

Short Communication

In vivo* activity of stem bark aqueous extract of *Khaya senegalensis* against *Trypanosoma brucei

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Aqueous extract of *Khaya senegalensis* A. Juss (Meliaceae) stem bark was used to treat trypanosomiasis in rats *in vivo* and changes in levels of aspartate transaminase (AST) and alanine transaminase (ALT) were studied. The treatment involved oral infusion of the crude extract at 60 mg/kg body weight (b.w) simultaneously with *Trypanosoma brucei* infection, and 60 and 100 mg/kg b.w infusion of the extract 3 days post infection (p.i). In all the rats treated with the extract, a significant decrease ($P < 0.05$) in parasitemia was recorded on day 6 p.i and there was also significant ($P < 0.05$) increase in the levels of AST and ALT when compared with rats that were neither infected nor given the infusion of the extract. It was concluded that orally infused *K. senegalensis* extract possessed *in vivo* activity against *T. brucei* but could not prevent the disease-induced liver damage.

Key words: *Khaya senegalensis*, *Trypanosoma brucei*, liver damage.

INTRODUCTION

Trypanosomiasis is an important protozoan disease of domestic animals and man in most parts of Africa (Igbokwe, 1989). The disease has been ranked high in the list of major problems facing mankind by World Health Organisation (Mhlanga, 1996). In Africa, trypanosomiasis is one of the major obstacles to livestock production whose eradication and control is principally based on chemotherapy and chemoprophylaxis that is challenged with problems comprising drug resistance, toxicity and expensive/limited drugs (Gutteridge, 1985). The need, therefore, to source for better alternatives becomes imperative. The use of herbal remedies in the treatment of trypanosomiasis holds a promising potential in that some ethnomedicinal plants used against the disease have been demonstrated to be potent trypanocides (Asuzu and Chineme, 1990; Nok et al., 1993; Nok, 2001).

Khaya senegalensis (Juss), a dry zone mahogany belonging to the family, Meliaceae, is highly reputed for its numerous medicinal uses and has been reported to be used indigenously in the treatment of trypanosomiasis

(Atawodi et al., 2001). The plant has also been reported to possess an *in vitro* antitrypanosomal activity (Wurochekke and Nok, 2004; Atawodi, 2005) but information on the *in vivo* action of the plant against the parasite and/or the possibility of the plant to prevent *T. brucei*-induced liver damage (Umar et al., 1999) is still lacking.

This study is, therefore, aimed at investigating the *in vivo* antitrypanosomal action of *K. senegalensis* extract. We also checked its ability to prevent *T. brucei*-induced liver damage to provide more information on its action.

MATERIALS AND METHODS

Sample collection

The stem bark of mature *K. senegalensis* was collected from three different locations in Ahmadu Bello University, Samaru Campus, Zaria and was identified at the Herbarium of Department of Biological Sciences, Ahmadu Bello University, Zaria. Its voucher specimen number is 900081. The bark of the plant was thoroughly washed and sun dried for about a week to a constant weight. The dried bark was pounded to a fine powder using pestle and mortar to a mesh size of about 60 and then stored in a dry container.

Experimental animals

A standard protocol was drawn up in accordance with the Good Laboratory Practice (GLP) regulations of the World Health Organi-

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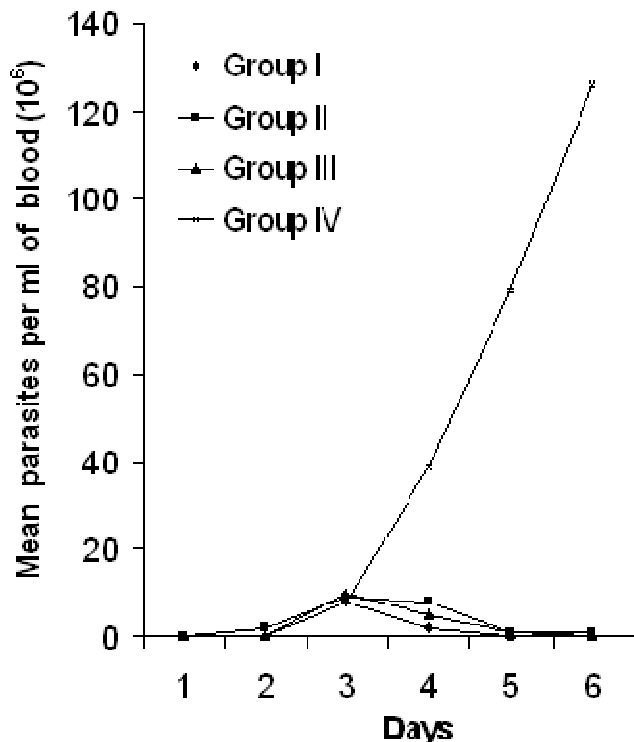


Figure 1. Effect of different oral doses of the aqueous extract of *K. senegalensis* stem bark on parasitemia.

sation (WHO Document, 1998). Apparently healthy white albino rats of both sexes weighing between 162-243 g were used for the work and were obtained from Department of Pharmacology, Faculty of Pharmaceutical Sciences, Ahmadu Bello University, Zaria, Nigeria. The animals were kept in well-ventilated laboratory cages with free access to commercial poultry feed (growers' mash), cassava flour (starch) and water ad libitum.

Sample preparation

Thirty grams of the fine powdered plant-part was soaked for 15 min in 500 ml of distilled water. It was then boiled for 15 min and allowed to cool and settle. The supernatant was then decanted and filtered through a thimble. The filtrate was evaporated to dryness with 0.32% yield and stored in a dark brown bottle at 4°C to avoid microbial growth.

Treatment of the experimental animals

Twenty-five rats were divided into five groups of 5 rats each. The rats in each group (except normal control) were intraperitoneally inoculated with about 10⁵ Bassa strain of *T. brucei* brucei from a donor rat obtained from Department of Parasitology, Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria. The level of parasitemia was monitored as described by Herbert and Lumsden (1976). The rats were then treated as follows: group I- daily oral treatment with 60 mg/kg b.w of the extract from day 1 p.i.; group II- daily oral treatment with 60 mg/kg b.w of the extract from day 3 p.i.; group III- daily oral treatment with 100 mg/kg b.w of the extract from day 3 p.i.; group IV- Infected and not treated (infected control); and group V: Uninfected and not treated (normal control).

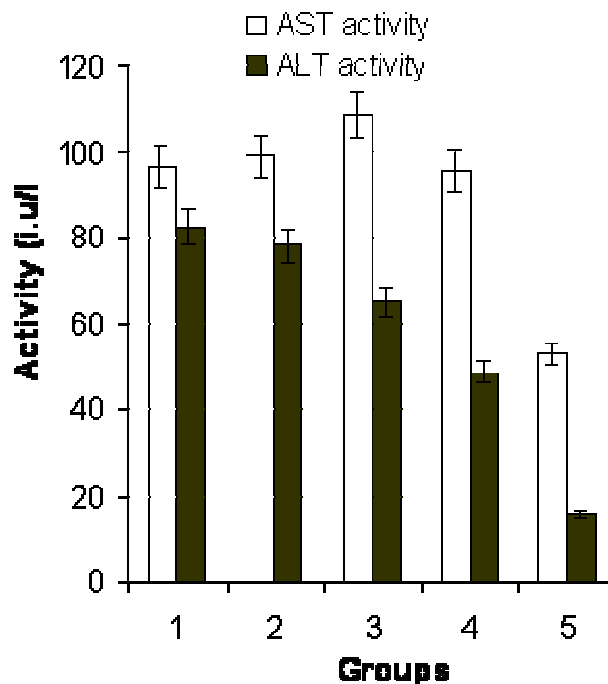


Figure 2. AST and ALT levels in *K.senegalensis* treated *T.brucei* infected rats.

All the rats were sacrificed after six days, blood was collected in dry centrifuge tubes and serum harvested.

AST and ALT assays

Serum aspartate transaminase (AST) and alanine transaminase (ALT) assays were carried out by the method of Reitman and Frankel (1957) using commercial reagent kits (Randox Laboratories, United Kingdom).

Statistical analysis

The results are presented as mean \pm standard deviation and Student's t-test was used to analyze the results.

RESULTS AND DISCUSSION

The parasitemia of both the infected control and the treated groups are presented in Figure 1. The treatments in groups II and III began on day 3 p.i. and a decrease in parasitemia in groups I, II and III on day 4 p.i. was observed. The parasites totally disappeared from the bloodstream on day 5 p.i in group I and significantly decreased ($P < 0.05$) in groups II and III. There was a progressive increase in parasitemia in the infected controls up to day 6 p.i when they eventually died (Figure 1).

Figure 2 presents the results of AST and ALT activities analyzed in the experiment. The *T. brucei* had increased

the AST activity from 53.00 ± 3.66 i.u/l in the normal controls to 96.33 ± 1.50 i.u/l in the infected controls. However, the activity was higher in the treated groups in a dose dependent fashion. The ALT activity in all the treated groups was higher than both the infected and the normal controls (Figure 2).

In the present study, *K. senegalensis*, a commonly used plant in the treatment/management of trypanosomiasis, has been shown to possess an *in vivo* antitrypanosomal activity. Other plants have also been reported to possess *in vivo* activities against *T. brucei* (Asuzu and Chineme, 1990; Nok et al., 1993). Although, the exact mechanism for the *in vivo* antitrypanosomal activity observed is not known, it is not surprising that the *K. senegalensis* extract may contain some phytochemicals that can interfere with the survival of the parasites *in vivo*. Nok (2001) reported an *in vivo* trypanocidal activity of azaanthraquinone, a flavonoid, isolated from a plant part. The observation of a faster antitrypanosomal action in the group of rats administered simultaneous infusion of the extract 60 mg/kg b.w. and infection with *T. brucei* than the groups treated 3 days p.i., suggests that the longer the parasites stay in the body of an infected host the longer it takes to be treated with the *K. senegalensis* extract and vice-versa. This might be as a result of the extract action being more effective when the parasites have not been fully established in the host.

T. brucei infection of the rats caused significantly ($P < 0.05$) increased serum levels of AST and ALT which supports earlier reports (Umar et al., 1999, 2007). Increases in the levels of these enzymes are indications of liver damage (Kaplan et al., 1988). That the AST and ALT levels in the *K. senegalensis*-treated groups were not lower than that of the infected untreated group suggest that the *K. senegalensis* extract could not reverse the *T. brucei* induced liver damage, despite its *in vivo* activity against the parasites. This could be attributed to free radicals generated by the parasites (Igbokwe, 1994) before the action of the extract.

It could therefore be deduced that *K. senegalensis* extract was effective in reducing the numbers of *T. brucei* in the bloodstream of infected animals but could not prevent the *T. brucei* induced liver damage.

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