

and controlled with early diagnosis and some countries lack the resources to do this effectively (Peter, 2017).

Methods

Study population

The study population consist of 384 randomly selected FCT residents made of 192 males and 192 females from 0 to >60 years visiting FCT Hospitals.

Sample size

The sample size was determined by:

$$n = Z^2 pq / ME^2 \text{ (} \textit{https://www.slideshare.net/zubis/sample-size-13281869} \text{)}$$

Where n= Sample size for this research

Z= $Z_{\alpha/2}$ -score =1.96 at 95% Confidence interval

p= Estimated proportion (50%, as actual proportion is unknown) of FCT residents with malaria parasitemia among different blood Genotypes.

ME= Marginal error= ± 0.05 .

$$\text{Therefore, } n = Z^2 pq / ME^2 = (1.96 \times 1.96 \times 0.5 \times 0.5) / 0.05 \times 0.05 = 384.16 \approx 384$$

Ethical approval

Ethical approval was obtained from the FCT Hospitals management board where all the subjects that were used for this study were confirmed to be asymptomatic for malaria and not on any antimalarial medication for at least seven days before sample collection.

Informed consent

Informed consent was obtained from volunteers and from parent's legal guardian of the under-aged participants.

Inclusion criteria

Included: written/thumb-printed informed consent obtained from the adult volunteers and parent or legal guardian of the under-aged participants, permanent resident in the Federal Capital Territory and all volunteers from 0 - >60 years of age.

Exclusion criteria

Exclusion criteria from the study were those of major congenital defects, any chronic disease, or anaemia defined as measured haemoglobin <6 g/dL and are not living in FCT. All asymptomatic subjects who are on anti-malaria were also excluded.

Sampling Area

Federal Capital Territory(FCT) is north of the confluence of the Niger and Benue Rivers. It has a land area of 8,000sqkm and it falls within latitude 8° 25' N and 9° 20' North of the Equator and longitude 5° 45' and 7° 39' Bordering the FCT are the states of Kaduna to the northeast, Plateau to the east and south, Kogi to the southwest, and Niger to the west and northwest. Abuja have been growing at 20% to 30% per year. Phase 1 of the city is divided into five (5) districts - Central, Garki, Wuse, Maitama, and Asokoro. Phase 2 is divided into five (5) districts - Kado, Durumi, Gudu, Utako and Jabi. Phase 3 districts is divided into four (4) districts - Mabuchi, Katampe, Wuye and Gwarimpa. Total population of Abuja FCT (1,406,239): Abaji (58642), Abuja Municipal-AMAC (776298), Bwari (229274), Gwagwalada (158618), Kuje (97233) and Kwali(86174) (NPC, 2006). More than 70% of the land is rural. Abuja is in tune with nature with abundant hills, highlands and other distinguishing features that make it a delight to behold. The largest indigenous group in Abuja are the Gbanyi (also known as Gwari); the next largest indigineous

group are Koro; smaller indigeneous group include; Gade, Egbura, Gwandara, Bassa and Ganagana (Awah, Nwanedo, *et al*, 2012).

Sampling

Using sterile needle and syringes, two milliliters (2mls) of venous blood was collected from 384 randomly selected FCT residents visiting FCT General Hospitals comprising of 192 males and 192 females by venipuncture (by tying a tourniquet around the upper arm and sterilizing arm with 70% ethanol to sterilize and increase blood pressure in the veins. The 2mls venous blood sample collected was dispensed into ethylene-diamine-tetra-acetic acid(EDTA) anti-coagulated blood containers, properly mixed by standard method and labeled appropriately(Cheesbrough,1998).

Malaria diagnosis

Subjects were tested for malaria parasite by x10 then, x100 microscopic objectives examination of Giemsa stained thick and thin blood films on grease free microscopic slide under oil immersion. The presence of even one parasite per microscopic field indicate positivity for malaria parasite. Level of parasitemia or degree of severity of malaria infection were graded as low+ (1 to 999 / μ L), moderate++ (1000 to 9999 / μ L) and severe+++ (>10,000 / μ L) (Idonije *et al.*, 2011 and Awah, Nwanedo, *et al*, 2012).

Determination of haemoglobin genotype

The haemoglobin genotypes were determined by the cellulose acetate membrane electrophoresis (CAME) as describe in John and Lewis (1986), Awah and Uzoegwu (2006) and Tidi *et al.* (2013). Briefly: fifty micro-liters of washed cells was added into khan tubes containing 50 micro-liters of 0.1% white saponin and mixed thoroughly (haemolysate). The haemolysate was centrifuged to remove any debris. The supernatant was used for the test. Haemoglobin lysates are spotted in triplicates at 0.5 cm apart of one end of well-dried cellulose acetate strips. Reference HbAS and HbA Chaemolysates was spotted alongside also as control. The spots were allowed to dry and the strips placed in an electrophoretic tank to form a bridge between the anode and cathode of the tank in such a way that haemoglobin bands migrate towards the anode. Both compartments of the tank contained the same working buffer–Tris-EDTA Borate buffer (pH 8.9). A constant voltage of 150 V (2mA) was applied through a Shandon power supply unit to achieve excellent separation within 5–10 minutes. After separation, the haemoglobin bands were visualized directly or otherwise stained for 2 minutes with 2% Ponceau S stain and then de-stained with distilled water. Pink spots of separated haemoglobin spots were observed upon white background of the cellulose acetate membrane. HbA migrated fastest followed by HbS then HbC, etc respectively (Evans, 1971 and Awah, Nwanedo, *et al*, 2012).

Statistical analysis of data

The data from this survey was analysed using Epi-Info Software Version 7. Only the test results with malaria parasitaemia among different genotypes were statistically analysed and presented in frequency tables, bar chart and percentages.

Results

The results were as presented in frequency Tables 1 to 5 and Figure 1 below: Malaria parasitaemia was more in female (58.8%) than male (41.4%) (Table1); and more in 0-10 (42.0%), 11-20 (35.2%) and 21-30(14.5%). Therefore, malaria parasitaemia was 91.7% within age range of 0-30 years of the studied group (Table 2); more (Table 3) in Gwagwalada (24.6%), followed by Bwari (21.4%), Kuje (18.7%), Kwali (17.7%), Abaji(15.5%) and was least in AMAC(2.1%). From figure 1 and Table 4 indicate that malaria parasitaemia was highest in blood genotype AA (69%), AS (24.2%), AC (4.7%) and least in SS(2.1%) and SC(0.0%) and blood genotype identified in FCT include: AA, AC, AS, SC and SS. Table 5 indicated that malaria parasitaemia was severe (Severe (+++) (> 10,000/ μ L)) in blood genotype AA (47.4%) than in AS(0.0%), AC(0.0%) and least in SS(0.0%) and SC(0.0%).

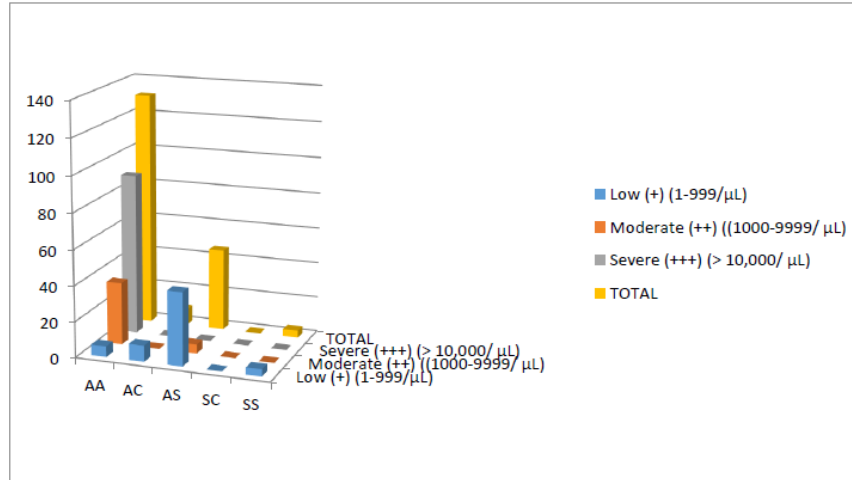


Figure 2. Bar chart distribution of malaria parasitaemia by blood genotype

Table 1. Frequency distribution of malaria parasitaemia by sex

Gender	Frequency	Percent	Cum. Percent	Exact 95% LCL	Exact 95% UCL
Male	77	41.40 %	41.40 %	34.24 %	48.84 %
Female	110	58.80 %	100.00 %	51.16 %	65.76 %
TOTAL	187	100.00 %	100.00 %		

**Note: N=384 (Total Volunteers Tested); n=187 (Volunteers with Malaria Parasitaemia).

Table 2. Frequency distribution of malaria parasitaemia by age in years.

Age (Years)	Frequency	Percent	Cum. Percent	Exact 95% LCL	Exact 95% UCL
0-10	78	41.97 %	41.97 %	34.92 %	49.27 %
11-20	66	35.23 %	77.20 %	28.51 %	42.42 %
21-30	27	14.51 %	91.71 %	9.86 %	20.28 %
31-40	11	5.70 %	97.41 %	2.88 %	9.97 %
41-50	3	1.55 %	98.96 %	0.32 %	4.48 %
>50	2	1.04 %	100.00 %	0.13 %	3.69 %
TOTAL	187	100.00 %	100.00 %		

**Note: N=384 (Total Volunteers Tested); n=187 (Volunteers with Malaria Parasitaemia).

Table 3. Frequency distribution of malaria parasitaemia by location in the fet

Location	Frequency	Percent	Cum. Percent	Exact 95% LCL	Exact 95% UCL
ABAJI	29	15.51 %	15.51 %	10.64 %	21.51 %
AMAC	4	2.14 %	17.65 %	0.59 %	5.39 %
BWARI	40	21.39 %	39.04 %	15.74 %	27.97 %
GWAGWALADA	46	24.60 %	63.64 %	18.61 %	31.41 %
KUJE	35	18.72 %	82.35 %	13.40 %	25.06 %
KWALI	33	17.65 %	100.00 %	12.47 %	23.88 %
TOTAL	187	100.00 %	100.00 %		

**Note: N=384 (Total Volunteers Tested); n=187 (Volunteers with Malaria Parasitaemia).

Table 4. Frequency distribution of malaria parasitaemia by blood genotype.

Malaria Parasitaemia	Blood Genotype					TOTAL
	AA	AC	AS	SC	SS	
Low (+) (1-999/ μ L)	6(3.2%)	9(4.7%)	41(21.6%)	0(0.0%)	4(2.1%)	59(31.6%)
Moderate (++) ((1000-9999/ μ L)	35(18.4%)	0(0.0%)	5(2.6%)	0(0.0%)	0(0.0%)	40(21.1%)
Severe (+++) (> 10,000/ μ L)	90(47.4%)	0(0.0%)	0(0.0%)	0(0.0%)	0(0.0%)	88(47.3%)
TOTAL	131(69%)	9(4.7%)	46(24.2%)	0(0.0%)	4(2.1%)	187(100.0%)

**Note: N=384 (Total Volunteers Tested); n=187 (Volunteers with Malaria Parasiteamia).

Table 5. Frequency distribution of malaria parasitaemia severity by blood genotype

Haemoglobin Genotypes	Malaria Parasitaemia (%)		
	Low (+) (1-999/ μ L)	Moderate (++) ((1000-9999/ μ L)	Severe (+++) (> 10,000/ μ L)
AA	3.2	18.4	47.4
AC	4.7	0.0	0.0
AS	21.6	2.6	0.0
SC	0.0	0.0	0.0
SS	2.1	0.0	0.0

**Note: N=384 (Total Volunteers Tested); n=187 (Volunteers with Malaria Parasiteamia).

Discussion

The aim of this research survey include: To determine in study group of FCT residents; (1) Malaria parasitaemia., (2) Haemoglobin genotype distribution, (3) relationship between haemoglobin genotype and malaria parasitaemia and (4) the severity of malaria parasitaemia amongst the various haemoglobin genotypes.

Malaria parasitaemia in FCT

In general, the result of this research work portrayed existence of malaria parasitaemia in FCT. From Table 4.3 above, malaria parasitaemia were more in Gwagwalada (24.6%), followed by Bwari (21.4%), Kuje(18.7%), Kwali (17.7%), Abaji(15.5%) and was least in AMAC(2.1%) which agreed with the work of Nasir (2015), who reported that the FCT is one of the endemic area for malaria with a high prevalence *malaria parasitemia* rate of 82.0% among tested subjects and 26.0% among control subjects and highest *malaria* density of >10,000. According to WHO (2012) and UNICEF (2000), malaria proliferates in conditions where awareness is low with weak health care systems and humid bushy areas supporting the result of this research work that found only 2.1% parasitaemia in AMAC, an urban area in FCT and higher parasitamia in Gwagwalada (24.6%), followed by Bwari (21.4%), Kuje(18.7%), Kwali (17.7%) and Abaji(15.5%) comprising mostly surban areas within FCT.

Again Ogbu (2015), prevalence of *malaria parasitaemia* among asymptomatic women at booking visit in a tertiary hospital in FCT North Central Nigeria was 38.8% which disagree with the result of this research work which found that malaria parasitaemia was more in female (58.6%) and male (41.4%) (Table 4.1 above).

From table 4.2 above showing the frequency distribution of malaria parasitaemia by age in years of volunteer subjects used in this study, malaria parasitaemia was more in the youngest 0-10 (42.0%), 11-20 (35.2%) and 21-30(14.5%). Therefore, malaria parasitaemia was 91.7% within young age range of 0-30 years of the studied group and agreed with WHO (2013) that reported that malaria remain a common and life-threatening disease in tropical and subtropical areas affecting mostly the young of over 100 countries

and territories, and these countries/territories are visited by more than 125 million international travellers every year.

Haemoglobin genotypes in fct

The work of Nwanedo *et al* (2012), reported that the gene for HbS is distributed widely throughout sub-Saharan Africa and countries with African immigrants, where carrier frequencies range from 5%–40% or more which disagreed with the findings of this research work which indicated AA(69%), AS(24.2%), AC(4.7%) and least in SS(2.1%) and SC(0.1%) as the percentage blood genotype identified in FCT (Bar Chart 4.1 and Table 4.4 above).

It could be adduced that the HbS and SS genes are endemic in FCT and again agreed with the work of Allison (2002) who hypothesized that the sickle cell gene arose spontaneously in malarious zones.

Relationship between haemoglobin genotype and malaria parasitaemia in fct

Nasir (2015), reported that the FCT is one of the endemic area for malaria with a high prevalence *malaria parasitemia* rate of 82.0% among tested subjects and highest *malaria* density of >10,000 and this research work in Bar Chart 4.1 and Table 4.4 , indicated that malaria parasitaemia was highest in blood genotype AA(69%), AS(24.2%), AC(4.7%) and least in SS(2.1%) and SC(0.0%) both supporting Modiano *et al* (2001) who emphasized that the high frequency of the sickle-cell haemoglobin (HbS) gene in malaria endemic regions is believed to be due to a heterozygote (HbAS) advantage against fatal malaria. This implied that HbS gene could shield human from malaria parasitaemia indicating that there exist strong relationship between haemoglobin genotypes and malaria parasitaemia as it has been suggested that HbAS would provide a protective advantage early in life before the acquisition of clinical immunity to malaria though definitive data to support this assumption especially in high malaria transmission areas, are lacking (Modiano *et al.*,2001 and Awah, Nwanedo, *et al*, 2012).

Severity of malaria parasitaemia among various haemoglobin genotypes in FCT

From table 4.5 above indicated that malaria parasitaemia was severe ((+++) (> 10,000/ μ L)) in blood genotype AA(47.4%) than in AS(0.0%), AC(0.0%) and least in SS(0.0%) and SC(0.0%) which agreed with the work of Michael Aidoo *et al* (2002), who also indicated that AS and AC *genotype* is associated with lower incidence of clinical *malaria* relative to AA *genotype* among the tested subjects and Esan (2015) who reported that *haemoglobin genotype* variants could reduce parasite replication within the red blood cells and enhance splenic clearance in parasitized erythrocytes. In addition, (Nwanedo, *et al* (2012) equally reported that HbAS is widely known to confer significant protection from severe and uncomplicated malaria, although underlying mechanisms for this phenomenon was not precisely defined and Kreuzberg (2010), reported that similar protection afforded by haemoglobin C (HbC) was more recently demonstrated; all supporting the result of this research.

Conclusion

Malaria had been shown as a complex disease that depends on many host genetic factors including haemoglobin genotypes.

The AC, AS and SC haemoglobin genotypes were associated with lower risk of malaria parasitaemia relative to normal genotype, AA amongst the studied volunteers.

These malaria, the haemoglobin C and haemoglobin S genes were endemic in FCT and that variant Haemoglobin C and S could confer protection to malaria parasitaemia.

Hence, evaluations of anti-malarial interventions in endemic regions like the FCT should consider Haemoglobin genotypes and compliance with national malaria guidelines as a potentially important.

Reference

- [1]. Aidoo, M., Terlouw, D.J., Kolezak M.S., McElroy P. D and TerKuile, F.O., (2002). Protective Effects of the Sickle Cell Gene against Malaria Morbidity and Mortality. *Lancet*. **359**,1311-1312.
- [2]. Allison, A.C (2002). The discovery of Resistance to Malaria by Sickle Cell Heterozygotes. *Biochemistry and Molicular Biology Education*, **30**, 279-287.
- [3]. Alouch AI. Sickle cell disease and Malaria parasitaemia in a tropical setting. *Journal*
- [4]. *Biological Chemistry*. 1997. **12**, 23-30.
- [5]. Awah, F M; Nwanedo, G.; Olalekan, S I; Augusta EhijieAzeke, A E; and Mbaik, N (June, 2012): A possible protective role of glucose-6-phosphate dehydrogenase deficiency and sickle haemoglobin genes against severe malaria in Madonna University, Elele Community. *Journal Medicinal and Medical Sciences* **3**(6), 375-381.
- [6]. Awah F.M. and Uzoegwu, P.N (2006). Influence of sickle heterozygous status and glucose-6-phosphate dehydrogenase deficiency on the clinicohaematolgoical profile of Plasmodium falciparum-infected children. *Biokemistri*. **18**(2), 89-97.
- [7]. Bell DR, Jorgensen P, Christophel EM, Palmer KL (2005). Malaria risk: estimation of the malaria burden. *Nature* 437: E3-E4.
- [8]. Bhandari PL, Raghuvver CV, Rajeev A, Bhandari PD (2008). Comparative study of peripheral blood smear, quantitative buffy coat and modified centrifuged blood smear in malaria diagnosis. *Indian Journal of Pathology*. 51: 108-112.
- [9]. Burn GW (1976). The Science of Genetics. 3rd Edition, Collier Macmillan Publications, London.
- [10]. CDC (2008). Malaria Facts. [Accessed September 10, 2018]. Available at: <http://www.cdc.gov/malaria/facts.htm>
- [11]. CDC. (2003) Local Transmission of Plasmodium vivax Malaria --- Palm Beach County, Florida, 2003. *MMWR* 52:908-911.
- [12]. Cheesbrough M (2000). District laboratory Practice in Tropical Countries Part 2. University Press, Cambridge: 271-340.
- [13]. Clark, I. A; Alleva, L. M.; Mills, A. C. and Cowden, W. B. (July, 2004): Pathogenesis of Malaria and Clinically Similar Conditions. *Clinical Microbiology Reviews*, 509–539.
- [14]. Diana Garza and Kathleen Becan-McBride (1993): Phlebotomy Handbook., 3rd edition. 103-148. Appleton & Lange, Norwalk, Connecticut.
- [15]. Endeshaw T, Gebre T, Ngondi J, Graves PM, Shargie EB, Ejigsemahu Y, Ayele B, Yohannes G, Teferi T, Messele A, Zerihun M, Genet A, Mosher AW, Emerson PM, Richards FO (2008). Evaluation of light microscopy and rapid diagnostic test for the detection of malaria under operational field conditions: a household survey in Ethiopia. *Malaria Journal*. 7: 118.
- [16]. Evans D.I.K (1971). Haemoglobin Electrophoresis on Cellulose Acetate Using Whole Blood Samples. *Journal Clinical Pathology* **2**,: 877–878.
- [17]. Harvey SA, Jennings L, Chinyama M, Masaninga F, Mulholland K, Bell DR (2008). Improving community health worker use of malaria rapid diagnostic tests in Zambia: package instructions, job aid and job aid-plus-training. *Malaria Journal*. 7: 160.
- [18]. Holland CA and Kiechle FL (2005). Point-of-care molecular diagnostic systems-past, present and future. *Current Opinion in Microbiology*. 8: 504-509.
- [19]. Lee SW, Jeon K, Jeon BR, Park I (2008). Rapid diagnosis of vivax malaria by the SD Biotline Malaria Antigen test when thrombocytopenia is present. *Journal of Clinical Microbiology*. 46: 939-942.
- [20]. Looareesuwan S (1999). Malaria. In: Looareesuwan S, Wilairatana P eds, Clinical Tropical Medicine. 1st ed. Bangkok, Thailand Medical Media. p 5-10.
- [21]. Ilozumba PC, Uzozie CR. 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*. 2009. **8**(2),1-8.
- [22]. John VD, Lewis M (1986). Practical Haematology 6th edition. Longman Singapore Publishers Pte Ltd., Churchill Livingstone.179-200.

- [23]. Koch AA, Olney RS, Yang Q (2000). Sickle Hemoglobin Allele and Sickle Cell Disease. *American Journal of Epidemiology* 9: 839-845.
- [24]. McMorrow ML, Masanja MI, Abdulla SM, Kahigwa E, Kachur SP. Challenges in routine implementation and quality control of rapid diagnostic tests for malaria-Rufiji District, Tanzania. *American Journal of Tropical Medicine and Hygiene* 2008; 79: 385-390.
- [25]. Modiano D, Luoni G, Sirima BS, Langrancotti A, Petrarca V, Cruciani F, Simpoire J, Ciminelli BM, Foglietta E, Grisanti P, Bianco I, Modiano G, Coluzzi M (2001). The Lower Susceptibility of Plasmodium falciparum Malaria to Fulani of Burkina Faso (West Africa) is Associated with Low Frequencies of Classic Malaria-Resistance Genes. *Transactions of the Royal Society of Tropical Medicine and Hygiene*. 95,149–152.
- [26]. Mwangi TW, Mohammed M, Dayo H, Snow RW, Marsh K. Clinical algorithms for malaria diagnosis lack utility among people of different age groups. *Tropical Medicine of Internal Health* 2005; 10: 530-536.
- [27]. Ngasala B, Mubi M, Warsame M, Petzold MG, Masele AY, Gustafsson LL, Tomson G, Premji Z, Bjorkman A(2008). Impact of training in clinical and microscopy diagnosis of childhood malaria on antimalarial drug prescription and health outcome at primary health care level in Tanzania: a randomized controlled trial. *Malaria Journal* 7: 199.
- [28]. NPC, (2006). The Nigerian Population Census Data, National Population Commission.
- [29]. Okoduwa, S. I. R. (Nov. 2013). Blood Group and Genotype Compatibility. *Infohealth Awareness Article*. 1 (2), 84-87.
- [30]. Ogbu G.I. ; Aimakhu, CO; Anzaku, SA; Ngwan, S and Ogbu, DA(2015). Prevalence of malaria parasitaemia among asymptomatic women at booking visit in a tertiary hospital, North Central Nigeria. *Journal of Reproductive Biology and Health* Vol 3
- [31]. Otajevwo, F D. (June, 2012): An Investigation Into Heterozygous Haemoglobin Genotype Association With Malaria Parasitaemia In A Community School Based In Benin City, Nigeria. *Global Research Journal of Medical Sciences*.2 (2).023 – 03.
- [32]. Ratsimbaoa A, Fanazava L, Radrianjafy R, Ramilijaona J, Rafanomezantsoa H, Mecnard D (2008). Evaluation of two new immunochromatographic assays for diagnosis of malaria. *American Journal of Tropical Medicine and Hygiene*. 79: 670-672.
- [33]. Reyburn H, Mbakilwa H, Mwangi R, Mwerinde O, Olomi R, Drakeley C, Whitty CJ (2007). Rapid diagnostic tests compared with malaria microscopy for guiding outpatient treatment of febrile illness in Tanzania: randomised trial. *British Medical Journal*. 334: 403.
- [34]. Reyburn H, Mbatia R, Drakeley C, Carneiro I, Mwakasungula E, Mwerinde O, Saganda K, Shao J, Kitua A, Olomi R, Greenwood BM, Whitty CJ. Overdiagnosis of malaria in patients with severe febrile illness in Tanzania: a prospective study. *BMJ* 2004; 329: 1212.
- [35]. Tangpukdee, N.; Duangdee,C.; Wilairatana, P. and Krudsood, S.(June, 2009). Malaria Diagnosis: An Overview. *Korean Journal of Parasitology*. Vol. 47, No. 2: 93-102.
- [36]. Tagbor H, Bruce J, Browne E, Greenwood B, Chandramohan D (2008). Performance of the OptiMAL dipstick in the diagnosis of malaria infection in pregnancy. *Therapy in Clinical Risk Management*. 4: 631-636.
- [37]. Tidi S.K, Amos J.T, Firyanda E (2013). Association between Plasmodium infection, Haemoglobin genotypes and Blood groups among Under-five nomadic Fulani of Northea eastern Nigeria.*International Journal of Malaria Research and Reviews*.1(2): 7-11.
- [38]. UNICEF (2000). Promoting Rational Use of Drugs and Correct Case Management in Basic Health Services, UNICEF’s Programme Division in cooperation with the World Health Organization.
- [39]. World Health Organization (2013). World Malaria Report 2013. World Health Organization, Geneva.
- [40]. WHO (2012). Management of severe malaria: a practical handbook, third edition. Geneva.
- [41]. WHO (2010). Guidelines for the treatment of malaria, second edition. Geneva.
- [42]. WHO (2006). Malaria vector control and personal protection: report of a WHO Study Group. Geneva, World Health Organization, 2006 (WHO Technical Report Series, No. 936).
- [43]. WHO (2000). WHO expert committee on MALARIA 20th REPORT. Who Technical Report Series 892. Geneva.

[44]. Zerpa N, Pabon R, Wide A, Gavidia M, Medina M, Caceres JL, CapaldoJ, Baker M, Noya O (2008). Evaluation of the OptiMAL test for diagnosisof malaria in Venezuela. *Investigations in Clinical Chemistry*. 49: 93-101.