

Research Article

## Hematological Evaluation of Ethanolic Extract of *Ficus Sur* in *Plasmodium Berghei* Infected Albino Rats

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### ABSTRACT

#### Background

Malaria, caused by *Plasmodium berghei*, leads to severe hematological alterations such as anemia, leukocytosis, and thrombocytopenia. *Ficus sur*, known for its anti-inflammatory and antioxidant properties, has been traditionally used for various ailments.

#### Objective

This research evaluated the haematological impacts of the ethanolic extract of *Ficus sur* in albino rats infected with *P. berghei*.

#### Methods

Thirty male albino rats (150–200g) were allocated into five groups (n=6). Group 1 (normal control) remained uninfected, but Groups 2–5 were infected with *P. berghei* (NK 65 strain). Group 2 (parasite control) received no intervention, whereas Groups 3–5 were administered treatments from days 4 to 8 with the ethanolic extract of *Ficus sur* at dosages of 400 mg/kg (high dose), 200 mg/kg (low dose), and 10 mg/kg of chloroquine, respectively. Hematological parameters, including RBC, WBC, PCV, Hb, platelets, and WBC differential counts, were analyzed on days 4 and 8 post-infection using standard hematological methods.

## Results

By day 4, the N.C. group had the highest RBC ( $6.14 \pm 0.05$ ), PCV ( $69.95 \pm 0.62$ ), Hb ( $17.96 \pm 0.33$ ), and Platelets ( $4.36 \pm 0.03$ ), while P.C. had the lowest RBC ( $2.84 \pm 0.06$ ), PCV ( $32.54 \pm 1.10$ ), Hb ( $7.58 \pm 0.27$ ), and Platelets ( $3.35 \pm 0.04$ ) but the highest WBC ( $14.41 \pm 0.18$ ), neutrophils ( $26.70 \pm 0.70$ ), and lymphocytes ( $80.66 \pm 0.87$ ). By day 8, P.C. RBC dropped further ( $2.62 \pm 0.05$ ), PCV ( $25.61 \pm 0.69$ ), and Hb ( $5.36 \pm 0.22$ ), while N.C. and S.C. maintained high RBC and platelets ( $\sim 4.32-4.35$ ).

## Conclusions

The ethanolic extract of *Ficus sur* improved RBC indices and modulated WBC differentials, suggesting its potential as an adjunct therapy for malaria-induced hematological disorders.

**Keywords:** Arteriov *Ficus sur*; *Plasmodium berghei*; Anemia; Hematological; Malaria; Leukocytosis

## INTRODUCTION

An infected Anopheles mosquito's bite can transmit Plasmodium parasites, responsible for the lethal disease malaria [1]. Neglected symptoms of the disease, such as elevated temperature, chills, and anemia, may result in severe repercussions and potential mortality (WHO, 2020). In 2020, the World Health Organization recorded 241 million documented cases of malaria, resulting in 627,000 global fatalities. Africa had the majority of cases and fatalities, with Nigeria being one of the nation's most severely affected by the pandemic [1].

In severe instances of malaria, anemia frequently occurs [2]. Malaria-induced typical normocytic and normochromic anemia is characterized by diminished hemoglobin, packed cell volume (PCV), and red blood cell (RBC) counts [3]. Malaria induces anemia via a complex, multifactorial mechanism involving immune system activation, inhibition of erythropoiesis, and the destruction of red blood cells (White, 2018). Malaria infection can result in severe and enduring effects on the blood, particularly in pregnant women and children [1]. Malaria infection induces anemia and elevates the risk of infection and hemorrhage due to alterations in white blood cell (WBC) and platelet counts [3].

Nigeria, a tropical African nation, hosts the *Ficus sur* tree, which belongs to the Moraceae family [4] observes that the herb has a longstanding application in traditional medicine, especially for malaria treatment. The extracts of the plant have been linked to several biological activities, including anti-inflammatory, antioxidant, and antibacterial effects [5]. Specifically, there is a deficiency of studies about the hematological effects of *Ficus sur* preparations in the context of malaria infection. Certain plant extracts demonstrate hematoprotective properties, suggesting their potential utility as adjuncts to existing therapies for malaria-induced anemia [3, 5].

This research aimed to investigate the impact of an ethanolic extract of *Ficus sur* on the hemoglobin levels of albino rats infected with *Plasmodium berghei*. Researchers anticipate that this study will contribute to the exploration of novel adjunctive therapies for malaria-related anemia by elucidating the potential benefits of *Ficus sur* in malaria management.



## METHODS

### *Collection and Preparation of Plant Extract*

The freshly picked leaves were sourced from a location in Amassoma, Bayelsa State, Nigeria, which is renowned for its profusion of the *Ficur sur* plant. The plant was identified by a Prof Inetiminebi Arrow Ogidi from the Department of Plant Science at Niger Delta University in Bayelsa State, Nigeria. The voucher specimen with the number NDUP/24/11 is located in the Herbarium Unit of Niger Delta University, which is situated on Wilberforce Island in Bayelsa State, Nigeria. The department is a component of the pharmacology faculty. The leaves were collected in large quantities and allowed to dry in a shaded area at room temperature for a period of two weeks. They were then reduced to a coarse powder. The powder, weighing 500 grams, was dissolved in 2 liters of methanol and left to stand for 72 hours, stirring occasionally. We used cheese cloth to filter the extract, and then we used a rotary evaporator set to 50 °C to evaporate the filtrate. Distilled water was added to the dry residue as needed to make it liquid again.

### *Experimental Animals*

Thirty male albino Wistar rats weighing 150-200g were obtained from the animal house of the Department of Pharmacology at the University of Port Harcourt in Rivers State. The rats were housed in the Faculty of Basic Clinical Sciences at the College of Health Sciences. It seemed like the rats were doing fine. These rodents were kept in standard rat cages at Niger Delta University's College of Health Sciences and Faculty of Basic Clinical Sciences on Wilberforce Island in Bayelsa State. The animals were allowed 14 days to acclimatize in a standard laboratory environment before the trial started. All they needed was a 12-hour light/dark cycle, lots of fresh air, commercial pelletised grower's mash (Delta Feeds), water when it was needed, and that was it.

### *Malaria Parasites*

*Plasmodium berghei* NK 65 strain collected in Lagos, Nigeria, at the Nigeria Institute of Medical Research (NIMR). Niger Delta University's pharmacology lab on Wilberforce Island, Bayelsa State, Nigeria, received the NIMR-passaged Swiss mice. This facility is part of the College of Health's Faculty of Clinical Science.

### *Inoculum Preparation*

Parasite-infected erythrocytes with a peripheral parasitemia of at least 20% were obtained from infected Swiss rodents through cardiac puncture in a heparin-coated tube. We were able to obtain a final inoculum concentration of 0.2 ml containing 10<sup>6</sup> parasitized red blood cells (RBCs) by diluting the stock with physiological saline [6].

### *Experimental Design*

Each of the five groups of six male Wistar rats was randomly assigned to a group after two weeks of acclimation. Group 1, which was not infected with plasmodium, acted as the control group. Groups 2 to 5, on the other hand, were intraperitoneally inoculated with 0.2 ml containing 1 × 10<sup>6</sup> infected erythrocytes of the *Plasmodium berghei* (NK 65 strain). Parasite control group 2 did not

undergo any therapy. From day 4 to day 8, groups 3, 4, and 5 were given different doses of the extract: 400 mg/kg b. w for the high dosage, 200 mg/kg b. w for the low dose, and 10 mg/kg b. w for chloroquine, respectively. The animals' submandibular blood was drawn on days 0, 4, and 8 for the purpose of testing parasitemia and blood count.

### ***Parasitemia Estimation***

The rats' tail veins were used to extract a small volume of blood (5  $\mu$ l), and a thin smear was made by placing a drop of blood at the edge of a clean microscope slide. To make a thin film, another slide was held at a 45° angle and utilized as a spreader to distribute the drop uniformly. In a dust-free atmosphere, the smear was allowed to air-dry entirely. After the slide had dried, it was fixed by immersing it in 100% methanol for approximately 30 seconds. After that, it was allowed to air-dry once again. Giemsa stain was applied to the fixed smear in a 1:10 dilution with buffered water (pH 7.2), and the smear was let to sit for 15 minutes. Slight rinsing with buffered water after staining, followed by tilting the slide to drain excess liquid and letting it air dry. Using separate fields, 1,000 RBCs were counted, including both total and parasitized RBCs [7]. The formula was used to compute the parasitemia percentage:

Parasitemia (%) = Number of infected RBC / Total number of RBCs counted X 100

### ***Packed cell volume (Hematocrit)***

The blood sample was drawn into the capillary tubes by capillary attraction until it reached three quarters of the tubes. After securing one end of the capillary tube with plasticine, the other end was inserted into a micro hematocrit centrifuge. To estimate the PCV, the samples were centrifuged at 3000 rpm for 5 minutes. The capillary tubes were then removed and inserted in the groove of a haematocrit reader. Every sample underwent this procedure [3].

### ***Hemoglobin Estimation***

A freshly made 0.1M HCl was filled to the 10 mark in the hemometer's dilution tube, and a blood sample was drawn from the EDTA bottle up to the 20mm mark using the hemometer pipette. A 20 mm blood sample was carefully blown into the acid in the hemometer dilution tube after the pipette tip was cleaned to avoid any extra sample. To make sure everything was mixed well and the red cells could hemolyze and produce acid hematocrit, the pipette was carefully sucked up and down twice. After precisely 5 minutes of letting the mixture sit, distilled water was added drop by drop while stirring with a glass rod. Under bright, diffuse day light, with white paper as a background, the colour of the hemolysed blood was compared to that of the hemometer standard. The meniscus level (X1%) was determined by continuously doing this until the dilution colour was slightly darker than the hemometer reference. When the dilution is just a hair lighter than the hemometer standard, more water is added. Additionally, the meniscus level reading (X2%) was recorded. The readings were averaged to get the percentage. A hemoglobin concentration of 14 g/100 ml of blood is equivalent to 100% on the Sahli scale, thus we can simply convert the mean (X%) to g/100 ml by using proportion [3].

### ***Red blood cell count***

The blood sample was accurately collected up to the 0.5 point on the pipette with a gentle compression of the mouthpiece. In order to prevent the transmission of cells to the diluting fluid, it was imperative to remove the superfluous blood from the pipette's exterior. We absorbed the diluting solution until it reached the 101-point mark. The sample and diluent are combined as the pipette is spun between the thumb and fingertips. The blending process is maintained for approximately two minutes to ensure that the cells are distributed evenly and the blood and diluting fluid are mixed properly. The pipette was maintained in a horizontal position for an additional two to three minutes. In order to guarantee that the clean hemocytometer cover slip was securely fastened to the counting chamber, the shoulders of the chamber where the cover slip was located were saturated. The pipette is angled at a 45-degree angle after 2 or 3 droplets of the undiluted fluid have been removed, and the counting chamber is placed on top of the pipette tip to facilitate the passage of the mixture between the cover slip and the counting chamber. The cells were examined under a microscope at a magnification of x40 after they had settled in the counting chamber for approximately three minutes [4]. The following was obtained after tallying the cells in five small squares:

#### Calculation

$$\text{RBC Count} = \frac{N \times DF \times 10^6}{A \times D}$$

N = number of cells counted

DF= the dilution factor (201)

10<sup>6</sup> = converts to cells per liter

A= area of chamber counted (0.04mm<sup>2</sup> x 5 = 0.2mm<sup>2</sup>)

D = depth of chamber (0.1mm)

### ***Platelet Count***

The blood sample was accurately collected up to the 0.5 point on the pipette with a gentle compression of the mouthpiece. In order to prevent the transmission of cells to the diluting fluid, it was imperative to remove the superfluous blood from the pipette's exterior. The diluting fluid was absorbed until the eleventh mark. The sample and diluent are combined as the pipette is spun between the thumb and fingertips. The blending process is maintained for approximately two minutes to ensure that the cells are distributed evenly, and the blood and diluting fluid are mixed properly. Following this, the pipette was left in a horizontal position for an additional 2-3 minutes. In order to guarantee that the clean hemocytometer cover slip was securely fastened to the counting chamber, the shoulders of the chamber where the cover slip was located were saturated. The pipette is angled at a 45-degree angle after 2 or 3 droplets of the undiluted fluid have been removed, and the counting chamber is placed on top of the pipette tip to facilitate the passage of the mixture between the cover slip and the counting chamber. The cells were examined under a microscope at

a magnification of x40 after they had settled in the counting chamber for approximately three minutes (Ogidi et al., 2020). The result of counting the cells in five small squares is as follows:

Calculation

$$\text{Platelet Count} = \frac{N \times DF \times 10^6}{A \times D}$$

N = number of cells counted

DF= the dilution factor (20)

$10^6$  = converts to cells per liter

A= area of chamber counted ( $0.04\text{mm}^2 \times 5 = 0.2\text{mm}^2$ )

D = depth of chamber (0.1mm)

### ***White Blood Cell Count***

The blood sample was accurately collected up to the 0.5 point on the pipette with a gentle compression of the mouthpiece. In order to prevent the transmission of cells to the diluting fluid, it was imperative to remove the superfluous blood from the pipette's exterior. The diluting fluid was absorbed until the eleventh mark. The sample and diluent are combined as the pipette is spun between the thumb and fingertips. The blending process is maintained for approximately two minutes to ensure that the cells are distributed evenly, and the blood and diluting fluid are mixed properly. The pipette was maintained in a horizontal position for an additional two to three minutes. In order to guarantee that the clean hemocytometer cover slip was securely fastened to the counting chamber, the shoulders of the chamber where the cover slip was located were saturated. The pipette is angled at a 45-degree angle after 2 or 3 droplets of the undiluted fluid have been removed, and the counting chamber is placed on top of the pipette tip to facilitate the passage of the mixture between the cover slip and the counting chamber. The cells were examined under a 10x microscope after they had settled in the counting chamber for approximately three minutes [7]. The following was obtained after tallying the cells in four large squares:

Calculation

$$\text{WBC Count} = \frac{N \times DF \times 10^6}{A \times D}$$

N = number of cells counted

DF= the dilution factor (20)

$10^6$  = converts to cells per liter

A= area of chamber counted ( $1\text{mm}^2 \times 5 = 5\text{mm}^2$ )

D = depth of chamber (0.1mm)

## RESULTS

### Blood Count Results

On Day 4, the N.C. group had the highest RBC ( $6.14 \pm 0.05$ ), PCV ( $69.95 \pm 0.62$ ), Hb ( $17.96 \pm 0.33$ ), and Platelets ( $4.36 \pm 0.03$ ), while the P.C. group had the lowest RBC ( $2.84 \pm 0.06$ ), PCV ( $32.54 \pm 1.10$ ), Hb ( $7.58 \pm 0.27$ ), and Platelets ( $3.35 \pm 0.04$ ) but the highest WBC ( $14.41 \pm 0.18$ ). By Day 8, N.C. ( $6.14 \pm 0.06$ ) and S.C. ( $6.10 \pm 0.04$ ) maintained high RBC, while P.C. RBC dropped further ( $2.62 \pm 0.05$ ), with PCV ( $25.61 \pm 0.69$ ) and Hb ( $5.36 \pm 0.22$ ) significantly reduced. Platelets were highest in N.C. ( $4.32 \pm 0.08$ ) and S.C. ( $4.35 \pm 0.03$ ), while P.C. had the lowest ( $3.14 \pm 0.02$ ), confirming severe hematological impairment in the P.C. group as shown in Tables 1 and 2 (Figures 1-10).

Table 1: Blood count day 4

	GROUPS					p-value	Inference
	N.C	P.C	H.D.E	L.D.E	S.C		
RBC (X $10^6/\mu\text{L}$ )	$6.14 \pm 0.05^b$	$2.84 \pm 0.06^a$	$4.44 \pm 0.10^{ab}$	$3.69 \pm 0.08^{ab}$	$5.37 \pm 0.13^{ab}$	0.000	Significant
WBC ( $\times 10^3/\text{L}$ )	$5.32 \pm 0.06^b$	$14.41 \pm 0.18^a$	$7.70 \pm 0.20^{ab}$	$10.79 \pm 0.20^{ab}$	$5.55 \pm 0.12^b$	0.000	Significant
PCV (%)	$69.95 \pm 0.62^b$	$32.54 \pm 1.10^a$	$54.87 \pm 0.77^{ab}$	$44.03 \pm 0.82^{ab}$	$64.04 \pm 1.40^{ab}$	0.000	Significant
Hb (g/dl)	$17.96 \pm 0.33^b$	$7.58 \pm 0.27^a$	$14.76 \pm 0.24^{ab}$	$11.70 \pm 0.23^{ab}$	$17.95 \pm 0.45^b$	0.000	Significant
Platelets ( $\times 10^5/\mu\text{L}$ )	$4.36 \pm 0.03^b$	$3.35 \pm 0.04^a$	$3.81 \pm 0.05^{ab}$	$3.50 \pm 0.04^a$	$4.22 \pm 0.04^b$	0.000	Significant

The values are presented as Mean  $\pm$  SEM for each cohort, where SEM represents the Standard Error of Mean. In comparison to Groups N.C. and P.C., the superscript 'a' and the superscript 'b' indicate the difference ( $p < 0.05$ ). The statistical level of significance (P) was determined using a one-way ANOVA and a Tukey post hoc test.

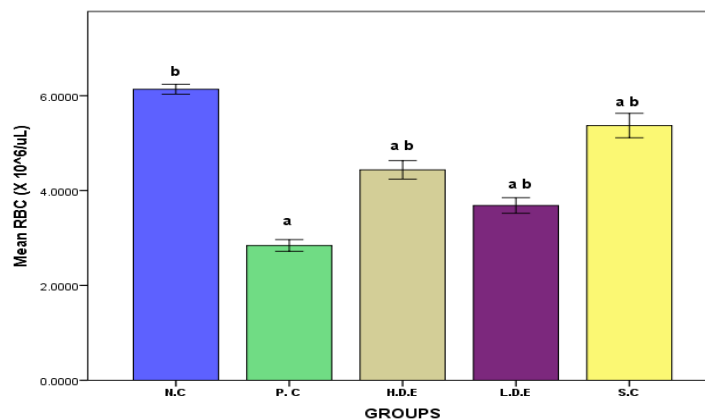
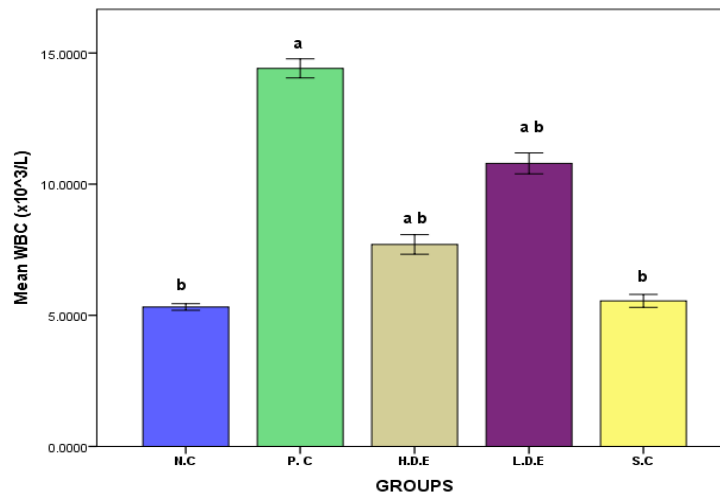
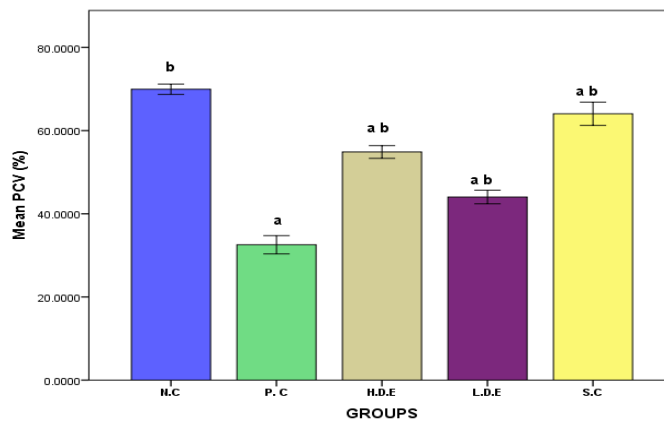


Figure 1. The effects of different treatment groups on RBC levels on day 4



*Figure 2.* The effects of different treatment groups on WBC levels on day 4



*Figure 3.* The effects of different treatment groups on PCV levels on day 4

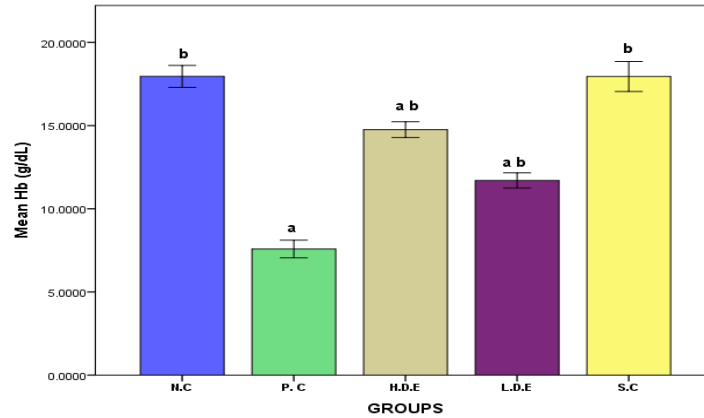


Figure 4. The effects of different treatment groups on Hb levels on day 4

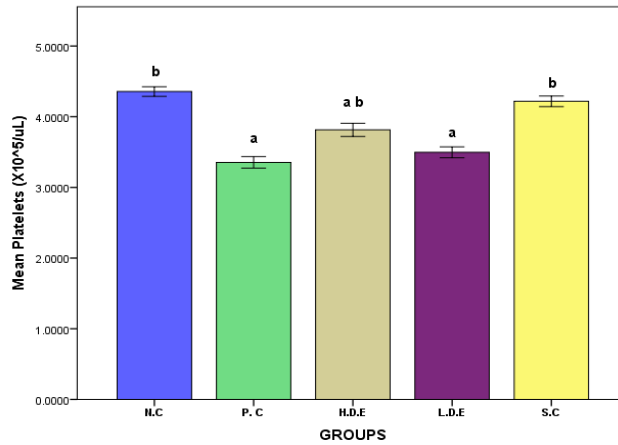


Figure 5. The effects of different treatment groups on platelets levels on day 4

Table 2: Blood count day 8

	GROUPS					p-value	Inference
	N.C	P.C	H.D.E	L.D.E	S.C		
RBC (X 10 <sup>6</sup> /μL)	6.14±0.06b	2.62±0.05a	5.52±0.16ab	4.18±0.04ab	6.10±0.04b	0.000	Significant
WBC (x10 <sup>3</sup> /L)	5.28±0.05b	16.98±0.14a	5.74±0.10ab	7.84±0.06ab	5.29±0.04b	0.000	Significant
PCV (%)	70,66±0.59b	25.61±0.69a	60.05±0.43ab	50.83±0.86ab	69.14±0.70b	0.000	Significant
Hb (g/dl)	17.92±0.26b	5.36±0.22a	16.03±0.17ab	12.74±0.50ab	18.66±0.10b	0.000	Significant
Platelets (X10 <sup>5</sup> /μL)	4.32±0.08b	3.14±0.02a	4.24±0.03b	3.63±0.04ab	4.35±0.03b	0.000	Significant

The values are presented as Mean  $\pm$  SEM for each cohort, where SEM represents the Standard Error of Mean. In comparison to Groups N.C. and P.C., the superscript 'a' and the superscript 'b' indicate the difference ( $p < 0.05$ ). The statistical level of significance (P) was determined using a one-way ANOVA and a Tukey post hoc test.

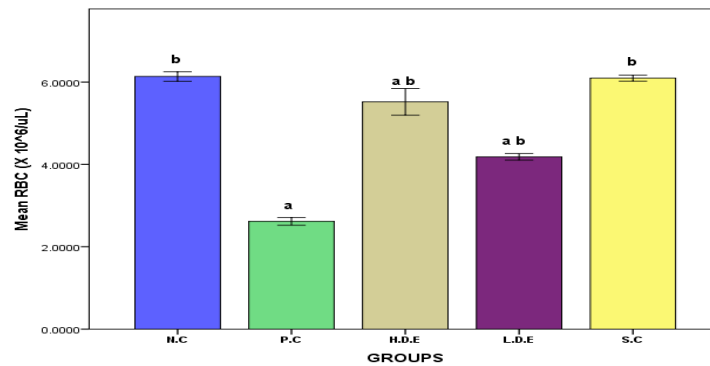


Figure 6. The effects of different treatment groups on RBC levels on day 8

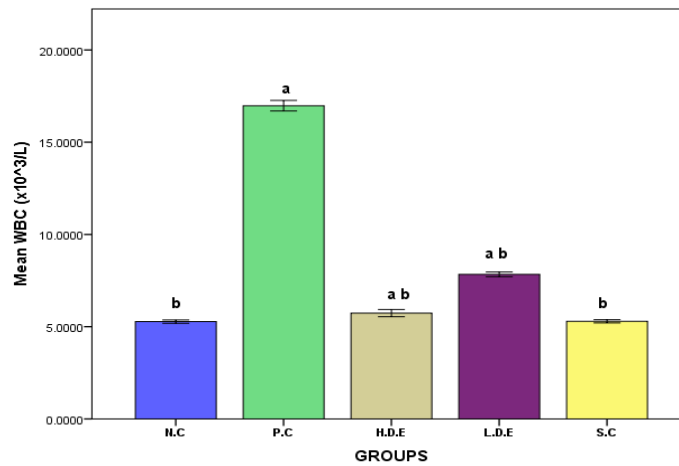


Figure 7. The effects of different treatment groups on WBC levels on day 8

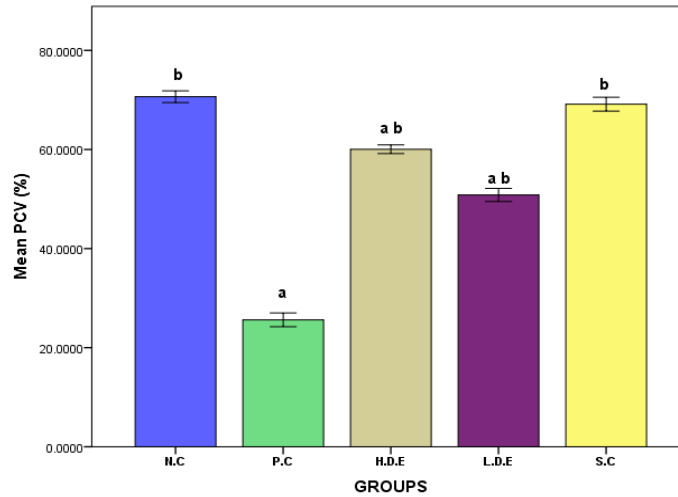


Figure 8. The effects of different treatment groups on PCV levels on day 8

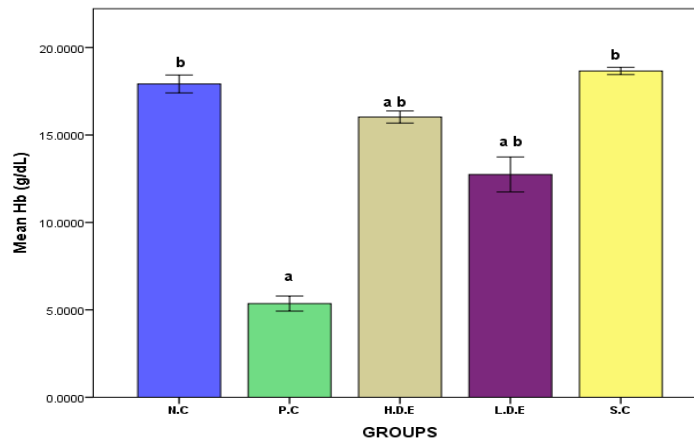


Figure 9. The effects of different treatment groups on Hb levels on day 8

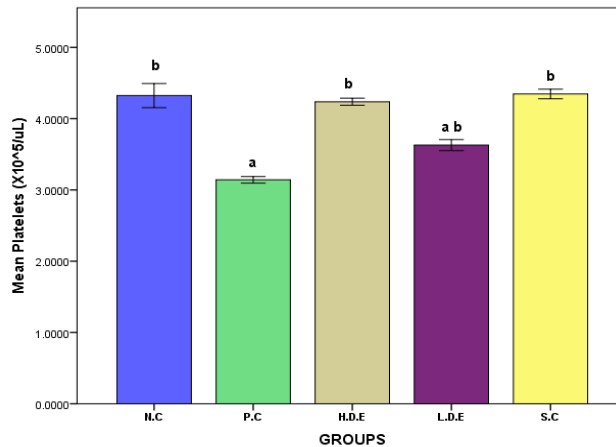


Figure 10. The effects of different treatment groups on platelets levels on day 8

### White Blood Cell Results

Tables 3 and 4 shows that on day 4, the P.C. group had the highest neutrophils (26.70±0.70), lymphocytes (80.66±0.87), monocytes (2.00±0.04), and eosinophils (3.55±0.07), while N.C. and S.C. had the lowest neutrophils (~18.81–20.06), lymphocytes (~66.05–72.58), monocytes (~1.42–1.53), and eosinophils (2.95±0.02). By Day 8, the P.C. group further increased in neutrophils (32.66±0.79), lymphocytes (85.23±0.54), monocytes (2.20±0.09), and eosinophils (3.60±0.07), while N.C. and S.C. maintained lower neutrophil (~18.24–18.69), lymphocyte (~66.24–67.04), and monocyte (~1.41) levels. The P.C. group had the lowest basophil count on both days (1.51±0.03; 1.58±0.03), whereas N.C. and S.C. had the highest (~2.02–2.12) (Figures 11-20).

Table 3: WBC Differential count day 4

	GROUPS					p-value	Inference
	N.C	P.C	H.D.E	L.D.E	S.C		
Neutrophil %	18.81±0.45b	26.70±0.70a	22.31±0.44ab	25.66±0.21a	20.06±0.50b	0.000	Significant
Lymphocytes %	66.05±2.31b	80.66±0.87a	74.62±0.54ab	78.35±0.77a	72.58±0.72b	0.000	Significant
Monocytes	1.42±0.04b	2.00±0.04a	1.59±0.04a b	1.88±0.02a	1.53±0.02b	0.000	Significant
Basophil %	2.08±0.05b	1.51±0.03a	1.78±0.02ab	1.63±0.01a	2.02±0.03b	0.000	Significant
Eosinophil %	3.00±0.06b	3.55±0.07a	3.17±0.04b	3.28±0.01ab	2.95±0.02b	0.000	Significant

The values are presented as Mean ± SEM for each cohort, where SEM represents the Standard Error of Mean. In comparison to Groups N.C. and P.C., the superscript 'a' and the superscript 'b' indicate the difference (p<0.05). The statistical level of significance (P) was determined using a one-way ANOVA and a Tukey post hoc test.

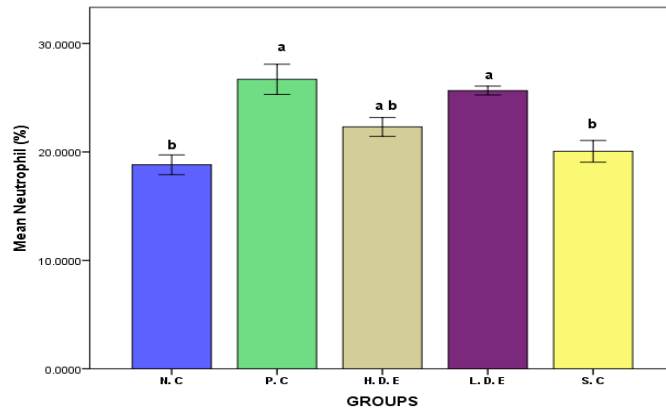


Figure 11. The effects of different treatment groups on Mean Neutrophil Percentage levels on day 4

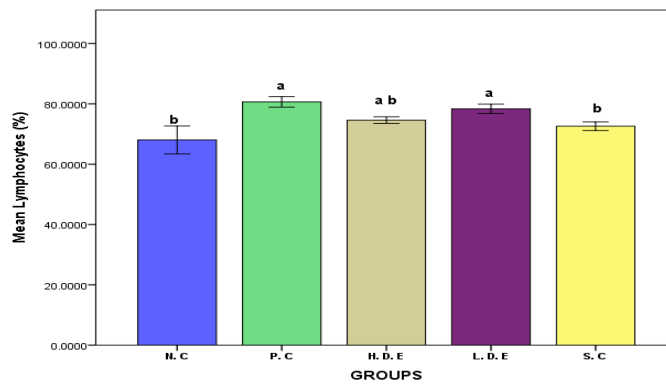


Figure 12. The effects of different treatment groups on Mean lymphocyte Percentage levels on day 4

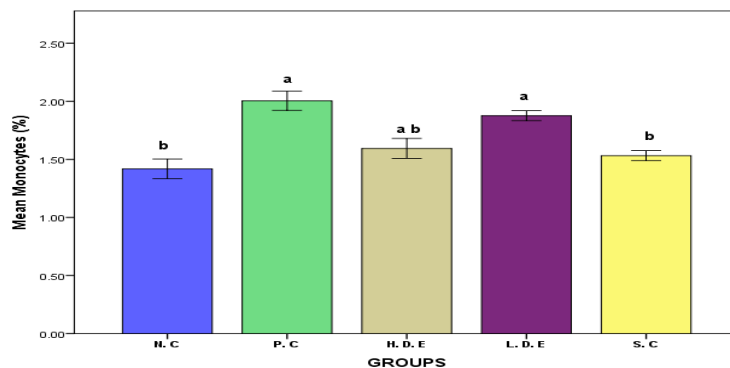


Figure 13. The effects of different treatment groups on Mean Monocyte Percentage levels on day 4

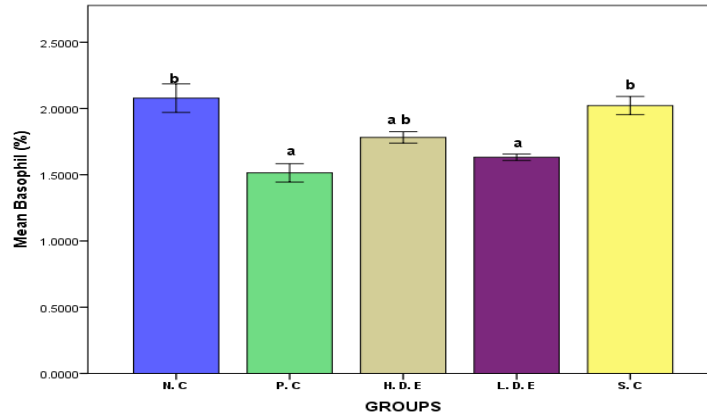


Figure 14. The effects of different treatment groups on Mean basophil Percentage levels on day 4

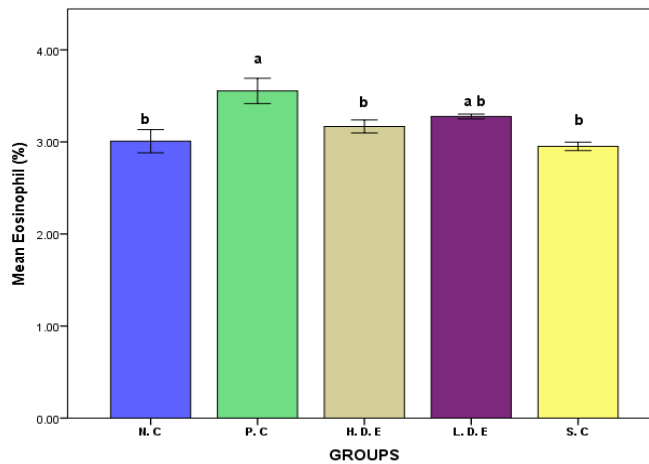


Figure 15. The effects of different treatment groups on mean eosinophil percentage levels on day 4

Table 4: WBC Differential count day 8

	GROUPS					p-value	Inference
	N.C	P.C	H.D.E	L.D.E	S.C		
Neutrophil %	18.69±0.42b	32.66±0.79a	20.29±0.41b	29.37±0.59ab	18.24±0.21b	0.000	Significant
Lymphocytes %	67.04±1.53b	85.23±0.54a	70.37±0.47b	75.65±0.83ab	66.24±0.73b	0.000	Significant
Monocytes	1.41±0.03b	2.20±0.09a	1.53±0.03b	1.71±0.02ab	1.41±0.01b	0.000	Significant
Basophil %	2.12±0.06b	1.58±0.03a	1.81±0.02ab	1.68±0.01a	2.05±0.02b	0.000	Significant
Eosinophil %	3.01±0.06b	3.60±0.07a	3.21±0.03b	3.26±0.03ab	2.90±0.03b	0.000	Significant

The values are presented as Mean  $\pm$  SEM for each cohort, where SEM represents the Standard Error of Mean. In comparison to Groups N.C. and P.C., the superscript 'a' and the superscript 'b' indicate the difference ( $p < 0.05$ ). The statistical level of significance (P) was determined using a one-way ANOVA and a Tukey post hoc test.

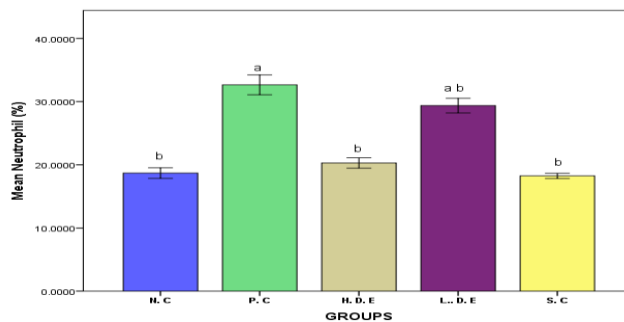


Figure 16. The effects of different treatment groups on Mean Neutrophil Percentage levels on day 8

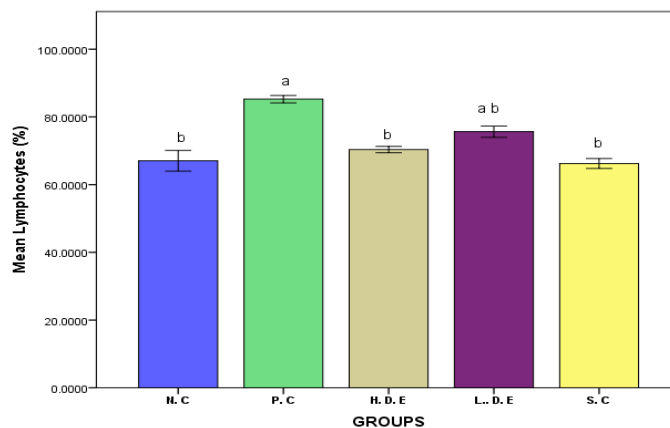


Figure 17. The effects of different treatment groups on Mean lymphocyte Percentage levels on day 8

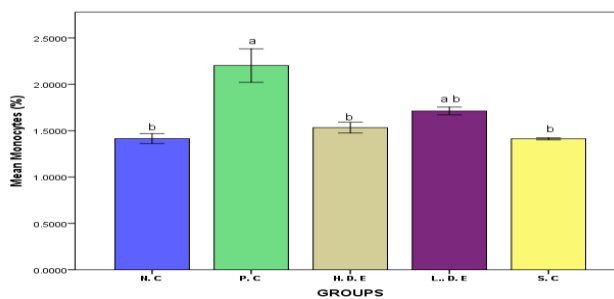


Figure 18. The effects of different treatment groups on Mean monocyte Percentage levels on day 8

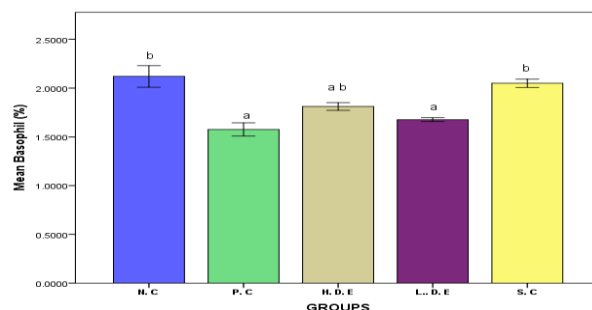


Figure 19. The effects of different treatment groups on Mean basophil Percentage levels on day 8

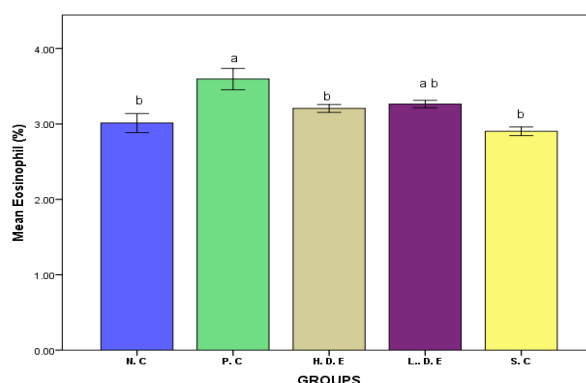


Figure 20. The effects of different treatment groups on Mean eosinophil Percentage levels on day 8

## DISCUSSION

The results of the blood count analysis conducted on days 4 and 8 are presented in Tables 1 and 2, respectively. The hydroethanolic extract of *Ficus sur* (H.D.E.) and its low dose extract (L.D.E.) dramatically modified the hematological parameters of the infected animals, as indicated by the findings. On day 4, red blood cell (RBC), packed cell volume (PCV), and hemoglobin (Hb) levels were markedly diminished in the positive control (P.C) group relative to the negative control (N.C) group, indicating that malaria infection induces anaemia [8]. In contrast to the P.C. group, the H.D.E. and L.D.E. groups demonstrated statistically significant elevations in RBC count, PCV, and Hb levels, suggesting that the extracts may have a hematoprotective effect.

The immune system was stimulated due to malaria infection, as evidenced by a markedly elevated white blood cell (WBC) count in the P.C group relative to the N.C group [9]. A significant reduction in white blood cell count was observed in the H.D.E. and L.D.E. groups relative to the P.C. group, suggesting that the extracts may exert an immunomodulatory effect. White (2018) reported that the platelet count in the P.C group was significantly lower than in the N.C group, indicating that malaria infection induces thrombocytopenia. The extracts seem to possess a

thromboprotective effect, as the groups administered H.D.E and L.D.E demonstrated markedly elevated platelet counts compared to the P.C group.

On day 8, persistent anaemia resulting from malaria infection was evidenced by the P.C groups significantly reduced red blood cell count, PCV, and Hb levels compared to the N.C group (Afolabi, 2018). The H.D.E. and L.D.E. groups exhibited significant enhancements in red blood cell count, platelet count, and hemoglobin level compared to the P.C. group, suggesting that the extracts may have a sustained hematoprotective effect. The elevated white blood cell count in the P.C group relative to the N.C group indicates that the immune system remains actively engaged in response to the malaria infection [10]. The white blood cell (WBC) count was markedly diminished in the H.D.E. and L.D.E. groups relative to the P.C. group, suggesting that the extracts may exert a prolonged influence on immunomodulation. White (2014) reported that the platelet count in the P.C group was significantly lower than in the N.C group, indicating that malaria infection exerts a persistent thrombocytopenic effect. The platelet counts in the groups administered H.D.E and L.D.E were considerably elevated compared to the P.C. group, suggesting that the extracts may confer a prolonged thromboprotective effect.

The differential count of white blood cells (WBCs) on days 4 and 8 is presented in Tables 3 and 4, respectively. The hydroethanolic extract of *Ficus sur* (H.D.E.) and its low dose extract (L.D.E.) considerably modified the white blood cell differential count characteristics in the infected animals, as indicated by the data. The immune system was activated in response to malaria infection on day 4, as the positive control (P.C) group demonstrated significantly elevated percentages of neutrophils and lymphocytes relative to the negative control (N.C) group (White, 2018). Notably, in comparison to the P.C group, the groups administered H.D.E and L.D.E demonstrated markedly reduced percentages of neutrophils and lymphocytes, suggesting that the extracts may influence immune system modulation. Other demonstrated that malaria infection activates the mononuclear phagocyte system, evidenced by a significantly elevated monocyte percentage in the P.C group relative to the N.C group [12]. The extracts seem to exert an anti-inflammatory effect, as the groups administered H.D.E and L.D.E had markedly reduced monocyte percentages relative to the P.C group. Other reported that the percentages of basophils and eosinophils were significantly diminished in the P.C group relative to the N.C group, indicating that the allergic response is inhibited during malaria infection [13]. The extracts seem to possess an immunoenhancing effect, as the groups administered H.D.E and L.D.E demonstrated markedly elevated percentages of basophils and eosinophils compared to the P.C group.

The P.C group exhibited persistent immune system activation in response to malaria infection on day 8, showing significantly elevated percentages of neutrophils and lymphocytes compared to the N.C group [13]. In contrast to the P.C. group, the H.D.E. and L.D.E. groups exhibited significantly reduced percentages of neutrophils and lymphocytes, suggesting that the extracts may have a prolonged immunomodulatory effect. The mononuclear phagocyte system's sustained activation in response to malaria infection was evidenced by a significantly greater proportion of monocytes in the P.C group relative to the N.C group [14]. The H.D.E. and L.D.E. groups exhibited a significant reduction in monocyte percentage relative to the P.C. group, suggesting that the extracts may possess enduring anti-inflammatory properties. Other reported that the proportions of

basophils and eosinophils were significantly decreased in the P.C group relative to the N.C group, indicating a persistent suppression of the allergic response during malaria infection. In comparison to the P.C. group, the groups administered H.D.E and L.D.E demonstrated markedly elevated percentages of basophils and eosinophils, suggesting that the extracts may exert a prolonged effect on the immune system [13].

## CONCLUSIONS

This work highlights the considerable hematological dysfunction induced by *Plasmodium berghei* infection, as evidenced in the parasite control group. By Day 4, the untreated infected group displayed significant declines in red blood cell count, packed cell volume, hemoglobin concentration, and platelet count, indicating the emergence of anemia and thrombocytopenia. Conversely, the normal control group exhibited optimum hematological values, signifying stable blood composition. The infected group exhibited a significant elevation in white blood cell count, especially neutrophils and lymphocytes, indicating an active immunological response to address the infection. By Day 8, the hematological deterioration in the untreated infected cohort became increasingly evident, with additional decreases in red blood cell count, packed cell volume, and hemoglobin concentrations. Simultaneously, the normal and standard control groups had steady red blood cell and platelet counts, indicating resistance to malaria-induced hematological damage. The persistent increase of neutrophils and lymphocytes in the infected cohort indicates ongoing immunological activation, although the persistently low basophil levels reflect a modified immune response. These data confirm the significant effect of malaria on hematological parameters and the body's continuous attempts to combat the infection.

*Plasmodium berghei* infection results in notable hematological abnormalities, including anemia, elevated leukocyte counts, and diminished platelet levels. The capacity of the normal and standard control groups to maintain stable blood parameters indicates that therapeutic approaches, especially the application of *Ficus sur* extract, may have protective effects against malaria-induced hematological injury. The extract's function in maintaining red blood cell integrity, hemoglobin concentration, and platelet counts, while modulating immune cell differentials, underscores its potential as an adjunct therapy for malaria. Subsequent research should concentrate on isolating its active components and examining their mechanisms of action for potential incorporation into malaria therapy regimens.

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## CONFLICT OF INTEREST

The authors declare that they have no competing interests.

## CONSENT FOR PUBLICATION

Not applicable.

## ETHICAL APPROVAL

The research and ethics committee of the biochemistry department at Bayelsa Medical University's faculty of basic medical sciences in Yenagoa, Bayelsa State, Nigeria, gave their stamp of approval to this work (Reference Number: FBMS/AD/BCH/REC/29/03).

## AUTHORS' CONTRIBUTIONS (CREDIT TAXONOMY)

Concept – O.I.O. Design – O.I.O., B.E.P., P.S.T. Supervision – O.I.O., P.S.T. Materials – O.I.O.; B.E.P. Data Collection and/or Processing – O.I.O.; Analysis and/or Interpretation – B.E.P., O.I.O.; Literature Search – P.S.T.; Writing – O.I.O., P.S.T., B.E.P.; Critical Reviews – O.I.O., P.S.T., B.E.P.

## DATA AVAILABILITY STATEMENT

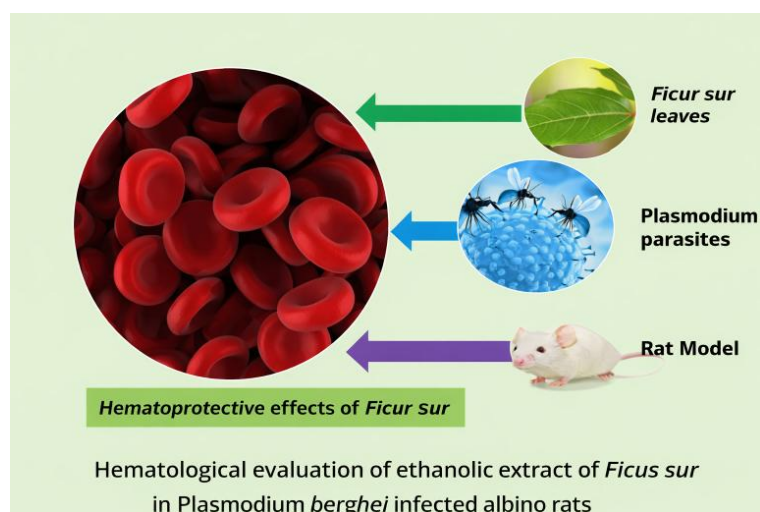
The datasets utilized and/or analyzed in the present study are available from the corresponding author upon reasonable request.

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### Graphical Abstract



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