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Effect of *Scoparia dulcis* on *Trypanosoma brucei* Induced Alterations in Serum Transaminase, Alkaline Phosphatase and Bilirubin in the Rabbit

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The present study reported the effect of *S. dulcis* on Trypanosome induced alterations in serum transaminases, Alkaline phosphatase and bilirubin in the rabbit. There were significant increases in Alkaline Phosphatase (ALP), Serum Glutamic Oxaloacetic Transaminase (SGOT), Serum Glutamic-Pyruvic Transaminase (SGPT), total bilirubin (T. Bil.) and conjugated bilirubin (C. Bil.) in infected animals relative to control. The values obtained for infected animals that were treated with *Scoparia dulcis* at a daily oral dose of 12.5 mg kg⁻¹ body weight compared well with controls and were significantly lower than those observed for infected but untreated animals. These results suggest that *S. dulcis* effectively resists these Trypanosome induced changes in the rabbit.

Key words: *Scoparia dulcis*, alkaline phosphatase, bilirubin, transaminase, *Trypanosoma brucei*

INTRODUCTION

Scoparia dulcis is an erect annual herb with serrated leaves, producing white flowers and measuring up to a half meter in height when fully grown. Its ethnomedicinal uses amongst various indigenous tribes is well-documented^[1,2]. Some of the many speculated medicinal values of *S. dulcis* have been validated by scientific research. These include hypoglycaemic activity^[3], antitumour promoting activity^[4] and antiviral activity^[5]. Phytochemical screening of the herb revealed that it is rich in flavonoids and terpenes and the pharmacological actions of *S. dulcis* are believed to be due to the presence of these phytochemicals^[5-9]. The present research interest/effort arose from the widely speculated efficacy of *S. dulcis* in the management of sickle cell anaemia in parts of Nigeria. Mrs. Hilda Ogbe has for over two decades employed the herb in the management of sickle cell anaemia with profoundly outstanding results. Not only were there claims of massive boost in haematocrit or Packed Cell Volume (PCV) and haemoglobin (Hb) levels, the patients and their parents were excited that the frequent crisis associated with the disease had also abated. The lack of animal model for sickle cell disease prompted us to investigate the efficacy of *S. dulcis* using animal models experimentally infected with *T. brucei brucei*. Progressive anaemia is widely accepted as a cardinal feature of *T. brucei brucei* infection^[10-11]. The compelling evidence in favour of the anti anaemic claim (paper under review) prompted us to investigate the effect of *S. dulcis* on *T. brucei* induced biochemical lesions. The present study determined the efficacy of *S. dulcis* in the management of trypanosome induced alterations in serum transaminases, alkaline phosphatase and bilirubin in the rabbit.

MATERIALS AND METHODS

Treatment of animals: A total of 15 New Zealand white rabbits (average weight=1.625 kg) obtained from a private farm in Benin City were used for the experiment. These were randomly divided into 3 groups of n=5 with each group allowed a 14 days acclimatization on growers mash (product of Bendel Feeds and flour Mill Ewu, Edo State, Nigeria) prior to the commencement of experiment. Group 1 served as control while group II and III were inoculated with *T. brucei brucei*. Inoculation was by intraperitoneal injection of 0.5 mL of a 1:1 (infected whole blood: normal saline) preparation and with each inoculum containing about 2×10^6 of the parasite. Parasite estimation was by the rapid "Matching" method^[12]. The original stock of *T. brucei* was obtained from the Nigerian Institute for Trypanosomiasis Research, Vom, Plateau State, Nigeria.

The control animals (groups 1) were given intraperitoneal injection of 0.5 mL of normal saline in lieu of parasite. All animals were allowed unlimited access to food and clean drinking water throughout the duration of the experiment. In addition the inoculated and treated animals (group II) were given *S. dulcis* at a daily oral dose of 12.5 mg kg^{-1} body weight. Preparation of *S. dulcis* involved only sun drying and blending of the entire shoot system. The required weight of pulverized herb was suspended in a little volume (3-5 mL) of clean drinking water and administered through gavage. All animals were sacrificed at the end of 4 weeks under ether anaesthesia and blood samples collected for subsequent biochemical analysis which were carried out within a few hours of sample collection.

Biochemical assays: Biochemical assays were carried out using previously described protocols. Serum transaminases (SGOT and SGPT) were assayed by the method of Reitman and Frankel^[13] using commercially available kits, product of Randox Laboratories, UK., Alkaline Phosphatase (ALP) was determined by the phenolphthalein monophosphate method using commercial kit prepared by Quimica Clinica Aplicada Spain. In both cases, the manufacturer's instructions were strictly adhered to. The method of Malloy and Evelyn^[14] was used in the determination of serum total and conjugated bilirubin.

The group Mean \pm S.E.M. was calculated for each analyte and significant difference between means evaluated at 5% level of significance.

RESULTS AND DISCUSSION

The present study evaluated the effect of *S. dulcis* on *T. brucei* induced alterations in serum transaminases alkaline phosphatase and bilirubin in the rabbit. There were significant increases in Alkaline Phosphatase (ALP), Serum Glutamic Oxaloacetic Transaminase (SGOT), Serum Glutamic-pyruvic Transaminase (SGPT), total bilirubin (T. Bil.) and conjugated bilirubin (C. Bil.) (Table 1) in infected animals relative to control. These changes were however remarkably resisted in the *S. dulcis* treated infected animals. For instance, there was no significant difference between the values obtained for ALP, SGOT, C. Bil. and T. Bil. in infected and treated animals when compared with controls. In addition, the values of all parameters determined were also significantly lower for group II (inoculated and treated) when compared with group III (inoculated untreated) (Table 1). This implies that infection with *T. brucei brucei* is accompanied with significant increases in ALP, SGOT, SGPT, as well as total and conjugated bilirubin. This has been repeatedly

Table 1: Control of *T. brucei brucei* induced changes in ALP, SGOT, SGPT and Bilirubin by *S. dulcis*

Parameters	Group I control	Group II inoculated and treated	Group III inoculated and untreated
ALP(U L ⁻¹)	16.00±1.45 ^a	15.60±1.25 ^a	34.60±1.63 ^b
SGOT(U L ⁻¹)	7.40±1.94 ^a	11.00±1.58 ^a	20.00±2.19 ^b
SGPT (U L ⁻¹)	8.40±1.08 ^a	11.60±0.68 ^a	19.20±1.96 ^b
SGOT: SGPT	0.88±0.17 ^a	0.94±0.10 ^a	1.04±0.08 ^a
T. BIL (mg dL ⁻¹)	0.70±0.03 ^a	0.70±0.04 ^a	0.90±0.03 ^b
C. BIL (mg dL ⁻¹)	0.38±0.06 ^a	0.46±0.05 ^b	0.56±0.02 ^b

Values are Mean±S.E.M. and values with different superscripts differ significantly (p<0.05)

demonstrated in our laboratory and other workers have reported elevations in serum transaminases following infection with the sister parasite, *T. congolense*^[15].

Elevations in ALP, SGOT and SGPT are usually secondary to tissue damage. This is because such damage results in the leakage of these enzymes from their intracellular stores into plasma. SGPT is most prevalent in the liver whereas SGOT may also be found in heart, skeletal muscle and liver to nearly the same extent. Significant increases in the transaminases commonly accompany such liver diseases as toxic hepatitis, acute liver necrosis or hepatic cirrhosis. Increases in SGOT are often seen in hemolytic anaemia, myocardial infarction and cholestatic diseases of the liver^[16-17].

The fractional increases in serum GOT and GPT or the ratio of SGOT: SGPT may be a useful tool in assessing the extent of liver damage. Liver cells contain more GOT than GPT and with GPT confined largely to the cytoplasm in which its concentration is higher than that of GOT. With inflammatory or infective conditions such as viral hepatitis, the cytoplasmic membrane sustains the greater damage and the relative increase in serum GPT is higher than that of GOT. The situation is reversed in infiltrative disorders in which both the cytoplasmic and mitochondrial membranes are affected, resulting in a proportionally greater increase in GOT relative to GPT^[16]. In this study, there was no significant difference in the SGOT: SGPT ratio, but the fractional increase in SGOT (1.7) was higher than the 1.3 observed for SGPT. It is pertinent to note that *T. brucei brucei* is essentially a tissue invasive parasite and that possible infiltration of the liver could result in hepatic lesions.

Serum ALP activity increases in Cholestatic disease of the liver and in bone diseases^[19]. This is usually secondary to increased enzyme synthesis and regurgitation of enzyme within the biliary tract into plasma^[17]. Increased red cell breakdown often results in elevated serum bilirubin and in the face of intra hepatic or extra hepatic obstruction as seen in cholestasis, conjugated bilirubin is regurgitated into the blood thus raising its level in plasma^[17]. Previous studies^[10-11] have

shown that progressive anaemia is a cardinal feature of *T. brucei* infection.

The changes in Trypanosome infected animals observed in this study, no doubt may have been complicated by multi system involvement^[18-22]. What is most remarkable however is the ability of *S. dulcis* to effectively control or resist these changes. It does not immediately appear, the exact mechanism(s) by which *S. dulcis* exerts its effect nor can this activity be readily ascribed to any one of the many biologically active compounds present in the plant.

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