

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/358445048>

The prevalence of malaria in children between the ages 2–15 visiting Gwarinpa General Hospital life-camp, Abuja, Nigeria

Article · February 2022

CITATIONS

63

READS

749

2 authors, including:



Alexander Philip

Bingham University

18 PUBLICATIONS 66 CITATIONS

SEE PROFILE

The Prevalence of Malaria in Children between the Ages 2-15 Visiting Gwarinpa General Hospital Life-Camp, Abuja, Nigeria

Nmadu P. M., Peter E., Alexander P. *, Koggie A. Z., Maikenti J. I.

Department of Biological Sciences, Bingham University karu, Nasarawa State, Nigeria

Abstract Malaria is a major cause of illness and death especially among children under 5 years and pregnant women. It is estimated that more than one million children living in Africa especially in remote areas with poor access to health services die annually from direct and indirect effects of malaria. Fatally affected children often die within less than 72hrs after developing the symptoms. In Nigeria, malaria consistently ranks among the five most common causes of death in children. As a result of increased mortality and morbidity there is need for proper understanding of the epidemiology of the disease among the most at risk groups. Two milliliters venous blood was collected from each of the 200 children and stored in an anticoagulant specimen bottle. Thick and thin films were prepared, stained and examined for malaria parasite under the microscope using the oil immersion objective. Malaria infection was found to be most prevalent among 2-5years old, (29%) while ages 6-10 and 11-15yrs both had 17.5% infection. There was no significant difference in prevalence among the male and female children, with 67 and 61%, respectively. The most prominent specie in the community is *Plasmodium falciparum* (62.5). There is need for mothers to protect their children from mosquito bite by ensuring that they sleep under Insecticide Treated Net.

Keywords Epidemiology, *Plasmodium, Falciparum*, Gwarinpa, Hypoendemic, Karimo, Idu, Gwagwa

1. Introduction

Malaria is a major public health problem and cause of suffering and premature death in tropical and subtropical countries (Cheesbrough, 2003). This preventable disease has reached epidemic proportions in many regions of the world and continues to spread unchecked (WHO, 2010). African children under five years and pregnant women are most at risk of malaria. Fatally afflicted children often die less than 72 hours after developing symptoms. In those children who survive, malaria drains vital nutrients from them impairing their physical and intellectual development (WHO, 2000).

It is estimated that more than one million children living in Africa die yearly from direct and indirect effects of malaria infection (Fawole & Onadeko, 2001). Malaria is a parasitic disease caused by single celled protozoan parasites of the genus *Plasmodium* belonging to the apicomplexan phylum (Krief *et al.*, 2009). Malaria parasites, (*Plasmodium* species) are spread from one person to another through the bites of haematogenous female adults of mosquitoes belonging to the insect genus *Anopheles*. These adult female *Anopheles*

mosquitoes are, hence said to be carriers of malaria parasites. These mosquitoes primarily inhabit the tropical and subtropical parts of the world (MMWR, 1999 and Epi *et al.*, 2008).

The four known species of *Plasmodium* genus that cause human malaria are *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale* and *Plasmodium malariae* and they contribute to majority of human health problem in malaria endemic regions of the world (Mohan *et al.*, 2007).

The major vectors of human malaria are *Anopheles gambiae*, *Anopheles funestus*, *Anopheles arabiensis* and *Anopheles melas*. *A.arabiensis* is most dominant in the savannah areas and cities. *A. gambiae* are found in highly dense forest areas, *A. funestus* has an uneven distribution while *A. melas* is a salt water species (Federal Ministry of Health, 1990). *Anopheles* mosquitoes can adapt to urban breeding sites over time e.g., in India, *Anopheles Stephensi* has developed into urban species and is found in much higher numbers in many cities in India than in the surrounding country side (WHO,2000). There is evidence that *Anopheles* mosquitoes are likewise becoming better adapted to the breeding site of Accra (Benneh *et al.*, 1993).

Transmission of malaria is intense and stable in Nigeria because the intensity of attack remains constant throughout the year or from year to year. In Nigeria, malaria is holoendemic in the rural areas and mesoendemic in the urban

* Corresponding author:

alexanderphlp@yahoo.com (Alexander P.)

Published online at <http://journal.sapub.org/health>

Copyright © 2015 Scientific & Academic Publishing. All Rights Reserved

areas. In the southern part of the country the transmission rate is approximately uniform throughout the year. In the far North there is a marked difference between the high transmission rate in the short wet season and low transmission rate in the long dry season (Lucas & Gilles, 1998).

Man and Malaria seem to have evolved together. It is believed that most, if not all, of today's populations of human malaria may have had their origin in West Africa (*P. falciparum*) and Central Africa (*P. vivax*) on the basis of the presence of homozygous alleles for hemoglobin C and RBC Duffy negativity that confer protection against *P. falciparum* and *P. vivax* respectively (Peter *et al.*, 2009). Recent molecular studies have found evidence that human malaria parasites probably jumped onto humans from the great apes, probably through the bites of vector mosquitoes (Krief *et al.*, 2010).

WHO (1951), reported that the degree of endemicity of malaria is measured based on the spleen rate in children aged 2-9 years in their order of severity. Hypoendemic malaria occurs when spleen rate in children is less than 10%. Mesoendemic malaria occurs when spleen rate in children is 11- 50%. Hyperendemic malaria occurs when spleen rate is 75% in children and > 25% in adults. Holoendemic malaria occurs when spleen rate is >75% in children but very low in adults. WHO (1998a) reported a prevalence of 58% malaria parasite among children in Banjul the capital of Gambia. Umar (2006) reported 94% prevalence of malaria parasite among children in Gombe metropolis. The prevalence of infection recorded for wet season in Udi Enugu State, was 59.8% according to Eneanya, 1998.

Mbanugo and Ejims (2000) in a study conducted in three hospitals and a Nursery School in Awka on prevalence of *Plasmodium* infections in children, discovered that out of 400 children, 233(58%) were positive and only *Plasmodium falciparum* were found. Matur *et al.* (2001), in a prevalence study reported 61% recorded in Abuja. Krogstad (1996), reported that *Plasmodium* infection was more prevalent in young children because of their relatively less developed immune system.

Malaria infections represent substantial social costs due to school absenteeism and reduced economic productivity. Malaria costs Africa up to US \$12billion annually. A poor family living in malaria affected area may spend up to 25% or more of its annual income on prevention and treatment of malaria. (WHO, 2000).

The *Plasmodium* species responsible for malaria infections in Nigeria are *Plasmodium falciparum*, *Plasmodium malariae* and *Plasmodium ovale*. Over 80% of malaria infections are caused by *P. falciparum* while up to 15% are caused by *P. malariae* and less than 5% are caused by *P. ovale* infections. Mixed infections with *P. falciparum* are common (Federal Ministry of Health 1990, Orajaka, 1996).

Although *P. vivax* and *P. malariae* had achieved the widest global distribution, today *P. malariae* has lost its

predominance and *P. vivax* and *P. falciparum* are the most commonly encountered malaria parasites. Almost 85% of the nearly 500 million annual malaria cases occur in sub-Saharan Africa and about 85% of cases in Africa are caused by *P. falciparum* with the remaining cases being caused by the other three strains. *P. vivax* is now the most geographically widespread of the human malarias, estimated to account for 100-300 million clinical cases across much of Asia, Central and South America, the Middle East, where 70-90% of the malaria burden is of this species and the rest due to *P. falciparum* (Lock *et al.*, 1997).

2. Materials and Methods

2.1. Sample Size

The study was conducted on a sample size of two hundred (200) children, within the age range of 2 – 15 attending Gwarinpa general hospital, Abuja Nigeria. The hospital serves people from Gwarinpa Estate, Jabi, Life-Camp and some suburbs of the city such as Karimo, Gwagwa and Idu industrial area.

2.2. Sample Collection

The blood samples were collected into EDTA bottles/containers which were labelled with information such as Name of the Patient, Investigation, Date, Sex, Age, Laboratory and Hospital Numbers and screened for the presence of malaria parasites within the months of June and July 2014.

2.3. Staining Technique

The laboratory method employed for staining and identification of malaria parasites in collected blood samples was as described by (Cheesbrough, 2003).

2.4. Smear Preparation

During this research, both thick and thin films smear were prepared. The thin film slide was flooded with Leishman stain for few minutes, two drops of buffered distil water of pH 6.8 was added and left for further 10 minutes, the slide was washed thoroughly under tap water to differentiate (the colour should be salmon pink), the slide was left to dry and the back of the slide were cleaned with cotton wool. The thick film were flooded with Giemsa stain and allowed to stand for 30minutes. The slides were then washed using clean water and the back of the slides were wiped with cotton wool and placed in a draining rack to air dry.

2.5. Microscopic Examination

The stained slides were examined for malaria parasites. Immersion oil was spread to cover about 10mm in diameter in the areas of the film. Both the thick and thin smears prepared were examined microscopically under oil immersion with the (x100) objective.

3. Results

Table 1 shows the distribution of malaria in relation to age and sex. In ages 2-5, 56males and 36 females were tested of which 33 and 25 were infected respectively. These represents 16.5% and 12.5% respectively. While in ages 6-10, 32males and 21females were examined out of which 17were positive for males and 18 for females representing 8.5% and 9% respectively. In the same vein, within the ages of 11-15, 23males and 32 females were tested out of which 18 and 17 were positive respectively representing 9% and 8.5%. In all, a total of 111males and 89 females were tested. Among these 68 (34%) male were infected and 60(30%) for females.

Table 1. Distribution of Malaria in Relation to age and sex

Age (year)	Number examined		Number infected		Prevalence (%)	
	Males	Females	Males	Females	Males	Females
2-5	56	36	33	25	16.5	12.5
6-10	32	21	17	18	8.5	9.0
11-15	23	32	18	17	9.0	8.5
Total	111	89	68	60	34	30

Overall distribution of Plasmodium parasites by sex and age which were calculated by chi-square test, concluded that there were significant differences between infection by sex and age since X² cal value in male is greater than X² tab value at P <0.05 and degree of freedom 2 respectively as shown below.

FOR INFECTED MALE

OBSERVED (O)	EXPECTED (E)	(O-E)	(O-E) ²
33	22.67	10.33	106.71
17	22.67	-5.67	32.15
18	22.67	-4.67	21.81
			160.67

$$X^2 = \frac{\sum(O-E)^2}{E} = \frac{160.67}{22.67} = 7.09$$

DEGREE OF FREEDOM d_f = n-1 (n=3)
3-1=2

X² tabulated @ 0.05 = 5.99

FOR INFECTED FEMALE

OBSERVED	EXPECTED	(O-E)	(O-E) ²
25	20.0	5	25
18	20.0	-2	4
17	20.0	-3	9
			38

$$X^2 = \frac{\sum(O-E)^2}{E} = \frac{38}{20} = 1.90$$

DEGREE OF FREEDOM d_f = n-1 (n=3)
3-1=2

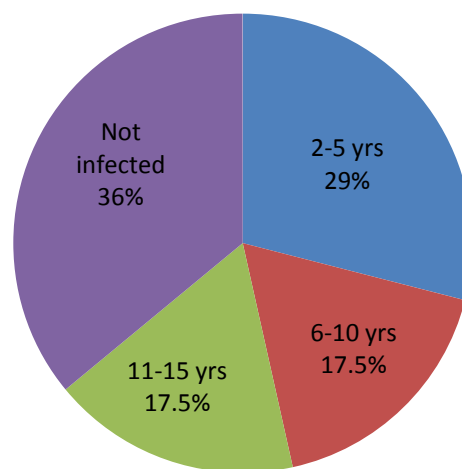
X² tabulated @ 0.05 = 5.99

- X² = chi square
- ∑ = summation
- O = observed frequency
- E = expected frequency

Table 2 shows that generally 58 children (2-5yrs) old, 34 children (6-10yrs old) and 33 children (11-15yrs) totalling 125 (62.5%) of the children sampled were infected with *P. falciparum*. Among those 6-10yrs, 1 child (2.9%) and 11-15yrs, 2 children (6.1%) had *P. malariae*. *P. vivax* and *P. ovale* however were not identified in any of the groups examined.

Table 2. Types of Plasmodium species involved in Malaria parasitaemia amongst Children 2-15 years

Variation in yrs	2-5		6-10		11-15	
	No. positive	%	No. positive	%	No. Positive	%
<i>P. Falciparum</i>	58	63	34	64	33	60
<i>P. Malariae</i>	0	0	1	2.9	2	6.1
<i>P. vivax</i>	0	0	0	0	0	0
<i>P. ovale</i>	0	0	0	0	0	0
Examined Number	58	63	35	67	35	66.1



Pie-chart showing percentage distribution of both infected and non infected children.

4. Discussion

In this study, it was observed that children between the ages of 2 - 5 years had the highest prevalence of *Plasmodium* infections (table 1) compared with the other age groups. This may be due to the fact that at that age, their immunity to parasitic infections has not been fully developed. Although it has been established that residual immunity derived from mothers could be very effective in younger children but environmental conditions and inability of children of this age in the study area to ward-off environmentally induced mosquito attacks predisposed them to malaria attack. The prevalence of *Plasmodium* infections has been found to reduce with other ages (6-10 and 11-15 years) this could be attributed to the fact that children of this age have developed immunity against *Plasmodium* parasite (Brown, 1980). The prevalence of parasitic infections among the different age

groups in the present study was not significant ($P < 0.05$) indicating that the occurrences of these infections on these age groups were the same (table 1).

The present study has shown that *Plasmodium* infections were more common in the male than in the female subjects (Table 2). The present result conforms with the recorded higher prevalence of *Plasmodium* infection in males than in females in the hospital. However, studies have shown that females have better immunity to parasitic diseases and this was attributed to genetic and hormonal factors (Krogstad, 1996).

5. Conclusions

From the result of this research, of the 200 children (2-15yrs) sample size, 128 children (64%) were infected with malaria parasite, out of which children between the age of 2-5 were observed to have the highest percentage (29%) of the infection followed by 6-10 and 11-15 years respectively as shown in Table 2, with *P. falciparum* having the highest prevalence among the causative agents as shown in Table 2. The following recommendations were therefore made:

- Public health education campaign for mothers and health care givers given to create awareness that may lead to reduction of vectors of malaria infection and control of the disease especially in young children.
- Free or subsidized Insecticide Treated Nets (ITN) should be made available to mothers so that the infection of malaria could be controlled in children.
- Mothers and other caregivers need to be empowered to treat malaria infection at home.
- Governments should train more health workers that will go into the rural areas to enlighten them about malaria (prevention and control).
- Children should be treated with antimalaria drugs every three months to prevent malaria and to kill (if any) the early stage of malaria parasite.

REFERENCES

- [1] A. Ariyasinghe, Sufi Reza M. Morshed, M. KaiissarMannoor, (2006). Protection against Malaria Due to Innate Immunity Enhanced by Low-Protein Diet. *The Journal of Parasitology*, 92 (3):531-538.
- [2] A. Kumar, NeenaValecha, Tanu Jain, Aditya P. Dash (2007). Burden of Malaria in India: Retrospective and Prospective View. *Am. J. Trop. Med. Hyg.* 77(6_Suppl):69-78.
- [3] Adeyemo A.A., Olumese P.E., Amodu O.K., Gbadesin R.A. (1999). Strengthening health infrastructure: correlates of hepatomegaly and splenomegaly among healthy school children in malaria endemic village. *Niger.J.Paediatr.*26:1-3
- [4] Brewer (2006). Suppression of adaptive immunity to heterologous antigens during Plasmodium infection through hemozoin-induced failure of dendritic cell function. *Journal of Biology*.5:5.
- [5] Brian M. Greenwood, David A. Fidock, Dennis E. Kyle, Stefan H.I. Kappe, Pedro L. Alonso, Frank H. Collins, Patrick E. Duffy (2008). Malaria: progress, perils, and prospects for eradication. *J. Clin. Invest.* 118:1266–1276.
- [6] Brown B. A. (1980). *Hematology: Principles and Procedures*. Lea & Febiger, Philadelphia. Third Edition, 75 – 87.
- [7] Carter R, Mendis KN. (2002) Evolutionary and Historical Aspects of the Burden of Malaria. *Clinical Microbiology Reviews.* 15(4):564-594.
- [8] Cheesbrough; M (1998). *District Laboratory. Practice in Tropical Countries*. Examination of blood for malaria parasites. Cambridge University press, Edinburgh, United Kingdom.239 - 242.
- [9] Cheesbrough; M (2003); *District Laboratory. Practice in Tropical Countries*. PCV and red cell indices. Cambridge University press Edinburgh, United Kingdom. 310 -313.
- [10] Claire L. Mackintosh, James G. Beeson, Kevin Marsh (2004). Clinical features and pathogenesis of severe malaria. *Trends in Parasitology*.20(12):597-603.
- [11] Coluzzi, M. (1997). Evoluzione Biologica and I Grandi Problemi della Biologia *Accademiadei Lincei*, Rome, Italy. 263 – 285.
- [12] Cox-Singh J, Singh B. (2008). Knowlesi malaria: newly emergent and of public health importance? *Trends in Parasitology*. 24(9):406-410.
- [13] Denise L. Doolan, Carlota Dobaño, J. Kevin Baird (2009). Acquired Immunity to Malaria. *Clinical Microbiology Reviews.* 22(1):13–36.
- [14] Eneanya, C.L (1996). Seasonal variation in malaria episodes among residents in Udi, a semi-urban community in southeast Nigeria. *The Nigeria journal of parasitology.* 19:39-43.
- [15] Epidi, T. T., Nwani, C. D. and Ugorji, N. P. (2008). Prevalence of malaria in blood donors in Abakaliki Metropolis, Nigeria. *Scientific Research and Essay* 3(4), 162-164.
- [16] Fawole, O.I. & Onadeko M.O. (2001). Knowledge and Management of Malaria in Under Five Children by Primary Health Care Workers in Ibadan South East Local Government Area. Nigeria. *Post Graduate Medical Journal.* 8 (1): 1-5.
- [17] Federal Ministry of Health (1990). Guidelines for Malaria control for physicians in Nigeria, Minsitry of Health, Lagos. *Federal Republic of Nigeria.* 1-45.
- [18] Federal Ministry of Health (2001). *National Malaria and Vector Control Division*. Federal Ministry of Health Abuja Nigeria.1-8.
- [19] G. Benneh, J. Songsore, J.S. Nabila, Amuzu, A.T., Tutu, K.A. and Yangyuoru, Y. (1993) Environmental Problems and the Urban Household in the Greater Accra Metropolitan Area (GAMA). *Ghana Stockholm Enviromental Institute*:44 – 45.
- [20] Kere, N.K.; J.F. Keni.; J.F. Kere, A Bobogara, R.H. Webber and B.A. Southgate(1993). The economic impact of *Plasmodium falciparum* malaria on education investment: A Pacific island case study: *Southern Asian J. Tropi Med. Public Health.* 24: 659 – 663.

- [21] Krief S *et al.*, 2010. On the Diversity of Malaria Parasites in African Apes and the Origin of *P. falciparum* from Bonobos. *PLoSPathog*;6(2):1000765.
- [22] Krongstad D.J (1996). Malaria as a re-emerging disease epidemiology. *Rev.* 18:77-89
- [23] Lock, B.and Well, S. (1977). Geographical distribution of malaria parasites. *A guide to Human Parasitology*: Crew W. (Editor) London, H.K.Lewis and co Ltd.,: 47.
- [24] Lucas, A.O. and Gilles H.M. (1998). *A New Short Textbook of Preventive Medicine for the Tropics*: Malaria, Great Britain ELBS with Edward Arnold. Publishers:188 – 192.
- [25] Mannoor MK, Weerasinghe A, Halder RC, Reza S, Morshed M, Ariyasinghe A, Watanabe H, Sekikawa H, Abo T.(2001). Resistance to malarial infection is achieved by the cooperation of NK1.1(+) and NK1.1(-) subsets of intermediate TCR cells which are constituents of innate immunity. *Cell Immunol.*1;211(2):96-104.
- [26] Mary M. Stevenson, Eleanor M. (2004). Riley. Innate immunity to malaria. *Nature Reviews Immunology.* 4:169-180.
- [27] Matur, D.M. Azare, B.A and Ugbong, L. (2001). Prevalence of malaria parasites among undergraduate students of University of Abuja. *The Nigeria Journal of Parasitology.* 22(1& 2):49-52.
- [28] Mbanugo J.I. and Ejim D.O. (2000). Plasmodium Infections in Children Aged 0-5yrs in Awka Metropolis, Anambra State, Nigeria. *Nigerian Journal of Parasitology.* 21: 55-59.
- [29] Mharakurwa S. and T. Mugochi (1994). Chloroquine-resistant falciparum malaria in an area of rising endemicity in Zimbabwe. *Journal Trop. Med. Hyg* 97: 39-45.
- [30] Mohan, D.R. and M. Ramaswamy, 2007. Evaluation of larvicidal activity of the leaf extract of a weed plant, *Ageratinaadenophora*, against two important species of mosquitoes, *Aedesaegypti* and *Culexquinqefasciatus*. *Afr. J. Biotechnol.*, 6: 631-638.
- [31] Morbidity and mortality weekly report (MMWR). (1999). Malaria. *MMWR*; 48 (12), 253-256.
- [32] National Surveillance Systems (2005). *Epidemiological Data Roll Back Malaria Monitoring and Evaluation Nigeria* W.H.O Publications Nigeria. 2.
- [33] Okafor H.U.; Nwaiwu (2001) Anaemia of Persistent malarial parasitemia in Nigerian children. *Journal Trop. Pediatr.* 47(5): 271-275.
- [34] Orajaka B.N (1996), Prevalence of Malaria Parasites and Antimalaria drugs in Use in Awka & Onitsha Anambra State. *M.Sc Thesis.:29 – 31* University Press Ibadan. 4 5 (5), *Serial No. 22, October, 2011. Pp. 264-281.*
- [35] Peter Perlmann, Marita Troye-Blomberg (2002). Malaria and the Immune System in Humans. In Perlmann P, Troye-Blomberg M (eds): Malaria Immunology. *Chem Immunol. Basel, Karger.*, vol 80, pp 229–242.
- [36] Peter Van den Eede, Hong Nguyen Van, Chantal Van Overmeir *et al.*, (2009). Human *Plasmodium knowlesi* infections in young children in central Vietnam.*Malaria Journal.* 8:249.
- [37] Putaporntip C, Hongsrimuang T, Seethamchai S *et al.*, (2009). Differential Prevalence of Plasmodium Infections and Cryptic *Plasmodium knowlesi* Malaria in Humans in Thailand. *The Journal of Infectious Diseases.*199:1143– 1150.
- [38] Ralf R. Schumann (2007). Malarial fever: Hemozoin is involved but Toll-free. *PNAS* 6.104(6):1743-1744.
- [39] Rich SM, Ayala FJ. (2006). Evolutionary Origins of Human Malaria Parasites. Krishna R. Dronamraju, Paolo Arese (Ed).Emerging Infectious Diseases of the 21st Century: Malaria - Genetic and Evolutionary Aspects. Springer US.pp.125-146.
- [40] Rich SM, Leendertz FH, Xu G *et al.*, (2009).The origin of malignant malaria. *PNAS* 106:14902-14907.
- [41] Richard Carter, Kamini N. Mendis (2002). Evolutionary and Historical Aspects of the Burden of Malaria. *Clinical Microbiology Reviews.* 15(4):564-594.
- [42] Roetyncck S, Baratin M, Vivier E, Ugolini S. NK cells and innate immunity to malaria. *Med Sci (Paris).* 2006 Aug-Sep; 22(8-9):739-44.
- [43] Tropical Disease Research (1987) “Malaria” UNDP/World Bank/WHO Special Programmes for Research and Training in Tropical Diseases. Geneva: 19.
- [44] Ukoli, (1990). *Introduction to Parasitology in Tropical Africa* Distinguishing features of the four species of malaria infecting man. John Wiley & Sons Text flow Ltd Ibadan, Nigeria.407-408.
- [45] Umar M.M (2006). NCE thesis on prevalence of malaria in Gombe Local Government Area.
- [46] W.H.O. (1951) Expert Committee on Malaria Fourth Session *Technical Report Series 39.* W.H.O publications Geneva 120.
- [47] WHO (1988A). *Urban Vector Pest Control Tech. Rep. Ser NO. 767* WHO. Geneva: 126.
- [48] WHO (1998a) Malaria, *WHO Information Fact Sheet No 94* Geneva, W.H.O press office Switzerland. : 1 - 6.
- [49] WHO (2000) Press Release *WHO/48 Fact Sheet* 5th July 2000. Geneva W.H.O publications: 1 – 18.