

**EVALUATION OF HAEMATOLOGICAL PROFILES OF WOMEN AND CHILDREN
INFECTED WITH MALARIAL PARASITES IN BOSSO AND PAIKORO LOCAL
GOVERNMENT AREAS OF NIGER STATE**

BY

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MTech/SLS/2017/6780

**DEPARTMENT OF MICROBIOLOGY
FEDERAL UNIVERSITY OF TECHNOLOGY
MINNA**

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**A THESIS SUBMITTED TO POSTGRADUATE SCHOOL FEDERAL UNIVERSITY OF
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ABSTRACT

Malaria continues to be a major public health challenge especially in the African region despite the availability of anti malarial drugs and the application of several preventive strategies such as the use of insecticide treated nets, indoor residual spraying, and intermittent preventive treatment in pregnancy. This study was aimed at investigating the prevalence of malaria parasite and its effect on the haematological indices among women and children attending selected government health facilities in Bosso and Paikoro Local Government Areas of Niger State, Nigeria. Blood samples were collected from 500 Patients attending selected Primary Health Care facilities in both local government areas. Examination for *Plasmodium* was carried out using rapid diagnostic test cassette for the qualitative detection of Histidine Rich Protein II (HRP-II) antigen of *Plasmodium falciparum* in human whole blood. The haematological analysis was carried out using automated standard methods. An overall prevalence of 49 % was obtained for the study area. Bossso LGA had a prevalence of 48 % while Paikoro LGA had 50 % prevalence. Of the total population, children recorded malaria prevalence of 70.7 %, pregnant women had 44.6 % while the non pregnant women recorded 33.9 % prevalence. Age, gender, educational background, occupation, haemoglobin content and blood group were all statistically significant at $p < 0.05$ indicating that these parameters could be regarded as risk factors that predispose to malarial infection. The haematological analysis revealed that there was association between anaemia and malaria as the severities were measured at $p < 0.05$. These severities ranged from mild, moderate and to severe anaemia. Genotype AA recorded more cases of malarial parasite infection than the other genotypes. A prevalence of 50.2 % was seen among the genotype AA population. The least prevalence of 16.6 % was recorded among subjects with genotype SS. Among the children, age group 10-12 years recorded the highest prevalence (75.0 %) of malaria and was followed by age group 0-4 with prevalence of 70.8 %. Age group 41-50 recorded the lowest malaria prevalence of 35.7 %. Age is seen from this study as a risk factor influencing malarial infection as P was at 0.05. To sustain the considerable progress made in the global fight to control and eliminate malaria, more efforts should be channeled into effective enforcement of the various control strategies such as the use of Insecticide Treated Nets (ITN), Intermittent Preventive Treatment in Pregnancy (IPTP), Larva Source Management (LSM) and Indoor Residual Spraying (IRS).

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ABBREVIATIONS, GLOSSARIES AND SYMBOLS

RBM:	Roll Back Malaria
MDGs:	Millennium Development Goals
GMAP:	Global Malaria Action Plan
SDGs:	Sustainable Development Goals
MP:	Malarial Parasite
ACT:	Artemisinin Combination Therapy
LLIN:	Long Lasting Insecticide Nets
IPT:	Intermittent Prevention Treatment
ITNs:	Insecticide Treated Nets
WHO:	World Health Organization
LGAs:	Local Government Areas
RDT:	Rapid Diagnostic Test
CSP:	Circum Sporozoite Protein
DC:	Dendritic Cell
EEFs:	Exo-Erythrocytic Forms
ICAM-1:	Intracellular Cell Adhesion Molecule-1
KC:	Killer Cell
TNF:	Tumor Necrotic Factor
IL:	Inter Leukine
INF:	Interferon
TTM:	Transfusion-Transmitted Malaria
HIV:	Human Immunodeficiency Virus

PfHRP2:	<i>Plasmodium falciparum</i> Histidine-Rich Protein
PCR:	Polymerase Chain Reaction
LAMP:	Loop-Mediated Isothermal Amplification
ELISA:	Enzyme-Linked Immuno-Sorbent Assay
IFA:	Immuno-Fluorescence Assay
EDTA:	Ethylene Diamine Tetraacetic Acid
SD:	Standard Diagnostic
HB:	Haemoglobin
MCV:	Mean Cell Volume
WBC:	White Blood Cells
MCH:	Mean Cell Haemoglobin
RBC:	Red Blood Cell
IRS:	Indoor Residual Spraying
IPTP:	Intermittent Preventive Treatment in Pregnancy
LSM:	Larval Source Management

CHAPTER ONE

1.0 INTRODUCTION

1.1 Background to the Study

Malaria is a life threatening parasitic disease transmitted by female *Anopheles* mosquitoes (Millicent and Gabriel, 2015). It is the most highly prevalent tropical disease with high morbidity, mortality, and with high economic and social impact (Dawaki *et al.*, 2016). The parasite attacks and destroys the red blood cell and may affect vital body organs like brain, liver and kidneys (Etusim *et al.*, 2013).

Malaria remains a major public health problem in some countries of the world, with Africa bearing the larger burden and Nigeria still bearing a high percentage in Africa in spite of interventions from international organizations over the previous years to reduce the incidence and mortality rate. These intervention programs include Roll Back Malaria (RBM), Millennium Development Goals (MDGs), Global Malaria Action Plan (GMAP), Sustainable Development Goals (SDGs) and Global Technical Strategy for Malaria 2016-2030 (Edogun *et al.*, 2017; Aregbeshola and Khan, 2017). Tremendous gains have been made in the fight against malaria. This success has been attributed to the adoption of the artemisinin combination therapy (ACT) as first line drug treatment in malaria endemic regions and also the scale - up of intervention efforts such as the use of long lasting insecticide nets (LLIN), intermittent prevention treatment (IPT) for pregnant women, vector control measures and more importantly increased funding (Aregbeshola and Khan, 2017). Despite these gains, malaria still remains a major health challenge in Nigeria with high morbidity and mortality. Malarial status as a major public health problem in Nigeria remains unchanged due to weak health system, high rate of poverty, poor investment in the health of the Nigerian population by governments at all levels and inequitable access to quality health care services (White *et al.*, 2011). The knowledge, attitude and

practices of households also contribute to the prevalence of malaria. Many households especially the vulnerable group which are children under 5 years of age, women and pregnant women still do not sleep under insecticide treated nets (ITNs) due to factors such as family size, level of education, gender of household head, place of residence, geo-political zone, knowledge that ITNs prevent malaria, and socio-economic status (Ezeama *et al.*, 2014). A lot of households seek malaria treatment in pharmacy stores and other types of places rather than public or private health facilities. This may be due to better accessibility and shorter waiting time. On the other hand, many caregivers and parents do not take their children for diagnostic testing. The disease reportedly accounts for an estimated 60% of outpatient hospital visits in Nigeria, 30% of hospitalizations, 30% of under-five mortalities, 25% of infant mortalities and 11% of maternal mortalities (Ali *et al.*, 2017). Malaria is holo-endemic in Nigeria, with a steady transmission rate throughout the year which comprises of a distinctive rainy and dry season (Millicent and Gabriel, 2015).

About 51% of malaria cases and deaths in Nigeria occur in rural villages away from effective diagnostic or treatment facilities (WHO, 2010). Early diagnosis and prompt effective treatment of malaria illness has been a cornerstone of malaria control. The reduction of morbidity and the interruption of parasite transmission by means of community-based anti-malarial treatment require an accurate, rapid and practical method of diagnosis. Over the past years, developments in rapid field diagnostic techniques based on the demonstration of parasite antigens have opened new possibilities for improved rural malaria diagnosis that is independent of centralized diagnostic services. There have been a considerable number of reports about knowledge, attitudes, and practices relating to malaria and its control from different parts of Africa. In spite of the giant strides in the control of malaria, misconceptions concerning malaria still exist and that practices for the control of malaria have been unsatisfactory. However, epidemiological patterns of malaria are widely different from one

place to another (White *et al.*, 2011). Specific data of a place collected can help in the making of a design of improved programme for strategic malaria control for a particular location. There are available effective low-cost strategies for the treatment of malaria, but any attempt to control a disease such as malaria in an area or locality should first of all be preceded by an extensive evaluation of the magnitude of the prevailing situation; a complete description of the health problems of the community comprising an account not only of the prevalence, but also of the community's view of its own problems and its use of existing health services.

In order to increase the chance of success of any malaria intervention, the planning and implementation must be based upon proper epidemiological analysis and application of interventions that address specific needs of localities and countries (Dawaki *et al.*, 2016). There is thus the need to complement the current research agenda primarily directed towards reducing morbidity and mortality, with one; that aims to identify key knowledge gaps and will define the strategies and tools that will result in further reduction in the burden of malaria. Operational research is one of the approaches for identifying gaps and areas of malaria control that need to be enhanced as well as for evaluating effectiveness of intervention for possible scale up. Operational research as defined by Ajayi *et al.* (2017) is the search for knowledge on interventions, strategies, or tools that can enhance the quality, effectiveness, or coverage of programmes in which the research is being done. Employing malaria operational research (MOR) findings in the planning of national malaria control programmes is gaining increased attention. Operational research can be very instrumental in providing answers to what changes, where, why, when and how these changes should be implemented to ensure smooth transition from control to elimination (Maduka, 2018). However, malaria operational research has continued to receive little attention and the relevance of the ones being conducted to ensure the needed progress from malaria control to elimination is not certain. However, poor attention has been

paid to exploring the priority research topics by researchers and malaria control implementers in Nigeria (Ajayi *et al.*, 2017). The challenging question faced by the world is whether we are winning the war against malaria or is malaria winning the war? The findings of this study will be useful in planning a sustainable malaria operational research agenda towards eliminating malaria in the nation.

1.2 Statement of the Research Problem

The Federal Ministry of Health in Nigeria has a targeted goal of reducing all malaria-related morbidity and mortality to zero level by the year 2020, but has so far been unable to achieve this as the target year is here with us and as such, malaria is still a serious issue of concern in public health.

Despite the fact that malaria is a preventable and curable disease and that treatment is currently cheap, it is still one of the leading causes of death in Nigeria. Several cases of maternal mortality, infant mortality, abortive pregnancy, and low birth weight have been traced to malaria. Moreso, most of the rural communities and even some of the urban areas still lack the proper knowledge of the vectors responsible for transmitting the disease. They still keep stagnant water and bushes which serve as breeding areas for the vector around their residence.

1.3 Justification for the Study

Over the years there has been intermittent rising and falling in malaria mortality. There is need therefore to periodically ascertain the status of malaria and thereby help in policy making and building of new intervention strategies that are locality based.

Prevalence studies and other community based malaria surveys are important tools for assessing the impact and effectiveness of malaria control measures and programmes at local and national levels.

This research will help in determining current malaria burden in the study areas. Apart from cases that are reported to hospitals there are no records of field surveys of malaria in the study areas.

1.4 Aim and Objectives of the Study

The aim of this study was to determine the prevalence of malaria burden and haematological profile among women and children attending selected government health facilities in Bosso and Paikoro local government areas (LGAs) of Niger State, Nigeria.

The specific objectives were to:

- i. determine the incidence of malaria infection among women and children attending government health facilities in Bosso and Paikoro LGAs of Niger State using the Rapid diagnostic kit.
- ii. determine the sensitivity of rapid diagnostic test cassette.
- iii. determine haematological profiles of *Plasmodium* infected subjects in comparison with those not infected with the parasite.
- iv. relate demographic factors to the transmission of malaria in the study area.

CHAPTER TWO

2.0

REVIEW OF LITERATURE

2.1 Overview of Malaria Parasite

Malaria is a parasitic life threatening disease, resulting in the destruction of the red blood cells (Okechukwu *et al.*, 2017). The term malaria originated from malaria which means bad air in Italian. The disease condition was named so because malaria was associated with wetlands (Tuteja, 2007). The scientific understanding of malaria started at the end of the 19th century when Charles Laveran, a French army surgeon noticed that there were parasites in the blood of his patients. Malaria was discovered to be transmitted by mosquitoes by Dr. Ronald Ross, a British medical officer in India while Giovanni Battista Grassi discovered that only Anopheles Mosquitoes transmitted malaria (Tuteja, 2007).

Five different species of parasites from the genus plasmodium cause malaria. These include; *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale*, *Plasmodium malariae* and *Plasmodium knowlesi* (Eledo and Sylvester 2018). *Plasmodium falciparum* is the predominant type in many African countries and it causes the most deadly form of malaria (Kuta *et al.*, 2014; Bawa *et al.*, 2014). During the process of sucking blood to nurture their eggs, malaria parasites are transmitted to humans from the bite of infected female anopheles mosquitoes. Malaria has a very complex life cycle, it starts when an infected anopheles mosquito injects sporozoites into the blood of its host. The sporozoites finds their way into the hepatocyte and multiplies in them, this matures and develops into merozoites. Merozoites launches attacks on the red blood cells and multiplication continues, due to invading of red blood cells and infection, they rupture causing the manifestation of symptoms arising from the disease. The parasite is taken up again by a mosquito that feeds on an infected host. It matures in the stomach of the mosquito and infects another host and the cycle continues (Tuteja, 2007). Humans show symptoms of infection in humans appears approximately 9 to 14 days after

infected bite. The symptoms include fever, headache, joint pains, vomiting, loss of appetite, chills and shivering (Cox, 2002; Tuteja, 2007)

2.2 Life Cycle of Malaria Parasites

Malaria is caused by protozoan parasites belonging to *Plasmodium* species. (phylum *Apicomplexa*). The life cycle is quite complex consisting of the sexual reproduction stage (sporogonic phase) in invertebrate host (mosquitoes) and an asexual reproduction stage (schizogonic phase) in vertebrate host (man) (Verra *et al.*, 2018). Transmission of the parasite to humans is through intravenous injection of sporozoites by the bite of an infected female Anopheline mosquito during blood meal. Subsequently, malaria transmission can also occur through transfusion of infected blood and also congenital transmission (Okocha *et al.*, 2005; Okonofua, 2001). Following the inoculation of sporozoites, they quickly invade hepatocytes and transform, multiply and develop into tissue schizonts. This asymptomatic, exoerythrocytic stage lasts for about 7 to 9 days but could be more depending on specie (White *et al.*, 2014). Tissue schizonts rupture thereby releasing thousands of merozoites into the bloodstream. They invade erythrocytes, and initiate the intra-erythrocytic developmental cycle (IDC). Most parasites undergo asexual development forming the ring stage trophozoites which in turn matures into erythrocytic schizonts, which rupture releasing merozoites during the intra-erythrocytic developmental cycle. In *P. falciparum* and *P. malariae* infections, no parasites remain in the liver but infection due to *P. vivax* and *P. ovale*, tissue parasites (hypnozoites) persist in the hepatocytes and can produce relapses months to years after the primary infection. Once the parasites enter the intra-erythrocytic developmental cycle, they lose potency to invade tissues, hence, malaria contracted by blood transfusion does not have a tissue stage (Verra *et al.*, 2018).

The asexual parasites digest the host haemoglobin in their acidic food vacuoles to provide in their amino acid requirements. This also is necessary to provide scope for parasite growth within the erythrocyte (Verra *et al.*, 2018).

The duration of intra-erythrocytic developmental cycle of *P. falciparum* which is the predominant specie in Africa is about 48hours. The synchronous merozoite release by blood schizonts causes the typical feverish attacks on days 1 and 3 in *falciparum* malaria. Following the release, more erythrocytes are invaded by the released merozoites and the next intra-erythrocytic developmental cycle begins.

The cycle continues until the death of the host or death of the parasites due to drug treatment or acquired partial immunity. A few erythrocytic parasites differentiate into sexual forms, named gametocytes (male; microgametocytes and female; macrogametocytes). When infected blood containing gametocytes is ingested by a female Anophelese mosquito during a blood meal, the parasites multiply in the mosquito; sporogonic cycle. While in the mosquito's stomach, the microgametes penetrate the macrogametes resulting in zygotes formation. The zygotes in turn become motile and elongated (ookinetes) which then invades the mid-gut wall of the mosquito where they develop into oocysts. The oocysts grows, rupture, and release sporozoites, which finds their way to the mosquito's salivary glands. Inoculation of the sporozoites into a new human host continues the malaria life cycle.

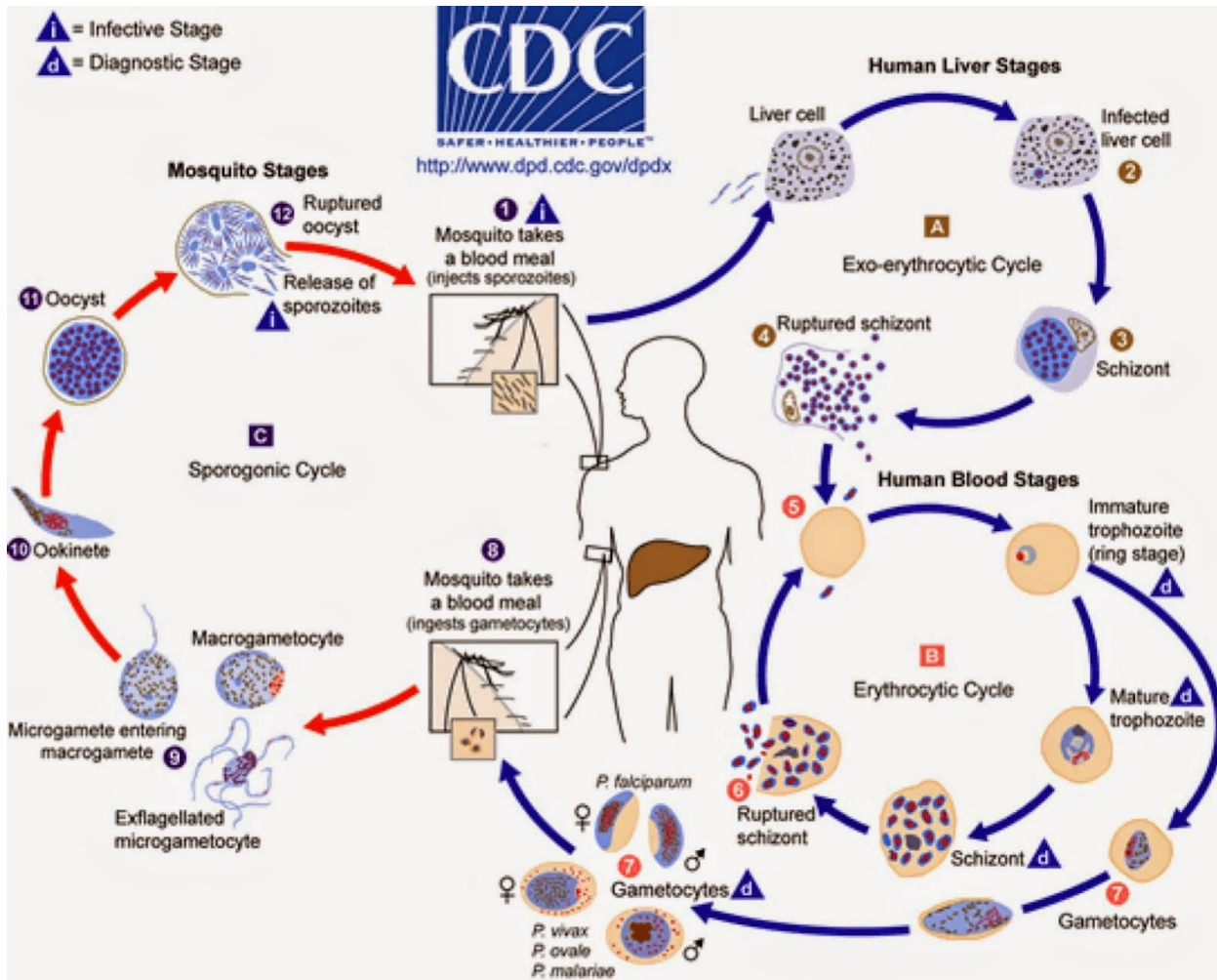


Figure 2.1: Life cycle of malaria parasite

Source: <https://www.cdc.gov/malaria/about/biology/>

2.3 Immuno-Pathogenesis of Malaria

2.3.1 The pre-erythrocytic stage

Some sporozoites, once injected into the skin presents antigen to CD11c+ dendritic cells in the proximal lymph node after draining into the lymphatic circulation, while others remain in the skin (Amino *et al.*, 2008). Although, the precise interaction of sporozoites with specialized dendritic cells

in the skin, such as Langerhans cells, and the role of innate immune signalling mechanisms (such as MyD88/Toll-like receptor signaling) remains unclear (Hafalla *et al.*, 2011).

The liver is capable of autonomously priming CD8⁺ T cell responses in the absence of infiltrating APCs (Klein and Crispe 2006). KCs, LSECs, Hepatic DCs, stellate cells and hepatocytes themselves are able to present antigen, although with limited effector outcome as the liver feature a unique tolerogenic environment (Knolle and Gerken 2003; Winau *et al.*, 2007). DCs are capable of processing circumsporozoite protein (CSP) in vitro and inducing CSP-specific CD8⁺ T cell clones that produce IFN- γ (Plebansk *et al.*, 2005).

CSP is the major sporozoite surface protein shed by the sporozoite during its motile behaviour and it has been expansively utilized to study antigen-specific cellular immune responses (Hafalla *et al.*, 2011). Upon merosome budding, the infected hepatocyte disintegrates and releases remnant parasite material which leads to the formation of small granulomas as macrophages and neutrophils perform phagocytosis (Hafalla *et al.*, 2011; Baer *et al.*, 2007). Granulomas, bundles of accumulated cells consisting primarily of macrophages, associated with severe inflammation, also form upon premature rupture of exo-erythrocytic forms (EEFs) and host cell necrosis (Hafalla *et al.*, 2011). This inflammatory response varies according to the host and the parasite specie. Merosomes avoid killer cells (KC) phagocytosis in the liver sinusoid by not expressing phosphatidyl serine on their cell membrane (Sturm *et al.*, 2006).

2.3.2 The erythrocytic stage

Onset of pathology in the affected individual occurs at the erythrocytic stage. Adults and older children develop non-sterilizing immunity against the malaria erythrocytic stage in malaria endemic countries (Crompton *et al.*, 2010). Erythrocytic rupture and subsequent release of soluble parasitic

products induces macrophages to produce IL-1 and TNF- α in non-immune individuals (Crompton *et al.*, 2010). With the rapid production of pro-inflammatory cytokines such as TNF- α and IFN- γ , there are paroxysms of malarial fever (Hafalla *et al.*, 2011). Macrophages, activated by TNF- α , secrete NO which up-regulates intracellular cell adhesion molecule-1 (ICAM-1) in leukocytes and endothelial cells (Sarah *et al.*, 2011). DCs are activated through TLR 9 and MyD88 and malarial GPI toxin activates TLR2 and haemozoin acts as a ligand for TLR9 (Hafalla *et al.*, 2011; Sarah *et al.*, 2011).

Infected erythrocytes cyto-adhere to the vascular endothelium to avoid clearance by the spleen (Crompton *et al.*, 2010). In doing so, they cause damage to organs and, via binding to ICAM-1 and other receptors, cause cerebral malaria (Aye *et al.*, 2016). This binding generates a large inflammatory cascade marked by an overriding Th1 response involving TNF- α and IFN- γ , which in turn up-regulates ICAM-1 on the endothelium and CD36 on platelets, which are primarily bound by the parasitic ligand PfEMP-1 (Sarah *et al.*, 2011; Aye *et al.*, 2016). IFN- γ plays a key role in malaria immunopathogenesis. It is first produced by NK cells and it is assumed this cell subset bridges the gap between the innate and adaptive response (Artavanis-Tsakonas and Riley, 2002; Hafalla *et al.*, 2011).

2.4 Malaria Mode of Transmission

Malaria transmission involves the transfer of the parasite from mosquitoes to man as much as from man to mosquitoes. Transmission of malaria to man are through three known ways which are vector transmission, blood transmission and congenital transmission.

2.4.1 Vector transmission

Anopheles gambiae, *Anopheles arabienses* and *Anopheles funestus* are the three most important species of Anopheles mosquitoes that are involved in malaria transmission (Hay *et al.*, 2005). The

disease is transmitted when an infected female *Anopheles* mosquito takes a blood meal with its proboscis in order to stimulate its reproduction. While probing for blood, she injects the microscopic parasite which then finds its way to the victim's liver. After which the cycle commences leading to the clinical manifestation of malaria.

2.4.2 Blood transfusion

Although blood transfusion is used in the management of emergencies involving patients with life-threatening illnesses as a life-saving venture, if not well managed, it also poses problems (Verra *et al.*, 2018). Malaria is also transmitted through transfusion of blood from an infected person to a healthy person. Transfusion-transmitted malaria (TTM) is a fortuitous *Plasmodium* infection caused by either the transfusion of whole blood or a component of blood from a malaria infected donor to a recipient, this was first described by Woolsey in 1911, that it may cause severe clinical symptoms in the recipients, particularly in those with no previous exposure to malaria or in immuno-compromised patients due to other coexisting diseases (Verra *et al.*, 2018). TTM is a serious risk, as the diagnosis of malaria in the recipient is unexpected and often missed (Kinde-Gazard *et al.*, 2000).

Plasmodium falciparum, *P. vivax* and *P. malariae* are the predominant species most frequently detected in TTM (Kitchen *et al.*, 2005). Various aspects of the parasite biology make this accidental infection possible such as the persistence of infection seen; *P. falciparum* infection can persist for at least 1 year before being cleared, *P. vivax* for 3 years whereas *P. malariae* is known to remain as a chronic infection at low density for decades (Verra *et al.*, 2018). All *Plasmodium* species are able to survive in stored blood, even if frozen, and retain their viability for at least 1 week, possibly well over 10 days depending on the conditions of storage, it has been proven also that microscopically detectable malaria parasites were present even after 28 days of storage at 4°C although a decrease of

infectivity after 2 weeks was observed (Verra *et al.*, 2018; Chattopadhyay *et al.*, 2001). An important difference between the natural infection and TTM is the absence of an initial pre-erythrocytic phase in the later. Infected blood transfusions directly release malaria parasites in the recipient's bloodstream triggering the development of high risk complications and potentially leading to a fatal outcome (Verra *et al.*, 2018; Garraud, 2006). It is notable to point out however that the mean incubation period for TTM is generally longer than the mean incubation period for the mosquito-transmitted malaria (MTM) for all Plasmodium species as reported by (Verra *et al.*, 2018).

2.4.3 Congenital transmission

Congenital malaria is the direct infection of an infant with malaria parasites from their mother prior to or during birth. It is now recognized that malaria parasites can cross the placental barriers and enter fetal circulation and give rise to congenital malaria (Olupot-Olupot *et al.*, 2018). Vertical transmission of malaria across the placenta from mother to fetus is diagnosed when parasitaemia is found in the neonate within 7 days of birth if there is no possibility of postpartum infection by mosquito bite or blood transfusion (Uneke, 2011; Menendez and Mayor 2007).

Congenital transmission of malaria results in low birth weight, still birth or death and abortion in new born babies. In endemic regions, the incidence of congenital malaria is usually extremely low or it may not result in any significant problem despite the high prevalence of placental infection. This is because sufficient antibodies would have also crossed from maternal compartment to the fetal compartment to protect the new born babies in the early phase of life (Olupot-Olupot *et al.*, 2018; Okonofua, 2001).

It has been stated by different authors (Uneke, 2011; Menendez & Mayor, 2007; Reynolds *et al.*, 2007) that the possible mechanisms in congenital transmission include direct penetration through

chorionic villi, premature separation of the placenta and the possible physiologic transfusion of maternal red blood cells to the foetal circulation in uterus or at the time of delivery. Uneke, (2011); Perrault *et al.*, (2009); Coll *et al.*, (2008) and Rogerson *et al.*, (2007) have attributed congenital transmission of malaria to the following factors; pre-existing level of malaria immunity in the pregnant woman, occurrence of severe malaria during pregnancy, absence of immunity especially pregnant women travelling to endemic areas, placental malaria especially among primigravid and secundigravid women and maternal human immunodeficiency virus (HIV) infection.

2.5 Clinical Manifestations of Malaria

The initial symptoms of malaria appear to be typical to flu like syndrome. Clinical manifestations in malaria are diverse and may range in severity from headache to more serious complications as in the case of cerebral malaria. Hence, on the basis of severity, clinical features of malaria may be classified into uncomplicated malaria and severe malaria.

2.5.1 Uncomplicated malaria

All signs and symptoms of uncomplicated malaria are non-specific and are caused by malaria parasite at the erythrocytic stage. Following the incubation period of 7 to 30 days depending on *Plasmodium* specie, the following symptoms are encountered; fever, malaise, headache, myalgias, nausea, dizziness, sense of dizziness and vomiting may be experienced (Crawley *et al.*, 2010). Fever is often high, spiking up to 40°C in children and naïve individuals, and can be associated with rigours in *P. vivax* infection (Bartoloni and Zammarchi 2012). The classic malaria paroxysm which consists of intermittent fever with chills and rigours occurring at the periodic interval of 24, 48 or 72 hours depending on the malaria species are also evident, this corresponds to the release of Plasmodium

merozoites from schizont rupture during the erythrocytic stage (Bartoloni and Zammarchi 2012; Crawley *et al.*, 2010).

2.5.2 Severe malaria

When uncomplicated malaria is not controlled either as a result of lack of treatment or partially treatment, the rapid progression to complicated or severe malaria can be fatal especially when it involves *P. falciparum*. The manifestations of severe malaria vary with both age and transmission level (Cowman *et al.*, 2016). In Africa, three dominant syndromes are associated with severe malaria which has fatal consequences especially in children, these syndromes include; cerebral malaria, severe anaemia, and respiratory distress (Tusting *et al.*, 2014). Clinical features of severe malaria may include; impaired consciousness, prostration, multiple convulsions (more than two episodes within 24 hours), Shock, Pulmonary oedema, significant bleeding, severe anaemia, Jaundice, renal impairment, acidosis, hypoglycemia and hyperglycemia (Cowman *et al.*, 2016; Tusting *et al.*, 2014).

2.6 Diagnosis

Timely and accurate diagnosis of malaria is vital in the effective management and surveillance of the disease. The diagnosis involves screening of blood for malaria parasites or antigens/products in patient blood. WHO recommends microscopy and RDT kits as diagnostic tools to be used before commencement of any anti-malarial treatment.

2.6.1 Microscopy

Microscopy technique is widely used in the management of malaria due to its simplicity, low cost, its ability to identify the presence of the parasites, the infecting species and assess parasite density. Microscopic detection and identification of Plasmodium species in Giemsa remain the gold standard

for laboratory diagnosis and remains relatively widespread as a point-of-care diagnosis in clinical and epidemiological settings (Wu *et al.*, 2015; Tangpukdee *et al.*, 2009; Bharti *et al.*, 2007). Thick and thin blood smears are prepared on a glass slide for the microscopic detection of parasites in the peripheral blood. Thick smears are useful for screening the presenting malaria parasite, parasite density and detecting of low-density malaria, whereas thin smears provide confirmation for malaria species. To prepare a thick blood smear, a blood spot is smeared in a circular motion with a spreader, and allowed to dry. After it is properly dried, without fixing the slide is then flooded with the chosen stain (Giemsa or Leshman stain) at the required proportion for the appropriate time (Tangpukdee *et al.*, 2009). Whereas, a thin blood smear is prepared by immediately placing the smooth edge of a spreader on a drop of blood, adjusting the angle between slide and spreader to 45° and then spreading the blood with a swift and steady sweep along the surface (Tangpukdee *et al.*, 2009).

2.6.2 Rapid diagnostic test (RDT)

Although the peripheral blood smear examination that provides the most comprehensive information on a single test format has been the gold standard for the diagnosis of malaria, the immunochromatographic tests for the detection of malaria antigens, developed in the past decades, have opened a new and exciting avenue in malaria diagnosis. Immuno-chromatographic tests are based on the capture of the parasite antigens from the peripheral blood using either monoclonal or polyclonal antibodies against the parasite antigen targets (Ayogu *et al.*, 2016).

Currently, immuno-chromatographic tests can target the *Plasmodium falciparum* histidine-rich protein 2 (PfHRP2), a pan-malarial Plasmodium aldolase, and the parasite specific lactate dehydrogenase (Ayogu *et al.*, 2016).

Blood sample is dropped on the sample well located on the RDT cassette, and then appropriate drops of buffer are added on the spot designated for the buffer on the cassette. The labeled antigen-antibody complex migrates up the test strip by capillary action towards test-specific reagents that have been pre-deposited during manufacture. These include (a) a line to capture antibody specific for the antigen under investigation (several lines are used if several antigens are being investigated) and (b) a procedural control line, with an antibody that will capture the labelled antibody. Visual coloured band(s) on the test strip indicate either positive or negative result. The complete test run time varies from 15-20 minutes.

Recent developments in molecular biotechnologies have permitted extensive characterization of the malaria parasite and generating new strategies for malaria diagnosis. Molecular diagnostic platforms display high sensitivity, high specificity and their ability to detect extremely low-level infections. Nevertheless, the significant barrier of these methods is in their requirement of specially trained technician, relatively high operational cost, prone to contamination, complex methodologies, and the amount of infrastructure needed in the form of equipment, stable power and reagent storage (Cordray and Richards-Kortum 2012). They are; polymerase chain reaction (PCR), loop-mediated isothermal amplification (LAMP), immuno-fluorescence (IFA), and enzyme-linked immuno-sorbent assay (ELISA) (Kitchen *et al.*, 2005).

2.7 Global Malaria Burden

Malaria burden is biased towards sub-Saharan African region. It remains one of the major public health problems and a leading cause of morbidity and mortality especially in sub-Sahara Africa. Reports from WHO region shows that 228 million cases of malaria occurred worldwide in 2018 compared with 231 million cases in 2017 and 251 million cases in 2010 (WHO, 2019). The WHO

African Region still bears the largest burden of malaria morbidity, with 213 million cases (93%) in 2018, followed by the WHO South-East Asia Region (3.4%) and the WHO Eastern Mediterranean Region (2.1%) (WHO, 2019). Almost 85% of all malaria cases globally were in 19 countries: India and 18 African countries, Nigeria inclusive in the list (WHO, 2019).

Between 2010 and 2018, the global incidence rate (the number of cases per 1000 population) of malaria dropped from 71 in 2010 to 57 in 2018 according to reports from WHO (2019).

The estimated deaths due to malaria globally declined from 585 000 to 405 000 cases between 2010-2018 with WHO Africa region having the highest absolute reduction in malaria deaths from 533 000 deaths in 2010 to 380 000 deaths in 2018.

Of all death cases due to malaria, globally, 272 000 (67%) were estimated to be in children aged less than 5 years (WHO, 2019; Aina *et al.*, 2013 and Onyesome and Onyemakonor, 2011).

Despite the impressive gains on the decline of death cases due to malaria, global malaria challenge remains enormous and the level of progress is slowing down.

2.8 Epidemiology

Over the years, several authors have carried out research findings on the prevalence of malaria, the transmission, diagnosis, symptoms, prevention and control strategies. These studies have gone great extent in trying to reduce and moderate the menace of malaria although attaining elimination status is still an issue of concern in many countries of the world particularly sub-Saharan African countries.

Mbuh *et al.* (2003) conducted a study to determine the rate of co-infection with malaria parasites and salmonella typhi in Zaria, Kaduna state, Nigeria. From a total of 218 blood samples that were collected from patients with a clinical suspicion of malaria and typhoid fever, sixty samples were

positive for malaria parasites, 22 of which were positive for typhoid by the Widal test and only one by the culture method. The rate of co-infection was significantly high when typhoid was diagnosed by Widal (10.1%) than by blood culture method (0.5%). A correlation analysis showed no specific relationship between malaria parasite load and the level of *Salmonella* antibody titres in malaria patients ($r = 0.05$ and 0.08 for somatic and flagella antigens of *S. typhi* respectively).

In a study by Mouatcho and Dean (2013), it was reported that due to the fact that parasitological confirmation of suspected malaria using microscopy, the gold standard, is cumbersome and requires trained personnel, microscopes and a source of electricity, malaria treatment based on RDTs, which are quick and easy to perform, is becoming more attractive. PfHRP2- and pLDH-based RDTs are the most commonly used. PfHRP2 RDTs appear to be more sensitive than pLDH RDTs, particularly at low parasite densities, although there are exceptions (Mouatcho and Dean, 2013). To date, both PfHRP2 and pLDH RDTs are more sensitive than aldolase-based tests. PfHRP2-based tests are less specific than pLDH-based tests, regardless of the level of parasitaemia (Abba *et al.*, 2011). A pLDH RDT should be employed when determining the efficacy of drug treatment, as PfHRP2 persists for long periods in the blood after parasites have cleared. As a result of the persistence of the PfHRP2 antigen, PfHRP2 RDTs can detect antigen when *P. falciparum* parasites are sequestered in placental tissues or elsewhere, and thus parasites are not present in peripheral blood for detection by microscopy. It was also reported from the study that the persistence of the PfHRP2 antigen after parasites decline in the blood leads to false positive results (Mouatcho and Dean, 2013). The Pfhrp2 gene undergoes antigenic variation, whilst the genes for Plasmodium pLDH and aldolase do not. The concentration of PfHRP2 has the potential to predict progression to severe malaria (Fox *et al.*, 2013), and to detect true severe malaria rather than severe non-malarial illness (Hendriksen *et al.*, 2011).

Bassey and Izah (2017) conducted a study on some determinant factors of malaria prevalence in Nigeria. From the study, it still showed that malaria is endemic in Nigeria in that over 95% of her population is at risk of falling victim of the disease. Certain factors including medical conditions, environment/season and human status; pregnancy, blood group, Rhesus factor, age, gender and educational status were outlined as the factors that upon which the prevalence of malaria is hinged. As such the predisposing factor of high prevalence of malaria within a location/locality needed to be ascertained in order to effectively manage the infection.

In another study by Millicent and Gabriel (2015) in determining the prevalence of malaria in patients attending the general hospital Makarfi, Kaduna State, North- Western Nigeria, the study classified patients into 3 groups ; under 5 years, 5 – 15 years and above 15. Of the total 1173 participants that were screened 419 (35.7%) were positive. The geometric mean parasite density was 15,108 parasite/ μ L of blood. Children aged 5 – 15 years had a statistically significant higher infection rate compared to the other groups. Furthermore, the study reported that more males were prone to malaria than females and that presence of parasitaemia, fever or anaemia did not correlate with the presence of malaria as Some of the positive patients had high parasite densities and yet asymptomatic.

In another case report study by Olupot-Olupot *et al.* (2018), neonatal and congenital malaria: a case series in malaria endemic eastern Uganda was carried out. Four cases (two congenital malaria cases and two neonatal malaria cases) were described after presenting for care at the Mbale Regional Referral Hospital Neonatal Unit (Mbale RRH-NNU). The maternal age was similar across the cases, but both neonatal malaria cases were born to primigravidae. Three cases had fever and history of fever, but one was hypothermic (34.8 °C) and no history of fever. One case of congenital malaria had low birth weight, while the other was born to an HIV positive mother. Both cases of congenital malaria presented with poor feeding, in addition one of them had clinical jaundice. All of the cases of

neonatal congenital malaria were treated with intravenous artesunate and all cases recovered and were discharged.

In another related study by Okechukwu *et al.* (2017) on methemoglobin levels among malaria parasite-infected blood donors in Nnewi, South - Eastern, Nigeria, a total of 100 apparently healthy blood donors aged 18–60 years were recruited and tested for malaria parasite, methemoglobin level, and packed cell volume (PCV). Methemoglobin was assayed by cyanmethemoglobin method, and malaria parasite was tested using thick and thin blood film and PCV by micro-haematocrit method. Among the 100 blood donors screened, 75 of the participants representing 75 % were positive for malaria parasite and 25 representing 25 % tested negative. *Plasmodium falciparum* was responsible for all cases of parasitemia. The mean values of methemoglobin were significantly higher in asymptomatic malaria parasite infected blood donors than in non-infected donors, while PCV was significantly reduced in malaria infected donors compared to non-infected donors ($P < 0.05$).

Umma *et al.* (2017) in a cross sectional study to determine the prevalence of malaria parasitaemia among febrile children in North-western Nigeria. 90 feverish children with severe PEM aged 6–59 months (44 males and 46 females) were enrolled as subjects and; 90 febrile well-nourished children age and sex matched children as controls. Both subjects and controls were enrolled consecutively in the emergency pediatrics unit of Aminu Kano Teaching Hospital. The prevalence of malaria parasitemia was 72.2 % in the subjects, which was significantly higher than 37.8 % in the controls. Age, sex, and type of PEM had no significant effect on the prevalence of malaria parasitemia and parasite density ($P > 0.05$). *Plasmodium falciparum* (*P. falciparum*) was seen in all the positive slides.

Afoakwah *et al.* (2016) carried out a study on relative susceptibilities of ABO blood groups to *Plasmodium falciparum* malaria in Ghana. It was presented from the study that the clinical outcome of *falciparum* malaria in endemic areas is influenced by erythrocyte poly-morphisms including the ABO blood groups. Association of ABO blood group to resistance, susceptibility, and severity of *P. falciparum* malaria infection was also reported. From their findings, individuals with blood group “A” were found to be highly susceptible to *falciparum* malaria whereas blood group “O” is said to confer protection against complicated cases. Out of the 293 samples analysed from young children less than six years old with malaria in the Korle Bu Teaching Hospital in Accra, Ghana, group O was present in about 16.1% of complicated cases weighed against 40.9% of uncomplicated controls. Individuals with complicated malaria were of blood groups A and B compared to group O. This study also reported that blood group O participants with complicated diseases had low parasitaemia compared to the other blood groups.

Olasehinde *et al.* (2019) reported malaria prevalence of 45.86 % from blood and corresponding saliva samples were collected from 1,243 subjects of all ages and sex presenting with fever and a parasitemia level $\geq 2,000$ in Ogun state. It was reported that male subjects had the highest incidence of 57.42%. Furthermore, point mutations of K76T and N86Y in the Pfcrt and pfmdr-1 genes, as well as non-synonymous mutations in Pfk13 genes, were screened for and sequenced. Pfcrt was detectable in 57.42 % of blood and 51.02 % of saliva samples, respectively. About 34.78% of the subjects that were microscopically confirmed to harbor the Pfmdr-1 mutated gene while 26.67% of the saliva samples revealed Pfmdr-1. Epidemiological studies identified the presence of wild-type Pfk13 genes in 21.84 % of blood and 44.44 % of saliva samples correspondingly. For each of the genes evaluated, saliva portrayed great diagnostic performance when compared with blood.

Etusim *et al.* (2013) conducted studies on the prevalence of malaria parasite among children with splenomegaly in Aba metropolis, Abia State, Nigeria, Of the 403 patients in clinical state with confirmed cases of splenomegaly tested with Care-start malaria test kit for the presence of malaria parasite, 338 (83.9 %) were infected with malaria while 65(16.1 %) were not. A significantly positive relationship between malaria incidence and splenomegaly was recorded. Moreso, age group 1 – 3 years recorded the highest infection rate 172 (89.5 %) while age group 13 – 15 years recorded the least infection rate 21(100 %). Of the 403 people found with SPR, 237 (58.8 %) were males while 166 (41.2 %) were females. The prevalence of malaria was higher 199 (84 %) in males than in female 139 (83.7 %).

In another study by Sam *et al.* (2014), from 384 blood samples screened for malaria parasites, 273 (71.1 %) were positive with malaria parasites. Species detected included *Plasmodium falciparum* (95.6 %), *Plasmodium malariae* (3.3%), *P. ovale* (0.7 %) and *P. vivax* (0.4 %). The age-group 1-11 years had the highest malaria prevalence 94 (81 %) while 51-60 had the least 6(54.5 %). The malaria prevalence in relation to age was significant ($p = 0.011$, $p < 0.05$). The males 110 (73.8 %) were slightly more affected than the females 163 (69.4 %), but not statistically significant ($p = 0.347$, $p > 0.05$). Malaria prevalence was significantly higher among those with no formal education 80 (81.6 %) and least among post-secondary educational. Prevalence among occupational groups showed significant difference, where the unemployed group were more infected and the retiree group had the least infection ($p = 0.014$, $p < 0.05$). It was also reported from the study that the singles (56.4 %) were more infected than the married (43.6 %).

In a study conducted by Kuta *et al.* (2014) to determine the prevalence of malaria among school aged children (0- 12years) in 3 primary schools in paikoro local government area of Niger State, from the two hundred and fifty (250) blood samples collected from children in 3 primary schools, one hundred

and forty four blood samples were positive representing 57.6 %. The study also highlighted that male children recorded prevalence of 37.6 %, while females had 20.0 % prevalence. Similarly, children within the age group of < 1-6 years recorded high prevalence of 48.8% compared to other age groups. Children living in rural areas recorded prevalence of 38.8% compared to those living in urban areas (18.8 %).

2.9 Prevalence of Exposure to Malarial Infection during Pregnancy

Anaemia is characterized by a decrease in the number of red blood cells in the blood or a decrease in haemoglobin (Hb) concentration to a level that impairs the normal physiological capacity of the blood to transport oxygen to cells around the body (WHO, 2019). Severity of anaemia is defined by the following Hb levels; Mild anaemia ≤ 11 g/dL Moderate anaemia ≤ 8 g/dL Severe anaemia ≤ 5 g/dL (White 2018). Deficiency in iron is thought to be the most common cause of anaemia. Maternal anaemia has several causes, mainly related to nutrition, infection and genetics (Ross *et al.*, 2006). Malaria infections cause anaemia through multiple mechanisms; direct destruction of red blood cells, clearance of infected and uninfected red cells by the spleen, and impaired red cell production by bone marrow. Individuals who are anaemic are at a greater risk of mortality, including from malaria. Single or repeated episodes of malaria may result in life-threatening anaemia, metabolic acidosis (Griffin *et al.*, 2014) and death. Exposure to malaria infection during pregnancy leads to maternal anaemia, which is associated with higher risk of obstetric haemorrhage and death. World Health Organization estimates of maternal anaemia (Hb concentration of <10.9 g/dl at sea level) by country were obtained for 38 moderate to high malaria transmission countries in sub Saharan Africa (WHO, 2019).

2.10 Malaria Prevention and Control Measures

Prevention and control of malaria is achieved through the use of the following measures; universal access to Long-Lasting Insecticide-treated nets (LLINs), indoor residual spraying (IRS), larval source Management (LSM), provision of Intermittent Preventive Treatment of malaria in pregnancy (IPTp), vector sentinel surveillance and resistance monitoring and quality assurance of commodities (Maduka, 2018).

2.10.1 Long lasting insecticide treated nets

LLINs have been lauded as the mainstay of malaria prevention, especially in sub-Saharan Africa (Albert *et al.*, 2015). As at 2015 net ownership across the Nigeria was 69 % (Albert *et al.*, 2015). This is a step increase from ownership of 2 % in 2003. Over 103.8 million LLINs were distributed in Nigeria between 2009 and 2015 with higher net ownership in rural compared to urban populations (Ajayi *et al.*, 2017). Across the regions in the country the northwest has the highest LLIN ownership while southwest has the lowest. Net utilisation is yet to come to par with net ownership. There are persistently lower rates for net utilisation compared to ownership (Maduka, 2018). However, net utilisation rates (measured as the proportion of persons who slept inside a treated net the previous night) have risen over the past decade. Among children, less than five years old, net utilisation has gone up from 1 % in 2003 to 39 % in 2015 while among pregnant women, utilisation increased from 5 % in 2003 to 43 % in 2015 (WHO, 2015).The challenges mitigating against increased net ownership and utilisation are manifold. In spite of all the social mobilisation campaigns around net ownership and use, many Nigerians still find sleeping under a treated net unacceptable. Socioeconomic and socio cultural barriers such as irregular power supply, the perception that net use is for women and children, perceptions surrounding itching, colour, odour, and heat production, among others, impede net use (Maduka, 2018).

Nigeria started piloting the school distribution channel in 2012 and the community-based distribution channels in 2013. The community channel is also being used to provide ITNs for internally displaced persons (IDPs) from the conflicts in the northeastern states (Maduka, 2018).

2.10.2 Indoor residual spraying (IRS)

Indoor residual spraying (IRS) is the practice of spraying insecticides on the interior walls of homes in malaria affected areas. After feeding, many mosquito species rest on a nearby surface while digesting the blood meal, so if the walls of dwellings have been coated with insecticides, the resting mosquitoes will be killed before they can bite another victim, and thereby transferring the malaria parasite (Vanhouwer *et al.*, 2007).

2.10.3 Intermittent preventive treatment in pregnancy

In 2014, Nigeria adopted the updated WHO IPTp policy of providing IPTp with SP, starting as early as possible in the second trimester, for all pregnant women at each scheduled antenatal care visit until the time of delivery, provided that the doses are given at least one month apart. IPTp entails giving three or more doses of sulphadoxine-pyrimethamine (SP) as directly observed treatment (DOT), one month apart from after the onset of quickening (Bawa *et al.*, 2014). The uptake of the three-dose IPTp in Nigeria is low at 19 % in 2015 (WHO, 2015). However, uptake of two-dose IPTp was 37 % (Maduka, 2018). Both figures reflect poor implementation of IPTp and poor knowledge and acceptance of three or more doses of IPTp.

2.10.4 Supplementary vector control

Supplementary vector control methods include larval source management (LSM) and personal protection. LSM has been advocated as useful for the control of breeding sites only where larval

breeding sites are few, fixed and findable (Maduka, 2018). Although LSM has been implemented in Rivers and Lagos states, it hasn't had much impact probably because of operational, technical, and logistical challenges associated with this vector control approach (Maduka, 2018).

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Study Area

This study was carried out in Bosso and Paikoro local government areas. Villages and towns that were covered in Bosso LGA included, Garatu, Begi, Bosso, Shatta and Maitumbi. Villages and towns that were covered in Paikoro local government area included, Chimbi, Paiko, Kaffin- koro, Kwakuti and Tunga Mallam. Sample analyses were carried out at the Microbiology unit, Centre for Genetic Engineering and Biotechnology, Federal University of Technology Minna and Heamatology laboratory, General Hospital, Minna, Niger State, Nigeria.

Bosso LGA covers a land mass of 1606.1 square kilometers, with Maikunkele as its headquarters. It has a population of 147,359 according to the 2006 population census. Paikoro LGA covers a land mass of 2,259.2 square kilometers, with Paiko as the local government headquarters. It has a population of 158,086 according to reports from the 2006 population census (Niger State Department of Budget & Planning, 2012).

The population of the study area is characterized with majority of children, followed by middle age adults and then the aged (Niger State Department of Budget and Planning, 2012). According to statistics provided by the Niger state department of budget and planning (2012), the study areas are characterized with more males than the female gender. Majority of the populace in the study areas (85

%) are farmers while the remaining (15 %) are involved in other vocations such as white collar jobs, business, craft and arts. The subjects of the study area are predominantly Muslims and Christians with very few Traditional worshipers and Atheists. Predominantly, three major languages are spoken in the study area, Nupe, Gbagyi, and Hausa. The settlement pattern in the study area is the semi-compact settlement which is also referred to as hamlet. Predominantly, lands are cultivated from one house to the other. This farm lands within residential areas could serve as breeding sites for the vector responsible for transmission of the disease.

Both local government areas have almost similar climate and it is that of tropical continental region which is characterized by relatively wide annual temperature range and a restricted rainfall. It is marked by two distinct seasons, namely, rainy and dry. The rainy season starts in April and lasts till October, with June to August as the months of peak rainfall. The dry season, extends from November to March and is characterized by harmattan with dust-laden cold winds swept-in by the Northeast trade wind (Ajayi *et al.*, 2014; Edogun *et al.*, 2017).

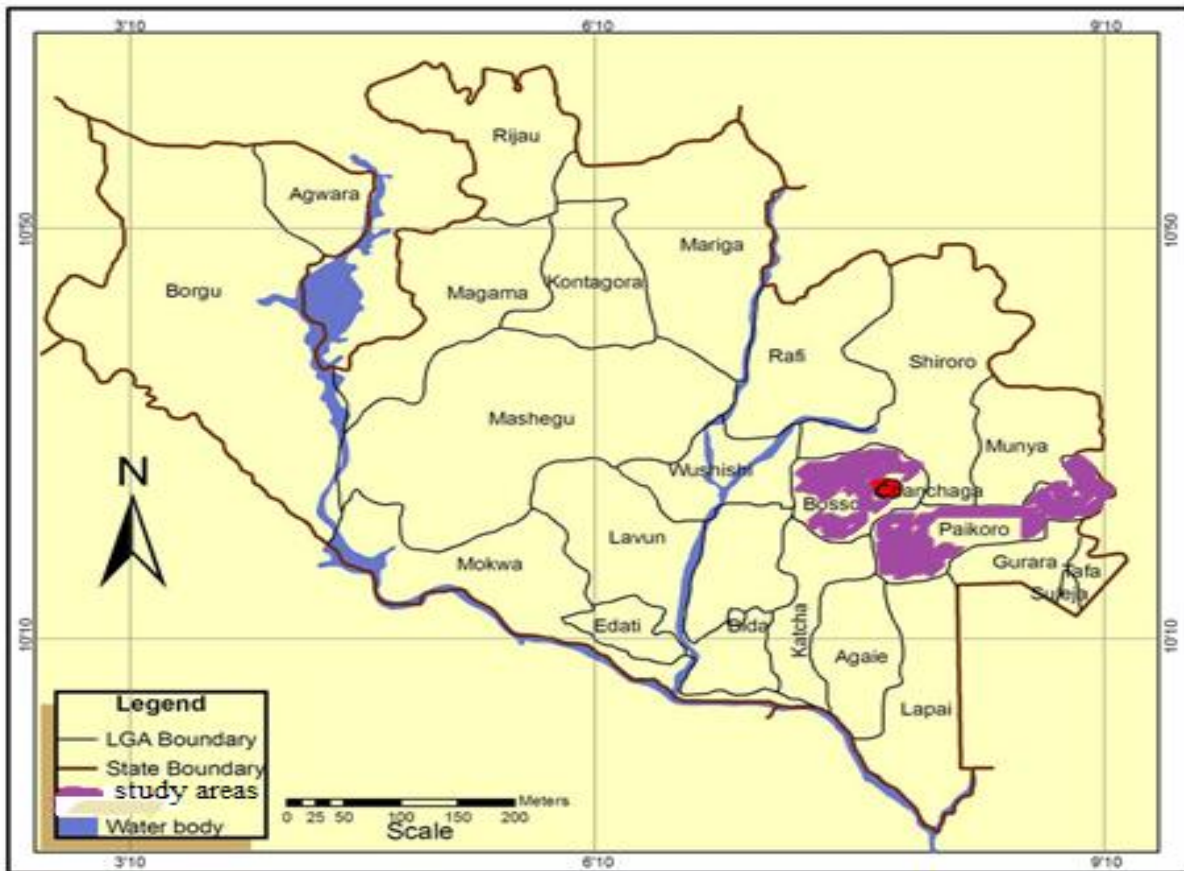


Figure 3.1: Map of Niger state showing the study areas
 Source: Ajayi *et al.* (2014).

3.2 Sample Size Determination

The sample size was determined using the formula;

$$n = \frac{N}{1 + N(e^2)} \quad (\text{Jaykaran and Tamoghna, 2013})$$

n = sample size

N = Population Size

e = Margin of error at (5 %) = 0.05 at 95 % confidence level

N= 33,711(DHIS2, 2019) Assessed from the office of Monitoring and Evaluation, Primary Health Care Department Bosso and Paikoro local government areas.

$$n = \frac{33711}{1+33711(0.05)^2}$$

n= 399.9 \approx 400 (Minimum Sample size)

n= 500

3.3 Ethical Approval

The study protocol was approved by the ethical committee, General Hospital Minna and Primary Health Care Department, Minna, Niger State. Before commencement of the study, verbal consent was sought from subjects or parent/legal guardian after details of study was explained to them in English and other languages (Hausa, Gbagyi and Nupe) for those who could not understand English language. The ethical approvals are presented in appendix V.

3.4 Sample Collection

Questionnaires were issued to randomly selected subjects who fall within the target group (women and children, adult males were excluded) after which verbal informed consent was sought for individually. Ethylene diamine tetraacetic acid (EDTA) container was labeled with subject's information. After which responses to questionnaires were collected and venous blood sample was aseptically collected from subjects attending the various government health facilities in selected communities within Bosso and Paikoro Local Government Areas using the method described by World Health Organization (2015). Venous blood sample of 2ml was aseptically collected from subjects using a disposable sterile needle

and syringe with a tourniquet through venipuncture in line with the method described by World Health Organization guidelines in the diagnosis and treatment of malaria. After which blood sample was transferred from the syringe into the labeled EDTA container to prevent clotting of the blood samples before being transported aseptically to the Center for Genetic Engineering and Biotechnology, Federal University of Technology, Minna, for analysis. Information contained in the questionnaire is presented in Appendix I.

3.5 Sample Analysis

3.5.1 Rapid diagnostic tests (RDTs)

The method used in this survey for the detection of malarial parasite was Rapid Diagnostic Test (RDT). The cassette used was SD, manufactured by Standard Diagnostics Incorporated, 65, Borahagal-ro, Giheung-do, Republic of Korea. After the overall prevalence was obtained from the study using SD RDT from the 500 samples, the sensitivity of the bioline RDT cassette was determined using about 5 % of the total sample size. The 5 % were tested first using microscopy to detect the presence of malarial parasite after which the samples were tested using SD RDT cassette to determine the sensitivity.

Thick and thin blood films as described by Cheesbrough (2009) were made from the fresh blood samples on clean grease free slides and labeled accordingly as recommended by WHO (2015).

In preparing the thick film, two drops of blood was dropped on a completely clean grease-free microscope slide using a small sterile Pasteur pipette. Without delay, the blood was then spread on the slide in a circular pattern to cover evenly an area of about 1-2 cm in diameter. The slide

was labeled accordingly with the patient's information provided at the point of sample collection. The smear was allowed to air-dry thoroughly with the slide in a horizontal position and placed in a safe place. After the smear was flooded with 3 % Giemsa staining solution for 30 minute in a staining rack, then washed with clean water. The back of the slide was then wiped clean, stained smear was air dried and then viewed under the oil immersion objective for the determination of the infective stages of malaria parasite in the erythrocytes.

For the thin film, with the use of sterile Pasteur pipette, a single drop of blood was placed at the center of a completely clean microscope slide. Using a smooth edged slide spreader the blood was immediately spread, then allowed to air-dry with the slide in a horizontal position and in a safe place. Absolute methanol (methyl alcohol) was used to fix the thin blood film, putting into considerations that the alcohol was completely free from water to properly fix the cells. The thin film was allowed to fix for 2 minutes (Cheesbrough *et al.*, 2009). The slides were labeled accordingly with the patient's information provided at the point of sample collection. The smear was allowed to air-dry thoroughly with the slide in a horizontal position and placed in a safe place. After which the smear was flooded with 3% Giemsa staining solution for 30 minute in a staining rack, then washed with clean water. The back of the slide was then wiped clean, stained smear was air dried and then viewed under the oil immersion objective (Cheesbrough *et al.*, 2009). Positive samples show either of these; trophozoite, schizont or the gametocyte stages of the parasite in the erythrocytes in the field depending on the duration of infection and disease establishment.

About 6 % (32) of the total sample was randomly selected to test sensitivity of the SD RDT used. Out of the 32 samples tested by both microscopy and RDT, 10 negative by microscopy were also seen to be negative by SD RDT cassette and the 10 samples that were positive for microscopy

also turned out positive for RDT. There by 95 % confidence level was assumed for the sensitivity of the SD RDT cassette in the detection of malarial parasite. The remaining 12 were positive by microscopy but turned out to be negative by RDT.

Although the venous blood smear examination that provides the most comprehensive information on a single test format has been the gold standard for the diagnosis of malaria, the immunochromatographic tests for the detection of malaria antigens, developed in the past decades, have opened a new and exciting avenue but not completely free of challenges of false positive and negative results in malarial diagnosis. Immuno-chromatographic tests are based on the capture of the parasite antigens from the peripheral blood using either monoclonal or polyclonal antibodies against the parasite antigen targets (Ayogu *et al.*, 2016).

Currently, immunochromatographic tests can target the *Plasmodium falciparum* histidine-rich protein 2 (PfHRP2), a pan-malarial Plasmodium aldolase, and the parasite specific lactate dehydrogenase (Ayogu *et al.*, 2016).

The test procedure was carried out appropriately following the manufactures manual on the test standard diagnostic (SD) kit. Anti-coagulated blood sample was dropped at the point for sample on the RDT cassette, and then 3 drops of buffer were added to the blood on the test cassette. The labeled antigen-antibody complex migrates up the test cassette by capillary action towards test-specific reagents that have been pre-deposited during manufacture. These include (a) a line of capture antibody specific for the antigen under investigation (several lines are used if several antigens are being investigated) and (b) a procedural control line, with an antibody that will capture the labelled antibody. After the test strip was observed for visual coloured line(s) on the test and control regions

of the test cassette as seen in figures 3 and 4. The complete test run time varies from 10 to 15 minutes.

3.6 Haematological Analysis

Haematological analysis was performed to determine the difference in haematological indices of healthy individual from that of infected subjects. Haematological analysis helps in the diagnosis of various disorders in whole blood which in-turn facilitates prescription of appropriate treatment. Haematological analysis of the blood samples of subjects was conducted using Abacus Junior automated haematological analyzer at the General Hospital, Minna, Niger State. Two ml of blood in an EDTA container was placed on a mixer, after which the sample was transferred to the analyzer and the machine switched on. About 50 microlitres of the sample was aspirated by the machine for the analysis. Results of the haematological parameters sought for were displayed on the screen. The time required for the test was about 15-20 minutes. The parameters that were sought for in the haematological analysis include, pack cell volume, haemoglobin concentration, mean cell volume, total white blood count, mean cell haemoglobin, neutrophils, eosinophils, monocytes, basophils, platelet count, red blood cell count, red cell distribution rate, blood group and genotype.

3.7 Data Analysis

Data analysis was done using Statistical Package for Social Science (SPSS) version 21, IBM for windows. Differences and proportions were tested by Chi-square (χ^2) tests and Kappa analysis was used to show the level of agreement between the use of RDT and microscopy in the diagnosis of malarial infection. Differences were considered significant at $p < 0.05$.

CHAPTER FOUR

4.0 RESULTS AND DISCUSSION

4.1 Results

4.1.1 Sensitivity pattern of rapid diagnostic tests for detecting malarial parasites

The results of sensitivity pattern of rapid diagnostic tests for detecting malarial parasites are shown in plate I - III. Out of the 32 confirmed results by microscopy, 10 that were negative by microscopy came out negative by RDT, another 10 that were positive by microscopy were also found to be positive by RDT.

The remaining 12 samples were reported as positive for malarial parasite by microscopy but turned out to be negative using the RDT. The results were not declared as false negative because blood films were prepared from those 12 samples and they appeared positive when viewed again under the microscope. The result obtained from this standardization was subjected to Kappa analysis to check for level of agreement between the use of Microscopy and RDT in the diagnosis of malaria and the level of agreement was on a fair scale of (0.363) which was not an acceptable scale for a diagnostic purpose. Although, under some circumstances, some RDT can either produce false positive and false negative results and this could be attributed to the antibody coating in the RDT. Some are coated with histidine rich protein II of *Plasmodium falciparum* (PfHRP II) which is a major protein produced by plasmodium and which is what the kit detects while others are coated with Plasmodium aldolase and others with parasite lactate dehydrogenase (pLDH). Detailed table containing the statistical analysis of the sensitivity pattern of the RDT is presented in appendix III.



Legend

Double red bands appearing on the control region and test region indicating positive result.

Plate I: SD RDT cassettes showing positive results for *P. falciparum*



Legend

Single red band appearing on the control region indicating negative result.

Plate II: SD RDT cassettes showing negative results for *P. falciparum*

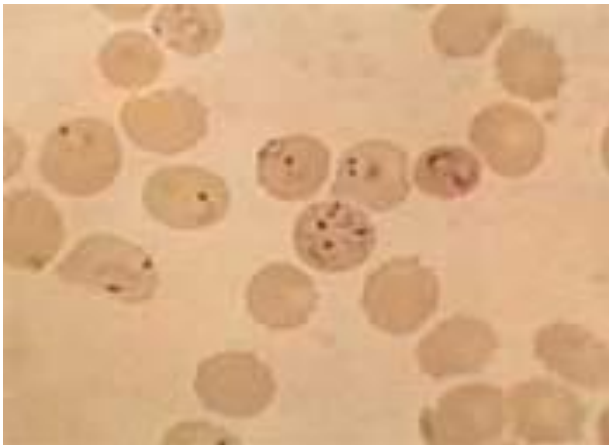


Plate III: Positive result by Microscopy

4.1.2 Prevalence of malarial parasites in Bosso and Paikoro LGAs in Niger State

Prevalence of malaria using RDT in the study area is shown in Figure 4.1. From the 500 participants in the study, 245 were positive for malaria parasite representing 49.0% prevalence. Out of which 120 were positive representing 48.0% prevalence in Bosso LGA, and 125 positive cases were recorded in Paikoro LGA representing 50.0% prevalence. The Table containing this information is presented in appendix II.

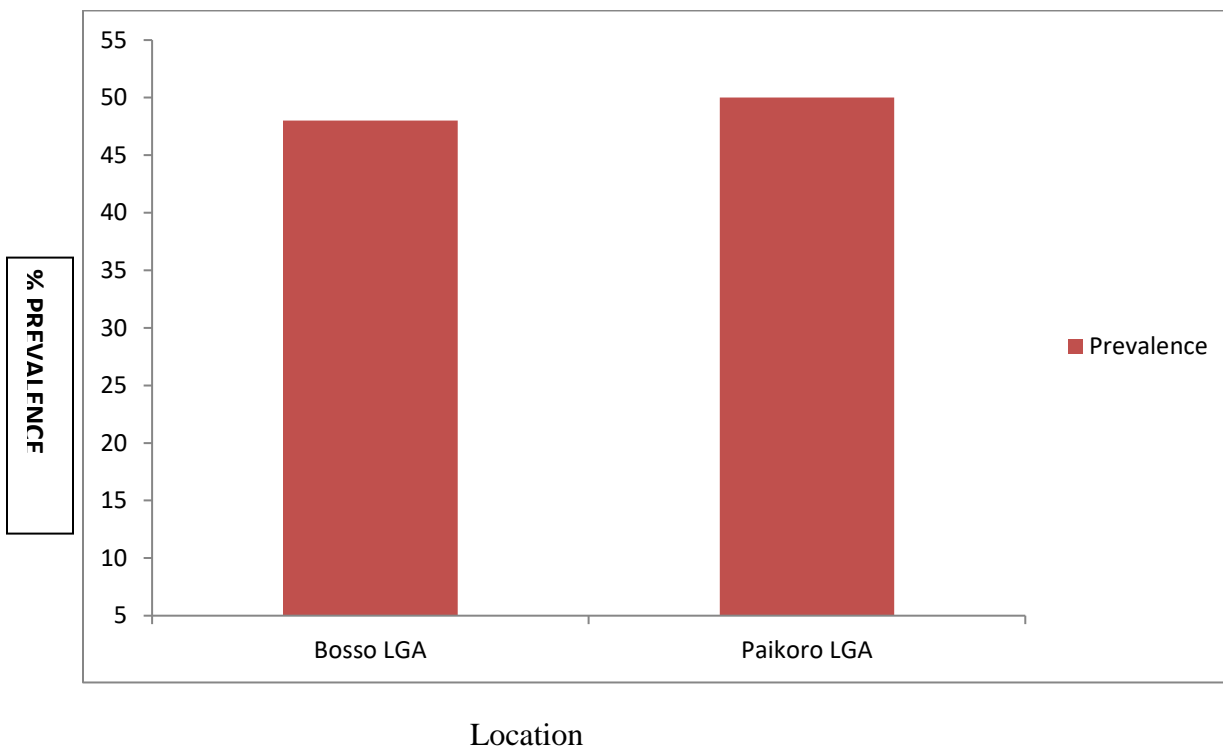


Figure 4.1. Prevalence of malarial parasites in Bosso and Paikoro LGAs in Niger State

4.1.3 Prevalence of malarial parasites in relation to socio-demographic parameters.

The prevalence of malarial parasites in relation to socio-demographic parameters are shown Table 4.1 below. From the results presented in the figure below, age group 10-12 had the highest prevalence of 75.0 % followed by age group 0-4 with a prevalence of 70.8 %. The least prevalence was recorded in age group 41-50. Although, women were considered for the study, it is evident that 75 male children were captured out of which 53 representing a prevalence of 70.7% for malaria parasite while 425 women including female children were captured from the study areas out of which 192 representing 45.2 % prevalence for malarial parasite. Of the total population sampled, the highest prevalence (56.0 %) was seen among those without any form of education. The least prevalence (38.2 %) was recorded among those with secondary education. Out the 500 samples, the highest prevalence (61.0 %) was recorded among the unemployed, this was followed in descending order by farmers (45 %), unskilled labourers (40.8 %), traders (33.7) and civil servants (32.7 %).

Table 4.1: Prevalence of malarial parasites in relation to socio-demographic parameters

Parameter	No. Examined (%)	Positive (%)	Negative (%)	P-value
Age				0.001
0-04	89(17.8)	63(70.8)	26(29.2)	
05-09	59(11.8)	41(69.5)	18(30.5)	
10-12	16(3.2)	12(75.0)	4(25.0)	
13-20	81(16.2)	34(42.0)	47(58.0)	
21-30	185(37.0)	68(36.8)	117(63.2)	
31-40	54(10.8)	22(40.7)	32(59.3)	
41-50	14(2.8)	5(35.7)	9(64.3)	
51-60	1(0.2)	0	1(100.0)	
>60	1(0.2)	0	1(100.0)	
Total	500(100)	245(49.0)	255(51.0)	
Gender				0.001
Male children	75(15.0)	53(70.7)	22(29.3)	
Female	425(85.0)	192(45.2)	233(54.8)	
Total	500(100)	245	255	
Education				0.019
None	223(44.6)	125(56.0)	98(43.9)	
Primary	126(25.2)	60(47.6)	66(52.4)	
Secondary	89(17.8)	34(38.2)	55(61.7)	
Tertiary	62(12.4)	26(41.9)	36(58.0)	
Total	500(100)	245	255	
Occupation				0.001
Farming Unskilled labour	100(20.0)	45(45.0)	55(55.0)	
Trading	71(14.2)	29(40.8)	42(59.1)	
Civil servants	80(16.0)	27(33.7)	53(66.3)	
Unemployed	49(9.8)	16(32.7)	33(67.3)	
Total	200(40.0)	128(61.0)	72(36.0)	
Total	500(100)	245	255	

At $P < 0.05$ age group and gender are significant risk factors influencing malaria infections. Education and occupation are significant risk factors influencing malaria infection at $P < 0.05$

4.1.4: Prevalence of malarial parasite in pregnant women

The positive cases of malaria in the study population with regards to pregnancy is shown in Figure 4.2. Gestational age and pregnancy was considered. And it was recorded that of the total of 118 pregnant women out of a total of 336 women in the study, 48.6 % malaria prevalence was recorded for women in their first and third trimesters, 44.0 % malaria prevalence was recorded in women in their second trimester. Appendix IV contains detailed information on this figure.

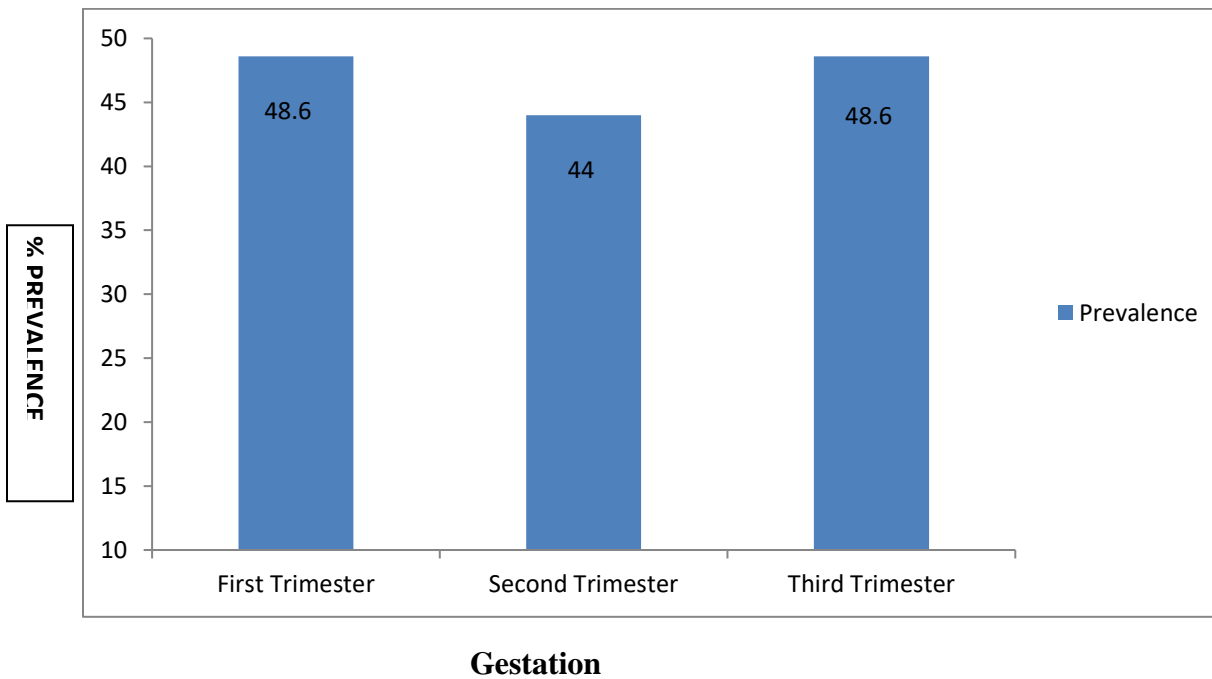


Figure 4.2: Prevalence of malarial parasite in pregnant women

4.1.5 Prevalence of malarial parasite among women and children

Prevalence of malaria among women and children was determined. Out of the 500 people sampled in the study areas, 118 were pregnant women, 218 were non pregnant women while 164 were children between the ages of 0-12 years old. Malaria prevalence for each of these sections of the study areas are represented in the figure below. The Table carrying this information is presented in appendix III.

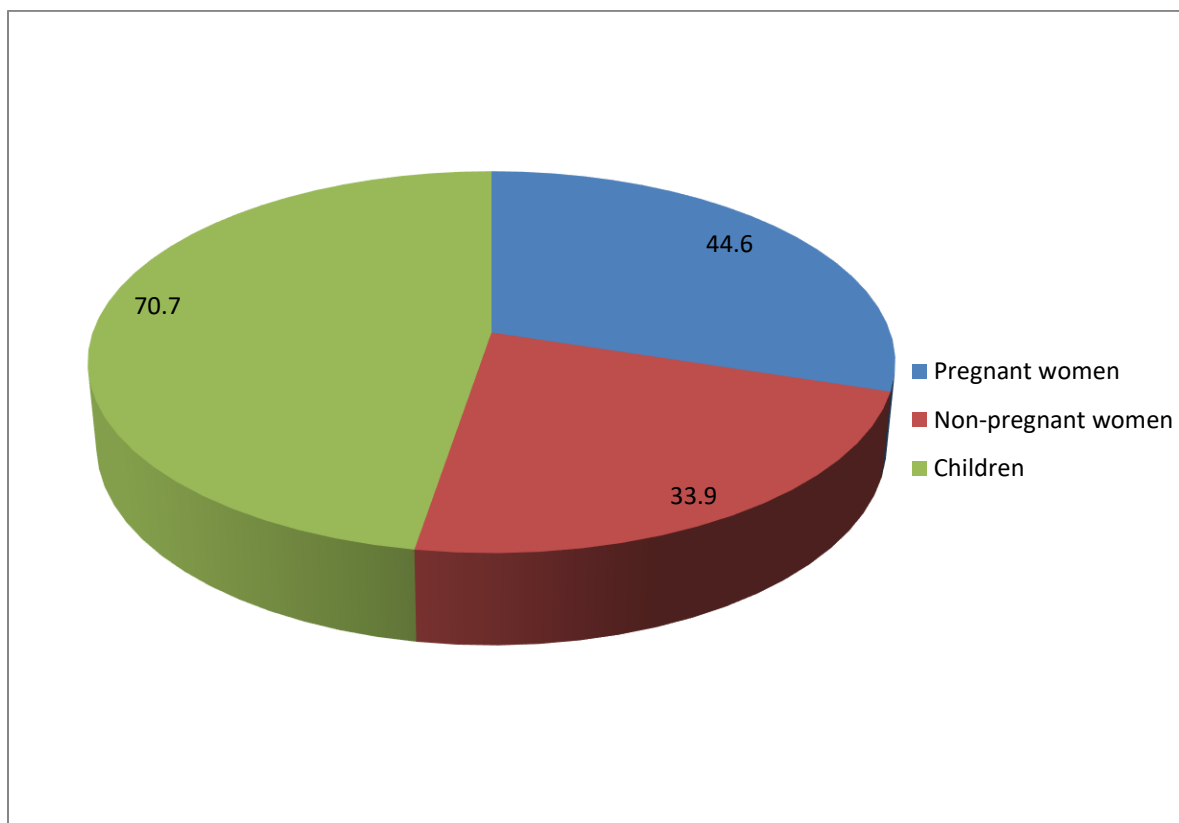


Figure 4.3: Prevalence of malarial parasite among women and children

4.1.6: Prevalence of malarial parasite with regards to knowledge of the Respondents

The prevalence of malarial parasite with regards to knowledge of the subjects on transmission is presented in Table 4.2. Out of the total population sampled, 58.0 % prevalence was recorded for those without the correct knowledge of transmission. While Respondents with the correct knowledge of transmission recorded prevalence of 45.1 %.

Table 4.2 Prevalence of malarial parasite with regards to knowledge of transmission

Response to knowledge of transmission	Num. of correct respondents (%)	Num. positive	Prevalence (%)	X ²	P-value
Correct knowledge	350(70)	158	45.1	11.26	0.0019
Incorrect knowledge	150(30)	56	58.0		

At $P < 0.05$ there is significant difference between respondents with the correct and incorrect knowledge of transmission of malaria infections.

The prevalence of malarial parasites in respect to the knowledge of signs and symptoms of malaria was described in Table 4.3. Of the sampled population, 59.1 % had knowledge of headache, 51.0% dizziness, followed in descending order by chills (50.4 %), loss of appetite (50.2 %), fever (46.6 %), weakness (46.4 %), body pain (45.6 %), vomiting (35.5 %) and 4.8 % had no knowledge of the signs and symptoms.

Table 4.3 Knowledge of signs and symptoms of malaria among Respondents

Signs & Symptoms	Num. of correct respondents (%)	Num. Positive	Prevalence (%)	X ²	P-value
Fever	231(46.6)	151	65.4	4464.0	0.459
Headache	293(59.1)	127	43.3		
Vomiting	176(35.5)	94	53.4		
Chills	250(50.4)	226	90.4		
Body pain	226(45.6)	202	89.3		
Dizziness	253(51.0)	226	89.3		
Loss appetite	249(50.2)	233	93.5		
Weakness	230(46.4)	196	85.2		
Don't know	24(4.8)	10	41.7		

Knowledge of signs and symptoms of malaria has no significant effect on malaria infection at $P > 0.05$.

Prevalence of malaria in respect to the knowledge of preventive strategies is shown in Table 4.4. From the study population, 51.8 % had knowledge of insecticide treated nets, 42.2 % knew of larval source management, 41.8 % had knowledge of indoor residual spraying while 30.0 % knew of intermittent preventive treatment in pregnancy.

Table 4.4 Prevalence of malaria in respect to knowledge of prevention strategies

Prevention strategies	Num. of respondents (%)	Num. of positive	Prevalence(%)	X ²	P-value
ITN	259(51.8)	117	45.2	1448.0	0.465
IRS	209(41.8)	94	44.9		
IPTP	150(30.0)	74	49.3		
LSM	211(42.2)	89	42.2		

At $P > 0.05$ respondents with the correct knowledge of prevention strategy do not have a significant effect on malaria infections.

ITN; Insecticide Treated Net, IRS; Indoor Residual Spraying, IPTP; Intermittent Preventive Treatment in Pregnancy, LSM; Larval Source Management.

4. 1.7 Prevalence of malaria with regards to the haematological parameters

The prevalence of malaria with regards to the haematological parameters of the subjects are presented in Table 4.5. Out of the total sampled population, blood group AB had the highest prevalence (75.3 %). This is followed in descending order by blood group B (68.4%), blood group A (62.1 %) and blood group O (18.3 %). Genotype AA subjects were the most susceptible (50.2 %) to malarial infection. The least prevalence (16.6 %) was seen among genotype SS. Haemoglobin concentration was statistically significant ($p = 0.036$). Subjects having severe anaemia recorded the highest prevalence 55.3 %, moderately anaemic subjects recorded prevalence of 40.0 %, subjects with mild anaemia had 50.5 % and the non anaemic subjects recorded malaria prevalence (47.8 %). Charts

representing information on haematological profile of subjects and other detailed tables are presented in appendices IV to VII.

Table 4.5 Prevalence of malaria with regards to the haematological parameters

Parameters		Num. tested (%)	Num. tested positive	Prevalence	P-value
Blood group	A+	82(16.4)	49	59.8%	0.047
	A-	21(4.2)	15	71.4%	
	B+	117(23.4)	79	67.5%	
	B-	16(3.2)	12	75.0%	
	AB+	51(10.2)	39	76.4%	
	AB-	22(4.4)	16	72.7%	
	O+	184(36.8)	32	17.3%	
	O-	7(1.4)	3	42.8%	
Genotype	AA	205	103	50.2	0.139
	AS	280	139	49.6%	
	AC	3	1	33.3%	
	SS	12	2	16.6%	
	SC	0	0	0	
HB	Severe anaemia	47	26	55.3%	0.036
	Moderate anaemia	35	14	40.0%	
	Mild anaemia	182	92	50.5%	
	Non-anaemia	236	113	47.8%	

Haematological parameter of the patients is a significant risk factor influencing malaria infection at $P < 0.05$ and at $p > 0.05$ there is no significant difference.

Legend: Severe anaemia; $HB \leq 5g/dL$, Moderate anaemia; $HB > 5-8g/dL$, Mild anaemia; $HB > 8-11g/dL$, Non-anaemic; $HB > 11$

4.2 Discussion

Malaria remains a prominent and ancient disease which has been profiled and studied. Nearly half of the world's population remains at risk and to a larger extent the African continent. Various research and studies have shown considerable progress in the global fight against malaria. Nigeria bears a higher burden of malaria in African region despite the various gains in the control and the quest for the elimination of malaria.

The world Health organization gave approval for the use of RDTs especially for field research in rural areas where access to light and microscopy is an issue. This has gone a long way to help in the diagnosis of the disease. The method of RDT employed for this survey also tested the sensitivity of the Bioline RDT used against the gold standard of microscopy for diagnosis of MP (malarial parasite). The Kappa value (0.363) revealed that there is a fair level of agreement between the use of RDT and microscopy in the diagnosis of malarial infection. The kappa value indicates that strong reliance should not be given to the use of RDT in the diagnosis of malaria as the agreement is between the use of RDT and microscopy is only fair. This could be as a result of lack of proper storage conditions of the RDT cassettes, inability of manufacturing companies to adhere to the right proportion of agents in the manufacture of the cassettes (Bassey and Izah 2017). Moreso, different

kits have specificity for the detection of specific species of *Plasmodium* and specific antigen coated in the strip might be different from the species of *Plasmodium* been sought after in the blood sample been tested and this could result in false negative results. Hence, except on emergency cases and in the absence of a microscope to perform microscopy, the use of RDT in the diagnosis of malaria should be discouraged and the gold standard; microscopy should be encouraged at all times (Beisel *et al.*, 2016; Boyce *et al.*, 2018).

Out of 500 samples, 245 were positive for malaria parasite. Bosso LGA had 120 positive samples representing 48.0 % while Paikoro LGA recorded 125 representing a prevalence of 50 %. The prevalence of malaria by RDT in the study area was 49.0%. The prevalence obtained in this study is lower than the prevalence of 57.6 % reported by Kuta *et al.* (2014) who worked on screening of blood samples from school aged children (0-12 years) for the detection of *Plasmodium* parasites in Paikoro Local Government Area of Niger State, Nigeria. The overall prevalence is also lower than what was reported by Edogun *et al.* (2017) who recorded an overall prevalence of 51.9 % in three selected Local Government Areas of Niger State, North Central, Nigeria. Millicent and Gabriel (2015) obtained a prevalence of 35.7 % in patients attending the General hospital Makarfi, Makarfi Kaduna State, North-Western Nigeria. Mbuh *et al.* (2003) recorded a prevalence of 27 % in a study titled rate of co-infection with malarial parasites and *Salmonella* Typhi conducted in Zaria, Kaduna State.

The varying prevalence reported in various regions across Nigeria could be as a result of different geographical locations and climate of the regions with respect to the presence and breeding of the insect vector and the habits of the area to preventive and control measures (Bassey and Izah 2017). Other factors such as age, educational status, and pregnancy could also be responsible for the varying prevalence recorded in various regions (Oladeinde *et al.*, 2014). Furthermore, the lower prevalence obtained in this study as against the higher prevalence(57.6 % and 35.7 % respectively) reported by

previous authors; Kuta *et al.* (2014) and Millicent and Gabriel (2015) in Niger State could be as a result improved awareness and preventive measures being put in place at various levels through research and government intervention programs. This decrease will help to reduce the financial burden incurred by the individuals in the study area and the State at large.

Although malaria affects all age groups, age group 0-4years had the highest prevalence of 70.8%. This high prevalence when compared with other age groups could be attributed to their vulnerability due to immature immune system and inability to adequately protect themselves from mosquito bites if not provided for by their parents. The high prevalence recorded in this age group was also reported by Edogun *et al.* (2017) who recorded prevalence of 48.4 % in children aged 1-5 years. It is worthy of note that age groups 5-9 years and 10-12 years also had very high malaria prevalence. The least prevalence of 35.7 % was seen in age group 41-50 years.

Malaria affects both males and females however, different studies have reported varying prevalence of malaria amongst the male children and female, some indicating higher prevalence for male children while others reported higher prevalence in female (Okonkwo *et al.*, 2010; Tela *et al.*, 2015). From the findings of this research, it was found that male children had higher prevalence (70.7 %) of malaria than females (45.2 %). Similar findings were reported by Millicent *et al.*, (2015) and Kuta *et al.* (2014) with higher prevalence in male children than in females.

From the results on the number of pregnant women, non-pregnant women and children captured in the study population, 46.6 % prevalence was obtained for malaria in pregnancy, 33.9 % prevalence for the non-pregnant while children had 70.7 % prevalence. The high prevalence in children could suggest that mortality in children arising from malaria could still be high (Aregbeshola and Khan 2017). More so, the prevalence of malaria in pregnant women is still high and this could lead to

complications in pregnancy and even maternal mortality. The vulnerability in pregnant women could be as a result of immune suppression in pregnancy.

Although previous literatures have established that malaria cuts across all educational status, this study also put to test the role of education in malarial infection. Malaria prevalence in patients with no formal education was 56.0 % making it the highest prevalence under educational status. This is followed in descending order by primary (47.6 %), tertiary (42.0 %) and secondary educational status (38.2 %) respectively. Education plays key role in malarial prevention and control as seen from the study. The highest malaria prevalence by RDT was recorded in patients without any formal education. The p-value, 0.019 indicate a statistically significant result for the role of education as a risk factor in malarial infection in the study population. This agrees with the findings of Valea *et al.* (2012), who recorded high malarial prevalence among those without any educational status. Aregbeshola and Khan (2017) also reported from their study a high prevalence (46.3%) of malaria among the group without any form of education. This could be as a result of little or no knowledge of transmission or preventive strategies by this group of people. This could have negative impact on the finance in terms of money that will be used for programs in the prevention and control of the Local Government Areas in the study.

On the basis of occupation, it was seen that the highest malarial prevalence by RDT was recorded in the unemployed with a prevalence of 61.0% this is followed by farmers out of 100 farmers tested 45 proved positive for malaria representing 45.0 % prevalence. The least prevalence was seen in civil servants having a total of 49 tested patients, with 16 positive for malaria by RDT representing 32.7 % prevalence. The study agrees with that of Edogun *et al.* (2017) where farmers had appreciably high prevalence (39.3 %) as against other occupations. This could be traced to outdoor mosquito bites experienced by the farmers. If the farmers are not able to cultivate in the farming seasons due to

infection by the parasite, it could have reduced the availability of food and this will affect the overall well-being of the community (Ughasoro *et al.*, 2013). The highest prevalence seen in the unemployed is probably traceable to their inability to access good and healthy living, lack of resources to afford the basic preventive measures and lack of money to access diagnosis and treatment (Pam *et al.*, 2015). The least prevalence recorded in civil servants can be traced or linked to their level of exposure and knowledge on the basic preventive measures and availability of money for early diagnosis and treatment (Bassey and Izah 2017). The difference in statistics among the various occupations was significant ($p = 0.001$, $p < 0.05$). Hence, occupation from this study is well considered as a risk factor influencing malaria infection in the study population. The findings agree with studies of Edogun *et al.* (2017) who reported that occupation is a risk factor in malarial infection.

Adequate knowledge of signs and symptoms of malaria was also tested among the participants in the research. The findings revealed that of the 500 patients, 59.1 % of the population had knowledge of headache as a symptom of malaria. Other signs and symptoms are recorded below in descending order according to knowledge of participants; dizziness (51.0 %), chills (50.0 %), loss of appetite (49.8 %), fever (46.2 %), weakness (46.0%), body pains (45.2 %), vomiting (35.2 %) and very little group; 4.8 % had no knowledge respectively. This is in agreement with results of Okwa and Ibidapo (2010) who reported headache (49.6 %) as the most known and common symptom within their study population. It is worthy of note that very small percent of the population had no knowledge of the signs and symptoms even in the rural communities where this study was carried out. This indicates that there is an appreciable knowledge of the signs and the symptoms of malaria; this can be traced to various levels of research done on the disease and the various international, federal and state

involvements in ensuring that the disease is adequately managed. Although the p-value (0.459) was statistically not significant varying levels of malarial prevalence was recorded for the various groups.

The participants were also subjected to test to ascertain the level of their knowledge on various preventive strategies. The preventive strategies include; insecticide treated nets (ITN), indoor residual spraying (IRS), intermittent preventive treatment in pregnancy (IPTP) and larval source management (LSM). Out of the four strategies sort for, 51.8 % of the study population had knowledge of ITN, 42.2 % for LSM, 41.8 % for IRS and 30.0 % for IPTP respectively. Okwa and Ibidapo (2010) and Chukwuocha (2012) in their independent studies also reported ITN prevention strategy as the most known by the study population. Statistically, p value (0.465) was not significant because the knowledge of prevention strategies without properly practicing same has little or no effect in reducing the incidence of malarial infection.

Response to correct knowledge of transmission was also tested and this revealed that 350 (70.7 %) subjects of the total population had the correct knowledge that infected mosquito bite brings about transmission and of this 350, 158 tested positive to malaria representing 45.1 % prevalence. The other 150 representing 30 % had no knowledge of transmission. From the 150, 87 tested positive for malaria representing 58.0 % prevalence. Okwa and Ibidapo (2010) in a study reported high knowledge of the cause of malaria among their study population. With the right knowledge of transmission, subjects are can prevent transmission of the disease. But subjects without the right knowledge of transmission are mostly at risk of infection (Okwa and Ibidapo 2010; Edogun *et al.*, 2017).

All 500 blood samples were screened for their ABO blood groups. The following malarial prevalence was recorded on the basis of blood group; blood group AB (75.3 %), blood group B (68.4 %), blood

group A (62.1 %) and blood group O (18.3 %) respectively. The high frequency of blood type O was also recorded by Tewodros *et al.* (2011) but the study disagrees with the findings of Uneke *et al.* (2006) with low frequency of blood type O.

Malarial infection showed significant association with blood group ($P=0.047$) with the highest malarial prevalence (75.3 %) recorded among individuals with blood group AB. Blood group O had the least malaria prevalence of 18.3 %. This agrees with the findings of Tewodros *et al.* (2011) who recorded lowest prevalence of malaria among the O blood type. But the study is not in credence with the findings from the studies of Okwa and Ibidapo 2010 in a work titled ‘The Malaria situation, perception of cause and treatment in a Nigerian University’ where the most infected with malaria were those of blood group O.

The present finding in this study substantiates the hypothesis about a selective (survival) evolutionary advantage of *P. falciparum* infection on blood group O cells compared with other blood group types (A, B or AB) in malarial endemic areas (Cserti and Dzik 2007; Fry *et al.*, 2008). Previous studies have tried to establish the reason for low level of infection among the O blood groups. Antigens of blood groups A and B have been suggested to play important roles in cytoadherence (Afoakwah *et al.*, 2016). Due to the absence of A and B antigens on the surface of blood group O erythrocytes, cytoadherence, and hence rosetting and sequestration, is reduced in individuals with blood group O (Tewodros *et al.*, 2011). Several mechanisms relate to these associations, including affinity for *Anopheles* species, shared ABO antigens with *P. falciparum*, impairment of merozoite penetration of RBCs, as well as cytoadherence, endothelial activation and rosetting (Tewodros *et al.*, 2011; Obisike and Thomas 2020).

The mechanism by which ‘A’ promotes susceptibility and ‘O’ confers a relative protective effect against high *P. falciparum* parasitaemia is not well understood. Nevertheless, different studies have come up with their reasonable explanations on the basis of rosette formation. Several reports support the hypothesis that blood group A represents a risk factor for high chance of rosette, which is usually characterized by high *P. falciparum* parasitaemia during malaria infection and a reducing effect of blood group ‘O’ on rosette (Tewodros *et al.*, 2011; Gayathri *et al.*, 2013).

The presence of several glycosylated adhesion molecules such as intracellular adhesion molecule, complement receptor, heparin sulfate-like glycosaminoglycan, platelet glycoprotein CD36, low arginine and nitrate levels, presence of cellular micro-particles and the nature of sugar molecules (trisacchrides) in blood group ‘A’ cells promote a high chance of binding with the rosette-forming surface molecules of the *P. falciparum* such as Duffy binding-like domain 1 alpha of *P. falciparum* erythrocyte membrane protein-1 (Fan *et al.*, 2011; Tewodros *et al.*, 2011; Afoakwah *et al.*, 2016). Conversely, blood group ‘O’ cells show deficiency of most of the above adhesive molecules and contain disaccharides sugar molecules which reduce the rate, size and stability of rosette formed during *P. falciparum* infection (Tewodros *et al.*, 2011; Afoakwah *et al.*, 2016).

The destruction of both parasitized and non-parasitized erythrocytes as well as rosetting and sequestration of parasitized erythrocytes has been cited to be the major cause of severe anaemia in complicated malaria (Afoakwah *et al.*, 2016). Malaria has long been recognized as one of the major causes of anaemia (Okwa and Adejoke 2010). Although a wide, and confusing variety of definitions of anaemia in general have been proposed, the most commonly used definitions in malaria studies, based on haemoglobin concentrations, are as follows; Mild anaemia ≤ 11 g/dL Moderate anaemia ≤ 8 g/dL Severe anaemia ≤ 5 g/dL (White 2018).

The findings from the present study give credence to previous studies on the reduction in haemoglobin (Hb) of blood of malaria infected subjects. Of the 500 participants in the study, 236 were non anaemic from their Hb values (ranging from >11 to 16.6 g/dL), out of these, 113(47.8 %) were positive for malaria. For the 113 that had malaria but presented to be non anaemic some factors such as blood type, stage of infection, nutritional state, level of immunity and possibly some intake of anti-malarial drugs before visiting the clinic facility could have been responsible for that. One hundred and eighty two (182) persons out of the study population had mild anaemia and of these, 92(50.5 %) were positive for malaria. This implies that slightly above half (50.5 %) of the study populations having mild anaemia were positive for malarial infection. Less than half (40.0 %) of the study population having malaria had moderate anaemia. More than half (55.0 %) of the study population living with malaria had severe anaemia. Consequently, the anaemic situation of the study population is high and this could have detrimental effects on the health and economic status of the area. Therefore, there is need to caution people on dieting and intake of iron rich foods as this will help in reducing the anaemic status of the study population. Chi square analysis revealed that p-value of anaemia and malaria in the study population was statistically significant ($p = 0.036$). This study agrees with the other studies by White (2018) and Burham *et al.* (2016) who in their independent studies reported a correlation between malarial infection and anaemia at different severities.

Participants of the study with genotype SS appeared to be more resistant to malaria than their counterpart AA, AS, AC and SC. This is perceived to be so because the red blood cells of the other genotypes except SS appeared to be more conducive for the growth and development of *Plasmodium* species (Okwa and Adejoke 2010). Okwa and Adejoke (2010) opined that there is higher resistance to *P. falciparum* infection in patients with homozygous sickle cell disease. In their study, malarial parasites were predominant in HbAA and least in HbSS. This study give credence to the findings of

Okwa and Adejoke (2010) as the least prevalence was found in subjects with genotype SS. The low level of malarial infection recorded in the SS genotype can be traced to low level of folic acid in the subjects with the sickle cell gene. Tetrahydrofolate is essential for DNA synthesis, survival and growth of malarial parasite in the blood. Hence low level of it in the SS genotype could be the reason for the low prevalence of malaria. Conversely, sufficient folic acid in the AA genotype could be attributed to the high prevalence of malarial infection recorded in the study population. About half (50.2 %) of the study population living with malaria were of the AA genotype, 49.6 % were of AS genotype while 16.6 % were of the SS genotype and 33.3 % were of AC genotype. The HbSS patients even though with the least prevalence are however not exempted from great risk for increased morbidity and mortality arising from malaria as a result of inherent HbSS complications and immune suppression. There is no relationship between malaria infection and genotype of a person as chi square analysis revealed no significant difference in the P-value ($p = 0.139$). This study agrees with the findings of Faga *et al.* (2020) having the least infection in SS genotype individuals and the highest infection recorded among genotype AA.

CHAPTER FIVE

5.0 CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

The incidence of malaria in Bosso and Paikoro LGA obtained in this study was 49.0%. Children are the most vulnerable groups affected by malaria in the study area, this was followed by the pregnant women. The study also revealed that malaria affects both males and females.

The rapid diagnostic test cassette used was not sensitive in the detection of malarial parasite.

It is also evident that anaemia and malaria infection cannot be separated, in most of the cases in the study, anaemia at various severities was an indicator of the presence of malarial parasite. The relationship between ABO blood group and malaria infection was also revealed from the study, subjects with blood group O exhibited low malarial prevalence when compared with the other blood groups. Blood groups AB were the most susceptible to malarial parasite with the highest prevalence. All genotypes were affected by the disease, although, the SS genotypes have low burden of the infection. The AS and the SS genotypes had the highest number of uninfected subjects.

There is a relationship between malarial infection and socio-demographic factors such as (age, occupation and education) as revealed from the research. Although malaria affects both the educated and uneducated, the study revealed that education plays a major role in malarial infection as the educated subjects recorded lower prevalence than their uneducated counterparts. It was also revealed from the study that occupation and age are factors that predisposes to malarial infection.

5.2 Recommendations

The following recommendations are drawn from this research;

1. Many cases of malaria will be missed if total dependence is placed on RDT in diagnosis. Microscopy which is regarded as the gold standard should be encouraged at all times where practicable.
2. Further studies are required to know the compliance to the various existing preventive strategies already in place. This will require researchers visiting the homes of the rural dwellers to check if the strategies are been practiced.
3. Adequate intake of iron rich foods such as sea foods, beans, legumes, dark green vegetables, liver and other organ meats should be encouraged in the rural communities. This will in turn help to build blood and help in the management of severe aneamia cases resulting from malarial infection.
4. Proper and adequate training should be organized for the health personnels in the rural areas on the need for standard procedures in the diagnosis of malaria in order to avoid administering false results.

5. Public awareness campaign using health care workers, community leaders, town criers, at social and religious gatherings on malarial preventive measures such as bush clearing, cleaning of drainages and other breeding grounds should be encouraged.

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APPENDIX I: QUESTIONNAIRE

Introduction: I am Ajanya, Benjamin Unekwuajo, an M. Tech student of the Department of Microbiology, Federal University of Technology Minna. I am conducting a research on haematological profile of women and children infected with malaria parasite in Bosso and Paikoro communities in Niger State.

Purpose of the research: The research will help in ascertaining the current prevalence of malaria in the study area and will also help in malaria control program to better understand the informational needs of the community on malaria issues.

Procedure: If you agree with the purpose of the research, I will ask you a few questions about your knowledge and attitude in relation to malaria diagnosis and treatment. The questioning will last approximately 10-15 minutes.

Benefits: There are no direct benefits for you being part of this research. However, your contribution will help immensely to a better malaria control program in the community. You are free not to participate in this research or not to answer any question you feel uncomfortable with. Should you agree to be part of the research, feel free to interrupt the interview process at any time. Confidentiality is guaranteed, your name will not appear in any oral or written report of this study. There are no wrong or right answers. Your openness and honest opinions are extremely important. In case you do not understand a question or issue, please ask me to repeat or clarify.

Would you like to participate in this study? Yes () No ().

Section 1

Demographical data

1. Age in years 0-3 () 4-13 () 14-24 () 25-35 () 36-50 () 51-60 () > 60 ()
2. Marital status Single () Married () Divorced () Widowed ()
3. Level of Education None () Primary () Secondary () Tertiary ()
4. Occupation Trader () Farmer () Civil servant () Unskilled labourer () Pensioner ()
5. Primigravidae () Multigravidae ()
6. Trimester 1st () 2nd () 3rd ()

Section 2

7. Do you know how malaria is transmitted? Yes () No ()
8. Have you been treated of malaria in the recent past? Yes () No ()

9. Which are the common signs and symptoms of malaria? Headache () Fever () Chill and shivering () Body pains () Loss of appetite () Vomiting () Weakness () Dizziness () Don't know ()
10. How important will you rate going for laboratory test before treatment of malaria? Very important () Barely important () Not important ()
11. Do you know any of these control strategies? Use of insecticide treated nets () Indoor residual spraying () Intermittent preventive treatment of malaria in pregnancy () Larval source management ()

APPENDIX II: Prevalence of Malaria by RDT in the study area

LGA	Number of samples	Number of positive samples	Prevalence (%)
Bosso	250	120	48.0
Paikoro	250	125	50.0
Total	500	245	49.0

APPENDIX III: Sensitivity pattern of rapid diagnostic tests for detecting malarial parasites

Test	MICROSCOPY		Kappa value	
	Negative (%)	Positive (%)		
RDT				
	Negative	10 (31.3)	12(37.5)	0.363
	Positive	0 (0.0)	10(31.3)	
Total		10 (31.3)	22 (68.7)	

The Kappa value (0.363) or the level of agreement shows that there is a fair level of agreement between the use of RDT and microscopy in the diagnosis of malaria infection.

Results interpretation of Kappa values:

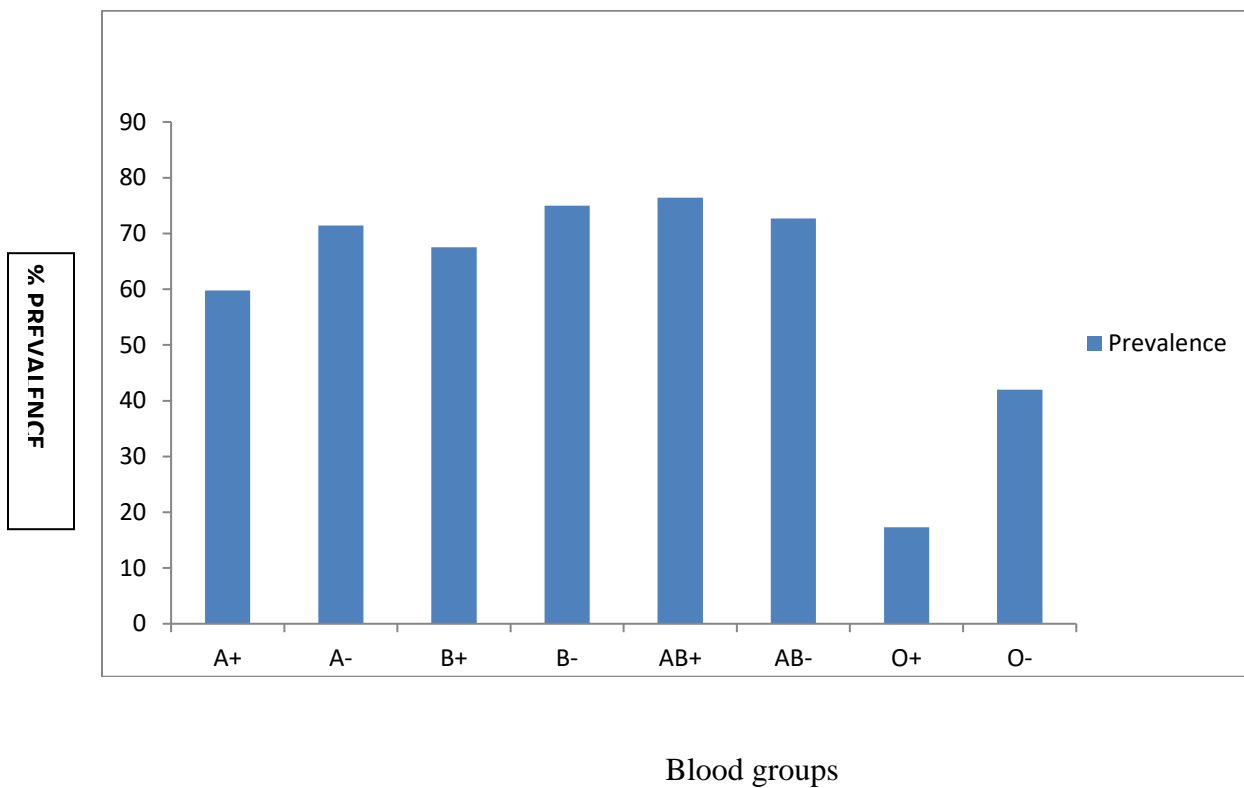
Kappa Values	Level of agreement
<0.01	Poor
0.01-0.2	Slight
0.21-0.40	Fair
0.41-0.60	Moderate
0.61-0.75	Substantial
0.76-1.0	Almost perfect

APPENDIX IV: Prevalence of malaria with regards to pregnant women in the study population

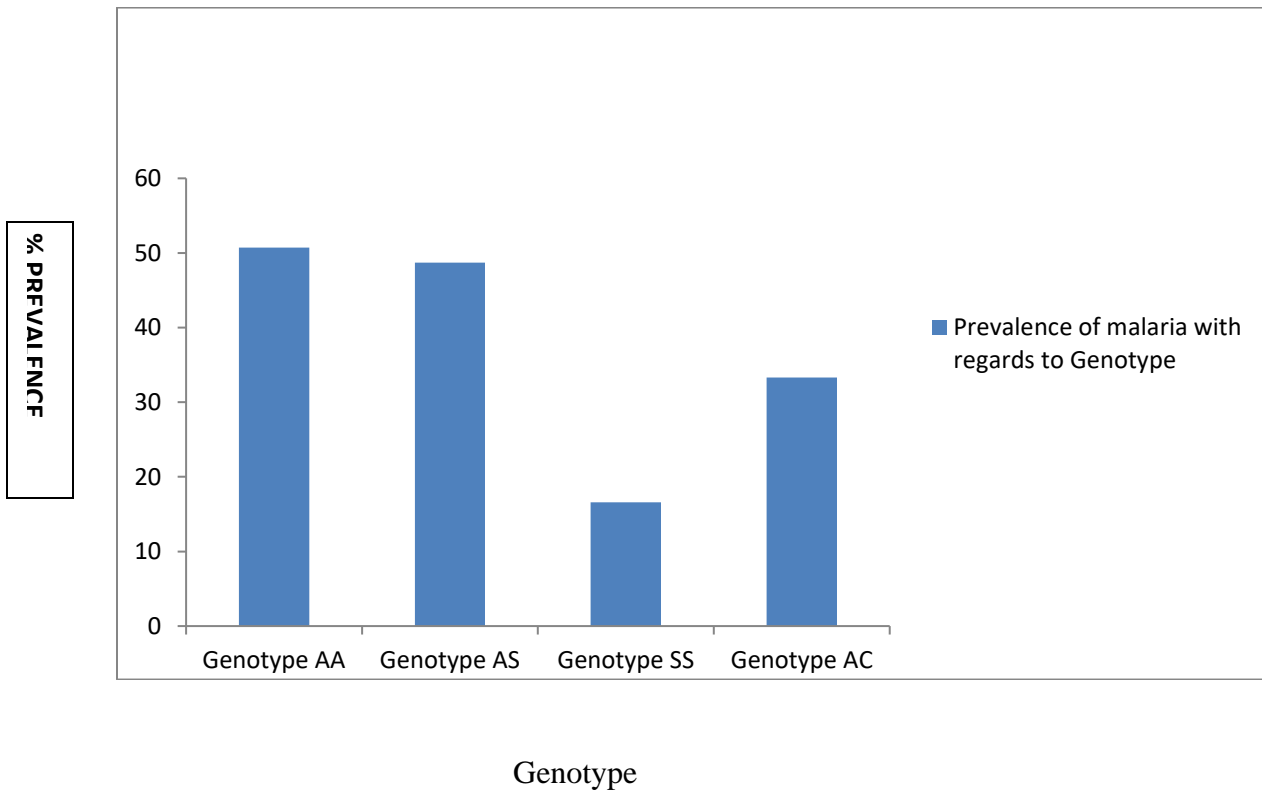
Parameter		Num. Tested(%)	Num. tested positive	Prevalence (%)	P-value
Trimester	First	35(29.6)	17	48.6	0.875
	Second	48(40.6)	21	44.0	
	Third	35(29.6)	17	48.6	
Non Pregnant		218(43.6)	74	33.9	
Children		164(32.8)	116	70.7	0.046
Pregnant Women		118(23.6)	55	46.6	

The subject, trimester is not a significant risk factor influencing malaria infection at $P>0.05$.
At $p<0.05$, some specific class of the study area show significant risk factor to malaria infection

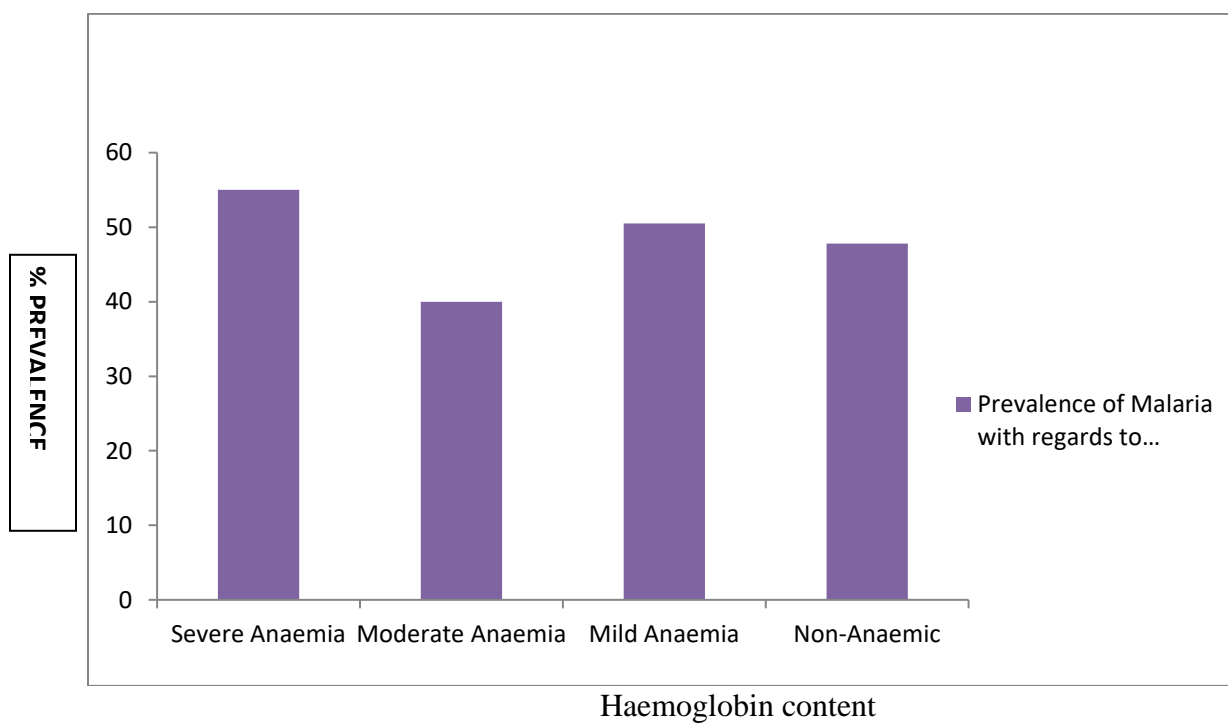
APPENDIX V: Prevalence of malarial parasite among patients with different blood group



APPENDIX VI: Prevalence of malaria with regards to haemoglobin genotype



APPENDIX VII: Prevalence of malaria with regards to haemoglobin concentration



Legend: Severe anaemia; $HB \leq 5g/dL$, Moderate anaemia; $HB > 5-8g/dL$, Mild anaemia; $HB > 8-11g/dL$, Non-anaemic; $HB > 11$

APPENDIX VIII: Heamatological parameters of sampled population

Patient ID	Genotype	Blood group	HB(g/dL)	PCV (%)	MCV (fL)	MCH (pg)	MCHC (g/dL)	RBC (10 ⁶ /μL)	PLC (1000/mm ³)	TWBC (/L)	RDWC (fL)	Eosinophils (%)	Neutrophils(%)
M1	AA	O+ve	10.5	32	86	28	34	4.1	240	9.3	13.8	6	63
M4	AA	A+VE	11.4	34	61	20	31	6.1	111		15.1	9	44
B2	AA	A+VE	13	39	72	23	33	5.3	255	6.1	14.4	4	49
B4	AS	O+ve	14.1	42	77	26	34	6.1	35	7.1	14.1	6	74
B7	AS	A+VE	9.1	27	68	28	38	3.1	243	6.1	45.1	8	66
B8	AA	O+ve	10	30	73	23	35	3.2	143	3.1	49.2	8	85
BJ10	AA	B-ve	10.1	30	72	28	32	2.3	164	7.1	37.3	22	56
BJ7	AA	B-Ve	10.2	30	70	28	33	2.5	204	8	32	21	57
BJ11	AS	B+ve	12.2	36	59	24	32	2	118	9.1	39.2	23	64
BJ16	AA	B+ve	15.5	35	62	23	35	2.2	162	5.1	33.1	29	46
BJ30	AC	B+ve	11.3	33	61	26	33	2.3	167	6.1	33.2	14	58
BJ39	AA	O+ve	15.7	46	69	22	36	2.1	121	5.2	32.3	18	58
BJ48	AA	A+VE	10.7	32	73	20	30	1.2	182	5.1	32.1	93	63
B30	AA	O+ve	12.1	36	65	23	37	1.4	190	3.4	37.8	10	52
B33	AC	O+ve	12	36	73	26	38	3	368	6.1	36.6	7	65
B40	SS	O+ve	5.1	15	60	18	28	1.8	100	16.5	39.1	8	50
M17	AA	O+ve	13.8	40	76	22	36	2.9	214	10.1	34.1	2	58
M25	AS	B+ve	9.5	28	62	20	35	12.1	148	6.1	35	3	53
M44	AA	B+ve	16.2	48	51	25	38	2	110	4.4	37	17	37
M50	AS	AB+ve	11	33	68	20	36	2.8	362	5.1	35	4	66
G1	AA	A+VE	10.4	31	54	21	35	2.1	274	6.1	34.7	9	69
G6	AA	O+ve	13.7	40	68	22	35	2.6	388	5.4	36.2	14	65

G22	AC	AB+ve	10.1	30	68	22	36	3	171	7.1	38.1	13	63
G24	AA	O+ve	10.3	30	66	23	35	2.4	186	9.1	37.4	8	72
G30	AS	B+ve	11.9	35	81	26	34	2.8	260	8.1	30.6	4	55
G39	AA	B+ve	9.8	29	58	23	36	1.7	184	4.1	36.2	14	60
G40	AA	O+ve	9.5	28	55	18	28	2.8	166	10.1	39	8	50
G49	AC	B+ve	11.6	33	68	21	37	3	207	6.6	32.8	5	62
G1	AA	O+ve	16.6	48	84	23	38	3	258	4.9	36	6	44
G3	AS	B+ve	10	30	61	26	398	2.1	203	6.1	30.9	8	58
G17	AS	B+ve	9.8	28	66	20	34	3	214	8.1	35.5	2	62
G20	AA	O+VE	13.8	42	78	25	38	2.8	284	9.9	37.8	6	60
G25	AS	B+ve	9.7	28	53	20	33	1.8	160	5.1	37	8	61
G30	AS	B+ve	11.3	33	66	21	36	2.1	345	6.1	36.4	15	60
G1	AS	A+VE	12.6	37	66	20	35	1.6	166	6.1	33.5	4	63
G3	AA	B+ve	12.6	38	64	20	36	3.6	305	8.8	36.6	4	60
G7	AS	B+ve	9.9	29	70	21	35	2.6	146	6.1	28.9	4	64
G16	AC	O+ve	10.1	30	71	22	37	2.5	214	6.1	36.5	8	68
G25	AA	B+ve	10.8	32	68	20	34	2.6	121	4.1	35.1	8	64
G39	AA	O+ve	8.8	26	63	19	28	2	116	7.1	29.6	3	77
G50	AA	O+ve	12.1	36	66	26	38	3	214	7.8	34.4	14	50
KW1	AS	A+VE	11.3	33	68	21	35	2	114	3.1	35.6	2	68
KW3	AA	O-ve	7.8	23	56	18	30	1.8	78	9.6	36.1	4	71
KW15	AA	A+VE	12.7	38	67	25	37	2.8	252	9.4	30.4	3	62
KW17	AA	O+ve	12.1	36	68	21	33	3.2	361	8.9	36.6	3	65
KW25	SS	B+ve	6.8	20	52	18	26	2	94	11.8	40.1	4	52
KW30	AC	B+ve	12	36	66	23	30	2.6	260	6.3	29.8	9	55
KW40	AS	AB+ve	11.9	35	58	20	35	3	211	7.1	35.4	10	60
KK2	AS	O+ve	10.1	30	64	20	35	2.7	214	7.9	34.4	3	62
KK5	AA	AB+ve	15.6	46	78	25	38	2.9	301	16.1	39.1	1	55
KK11	AA	A+VE	13.6	40	83	25	39	3.4	406	8.7	38.9	5	60
KK16	AS	AB+ve	9.8	29	55	20	35	3	235	5.4	37.5	7	58
KK36	AS	O+ve	11.6	35	65	20	34	2.6	183	7.1	35.3	8	68
KK49	AA	O+ve	12.5	37	58	23	34	3	215	6.1	37.8	3	73
KK39	AA	AB+ve	13.5	40	72	26	38	3	268	5.4	32.1	4	72
C3	AA	O+ve	6.8	20	72	18	29	2.5	120	4.1	34.5	10	59
C10	AA	O+ve	12.1	3.6	73	22	36	3.1	168	3.1	39.1	8	62
C18	AS	O+ve	12	36	68	21	37	2.1	198	4.6	35	1	84
C24	SS	O+ve	6.2	18	52	18	31	2.1	111	8.1	46.1	8	46
C31	AA	O+ve	9.8	29	66	23	34	2.6	85	7.1	36	2	78
C43	AA	B+ve	9.5	29	52	16	28	3.1	213	6.1	38.8	11	58
C50	SS	B+ve	4.1	12	52	17	29	3	118	29.4	38.6	14	36

F1	AA	A+VE	16.2	48	78	26	37	3.7	248	8.1	32.4	3	61
F11	AA	O+ve	14.3	42	73	28	37	3.7	348	6.8	36.6	6	56
F29	AA	O+ve	12	36	74	25	36	2.9	216	3.6	36.1	11	51
F32	AS	O+ve	10.5	31	64	19	31	2.8	184	8.5	33.6	5	65
F40	AA	AB+ve	10.6	32	68	25	32	2.6	262	6.8	33.6	10	68
F47	AA	A+VE	13	39	72	25	39	3	256	5.5	34.1	68	68

HB; Haemoglobin, PCV; Packed Cell Volume, MCV; Mean Cell Volume, MCH; Mean Corpuscular haemoglobin, MCHC; Mean Cell Haemoglobin Concentration, RBC; Red Blood Cell, PLC; Platelet Count, TWBC; Total White Blood Count, RDWC; Red blood Cell Distribution Width.

APPENDIX IX: Haematological parameters of some participants on the basis of genotype

Patient's ID	Age	Sex	RDT	Genotype	HB	PCV	TWBC	Eosinophil
M1	28	F	Negative	AA	10.5	32	9.3	6
M4	35	F	Positive	AA	11.4	34	5.1	9
B2	26	F	positive	AA	13	39	6.1	4
B8	25	F	positive	AA	10	30	3.1	8
BJ10	25	F	positive	AA	10.1	30	7.1	22
BJ7	25	F	positive	AA	10.2	30	8	21
BJ16	5	F	Negative	AA	15.5	35	5.1	29
BJ39	4	M	positive	AA	15.7	46	5.2	18
BJ48	37	F	Negative	AA	10.7	32	5.1	13
B30	2	F	positive	AA	12.1	36	3.4	10
M17	5	M	Positive	AA	13.8	40	10.1	2
M44	22		Negative	AA	16.2	48	4.4	17

F								
G1	5	M	Positive	AA	10.4	31	6.1	9
G6	23	F	Positive	AA	13.7	40	5.4	14
G24	30	F	Negative	AA	10.3	30	9.1	8
G39	6	M	Positive	AA	9.8	29	4.1	14
G40	30	F	Positive	AA	9.5	28	10.1	8
S1	9Months	M	Positive	AA	16.6	48	4.9	6
S20	1	M	Negative	AA	13.8	42	9.9	6
P3	2	F	Positive	AA	12.6	38	8.8	4
P25	20	F	Positive	AA	10.8	32	4.1	8
P39	25	F	Positive	AA	8.8	26	7.1	3
P50	5	F	Negative	AA	2.1	36	7.8	14
KW3	45	F	Positive	AA	7.8	23	9.6	4
KW15	16	F	Positive	AA	12.7	38	9.4	3
KW17	2	F	Positive	AA	12.1	36	8.9	3
KK5	6Months	M	Positive	AA	15.6	46	16.1	1
KK11	1	M	Negative	AA	13.6	40	8.7	5
KK49	40	F	Positive	AA	12.5	37	6.1	3
KK39	16	F	Positive	AA	13.5	40	5.4	4
C3	24	F	Positive	AA	6.8	20	4.1	10
C10	29	F	Positive	AA	12.1	36	3.1	8
C31	24	F	Positive	AA	9.8	29	7.1	2
C43	45	F	Positive	AA	9.5	29	6.1	11
T1	18	F	Negative	AA	16.2	48	8.1	3
T11	21	F	Negative	AA	14.3	42	6.8	6
T29	25	F	Positive	AA	12	36	3.6	11
T47	29	F	Positive	AA	13	39	5.5	8
B4	23	F	Negative	AS	14.1	42	7.1	6
B7	30	F	Positive	AS	9.1	27	6.1	8
BJ11	8	F	Positive	AS	12.2	36	9.1	23
M25	23	F	Positive	AS	9.5	28	6.1	3
M50	3	F	Positive	AS	11	33	5.1	4

G30	4	M	Negative	AS	11.9	35	8.1	4
S3	27		F Positive	AS	10	30	6.1	8
S17	33		F Positive	AS	9.8	28	8.1	2
S25	31		F Negative	AS	9.7	28	5.1	8
S30	5		F Positive	AS	11.3	33	6.1	15
P25	35		F Negative	AS	12.6	37	6.1	4
P7	20		F Positive	AS	9.9	29	6.1	4
KW1	25		F Negative	AS	11.3	33	3.1	2
KW40	30		F Negative	AS	11.9	35	7.1	10
KK2	28	F	Positive	AS	10.1	30	7.9	3
KK16	22		F Positive	AS	9.8	29	5.4	7
KK36	30		F Negative	AS	11.6	35	7.1	8
C18	34		F Negative	AS	12	36	4.6	1
T32	21		F Positive	AS	10.5	31	8.5	5
T40	25		F Negative	AS	10.6	32	6.8	10
BJ30	2		F Positive	AC	11.3	33	6.1	14
B33	3	M	Positive	AC	12	36	6.1	7
G22	20		F Positive	AC	10.1	30	7.1	13
G49	32		F Negative	AC	11.6	33	6.6	5
P16	35		F Positive	AC	10.1	30	6.1	8
KW30	33		F Positive	AC	12	36	6.3	9
B40	19		F Positive	SS	5.1	15	16.5	8
KW25	24		F Negative	SS	6.8	20	11.8	4
C24	3		F Positive	SS	6.2	18	15.1	8
C50	1	F	Negative	SS	4.1	12	29.4	14

AC: Sickle cell carrier
AS: Sickle cell carrier
SS: Complete sickle cell
F: Female
M: Male children

APPENDIX X: Hematological Parameters of Some Participants on the basis of ABO blood group

Patient's ID	Age	Sex	RDT	Blood group	HB	PCV	TWBC	Eosinophils
M1	28	F	Negative	O+ve	10.5	32	9.3	6
B4	23	F	Negative	O+ve	14.1	42	7.1	6
B8	25	F	Positive	O+ve	10	30	3.1	8
BJ39	4	M	Positive	O+ve	15.7	46	5.2	18
B30	2	F	Positive	O+ve	12.1	36	3.4	10
B33	3	M	Positive	O+ve	12	36	6.1	7
B40	19	F	Positive	O+ve	5.1	15	16.5	8
M17	5	M	Positive	O+ve	13.8	40	10.1	2
G6	23	F	Positive	O+ve	13.7	40	5.4	14
G24	30	F	Negative	O+ve	10.3	30	9.1	8
G40	30	F	Positive	O+ve	9.5	28	10.1	8
S1	9Months	M	Positive	O+ve	16.6	48	4.9	6
S20	1	M	Negative	O+ve	13.8	42	9.9	6
P16	35	F	Positive	O+ve	10.1	30	6.1	8
P39	25	F	Positive	O+ve	8.8	26	7.1	3
P50	5	F	Negative	O+ve	12.1	36	7.8	14
KW3	45	F	Positive	O+ve	7.8	23	9.6	4
KW17	2	F	Positive	O+ve	12.1	36	8.9	3
KK2	28	F	Positive	O+ve	10.1	30	7.9	3

KK36	30	F	Negative	O+ve	11.6	35	7.1	8
KK49	40	F	Positive	O-ve	12.5	37	6.1	3
C10	29	F	Positive	O+ve	12.1	36	3.1	8
C18	34	F	Negative	O+ve	12	36	4.6	1
C24	3	F	Positive	O+ve	6.2	18	15.1	8
C31	24	F	Positive	O+ve	9.8	29	7.1	2
T11	21	F	Negative	O+ve	14.3	42	6.8	6
T29	25	F	Positive	O+ve	12	36	3.6	11
T32	21	F	Positive	O+ve	10.5	31	8.5	5
M4	35	F	Positive	A+ve	11.4	34	5.1	9
B2	26	F	Positive	A+ve	13	39	6.1	4
B7	30	F	Positive	A-ve	9.1	27	6.1	8
BJ48	37	F	Negative	A+ve	10.7	32	5.1	13
G1	5	M	Positive	A+ve	10.4	31	6.1	9
P2	32	F	Negative	A-ve	12.6	37	6.1	4
KW1	25	F	Negative	A+ve	11.3	33	3.1	2
KW15	16	F	Positive	A+ve	12.7	38	9.4	3
KK11	1	M	Negative	A+ve	13.6	40	8.7	5
C3	24	F	Positive	A+ve	6.8	20	4.1	10
T1	18	F	Negative	A+ve	16.2	48	8.1	3
T47	29	F	Positive	A+ve	13	39	5.5	8
BJ10	25	F	Positive	B-ve	10.1	30	7.1	22
BJ11	8	F	Positive	B+ve	12.2	36	9.1	23
BJ7	30	F	Positive	B-ve	9.1	27	6.1	8
BJ16	5	F	Negative	B+ve	15.5	35	5.1	29
BJ30	2	F	Positive	B+ve	11.3	33	6.1	14
M25	23	F	Positive	B+ve	9.5	28	6.1	3
M44	22	F	Negative	B+ve	16.2	48	4.4	17
G30	4	M	Negative	B+ve	11.9	35	8.1	4
G39	6	M	Positive	B+ve	9.8	29	4.1	14
G49	32	F	Negative	B+ve	11.6	33	6.6	5
S3	27	F	Positive	B-ve	10	30	6.1	8
S17	33	F	Positive	B+ve	9.8	28	8.1	2
S25	31	F	Negative	B+ve	9.7	28	5.1	8
S30	5	F	Positive	B+ve	11.3	33	6.1	15
P3	2	F	Positive	B+ve	12.6	38	8.8	4
P7	20	F	Positive	B+ve	9.9	29	6.1	4
P25	20	F	Positive	B+ve	10.8	32	4.1	8
KW25	24	F	Negative	B+ve	6.8	20	11.8	4
C43	45	F	Positive	B+ve	9.5	29	6.1	11
C50	1	F	Negative	B+ve	4.1	12	29.4	14
KW30	33	F	Positive	B+ve	12	36	6.3	9
KW40	30	F	Negative	AB+ve	11.9	35	7.1	10

kk5	6Months	M	Positive	AB+ve	15.6	46	16.1	1
KK16	22	F	Positive	AB+ve	9.8	29	5.4	7
KK39	16	F	Positive	AB+ve	13.5	40	5.4	4
T40	25	F	Negative	AB-ve	10.6	32	6.8	10
M50	3	F	Positive	AB+ve	11	33	5.1	4
G22	20	F	Positive	AB+ve	10.1	30	7.1	13

APPENDIX XI: Ethical Approval to conduct research



NIGER STATE HOSPITALS MANAGEMENT BOARD GENERAL HOSPITAL MINNA

ADDRESS:

No 1 Hospital Road,
P.M.B 2 Minna,
Niger State,
Tel: 08138105225

E-mail: genhosp.minna@yahoo.com

website: www.generalhospitalminna.com

10th June, 2019

HMB/GHM/STA/136/VOL.IV/548

The Head of Department,
Microbiology,
Federal University of Technology,
Minna.

Through:

Ajanya Benjamin Unekwujo
Microbiology Department,
Federal University of Technology,
Minna.

Sir,

AJANYA BENJAMIN UNEKWUOJO (M TECH/SLS/2017/6780)
RESEARCH, ETHICS AND PUBLICATION COMMITTEE'S
APPROVAL TO CONDUCT RESEARCH

The bearer a student of your department sought for permission to conduct research on "Parasitological and haematological Profile of Women and Children Infected with Plasmodium in Bosso and Paikoro Local Government Areas of Niger State".

The committee after going through his proposal has given him the approval to conduct the research.

A copy of his final findings must be submitted to the committee as a pre-requisite to this approval.

Thank you for your cooperation



Amuda O. Amuda BDS, Mph, Msc, Phd
Chairman Research ethics and publication committee



PAIKORO LOCAL GOVERNMENT

Paikoro Local Government Secretariat,
Paiko, Niger State



Telegram _____

Telegram No _____

Your Ref: _____

Ref No. _____

Date 22-08-2019

PRIMARY HEALTH CARE
DEPARTMENT

THE INCHARGE
PRIMARY HEALTH CARE CENTRE
KWAKUTI.

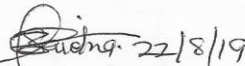
Sir,

LETTER OF INTRODUCTION

The above department wish introduce to you a student on research work from Federal University of Technology Minna to enable him meet up with the fulfillment to obtain his master's degree.

I solicit for your cooperation and needed assistance to him.

Thanks for usual cooperation.



ILIASU IBRAHIM BAIDNA
Monitory and Evaluation officer
For Director PHC

*Ajanya Benjamin unekwajofa
reported to our clinic on 15/9/2019
and he was receive by
Shuaib Mohammed WFP
Kwalati ward. 20/9/2019*

BOSSO LOCAL GOVERNMENT COUNCIL
PRIMARY HEALTH CARE DEPARTMENT



OFFICE:
Local Gov't Secretariat Complex
P.M.B 153,
Maikunkele, Niger State.
066/221072

Our Ref: B/LG/PHC/M&E/FUT/2019
Your Ref: _____

INTRODUCTION LETTER

1/c PHCCB/51

Date: 27/5/19

RE-AJANYA, BENJAMIN UNEKWUOJO

1. I write to introduce the above named student of FUT with Reg. no Mtech/SLT/2017/6780. He is on field research to Bosso LGA and will need some research information and data from your health facility.
2. Please give him the necessary support and adequate co-operation towards this work.
3. You may also need to contact the office in any area you are not clear with in this exercise.

9/7/2019

Received

Student Affairs Officer

PHCC B&E

[Signature]

Sign [Signature]

M&E PHC, Bosso L.G.A