09.30.2024

JOURNAL OF

Health, Metabolism & Nutrition Studies (JHMNS) Vol. 5 No. 3

SUSCEPTIBILITY OF SEVERE MALARIA IN ASSOCIATION WITH ABO BLOOD GROUPING SYSTEM AMONG PATIENTS ATTENDING FEDERAL POLYTECHNIC BAUCHI MEDICAL CENTER, BAUCHI STATE

AMBI AHMAD ADAMU[1], ADAMU ABUBAKAR ADAMU[1], JIBRIL IBRAHIM[1], UMAR ABDULLAHI[2], WADA NAFIU MUHAMMAD[2], UDOSEN EDWARD[2] AND SA'ADATU BELLO KIRFI[2]

[1]Department of Biochemistry, Federal Polytechnic Bauchi. [2]Department of Microbiology, Federal Polytechnic Bauchi

Corresponding Author: ahmadambi@fptb.edu.ng

Abstract

The chemistry between malaria parasites and blood group antigens remains a fascinating subject with potential to contribute to the development of new interventions to reduce the global burden of malaria. Elucidation of the association between the blood groups status and infection with P. falciparum can bring about understanding of the differences noted in the ABO blood group variation. A cross-sectional study of 100 patients were clinically examined to determine the ABO blood group system and its association with severe malaria pathogenesis of patients who were tested

Introduction

Malaria is one of the major endemic disease in Africa have shown that report, there were 241 million cases in 2020 compare to 227 million cases in 2019 the estimated number of malaria death stood at 627000 in 2020- an increase of 69000 death over previous year (World Malaria Report, 2020). Malaria is caused by an obligate, intracellular protozoan parasite of the genus Plasmodium, it is

E-ISSN 3026-8664 P-ISSN3027-2238

positive to malaria infection via microscopy at Federal Polytechnic Bauchi Medical Center, Bauchi State. Data entry was done in MS word, database management and analysis were done using Statistical Package for Social and Sciences (SPSS) version 20 software. The result shows that 17 (16%), 4 (4%), 3 (3%) and 77 (77%) of respondents belong to the A, B, AB and O blood groups. Packed Cell Volume (PCV), Red Blood Cell (RBC) and Parasite Density were significantly associated ($F=2.906$, $p=0.038<0.05$), ($F=4.996$, $p=0.003<0.05$) and ($F=3.108$, $p=0.030<0.05$) with blood groups. This study suggests that during *P. falciparum* infection, blood group O individuals are conferred with a protective advantage, blood group A individuals are at a disadvantage, while blood groups B and AB has an intermediate effect.

Keyword: Severe Malaria, Blood Grouping System, Patients, Federal Polytechnic Bauchi, Medical Center.

Transmitted by infected female anopheles' mosquito when feeding on blood. There are four species that infect humans namely: *Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale, Plasmodium malariae and Plasmodium knowlesi. The P. falciparum* is the principal cause of severe clinical manifestations to some extent *P. vivax* and *P. knowlesi*. The virulence of *P. falciparum* has been associated with the capacity of infected red blood cells (iRBCs) to adhere to uninfected red blood cells (uRBCs), leading to rosetting of cells (Pathirana *et al.,* 2010).

Blood grouping is a classification of blood based on the presence or absence of inherited antigenic substances on the surface of red blood cells (RBC). The ABO blood group refers to system of carbohydrate antigens expressed on human erythrocytes (Loscertales *et al.,* 2007) and other human cells. The ABO system is the most important blood group system in human blood transfusion. The "A" and "B" antigens on erythrocyte are trisaccharides ("A" GalNAcα1-3(Fucα1-2) Gal1β1; and "B" Gal1α1-

3(Fucα1-2) Galβ1) (Daniels *et al.,* 2020) that are attached to different glycolipids and glycoproteins.

Malaria is one of the world most threatening diseases with a high rate of mortality especially in Africa. According to world malaria report, there were 241 million cases in 2021 compare to 227 million cases in 2020 the estimated number of malaria death stood at 627000 in 2020- an increase of 69000 death over previous year while about two thirds of these death [4700] were due to disruptions during the covid-19 pandemic, the remaining one third death [22000] reflect a recent change in (WHO's) methodology for calculating malaria mortality irrespective of covid-19 disruption The new cause of death methodology was applied to 32 countries in sub-sahara Africa that shoulder about 93% of all malaria death globally. Applying the methodology revealed that malaria has taken a considerably higher toll on Africa children every year since 2000 than previous thought (World Malaria Report , 2020). Blood group "O" occurs in approximately 40-80% of the population in different part of Africa (Troy *et al.*, 2013). If the blood group "O" protect against life threatening malaria, why then is the frequency of "O" not higher in malarias. Prone countries.

Since malaria is a major world's threatening disease with high rate of mortality especially in Africa and it were estimated that about nearly half of the world's population are at risk recently by world health organization, the fact that the effect of ABO blood group to malaria, it would be useful to know whether there is any association between malaria with blood group. This study was aimed at investigating the susceptibility of severe malaria (*P. falciparum*) in association with "ABO" blood groups in respondents attending the Federal Polytechnic Bauchi Medical Center.

MATERIALS AND METHODOLOGIES

A descriptive cross-sectional study was designed and conducted to assess the effect of ABO blood group system on the development of severe malaria. The study was conducted at Federal Polytechnic Bauchi Medical Center, Bauchi State within the month of July, 2023.

Laboratory Analysis

The sample was collected using sterile needle and syringes, five milliliters (5ml) of blood samples was collected from the respondent with the assistances of the laboratory technologists via vein puncture technique. Blood collected was dispensed into ethylene-diamine-tetra-acetic acid (EDTA) anti-coagulated blood sample bottles, properly mixed and labeled appropriately.

To conduct microscopy study, thick and thin film were prepared on two different clean and grease free slides after appropriate labeling and allowed to air dry, on the laboratory work bench. Thin films were fixed with methanol and allow to evaporate while the thick films were not fixed with methanol. The slides are arranged on a staining rack and flooded with 10% Giemsa stain solution for 10-15 minutes. The slides were washed under a slow running tap water and allowed to air dry. Finally, the films were carefully examined under oil immersion microscope objective (x100).

Determination of parasite density

The malaria parasite density (PD) was determined by applying a counting technique in which the number of parasites counted in microscopic field (i.e. 100 field was used) against 200leucocytes within that field multiply by a given white cell count to give an approximate number of parasites per µl. In this study, the white cell count 10000/µl of blood was used (Dennis *et al*, 2012).

Before calculation of parasites density, number of parasites were considered, as if:

>10parasite were counted, 200leucocytes used in calculation of parasites density, but if <10parsites were counted, 500leucocytes used in parasites density calculation.

i.e. >10 parasites, the (PD) $= \frac{\text{Number of parasites counted}}{200\text{leucocytes}} X\ WBC$

If <10 parasites, the (PD) $= \frac{\text{Number of parasites counted}}{500\text{leucocytes}} X\ WBC$

(Moody, 2019).

Determination of blood group and rhesus test

The ABO blood group of each of respondents was determined using commercial cell grouping anti-sera (A, B and D).

Three (3) drops of each respondent blood sample (serum) is placed on separate point on a sterile white tile divided into three cells. A drop of anti-sera A, B and D were placed beside the blood which was properly mixed to a strain homogenous mixture with the aid of a sterile rod and the tiles was rocked gently to ensure uniform mixture. The mixtures were carefully observed to determine blood group of the patients by presence or absence of agglutination. Anti-serum D was used to determine Rhesus factor. Finally, the blood was grouped based on these observation and confirmation into blood group A+, A-, B+, B-, AB+, AB-, O+, and O-.

ABO blood groups were typed by agglutination using commercial antisera (Sharana *et al*, 2016).

Determination of hemoglobin concentration

Finger-prick samples were drop on strip and the strip was inserted into haemoglobin measured aid of Hemocue.

Determination of packed cell volume (PCV)

Packed cell volume (PCV) is determined by micro hematocrit method using a small quantity of blood, a capillary tube and a high speed centrifuge. PCV measurement is used in determining anemia and it can also be used to calculate hemoglobin concentration (Hb) expressed in g/dl and also to calculate Mean Corpuscular hemoglobin concentration (MCHC) which is the average hemoglobin concentration within the red blood cells. The pathophysiology of the anemia of falciparum malaria is both complex and multifactorial, and results in a condition which is a major cause of mortality and morbidity in patients, especially children and pregnant women, living in malarial endemic areas. (Meraiyebu *et al.*, 2012).

Inclusion and Exclusion Criteria

Patients who were suspected to have malaria and tested positive for malaria using thick and thin blood smear and microscopy or using Rapid Test Diagnosis (RDTs) kit were included in the study. While patients whose blood was not tested for malaria and febrile patients who had taken any antimalaria drugs within the last two weeks before the blood test. Patients who are critically ill and patients who did not consent or assent to the study were also excluded.

Statistical Analysis of Data

Data were entered in Microsoft Excel (MS-Excel) and exported into SPSS version 20 and analyzed. ANOVA was used to test the differences between parasites density and ABO blood grouping system. Observed differences was considered to be significant for ($p<0.05$).

RESULTS

Figure 1 give the distribution of respondents by gender. Exactly 58% were female and 42% were male. Figure 2 give the distribution of respondents by age. Children constitute about 9%, adolescent 7% and adult 84%. Figure 3 present the blood group of all respondents. From the figure, blood group A, B, AB and O. Were 16%, 4%, 3% and 77%. Figure 3.4 present density (parasites/liter) of various blood group of sampled respondents.

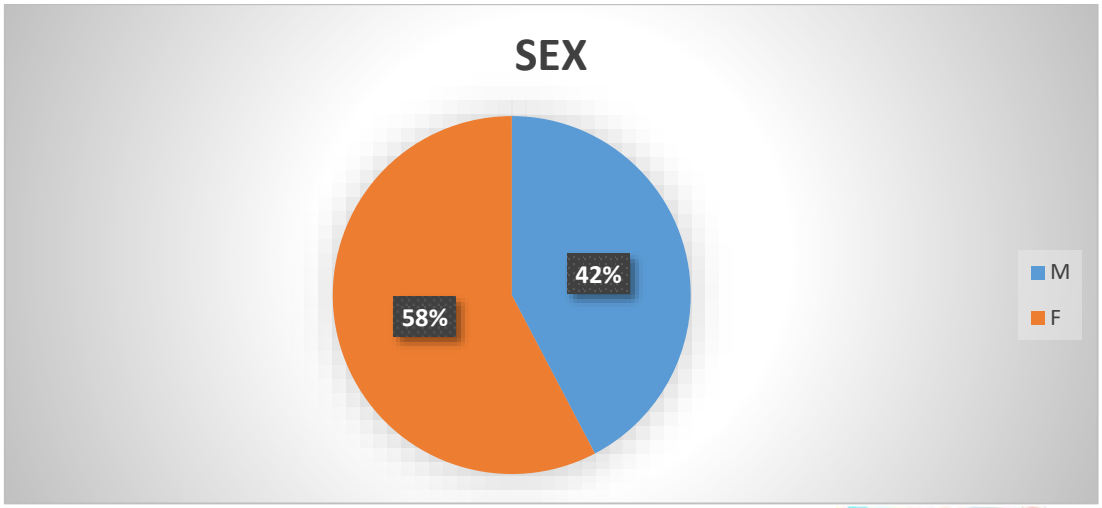

Figure 1: Distribution of Study Respondents According to Gender.
Keys: Male (M)
Female (F)

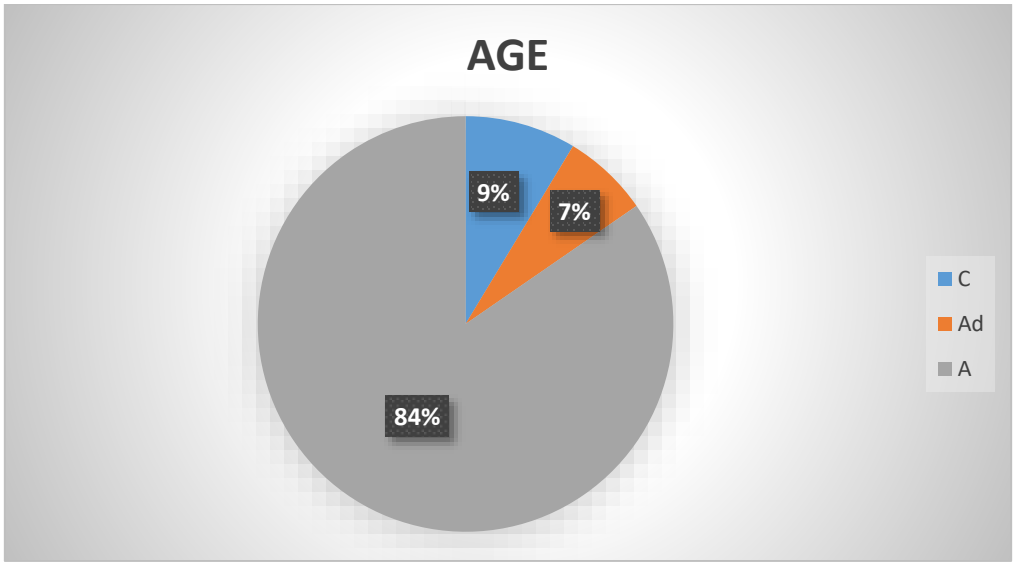

Figure 2: Distribution of Study Respondents Based on Age group.
Keys: Child (C)
Adolescents (Ad)
Adult (A)

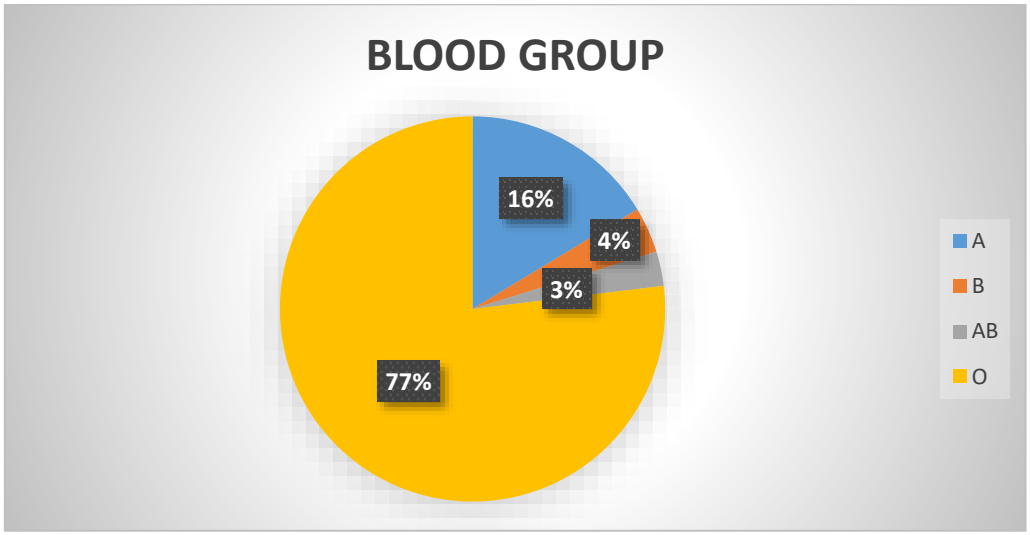

Figure 3: Distribution of Study Respondents Based on Blood Grouping.

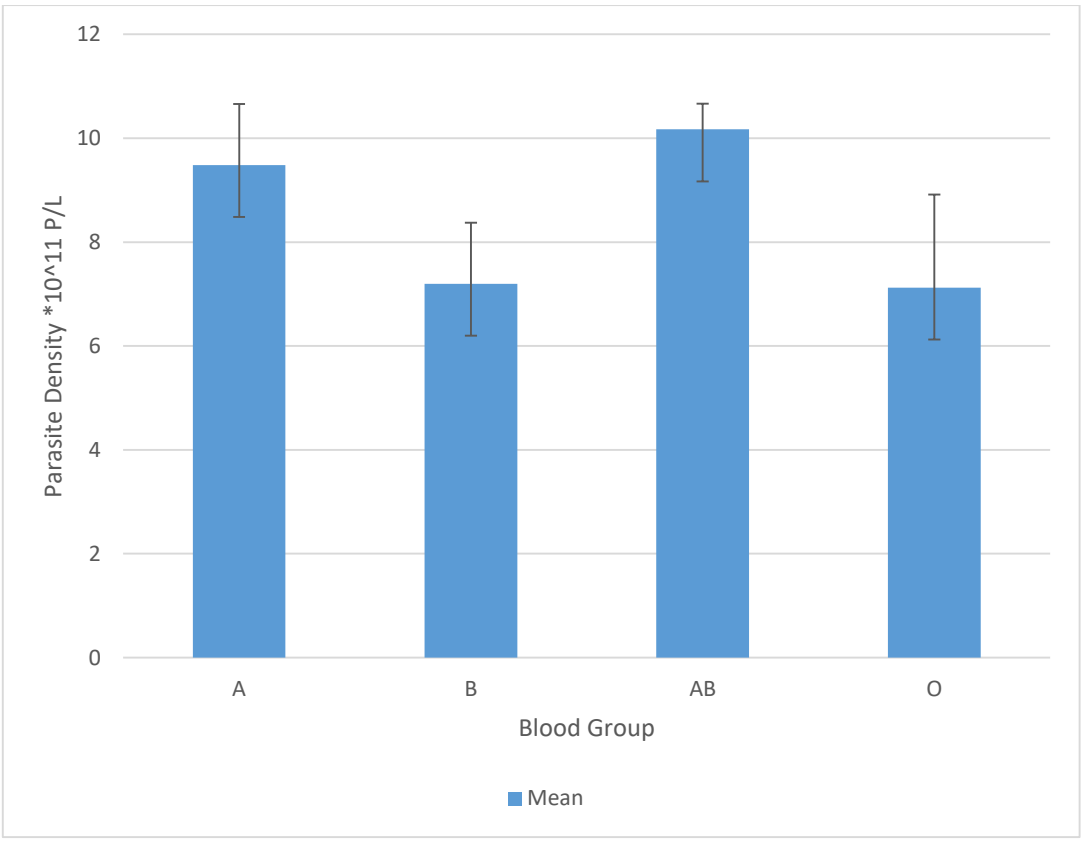

Figure 4: Parasite Density (parasites/liter) of Various Blood Groups of Sampled Respondents.

Table 1: Distribution of parasitaemia between ABO blood grouping system shows that about 25.00% (1/4), and 52.50% (42/80) of those infected individuals of blood group B and O respectively had parasite density of less than 200 parasite/ µL of blood. In contrast 29.41% (5/17), 66.67% (2/3) and 22.50% (18/80) *P. falciparum* infected individuals of blood group A, AB and O respectively had parasite density ranging from above 601-999 parasites / µL of blood.

Table 2 homogeneity of variance shows significance values of p=0.544, and 0.093 for PCV and RBC respectively, while that of the PD p=0.000 yield significant to homogeneity test of variance which gives the reason for the use of ANOVA to analyze the association between parasite density and ABO blood grouping system.

Table 3 ANOVA test presented (F=3.108, p=0.030) at 95% confidence interval and degree of freedom between groups (df=3) and within groups (df=100) is statistically significant.

Table 1: The Distribution of Parasitemia between ABO Blood Grouping System

Variables	Parasitaemia			Total
	A	B	C	
	<200 parasites /µL of blood	601-999 parasites /µL of blood	>601-999 parasites /µL of blood	
Blood Group				
A	0(0.00%)	12(70.59%)	5(29.41%)	17(100%)
B	1(25.00%)	3(75.00%)	0(0.00%)	4(100%)
AB	0(0.00%)	1(33.33%)	2(66.67%)	3(100%)
O	42(52.50%)	20(25.00%)	18(22.50%)	80(100%)

Table 2: Test of Homogeneity of Variances

	Levene Statistic	df1	df2	Sig.
PCV (%)	.718	3	100	.544
RBC (×10^12/)	2.197	3	100	.093
P.D (×10^11 P/L)	11.562	3	100	.000

Table 3: Relationship between Blood Groups and Parasite Density.

PD	Sum of Squares	Df	Mean Square	F	Sig.
Between Groups	134.715	3	44.905	3.108	.030
Within Groups	1445.024	100	14.450		
Total	1579.739	103			

DISCUSSION

This study shows that, the frequency of females is higher than that of the males. The frequency of adults is greater than that of adolescents and children. The frequency of blood group O is higher than that of blood group A, B and AB. The frequency of blood group O is greater in the uncomplicated malaria than that of blood group A, B and AB. The frequency of blood group AB is higher in complicated malaria than that of blood group A, B and O.

In contrast, group A is the predominant blood group in the colder regions of the Earth where malaria has not been common (Cserti *et al*., 2007). This is consistent with the findings of this study which found that the highest percentage of the study participants had the O blood group phenotype followed by blood group phenotypes A, B and AB. Blood group B seems to have an intermediate effect as proposed in the hypothesis.

P. falciparum infected group O erythrocytes are also more efficiently cleared by macrophages than infected A and B erythrocytes. This was shown in a study where human macrophages in *vitro* and mouse monocytes in *vivo* phagocytized *P. falciparum* infected O erythrocytes more eagerly than A and B infected erythrocytes (Panda *et al*., 2011). The enzymatic conversion of B erythrocytes to type O erythrocytes before infection also significantly enhanced their uptake by macrophages to levels comparable to those with infected O wild-type erythrocytes. This study not only showed that the ABO blood group antigens influence macrophage clearance of *P. falciparum* infected erythrocytes but also suggested an

additional mechanism by which blood group O may confer resistance to malaria (Wolofsky *et al*., 2012).

Blood group O was the dominant blood type in both complicated and uncomplicated malaria cases. Blood group A patients were more prone to severe mixed malaria infections. Blood group O malaria cases had a favorable clinical outcome (Sharana *et al*, 2016).

Several other studies have established that parasitized erythrocytes form rosettes more readily with RBCs of blood groups A, B, or AB than with blood group O RBCs, with parasite-triggered RBC rosette formation being associated with increased severity of clinical disease (Uneke, 2007). Rosette formation is governed by strong adhesive forces, with lectern-like bindings between parasite-derived proteins exposed on the surface of *P. falciparum* infected RBCs and various carbohydrate moieties present on the uninfected erythrocyte. The strongest carbohydrate receptors have been shown to be contained within the blood group A and B antigens (Carlson *et al*., 2018), thereby justifying the ease with which group A and B infected erythrocyte form rosettes, and accounting for the formation of larger and stronger rosettes by the non-O blood groups (Carlson *et al*., 2018). Being stronger, the rosettes formed by the group A and B *P. falciparum* infected erythrocytes are less easily disrupted than those formed by group O erythrocytes. This allows the group A and B parasitized cells to avoid the host's normal spleeny clearance mechanisms that remove aged or damaged erythrocytes (Mebius *et al*., 2020). Consequently, parasitized group A and B erythrocytes persists longer in the body, allowing multiplication of the *plasmodium* parasite, thereby predisposing individuals with this blood groups to clinical disease.

This is also consistent with reports suggesting that individuals with blood groups A, B and AB are more susceptible to *P. falciparum* infection than those with O blood group (Rowe *et al*., 2009)

In comparison with the above studies, the current study shows that blood group A individuals had higher percentage for severe malaria than other

blood groups while blood group O had higher percentage in uncomplicated than any other blood groups.

This study does not account for the effect of other determinants such as genetic polymorphism that might also play an important role in severity of falciparum infection.

CONCLUSION

The findings of this study showed that blood group O are most susceptible in the malaria area of Federal Polytechnic Bauchi Medical Center, Bauchi State. It also established a significant association between the ABO blood groups with *falciparum* malaria, where individuals of blood group O were found less likely to develop severe malaria when infected with *P. falciparum*, whereas individuals of blood group A were found more likely to develop severe malaria. Blood group B appeared to confer an intermediate effect. The findings of this study therefore agreed with the proposed hypothesis that during an infection with *P. falciparum*, blood group O offers a survival advantage, group A offers a disadvantage, and group B and AB has an intermediate effect.

REFERENCES

AbdulLatif Eunsin Ju, Chang-SukKong, YoungwanSeo, Yeon-JuLee, Seema R Dalal, Maria B Cassera & David GI Kingston, (2018). Antimalarial Activity of the Isolates from The Marine Sponge Hyrtioserectus against The Chloroquine-Resistant Dd2 Strain Of *Plasmodium Falciparum. Zeitschriftfür Nature For Schung* C73(**9-10**), 397-400.

Adams Joanna Furnival, Evelyn A Olanga, Mark Napier & Paul Garner, (2019). Housing Interventions For Preventing Malaria. *The Cochrane Data base of Systematic Reviews* (8).

Blythe Jane E, Makhtar Niang, Kevin Marsh, Anthony A Holder, Jean Langhorne, Peter R Preiser Jane E Blythe, Makhtar Niang, Kevin Marsh, Anthony A Holder, Jean Langhorne & Peter R Preiser, (2009). Characterization of the Repertoire Diversity of The *Plasmodium Falciparum* Stevor Multigene Family In Laboratory And Field Isolates. *Malaria Journal*. 8, 1-13.

Carlson Jenny S, Brittany Nelms, Christopher M Barker, William K Reisen, Ravinder NM Sehgal & Anthony J Cornel, (2018). Avian Malariaco-Infections Confound Infectivity and Vector Competence Assays of Plasmodium Homopolare. *Parasitology Research* 117, 2385-2394.

Center for Disease Control (CDC), (2020). Evaluation Two Malaria Surveillance System in YEMEN Using Updated CDC Guideline: Lessons Learning and Future Perspective. *Journal Health Care Organization,* 56: 469-1998.

Chen Q, Barragan A, Fernandez V & Sundström A (2000). Identification of *Plasmodium falciparum* Erythrocyte Membrane Protein 1 (PfEMP1) as the Rosetting Ligand of the Malaria Parasite P. *falciparum*. *Jounal of Experiment Medicine*.187:15–23.

Cheng Qin, Michelle L Gatton, John Barnwell, Peter Chiodini, James Mc Carthy, David Bell & Jane Cunningham, (2014). Plasmodium Falciparum Parasites Lacking Histidine-Rich Protein 2 And 3: A Review And Recommendations For Accurate Reporting, *Malaria Journal*. 13, 1-8.

Christine M Cserti and Walter H Dzik, (2007). The ABO Blood Group System and Plasmodium Falciparum Malaria. Blood. *The Journal of the American Society of Hematology* 110(**7**), 2250-2258.

Cooke BM, Mohandas N & Coppel RL. (2001). The Malaria-Infected Red Blood Cell: *Structural and Functional Changes. Advance Parasitol* 50: 1–86.

Cserti CM and Dzik WH. (2007). The ABO *Blood Group System and Plasmodium Falciparum Malaria. Blood*.110:2250-2258.

Daniels F Rachel, Richard Toll, Stephen F Schaffner, Yakou Dieye, Gnagna Dieng, Michael Hainsworth, Fatou B Fall, Coumba Ndoffene Diouf, Medoune Ndiop, Moustapha Cisse, Alioune Badara Gueye, Oumar Sarr, Philippe Guinot, Awa B Deme, Amy K Bei, Mouhamad Sy, Julie Thwing, Bronwyn MacInnis, Duncan Earle, Caterina Guinovart, Doudou Sene, Daniel L Hartl, Daouda N diaye, Richard W Steketee, Dyann F Wirth & Sarah K Volkman, (2020). Genetic evidence for imported malaria and local transmission. *Malaria Journal* 19(**1**), 1-8.

Deepa, Alwar VA & Rameshkumar K, Ross C. (2011). ABO Blood Groups and Malaria Related Clinical Outcome. *Journal Vector Borne Disease* (**48**) 7–11.

Fernandez-Arias Cristina, Jean Pierre Lopez, Jean Nikolae Hernandez-Perez, Maria Dolores Bautista-Ojeda, Ora Lee Branch & Ana Rodriguez (2013). Malaria In Hibits Surface Expression Of Complement Receptor 1 Inmonocytes/ Macrophages, Causing Decreased Immune Complex Internalization. *The Journal Of Immunology* 190(**7**), 3363-3372.

Fischer S Hannah,Mirjam Groger, Luzia Veletzky, Albert Lalremruata & Michael Ramharter , (2017). A Systematic Review of the Clinical Presentation, Treatment and Relapse Characteristics of Human Plasmodium Ovale Malaria. *Malaria Journal* 16(**1**),1-16.

Garnham Cyril & Keith Vickerman Protist, (2005). The lure of lifecycles: Cyril and the malaria parasites of primates. cytoadherence and its Association With Clinical Disease in Plasmodium Falciparum Infections in Srilanka. *NSF: Colombo.* 156(**4**), 433-449.

Grau GE & Craig A. G., (2012). Cerebral Malaria Pathogenesis: Revisitng Parasite and Host Contributions. *Future Microbiol*. 7**(2):** 299-302.

Grau GE, Mackenzie CD, Carr RA, (2003). Platelet Accumulation in Brain Microvessels in Fatal Pediatric Cerebral Malaria. *Journal of Infectious Disease*. 187**(3):**461–466.

Ilozumba PCO & Uzozie CR. (2009). Prevalence of Malaria Parasitaemia and Its Association With ABO Blood Group In Odoakpu Area of Onitsha South Local Government Area, Anambra State, Nigeria. *Nigerian Annals of Natural Sciences,* 8**(2),** Pp 1-8.

Kyes A Sue, Susan M Kraemer & Joseph D Smith, (2007). Antigenic Variation in *Plasmodium Falciparum*: Gene Organization and Regulation of the var Multigene Family. *Eukaryotic cell* 6(**9**), 1511-1520.

Loscertales María Paz, Stephen Owens, James O'Donnell, James Bunn, Xavier Bosch-Capblanch & Bernard J Brabin, (2007) ABO Blood Group Phenotypes And Plasmodium Falciparum Malaria: Unlocking Pivotal Mechanism. *Advances In Parasitology.* 65, 1-50.

Marsh K, Alan F Cowman, Julie Healer and Danushka Marapana, (2016). Cell Malaria: **Biology and Disease**. 167(**3**), 610-624.

Mckenzia FE, Wendy AP, Alan JM, Forney JR, Barnyen P, Carmen L, Robert AGJ, Chansuda W, (2008). White Blood Cell Count and Malaria. *Journal Of Infectious Disease.* 6:23.

Mclnerney P Curationis & Lily CKumbani, (2006). Primigravidae's Knowledge About Obstetric Complications In an Urban Health Centre in Malawi, *Malaria Journal,* 29(**3**), 41-49.

Mebius M Mirjam, Ayola A Adegnika, Jeannot F Zinsou, Jean Claude Dejon Agobe, YJ Honkpehedji, Maria Yazdanbakhsh, Govert Jvan Dam, Paul LAM Corstjens, Aloysius GM Tielens, Philip Gde Groot, Rolf T Urbanus & Jaap Jvan Hellemond,N (2020). Haemostatic Changes in Urogenital Schistosomiasis Haematobium: A case-Control Study In Gabonese School Children. *Journal of Helminthology*. e84, 94.

Meraiyebu A, Akintayo CO & Nenchi DY, (2012). Evaluations Of PCV And Haemoglobin Variations Among Malaria Positive And Negative Patients At ECWA Community Health Centre, Bukuru, Jos. Nigeria. e-ISSN: 2250-3013 p-ISSN: 2319-4219.

Miller LH & Baruch DI, Marsh K, (2002). The Pathogenic Basis of Malaria. *Nature Review Microbiology.* 415: 673–679.

Newbold C, Craig A & Kyes S, (2000). Cytoadherence, Pathogenesis and the Infected Red Cell Surface in *Plasmodium falciparum. International Journal for Parasitology.* 29:927-937.

Panda A. K, Panda S. K & Sahu A. N, (2011). Association of ABO Blood Group With Severe *Falciparum* Malaria In Adults: Case Control Study And Meta-Analysis. *Malaria Journal* 10:309.

Pathirana SL, KE kanayake, RJ Howard, KN Mendis & SM Handunnetti, (2010). Cytoadherence and Its Association with Clinical Disease in Plasmodium Falciparum Infections In Srilanka. *NSF:Colombo*, 9, 2010

Paul Horrocks and David Muhia, (2005). Pexel/VTS: A Protein-Export Motifin Erythrocytes Infected With Malaria Parasites. *Trends In Parasitology* 21(**9**), 396-399.

Penman B & Gupta S. (2008). Evolution of Virulence in Malaria. *Journal of Biology* 77:22.

Penman S Bridget, Bronner P Gonçalves & Sunetra Gupta, (2016). Sickle Haemoglobin, Haemoglobin C and Malaria Mortality Feedbacks. *Malaria Journal* 15(**1**), 1-7.

Perlmann Peter & Marita Troye Blomberg, (2002). Malaria and The Immune System in Humans. *Malaria Immunol* 80, 229-242.

Pizzi Elisabetta, Anna Olivieri, Rebecca S Lee, Federica Fratini, Cyrianne Keutcha, Mudit Chaand, Valentina Pizzi Mangano, Francesco Celani, Stefania Mochi, Cecilia Birago, Silvio Paone, Felicia Grasso, Valentina Tirelli, Mario Falchi, Estela Shabani, Stefania Bertoncini, Bienvenu Sodiomon Sirima, David Modiano, Manoj T Duraisingh and Marta Ponzi, (2021). Structural Organization of Erythrocyte Membrane Micro Domains and Their Relation with Malaria Susceptibility. **Communications Biology** 4(**1**),Pp 1375.

Rowe J Alexandra, Ian G Handel, Mahamadou A Thera, Anne- Marie Deans, Kirsten E Lyke, Abdoulaye Koné, Dapa A Diallo, Ahmed Raza, Oscar Kai, Kevin Marsh, Christopher V Plowe, Ogobara K Doumbo & Joann M Moulds, (2007). Blood Group O Protects Against Severe *Plasmodium Falciparum* Malaria Through the Mechanism of Reduced Resetting Proceedings of the National Academy of Sciences, *Malaria Journal* 104(**44**), 17471-17476.

Rowe J. A., D. Opia D. H & Williams T. N. (2009). Blood Groups And Malaria: Fresh Insights Into Pathogenesis and Identification of Targets For Intervention. *Current Opinion in Hematololgy*; 16**(6)**, 480–487.

Sharana H., Ruchi S., Shrijeet C & Lavnish O. (2016). Relationship Between Malaria and ABO Blood Group Types. *Malaria Journal*:5**(5)** 2319-7064.

Snow W Robert, Carlos A Guerra, Abdisalan M Noor, HlaY Myint & SimonI Hay, (2005). The Global Distribution of Clinical Episodes of Plasmodium Falciparum Malaria. *Nature* 434(**7030**), 214-217.

Suchi Goel, Mia Palmkvist, Kirsten Moll, Nicolas Joannin, Patricia Lara, Reetesh R Akhouri, Nasim Moradi, Karin Öjemalm, Mattias Westman, Davide Angeletti, Hanna Kjellin, Janne Lehtiö, Ola Blixt, LarsIdeström, Car lG Gahmberg, Jil lR Storry, Annika K Hult, Martin LO lsson, Gunnar Von Heijne, Ing Marie Nilsson, Mats Wahlgren, (2015). Rifins are Adhesion Simplicated in Severe *Plasmodium Falciparum* Malaria. *Nature Medicine* 21(**4**), 314-317.

Tekeste Z & Petros B. (2010). The ABO Blood Group And *Plasmodium Falciparum* Malaria In Awash, Metehara and Ziway Areas, Ethiopia. *Malaria Journal* 9:280.

Torres JR, (2003). Malaria And Babesiosis. Therapy Of Infection Disease (Edited by Baddor LM, Gorbach SL). Philadelphia, PA: Saundess; 597-613.

Troy CLund, Heather Hume, Jean PAllain, Jeffrey Mc Cullough & Walter Dzik (2013). The Blood Supply in Sub-Saharan Africa: Needs, *Challenges, And Solutions Transfusion and Aphaeresis Science* 49(**3**), 416-421.

Udujih HI, A A Amaechi, BEB Nwoke, CMU Ajero, OG Udujih & CC Iwuala (2019). Studies on Malaria, Bancroftian Filariasis and Insecticide Treated Bed Netsin Mmiata Anam, Anambra West Local Government Area, Nigeria. *International Journal of Current Microbiology and Applied Sciences* 8(10).

Uneke C. J. (2007). Plasmodium *Falciparum* Malaria And ABO Blood Group: Is There Any Relationship? *Parasitology Research*. 100, 759–765.

Wolofsky Kayla T, Kodjo Ayi, Donald R Branch, Annika K Hult, Martin LO lsson, W Conrad Liles, Christine M Cserti-Gazdewich & Kevin CKain, (2012). ABO Blood Groups Influence Macrophage-Mediated Phagocytosis of Plasmodium falciparum-Infected Erythrocytes, *Public Library of Science* 8(**10**), 1002942.

World Health Organization (WHO), (2014). Malaria Policy Advisory Committee to the WHO: Conclusion and Recommendation of Fifth Biannual Meeting. *Malaria Journal*. 13, 1-8.

World Health Organization (WHO), (2020). Malaria and Covid-19: Common and Different Findings. *Tropical Medicine and Infectious Disease*, 5(**3**),141.

Wu Xianzhu & D Channe Gowda, (2018). Parasite Recognition and Signaling Mechanisms in Innate Immune Responses to Malaria. *Frontiers in Immunology* 9, 3006.

E-ISSN 3026-8664 P-ISSN3027-2238