Anti-diarrhoeal activity of *Abutilon indicum* Linn leaf extracts

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Abstract

**Objective:** The present study is aimed to evaluate the leaf extracts of *Abutilon indicum* for acclaimed anti-diarrhoeal activity using albino rats. **Materials and methods:** Anti-diarrhoeal activity of *Abutilon indicum* was evaluated by gastrointestinal motility, castor oil-induced diarrhoea and prostaglandin E₂-induced enteropooling in rats. Loperamide was used as a standard drug. **Results:** The study revealed that, the methanolic extract and aqueous extract possessed significant antidiarrhoeal activity in castor oil-induced diarrhoea and prostaglandin E₂-induced diarrhoea, compared to the control group. **Conclusion:** *Abutilon indicum* showed significant anti-diarrhoeal activity as compared to loperamide and can be recommended for further studies.  

**Key words:** *Abutilon indicum*, Loperamide, Antidiarrhoeal activity, Albino rats.

1. Introduction

*Abutilon indicum* linn (malvaceae) is an herb, commonly found in the Indian Sub-continent and in tropical countries [1]. The different parts of this plant are used in traditional system of medicines as diuretic, antiulcer, demulcent and in pains, the vapour form of hot decoction is used in chronic form of dysentery and diarrhoea [2]. It was reported that, this plant possess antimicrobial (3), antifertility (4) and analgesic activity (5). The aim of the present study is to screen the leaf extracts of *Abutilon indicum* for antidiarrhoeal activity in albino rats.

2. Materials and methods

2.1 Plant material

The leaves of *Abutilon indicum* were collected from the region of Harapanhalli, Davanagere (District), Karnataka, in the month of December. The plant was authenticated by the Botanist and a voucher specimen of the plant was kept in the

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The dried leaves of *Abutilon indicum* were subjected to successive solvent extraction using petroleum ether (60-80°C), methanol and distilled water in a soxhlet extraction apparatus and concentrated to get a semisolid residue. All the extracts were vacuum dried and finally suspended in 1% w/v Tween-80 solution.

2.2 Drugs

Following drugs were chosen for the present study.

1. Castor oil (Paras chemical Industries) Kalol, Gujarat.
2. Loperamide (Torrent Pharmaceuticals Ltd., Ahmedabad).
5. Prostaglandin E$_2$ (Astra-IDL Ltd., Bangalore, India).

2.3 Experimental animals

Wistar albino rats of either sex (150-250 g) were obtained from the animal house of Indian Institute of Science, Bangalore. They were fed with synthetic diet viz., Gold Mohur (Lipton India Ltd., Bangalore) and water was given *ad libitum*.

2.4. Intestinal motility test

Animals were starved 24 h prior to the experiment with free access to water and placed in five cages containing six in each as shown in Table-1. They were given the extract or vehicle or Atropine sulphate 5 mg/kg (i.m.). Subsequently, after 30 min, each individual rat was administered 1 ml of charcoal meal (5% activated charcoal in 10% aqueous tragacanth suspension) by oral route. These animals were sacrificed after 30 min and the abdomen was opened. The movement of charcoal meal in small intestine from pylorus was measured and it is expressed as a percentage of distance movement from pylorus to caecum [6].

2.5 Castor oil-induced diarrhoea

The method of Awouters *et al.* [7] was used. The rat were divided into five group each containing six as shown in Table-2. The extract or vehicle or Loperamide (1 mg/kg) was administered. Loperamide was used as a standard drug. Castor oil (10 ml/kg) was administered orally after 30 min and each rat was then housed in the cages, each provided with a clean filter paper at the bottom. These animals were observed for the characteristic stool and time of onset of diarrhoeal episodes. The observations were recorded every hour up to six hours.

2.6 Prostaglandin E$_2$-induced enteropooling

The method of Pulok K. Mukerjee *et al* was used [8]. In this method, animals were deprived of food and water for 18 h and six animals were placed in each perforated cage. The first and second groups were treated with 1 ml of 5% v/v ethanol in normal saline (i.p). The first group was treated with Tween-80 (1% w/v), which served as control.

The third, fourth and fifth group were treated with extracts. Prostaglandin E$_2$ (100 µg/kg) was administered orally to each rat in 5% v/v ethanol in normal saline after the above said treatment. The animals were sacrificed after 30 min and the whole length of the intestine from the pylorus to the caecum was dissected out and contents were collected in a test tube and volume was measured.

2.7 Statistical Analysis

The determination for the significant intergroup difference, each parameter was analysed separately and Student’s *t*- test was used for comparison (P<0.05).
3. Results

The standard anti-muscarinic drug, atropine sulphate, methanol extract and aqueous extract showed significant decrease in the propulsion of the charcoal meal through the gastrointestinal tract, as compared to the control group (Table-1). The petroleum ether extract did not show any significant intestinal motility.

In castor oil-induced diarrhoea the methanolic extract, aqueous extract and loperamide showed significant reduction in diarrhoeal episodes (Table-2). The petroleum ether extract did not show any significant activity.

In Prostaglandin E₂-induced enteropooling, the methanolic extract and aqueous extract showed significant decrease in the fluid volume of rat intestine as compared to control group (Table-3). The pertroleum extract did not show any significant decrease in the fluid volume of rat intestine.

4. Discussion

There has been a statistically significant reduction in the incident and severity of diarrhoea produced in experimental models. Abutilon indicum leaf extracts, like the standard antidiarrhoeal agent Loperamide, inhibited significantly the frequency of defecation, faecal droppings, when compared with untreated control rats.

The antimuscarinic drug atropine, methanolic and aqueous extract of A.indicum decreased intestinal propulsive movement in charcoal meal treated animal models. The mechanism for this inhibition of motility may be due to the

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Group</th>
<th>Dose (mg/kg)</th>
<th>Mean% of movement of charcoal ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Control (Tween-80)</td>
<td>1% w/v</td>
<td>86.73± 1.05</td>
</tr>
<tr>
<td>2</td>
<td>Atropine sulphate</td>
<td>5</td>
<td>37.65 ± 1.70*</td>
</tr>
<tr>
<td>3</td>
<td>Petroleum ether extract</td>
<td>500</td>
<td>86.39± 3.78</td>
</tr>
<tr>
<td>4</td>
<td>Methanolic extract</td>
<td>500</td>
<td>42.33 ± 3.83*</td>
</tr>
<tr>
<td>5</td>
<td>Aqueous extract</td>
<td>500</td>
<td>45.13 ± 3.67*</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SEM; * P<0.001 vs respective control group; n=6.

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Group</th>
<th>Dose (mg/kg)</th>
<th>Mean weight of defecation ± SEM after 6 h (gms/kg body weight)</th>
</tr>
</thead>
<tbody>
<tr>
<td>01</td>
<td>Control (Tween-80)</td>
<td>1% w/v</td>
<td>23.52 ± 0.34</td>
</tr>
<tr>
<td>02</td>
<td>Loperamide</td>
<td>1</td>
<td>3.86 ± 0.27*</td>
</tr>
<tr>
<td>03</td>
<td>Petroleum ether extract</td>
<td>500</td>
<td>21.93 ± 0.73</td>
</tr>
<tr>
<td>04</td>
<td>Methanolic extract</td>
<td>500</td>
<td>10.25 ± 0.22*</td>
</tr>
<tr>
<td>05</td>
<td>Aqueous extract</td>
<td>500</td>
<td>15.38 ± 0.53*</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SEM; * P<0.001 vs respective control group; n=6.
nonspecific spasmolytic activity of *A.indicum*. Similarly *A.indicum* inhibit significantly PGE$_2$ induced enteropooling. These observations suggest that *A.indicum* reduced diarrhoea by inhibiting intestinal peristalsis, gastrointestinal motility and PGE$_2$ induced enteropooling.

Prostaglandins contribute to the pathophysiological functions in the gastrointestinal tract [9]. Castor oil and arachidonic acid increase peristaltic activity and produce permeability changes in the intestinal mucus membranes to electrolyte and water, effects associated with prostaglandin release [10, 11]. Thus a component of the antidiarrhoeal activity of the *A.indicum* leaf extracts may be due to not only the inhibition of prostaglandin synthesis and release, but also to its actions, as it showed in activity against prostaglandin E$_2$ induced diarrhoea.

Preliminary Phytochemical investigation of *A.indicum* leaf revealed the presence flavonoids. [12]. Flavonoids play an important role in inhibition of prostaglandin E$_2$ that may be responsible for the different therapeutic activities of this plant [13]. Flavonoids inhibit several enzymes, including those involved in arachidonic acid metabolism [14, 15]. There is a need to pursue the characterization of active principles, to optimize the observed activities.

Table 3.
Effect of *Abutilon indicum* Linn leaf extracts on Prostaglandin E$_2$ - induced diarrhoea.

<table>
<thead>
<tr>
<th>Sl.No.</th>
<th>Group</th>
<th>Dose (mg/kg)</th>
<th>Mean volume of intestinal fluid in ml ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>01</td>
<td>Control (Tween-80)</td>
<td>1% w/v</td>
<td>0.73± 0.016</td>
</tr>
<tr>
<td>02</td>
<td>PGE$_2$ in ethanol</td>
<td>100 µg/kg</td>
<td>2.90 ± 0.106</td>
</tr>
<tr>
<td>03</td>
<td>Loperamide</td>
<td>1</td>
<td>1.58 ± 0.260*</td>
</tr>
<tr>
<td>04</td>
<td>Pet. ether extract</td>
<td>500</td>
<td>2.78 ± 0.094*</td>
</tr>
<tr>
<td>05</td>
<td>Methanolic extract</td>
<td>500</td>
<td>2.01± 0.164*</td>
</tr>
<tr>
<td>06</td>
<td>Aqueous extract</td>
<td>500</td>
<td>2.40 ± 0.08*</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SEM; * P<0.001 vs respective control group; n=6.

**References**


