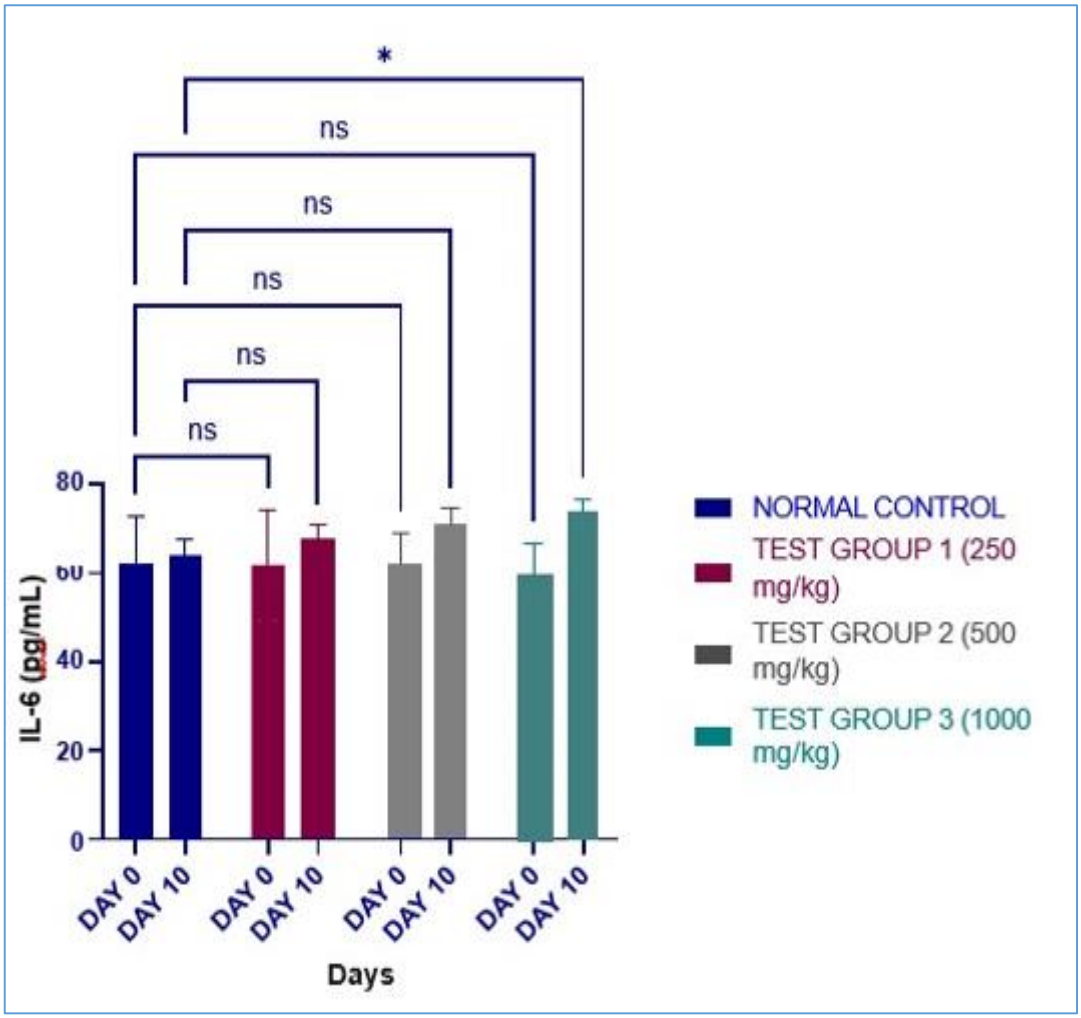

Fig 8. IL 6

Values are expressed as mean ± SD, n=6, *p<0.05, **p<0.01, *** p<0.001, **** p<0.0001. Groups were compared with Normal Control by t-test. Values of test groups were compared with lowest conc. by Two-way ANOVA by Dunnett's test.

Table 5. IL 6

Groups	**Day 0**	**Day10**
Normalcontrol	62.333 ±10.541	64.000 ±3.496
Testgroup1(250mg/kg)	61.778±12.546	67.889±2.722
Testgroup2(500mg/kg)	65.111 ±3.90	72.333 ±2.11
Testgroup3(1000mg/kg)	62.889±3.897	*75.111±1.361

3) IL-8

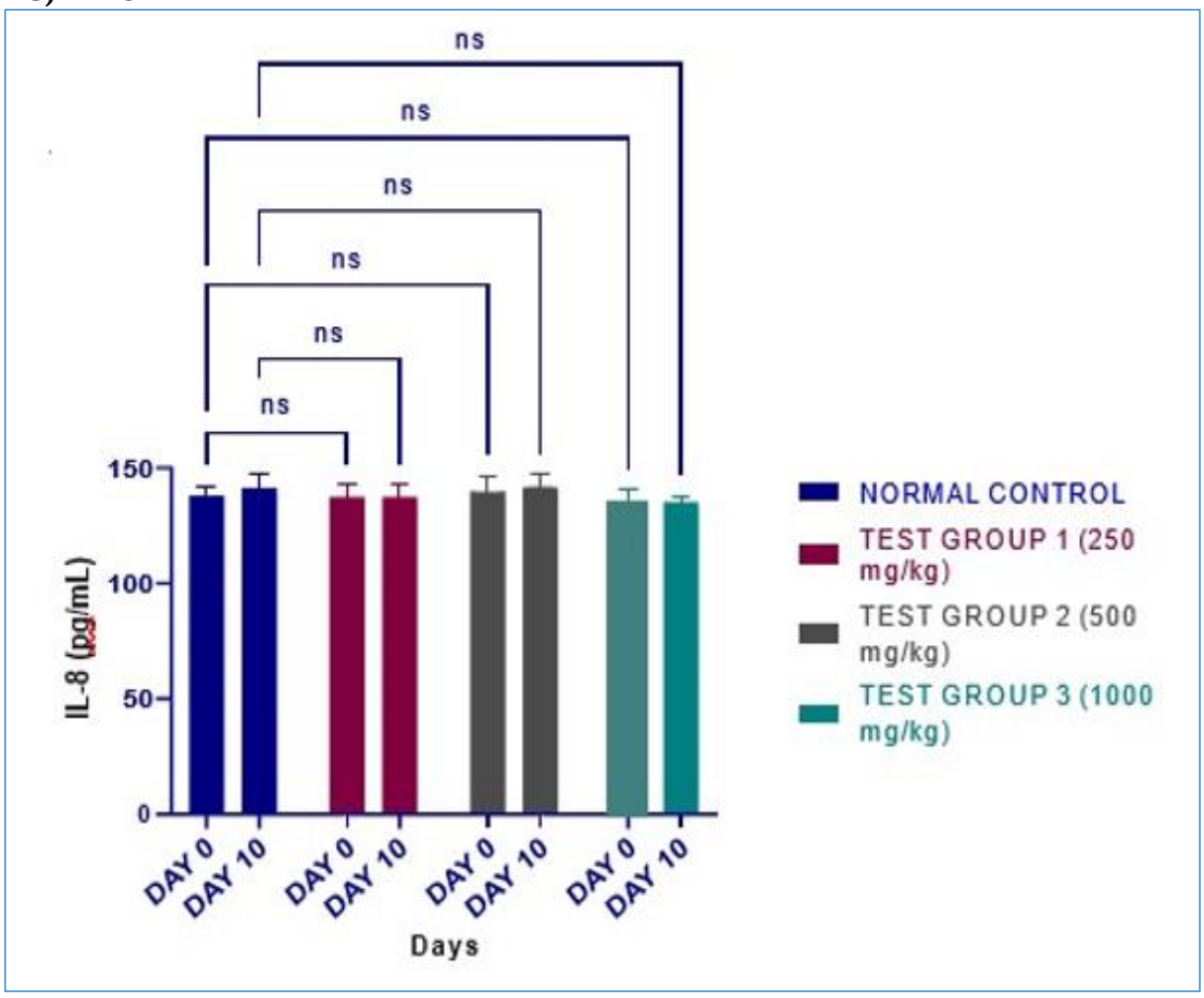

Fig 9. IL-8

Values are expressed as mean ± SD, n=6, *p<0.05, **p<0.01, *** p<0.001, **** p<0.0001. Groups were compared with Normal Control by t-test. Values of test groups were compared with lowest conc. by Two-way ANOVA by Dunnett's test.

Table 6. IL 8

Groups	**Day 0**	**Day10**
Normalcontrol	138.208 ±3.862	141.274±6.351
Testgroup1(250mg/kg)	137.583 ±4.878	137.613±4.910
Testgroup2(500mg/kg)	139.786±6.75	141.810±5.06
Testgroup3(1000mg/kg)	137.375 ±3.103	135.202±2.109

DISCUSSION

The anti-diarrheal effects of *Acacia farnesiana* have been extensively studied and have shown promising results. First, the effect of *Acacia farnesiana* on body weight was verified. Changes in animal body weight were observed weekly between acclimation day and the day of treatment in experimental mice.

The body weight of test animals in the Normal Control, Disease Control, Standard, and Experimental groups increased throughout the study period. Second - Clinical signs and symptoms were checked. Daily observations recorded no changes in skin, hair, eyes, mucous membranes, respiratory, circulatory, autonomic and central nervous systems, physical activity, activity on behavioral patterns judged to be abnormal as animals were weak and lethargic.

There were no signs of tremors, convulsions, salivation and diarrhoea. Third bowel mass unverified. Compared with the disease control group, total stool weight after 4 hours decreased in the test group and the standard group. The disease control group gained a total weight of stools after castor oil induction.

CONCLUSION

Acacia farnesiana was assessed for acute oral toxicity in Wistar rats according to OECD 423 guidelines. No clinical pathogenesis or mortality was observed, indicating its safety under laboratory conditions. In the carrageenan-induced paw edema model, significant anti-inflammatory activity was demonstrated. The test group showed 58.79% inhibition at ½ hour, 55.72% inhibition at 1 hour, and 58.08% inhibition at 2 hours. Moreover, at 12 hours, the percentage inhibition of acute inflammation was greater in the test group compared to the standard drug-treated animals.

Acacia farnesiana exhibited significant anti-inflammatory activity compared to the disease control group, with a reduction in paw edema observed at ½ hr, 1 hr, 2 hr, 4 hr, 8 hr, and 12 hr post-carrageenan induction. In the castor oil-induced diarrhoea model, *Acacia farnesiana* demonstrated significant anti-diarrheal activity. After 4 hours, the test group showed 82.88% inhibition of diarrhoea by weight and 13.21% inhibition by volume. Total fecal weight was reduced in both the test and standard groups compared to the disease control group, indicating the effectiveness of *Acacia farnesiana* in alleviating diarrhoea.

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