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Evaluation of the antidiarrhoeal effect of *Sanseviera liberica* Gerome & Labroy (Agavaceae) root extract

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ABSTRACT

Ethnopharmacological relevance: The aqueous root extract of *Sanseviera liberica* (Agavaceae), SL, is used in Traditional African Medicine (TAM) for the treatment of diarrhoea. However, the scientific basis for this usage has not been established.
Aim of the study: To evaluate the antidiarrhoeal activity of SL using various pharmacological models.
Materials and methods: The intestinal transit, castor oil induced diarrhoea, enteropooling, and gastric emptying methods were used in this study.
Results: SL (25–400 mg/kg, p.o.) produced significant ($P < 0.05$) dose dependent reduction in propulsive movement in both the normal and castor oil induced intestinal transit tests in mice. Peak effect was elicited at 200 mg/kg but this effect was lower than that produced by morphine (10 mg/kg, s.c.). The effect of SL on castor oil induced intestinal transit was antagonized by isosorbide dinitrate, IDN (150 mg/kg, p.o.) but not by yohimbine (1 mg/kg, s.c.). In the castor oil induced diarrhoea test, SL significantly delayed the onset and decreased the frequency and severity of diarrhoea. The effect at 200 mg/kg was comparable to that of morphine and was reversed by IDN. SL at the dose of 200 mg/kg significantly reduced the volume of intestinal secretion induced by castor oil but produced no effect on gastric emptying.

The extract was practically nontoxic administered p.o. The LD_{50} was 631 mg/kg given i.p. Phytochemical analysis revealed the presence of oils, reducing sugars, alkaloids, saponins, anthraquinones, and tannins in the extract.
Conclusion: The results obtained in this study suggest that the aqueous root extract of *Sanseviera liberica* possesses antidiarrhoeal property due to inhibition of gastrointestinal propulsion and fluid secretion, possibly mediated through inhibition of the nitric oxide pathway. This justifies the use of the plant extract in TAM for the treatment of diarrhoea.

1. Introduction

Sanseviera liberica (Agavaceae) is a perennial plant with thick woody rhizomes. It is widely distributed in the tropical, subtropical and temperate zones of the world, commonly located in shady places near streams and rocky parts. It is found in different parts of Nigeria and ascribed local names include: mooda (Hausa), ebube – agu (Igbo), okonno (Efik) and oja ikoko (Yoruba). The common names of this plant include "Mother in-law tongue", "African bowstring" and "Leopard lily". In Traditional African Medicine (TAM), *Sanseviera liberica* is used for the treatment of ear and eye infections, inflammation (leaf juice); tooth ache (fruit juice together with fluid from snails); fever, headache and cold (fume from burning leaves inhaled); cough, pain, inflammation, infections, convulsion, diarrhoea and as stimulating tonic (root decoction) (Watt and Breyer-Brandwijk, 1962).

Diarrhoea is a common gastrointestinal disorder characterized by an increase in stool frequency and a change in stool consistency (Farthing, 2002). It remains one of the major health threats to populations in the tropical and subtropical poor countries. According to Heinrich et al. (2005), the World Health Organization (WHO) estimates that 3–5 billion cases occur each year (1 billion in children less than 5 years of age), and that approximately 5 million deaths are due to diarrhoea annually (2.5 million in children less than 5 years of age). A study by Martinez et al. (1998) which looked at the form of treatment that is administered by primary caretakers of young children including mothers showed that herbal remedies are still important in the treatment of diarrhoea.

In view of these facts, this study was conducted to investigate the antidiarrhoeal activity of the aqueous root extract of *Sanseviera liberica*. No report of such study was found in the course of literature search.

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2. Materials and methods

2.1. Plant material

The plant material was collected in the premises of the Lagos University Teaching Hospital (LUTH), Idi-Araba, Lagos, Nigeria. Botanical identification and authentication was done by Professor J.D. Olowokudejo of the Department of Botany, Faculty of Science, University of Lagos, Lagos, Nigeria, and Mr. T.K. Odewo, Senior Superintendent of the Forestry Research Institute of Nigeria (FRIN), Ibadan, Nigeria. A voucher specimen, with identification number FHI 107621, was deposited in the herbarium of the institute.

2.2. Preparation of plant extract

Two hundred grams of the dried root of the plant was washed and chopped into pieces. The plant material was boiled with 2 l of distilled water in a conical flask for 3 h. The liquid was decanted 24 h after and the resultant filtrate was evaporated to dryness in the oven (Griffin 2/250 FC) at 40 °C (Adeyemi et al., 2007; Amida et al., 2007). The yield of the dried extract obtained from the process was 24.01%. The dried extract was reconstituted in distilled water just before use on each day of the experiment. The extract was administered orally at doses of 25, 50, 100, 200, and 400 mg/kg based on results from the acute toxicity study and preliminary studies on pharmacological effect.

2.3. Animals

Albino mice (20–25 g) and rats (150–200 g) of both sexes obtained from the Laboratory Animal Center of the College of Medicine, University of Lagos, Lagos, Nigeria, were used for this study. The animals had free access to food (Livestock Feeds PLC) and water *ad libitum* and were maintained under standard environmental conditions. The experimental protocol was approved by the Experimentation Ethics Committee on Animal Use of the College of Medicine, University of Lagos, Lagos, Nigeria.

2.4. Acute toxicity test

Groups of mice of both sexes (5 per group) fasted for 12 h prior to the test were given SL orally at doses of 0.1, 1, 5, 10, and 20 g/kg. A different set of groups of animals received the extract at doses of 100, 200, 400, 800, and 1600 mg/kg intraperitoneally. Animals in each group were observed for any immediate signs of toxicity and mortality within 24 h. The LD_{50} was estimated by the log dose–probit analysis (Litchfield and Wilcoxon, 1949; Adeyemi et al., 2008). Surviving animals were observed for further 7 days for any signs of delayed toxicity.

2.5. Normal intestinal transit

This test was conducted according to the method of Hsu (1982). Mice were allotted to groups of six animals each. Treatment was then carried out as outlined below:

Group 1: Distilled water, 10 ml/kg, p.o.,
Group 2: SL, 25 mg/kg, p.o.,
Group 3: SL, 50 mg/kg, p.o.,
Group 4: SL, 100 mg/kg, p.o.,
Group 5: SL, 200 mg/kg, p.o.,
Group 6: SL, 400 mg/kg, p.o., and
Group 7: Morphine, 10 mg/kg, s.c.

Thirty minutes after, charcoal meal (10% charcoal in 5% gum acacia; 0.2 ml/mouse, p.o.) was administered to each animal. The mice were killed 30 min later and the small intestine was immediately isolated. The Peristaltic Index (PI), which is the distance traveled by the charcoal meal relative to the total length of small intestine expressed in %, was determined for each mouse (Aye-Than et al., 1989).

2.6. Castor oil induced intestinal transit

The same procedures as in the normal intestinal transit test were followed except that castor oil (0.2 ml/mouse, p.o.) was administered 30 min before administration of charcoal meal, i.e. 30 min post-treatment (Hsu, 1982; Aye-Than et al., 1989). Additional groups comprised sets of animals that received yohimbine (1 mg/kg, s.c.; Group 8) and isosorbide dinitrate, IDN (150 mg/kg, s.c.; Group 9) 15 min before SL (200 mg/kg; p.o.).

2.7. Castor oil induced diarrhoea

Groups of six mice each were treated as outlined below:

Group 1: Distilled water, 10 ml/kg, p.o.,
Group 2: SL, 25 mg/kg, p.o.,
Group 3: SL, 50 mg/kg, p.o.,
Group 4: SL, 100 mg/kg, p.o.,
Group 5: SL, 200 mg/kg, p.o.,
Group 6: SL, 400 mg/kg, p.o.,
Group 7: Morphine, 10 mg/kg, s.c., and
Group 8: IDN, 150 mg/kg, p.o. (given 30 min prior to the administration of SL, 200 mg/kg, p.o.).

Thirty minutes after, castor oil (0.2 ml/mouse) was administered to each mouse. The animals were then placed under separate glass funnels, with the floor lined with blotting paper, for observation for 4 h (Izzo et al., 1992). The following parameters were observed: onset of diarrhoea, number of wet faeces, total number of faecal output, total weight of wet faeces, and total weight of faecal output. A numerical score based on stool consistency was assigned: 1 (normal stool), 2 (semi-solid stool), and 3 (watery stool). The correspondent percentages and purging index were computed, the later by comparison with the control group. Using the method of Aye-Than et al. (1989), the in vivo antidiarrhoeal index ($ADI_{in\ vivo}$) was expressed according to the formula:

$$ADI_{in\ vivo} = \sqrt[3]{Dfreq \times Gmeq \times Pfreq}$$

where Dfreq is the delay in defaecation time or diarrhoea onset (in % of control), Gmeq is the gut meal travel reduction (in % of control), and Pfreq is the purging frequency, as number of stool reduction (in % of control).

2.8. Intestinal fluid accumulation

Following the method of Robert et al. (1976), rats divided into six animals per group were pretreated with distilled water (10 ml/kg, p.o.), and SL (100 and 200 mg/kg, p.o.). One hour after, the rats received castor oil (2 ml/rat) intragastrically. The animals were killed 1 h later and the small intestines were removed after ligation at the pyloric end and ileocaecal junction, respectively, and weighed. The contents of the intestine were then expelled into a graduated tube and the volume measured. The intestines were reweighed and the differences between the full and empty intestines were calculated.