Evaluation of anti-plasmodial, anti-pyretic and mean survival time of rodents treated with ethanolic extract of *Enantia chlorantha* stem bark

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Abstract

**Background and aim of study:** *Enantia chlorantha* is a medicinal plant that is used all over Nigeria for treatment of malaria and other febrile illnesses. Anti-plasmodial effects of the extract and its different fractions (n-hexane, chloroform, ethyl acetate, methanol and aqueous) were tested at doses of 32.40 mg/kg, 64.80 mg/kg and 96.20 mg/kg in Chloroquine sensitive *Plasmodium berghei berghei* infected mice. The antipyretic effects were evaluated also.

**Experimental:** Parasitized blood donor of Swiss albino mice with at least 1.0 x 10^7 parasitized red blood cells was used. Suppressive, prophylactic and curative models were investigated. The mean survival time was observed. Evaluation of anti-plasmodial activity of the fractions using the 4-day test was done. The effect of extract on 2, 4-Dinitrophenol (DNP)-Induced pyrexia in adult albino rats (150-180 g) was also evaluated.

**Results:** Statistically significant dose-dependent reduction in parasitaemia was noted in all three models (p<0.001) when compared to control. The mean survival times were increased in a dose-dependent manner which was statistically significant (p<0.001). N-hexane, chloroform, ethyl acetate and methanol fractions exerted varying degrees of anti-plasmodial activity with methanol being the highest. The extract treated groups showed dose-dependent reduction in temperature that was statistically significant (p<0.05 – 0.001).

**Conclusion:** The above results provide support for the traditional use of *Enantia chlorantha* stem bark in treating febrile illnesses such as malaria in Akwa Ibom state. This extract shows a lot of promise and is a good candidate for evaluation as a potential new drug for malaria and other febrile and inflammatory conditions.

**Key words:** *Enantia chlorantha*, ethanol extract, malaria, rodents, pyrexia

1. Introduction

*Enantia chlorantha* is a fair sized ornamental forest tree that can reach heights of 30m. It grows in dense shade and may be recognized by its bright yellow slash and conspicuous black fruits (Vivien and Faura, 1985) [29]. It is located in the West African region and extends from southern Nigeria to Gabon, Zaire and Angola. It is commonly called African white wood, Moambe Jaune and Annikia chlorantha. Locally, the name varies from place to place. The Ibios of Akwa Ibom call it Uno eto, the Yoruba’s call it Osupupa or dokita Igbo. The Edo people refer to it as Erenbav bogo while Ikale and Boki tribes refer to it as Osomolu and Kakerim respectively. The family is Annonaceae and the specie is chlorantha. There are over 40 different species that grow in Nigeria as is seen by the various names that it is called and the varied uses to which it is applied. Enantia chlorantha is used to treat a wide variety of conditions using its roots, stem bark, fruit and leaves. In Akwa Ibom it is used to treat malaria fever, typhoid fever, jaundice, dysentery, wounds, infections, high blood pressure and many other related illnesses. It has been used also as anti-viral, anti-candidal and for gastroenteritis (Moody et al, 1995) [18], (Gill and Akinwunmi, 1986) [9]. A decoction of the stem bark in illicit gin is usually taken to relieve painful and swollen joints, fever, headache and toothache. A literature search showed that the plant has been worked on by many investigators who have examined different aspects of the plant. We aim by this work to use other models in the hope that the mechanism of action of the plant may be inferred.
2. Materials and Methods

2.1 Plant material
The plant *Enantia chlorantha* was collected in January 2012 in Uyo the capital city of Akwa Ibom State, Nigeria. It was identified and authenticated by a Taxonomist in the Department of Botany and Ecological Studies, University of Uyo, Akwa Ibom state Nigeria. A voucher specimen (voucher number UUH 018/13) was deposited with the herbarium of the Department of Pharmacognosy and Natural Medicine, Faculty of Pharmacy, University of Uyo.

2.2 Extraction
The plant stem bark was washed and partially air dried for about 2 weeks at ambient temperature (25°C ± 1°C). It was then ground to powder using pestle and mortar. The pulverized sample was divided into two parts. One part was cold macerated in 70% ethanol at room temperature for 72 hr and then filtered. The filtrate was dried in a rotary evaporator at 40°C. This extract is referred to as crude. The other part was successively and gradiently macerated for 72 hr at room temperature in the following solvents: n-hexane, chloroform, ethyl acetate, methanol and water to obtain different fractions.

The crude extract and the fractions were stored in the freezer at -40°C until required.

2.3 Acute toxicity study of the extract
The method of Lorke (1983) was used to determine the LD$_{50}$ of the extract in Swiss albino mice. The extract was administered to three groups of mice containing three mice each at a dose range of 100-1000 mg/kg, (i.p). The animals were observed for physical signs of toxicity and the number deaths in each group within 24 h were recorded. The animals were fasted for 24 h prior to the experiment but allowed water *ad libitum*. The LD$_{50}$ was calculated as the geometric mean of the maximum dose producing 0 % mortality (A) and the minimum dose producing 100 % mortality (B). LD$_{50}$ = $\sqrt{AB}$

2.4 Phytochemical screening
The phytochemical screening of the extract was carried out according to the methods of [Odebiyi and Sofowora, 1978] [19], [Trease and Evans, 1996] [28]. The following bioactive compounds were screened for their presence: saponins, tannins, and alkaloids. Others were flavonoids, antraquinones, cardiac glycosides and reducing sugars.

2.5 Animal stock
Swiss albino wistar rats weighing 150-180 g of both sexes were used for the experiments. They were obtained from the Department of Pharmacology Animal House in the University of Uyo, Uyo. The animals were housed in standard plastic cages at room temperature and moisture under naturally illuminated environment of 12:12 h dark/light cycle. They were fed with pelleted feeds (Bendel Feeds) and allowed water *ad libitum*.

3. Evaluation of Anti-Malarial Activity

3.1 Parasite Inoculation
A parasitized blood donor of Swiss albino mice with at least 1.0 x 10$^7$ parasitized red blood cells was anaesthetized with chloroform and blood collected through a cardiac puncture using a sterile syringe and a heparinized bottle. Each mouse was given 0.2 ml of infected blood intraperitoneally. Each inoculum contained at least 1.0 x 10$^7$ Plasmodium berghei berghei infected erythrocyte per ml. This was prepared by determining both the percentage parasitaemia and the erythrocyte count of the donor mouse and diluting the blood with isotonic saline (Odetola and Basir, 1980) [20].

3.2 Determination of Suppressive Activity on Early Infection (4-day Test)

The suppressive activity of the extract was measured using the method of Knight and Peters (1980) [14]. An intraperitoneal inoculation of each mouse was done on the first day (day 0) using 0.2 ml of infected blood containing 1.0 x 10$^7$ Plasmodium berghei berghei parasitized erythrocytes. The animals were then randomly divided into six groups of 6 animals each. Ten minutes after the inoculum was given, mice in group 1 were administered with distilled water (10 ml/kg, p.o.). Groups 2-4 were administered with 32.40, 64.80 and 96.20 mg/kg, (i.p.) of the extract respectively. The fifth group received artesunate (5 mg/kg, i.p.) while group 6 was administered with artesunate (5 mg/kg, i.p.) and then 10 min later, the extract (64.40 mg/kg) was given. Administration of extract and drug was continued for 4 days (D0-D3) between 8.00am and 9.00am. On the fifth day (D4), a thin blood smear was taken from the tail of each mouse. The films then stained with Leishman’s stain and examined under high power field. The percentage parasitaemia was determined by counting the number of parasitized red blood cells in a minimum of 500 erythrocytes in random high power fields of the microscope. Percentage (%) parasitaemia = No. of parasitized RBC/Total no. of RBC counted x 100/1

Average Percentage (%) chemosuppression was calculated as A-B/A x 100.

Where A is the average parasitaemia in the negative control group and B is the average Percentage (%) parasitaemia in the test group.

3.3 Determination of Repository (Prophylactic) Activity

The method of Peters (1965) [22] was adopted for the test. Six groups of Swiss albino mice were randomly selected with six mice per group. Mice in group 1 were administered with distilled water (10 ml/kg, p.o.). Groups 2-4 were administered with 32.40, 64.80 and 96.20 mg/kg, (i.p.) of the extract respectively. Group 5 received pyrimethamine, 1.2 mg/kg and served as positive control. Group 6 received 1.2 mg/kg of pyrimethamine and middle doses of extract (64.80 mg/kg). The animals were pre-treated with the extract for three consecutive days (D0-D2). On the fourth day, the mice were inoculated intraperitoneally with 0.2 ml of infected blood containing 1.0 x 10$^7$ Plasmodium berghei berghei parasitized erythrocytes. Tail blood was taken and parasitaemia evaluated by thin blood smear and microscopy after 72 h. The percentage parasitaemia and average chemosuppression were calculated as shown in section (3.2) above.

3.4 Determination of Effect of Extract on Established Infection (Curative Test)

The method of Ryley and Peters (1970) [25] was used to determine schizonticidal activity of the extract in the presence of established infection. Thirty six mice were inoculated intraperitoneally with 1.0x10$^7$ Plasmodium berghei berghei infected erythrocytes on day 1 (D0). 72 h later, the animals were randomly selected and divided into 6 groups of 6 mice per group. Mice in group 1 were administered with distilled water (10 ml/kg, p.o.) as negative control. Groups 2-4 were administered with 32.40, 64.80 and 96.20 mg/kg, (i.p.) of the extract respectively. Group 5 received 5 mg/kg of artesunate and served as positive control. Group 6 received artesunate (5 mg/kg). Ten minutes later, the extract (64.80 mg/kg) was
The extract was administered once daily for 5 days. Thin
smears were made from tail blood and stained with
Leishman’s stain to monitor the level of parasitaemia. The
mean survival time of each group was determined over a
period of 30 days (D0-D29) by evaluating the average
survival time for each group.

3.5 Evaluation of Anti-plasmodial Activity of the Fractions
using the 4-day Test
Different fractions of Enantia chlorantha (n-hexane,
chloroform, ethyl acetate, methanol and aqueous) were
administered orally to the animals. The rats were divided into
six groups of six rats per group. A single dose of the fractions
(64.80 mg/kg) was administered to each group. Groups 1-3
were administered with n-hexane, chloroform, and ethyl
acetate respectively while groups 4 and 5 were given
methanol and aqueous fractions equally. Group 6 was given
distilled water (10 ml/kg, p.o.)

After 30 min, 0.2 ml of infected blood containing about
1.0x10⁷ Plasmodium berghei berghei was administered
intraperitoneally. The administration of fractions and distilled
water was done for 4 days. Thin blood films were made on
day 5 from tail blood and the level of parasitaemia evaluated
by counting the number of parasitized erythrocytes per
random high power field of not less than 500 red blood cells.
The mean survival time was determined over a period of 30
days. The percentage chemo suppression was then calculated.

3.6. Evaluation of Anti-pyretic Activity of Extract in Rats
3.6.1 Effect of Extract on 2, 4-Dinitrophenol (DNP)-
Induced Pyrexia in Rats
Adult albino rats (150-180 g) of both sexes were used for this
experiment. The animals were fasted for 24 h but allowed
access to water ad libitum. The basal rectal temperature was
taken before administration of any drug. DNP (10 mg/kg) was
given to all animals intraperitoneally (i.p). Hyperthermia was
induced at least 30 min after the administration of DPN.
Rectal temperatures were then taken at hourly intervals and animals with a 10°C rise in temperature
were selected for the experiment. The extract was administered at doses of 32.40, 64.80 and 96.20 mg/kg, (i.p.)
to animals in groups 2-4 respectively. Group 1 received 10
ml/kg, (p.o.) of distilled water while group 6 was administered acetylsalicylic acid (100 mg/kg, i.p.). Group 5
animals received middle dose of extract and 10 min later, ASA (100 mg/kg, p.o.) was administered (Agrawal and

3.6.2 Effect of Extract on D-amphetamine-induced
Pyrexia in Rats
Adult albino rats of both sexes were used for this study. They
were fasted for 24 hr and allowed access to water ad libitum.
The basal rectal temperatures of the animals were taken.
Amphetamine (5 mg/kg, i.p.) was given and hyperthermia
developed 30 min after administration. Rectal temperatures were taken within 30 min after receiving amphetamine. Those
with 10°C rise in temperature were selected for the study. Thereafter, they were randomized into six groups of 6 rats
each. Group 1 was given 10 ml/kg, (p.o). Groups 2-4 were
administered the extract (32.40, 64.80 and 96.20 mg/kg, i.p.)
respectively. Group 5 animals received middle dose of extract
and 10 min later, acetylsalicylic acid (ASA, 100 mg/kg, p.o.)
was administered. Group 6 was administered with ASA (100
mg/kg, p.o.). Rectal temperatures were then taken hourly for 5
h (Bamgbose and Noamesi, 1981; Blackhouse et al., 1994;
Mbagwu et al., 2007) [5, 6, 17].

3.6.3 Effect of Extract on Yeast-induced Pyrexia in Rats
Adult albino rats (150-180 g) of both sexes were fasted for 24
h and were allowed access to water ad libitum. They were
randomized into 6 groups of 6 animals each. The basal rectal
temperatures were determined. Aqueous suspension of
Brewer’s yeast (10 ml/kg of 15 % w/v) was administered
subcutaneously. Rectal temperatures were then taken at hourly intervals and animals with a 10°C rise in temperature
were selected for the experiment. The extract was administered at doses of 32.40, 64.80 and 96.20 mg/kg, (i.p.)
to animals in groups 2-4 respectively. Group 1 received 10
ml/kg, (p.o.) of distilled water while group 6 was administered acetylsalicylic acid (100 mg/kg, i.p.). Group 5
animals received middle dose of extract and 10 min later, ASA (100 mg/kg, p.o.) was administered (Agrawal and
Paridhavi, 2007) [3].

3.7 Statistical analysis
Results were expressed as multiple comparisons of mean ±
SEM. Significance was determined using One-way Analysis
of Variance (ANOVA) followed by Turkey-Kramer multiple
comparison post test. A probability level of less than 5 % was
considered significant.

4. Results/Discussions

4.1 Repository/Prophylactic Activity of Extract
The results of prophylactic activity of extract is as shown in
Table1. The extract produced a dose-dependent chemo suppressive effect at the different doses employed in the
study. The percentage chemo suppressions were 47.35, 56.37
and 76.60 % for 32.40, 64.80 and 96.20 mg/kg/day doses
respectively. These effects were statistically significant
(p<0.001) when compared to control. The effects of the various
doses of the extract were incomparable to that of
artesunate, 5 mg/kg.

4.2 Effect of Extract on Established Infection in Mice
(Curative Test)
The effect of extract on established infection of P. berghei is
as shown in Figure 1. There was a dose–dependent reduction
in parasitaemia in the extract-treated groups similar to that of
the artesunate treated group. The reductions were statistically
significant (p<0.001) when compared to control. The control
group showed daily increase in parasitaemia. On Day7, the
percentage chemo suppression for the groups were 77.72,
79.70, 85.07, 88.76 and 91.21 for 32.40 mg/kg, 64.80 mg/kg,
96.20 mg/kg, extract (64.80) plus artesunate (5 mg/kg),and
artesunate groups respectively (figure 1).
4.3 Effect of Extract on Mean Survival Time

The result of mean survival time (MST) is shown in Table 3. The MST of the extract treated groups were significantly ($p<0.01$) longer than that of the control though less than that of the artesunate treated group. Artesunate treated group had MST value of 30.00 days.

<table>
<thead>
<tr>
<th>Drug/Extract</th>
<th>Dose (mg/kg)</th>
<th>Parasitaemia</th>
<th>% Chemosuppression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distilled water</td>
<td>10 ml</td>
<td>8.33 ±2.58</td>
<td>55.00 + 1.06</td>
</tr>
<tr>
<td>Extract</td>
<td>32.40</td>
<td>15.80 ± 2.50 a</td>
<td>61.24</td>
</tr>
<tr>
<td>Extract</td>
<td>64.80</td>
<td>20.50 ±1.41 a</td>
<td>73.68</td>
</tr>
<tr>
<td>Artesunate</td>
<td>5.00</td>
<td>24.57 ± 2.25 a</td>
<td>77.01</td>
</tr>
<tr>
<td>Artesunate + Extract</td>
<td>5.00 + 64.80</td>
<td>28.35 ± 0.57 a</td>
<td>77.45</td>
</tr>
</tbody>
</table>

Values are expressed as mean ±SEM Significance relative to control: $p<0.001$ (n=6).

4.4 Anti-plasmodial Activity of Fractions of Extract

The results of suppressive activity of the various fractions of the extract are shown in Table 4. N-hexane, chloroform, ethyl acetate and methanol fractions exerted degrees of antiplasmodial activity. The chemosuppressive activities of the fractions were 61.24, 73.68, 77.01 and 77.45 % n-hexane, chloroform, ethyl acetate and methanol respectively. However, methanol fraction had the highest activity.

<table>
<thead>
<tr>
<th>Drug/Extract</th>
<th>Dose (mg/kg)</th>
<th>Parasitaemia</th>
<th>% Chemosuppression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distilled water</td>
<td>10 ml</td>
<td>45.80 ± 0.71</td>
<td>61.24</td>
</tr>
<tr>
<td>n-hexane</td>
<td>64.80</td>
<td>17.75 ±0.65 a</td>
<td>73.68</td>
</tr>
<tr>
<td>chloroform</td>
<td>64.80</td>
<td>12.05 ± 0.74 a</td>
<td>77.01</td>
</tr>
<tr>
<td>ethyl acetate</td>
<td>64.80</td>
<td>10.35 ± 1.82 a</td>
<td>77.45</td>
</tr>
<tr>
<td>methanol</td>
<td>64.80</td>
<td>10.15 ± 0.92 a</td>
<td>77.45</td>
</tr>
</tbody>
</table>

Values are expressed as mean ±S.E.M. Significance relative to control: $p<0.001$ (n=6).
4.5 Anti-pyretic Activity

4.5.1 Effect of Extract on Dinitrophenol-Induced Pyrexia in Rats

The anti-pyretic effect of the extract on 2, 4 dinitrophenol (DNP)-induced pyrexia is as shown in Table 5. Administration of the extract (32.40, 64.80 and 96.20 mg/kg) in the presence of the pyrogen caused a significant (p<0.05 – 0.001) reduction in the temperatures of the extract treated rats when compared with the control. The anti-pyretic effect was dose-dependent though not as high as that of the standard drug, ASA (100 mg/kg).

### Table 5: Effect of extract on dinitrophenol-induced pyrexia in rats

<table>
<thead>
<tr>
<th>Treatment/ Dose (mg/kg)</th>
<th>Time Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Basal Temp</td>
</tr>
<tr>
<td>Control</td>
<td>36.94±0.05</td>
</tr>
<tr>
<td>Extract 32.40</td>
<td>36.96±0.06</td>
</tr>
<tr>
<td>64.80</td>
<td>36.94±0.05a</td>
</tr>
<tr>
<td>96.20</td>
<td>37.01±0.04c</td>
</tr>
<tr>
<td>ASA 100</td>
<td>36.96±0.06b</td>
</tr>
<tr>
<td>ASA 100+ 64.80 mg/kg</td>
<td>36.91±0.12b</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM. Significant at *p<0.05, *p<0.01 when compared to control (n = 6).

ASA = Acetyl salicylic Acid

4.5.2 Effect of Extract on Amphetamine-induced Pyrexia in Rats

Table 6 shows the effect of the extract on amphetamine-induced pyrexia. The extract exerted a significant (p<0.05–0.001) dose-dependent anti-pyretic effect especially in fourth and fifth hours when compared to control.

### Table 6: Effect of extract on amphetamine-induced pyrexia in rats

<table>
<thead>
<tr>
<th>Treatment/ Dose (mg/kg)</th>
<th>Time Intervals (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Basal Temp</td>
</tr>
<tr>
<td>Control</td>
<td>36.10±1.70</td>
</tr>
<tr>
<td>Extract 32.40</td>
<td>36.48±1.80b</td>
</tr>
<tr>
<td>64.80</td>
<td>36.60±0.80b</td>
</tr>
<tr>
<td>96.20</td>
<td>36.15±0.60b</td>
</tr>
<tr>
<td>ASA 100</td>
<td>36.58±0.80b</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM. Significant at *p<0.05, *p<0.01, *p<0.001 when compared to control (n = 6).

ASA = Acetyl salicylic Acid

4.5.3 Effect of Extract on Yeast-Induced Pyrexia in Rats

The result of the effect of the extract against yeast-induced pyrexia is as shown in Table 7. The extract caused considerable and sustained reductions in temperature change in rats treated with the extract. These reductions were statistically significant (p<0.05–0.001) relative to control.

### Table 7: Effect of extract on brewer’s yeast-induced pyrexia in rats

<table>
<thead>
<tr>
<th>Treatment/ Dose (mg/kg)</th>
<th>Time Interval (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Basal Temp</td>
</tr>
<tr>
<td>Control</td>
<td>36.50±0.17</td>
</tr>
<tr>
<td>Extract 32.40</td>
<td>36.00±0.16a</td>
</tr>
<tr>
<td>64.80</td>
<td>35.83±0.11a</td>
</tr>
<tr>
<td>96.20</td>
<td>36.67±0.06c</td>
</tr>
<tr>
<td>ASA 100</td>
<td>37.70±0.20c</td>
</tr>
<tr>
<td>ASA 100+ 64.80 mg/kg</td>
<td>36.43±0.45a</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM. Significant at *p<0.05, *p<0.01, *p<0.001 when compared to control (n = 6).

ASA = Acetyl salicylic Acid

5. Discussion

The anti-plasmodial evaluations of the extract showed a significant activity in suppressive, repository and curative models. These effects were comparable to those of the standard drugs employed in this investigation namely artemesunate and pyrimethamine. Secondary metabolites such as alkaloids and terpenes like sesquiterpenes and monoterpene have been implicated in anti-plasmodial activity of plants (Philipson and Wright, 1991) (23), (Christensen and Kharazmi, 2001) (8). The extract also contains protoberberine and berberine alkaloids which have been reported to be responsible for the anti-malarial activity of this plant (Kimbi et al., 1996) (13), (Tan et al., 2000) (20); (Tan et al., 2002) (22). That the extract contains the above secondary metabolites suggest that the anti-plasmodial activity exhibited by the plant may in part be due to these active ingredients. The result of this study corroborates well with that of Kimbi et al. (1996) (13); Tan et al. (2000) (20); Tan et al. (2002) (22) and (Ogbonna, Sokari, and Agomoh, 2008) (21), who reported significant anti-plasmodial activity in the root of this plant. Artemisinin, sesquiterpene endoperoxide is reported to exert anti-plasmodial activity by generating reactive oxygen species
which alkylate with the heme protein resulting in the death of the parasite (Robert, Benoit-vical, Dechy-cabaret, and Meunier, 2001) [24]. Sesquiterpenes and monoterpenes like limonene have been implicated in endoperoxidation leading to plasmodial activity (Hatzakis, Opesenica, Solaja, and Stratakis, 2007) [11]. Chloroform, ethyl acetate, n-hexane and methanolic fractions had comparative activity indicating possible localization of the active ingredients in these fractions. This study has demonstrated that the extract possess anti-malarial activity which justifies its use in traditional medicine. On anti-pyretic activity, the extract inhibited significantly dinitrophenol, amphetamine and yeast-induces pyrexia. Dinitrophenol induces hyperthermia by uncoupling oxidative phosphorylation causing release of calcium from mitochondrial stores and also prevents calcium reuptake. This results in increased level of intracellular calcium, muscle contraction and hyperthermia (Kumar, Baker, and Seger, 2002) [15]. Yeast induces pyrexia by increasing the synthesis of prostaglandins (Al-Ghamdi, 2001) [1]. The extract may in part reduced pyrexia by reducing brain concentration of prostaglandin E2 especially in the hypothalamus through its action on COX-2 or by enhancement of the production of the body’s own anti-pyretic substances such as vasopressin and arginine (Chandrasekharan, 2002) [7]. The hypothermic activity of the extract could have also been mediated by vasodilatation of superficial blood vessels leading to increased dissipation of heat following resetting of hypothalamic temperature control center. Diterpenes such as those present in the leaf of Croton zambesicus have been reported to possess vaso-relaxant activity (Baccelli et al., 2005) [6]. Therefore, the temperature lowering activity of the extract may not be unconnected with the inhibition of one or combination of the mechanisms mentioned above. This finding corroborates an earlier report by Adesokan et al. (2008) [1] who reported the anti-pyretic activity of the extract against brewer’s yeast.

6. References


25. Ryley JF, Peters W. The Antimalarial Activity of some Quinone Esters. Annals of Tropical Medicine and


