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Distribution of Malaria Infection Among Different Genotypes in Three Senatorial Zones of Taraba State Nigeria

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ABSTRACT

Malaria infection is recognized as a severe public health problem linked to most cases of morbidity and mortality in malaria endemic areas. The study was used to determine the distribution of malaria infection among the different genotypes in three senatorial zones in Taraba state. The study employed a community and laboratory based cross sectional study. The electrophoresis machine was used to determine the genotypes of the research subjects while Rapid diagnostic technique and the gold standard microscopy was used to determine the prevalence of malaria infection. The haematology auto-analyzer (Sysmex XTI 2000) was used to determine the haematological parameters of the subjects. A total of 3084 blood samples was obtained by venepuncture. The distribution of the genotypes is AA 1721 (55.8%), AS 966 (31.3%) and SS 397 (12.9%). The overall prevalence of malaria was 620 (20.1%). Malaria infection was highest in the southern senatorial zone (29.1%) than in the north and central zones ($p<0.05$). Infection was high with the males (21.2%), age-related malaria infection was significant ($p<0.05$) with age 1-10yrs recording the highest infection (28.0%). No significant difference ($p>0.00$) was recorded in the marital status of the patient with the widows/widower having 23.4% while degree of infection was significant for education-related infection ($p<0.05$) with the non-educated subjects recording highest infection (34.4%). Occupational related prevalence was significant ($p<0.05$) with high infection among traders (162 (28.7%). Significant difference was recorded in the marital status with the widow/widower recording a high prevalence of malaria 112 (23.4%). Our finding in the current study confirm that malaria remains a major challenge and there is need for periodic prophylactic administration of malaria drugs in the treatment regime of sickle cell anaemia patients.

Keywords: Malaria infection, genotypes, distribution, senatorial zones, Taraba State, Nigeria

INTRODUCTION

Background to the Study

Malaria caused by the protozoan parasite belonging to the species Plasmodium falciparum is considered the most significant public health problem worldwide and ranks top in its socio-economic, community and public health burden in tropical, sub-tropical areas, sub- Saharan Africa and South-West Asian countries

(Minakawa *et al* 2006; Hochman & Kim 2009; WHO 2011). It is a major cause of morbidity and mortality in malaria endemic communities in many African countries (Aninagyei *et al* 2022). *Plasmodium falciparum* accounts for the greater part of malaria linked mortality in Nigeria since more than 90% of the populace lives in malaria endemic areas (Guerra *et al* 2008 & Aninagyei *et al* 2022). *Plasmodium falciparum* is vectored by the female *Anopheles* mosquito and is the most dangerous form of malaria accounting for the highest rates of complications and mortality.

Plasmodium falciparum infection is more prevalent in sub-Saharan Africa than in other regions of the world. And almost every malarial death is caused by this unicellular protozoa (WHO 2017 & WHO 2021). In 2019, malarial cases were estimated to about 229 million globally in 87 malaria endemic countries, declining from 238 million in 2000 while malarial deaths reduced from 736,000 in 2000 to 409,000 in 2009. Nigeria accounts for 27% and 23% global cases and deaths respectively (WHO 2020). The endemicity of *Plasmodium falciparum* in Nigeria was established by the World Health Organization in 2017 and the population at risk includes children, pregnant women and the non-immune (Carrington 2001 & WHO 2017). *Plasmodium falciparum* infection is associated with serious co-infecctions, it causes severe anaemia leading to high mortality rates, impaired physical and cognitive development in children as well as reduced immune functioning (WHO 2011). *Plasmodium falciparum* infection is characterized by acute or intermittent or continuous fever which is accompanied by shivering, sweating, fatigue, vomiting, joint pains and headache. In severe infection, it causes yellow discoloration of the skin, seizures, coma and death (Caraballo 2014 & WHO 2017). A high malaria infection rate within a country is attributed to poverty promoting condition (Hotez *et al* 2006).

The factors that contribute to the spread and transmission of malaria depend on the interaction between the human host, the anopheles vector, malaria parasite and environmental conditions (Arora & Arora 2009). However, there is significant risk of infection in urban areas, where indiscriminate waste disposal and the presence of swamps, gutters and thick vegetation encourage the breeding of the mosquito vector that causes malaria (Anumudu 2006). In rural and urban areas, breeding sites of the female Anopheles mosquito is common during the rainy season where there is abundant of bushes, stagnant water around residential homes, while in the dry season, stagnant and smelling streams, irrigation ponds for dry season farming, indiscriminate disposal of domestic, commercial and industrial waste provides suitable environment for the infected mosquito to breed and proliferate. In the northern parts of Nigeria, due to the high shortage of water, many residents in both urban and rural areas harvest and store their water in commercial plastic tanks, clay pot, open buckets and basins. These water storage containers have been identified as good breeding sites of the mosquito vector.

In Nigeria, *Plasmodium falciparum* malaria exist all year round since hospitals have reported to treating malarial cases in all seasons and months of the year. The problems of rural-urban migration, persistence of poverty, environmental degradation and intractable problems of providing decent housing, potable water and sanitation are common in many cities and they cumulatively encourage the risks of malaria infection and parasite resistance through inconsistent malaria treatment options (Koram *et al* 1995 &Anumudu 2006).

The connection between sickle cell disease and malaria was first discovered in the 1940s (Esoh &Wonkam 2021). Patients with sickle cell traits (AS) have shown some resistance to severe forms of malaria (Depetris-Chauvin & Weil, 2018) because the sickle cell traits confer some resistance

to malaria (Piel *et al*., 2010). Individuals with sickle cell anaemia (SS) do not have protection from malaria. Malaria is both a precipitating factor of vaso-occlusion and a cause of haemolytic anaemia. Malaria causes destruction of erythrocytes by direct red cell lysis, phagocytosis and immune destruction with liberation of the parasites and erythrocyte material into the circulation, thus resulting in haemolytic crisis ((Serjeant, 2001. Gullet, 2001 & Luzzatto, 2012). Haematological derangements such as low platelet count, low white blood cell count and low lymphocyte counts are the most important predictors of *Plasmodium falciparum* infection.

The association of Plasmodium falciparum malaria with sickle cell anaemia is well described in Sub-Saharan Africa but is rare in the United States of America (Glickman *et al* 2021). However, this relationship has remained undocumented in Nigeria.

Sickle cell anaemia (SCA), an autosomal recessive disorder due to the presence of a mutated form of hemoglobin S (HbS) and is a neglected non-communicable disease of public health concern worldwide (Lopez *et al* 2014; Pecter *et al* 2021 & Sedrak *et al* 2023). Sickle cell anaemia is an inherited blood disorder from two abnormal copies of the β-globin gene which occurs in chromosome 11. The disease alters the shape of the red blood cells to a sickle shape which makes the red blood cells sticky and rigid and prone to getting trapped in small vessels, hence blocks blood from reaching the different parts of the body to cause pain and tissue damage (Lopez *et al* 2010). Sickle cell anaemia is a serious public health concern, present mainly in African countries (Makani *et al* 2007 & Rees *et al* 2010). The World Health Organization (WHO) estimates that 300,000 children are born of sickle cell disease each year, 75% of whom are in sub-Saharan Africa (Silver-Nunes & Ferreira 2007; Roucher *et al* 2012). Malaria remains a menace and is a public health concern in Nigeria, because it impacts on the health of the populace, affects income and capital which in turn constitutes a huge burden on the dwindling economy and result in poor health outcomes and an increasing severity of diseases among patients with sickle cell anaemia (Carrington, 2001 & WHO 2021).

Statement of problem

Despite the various interventions introduced by WHO, UNICEF, governmental and non-governmental organizations, malaria still remains a disease of public health concern since it continuously inflicts tremendous medical, social and economic burden on human population resulting in millions of death globally (WHO 2011; Minakawa *et al*, 2006). Malaria accounts for a quarter proportion of malaria linked morbidity and mortality in sickle cell patients. Some sickle cell patients are symptomatic while some are asymptomatic of *Plasmodium falciparum* malaria. Individuals with sickle cell anaemia are more vulnerable to life threatening malaria in malaria endemic countries hence, require hospitalization. Malaria infection causes haematological derangement hence patient with HbSS had a least chance of surviving malaria infection because even low-level malaria infection can precipitate severe anaemic crises that would likely prove fatal without rapid access to blood transfusion services (Uyoga *et al*, 2022). Since diagnosis of sickle cell anaemia is often overlooked and delayed until the disease becomes chronically manifested, studies could not have linked - malaria deaths in undiagnosed sickle cell anaemia patients resulting in dearth of information in the study location. Although, several epidemiological studies have been carried out on the prevalence of malaria in most parts of Taraba State, no research study have been carried out on *Plasmodium falciparum* malaria infection in sickle cell patients in the study area resulting in limited knowledge related to malaria, thus hindering appropriate and effective malaria intervention program to the targeted vulnerable population.

MATERIALS AND METHODS

Study Area

The study was carried out in Taraba State, located in the north-eastern part of Nigeria. The state lies within the coordinates $8^{0}00'N10^{0}'E$ with total area of 54,437km and the total population of 2,294,800 (National Population Commission, 2017). The river Taraba, Benue, Ibi and Donga which arises from the Cameroun Mountain supplies the state with adequate water supplies for its agricultural activities. The climate of the state is tropical with vegetation characterized by a typical Guinea Savannah. There are two distinct seasons, the wet and dry season. The residents of the state are mostly involved in commercial and subsistence farming and in livestock production. Communities living on the banks of the rivers engage in fishing all year round. Other occupational activities include: pottery, cloth-weaving, dyeing, mat-making, carving, embroidery and blacksmithing.

The Mumuye, Fulani, Jenjo, Wurkum and Kona tribes are predominantly located in the northern part of the state. While the Jukun, Chamba, Tiv, Kuteb and Ichen are the tribes that inhabits the southern parts of the state.

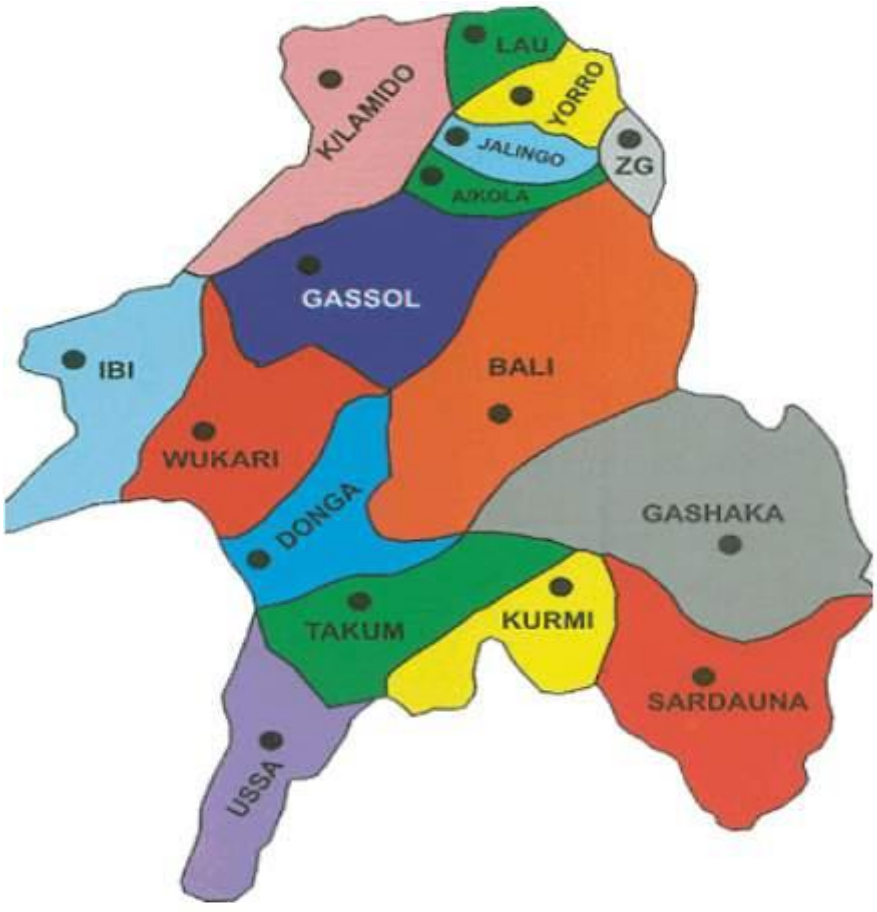

Fig 1: Map of Taraba State showing the selected study areas

Study Design

This study employed a cross-sectional research study designed to quantify the level of *Plasmodium falciparum* among patients with sickle cell anaemia in health facilities in three senatorial zones in Taraba State. Sickle cell anaemia (SS) patients with *Plasmodium falciparum* infection will serve as the test subjects while patients with the sickle cell trait (AS) without *Plasmodium falciparum* infection will serve as the control.

Research Instrument

Structured questionnaire was used to collect information on the respondents' age,

gender, occupation status, educational status, use of malarial chemoprophylaxis, use of long-lasting insecticide treated nets and proximity of residence to mosquito breeding sites.

Informed consent

Informed consent for participation is sought from all participants according to the standards for human experimentation and the Helsinki declaration.

Study Population

The study population comprises] of all consenting patients who present themselves in the Out-Patients Department (OPD) in designated hospitals and Primary Health Care Centres with various ailments from December 2022 to November 2023.

Sample size determination

The method of Sapoka (2006) was used to determine the sample size at 0.05 significant levels.

$$N = \frac{z^2pq}{d^2}$$

Where:

N= Desired sample size

z^2= Standard normal deviate set at 1.9^2

P= Proportion in the target population estimated to have a particular characteristic.

q= 1-p (either the patient has or does not have the characteristics)

d= Degree of accuracy set at 0.05.

Substituting the numbers into the formula

$$N=\frac{1.9^2 \text{ X } 820 \text{ X } 0.5}{0.5^2}$$

$$= \frac{3.61 \text{ X } 830 \text{ X } 0.5}{0.0025}$$

$$= \frac{1498.15}{0.0025}$$

$$=599.260$$

The calculated sample size N was approximately 600

Ethical Permission

Ethical permission was obtained from the ethical committee of the State Ministry of Health and the directors of the health facilities where blood specimens will be collected.

Specimen collection

Blood samples was collected from consenting participants in the three senatorial zones. Blood samples will be collected from the participants regardless of their age, gender, religion, occupational and educational status, who have not received any anti-malarial drugs for the past two months but with clinical presentation of fever, headache, rigors, vomiting, diarrhea, general malaise, weakness, enlarged spleen and liver. The method of sample collection will be by venepuncture (Cheesebrough, 2006). A 10.0ml of blood will be collected aseptically into labeled EDTA containers.

Screening of Blood Samples

Blood samples was introduced into acetate paper and place in the gel electrophoresis machine to determine the genetic status of the patient. All blood samples carrying the sickle cell traits (AS) and the sickle cell anemia (SS) was be screened for *Plasmodium falciparum* infection using the rapid diagnostic test (RDT). The RDT test that was positive for malaria was further subjected to the gold standard microscopy for malaria parasite using the thick and thin blood films flooded with Giemsa stain and examined under the x100 objective lens microscope. A rough estimate of parasitaemia was made from the positive blood films. The level of parasitemia was quantified as low (+), moderate (++) and high (+++). Blood samples showing large ring stage of *Plasmodium falciparum* was further processed for other haematological parameters (WHO, 2022).

Hb Electrophoresis for Screening of Sickle Cell Trait (AS) and Sickle Cell Anaemia (SS)

The collected blood samples were centrifuged at 1200g for 5 minutes, the electrophoresis tank is prepared with TEB buffer, and the cellulose acetate was soaked in the buffer for 5 minute. The well plate

was filled with diluted samples and covered with glass slide to prevent evaporation. A second well plate was loaded with Zip Zone Prep solution. Both was applied to a blotting paper and the cellulose acetate strip was blotted twice between two layers of clean blotting paper. The cellulose acetate strip was not allowed to dry. The applicator was loaded by depressing the tips into the sample wells twice and the cellulose acetate plates was placed across the bridges. After 25 minutes of electrophoresis, the cellulose acetate was immediately transferred to ponceau S, fixed and stained for 5 minutes. Excess stain was removed by washing for 5 minutes in the first acetic reservoir. The result was interpreted according to the manufacturer's instruction.

Preparation of Thin Blood Films for Malaria Investigation

Thin blood film was prepared according to the method described by Cheesbrough (2006) and Baker *et al* (2007). A drop of blood was placed on a clean grease free microscope slide, about 1cm from one end of the slide and a spreader with smooth edge was placed in front of the drop of blood inclined at angle 45^0. The spreader was drawn backward to make contact with the blood. A quick forward movement was made to enable the blood spread out. This film was made to cover about half of the slide and to assume a tongue shape. The thin film was air-dried and labeled accordingly.

Preparation of Thick Film for Malaria Investigation

Thick blood film was prepared according to the method described by Cheesbrough (2006). Using a clean grease-free microscope slide, a drop of blood was added to the centre of the slide and a large drop of blood was added about 15mm to one end of the slide. Using a smooth edge slide spreader, the large drop was spread to make a thick film to cover an area of about 15 x 15mm. The film was fixed with absolute methanol for 15 minutes and labelled

The parasite density will be counted according to the WHO standard formula (WHO, 2016).

$$\text{Parasite}/\mu l = \frac{\text{number of parasites counted x8000 white cells}/\mu l}{\text{Number of white cells counted}}$$

Where 8000 = putative means of leucocytes. The level of parasitemia will be quantified as low (+), moderate (++) and high (+++).

STATISTICAL ANALYSIS

The data obtained from this study was entered into Microsoft Excel and exported to Statistical Package for Social Sciences (SPSS) version 20.0 for data analysis. Chi square (χ^2) test was used to compare the relationship between infection and demographic profiles of the participants.

RESULTS

Distribution of genotype in 3 Senatorial Zones

Table 1 shows the distribution of genotype in the 3 Senatorial Zones. Results obtained indicated south zone had genotype AA (56.0%), AS (31.5%) and SS (12.5%). The central zone had genotype AA (51.3%), AS (32.7%) and SS (16.1%). North zone had genotype AA (60.2%), AS (29.7%) and SS (10.0%). The degree of distribution was significant for genotype in the 3 senatorial zones ($\chi 2$= 24.092, P<0.05).

Table 1: Distribution of genotype in 3 Senatorial Zones

Senatorial Zones	AA	Genotype AS	SS	Total Examined
South	567(56.0)	319(31.5)	125(12.5)	1012
Central	542(51.3)	345(32.7)	170(16.1)	1056
North	612(60.2)	302(29.7)	102(10.0)	1016
Total	1721(55.8)	966(31.3)	397(12.9)	3084

χ^2 = 24.092; df=2, P <0.05 for distribution of Genotype

Distribution of genotype in Relation to gender

Table 2 shows the distribution of genotype in relation to sex. Results obtained indicated male had genotype AA (44.6%), AS (48.0%) and SS (47.3%). The Females genotype AA (55.3%), AS (52.0%) and SS (53.0%) respectively. The degree of distribution was not significant for genotype of sex-related (χ^2=2.791, P>0.05).

Table 2: Distribution of genotype according to Gender

Gender	AA	Genotype AS	SS	Total Examined
Male	769(44.6)	462(48.0)	188(47.3)	1419
Female	952(55.3)	504(52.0)	209(53.0)	1665
Total	1721(55.8)	966(31.3)	397(12.9)	3084

χ^2 = 2.791; df=1, P >0.05 for distribution of Genotype

Distribution of genotype in relation to age

Table 3 shows the distribution of genotype in relation to age. Results obtained indicated age group 1-10yrs genotype had AA (55.0%), AS (25.1%) and SS (20.0%) respectively. 11-20yrs had genotype AA (54.8%), AS (30.8%) and SS (14.3%). The 21-30yrs had genotype AA (54.5%), AS (34.4%) and SS (11.1%). 31-40yrs had genotype AA (51.2%), AS (39.8%) and SS (9.0%). While the >40yrs had genotype AA (70.0%), AS (24.1%) and SS (6.0%) respectively for distribution of genotype in relation to age group. The degree of distribution was significant for age-related genotype (χ^2= 99.947, P<0.05).

Table 3: Distribution of genotype according to Age

Age	AA	Genotype AS	SS	Total Examined
1-10	405(55.0)	185(25.1)	146(20.0)	736
11-20	386(54.8)	217(30.8)	101(14.3)	704
21-30	347(54.5)	219(34.4)	71(11.1)	637
31-40	333(51.2)	259(39.8)	58(9.0)	650
>40	250(70.0)	86(24.1)	21(6.0)	357
Total	1721(55.8)	966(31.3)	397(12.9)	3084

χ^2 = 99.947 ; df=4, P <0.05 for distribution of Genotype

The results obtained indicates *Plasmodium falciparum* infection in the South zone is 29.1%, Central zone had 19.0.1% and North zone had 12.3. The result also indicated that male genotypes were AA genotype had malaria infection of (83.4%), AS (7.3%) and SS (9.3%). The female genotypes show malaria infection of AA (85.0%), AS (7.2%) and SS (7.8%).

The results obtained revealed that age group 1-10yrs old with AA genotype had malaria infection of 76.7%, AS had malaria of 9.7%, SS had malaria 13.6%, 11-20yrs old with AA genotype had 83.3%, AS had 8.7%, SS had 8.0%, 21-30yrs old with AA genotype had 92.4%, AS had 3.2%, SS had 4.4%, 31-40yrs old with AA genotype had 87.8%, AS had 6.1%, SS had 6.1% and >40yrs old with AA genotype had 87.5%, AS had 8.3%, SS had 4.2%.

Marital status revealed that single genotypes with AA genotype had malaria infection of 81.2% AS had 8.0%, SS had 10.8% Married genotypes with AA had 87.7%, AS had 5.3%, SS had 8.3%.

Malarial infection result indicated that non-educated patients with AA genotype had 77.7% AS had 8.9%, SS had 13.4% primary genotypes with AA had 84.8%, AS had 7.9%, SS had 7.3%, secondary genotype with AA genotype had 86.7%, AS had 6.3%, SS had 7.0% and Tertiary genotypes with AA genotype had 91.9%, AS had 4.5%, SS had 3.6%.

Malaria infection according to patients occupation indicated that artisan with the

AA genotype had 78.3% AS had 10.1%, SS had 11.6%. Traders with AA genotype had 84.6%, AS had 6.8%, SS had 8.6%, Farmers with AA genotype had 86.0%, AS had 7.6%, SS had 6.4%, Students with AA genotype had 84.6%, AS had 5.8%, SS had 9.6% and Civil servant with AA genotype had 90.7%, AS had 3.7, SS had 5.6%.

Distribution of malaria according to patients socio-demographic profile

Socio-demographic profile	Overall sample examined	AA No. infected (%)	AS No. infected (%)	Genotype SS No infected (%)	$\chi 2$	P-value
Gender						
Male	1419	251 (17.6)	22 (1.5)	28 (2.0)	0.436	0.005
Female	1665	271 (16.2	23 (1.3)	25 (1.5)		
Age						
1-10	736	158 (76.7)	20 (9.7)	28 (13.6)	19.19	0.005
11-20	704	125 (83.3)	13 (8.7)	12 (8.0)		
21-30	637	146 (92.4)	5 (3.2)	7 (4.4)		
31-40	650	72 (87.8)	5 (6.1)	5 (6.1)		
>40	657	21 (87.5)	2 (8.3)	1 (4.2)		
Marit. status						
Single	965	145	14 (1.4)	16 (1.6)	3.756	0.005
Married	906	164	10 (1.1)	13 (1.4)		
Widowed	479	93	9 (1.8)	10 (2.0)		
Separated	734	120	12 (0.2)	13 (1.7)		
Edu. Status						
Non-edu	587	157 (26.7)	18 (3.0)	27 (4.6)	13.460	0.005
Pr. sch	765	139 (18.2)	13 (1.7)	12 (1.5)		
Sec. sch	804	124 (15.4)	9 (1.1)	10 (1.2)		
tertiary	928	102 (11.0)	5 (0.5)	4 (0.4)		
Occu. status						
Artisan	569	101 (78.3)	13 (10.1)	15 (11.6)	6.656	0.005
Trader	563	137 (84.6)	11 (6.8)	14 (8.6)		
Farmer	700	147 (86.0)	13 (7.6)	11 (6.4)		
Student	650	88 (84.6)	6 (5.6)	10 (9.6)		
Civil serv	602	49 (90.7)	2 (3.7)	3 (5.6)		

Comparison of RDT and Microscopy

A total of three thousand and eighty-four blood samples were examined using the stained blood film microscopy and the rapid diagnostic test. 596 (96.1%) was positive for the gold standard microscopy while in rapid diagnostic test, 330 (13.4%) was positive for malaria infection. This shows that the gold standard microscopy was more sensitive to malaria infection.

Comparison of RDT and Microscopy

RDT	Microscopy		Total
	-ve	+ve	
Negative	2134 (86.6%)	24 (3.8%)	2158
Positive	330 (13.4%)	596 (96.1%)	926
Total	2464	620	3084

DISCUSSION

Malaria and sickle cell anaemia are major public health concern because they are attributed to high rates of morbidity and mortality in sub-Saharan Africa and especially among citizens with low socio-economic status and illiteracy.

The current study revealed a high haemoglobin genotype AA 1721 (55.8%), AS 966 (31.3%) and SS 397 (12.9%). This differs with the haemoglobin distribution of AA 84 (84.0%), AS 16 (16.0%) and SS 0(0.0%) in Buea Health district of Cameroun (Ngwengi *et al* 2020), and the haemoglobin genotype distribution AA (81.54%), AS (16.92%) and SS (1.54%) in Madonna University, Okija, Anambra state (Eledo *et al* 2018). Also, the AA (88.1%), AS (10.2%) and SS (0.7%) in Akure, Ondo

state, Nigeria (Akinboro *et al* 2016). Mustapha & Abubakar (2001) recorded high SS genotype (17.1%) in Kano metropolis and Tukur et al (2017) recorded AA (63.1%) AS (26.0%) and SS (6.8%). The high rate of AS and SS haemoglobin genotype in the study area is attributed to the fact that the research was focused in the rural areas where there is high rate of illiteracy, ignorance, early forced marriages/child birth, out of wedlock teenage pregnancies and also the lack of genetic testing facilities. While the high AA variant could be due to intermarriages between families, tribes and ethnic groups.

The study recorded high haemoglobin variant among females AA (55.3%) AS (55.0%) and SS (53.0%) than males AA (44.6%), AS (48.0%) and SS (47.3%). This differs with the study of Mohammed-Nafiu *et al*., (2020) where more males had low haemoglobin variants among newborn infants in National Hospital Abuja. There was no explanation proffered for the gender predilection.

High prevalence was recorded in the AA genotypes 250 (70.0%) among this >40yrs while low prevalence 333 (51.2%) was recorded among 31-40 yrs group. The prevalence of AS increases with age such that 1-10yrs had 185 (25.1%), 11-20yrs recorded 217 (30.8%), 21-30yrs had 219 (34.4%), while 31-40yrs had 259 (39.8%). This agrees with the study of Weatherall & Clegg (2001)., Modell & Darlison (2008)., Otoikhian (2019) and Mohammed-Nafiu *et al* (2020). This could be attributed to high mortality associated with the SS in childhood. In this study, the SS genotypes decreases with age;1-10yrs 146 (20.0%), 11-20yrs 101 (14.3%), 21-30yrs 71 (11.1%), 31-40yrs 58 (9.0%) and >40yrs 21 (6.0%). The decrease in the SS genotype could be due to high mortality associated with SS in childhood resulting in few survivors among adults especially in rural areas where there is no routine screening, early diagnosis, blood transfusion services and other medical interventions.

The current study corroborates with the research findings of previous researchers on the endemicity of malaria infection in different communities in Nigeria. The prevalence of 620 (20.1%) falls within the Nigerian malaria risk map estimates of less than 20% in certain zones to more than 70% in other zones (Okonko *et al* 2010). The 20.1% prevalence is lower than those reported in previous study in Akure in Southwestern Nigeria and among rural inhabitants of Gabon (Awosulu *et al*., 2019 & Woldearegal *et al*., 2019). The low prevalence is attributed to the use of local/indigenous plants and tea as malaria prophylactic treatments and reduced mosquito breeding sites due to the dry weather condition.

High malaria infection 522 (19.9%) was recorded in the AA haemoglobin variant than in the AS 45 (1.5%) and the SS 53 (1.7%). This is consistent with the results among Kenyan children (Sultana *et al*., 2017), among children in Korea (Kuesap & Na-Bangchang 2018), among children in Jos (Njila *et al.*,2022) and in Yemen (Albiti & Nsiah.,2014). The current result from this study and previous study shows that malaria infection is low for the HBSS (Kepha *et al*., 2016 & Ebadan et al., 2017). The high prevalence of malaria in the AA haemoglobin variant as compared to the SS variant may be due to the fact that the red blood cells create favorable environment for the Plasmodium parasite to thrive than the SS genotype. This could be due to the high rate of oxygen consumption and a large amount of haemoglobin ingested in the peripheral blood during the mosquito vector replication stage. (Njila *et al*., 2022, Albiti *et al* 2014 & Ebadan *et al* 2017).

Gender distribution of malaria infection reveals that males have higher infection301 (21.2%) than their female counterparts (19.2%). this agrees with previous research findings from other malaria endemic areas in Nigeria (Gebretsadik *et al* 2018 & Escobar *et al*., 2020). The mosquito vector is mostly active in its biting activities in the night when males are engaged in outdoor

activities that exposes them to the mosquito bites, they are move without wearing clothes that covers/protects them from mosquito bites and they are less concerned about malaria prevention than the females.

The age-related prevalence of malaria infection shows that malaria was higher in the 1-10yrs old 206 (28.0%), 11-20yrs old 150 (21.3%) and 21-30yrs 158(24.8%) than the 31-40yrs old 82(12.6%) and the >40 yrs old 24 (3.7%) in all the haemoglobin variants. This agrees with research findings of 97 (53.9%) among ages 1-10yrs by Ibrahim *et al* (2023) in rural southwestern Nigeria. According to WHO (2018), children between ages 1-5yrs are more susceptible to malaria infection and this could be attributed to low level of immunity while children between 6-10yrs suffer malaria infection due to exposure to the mosquito vector by playing in stagnant water bodies habouring the mosquito vector, and playing without clothes. Malaria infection decreases with age (Ibrahim *et al* 2023).

Concerning education related prevalence, this study revealed that malaria infection is higher among the non-educated subjects 202 (34.4%) than the educated subjects, primary 164 (21.4%), secondary 143 (17.7%) and tertiary 111 (11.9%). This agrees with the findings of Obimakinde *et al* (2018) who revealed that non educated have high malaria infection than the educated. The non-educated are not knowledgeable about the preventive practices of malaria infection. They engage in outdoor activities especially in the evenings and night during the active biting period of the female anopheles mosquito.

Result from this study shows that malaria infection is high among low-income earners such as traders 167 (28.7%), farmers 171 (24.4%) and artisans 129 (22.7%) than students 104 (18.0%) and civil servants 54 (9.0%). This agrees with the findings of Obimakinde *et al* (2018). Low-income earners are mostly exposed to mosquito bites in their daily quest for survival. Majority of them work bare-bodied and late into the night. While the students and civil servants make use of mosquito repellant creams, socks, long sleeves and sleep under long lasting insecticide treated nets to protect themselves against mosquito bites.

The sensitivity, specificity, PPV and NPV for RDT in comparism to microscopy was 53.4%, 86.6.%, 64.3% and 98.8% respectively. Most comparative study of RDT and microscopy showed both to be sensitive in the diagnosis of malaria (Reybun *et al* 2007., Azikiwe *et al* 2012., Ojurongbe *et al* 2013, Nwachukwu 2014). The study got a relatively high sensitivity of 53.4%. This differs from the research done in Angola (Fancony *et al* 2013) and Uganda (Hopkins *et al* 2008), where RDT recorded a higher sensitivity and specificity of 78.4% and 62.0% respectively. Research study by Elechi *et al* (2015) recorded only 8.3% sensitivity for RDT in under five children with acute uncomplicated malaria. The sensitivity to RDT was lower than 69.6% Ben-Edet *et al* (2004) in Lagos state, 66.0% in Obimakinde *et al* 2018 in Akure and 82.0% Adesanmi *et al* 2011 in Enugu state, Nigeria.

The variation in the sensitivity of RDT to microscopy could be attributed to the fact that different populations show variation in the diagnostic kits mainly due to the differences in the types of RDTs used or the test methods adopted by the microscopist (Garba *et al*, 2016). The level of the Plasmodium falciparum density could affect or alter the RDT result since RDT does not detect low parasite density of <50ml.Furthermore, the presence of other plasmodium species could also be a factor of interest, exposure to extreme temperature during transportation and storage could contribute to poor performance of RDT as observed by (Garba *et al* 2024). Another reason for the variation in the RDT sensitivity could be due to the fact that majority of pharmaceutical shops that deals with the sales and distribution of RDTs keep the packs on the counter shelves without considering sunlight and humidity instead of storing them in the appropriate temperature

as specified by the manufacturers for optimal performance of the RDT kits.

CONCLUSION

Findings from the study revealed a high distribution of HbAS but low HbSS among the study populace despite increased awareness of genetic counselling and testing advocated by the World Health Organization. The study reveals that malaria infection caused by *Plasmodium falciparum* is endemic in different parts of Taraba state and it impacts on the heamatological parameters of the patients. Comparing the diagnostic tools, PCR was more sensitive and specific than microscopy and RDT.

Contribution to knowledge

The current study which is believed to be the first attempt to document systematically the distribution of genotypes and quantitative analysis of Plasmodium falciparum among sickle cell anaemia patients in three senatorial zones in Taraba state has the following contributions to knowledge.

1. The finding has shown that Taraba state has a high distribution of sickle cell anaemia patients (both diagnosed and undiagnosed). The data collected could provide baseline information necessary for reducing and eliminating this genetic disorder in the state.
2. The significant association in the prevalence of malaria in HbAA, HbAS and HbSS genotype variant could be used as a criterion for including malaria prophylaxis in the treatment regime of sickle cell patients.

RECOMMENDATION

Based on the findings of this study, the following recommendations were made:

1. There is need to introduce genetic counselling and testing in all health facilities in the state.
2. There is need to intensify the malaria control programs by the PMI, USAID and all donor organizations and improve strategies to reduce/eradicate malaria morbidity and mortality among patients with sickle cell anaemia.
3. Regular public enlightenment campaigns, health education workshops and mass media campaign should be organized to educate the populace on the risk factors of malaria infection.
4. Monthly environmental sanitation should be organized and embarked upon to keep all drainages clean and clear the bushes around homes.
5. There should be yearly distribution of mosquito repellants and long-lasting insecticide treated nets.
6. The health sector should advocate the wearing of protective clothing.
7. Sickle cell anaemia patients should be placed of regular malaria prophylaxis at all times.

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REFERENCES

1. Akinyanju O.O, Otaigbe A.I Ibidapo M.O (2005). Outcome of holistic care in nigerian patients with sickle cell anaemia. Clinical Lab Haematol. 2005 Jun;27(3):195-199.
2. Aninagyei, E., Tettey, C.O., Kwansa-Bentum, H., Boakye, A.A, Ghartey-Kwansah, G., Boye, A (2022). Oxidative stress and associated clinical manifestations in Malaria and sickel cell (HbSS) co-morbidity. PLOSONE 76: https: doi.org/10.1371/journal.pone.0269720
3. Anumudu, C. I., Adepoju, A., Adediran, M., Adeoye, O., Kassim, A., Oyewole, I., & Nwuba, R. I. (2006). Malaria prevalence and treatment seeking behaviour of young Nigerian adults. *Annals of African Medicine*, *5*(2), 82-88.
4. Arman M, Raza A, Tempest L. J, Lyke K.E, Thera M. A, Kone A, et al. Platelet-mediated clumping of Plasmodium falciparum infected erythrocytes is associated with high parasitemia but not severe clinical manifestations of malaria in

African children. Am J Trop Med Hyg. 2007;77(5):943–6.
5. Bilal, A.J., Gasim, I.G., Karsani, H. A., Elbashir, L.M., Adam, I (2016). Malaria parasite density estimation using actual and assumed white blood cells count in children in Eastern Sudan. Journal of Tropical Pediatrics, vol 62, issue 2, April 2016, pg 171-175.
6. Caraballo, H., & King, K. (2014). Emergency department management of mosquito-borne illness: malaria, dengue, and West Nile virus. *Emergency medicine practice*, *16*(5), 1-23.
7. Carrington, A. (2001). Malaria: its human impact, challenges, and control strategies in Nigeria. *Harvard health policy review*, *2*(2), 1-3.
8. Depetris-Chauvin, E., & Weil, D. N. (2018). Malaria and early african development: Evidence from the sickle cell trait. *The Economic Journal*, *128*(610), 1207-1234.
9. Elkanah, O.s, Obiorah, S.C, Onyeukwu, O.C, Elkanah, D.S, Egeonu S.U (2020). Haematological derangement due to P.falciparum malaria in patients attending selected health centres in Ardo-kola,L.G.A, Taraba state. Biomedical Sciences. Vol 6 no 3, 2020 pp 67-73
10. Esoh, K., & Wonkam, A. (2021). Evolutionary history of sickle-cell mutation: implications for global genetic medicine. *Human molecular genetics*, *30*(R1), R119-R128.
11. Gullet P. (2001) Insecticides treated nets in Africa: where do we stand? Africa Health 2001: Vol 23(6): 20-23.
12. Hochman, S., & Kim, K. (2009). The impact of HIV and malaria coinfection: what is known and suggested venues for further study. *Interdisciplinary perspectives on infectious diseases*, *2009*.
13. Hoontrakoon S, Suputtamongkol Y. Exchange transfusion as an adjunct to the treatment of severe falciparum malaria. Trop Med Int Health. 1998;3(2):156–61.
14. Horstmann RD, Dietrich M. Haemostatic alterations in malaria correlate to parasitaemia. Blut. 1985;51(5):329–35.
15. Hotez, P. J., Molyneux, D. H., Fenwick, A., Ottesen, E., Ehrlich Sachs, S., & Sachs, J. D. (2006). Incorporating a rapid-impact package for neglected tropical diseases with programs for HIV/AIDS, tuberculosis, and malaria: a comprehensive pro-poor health policy and strategy for the developing world. *PLoS medicine*, *3*(5), e102.
16. Kreil A, Wenisch C, Brittenham G, Looareesuwan S, PeckRadosavljevic M. Thrombopoietin in Plasmodium falciparum malaria. Br J Haematol. 2000;109(3):534–6.
17. Lamikanra AA, Theron M, Kooij TW, Roberts DJ. Hemozoin (malarial pigment) directly promotes apoptosis of erythroid precursors. PLoS One. 2009;4(12):e8446.
18. López, C., Saravia, C., Gomez, A., Hoebeke, J., & Patarroyo, M. A. (2010). Mechanisms of genetically-based resistance to malaria. *Gene*, *467*(1-2), 1-12.
19. Luzzatto, L., (2014) Sickle cell anaemia and malaria. *Mediterranean Journal of Haematology and Infectious Diseases*. 2012;4(1):E2012065-E2012065
20. Makani, J., Williams, T. N., & Marsh, K. (2007). Sickle cell disease in Africa: burden and research priorities. *Annals of Tropical Medicine & Parasitology*, *101*(1), 3-14.
21. Minakawa, N., Omukunda, E., Zhou, G., Githeko, A., & Yan, G. (2006). Malaria vector productivity in relation to the highland environment in Kenya. *The American journal of tropical medicine and hygiene*, *75*(3), 448-453.
22. Mustapha, O.T., Abubakar, F.H (2001). Study of the prevalence of sickle cell disease in Kano metropolis and its suburbs in northern Nigeria. Nigerian Journal of Basic Applied Biology. 2001; 10: 219-225
23. Nakazawa S, Looareesuwan S, Fujioka H, Pongponratn E, Luc KD, Rabbege J (1995). A correlation between sequestered parasitized erythrocytes in subcutaneous tissue and cerebral malaria. *American Journal of Tropical Medical Hygiene*. 1995;53(5):544–6.
24. Pecker, L. H., & Lanzkron, S. (2021). Sickle cell disease. *Annals of internal medicine*, *174*(1), ITC1-ITC16.
25. Piel F.B., Patil A.P., Nyangiri O.A., Gething P.W., Williams T.N., Weatherall D.J., Hay S.I (2010) Global distribution of sickle cell gene and geographical confirmation of malaria hypothesis. *American Journal of Nature Communication*. 2010; 1:104.
26. Rees D.C, Williams T.N Gladwin M.T (2010). Sickle cell disease. The Lancet 2010 376(9757). 2018-2031.
27. Rees, D. C., Williams, T. N., & Gladwin, M. T. (2010). Sickle-cell disease. *The Lancet*, *376*(9757), 2018-2031.

28. Roucher, C., Rogier, C., Dieye-Ba, F., Sokhna, C., Tall, A., & Trape, J. F. (2012). Changing malaria epidemiology and diagnostic criteria for Plasmodium falciparum clinical malaria.
29. Sedrak, A., & Kondamudi, N. P. (2021). Sickle cell disease. In *StatPearls [Internet]*. StatPearls Publishing.
30. Serghides L, Smith TG, Patel SN, Kain KC. CD36 and malaria: friends or foes? Trends Parasitol. 2003;19(10):461–9.
31. Serjeant G.R & Serjeant B.E (2001). Sickle cell disease. 3rd Ed. Oxford University Press 2001.
32. Sowunmi A, Akindele J.A, Balogun M.A. Leukocyte counts in falciparum malaria in African children from an endemic area. Afr J Med Med Sci. 1995;24(2):145–9.
33. Sultana M, Sheikh N, Mahumud RA, Jahir T, Islam Z, Sarker AR (2017). Prevalence and associated determinants of malaria parasites among Kenyan children. Trop Med Health 2017; 45(1):25. https://doi.org/10.1186/s41182-017-0066-5 PMID: 29085254
34. UNICEF. (2010). Building on Good Practice: Advancing Gender Equality and Girls' Education March 2015 to April 2018.
35. Waitumbi JN, Opollo MO, Muga RO, Misore AO, Stoute JA. Red cell surface changes and erythrophagocytosis in children with severe Plasmodium falciparum anemia. Blood. 2000; 95(4):1481–6.
36. White R.H, (1973). Quartan malarial nephrotic syndrome. Nephron. 1973;11(2): 147–62
37. WHO (2001). The prevention and management of severe anaemia in children in malaria- endemic regions of Africa: A review of research: WHO; 2001
38. WHO (2010). Basic malaria microscopy Part 1. Learners' Guide. Second Edition.
39. WHO (2015). QA manual for malaria microscopy. 2nd Edition. Geneva. 2015.
40. WHO (2017). World malaria report. Geneva, Switzerland: World Health Organization, pp 32 – 42
41. WHO (2020). World Malaria Report 2020. World Health Organzation. ISIPP. https: //www.who.int/publications/i/item/9789240 015791
42. WHO (2021). World Malaria Report, 2021. World
43. World Malaria Report. (2017). World Health Organization, 2017.
44. Woldearegal T.G, Lalremruata A, Nguyen T.T, Gmeiner, M,Veletzky L, (2019). Characterization of plasmodium infections among inhabitants of rural areas in Gabon. Sci, Rep; 9 (2019). P 9784.
45. Yagmur Y, Kara IH, Aldemir M, Buyukbayram H, Tacyildiz IH, Keles C. Spontaneous rupture of malarial spleen: two case reports and review of literature. Crit Care. 2000;4(5): 309–13.
46. Wyler DJ. Peripheral lymphocyte subpopulations in human falciparum malaria. Clin Exp Immunol. 1976;23(3): 471–6.
47. Zhang Y, Telleria L, Vinetz J.M, Yawn D, Rossmann S, Indrikovs A.J. Erythrocytapheresis for Plasmodium falciparum infection complicated by cerebral malaria and hyperparasitemia. J Clin Apher. 2001;16(1):15–8.
48. Zingman BS, Viner BL. Splenic complications in malaria: case report and review. Clin Infect Dis. 1993;16(2):223–32.
49. Zucker JR, Campbell CC. Malaria. Principles of prevention and treatment. Infect Dis Clin North Am. 1993;7(3):547–67.

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