Original Research Article

Assessment of some brain antioxidants profile in *Plasmodium berghei berghei* infected mice treated with ethanol leaf extract of *Musa paradisiaca.*

A B S T R A C T

Background and aim: This study evaluated brain antioxidants of locally formulated herbal antimalarial therapy, dry plantain leaf extract (Musa paradisiaca) on mice infected with Plasmodium berghei

Experimental procedure: Five groups of ten mice each namely control, P. berghei, artesunate, P. berghei infected mice using curative, suppressive and prophylactic models were orally administered with 250 and 1000mg/kg leaf extract of Musa paradisiaca. On the 10th day, the mice were sacrificed and blood samples collected for Catalase, Glutathione Peroxidase(GPX), Glutathione(Glut) and Malondialdehyde(MDA) estimation by standard method. Data was analyzed using SPSS version 21.

Results and conclusion: Catalase(μ/L) was 18.44±1.31, 9.65±0.16, 11.64±1.68, 12.83±0.43, 12.39±0.67, 12.80± 2.20, 9.90± 0.30, 11.54± 0.30, 15.59±0.86, 12.91± 0.33 and 12.0± 0.30, GPX(μ/L) was 63.58±0.62, 19.13±2.4, 42.24±7.15, 17.62±12.70, 55.27±0.50, 42.24±7.15, 17.62±12.70, 55.27±0.50, 37.8±5.20, 39.40± 0.30 and 34.50± 0.50, Glut(μ/m) was 1.05±0.35, 0.61±0.59, 0.67±0.53, 0.74±0.46, 0.94±0.18, 0.67±0.53, 0.74±0.46, 0.94±0.18, 0.74± 0.18, 0.68± 0.40 and 0.81± 0.30 while MDA(μ/m) was 47.9±0.45, 11.2±0.8, 6.4±0.2, 6.0±0.10, 8.30±0.40, 6.4±0.2, 6.0±0.10, 8.3±0.4, 6.10± 0.50, 6.00±0.10 and 5.90± 0.00 in control, *P bergei*, Artesunate, 250mg, 1000mg, Artesunate, 250mg, 1000mg, Artesunate, 250mg and 1000mg curative, Suppressive and prophylactic doses respectively. Results showed *Musa paradisiaca* leaf extract protects brain of *Plasmodium berghei* infected mice from free radicals.

Keywords: Musa paradisiaca, Plasmodium berghei, Brain, antioxidant, curative, Suppressive, Prophylaxis

1. Introduction

Plasmodium berghei is a species in the genus *Plasmodium* subgenus *Vinckeia* . It is a protozoan parasite that causes malaria in certain rodents. Originally, isolated from thicket rats in Central Africa, *P. berghei* is one of four *Plasmodium* species that have been described in African marine rodents, the others being *Plasmodium chabaudi*, *Plasmodium vinckei*, and *Plasmodium yoelii*. Due to its ability to infect rodents and relative ease of genetic engineering, *P. berghei* is a popular model organism for the study of human malaria (1). Like all malaria parasites of mammals, including the four human malaria parasites, *P. berghei* is transmitted by *Anopheles* mosquitoes and it infects the liver after being injected into the bloodstream by a bite of an infected female mosquito. After a short period (a few days) of development and multiplication, these parasites leave the liver and invade erythrocytes (red blood cells). The multiplication of the parasite in the blood causes the pathology such as anaemia and damage of essential organs of the host such as lungs, liver, spleen. *P. berghei* infections may also affect the brain and can be the cause of cerebral complications in laboratory mice. These symptoms are to a certain degree comparable to symptoms of cerebral

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malaria in patients infected with the human malaria parasite *Plasmodium falciparum* (1). The fruit of *Musa paradisiaca* is an exceptional fruit offering different forms of nutrition. It is an excellent source of potassium and a single banana is said to provide 23% of potassium on a daily basis (2). The flowers are useful in bronchitis, dysentery, and ulcers, whereas cooked flowers are given to people with diabetes. The fruit and leaves of Plantain are useful in treating burns and wounds (2). The fruit has a mild laxative property whereas the plant sap is an astringent which is used in cases of hysteria, epilepsy, leprosy, fevers, hemorrhages, dysentery and diarrhea (2). The unripe fruits have been evaluated for its antihyperglycemic, antioxidant, wound healing, hypolipidemic, hair growth promoter, cardioprotective and against thyroid dysfunction (3). Plantain is also considered to be a rich source of antioxidants (4). Bioactive compounds in Plantain leaf include phenolic compounds, carotenoids, biogenic amines, and phytosterols (4). The aim of this study is to determine the level of some brain antioxidants profile in *Plasmodium berghei berghei* infected mice, treated with ethanol leaf extract of *Musa paradisiaca* using Catalase, Glutathione, Malondialdehyde and Glutathione Peroxidase

2. Materials and methods

2.1 Animal

One hundred and six (106) 2-3 months old healthy albino mice weighing between 13-36g were obtained from animal house of the Federal University of Technology, Owerri, Nigeria for the study. They were kept under light/darkness cycles in aluminum cages at room temperature in the animal house of Madonna University, Elele campus. The mice were fed rat pellet with clean drinking water *ad libitum* and were acclimatized for two weeks before administration. They were maintained in accordance with recommendations of the guide for the care and use of laboratory animals and experimental protocol.

2.2 Extraction and preparation of plant materials

The leaves of *Musa paradisiaca* obtained from a plantain plantation in Elele, Rivers State, Nigeria, were cut into pieces, washed and dried. The dried pieces were grounded into fine powder using a manual grinder. Ten kilogram (10kg) of the grounded powder was soaked in 10litres of 80% ethanol for 72 hours with intermittent stirring of the solution and subsequently filtered through Whatman filter paper (125mm). Rotary evaporator was used to concentrate the extract at 45°C and then dried with a water bath at 39°C to produce 40g of dark semi-solid extract which was preserved at 0-4°C until needed for use.

2.3 Parasite innoculation

A chloroquine sensitive strain of *Plasmodium berghei* (NK 65 strain) parasite was obtained from the Department of Pharmacology and Toxicology, Faculty of Basic Clinical Sciences, University of Port Harcourt, Rivers State, Nigeria. Constant re-infestation of parasitized erythrocytes using *Plasmodium berghei* from a donor-infected mouse by the tail via a heparinized syringe and made up to 20ml with normal saline was done to sustain the *Plasmodium berghei.* The mice were inoculated with 0.2ml of infected blood suspension and were monitored for signs of infection such as lethargy, anorexia, shivering and heat-seeking environment. Daily microscopic examination of thick film stained by Giesma method was used to monitor the parasitaemia while Leishman stain on thin blood film was used to detect malaria parasite infection and the parasite density was determined using the formular.

Parasite density =Number of parasites counted × 100white blood cells/Number of white blood cells counted.

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2.4 Acute toxicity study (LD_{50})

The ***Acute toxicity study (LD_{50}) of*** crude extract of *Musa paradisiaca* leaf was evaluated using, modified Locke's (5) method of determining toxicity level of extract in mice. twelve mice randomized into three groups of four mice each and were given (500, 1000 and 3000) mg/kg body weight respectively of the extract orally and observed for changes in physical appearance, gross behavioral change and death in the first four hours and subsequently daily for five days in the first phase. Based on the result obtained from phase I treatment, phase II treatment was carried out using another fresh set of twelve mice randomized into three groups of four mice each and were given (500, 1000 and 3000) mg/kg body weight of the extract orally and subsequently observed for signs of toxicity and mortality for the first four hours and thereafter daily for ten days (5). The LD_{50} was then calculated as the square root of the product of the lowest lethal dose and highest non-lethal dose, i.e., the geometric mean of the consecutive doses for which 0 and 100% survival rates were recorded in the second phase. The oral median lethal dose was calculated using the formula: $LD_{50} = \sqrt{\text{maximum dose for all survival} \times \text{minimum dose for all death}}$ (5).

2.5 Experimental design

Fifty (50) mice weighing 10-37g were assigned into five groups of ten mice each. The group 1 were fed mice diet and water *adlibitum* to serve as control while the group 2 mice was infected with *P. berghei* and the group 3 mice was administered Artesunate. The group 4 mice was administered with 250 mg/ kg body weight *M paradisiaca* extract plus *P. berghei* parasite, group 5 mice was administered with 1000 mg/ kg body weight *M paradisiaca* extract plus *P. berghei* parasite using curative, suppressive and prophylactic model for 5 days..

2.6 Curative or Rane Test by evaluating Schizontocidal activity in Established Infection

A method described by Ryley and Peter (6) was used to evaluate the curative potential of the extract. This was done by intraperitoneal injection of the standard inoculums of $1x10^7$ *P. berghei* infected erythrocytes into 40mice on first day (Day 0) while 10mice were administered diet and water *adlibitum* to serve as control. The 40 mice were assigned into four groups of 10 mice each seventy-two hours later (3days) and each group were orally administered with 250, 1000mg/kg/day *Musa paradisiaca leaf* extract and 50 mg/kg/day artesunate while the last group served as *P. berghei* infected. The drug extract was given orally once daily for 5days while thin films stained with 10% Giemsa for parasitaemia (7) was examined microscopically on each day of treatment to monitor parasiteamia level.

2.7 **Evaluation of Blood Schizontocidal Activity on Early Infection (Suppressive Test)**

The method of Knight and Peters (8) was used to evaluate Schizontocidal activity of the extract fraction and artesunate against early *P. berghei* infection. Forty mice were inoculated with 0.2ml of infected blood containing about 1 X 10^7*Plasmodium berghei* parasitized erythrocytes using intraperitoneal route and subsequently divided into four groups of ten mice each, while another 10 mice were administered diet and water *adlibitum* to serve as control. The 250, 1000 mg/kg/day doses of the *Musa paradisiaca* leaf extract and 50 mg/kg/day artesunate were orally administered shortly after inoculation while the last group was *P. berghei infected group.* Thin films were made from blood collected from the tail of each mouse on the 5th day (day 5), stained with 10% Giemsa stain according to Cheesbrough (7) and examined microscopically while the

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parasitaemia level was determined by counting the number of parasitized erythrocytes out of 200 erythrocytes in random fields of the microscope.

Average percentage chemo suppression was calculated as: 100[(A−B)/A], where A is the average percentage parasitaemia in the negative control group and B is the average parasitaemia in the test/standard group.

2.8 Evaluation of prophylactic activity (Repository test)

Prophylactic activity of *M. paradisiaca* was determined using the method described by Peters (9) In this method, Fifty mice randomly divided into five groups of 10 mice was assigned to control group, artesunate treated, *P. berghei* infected and the experimental groups of 250 and 1000mg/kg/day body weight *Musa paradisiaca* leaf extract. The control were administered diet and water *adlibitum*, the artesunate treated was given 50 mg artesunate per kg body weight intraperitoneally while the experimental groups were administered with 250 and 1000mg/kg/day body weight *Musa paradisiaca* leaf extract and the last group was infected with *P. berghei*. Treatments were initiated on day 0 and continued until day 4; the mice were all infected with the parasite. Blood smears were then made from each mouse 72 hours after treatment (10) while the increase or decrease in parasitaemia was then determined.

2.9 Sample collection and analysis

The treatment was done for 10days, blood samples collected into plain containers and centrifuged at 1500rpmfor 10minutes. After centrifugation, the serum obtained was used for the analysis.

2.10 Catalase estimation

Estimation of Catalase was done by using spectrophotometric method (11). This assay method is based on the measurement of the hydrogen peroxide substrate remaining after the action of catalase. The catalase initially converts hydrogen peroxide to water and oxygen (catalytic pathway) and then stopped with addition of sodium azide while an aliquot of the reaction mix is then assayed for the amount of hydrogen peroxide remaining by a colorimetric method. The colorimetric method uses a substituted phenol (3,5-dichloro-2-hydroxybenzenesulfonic acid), which couples oxidatively to 4-aminoantipyrine in the presence of hydrogen peroxide and horseradish peroxidase (HRP) to give red quinoneimine dye (N-(4-antipyryl)-3-chloro-5-sulfonatep-benzoquinone monoimine) that absorbs at 520 nm.

Procedure: The assay reaction was performed at room temperature (25 °C). Into a test tube, 25ul of sample and 75ul of assay buffer were dispensed, then the reaction was started by addition of 25ul of substrate B to the test tube. It was mixed by inversion and incubated at 25°C for 15 minutes. 825ul of the stop solution was added and mixed. 10ul aliquot of the mixture was removed, added to another test tube and 1ml of chromogen reagent was added and mixed well. Then it was allowed for 15 minutes at room temperature for color development. Change in absorbance was read at 520nm wavelength.

2.11 Glutathione peroxidase (GPX) estimation

GPX estimation was carried out using Spectrophotometric method of Charmagnol *et al*., (11) as modified by Sigma Aldrich diagnostic. The Principle of this assay showed that Glutathione peroxidase catalyses the oxidation of Glutathione (GSH) by cumene hydroperoxides. The oxidized glutathione is converted to the reduced form in the presence of glutathione reductase

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and NADPH. In this reaction the NADPH is oxidized to $NADP^+$ simultaneously. The decrease in absorbance at 340nm is then measured. **Procedure**: 20ul of sample was added into a clean microcuvette and 20µl of distilled water into another cuvette to serve as reagent Blank, then 1ml of working reagent was added to each cuvette. 40ul of cumene hydroperoxide solution was added to each cuvette. It was mixed and initial absorbances of sample and reagent blank were read after 1 minute and timer was started simultaneously. It was read again after 1 and 2 minutes. The reagent blank value was subtracted from that of the sample.

2.12 Malondialdehyde (MDA) estimation

MDA estimation was done by Colorimetric method. The Principle of **t**his assay is based on the reaction of a chromogenic reagent, 2-thiobarbituric acid, with MDA at 25°C. One molecule of MDA reacts with 2 molecules of 2-thiobarbituric acid via a Knoevenagel-type condensation to yield a chromophore with absorbance maximum at 532 nm.

Procedure: The Malondialdehyde (MDA) estimation was done by estimating Free MDA and Total MDA. To estimate Free MDA**,** 200ul of standard, sample and 200ul of indicator solution were added into glass tubes labelled standards, samples and blank. This was followed by addition of 200ul of indicator solution to all the tubes, mixed well and allowed to react for 45 minutes at room temperature to develop a pink color that is stable for several hours at room temperature. The absorbance of the resulting solution was measured at 532 nm. Measurement of Total MDA required heat at 65˚C. 200ul of standard, sample and 200ul of indicator solution were added into glass tubes labelled standards, samples and blank, followed by addition of 200ul of indicator solution to all the tubes and mixed well. The content of sample tube was heated at 65˚C in waterbath for 45 minutes. The absorbance of the resulting solution was measured at 532 nm.

2.13 Reduced glutathione (GSH) estimation

GSH estimation was done using Spectrophotometric method of Ellman (13) as modified by Sigma Aldrich diagnostic. The spectrophotometric procedure showed that 5,5'-dithiobis- (2,-nitrobenzoic acid) is reduced by SH groups to form 1 mole of 2-nitro-5-mercaptobenzoic acid per mole of SH. The nitromercaptobenzoic acid anion has an intense yellow color which when measured at a wavelength of 412nm can be used to measure SH groups. **Procedure:** 100ul of standard and samples were dispensed into labelled microcuvette followed by addition of 880ul of GSH dilution buffer and 20ul of GSH chromogen to the microcuvette respectively in that order. The microcuvette, were mixed well and absorbance of resulting solution was measured at 412nm wavelength within 5 minutes and unknown concentration was determined using the absorbance and concentration of standard.

2.14 Statistical analysis

Data obtained were analysed using statistical package for social science version 21 employing statistical tools like t-test and analysis of variance (ANOVA). Results were expressed as Mean ± Standard Deviation (X±SD).The values of P<0.05 was considered significant.

3. Results

3.1 Catalase, GPX,MDA and glutathione concentrations in P.berghei infected mice treated with curative ethanol leaf extract of Musa paradisiaca

There was reduction in catalase (µ/L) activities by *P bergei from* 18.44±1.31 in control to 9.65±0.16 in *P bergei while* Artesunate, 250 mg curative and 1000 mg curative doses increased the catalase (µ/L)activities to 11.64±1.68, 12.83±0.43 and 12.39±0.67. Glutathione peroxidase (µ/L) activities was 63.58±0.62, 19.13±2.4, 42.24±7.15, 17.62±12.7 and 55.27±0.5 in Control, *P*

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bergei, Artesunate, 250 mg and 1000 mg curative respectively. There was reduction in Malondialdehyde (µ/m)by *P bergei from* 47.9±0.45 in control to 11.2±0.8in *P bergei with further decrease by* Artesunate, 250 mg curative and 1000 mg curative doses to 6.4±0.2, 6.0±0.1and 8.3±0.4. Glutathione (µ/m) was 1.05±0.35, 0.61±0.59, 0.67±0.53, 0.74±0.46 and 0.94±0.18in Control, *P bergei,* Artesunate, 250 mg curative and 1000 mg curative respectively (Table 1).

3.2: Catalase, GPX, MDA and glutathione concentrations in P.berghei infected mice treated with suppresive ethanol leaf extract of Musa paradisiaca

There was reduction in catalase (µ/L) activities by *P bergei from* 18.44±1.31 in control to 9.65±0.16 in *P bergei while* Artesunate, 250 mg and 1000 mg Suppressive doses were12.8± 2.2, 9.9± 0.3 and 11.54± 0.3. Glutathione peroxidase (µ/L) activities was 63.58±0.62, 19.13±2.4, 42.24±7.15, 17.62±12.7 and 55.27±0.5 in Control, *P bergei,* Artesunate, 250 mg and 1000 mg Suppressive respectively. There was reduction in Malondialdehyde (µ/m)by *P bergei from* 47.9±0.45 in control to 11.2±0.8in *P bergei with further decrease by* Artesunate, 250 mg and 1000 mg Suppressive doses to 6.4±0.2, 6.0±0.1and 8.3±0.4. Glutathione (µ/m) was 1.05±0.35, 0.61±0.59, 0.67±0.53, 0.74±0.46 and 0.94±0.18in Control, *P bergei,* Artesunate, 250 mg and 1000 mg Suppressive respectively(Table 2).

3.3: Catalase, GPX,MDA and glutathione concentrations in P.berghei infected mice treated with prophylactic ethanol leaf extract of Musa paradisiaca

There was reduction in catalase **(µ/L)** activities by *P bergei from* 18.44±1.31 in control to 9.65±0.16 in *P bergei* . Artesunate, 250 mg and 1000 mg prophylactic doses were 15.59±0.86, 12.91± 0.33 and 12.0± 0.3 respectively. **Glutathione peroxidase (µ/L) activities was** 63.58±0.62, 19.13±2.4, 37.8±5.2, 39.4± 0.3 and 34.5± 0.5 in Control, *P bergei,* Artesunate, 250 mg and 1000 mg prophylactic respectively. There was reduction in **Malondialdehyde (µ/m)**by *P bergei from* 47.9±0.45 in control to 11.2±0.8in *P bergei with further decrease by* Artesunate, 250 mg and 1000 mg prophylactic doses to 6.1± 0.5, 6.0±0.1and 5.9± 0.0. **Glutathione (µ/m) was** 1.05±0.35, 0.61±0.59, 0.74± 0.18, 0.68± 0.4and 0.81± 0.3in Control, *P bergei,* Artesunate, 250 mg and 1000 mg prophylactic respectively (Table 3).

4. Discussion

The *Musa paradisiaca* plant has been known to carry out some anti-oxidation function in the body when taken as herbs (2). The study was done to evaluate the concentration of some serum brain antioxidants like Catalase, Glutathione, Glutathione peroxidase and Malondialdehyde in mice infected with *Plasmodium berghei berghei* treated with artesunate and *Musa paradisiaca* ethanol leaf extracts given in doses of 250mg/kg body weight and 1000mg/kg body weight of different models of curative, prophylactic and suppressive treatments. Oxidative stress may play a central role in the onset of many diseases. Malondialdehyde (MDA) is a marker of lipid peroxidation (14).

The result of the study showed that *P bergei* caused reductions in Catalase, GPX, MDA and glutathione concentrations. This is suggestive that *P bergei* caused brain oxidative stress by reducing the brain antioxidants. Administration of curative ethanol leaf extract of *Musa paradisiaca* reduced the brain oxidative stress by increasing catalase and GPX. Free radicals are

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too short-lived to be detected directly in clinical systems, but oxygen free radicals react with lipids to produce lipid peroxidation products, which when measured serve as indirect biomarkers of *in vivo* oxidative stress status and related diseases. Administration of suppressive and prophylactic ethanol leaf extract of *Musa paradisiaca* also reduced the brain oxidative stress by increasing catalase and GPX. The extent of oxidative damage depends not only on ROS levels, but also on mechanisms of cellular antioxidant defenses. Low level of GSH, a molecule of critical importance in maintaining the stability of erythrocytes membranes, is related to cellular defense against xenobiotics and harmful compounds such as free radicals and hydroperoxides (15). Glutathione acts as the first line of defense against free radicals produced by antitumor molecules. Decreased GSH levels can be explained by a decrease in GSH synthesis and/or increased consumption to remove peroxides and xenobiotics (16).

The extract was effective in the reduction of the elevated levels of brain oxidants caused by the parasite. This suggests that the parasite increases the levels of these markers. This is suggestive that *Musa paradisiaca* leaf extract reduced the brain oxidation caused by *Plasmodium berghei berghei*. The study concludes that the curative, prophylactic and suppressive extracts of *Musa paradisiaca* extract caused reductions in the brain oxidative stress by increasing catalase and GPX. This study has shown that leaf extract of *Musa paradisiaca* has some brain antioxidants that protects the brain of *Plasmodium berghei* infected mice from free radicals

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Tables

Table 1: Catalase, GPX,MDA and glutathione concentrations in *P.berghei* infected mice treated with curative ethanol leaf extract of *Musa paradisiaca*

Group	Catalase (µ/L)	Glutathione peroxidase (µ/L)	Malondialdehyde (µ/m)	Glutathione (µ/m)
Control	18.44±1.31	63.58±0.62	47.9±0.45	1.05±0.35
P bergei	9.65±0.16*	19.13±2.4*	11.2±0.8*	0.61±0.59*
Artesunate	11.64±1.68*	42.24±7.15**	6.4±0.2*,**	0.67±0.53*
250 mg curative	12.83±0.43*	17.62±12.7*	6.0±0.1*,**	0.74±0.46*
1000 mg curative	12.39±0.67*	55.27±0.5**	8.3±0.4*	0.94±0.18*,**

*= Compare with the control
**= Compare with the *P.berghei* treated

Table 2: Catalase, GPX, MDA and glutathione concentrations in *P.berghei* infected mice treated with suppressive ethanol leaf extract of *Musa paradisiaca*

Group	Catalase (µ/L)	Glutathione peroxidase (µ/L)	Malondialdehyde (µ/m)	Glutathione (µ/m)
Control	18.44±1.31	63.58±0.62	47.9±0.45	1.05±0.35
P bergei	9.65±0.16*,	19.13±2.4*	11.2±0.8*,	0.61±0.59*
Artesunate	12.8± 2.2*,**	47.3±1.2*,**	5.6± 0.1*,**	0.84± 0.31**

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250 mg Suppressive	9.9± 0.3*	48.4±0.01*,**	5.9± 0.1*,**	0.71± 0.9*
1000 mg Suppressive	11.54± 0.3*,**	31.0±11.3*,**	4.1±1.0*,**	0.94±0.19**

*= Compare with the control
**= Compare with the *P.berghei* treated

Table 3: Catalase, GPX,MDA and glutathione concentrations in *P.berghei* infected mice treated with prophylactic ethanol leaf extract of *Musa paradisiaca*

Group	Catalase (μ/L)	Glutathione peroxidase (μ/L)	Malondialdehyde (μ/m)	Glutathione (μ/m)
Control	18.44±1.31	63.58±0.62	47.9±0.45	1.05±0.35
P bergei	9.65±0.16*	19.13±2.4*	11.2±0.8*	0.61±0.59*
Artesunate	15.59±0.86*,**	37.8±5.2*,**	6.1± 0.5*,**	0.74± 0.18*
250 mg prophylactic	12.91± 0.33*,**	39.4± 0.3*,**	6.0± 0.1*,**	0.68± 0.4*
1000 mg	12.0± 0.3*	34.5± 0.5*,**	5.9± 0.0*,**	0.81± 0.3**

*= Compare with the control
**= Compare with the *P.berghei* treated

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