

Haemoprevalence of malaria and haematological parameters of febrile patients in a hospital in Dutsin-Ma town, North Western Nigeria

Auta T^{1*}, Runka JY², Bawa JA¹, Sa'adatu SK¹

¹Department of Biological Sciences, Federal University, Dutsin-Ma, Katsina State Nigeria

²Department of Animal Production & Health, Federal University, Dutsin-Ma, Katsina State Nigeria

ABSTRACT

A study to assess the haemoprevalence and associated haematological effect of malaria infection was conducted among febrile patients. 86 febrile patients (37 males and 49 females) attending Dutsin-Ma General hospital, Katsina State, North-western Nigeria were used for the study. 5ml of peripheral blood samples were collected from each patient; 3 ml was placed in EDTA containers for malaria parasite test and hematologic parameters test. All samples were stored at -2°C to +8°C and transported to the laboratory for analysis. An overall herd prevalence of 61.6% was observed. The percentages of infected male and female individuals were 59.5% and 63.3%, respectively. The least (40%) and the highest prevalence (77.8%) were recorded in age groups 25-36 and >36 years respectively. The odd of having a malaria positive individual is 0.9 (95% CI = 0.355-2.046). The difference in Prevalence across the age groups is statistically not significant ($P>0.05$). The odd of having a malaria positive individual across the Age groups is 1.7 (95% CI = 0.613-4.494). The herd level prevalence of malaria in the study area is high. Therefore, Proactive steps such as use of Insecticide Treated Nets (ITNs), ensuring the screening of windows and doors, maintaining a healthy environment condition (which discourages the breeding of mosquitoes) and other effective malaria control strategies would all have a synergistic effect in controlling malaria infection.

Citation: Auta T, Runka JY, Bawa JA, Sa'adatu SK (2016). Haemoprevalence of malaria and haematological parameters of febrile patients in a hospital in Dutsin-Ma town, North Western Nigeria. *Biosciences Research in Today's World* 1: 113-119.

Received February 29, 2016; **Accepted** March 17, 2016; **Published** May 13, 2016.

Copyright: © 2016 Auta et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited. BRTW is the official journal publication of BRSF.

Competing Interests: The authors have declared that no competing interests exist.

* **E-mail:** autatimz@gmail.com

Keywords: haemoprevalence; malaria; haematology; Dutsin-Ma; Katsina

1. INTRODUCTION

Malaria is a life-threatening disease caused by parasites that are transmitted to people through the bites of infected mosquitoes. According to the latest estimates, released in December 2014, there were about 198 million cases of malaria in 2013 (with an uncertainty range of 124 million to 283 million) and an estimated 584 000 deaths (with an uncertainty range of 367 000 to 755 000). Malaria mortality rates have fallen by 47% globally since 2000 and by 54% in the WHO African Region [1].

Malaria occurs mostly in poor tropical and subtropical areas of the world. In many of the countries affected by malaria, it is a leading cause of illness and death. In areas with high transmission, the most vulnerable groups are

young children, who have not developed immunity to malaria yet, and pregnant women, whose immunity has been decreased by pregnancy. The costs of malaria – to individuals, families, communities, nations – are enormous [2].

Malaria has emerged as one of the top 10 killer diseases around the globe. It is the major cause of mortality in various tropical and subtropical regions. More than 500 million people reported positive cases of malaria and leading to death in 2 to 3.0 million cases [3].

Malaria is a major cause of morbidity in the tropics, thus the disease is of global importance that results in 300–500 million cases and 1.5–2.7 million deaths yearly [4]. Four species of

malaria parasite cause this disease (*Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium malariae*, and *Plasmodium ovale*) but *Plasmodium falciparum* is the foremost cause of malaria and death [3]. *Falciparum* malaria is the most dangerous form of the disease resulting in life threatening complication such as anaemia and cerebral malaria. The pattern of exposure to malaria infection, the type of treatment and the degree of compliance with the anti-malaria regimen, local drug resistance patterns, and an individual's age and genetic makeup all tend to influence the severity of the disease [5].

Most deaths occur among children living in Africa where a child dies every minute from malaria. Malaria mortality rates among children in Africa have been reduced by an estimated 58% since 2000. It poses a threat to public health with 80 to 90% of morbidity and mortality occurring in Africa, afflicting both young and old [6-8].

Thirty countries in Sub-Saharan Africa account for 90% of global malaria deaths. Nigeria, Democratic Republic of Congo (DRC), Ethiopia, and Uganda account for nearly 50% of the global malaria deaths. Malaria is the 2nd leading cause of death from infectious diseases in Africa, after HIV/AIDS. Almost 1 out of 5 deaths of children under 5 in Africa is due to malaria. Malaria is a major public health problem in Nigeria where it accounts for more cases and deaths than any other country in the world. Malaria is a risk for 97% of Nigeria's population. The remaining 3% of the population live in the malaria free highlands. There are an estimated 100 million malaria cases with over 300,000 deaths per year in Nigeria. This compares with 215,000 deaths per year in Nigeria from HIV/AIDS. Malaria contributes to an estimated 11% of maternal mortality [9].

Malaria in Nigeria is endemic and constitutes a major public health problem. Despite the curable nature of the disease; malaria related deaths accounts up to 11% of maternal mortality, 25% of infant mortality and 30% of under-five mortality [10], resulting in about 300,000 childhood death annually. The vast majority of deaths occur among children below five years of age and pregnant women [11], especially in remote rural area with poor access to health. Despite the combined efforts by 102 countries to eradicate malaria, it remains the major disease in the world today in terms of lives lost and economic burden. Progress has been made however in some countries. In countries such as United States, eradication of endemic malaria is complete [5].

Changes in haematological parameters are likely to be influenced by any disease condition including endemic diseases, such as malaria, that can affects health of mankind with various clinical presentations. Malaria is a major cause of deaths in the tropical area of the world. Two

hundred and nineteen million cases were reported worldwide in 2010 [10] Haematological changes are some of the most common complications in malaria and they play a major role in malaria pathogenesis. These changes involve the major cell types such as RBCs, leucocytes and thrombocytes [12]. Malaria infected patients tended to have significantly lower platelets, WBCs, lymphocytes, eosinophils, RBCs and Hb level, while monocyte and neutrophil counts were significantly higher in comparison to non-malaria infected patients [13]. One study showed patients with higher WBCs count compared with community controls. The most common complication during malaria infection is thrombocytopenia [14].

Anaemia is the commonest consequences of *Plasmodium falciparum* malarial infection [15]. The severity and type of anaemia can be determined by the levels of haematological indices such as haemoglobin concentration, Packed Cell Volume (PCV), Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin Concentration (MCHC) and Mean Corpuscular Haemoglobin (MCH) [13]. Hence, this study assessed the haemoprevalence of malaria and associated haematological effect of malaria infection.

2. MATERIAL AND METHODS

2.1. Study area

The study was carried out in Dutsin ma Local Government Area of Katsina State. Dutsin-Ma LGA lies on latitude 12°26'N and longitude 07°29'E. It is bounded by Kurfi and Charanchi LGAs to the north, Kankia LGA to the east, Safana and Dan-Musa LGAs to the west, and Matazu LGA to the southeast. Dutsin-Ma LGA has a land size of about 552.323 km² with a population of 169 829 as at 2006 national census. The people are predominantly agro pastoralists [16].

2.2. Study design and study population

The study design is a cross sectional study involving participants from all the geopolitical wards. The study population includes 86 febrile patients that attended general hospital Dutsin-Ma.

2.3. Sampling and sample collection

Judgmental or purposive sampling was used based on the consent of the participants. 5ml of peripheral blood sample was collected from each patient; 3 ml was placed in EDTA containers for malaria parasite test and hematologic parameters test. All samples were stored at -2°C to +8°C and transported to the laboratory for analysis.

2.4. Laboratory analysis of samples

2.4.1. Malaria parasite test

Both thick and thin blood films were prepared. Place a large drop of blood at one end of the slide; spread the blood over an area of about 2cm in diameter. Allow to air dry. Place a drop of blood at the end of the slide. Spread the blood to prepare a thin film. Allow the film to air dry by placing the slide in horizontal position. Fix in absolute methanol for 1-2 minutes. Pour the stain into the staining trough. Place the slide face down ward in a rack. Allow to stain for 10minutes. Wash off the stain to prevent a fine deposit of stain to carrier the film. Examine microscopically. Trophozoites and ring forms of malarial parasites within red blood cells will be observed.

2.4.2. Hematologic parameters test

These include measurement of Hemoglobin, Packed Cell Volume (PCV), White Blood Cell Count (WBC Count), Red Blood Count (RBCs) and Erythrocytes Sedimentation Rate (ESR).

2.4.2.1. Measurement of hemoglobin

Cyanomethae-moglobin reaction was used for the determination of haemoglobin concentration [17]. About 0.2 ml of each blood sample was mixed with 4ml of diluting fluid. The sample was left for 5 minutes and examine at 540nm wavelength.

2.4.2.3. Packed cell volume (PCV)

Blood sample was mixed gently but thoroughly with a blood mixer, and then fill the capillary tube with blood at least 2/3 but not more than 3/4 and excess blood on the tube should be cleared with a piece of cotton wool, sealed the dry end of the tube with the sealant or Bunsen flame, Place the tube the radial grooves of the Haematokrit centrifuge head with the open ends towards the centre replace the lid, Centrifuge for 5minute at 10,000rpm. Allow the centrifuge to stop on its own, Remove the tubes and use the reader to read the PCV, Align the base of the blood in the column with O and the bottom of the menials of the plasma with 100, the volume of the packed cells is taken directly from the PCV reader.

PCV reading of 30% - 45% in males is normal and 29% - 35% in females is normal. In infants and very small children, PCV reading can be up to 45% and above.

2.4.2.4. White blood cell (WBC) count

About 0.2 ml of well mixed blood sample was mixed with 0.3 ml of diluting fluid. The grid of the counting chamber will be filled with the sample using a pastuer pipette. The chamber will be left undisturbed for 2 minutes. The sample will be read microscopically using x10 objective with the condenser iris closed sufficiently to give a good contrast [17].

2.4.2.5. Red blood cell count (RBC Count)

About 0.2 ml of well mixed blood sample was mixed with 0.3 ml of diluting fluid. The grid of the counting chamber will be filled with the sample using a pastuer pipette. The chamber will be left undisturbed for 2 minutes. The sample was read microscopically using x10 objective with the condenser iris closed sufficiently to give a good contrast [17].

2.4.2.6. Erythrocytes Sedimentation Rate (ESR)

The erythrocytes sedimentation rate was determined using the Westergren method. The method involves collecting 2ml of venous blood into a tube containing 0.5ml of sodium citrate. The blood is placed into a Westergren-Katz tube, the tube is placed in a rack in a vertical position for 1 hour at room temperature. The distance from the lowest point of the surface meniscus to the upper limit of the red cell sediment is measured. The distance is expressed as millimeters in 1 hour. Expected range for a healthy males and females are <15 mm/hr and <20 mm/hr.

2.5. Statistical analysis

Odds ratio (OR) and 95% confidence interval (CI) on the Odds ratio were calculated using Winepiscope® 2.0 to measure strengths and statistical significance of associations between variables and malaria Prevalence. Values of $p \leq 0.05$ were considered significant. Descriptive statistics was carried out using Microsoft excel® 2007.

3. RESULTS

3.1. Malaria prevalence by gender in Dutsin-Ma LGA, Katsina State

A total of eighty six patients were randomly sampled and screened for malaria parasites. Out of the eighty six patients, thirty seven (43%) were males and forty nine (57%) females. In the male category, twenty two (59.5%) males were positive for malaria parasite. Similarly, in the female category, thirty one (63.3%) females were positive for malaria parasite. Distribution of malaria positive individuals by gender in Dutsin-Ma LGA, Katsina state is presented in Table 1. The herd level haemo-Prevalence of malaria in Dutsin-Ma LGA based on the study was 61.6%. The odd of having a malaria positive individual is 0.9 (95% CI = 0.355-2.046). However, the result is not statistically significant ($P = 0.09$).

3.2. Malaria prevalence by age group in Dutsin-Ma LGA, Katsina State

The participants to be screened were grouped into 0-12, 13-24, 25-36 and >36years age groups (Table 2). The highest Prevalence was recorded in >36years age group with a value of 77.8% and the lowest Prevalence was recorded

in 25-36years age group with a value of 40%. The difference in Prevalence across the age groups is statistically not significant ($P=0.053$). The odd of having a malaria positive individual across the Age groups is 1.7 (95% CI = 0.613-4.494).

3.3. Comparative haemogram of malaria positive and malaria negative patients in Dutsin-Ma LGA, Katsina State

Mean hematologic parameters among the study population are presented in Tables 3. In malaria

positive patients, results obtained shows a mean packed cell (PCV) of 35.8, mean hemoglobin concentration of 10.168g/dl, mean white blood cell count (WBCs) of 6.836×10^3 , mean red blood cell count (RBCs) of 4.153 and Erythrocyte sedimentation rate (ESR) of 21.05mm/hr while in malaria negative patients, mean packed cell volume (PCV) was 38.1%, mean hemoglobin concentration was 13.82g/dl, mean white blood cell count (WBCs) was 5.35×10^3 , mean red blood cell count (RBCs) was 5.361 and Erythrocyte sedimentation rate (ESR) was 15.67.

Table1. Distribution of Malaria positive individuals according to gender in Dutsin-Ma Katsina State.

Sex	No. Screened	No. Positive	Prevalence (%)	OR	95%CI on OR	P-Value
Male	37	22	59.5	0.9	0.355-2.046	0.088
Female	49	31	63.3			
Total	86	53	61.6%			

Table2. Prevalence of malaria infection according to age-group.

Age group (Years)	No. Screened	No. Positive	Prevalence (%)	OR	95% CI on OR	P-Value
0-12	38	23	60.5			
13-24	29	19	65.5	1.7	0.613-4.494	0.530
25-36	10	4	40.0			
>36years	9	7	77.8			
Total	86	53	61.6			

Table3. Mean Blood Haemogram in relation to malaria infection

Blood parameter	Leucocytes \pm SE (Cell/ μ l/ mm^3)	Hemoglobin Concentration (g/dl)	Erythrocyte Sedimentation Rate (ESR)
Infected	$6.84 \times 10^3 \pm 2015.83$	10.17 ± 19.85	21.05 ± 5.79
Non-infected	$5.35 \times 10^3 \pm 3426.14$	13.82 ± 5.21	15.67 ± 8.64

The differences in the mean values of the infected and non-infected subjects were not significant ($P>0.05$).

Table4. Mean (\pm SE) PCV in relation to age and malaria infection status

Age (years)	0-12	13-24	25-36	>36*
Infected	$24.50 \pm 6.34a$	$23.00 \pm 6.97a$	$28.33 \pm 5.67a$	$35.82 \pm 4.90a$
Non-infected	$29.43 \pm 5.01a$	$31.15 \pm 5.25a$	$32.00 \pm 8.44a$	$38.15 \pm 8.04b$

*The differences in the mean values of PCV of infected and non-infected subject was significant ($P>0.05$) in age groups >36years.

4. DISCUSSION

Prevalence of malaria in urban environments is generally lower than in the rural communities. The low levels of malaria incidence in the urban settlements could be as a result of relatively good effective alert systems on malarial control. Anopheles mosquitoes may also be less abundant due to the urban pollution. However, high disease impact may result due to lack of

repeated infections with multiple strains of malaria parasites in urban settings [18]. Tolerance to malaria parasitaemia does occur naturally, but only in response to repeated infection with multiple strains of malaria, especially among adults in areas of moderate or intense transmission conditions [19,20].

The overall prevalence of malaria in this study was high (61.6%). It was higher than 17% and

7.7% overall prevalence reported by Anumudu *et al.* [21] and Oyibo *et al.* [22] respectively, among the University of Ibadan campus students and pregnant women in Lagos, South-western Nigeria, respectively. On the other hand, the overall pre-valence of malaria reported in this study is at variance with previous estimates from other studies in peri-urban areas of Nigeria and other parts of West Africa [23, 24, 25]. This high prevalence could be attributed to the raining season during which the study was carried out. High rainfall and humidity increases mosquito longevity and give room to the collection of clear, still, sun exposed waters, all of which enhance malaria transmission, serving as good vector breeding sites [26].

Moreover, in this study, it was found that female individuals have a higher risk of being infected with malaria compared to the male participants. This is in agreement with other reports [27,28]. However, the reverse trend has been reported in some other studies [29,30]. Attitudes of women such as getting up before dawn to perform household chores may expose them more to mosquitoes and consequently to malaria infection than their male counterparts [31]. Our study is at variance with other findings that showed higher prevalence among children [32,33]. The 60.5% prevalence in the age group 0 - 12 years reported in this study corroborates with the reports of WHO [1] and UNAIDS/WHO [34]. This age group therefore constitutes the group with significantly high risk of malaria.

The haematological abnormalities previously reported included changes in haemoglobin, leukocyte count, platelet abnormalities resulting in defective thrombo-plastin, and disseminated intravascular coagulation (DIC) [35]. In this study, although leukocytosis was frequently seen in the malaria-infected subjects, no significant difference in WBC was found between the two groups. This finding agrees with other reports. In contrast, other studies have demonstrated leucopenia [12,37] or leukocytosis [13]. These findings are comparable with those of other studies [25, 38] that reported no significant difference in WBC between the malaria infected and non-infected groups. Higher lymphocyte count reported among malaria infected subjects in our study was also in contrast to other studies that showed that decrease lymphocyte count was associated with malarial parasites infection [12, 35]. The high level of leucocytes could be due to its distribution into the peripheral blood during malarial infection.

Anaemia is one of the most common complications in malaria, especially in younger children and pregnant women in high transmission areas [39]. It is thought to result from a combination of haemolytic mechanisms and accelerated removal of both parasitized and non-parasitized red blood cells, depressed and ineffective erythropoiesis [40]. Generally, the

present study showed higher susceptibility in the age group >36 years to anaemic condition than the children population. This contradicts the reports of Adedotun *et al.* [36] where they reported more susceptibility in the children age group. The low PCV values recorded among few non-parasitized subjects may in part reflect poor nutritional status, background haemoglobinopathy, intestinal worm infestation and previous and/or repeated malaria infections in this area [25]. ESR of malaria patients were above the reference intervals of ESR = 0-15 mm/h. Elevation of ESR has been reported in acute and chronic infections [41]. Supcharoen *et al.* [42] used ESR as basis for the diagnosis and monitor of therapeutic intervention of malaria. They suggested that ESR was elevated during acute malaria infection and declined with recovery.

4. CONCLUSION

Malaria prevalence in the study area was high compared to some other investigations. Although being an urban setting, ineffective and inadequate system could be responsible for the high level. However, this cannot be fully justified until similar study is carried out in the study area during the dry season. This will enable us to evaluate the influence of seasons in the dynamics of the disease.

Proactive steps such as use of Insecticide Treated Nets (ITNs), ensuring the screening of windows and doors, spraying of homes and surroundings with insecticide, cleaning of waterways, maintaining a healthy environment condition (which discourages the breeding of mosquitoes) and other effective malaria control strategies would all have a synergistic effect in controlling malaria infection.

ETHICAL APPROVAL

Ethical approval was gotten from the ethical committee of the Katsina state ministry of health before the commencement of this research. All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

ACKNOWLEDGMENTS

The authors are very grateful to the entire Hospital Board Management (HBM) of General Hospital Dutsin-Ma, Katsina State, Nigeria for granting permission for the study.

REFERENCES

1. WHO (2014). Malaria Fact Sheet. www.who.malariafact.org.
2. CDC (2014). Malaria worldwide www.cdc.gov/malaria/malaria_worldwide/index.html.

3. Godse RR. Hematological and biochemical evaluation in malaria patients with clinical correlation. *Inter. J. Appl. Res.* 2013;3(4):28-31.
4. Snow RW, Guerra CA, Noor AM, Myint HY, Hay SI. The global distribution of clinical episodes of *Plasmodium falciparum* malaria. *Nature.* 2005;434:214-217.
5. Etusim PE, Kalu C, Nduka FO, Kalu EC, Melariri PE, Nwoke M, Aduaka AC. Studies on the Prevalence of Malaria Parasite among Children with Splenomegaly in Aba Metropolis, Abia State, Nigeria. *J. Med. Appl. Bios.* 2013;5:56-66.
6. Afolabi BM. Malaria: The global scourge. *Nig. Clin. J.* 2001;5:9-12.
7. Ikekpeazu EJ, Neboh EE, Maduka IC, Nwagbara IJ, Nwobodo MW. Type-2 diabetes mellitus and malaria parasitaemia: Effect on liver function tests. *As. J. Med. Sci.* 2010;2:214-217.
8. Ogbodo SO, Okeke AC, Obu HA, Shu EN, Chukwurah EF. Nutritional status of parasitemic children from malaria endemic rural communities in eastern Nigeria. *Cur. Ped. Res.* 2010;14(2),131-135.
9. United States Embassy in Nigeria. Nigeria Malaria Fact Sheet (2011). Available from: <http://photos.state.gov>. [Last accessed on 2013 Apr 17].
10. WHO (2012). World malaria report 2012. Geneva: World Health Organization; 2012.
11. Philips RS. Current Status of Malaria and Potential for Control. *Clin. Mic. Rev.* 2011;14:208-226.
12. Erhart LM, Yingyuen K, Chuanak N, Buathong N, Laoboonchai A, Miller RS, Meshnick SR, Gasser Jr RA, Wongsrichanalai C. Hematologic and clinical indices of malaria in a semi-immune population of western Thailand. *Amer. J. Trop. Med. Hyg.* 2004;70(1):8-14.
13. Ladhani S, Lowe B, Cole AO, Kowuondo K, Newton CR. Changes in white blood cells and platelets in children with falciparum malaria: Relationship to disease outcome. *Br. J. Haematol.* 2002;119:839-847.
14. Mahmood A, Yasir M. Thrombocytopenia: a predictor of malaria among febrile patients in Liberia. *Infect. Dis. J.* 2008;14:41-44.
15. Uneke CJ. Impact of placental *Plasmodium falciparum* malaria on pregnancy and perinatal outcome in sub-Saharan Africa: Part III: Placental malaria, maternal health and public health. *Yale J. Biol. Med.* 2008; 81:1-7.
16. Runka YJ, Jibia AB, Janaidu S., Nana, A.B. A field survey on the assessment of awareness about contagious bovine pleuropneumonia (CBPP) in rural pastoral communities of Dutsinma Region, Katsina State, Nigeria. *Sci. J. Vet. Adv.* 2015;4(1):1-12.
17. Cheesbrough M. District laboratory practice in tropical countries. Cambridge University Press, Cambridge. 2006.
18. Klinkenberg E, McCall PJ, Hastings IM, Lengeler C, Bates I, D'Alessandro U, Barnish G, Klinkenberg E, Townson H, Trape JF. Malaria and irrigated crops, Accra, Ghana. *Emerg. Inf. Dis.* 2005;11(8):1290-1293.
19. Färnert A, Williams TN, Mwangi TW, Ehlin A, Fegan G, Macharia A et al. "Transmission dependent tolerance to multiclonal *Plasmodium falciparum* infection". *J Infect Dis.* 2009;200(7):1166-1175.
20. World Health Organization. World Malaria Report 2008. Geneva: World Health Organization; 2008.
21. Anumudu CI, Adepoju A, Adeniran M. Malaria prevalence and treatment seeking behaviour of young Nigerian adults. *Ann. Afr. Med.* 2006;15:82-88.
22. Oyinbo WA, Agomo CO (2009). Effects of malaria and human immunodeficiency virus co-infection during pregnancy. *IJHS* 2009;2(3):237.
23. Ojo DA, Mafiana CF. Epidemiological studies of malaria parasitaemia in Abeokuta, Ogun State, Nigeria. The Book of Abstract of the 29th Annual Conference and General Meeting (Abeokuta 2005) on Microbes as Agents of Sustainable Development. Organized by the Nigerian Society for Microbiology (NSM), University of Agriculture, Abeokuta, 6-10th November, 2005. 50 pp.
24. Umeanaeto PU, Ekejindu IM. Prevalence and intensity of malaria in blood donors at Nnamdi Azikiwe University Teaching Hospital (NAUTH) Nnewi, Anambra State, Nigeria. *Nig. J. Parasitol.* 2006;27:1-15.
25. Maina RN, Walsh D, Gaddy C, Hongo G, Waitumbi J, Otieno L, Jones D, Ogutu BR. Impact of *Plasmodium falciparum* infection on haematological parameters in children living in Western Kenya. *Malar J.* 2010; 9(3):S4. doi: 10.1186/1475-2875-9-S3-S4.
26. Bremen JG. The ears of hippotamus: Manifestations, determinants, and estimates of the malaria burden. *Am. J. Trop. Med. Hyg.* 2001;64(1,2):1-11.
27. Ibekwe AC, Okonko IO, Onunkwo AI, Ogun AA, Udeze AO. Comparative Prevalence Level of *Plasmodium* in Freshmen (First Year Students) of Nnamdi Azikiwe University Awka, South-Eastern, Nigeria. *Malay. J. Microbiol.* 2009;5(1):51-54.
28. Okonko IO, Donbraye-Emmanuel OOB, Donbraye E, Abubakar MJ, Fowotade A, Fadeyi A, Babalola ET, Ojezele MO, Adeyi AO. Malaria parasitaemia among patients in Ibadan, Southwestern Nigeria. *J. Appl. Biosci.* 2010;29:1774-1780.
29. Asklung HH, Nilsson J, Tegnell A, Jason R, Ekdahl K. Malaria risk in travelers. *Emerg. Inf. Dis.* 2005;11(3):436-441.
30. Abdullahi K, Abubakar U, Adamu T, Daneji AI, Aliyu RU, Jiya N, Ibraheem MTO, Nata'ala SU. Malaria in Sokoto, North Western Nigeria. *Afr. J. Biotech.* 2009; 8 (24):7101-7105.
31. Vlassoff C, Manderson L. Incorporating gender in the anthro-pology of infectious disease. *Trop. Med. Int. Health.* 1998; 3(12):1011-1019.
32. Umar RA, Hassan SW. The Relationship between levels of parasitaemia and anaemia in children with malaria. *Sahel Med. J.* 2002; 5(1):58-62.
33. World Health Organization. Susceptibility of *Plasmodium falciparum* to antimalarial drugs: report on global. 2005.
34. United Nations Programme on HIV/AIDS/ World Health Organization. Global Facts and Figures. AIDS epidemic update, December 2009.
35. Richards MW, Behrens RH, Doherty JF. Short report: Haematological changes in acute, imported *Plasmodium falciparum* malaria. *Am. J. Trop. Med. Hyg.* 1998; 59:859.
36. Adedotun AA, Salawu OT, Morenikeji OA, Odaibo AB. Plasmodial infection and haematological parameters in febrile patients in a hospital in Oyo town, South-western Nigeria. *Journal of Public Health and Epidemiology.* 2013; 5(3):144-148.
37. Lathia TB, Joshi R. Can hematological parameters discriminate malaria from non malarious acute febrile illness in the tropics? *Indian J. Med. Sci.* 2004; 58:239-244.
38. Bashawri LAM, Mandi AA, Bahnassy AA, Ahmed MA. Malaria: Haematological aspects. *Annals of Saudi Medicine.* 2002; 372-376.

39. Menendez C, Fleming AF, Alonso PL. Malaria-related anaemia. *Par. Today*. 2000; 16:469-476.
40. Weatherall DJ, Miller LH, Baruch DI, Marsh K, Doumbo OK, Casals-Pascual C, Roberts DJ. Malaria and the red cell Hematology. *Am. Soc Hematol. Educ. Program*. 2002; 35-57.
41. Kwiatkowski D, Cannon JG, Manogue KR, Cerami A, Dinarello CA, Greenwood BM. TNF production in falciparum malaria and its association with schizont rupture. *Clin. Exp. Immu.* 1989; 77:391-396.
42. Supcharoen O, Widjaja H, Ali KB, Kitayaporn D, Pukrittayakamee S, Wilairatana P et al., A study of erythrocyte sedimentation rate in malaria. *J. Infect. Dis. Antimicrob. Agents*. 1992; 9(4):193-199.