

UNIVERSITY OF CAPE COAST

MALARIA: EPIDEMIOLOGY AND HERBAL TREATMENT OPTIONS IN
SOME SELECTED AREAS OF CENTRAL REGION OF GHANA

BY

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DEGREE IN BOTANY.

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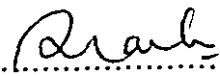
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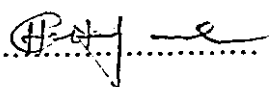
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ABSTRACT

Incidence of malaria in the Central Region of Ghana was investigated to determine age group and sex vulnerability as well as the parasite species distribution, haemoglobin status of patients and herbs used in treating the disease. Four Hospitals were used as study centers namely, Cape Coast District Hospital and the University of Cape Coast Hospital constituting the coastal zone; Saint Francis Xavier Hospital at Assin Foso and Our Lady of Grace Hospital at Breman Asikuma forming the forest zone.

Microscopic examination of thin blood smears of the patients examined revealed high malaria incidence rates of 34.4% and 26.9% of coastal zone and forest zone respectively. Generally, children up to 10 years old and females were more vulnerable to the disease. In the forest zone, older females were more vulnerable. *Plasmodium falciparum* was the most prevalent malaria parasite (>90%) infecting people in the region. Few cases of *P. malariae*, *P. ovale* and mixed infection of *P. falciparum*+ *P. malariae* were recorded. *P. vivax* was not encountered. The most predominant erythrocytic stage identified was trophozoites (92.1%) and few (7.9%) gametocytes and schizonts observed.

The haemoglobin levels of patients decreased with increase in parasitaemia and subsequently anaemia with haemoglobin levels <11g/dL.

A total of 89 plant species distributed into 41 families was documented as anti malaria plants in the region. Among these, 60 species in 36 families were mentioned for the first time as anti malaria plants. All the plants recorded in this study were reported to be abundant except one species.

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DEDICATION

To my children, Dennis and Bennet.

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LIST OF ABBREVIATIONS AND THEIR MEANINGS

Abbreviation	Meaning
CCDH	Cape Coast District Hospital
DALY	Disability Adjusted Life Year
DDT	Dichlorodiphenyltrichloroethane
GPRS	Growth and Poverty Reduction Strategy
HRP2	Histidine Rich Protein II
ICT	Immunochromatography
MVI	Malaria Vaccine Initiative
OA	Acridine Orange
OLGH	Our Lady of Grace Hospital
OPD	Out Patient's Department
PCI	Project Concern Initiative
PLDH	<i>Plasmodium</i> Lactate Dehydrogenase
QBC	Quantitative Buffy Coat
SFXH	Saint Francis Xavier Hospital
TBAs	Traditional Birth Attendants
TDR	Tropical Disease Research Programme
UCCH	University of Cape Coast Hospital
UNICEF	United Nations Initiative on Children Education Fund
WHO	World Health Organization

CHAPTER ONE

INTRODUCTION AND LITERATURE REVIEW

Background

Malaria is a major global public health problem especially in developing and underdeveloped countries and has been recognized as an important parasitic disease of humans for centuries (WHO, 1996; 1997). Despite the introduction of control programmes in many parts of the world over the past decades, impact of malaria on human populations continues to increase more especially throughout much of the tropics and subtropics (WHO, 1998; de Souza and Riley, 2002; Sachs, 2002; Sachs and Melaney, 2002; Korenromp *et al.* 2003).

Estimates suggest that about 1.5 billion persons live in areas of the world where malaria is an endemic disease (WHO, 1998; Garvey, 2006), and the number of infected humans exceeds 500 million (Oaks *et al.* 1991; WHO 2000b; MVI, 2003a; Mercola, 2004). About 1.5 – 2.7 million persons die from malaria each year (Cheng, 1986; WHO, 1996; 1997; 2004; MVI, 2002; Ahorlu *et al.* 2006) and a child dies every 15 – 20 seconds (Oaks *et al.* 1991; WHO, 2000b; Mercola, 2004; MVI, 2003b; Garvey, 2006). The effect of the disease threatens public health productivity on a broad scale and impedes the progress of many countries toward democracy and prosperity (Oaks *et al.* 1991; Lederberg *et al.* 1992; MVI, 2002; Sachs and Melaney, 2002). Young children and pregnant women are more

vulnerable to the disease. Expectant mothers are more susceptible and may suffer miscarriage or premature labour. Malaria also poses a risk to travelers and immigrants and Goodman *et al.* (1999) reported that imported cases were increasing in non-endemic areas.

As indicated earlier, malaria has immense global importance. It is the most prevalent vector-borne disease in the world threatening some 2.4 billion people constituting 40 percent of the world's population (MVI, 2002; Sachs and Melaney, 2002). Malaria has many manifestations and its impact varies depending on the epidemiological setting. As shown in figure 1, the disease exists in five continents (Folasade, 2003), and involves about one hundred countries but is mainly confined to poorer tropical areas of Africa, Asia and Latin America, certain Caribbean Islands, Islands of the South, West and Central Pacific Ocean and Turkey (Folasade, 2003). Malaria generally occurs in areas where environmental conditions allow parasite multiplication in the vector. Thus, malaria is usually restricted to tropical and subtropical areas and altitudes below 1,500 metres. However, this distribution might be affected by climatic changes, especially global warming and population movements. Both *Plasmodium falciparum* and *P. malariae* are encountered in all shaded areas of the map (with *P. falciparum* by far the most prevalent). *P. vivax* and *P. ovale* are traditionally thought to occupy complementary niches, with *P. ovale* predominating in Sub-Saharan Africa and *P. vivax* in other areas (Crutcher and Hoffman, 2003).

More than 90 percent of malaria deaths occur in Tropical Africa and *P. falciparum* is the main cause of severe clinical malaria and death (TDR, 1997;

MVI, 2002). The public health importance of malaria in Africa is evidenced by the degree of morbidity and mortality among children and pregnant women throughout tropical Africa (Greenwood *et al.* 1987; 1991; WHO, 1992; 2000a; Binka *et al.* 1994; Afari *et al.* 1995; Goodman *et al.* 1999) with about 800,000 of African children dying every year (MVI, 2003b; GPRS II, 2005; Garvey, 2006).

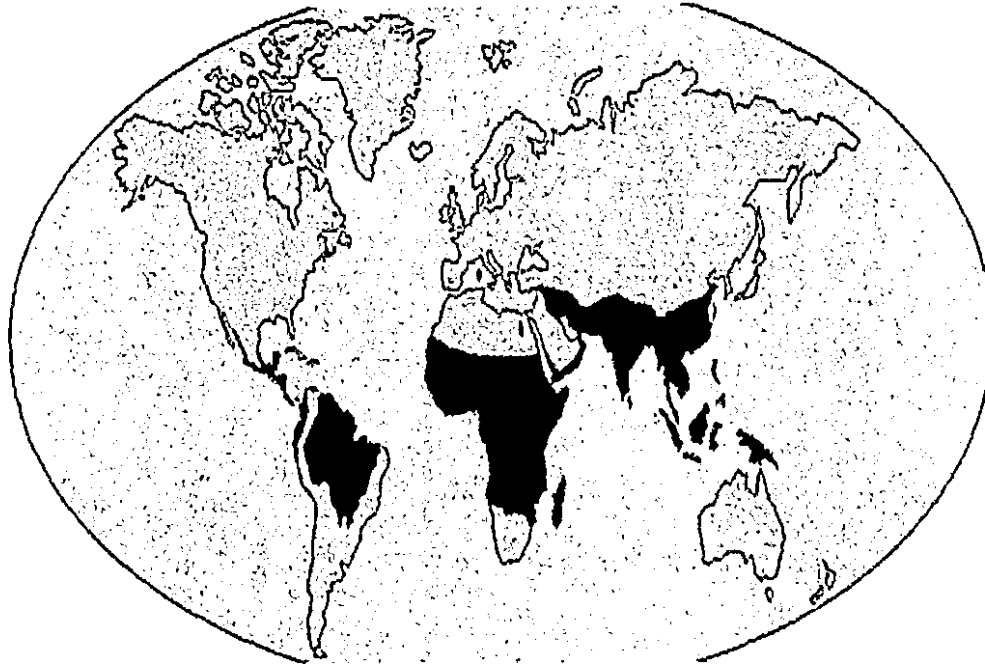


Fig. 1: Global distribution of malaria.

(Source: Centers for Disease Control and Prevention, 2004)

Plants have provided man with all his needs in terms of shelter, clothing, food, flavors and fragrances as well as medicines. Plants have formed the basis of sophisticated traditional medicine systems, which have given rise to some important drugs in use today (Gurib-Fakim, 2006). In Africa, the practice of traditional medicine still requires considerable improvement when compared with

situations in India and China. The increasing debt of African nations and the increasing cost of modern health care make the role of traditional healthcare delivery more important to the African population which lives in rural areas (Sofowora, 1993). Even people in the urban areas and from the developed countries go back to nature for their health care. The recent trends and development in medicinal plant research and development of traditional medicine in Africa, are based on the sound recognition of the role that traditional medicine is already playing in health care programmes in most developing countries especially in Africa, Asia and Latin America.

Malaria disease has become very critical and widespread and one of the main reasons for this is that the anti-malarial drugs, including chloroquine, are no longer effective against the disease as their efficacy have been decreased by the spread of drug-resistant strains of the parasite. This loss in efficacy has been a major barrier to the treatment of malaria and has posed an urgent challenge to discover new anti-malarial drugs especially in plants.

The WHO and UNICEF in 1978 as cited by Oku-Ampofo (1992), gave recognition to the peculiar circumstances regarding traditional medicines and healthcare delivery. These organizations resolved among other issues that member states should initiate comprehensive programmes for the identification, evaluation, cultivation and conservation of medicinal plants. This makes the inclusion of plant inventory in the study of diseases imperative.

The Disease and Symptoms

Four species of the genus *Plasmodium*: *P. ovale*, *P. falciparum*, *P. malariae* and *P. vivax* infect humans and cause malaria. The symptoms of malaria are very well known and have been described by various authors (eg. Russell *et al.* 1963; Schmidt and Roberts, 1985). According to these authors the classical malarial fever has 3 stages. Cold stage (shivering) lasts for 2 hours; hot stage lasts for 3-4 hours and sweating stage lasts for 2-4 hours. These symptoms occur at regular intervals i.e show periodicity. Periodicity becomes apparent only after the first few days of illness and depending upon the species it could be tertian (every 2nd day for *vivax*, *falciparum* and *ovale*) or quartan (every 4th day for *malariae*). However, these clinical symptoms are not present in many patients who have the infection. Malaria, especially the type caused by *P. falciparum* is a very variable disease mimicking many other conditions such as typhoid, meningitis, and gastroenteritis etc (Trampuz *et al.* 2003). Symptoms due to malaria depend upon the age, immune status, intensity of transmission and prevalent species of malaria parasite. In those living in non-endemic areas, symptoms are very vague because, most of the people are non immune. The disease often presents like flu. Presence of rigors and a rapid increase in temperature is an indicator of the possibility of malaria. In those living in endemic areas, the attacks are modified by immunity. Fever is often intermittent and may have periodicity. In children, fever due to malaria is variable and has no periodicity. It is mostly irregular, may be high and continuous or low grade. Other manifestations in children are pallor, nausea, vomiting, and refusal of feeds, lethargy, restlessness, headache, diarrhoea and

unproductive cough (Russell *et al.* 1963; Schmidt and Roberts, 1985; Stauffer and Fischer, 2003; Wattanakoon *et al.* 2003). Severe forms of malaria are seen with *P. falciparum* species which can progress to potentially fatal forms with central nervous system involvement (cerebral malaria), acute renal failure, severe anaemia (Armah *et al.* 2007), or adult respiratory distress syndrome or pulmonary edema, inability to sit up without help, impaired consciousness, seizures, circulatory collapse, abnormal bleeding, jaundice, haemoglobinuria or severe anaemia (haemoglobin less than 5.0 g/dL, or hematocrit less than 15%). The following symptoms have also been described by the above authors: Prostration and altered consciousness occur frequently in both children and adults with severe disease. Respiratory distress, seizures and severe anaemia are more common in children, whereas renal failure and jaundice occur more frequently in adults. Acute respiratory distress syndrome, an immune-mediated complication, often occurs during the second to fourth day of treatment, even when parasitaemia is decreasing. Severe malaria usually occurs with parasitaemia of 5% or more, and even with optimal management, the mortality rate exceeds 20% (Russell *et al.* 1963; Schmidt and Roberts, 1985; Stauffer and Fischer, 2003; Wattanakoon *et al.* 2003). At highest risk of complications from malaria are non-immune people, children and pregnant women who live in endemic areas. Complications generally involve the central nervous, pulmonary, renal and hematopoietic systems. Hypoglycemia occurs because of parasite consumption of glucose and treatment with quinine could also be a factor. Another common metabolic derangement associated with the disease is acidosis. Bacterial infection may occur as a

complication of malaria itself (e.g., aspiration pneumonia). One of the most serious complications is cerebral malaria, manifested by altered level of consciousness, focal neurologic findings and seizures. Mortality is high (15% to 25%), and survivors may have residual neurologic deficits (WHO, 2000c). Severe malaria occurs when *P. falciparum* infections are complicated by serious organ failures or abnormalities in the patient's blood or metabolism (Boivi, 2002). Complications of *P. vivax* malaria include splenomegaly (with, rarely, splenic rupture), and those of *P. malariae* include nephrotic syndrome (Bruce-Chwatt, 1985; Aikawa, 1988; Greenwood *et al.* 1987; 1991; Garvey, 2006). Some of the manifestations are severe vomiting and diarrhea, cerebral malaria, algid malaria, hepato renal syndrome, black water fever and malaria shock lung (Banzal *et al.* 1999).

Malaria and Anaemia

Anaemia is a common manifestation of all types of malaria and poses a problem for pregnant women and children (Kakkilaya, 2006). Anaemia in malaria has been described as multifactorial. The causes include obligatory destruction of red cells at merogony, accelerated destruction of non-parasitised red cells which is a major contributor in anaemia of severe malaria, bone marrow dysfunction that can persist for weeks, shortened red cell survival and increased splenic clearance. Massive gastrointestinal haemorrhage can also contribute to the anaemia of malaria (Kakkilaya, 2006). Anaemia therefore can be described as a condition in which the red cells of the blood (erythrocytes) are reduced in number

or volume or are deficient in haemoglobin, the oxygen carrying pigment. There are about 100 different varieties of anaemia distinguished by the cause, the size and haemoglobin content of the abnormal cells, and symptoms (The New Encyclopaedia, 2003). In falciparum malaria, anaemia can develop rapidly due to severe haemolysis and the degree of anaemia has been found to correlate with parasitaemia and schizontemia (The New Encyclopaedia, 2003). Haemoglobin is an iron-containing protein of the blood that binds oxygen in the lungs and transports it to tissues in the body. Normal haemoglobin level of an individual is relative to age, sex and even race (Crawley, 2004). In males, the normal haemoglobin ranges from 14-16g/dL, and 12-14g/dL for females (Crawley, 2004). Children may have severe anaemia even with low parasitaemia and in such cases the reticuloendothelial cells exhibit abundant malarial pigments (Kakkilaya, 2006).

Modes of Transmission

All the four species of *Plasmodium*, the causative agent of malaria are vector-borne and spread by anopheline mosquitoes (Coatney *et al.* 1971; Miller *et al.* 1986). Inoculation is through the bite of infected blood feeding female mosquito of the genus *Anopheles* which breeds in swamps and marshes (Brown, 1970) and other stagnant freshwater bodies. The mosquitoes transfer parasites from human to human; however, male anopheles mosquitoes do not bite (TDR, 1997). About 60 species of mosquitoes are possible vectors for the disease under natural conditions. Malaria can also be transmitted by blood transfusions from infected persons and through contaminated needles and syringes. Blood

malaria parasites. Malaria parasites can also be transmitted congenitally where infected mothers transmit parasites to their children before or during birth (Riley *et al.* 2000).

Life Cycle of Malaria Parasite

The life cycle of the malaria parasite alternates between the mosquito vector, the intermediate host and humans, the definitive host (Fig.2). These two hosts are also referred to as invertebrate and vertebrate hosts respectively in view of the controversy regarding their status in terms of stage of the parasite's life cycle they harbour.

Malaria Parasite in Mosquito

When a female *Anopheles* mosquito bites a human host harbouring malaria parasite, it ingests the parasites along with the blood meal. The asexual forms are digested, while the mature sexual forms called gametocytes undergo further development in the mosquito. In the male gametocytes the nucleus divides into 4-8 nuclei, each forming thread like structures called microgametes. Female gametocytes undergo maturation process and form macrogametes. In the mosquito's stomach, microgamete fuses with the macrogamete (fertilization) resulting in a product called zygote. Within 18-24 hrs, the zygote develops into a long mobile worm-like form called the ookinete. The ookinete passes between epithelial cells to the outer surface of the mosquito's stomach wall and becomes rounded up into a small sphere called oocyst. The oocyst increases in size and the nucleus divides repeatedly to form sporoblast. The divided nuclei of the

sporoblast form elongated sporozoites and are released into the body cavity. These sporozoites migrate to the salivary gland of the mosquito and are now ready for transmission to human host. When the mosquito feeds on blood, sporozoites are released into the bloodstream of the human host.

Malaria parasite in human

The parasite undergoes two distinct phases of development in the human body.

Exo-erythrocytic schizogony or tissue phase

Sporozoites inoculated by a mosquito into the human host circulate for about half an hour during which many are destroyed by the phagocytes. Some sporozoites enter the parenchymal cells of the liver and undergo a process of development known as pre-erythrocytic schizogony. During pre-erythrocytic schizogony (In *P. falciparum* and *P. malariae*,) sporozoites develop into schizonts. In *P. ovale* and *P. vivax*, within 40-48 hrs, after an infective bite, sporozoites shrink and form narrow cytoplasmic rim structures called hypnozoites. Hypnozoites remain dormant in hepatocytes and then grow into schizonts by a process known as delayed schizogony.

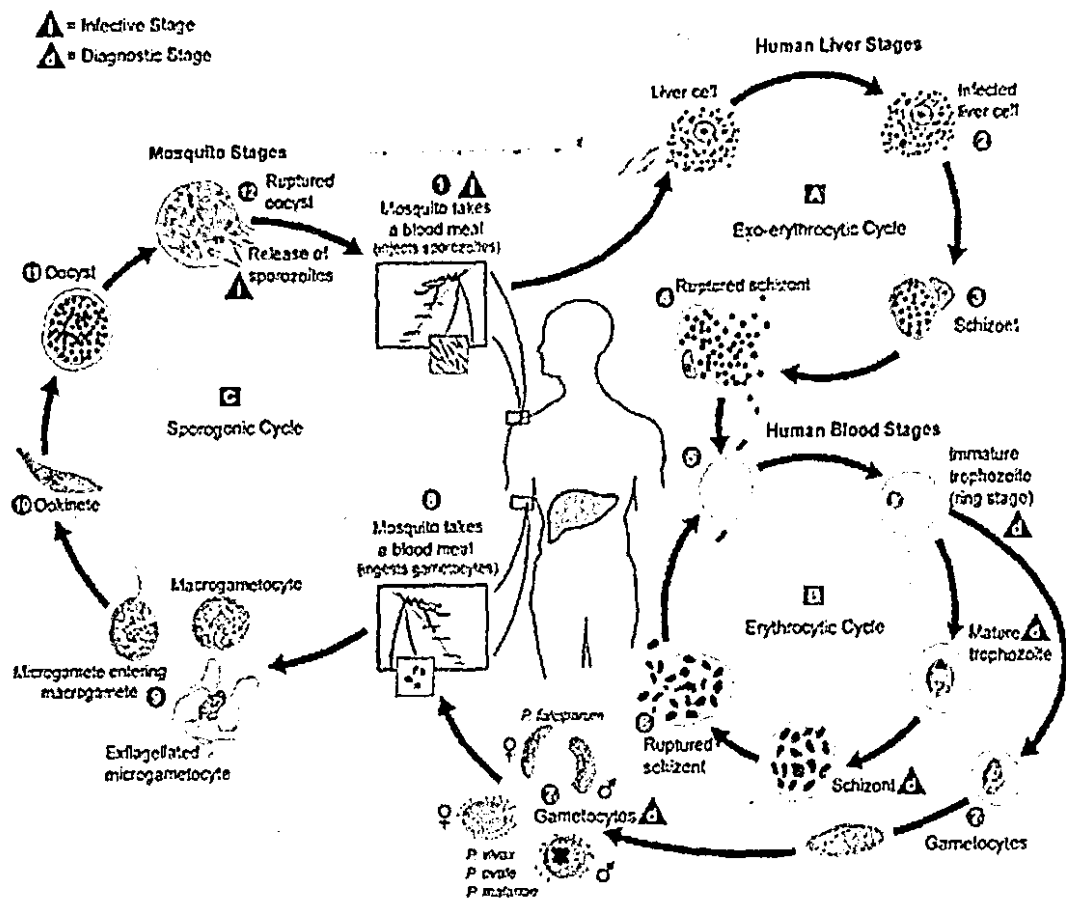


Fig. 2. Life cycle of the *Plasmodium* parasite

(Source: Centres for Disease Control and Prevention, 2004)

After about 6 to 16 days from the time of infection, mature schizonts enlarge and burst to release thousands of merozoites into the blood. Merozoites re-enter fresh liver cells and repeat the process of schizogony, thus causing relapses of infection during the secondary exo- erythrocytic schizogony.

Erythrocytic schizogony

Merozoites released from the tissue schizont enter red blood cells (RBCs) in five stages: i. Initial recognition and attachment. ii. formation of junction, iii. creation of vacuole membrane continuous with the red cell membrane, iv. entry into the vacuole through the moving junction, v. and sealing of the erythrocytes after entry (Boampong *et al.* 2007).

After entering the erythrocytes, merozoites assume rounded forms. These youngest stages of parasites in RBCs are rounded bodies with annular appearance called ring forms. The ring forms grow into irregular shapes called trophozoites. The parasite lives on cytoplasm of the RBC, absorbing haemoglobin and leaves a product of digestion, a pigment called haemozoin (combination of haematin with protein). After a period of growth, trophozoites divide asexually forming schizonts by a process known as erythrocytic schizogony. Mature schizonts divide into small round forms called merozoites. When the process of schizogony is completed the red blood cell bursts, releasing the merozoites into the blood stream. They invade fresh erythrocytes and repeat the erythrocytic schizogony cycle. After several rounds of erythrocytic schizogony, some merozoites develop into sexual forms, the gametocytes. The number of gametocytes increases with increasing number of erythrocytic schizogony. According to Brown (1970), after a human accumulates a billion or more of *Plasmodium* individuals, the production of gametocytes begins and the human now may infect mosquitoes.

Diagnosis

Two main approaches are used in diagnosing malaria, namely microscopic and non-microscopic.

Microscopic diagnosis

A. Blood films

Malaria is diagnosed by making blood smears stained with Giemsa and examining through microscope using 100x oil immersion objective. Microscopy is sensitive, can detect densities as low as 5-10 parasites per microlitre of blood. When parasites are found, their species and circulating stage can also be seen. Examination of a thick blood film as the first step has the advantage of concentrating the parasites by twenty folds although parasites may appear distorted. This is the one for quantification. The presence of the parasite relating to the severity of parasitaemia is reported as:

+ = 1 to 10 parasites per 100 microscopic fields;

++ = 11 to 100 parasites per 100 microscopic fields;

+++ = 1 to 10 parasites per 1 microscopic field;

>++++ = more than 10 parasites per 1 microscopic field.

Thin blood film examination affords the clear identification and stage of the parasite species involved (Gilles and Warell, 1993).

B. Buffy coat (QBC; Becton Dickinson) method.

This involves centrifuging the patient's blood in special capillary tubes precoated with Acridine Orange (OA). A small precision moulded plastic float presses the parasitized red cells against the wall of the tube where they can be viewed by ultra violet light microscopy. The sensitivity of this method is claimed to be very high however the young trophozoites of *P. falciparum* and *P. vivax* cannot be distinguished with any degree of certainty and that confirmatory blood films should be examined.

In recent years however, a number of techniques have been employed for the diagnosis of human malaria.

Non-microscopic diagnosis

A. Detection by Polymerase Chain Reaction (PCR)

This method uses a non isotopically labeled probe following PCR amplification. It is possible to detect less than ten parasites per 10 microlitre of blood and may prove to be a valuable addition to the examination of blood films for the diagnosis and speciation of malaria.

B. The fluorescent antibody test for malaria

Antibodies to malaria can be detected using enzymatic immunoassays or immunofluorescence techniques. The antibodies to the asexual blood stages appear days to weeks after the infection and may persist for months. The method

is very useful in survey work and for screening blood donors. It also reduces wastage. However, it is of little value in the acute malaria situation.

C. Detection by ICT-Malaria Pf and; OptiMALr and Kat-Quick kits

ICT method

The method is based on the principle of the detection of plasmodial histidine rich protein-2 (PfHRP-2). The protein is a constituent of the knobs on the membranes of red blood cells which are infected by *Plasmodium falciparum* and makes use of primary and secondary antibodies specific for the PfHRP-2 antigens which are attached to a paper strip. One of the antibodies is coupled to a colloidal gold and applied to where blood sample is to be applied. The secondary antibody is fixed elsewhere on the strip in a band where test result is read. A positive blood sample when applied forms an antigen-antibody complex yielding a clear purple band on the strip. The test has 90-95% sensitivity for parasitaemia of more than 100 parasites per microlitre of blood.

OptiMALr method

The "OPTiMAL" test is based on the detection of parasite specific lactate dehydrogenase (pLDH), which is present in *P. falciparum* infections. It consists of a dipstick coated with monoclonal antibodies to pLDH. Differentiation of the parasite species is based on antigenic differences between various pLDH isoforms. The pLDH is only produced by live parasites and also by gametocytes. Specificity and sensitivity of the test varies depending on the erythrocytic stage

and may be positive by the presence of circulating gametocytes even when patients have been clinically cured of trophozoites and schizonts.

D. The dipstick method

The method has the potential of enhancing the speed and also the accuracy of diagnosing *P. falciparum*, the dipstick kit is found to be very useful for screening or confirmatory tests especially when there is difficulty in identifying scanty ring forms in blood films. However, dipstick methods are unable to indicate parasite load. The potential problem with this method is that the circulating antigen may be detected for many days after the elimination of viable parasites from circulation.

Problems with diagnosis and cure

The popular use of self medication as a first choice of action for treating malaria hampers the effective and efficient diagnosis and treatment of the malaria disease (Swartout, 1951; Adasi, 2005). Parasites tend to develop resistance to low dosages of drugs and poor time management. They also hide in the tissues and are not seen in peripheral blood making it difficult for diagnosis and effective treatment. Particularly in Ghana, a major obstacle to effectively implement the WHO malaria control strategy is due to the inappropriate and inadequate doses of anti-malarials given at home. The final recourse after the sickness has failed to respond to home treatment is the formal sector (Ahorlu *et al.* 1997), and this results in delays in effective treatment and development of drug resistant strains.

Plants as sources of anti-malarial drugs

Herbs are effective in disease prevention as well as cleansing the body of harmful substances. All human beings need some help for their many diseases and illnesses and this help comes from nature-herbs (Abukari-Sadik, 2007).

Out of the six tropical diseases of WHO's a TDR programme, malaria is the one that takes the largest toll of human life in Africa (Sofowora, 1993) this is due to the resistance of the *P. falciparum* species to chloroquine in many countries. This awareness has encouraged the increase in the search of anti-malarials from African plants. As a result, new drugs and drug combinations are needed. In some parts of the world where antimalarial drugs are failing due to resistance, or are not available to everyone, people often turn to traditional herbal remedies instead. The development of medicinal plants of Africa will naturally encourage more trading in the commodity and is necessary therefore, for quantitative pharmacognostical analysis to be carried out on some African medicinal plants and to recommend standards to be used in their quality control (Sofowora, 1993).

Within the context of traditional practice, malaria (and/or malaria symptoms) is commonly treated by decoctions or infusions from bitter plants and are all derivatives from quinine, a substance which is extracted from the bark of the cinchona tree which is native to South America.

A number of medicinal plants abound in Ghana's flora and most of them are used in the crude form and in various doses. Some are used whole, crushed,

powdered forms, decoctions, dried extracts, infusions, poultices and tinctures. Many of these plants have already been investigated and they are being used while many more are yet to be scientifically investigated. The formal documentation of Ghana's herbal heritage has been lacking (Oku Ampofo, 1992)

Ghanaian medicinal plants have been passed on over the years through oral traditions from one herbalist to another. In the midst of the rapid destruction and degradation of our environment, the loss of useful medicinal plant species is obvious. The passing away of the custodians' knowledge and the lack of documentation pose a threat to Ghana's traditional medicine heritage. Thus, it is worth documenting the ethnobotanical data, and testing the antiplasmodial activity of the extractives from plants (Randrianariveლოსია *et al.* 2003), and in Ghana, it is very incumbent on Ghanaian scientists and laymen to document information on various medicinal plants, most importantly on anti-malarials, since malaria kills about 15 people each day in Ghana (Dr. Azeez, U.C.C. Hospital, Pers.communication). This documentation therefore should consider the distribution, status and conservation for posterity.

In this study therefore ethno-botanical data was compiled in the Central Region of Ghana on plants which are used as anti-malaria drugs.

Malaria situation in Ghana

In Ghana malaria is the single most important cause of mortality especially among children under five years and pregnant women (Adasi, 2005; Ghana Ministry of Health, 1999; GPRS II, 2005). In 2002, malaria was estimated

to account for 44.5% of all out patient illnesses and 36.9% of all admissions and 13.2% of all deaths in health facilities in the country (PCI, 2003; GPRS 1, 2001; GPRS II, 2005). The disease is responsible for a substantial number of miscarriages and low birth weight babies (GPRS II; 2005). It is estimated that a single bout of malaria costs an amount equivalent to over 10 working days in Africa. According to GPRS II (2005), malaria in Ghana accounts for a significant portion of the disease burden, causing about 10.6% of lost Disability Adjusted Life Year (DALY) and costing an equivalent of about 3% of GDP annually in economic terms. The cost of treating a single bout of malaria stands at \$39. Ghana's infants, child and maternal mortality are among the worst 25% of all countries in the world. Malnutrition and the preventable childhood diseases of malaria, measles, pneumonia and diarrhoea claim the lives of 70% of children who die before the fifth birthday each year. Studies conducted by Dugbartey *et al.* (1998) concluded that the long-term emotional and cognitive effects of malaria infection in non migratory Ghanaian adults without a lifetime medical diagnosis of malaria showed the presence of an enduring but albeit sub-clinical, mixed anxiety depression syndrome after medical recovery from *falciparum* malaria. A community study in Ghana by Mitchell *et al.* (1986) revealed that malaria may be a risk factor for psychiatric morbidity and is also concerned with socio-cultural determinants of childhood morbidity and mortality. About 27 % of untreated cases develop permanent neurological damage (Koram *et al.* 2003). Owusu-Agyei *et al.* (2000) reported that in Kintampo area the incidence of clinical malaria (any level of parasitaemia with recorded fever or reported fever) was high with about

eight episodes per child per year among children under five years. More than 50% of all children less than 10 years were anaemic (haemoglobin level <11g/dL) with the prevalence of severe anaemia (haemoglobin <8g/dL being (12%). Severe anaemia resulted from repeated episodes and affected the growth and intellectual development of the child (Koram *et al.* 2003).

Treatment and preventive methods in Ghana

An effective treatment for malaria was known long before the cause of the disease was understood. The bark of the cinchona tree, whose most active principle is quinine, was used to alleviate malarial fevers from 1700 until World War II, when more effective, synthetic drugs were developed.

An effective treatment for malaria in Ghana is by chemotherapy. Chief among the drugs are chloroquine, primaquine, pyrimethamine, artesunate and amodiaquin, all of which can destroy the malaria parasites while they are living inside red blood cells. Chloroquine and related drugs could wipe out the *Plasmodial* infection entirely until recently when strains of the parasites developed resistance. Currently, combination of drugs like Artesunate-Amodiaquin is being used to treat the disease. Due to widespread poverty, various parts of different plants serve immense purpose for the treatment of malaria in Ghana. Popular among the rural areas where health facilities are not available, infusions and concoctions prepared from many different plant parts serve as rich remedy against the disease (Agyepong, 1992; Ahorlu *et al.* 1997). However, since malaria disease mimics a variety of other ailments, some people

term it as spiritual and resort to healing by Divine intervention thereby causing a delay in the diagnosis and effective treatment of the disease which results in high mortality rate.

Prevention of the disease is also of paramount concern in the country. The basic methods of prevention are to eliminate the breeding places of *Anopheles* mosquitoes by draining and filling marshes, swamps, stagnant pools, and other large or small bodies of standing fresh water. DDT, dieldrin, and other less toxic insecticides have proved potent in controlling mosquito populations in affected areas. Window screens and treated mosquito netting are widely in use to secure interior spaces from the mosquitoes, which are mainly active at night. Mosquito repellent creams are also used as a protective measure.

Statement of the research problem

Efforts by the World Health Organization to eradicate malaria in developing countries have so far proven unsuccessful. The growing problem of drug resistant *Plasmodium* parasites makes adequate treatment of malaria increasingly difficult. Hence more and more money continues to be spent to develop new drugs to keep pace with the rate of development of new strains of the parasite.

In the absence of functional, safe and widely available vaccine, efforts to develop new strategies to attack the parasite are extremely important so that increased incidence of the disease can be prevented. In view of the difficulty in eradicating the disease, there is the need to conduct further studies to understand better the

biology of the parasite, the epidemiology of the disease and alternative treatment options. Research questions should therefore include the following:

- i. What is the incidence rate of the disease?
- ii. Are there any age and sex differences with regard to the incidence of the disease?
- iii. What is the dominant *Plasmodium* species, and does the dominance exhibit seasonal variation?
- iv. Does the haemoglobin status of malaria patients vary in terms of parasite density and gender?
- v. What types of herbs are used in the treatment of the disease?

Justification

Ghana is a malarious country and being a developing one suffers a heavy economic burden imposed by the disease. Further research into the malaria disease will be of immense benefit to the global community as well as the tropical regions where environmental conditions allow parasite multiplication in the vector. Previous studies conducted on malaria all aiming at helping to control the disease in various parts of Ghana include Commey, 1989; Meima, 1989; Adiama *et al.* 1993; Afari *et al.* 1993; Koram *et al.* 1995; Wagner *et al.* 1998; McGuinness *et al.* 1998; Kurtzhals *et al.* 1999; Dunyo *et al.* 2000; Riley *et al.* 2000; Afenyadu *et al.* 2005; and Chandramohan *et al.* 2005.

Two previous preliminary studies have shown that in the Central Region of Ghana, children under age of five and the productive age groups of 21-45 are

the most vulnerable groups (Safua-Appenteng, 1994; Ayirebi-Akomea, 1993). This finding emphasizes the economic hardship that is brought to the nation by the disease.

The Central Region of Ghana with its diversity of climatic conditions and habitats is faced with more serious threat from the *Plasmodium* parasite and the consequence of its treatable but deadly disease. The incidence in the region is very high (J. Boampong, Pers.communication) thereby causing high morbidity and mortality and rendering the youth unproductive. The frequent recurrence of the disease among individuals has had a very bad effect on the economic prosperity and thus compelling the Government of Ghana to brand the region as the 4th poorest region in the country. It is therefore crucial to do further research on the malaria disease in the region to compliment the effort of other health workers to reduce the incidence of the disease thereby creating an environment for sound health and vibrant economy.

Study objectives

The main aim of the present study therefore was to gather information that will contribute to enhanced understanding of the dynamics and the control of malaria.

The specific objectives were to:

1. investigate further the prevalence of malaria in the Central Region.
2. determine the incidence of malaria among the various age groups of people in the Region.
3. identify the most prevalent *Plasmodium* species in the region and determine its distribution in time and space.
4. identify the predominant stage of the parasite in human blood.
5. determine the haemoglobin status of malaria patients in the Region.
6. determine the types, distribution and status of plants used as antimalarials in the Central Region of Ghana.

CHAPTER TWO

MATERIALS AND METHODS

Study areas

The research was conducted in the coastal and forest zones of the Central Region of Ghana (Figs. 3 and 4). The Cape Coast District Hospital and the University of Cape Coast Hospital both with wide catchment areas represented the coastal zone. The forest zone was represented by Saint Francis Xavier Hospital at Assin Foso and Our Lady of Grace Hospital at Breman Asikuma. These constituted the four study areas.

The Central Region covers an area of about 9, 826 square kilometre of land in Ghana and is located along the littoral region of the country. It lies between the major industrial and agricultural Western, Ashanti, Greater Accra and Eastern Regions, which is bordered on the south by a coastline of about 160 kilometres (Fig. 3). It is located on latitude $6^{\circ} 15' S$ and longitude $0^{\circ} 30' N$. The region has twofold vegetation; the dry coastal savanna and the wet rain forest with about one-tenth of the land mass forming various reserved forests some of which have provided natural habitat for rare and exotic species of animals. The region has a population of 1,593,823 and is the second most densely populated in the country. It has growing rate of 2.1% per annum. Central Region has a relatively stable climatic condition with a bimodal rainfall pattern.

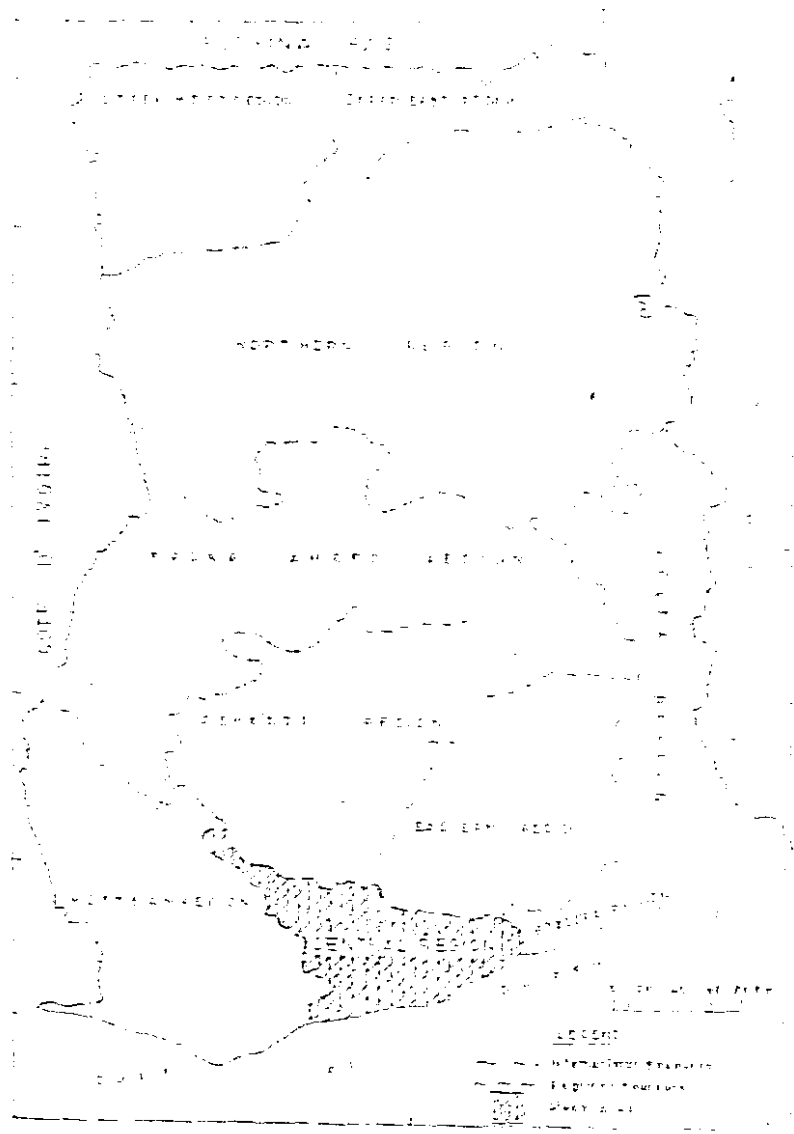


Fig. 3: Map of Ghana showing Central Region

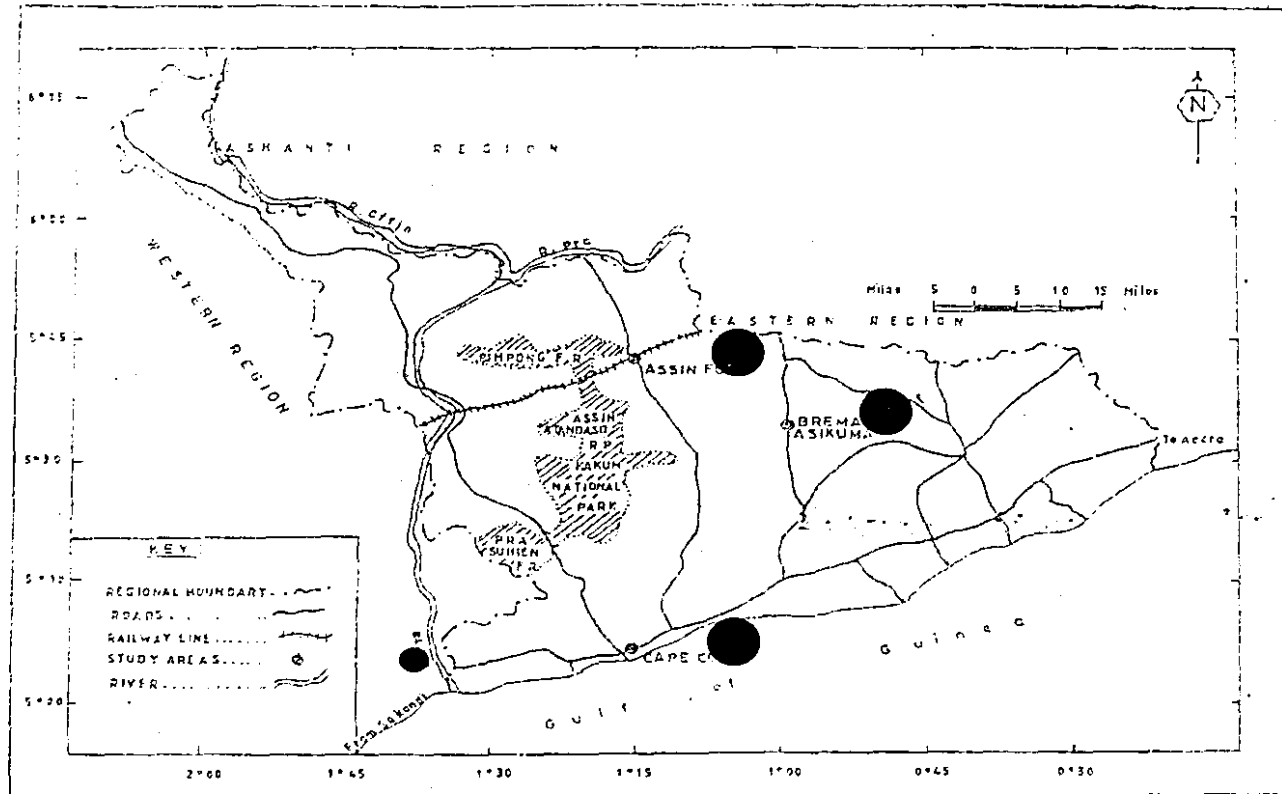


Fig. 4: Map of Central Region showing the study areas.

The major rainy season occurs in April to July while the minor one occurs in September to November. The Region also experiences high temperatures in February and March while low temperatures are experienced in June and August. Economic activities of the Region rests largely on traditional agriculture inland and small -scale fisheries along the coast.

Cape Coast District Hospital

The current Cape coast District Hospital was formerly the Central Regional Hospital which dealt with all referral cases from the various district and mission hospitals. The facility is situated in the heart of Cape Coast and is very close to the sea. Currently, it is one of the four government health facilities in the Cape Coast municipality in addition to a few private ones. The hospital is strategically sited such that it takes care of a chunk of the entire population of the municipality. The hospital also has a referring catchment population from Elmina, Twifu-Hemang Lower Denkyira, Abura-Asebu-Kwamankese and Mfantsiman districts all in the southern zone of the Central Region. Cape Coast is the capital of the Central Region and is also the seat of the Central Regional Co-ordinating Council. The Municipality can boast of numerous educational and financial institutions. It also hosts the regional and district offices for all the government ministries. The main occupation of the indigenous people in Cape Coast is fishing and trading. The town is densely populated along the beaches where there are no proper houses for accommodation compelling people to

mostly sleep in the open at night.

University of Cape Coast Hospital

This hospital was established at the University of Cape Coast to take care of the health needs of the students, staff and their dependants and also as a research centre for students of the University. Additionally, the hospital takes care of the pupils of the university's basic and secondary schools and also has patients' inflow from the several rural communities surrounding the university as well as from the Cape Coast municipality. It is situated along the main Accra-Takoradi highway facing the Atlantic Ocean in the south. Majority of the people who patronize the facility are students and staff of the university. They live in relatively clean environment and sleep in good rooms. The communities around the University are quite overcrowded and inhabitants enjoy trading by taking advantage of the overpopulated student community.

Saint Francis Xavier Hospital

St. Francis Xavier hospital was formerly known as Foso Catholic Hospital established in the early 1950s. The hospital was initially being manned by the District Council then transferred to Our Lady of Apostles Sisters followed by the Dutch Lay Organization until in 1965 when it was entrusted to Sisters Hospitallers of the Sacred Heart of Jesus, a religious sect in the Catholic Church to date. The facility remains the district hospital for both Assin North and South districts.

St. Francis Xavier Hospital is situated at Assin Foso, the district capital for Assin North and is located along the main Cape Coast - Kumasi highway. The two Assin districts cover a land surface area of 2,375 square kilometres out of 9,500 square kilometres of the whole Central Region of Ghana. The hospital has a catchment population of 207,000 and a referring catchment population from Twifu-Hemang Lower Denkyira, Abura-Asebu-Kwamankese, Asikuma-Odoben-Brakwa, Ajumako-Enyan-Essiam and Mfantsiman districts. The people of Assin Foso and its environs are mainly peasant farmers and petty traders. The youth are engaged mainly in the latter.

Our Lady of Grace Hospital

Our Lady of Grace is quite a recent christened name given to this hospital. It was formerly called Breman Asikuma Catholic Hospital. The hospital is a property of the Roman Catholic Church and is manned by the Sister's Hospitallers of the sacred heart of Jesus, a religious sect in the Catholic Church. It is situated in Breman Asikuma, the district capital for Asikuma-Odoben-Brakwa district. The district is in the thick rainforest zone and farming is the main occupation for the people. People living in the various villages outside the district capital live deep in the forests with no proper housing. Children walk long distances to and from school. The houses of most people in the villages and the outskirts of the capital are built with mud and have open eaves. The hospital has inflow of patients from all over the southern part of the country because of an eye clinic facility.

Collection of data from Out-Patients Departments

Monthly data were collated from the Out- Patients Department (OPD) records on patients who were diagnosed as having malaria (Appendix 1). Records of patients whose symptoms were not explicit and were therefore referred to the laboratory for confirmation were also obtained. This was done from November 2005-October, 2006 at all the four hospitals.

Slide preparation and parasite identification

About 20-30 malaria positive thick and thin blood smeared slides made from peripheral blood were collected from each hospital monthly for twelve months. The thin film parts of the prepared slides were already fixed in absolute methanol to enable storage for a longer period. Slides were carefully packed in slide boxes and transported to the Cape Coast District Hospital laboratory where detailed studies involving staining, identification of species and life cycle stages were undertaken.

Stock stain preparation

Giemsa powder weighing 3.8g was crushed in a mortar and 250 mls of glycerol was added and mixed to form a uniform paste. The mixture was poured into a dark bottle and 250 mls of methanol was added and kept at room temperature for storage. The concentrated stain was diluted when needed. The dilution was performed by adding one part of the stain to nine parts of Phosphate

buffer of pH 7.2. This was the method adopted at the Cape Coast district hospital laboratory.

Staining

Smear slides were arranged on a staining rack. A part of the stock stain was pipetted into a staining jar and diluted by a factor of 1 part of stain to 9 parts of phosphate buffer (1 in 10 dilutions). (Note: quantity of stain depended on number of slides to be stained). A plastic teething pipette was used to flood the slides with the stain, and were left for 25 minutes at room temperature. The slides were then drained of the stain and gently washed under running tap water of pH 7.2 to get rid of all debris and artifacts. They were then arranged on a drying rack to dry at room temperature. The stained slides were packed into slide boxes and labelled according to study area and date.

Parasite identification

Identification of the malaria parasites was made using the Bench Aids for the Diagnosis of malaria by Coatney *et al.* (1971) (Plates 1 – 4).

A drop of immersion oil was added to both parts containing the thick and thin films to minimize refraction and give a good contrast. Using the 10x eye piece and 100x objective lens with the condenser iris partly opened, the thick film was first examined to detect the parasites before the thin film was thoroughly examined to identify the species and life cycle stages of the parasite present.

Collection of blood samples for haemoglobin analysis.

The ball of the middle finger of malaria positive patients was wiped clean with cotton wool soaked in 70% ethanol. A sterile lancet was used to prick the finger ball and the first drop of blood cleaned. A capillary tube was used to collect blood of about 0.5mls into a test tube containing Drabkin's solution (Appendix 5) and stirred to mix well before using the automated haemoglobin analyzer for reading. The work was carried out in each hospital for a period of one month when 50-90 samples were obtained (Appendix 2). Each patient was given a laboratory code for reference.

Types, distribution and status of anti-malarial plants

This aspect of the study was conducted using an interview guide questionnaire (Appendix 3). The questionnaire was administered to 50 people from each district. A cross-section of the people made up of herbalists/herbal practitioners, medicinal plant vendors, pastors, traditional birth attendants, chiefs, opinion leaders who are well known to be using herbs in treating diseases in a particular district as well as individuals who seldomly use herbs as anti-malarial drugs were interviewed. The sampling was therefore not random but 'directed' since the objective was to get the names and other relevant information about the plants being used to treat malaria in the region.

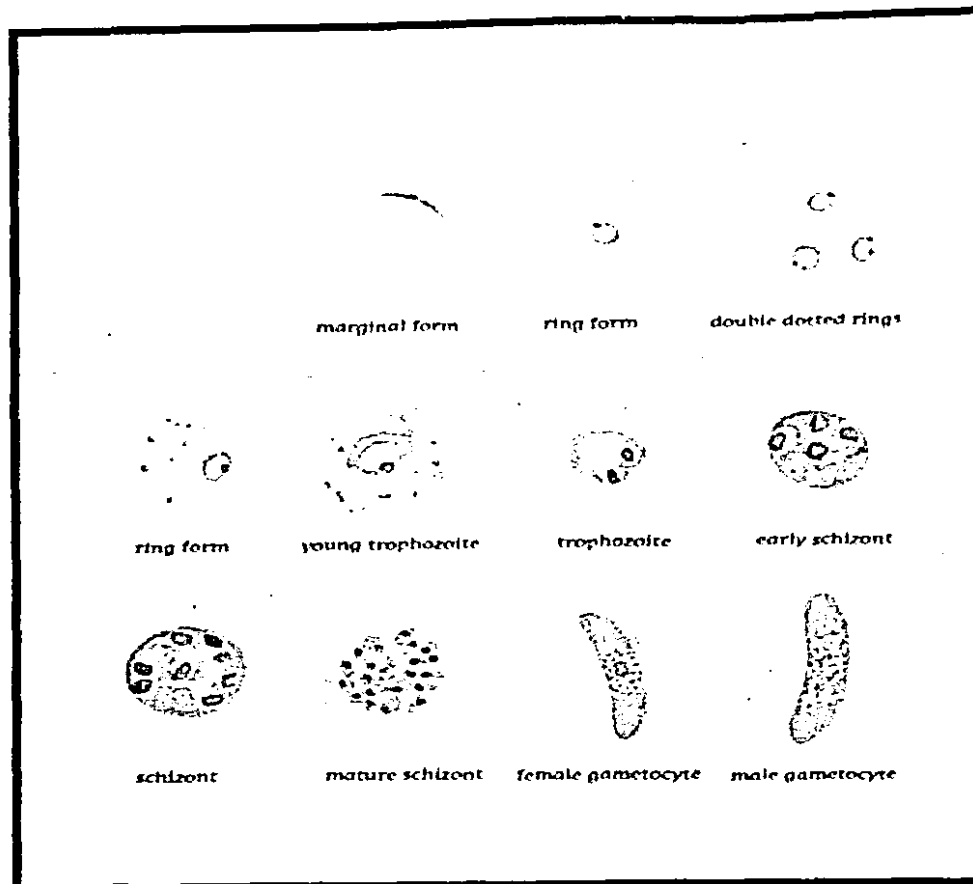


Plate 1: Life Cycle Stages of *P. falciparum*. After Coatney *et al.* (1971)

Diagnostic Points:-

1. Red Cells are not enlarged.
2. Rings appear fine and delicate and there may be several in one cell.
3. Some rings may have two chromatin dots.
4. Presence of marginal or applique forms.
5. It is unusual to see developing forms in peripheral blood films.
6. Gametocytes have a characteristic crescent shape appearance. However, they do not usually appear in the blood for the first four weeks of infection.
7. Maurer's dots may be present

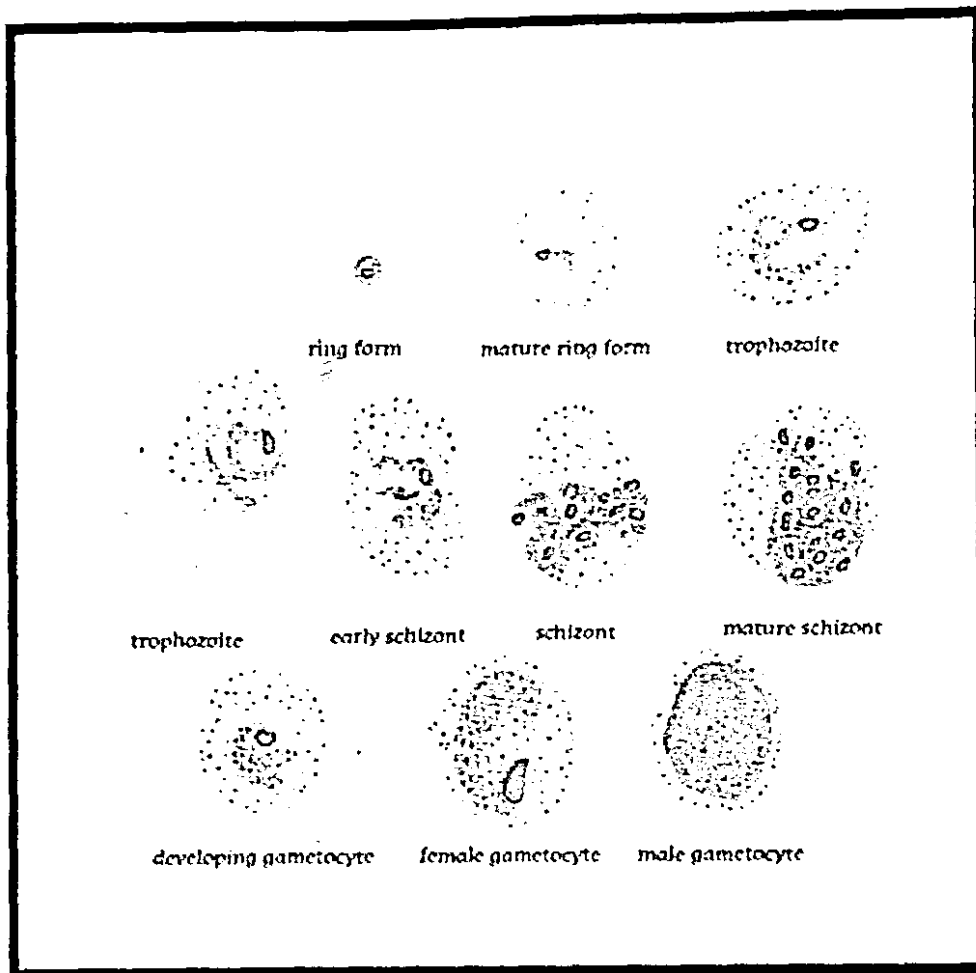


Plate 2: Life Cycle Stages of *P. vivax*. After Coatney *et al.* (1971)

Diagnostic Points:-

1. Red cells containing parasites are usually enlarged.
2. Schuffner's dots are frequently present in the red cells as shown above.
3. The mature ring forms tend to be large and coarse.
4. Developing forms are frequently present.

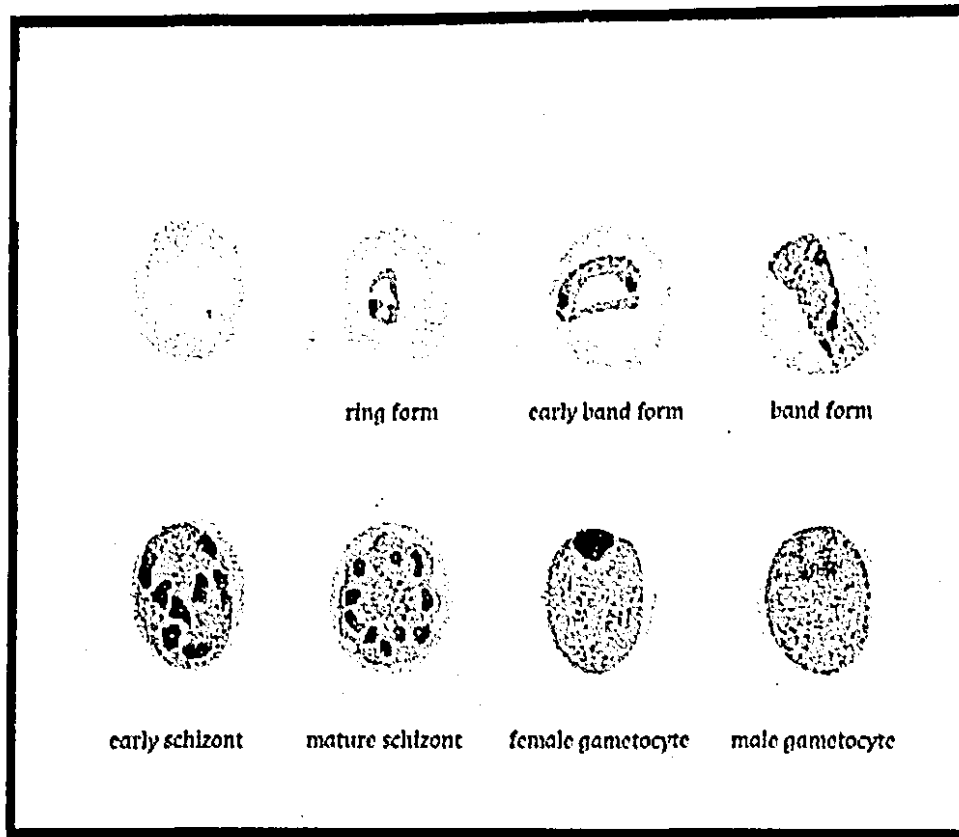


Plate 3: Life Cycle Stages of *P. malariae*. After Coatney *et al.* (1971)

Diagnostic Points:-

1. Ring forms may have a squarish appearance.
2. Band forms are a characteristic of this species.
3. Mature schizonts may have a typical daisy head appearance with up to ten merozoites.
4. Red cells are not enlarged.
5. Chromatin dot may be on the inner surface of the ring.

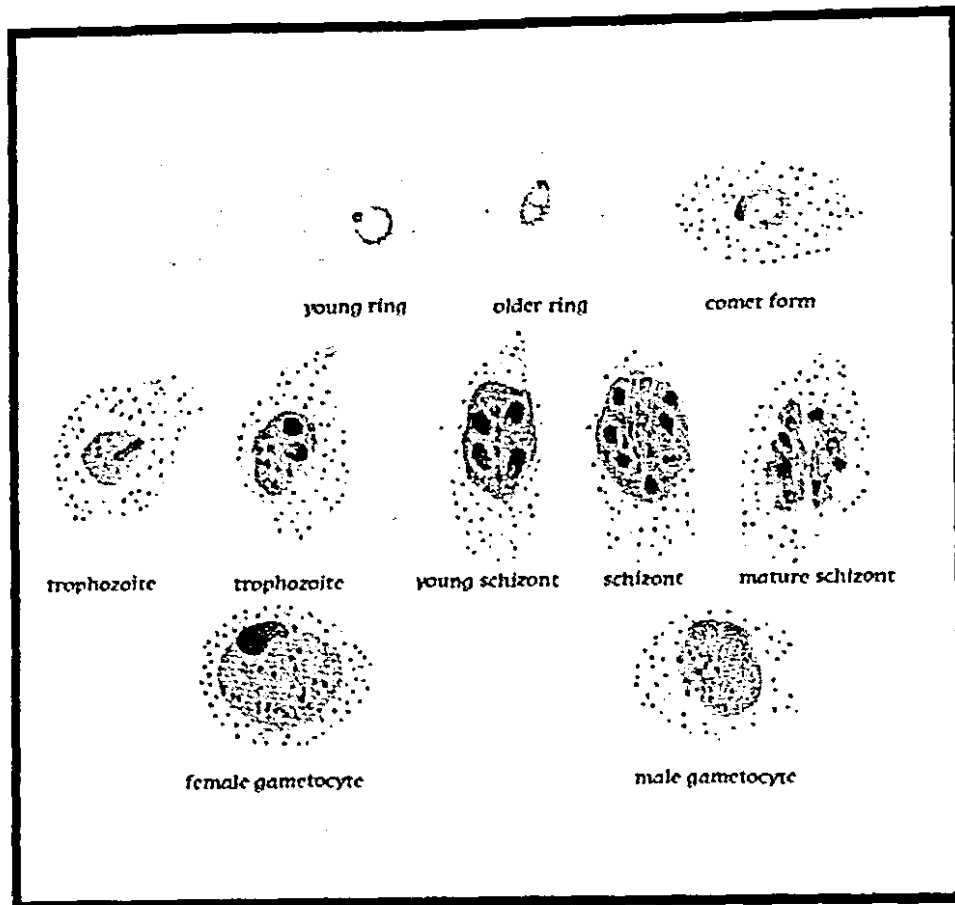


Plate 4: Life Cycle Stages of *P. ovale*. After Coatney *et al.* (1971)

Diagnostic Points:-

1. Red cells enlarged.
2. Comet forms common (top right)
3. Rings large and coarse.
4. Schuffner's dots, when present, may be prominent.
5. Mature schizonts are similar to those of *P. malariae* but larger and coarser.

CHAPTER THREE

RESULTS

Incidence of malaria in the four hospitals

A total of 199,802 patients attended the Cape Coast District Hospital (CCDH), University of Cape Coast Hospital (UCCH), Saint Francis Xavier Hospital (SFXH) and Our Lady of Grace Hospital (OLGH) during the period of study. The raw data are presented in Appendix 1 (A, B, C & D). Patients diagnosed for malaria in the four hospitals were 57,105 giving an overall incidence of malaria among the patients or reported O.P.D cases of malaria patients as 28.6%. The incidence at the various hospitals is presented in Table 1. UCCH recorded the highest incidence (36.3%), followed by CCDH (32.4%), SFXH (24.3%) and OLGH (19.5%) in that order. The mean incidence at the four hospitals was 28.1%.

Monthly incidence of malaria in the four hospitals

The monthly malaria cases in the 4 different hospitals are shown in figures 5 (a, b, c & d). At the CCDH the highest incidence of (43.0%) occurring in August 2006 while the lowest (18.9%) was recorded in April the same year. At UCCH, July 2006 recorded the highest incidence (42.7%) and the lowest

(33.0%) occurred in April. At SFXH the highest (41.1%) and lowest (16.0%) incidence were recorded in December 2005 and October 2006 respectively. OLGH recorded the highest incidence (27.3%) in April 2006 and the lowest (15.4%) in July the same year.

Table 1: Incidence of malaria in the four hospitals.

Hospital	Total Number of Patients	Total Number of Malaria Patients	% Malaria Patients
Cape Coast Dist. Hospital. (CCDH)	43,096	13,949	32.4
University of Cape Coast Hospital. (UCCH)	58,798	21,351	36.3
St. Francis Xavier Hospital. (SFXH)	56,914	13,803	24.3
Our Lady of Grace Hospital. (OLGH)	40,994	8,002	19.5
TOTAL	199,802	57,105	28.6

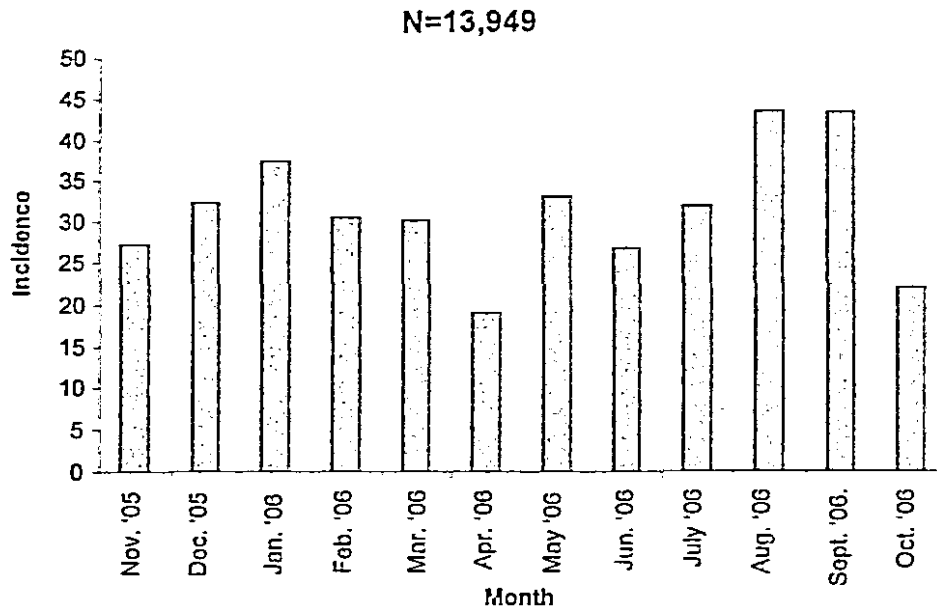


Fig. 5a: Monthly incidence of malaria at CCDH

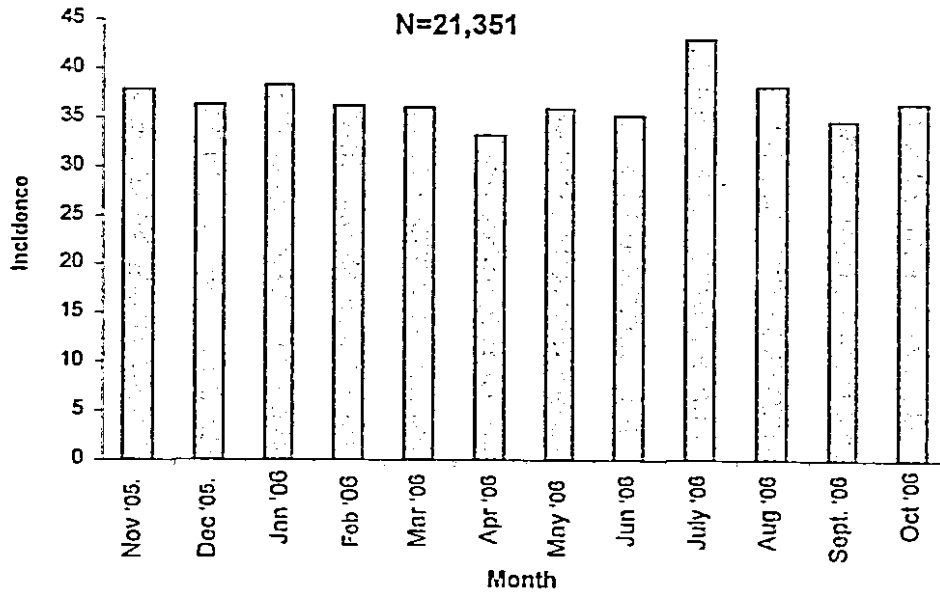


Fig. 5b: Monthly incidence of malaria at UCCH

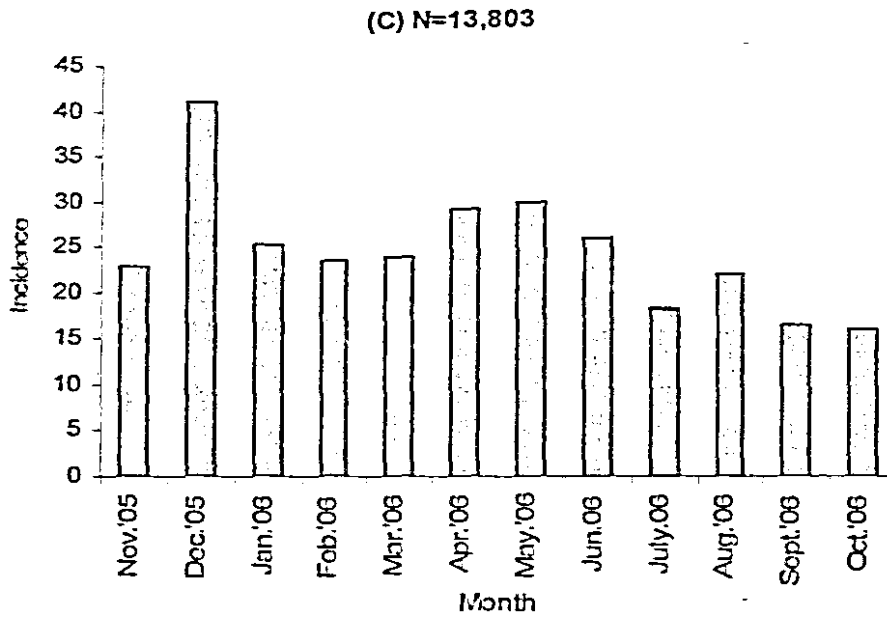


Fig.5c: Monthly incidence of malaria at SFNH

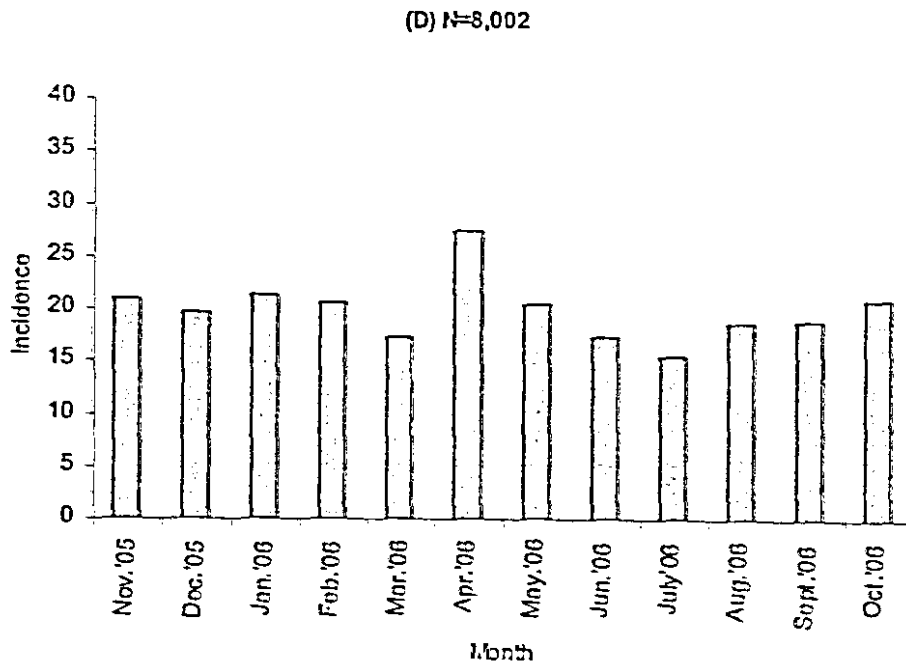


Fig. 5d: Monthly incidence of malaria at OLGH

The pooled data for the four hospitals showed December 2005 as the month with the highest incidence (34.3%) while July 2006 recorded the lowest (26.0%) (Fig.6).

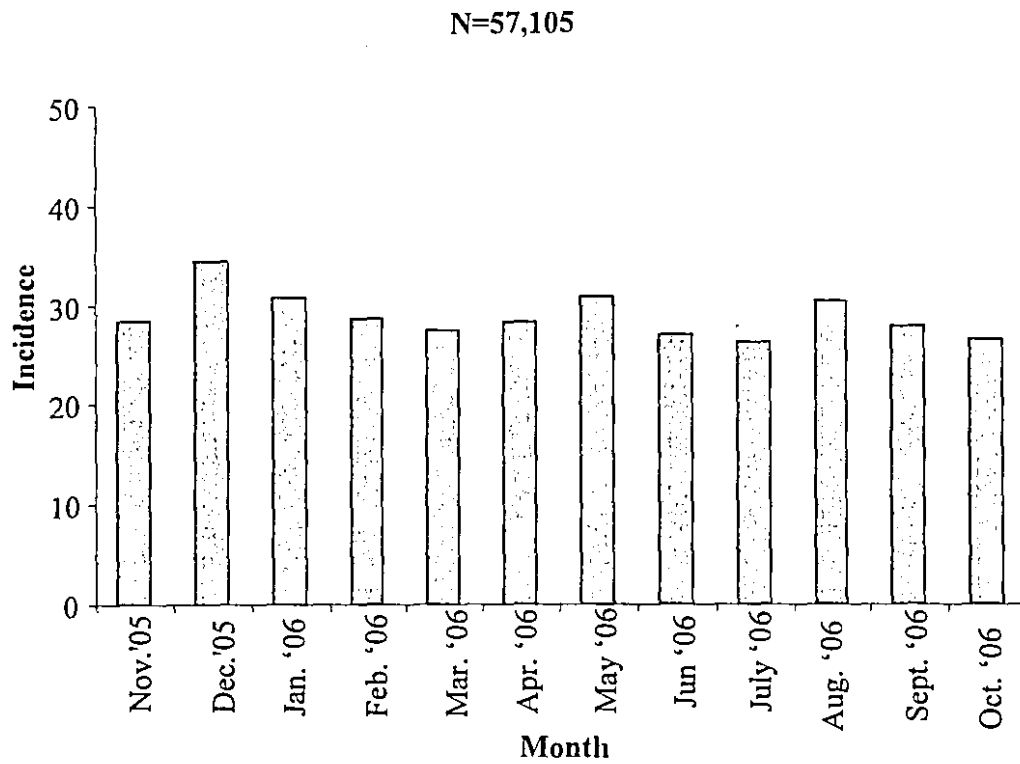


Fig. 6: Pooled monthly incidence of malaria at the four hospitals

Incidence of the disease in different age groups

A total number of 57,105 patients of age <1-70+yrs. were diagnosed as having the malaria disease in the four hospitals during the period of study. Incidence among the various age groups has been presented in (Fig. 7(abcd)).

At the CCDH, age group 11-20yrs had the highest incidence rate of 23.60%, while age group under 1yr had the lowest (5.3%). The rest were: 1-10yrs, 18.0%; 21-30yrs, 19.0%; 31-40yrs, 12.1%; 41-50yrs, 9.0%; 51-60yrs, 6.1% and 61-70+, 7.0%. UCCH diagnosed 21,351 patients as having malaria. The highest incidence of 37.7% occurred in the age 21-30yrs throughout the study period. Age group 61-70+yrs had the lowest incidence of 2.3%. Others were <1yr., 2.5%; 1-10yrs, 10.7%; 11-20yrs, 12.7%; 31-40yrs, 20.5%; 41-50yrs, 9.8% and 51-60yrs, 3.8%. SFXH recorded the highest incidence among the age group 1-10yrs with a rate of 30.0% from a total of 13,803 malaria patients. The rest of the age groups recorded the following: 21-30yrs., 13.1%; 31-40yrs., 11.4%; 11-20yrs., 10.9%; <1yrs, 10.3%; 61-70+, 9.6%; 41-50yrs, 8.6%; and 51-60yrs, 6.3%. OLGH diagnosed 8,002 patients to have malaria during the period of study. The incidence were 20.1% among the 11-20yrs, 19.6% for 1-10yrs, 16.0% for 21-30yrs, 10.2% for 31-40yrs, 9.2% for 61-70+yrs, and 8.9% for 41-50yrs, 8.8% for <1yr. and 6.5% for 51-60yrs respectively.

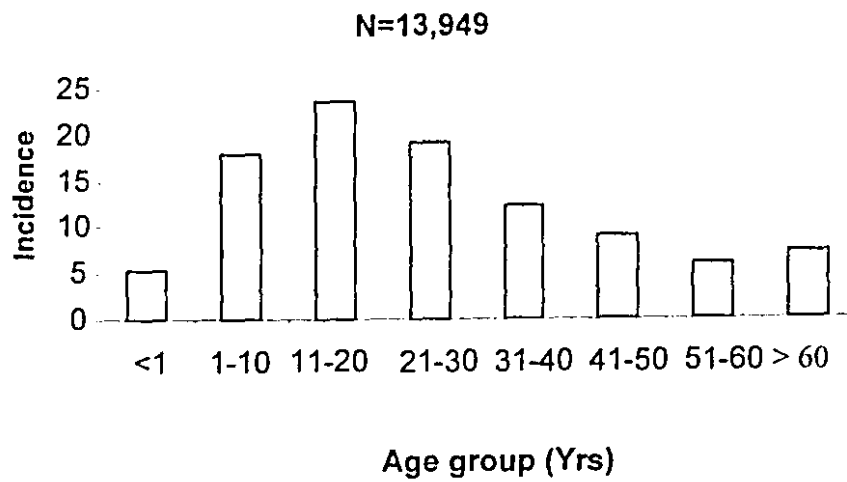


Fig. 7a: Incidence of malaria among various age groups at CCDH

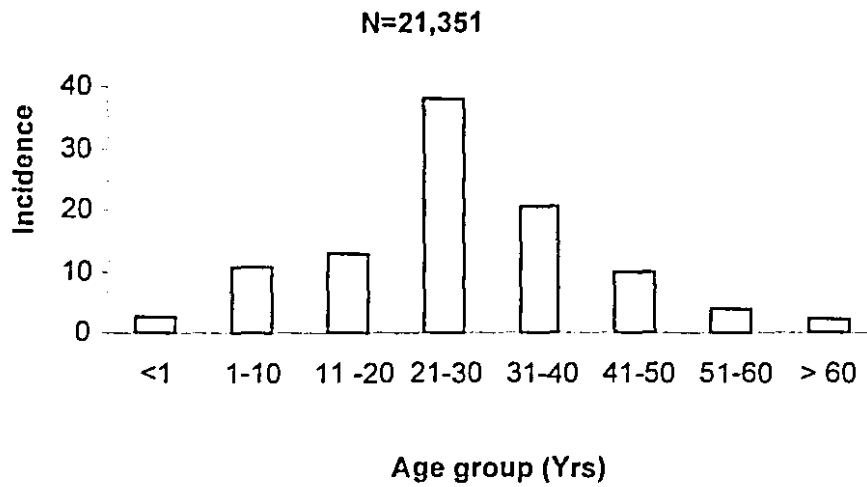


Fig. 7b: Incidence of malaria among various age groups at UCCH

(C) N=13,803

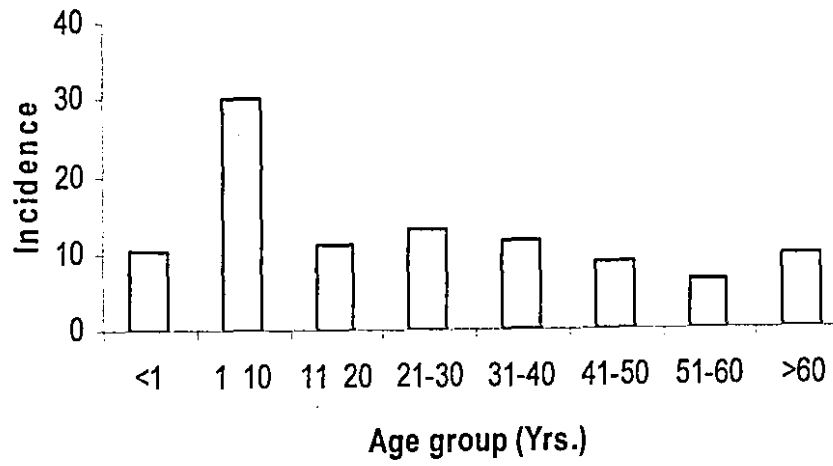


Fig. 7c: Incidence of malaria among various age groups at SFXH

N=8002

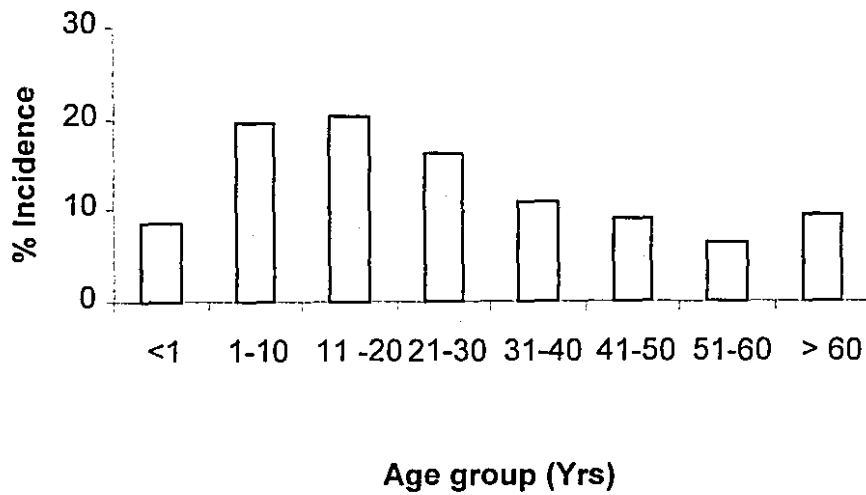


Fig. 7d: Incidence of malaria among various age groups at OLGH

It should be noted that the age distribution of malaria patients at UCCH might have been affected by the age structure of the University of Cape Coast students who are the major patrons of the facility. In view of this apparent unnatural distribution (Fig.7b) the subsequent analyses pertaining to age did not include the UCCH data. Combined incidence of the disease among the various age groups from three hospitals without UCCH indicated that age group 1-10 yrs. recorded the highest incidence rate of 23.0% in the region during the year under review (Fig.8). The rest followed with 17.9% (11-20yrs), 16.0% (21-30yrs), 11.6% (31-40yrs), 8.8% (41-50yrs), 8.5% (61-70+yrs), 8.0% (Under 1yr) and finally 6.3% (51-60yrs) respectively. It should be noted that if the less than 1 yr. group are added to the 1-10 yrs. group then the children up to 10 yrs old are the most affected in the population.

Incidence of malaria among various age groups and sex

The results of malaria incidence organized according to age groups and sex are presented in figure 9(A,B&C). At CCDH there were no clear-cut differences in the incidence of the disease among the sexes in the various age groups. At SFXH and OLGH incidence in the males appeared to be higher in the younger population (up to 10years) while the females generally recorded higher incidence in the older age groups. By ignoring the UCCH data for reasons indicated earlier and combining the results from 3 hospitals as shown

in figure 10, it is evident that the regional pattern (Afari *et al.* 1993) is reflected by the results in the 3 hospitals separately (see Fig. 9).

In figure 11 the sexes of malaria patients in the 3 hospitals were pooled and presented separately. It is seen that females recorded higher incidence rates than males in all cases- approximately 52% for both CCDH and OLGH, and 55% for SFXH (Fig.11). The calculated Chi-square values were 20.982, 132.624 and 25.532 for CCDH, SFXH and OLGH respectively compared with 3.841 at 5% level. This indicates significant differences among the sexes.

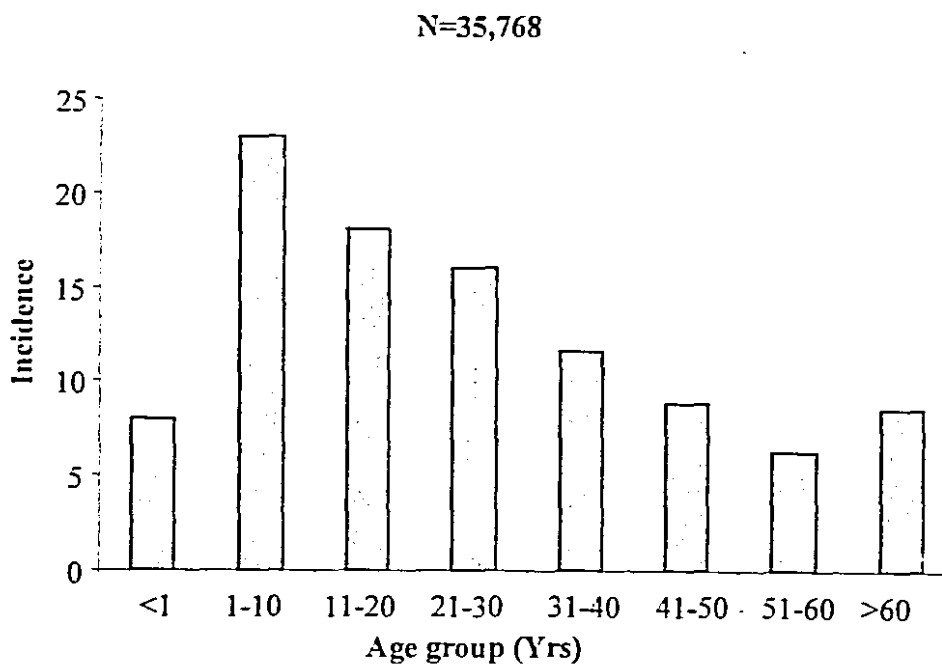


Fig. 8: Combined incidence of malaria in various age groups at CCDH, SFXH and OLGH

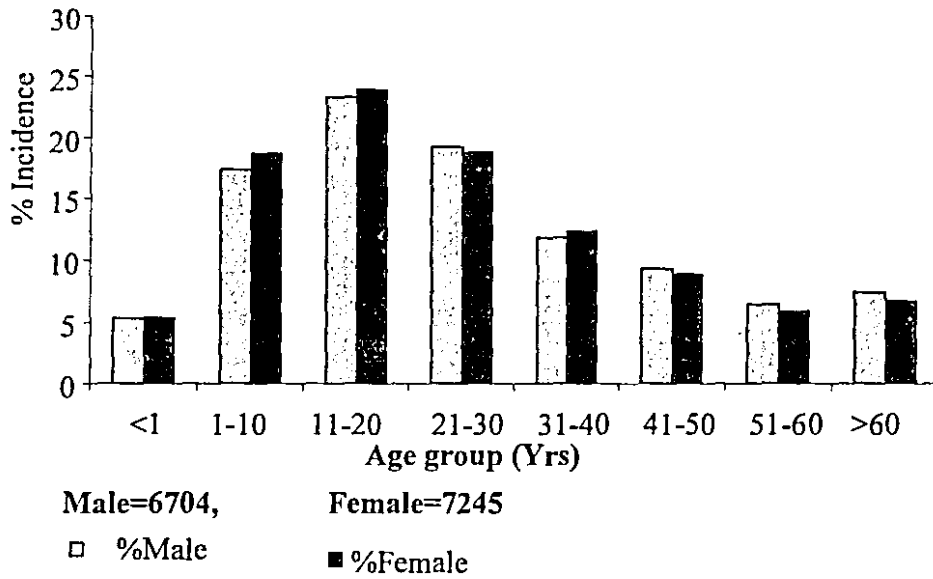


Fig. 9a: Incidence of malaria among various age groups and sex at CCDH

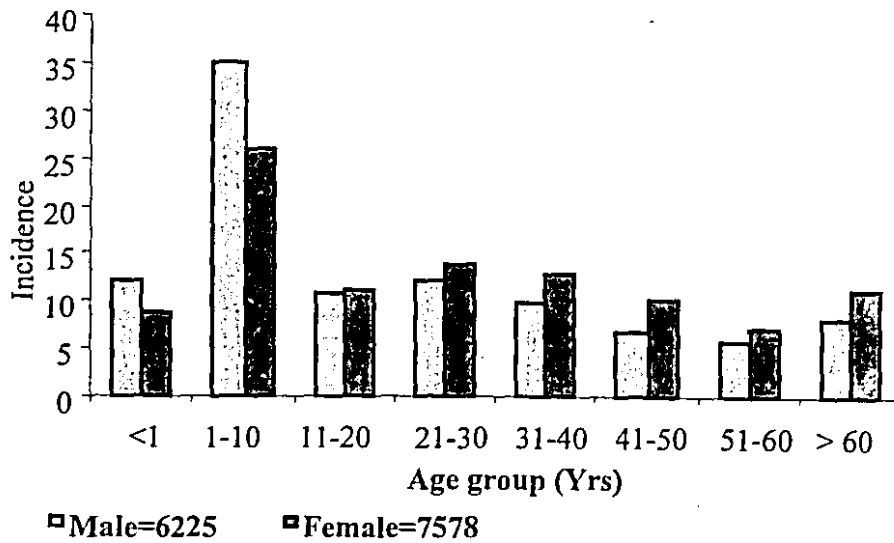


Fig. 9b: Incidence of malaria among various age groups and sex at SFXH

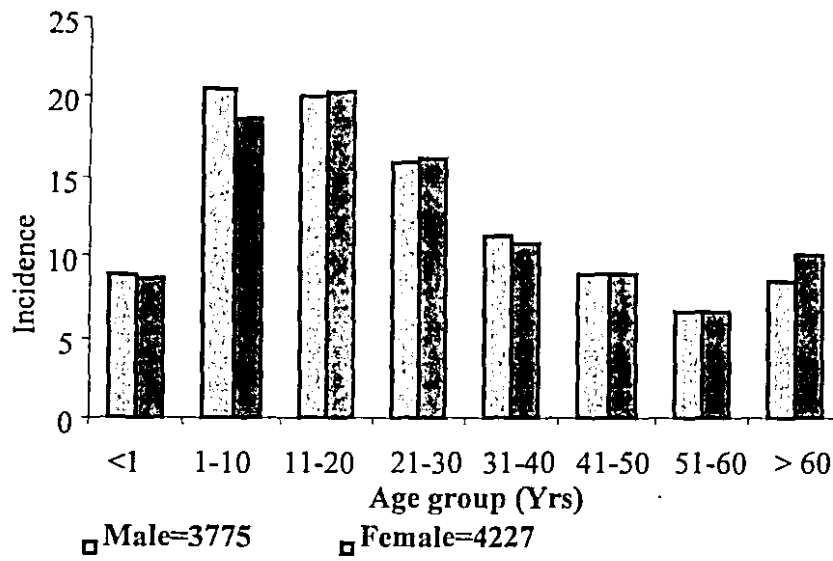


Fig. 9c: Incidence of malaria among various age groups and sex at OLGH

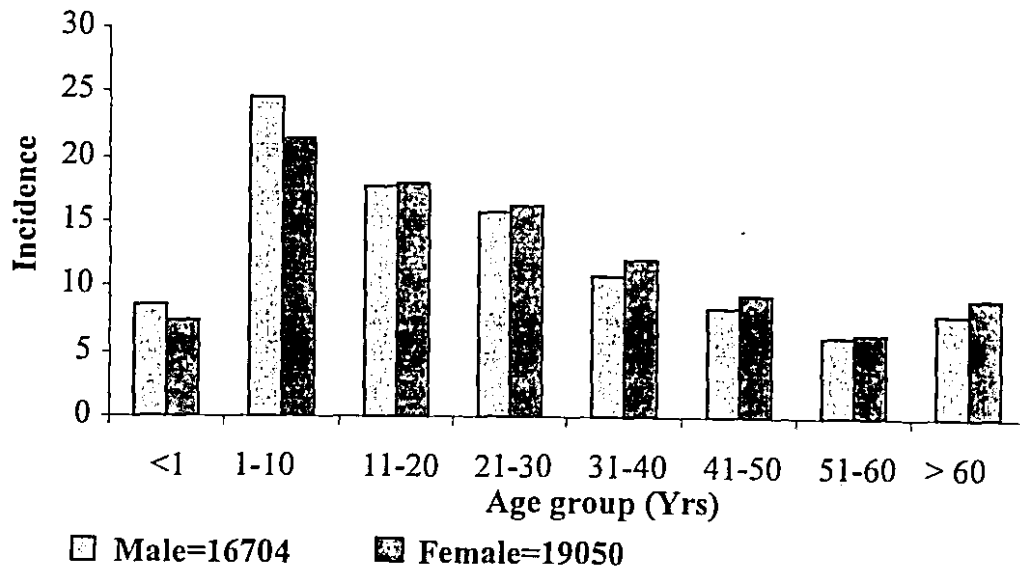


Fig.10: Combined incidence of malaria among age groups and between the sexes in three hospitals

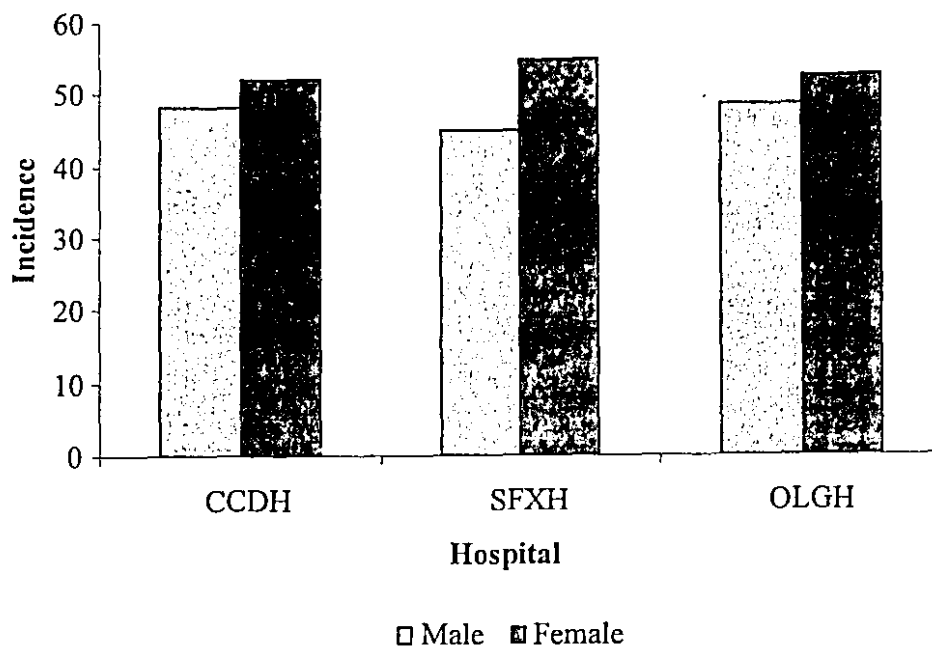


Fig.11: Incidence of malaria by sex of patients in three hospitals

Distribution of Malaria Parasite Species

The distribution of *Plasmodium* species in the region is presented spatially and temporally below.

Spatial distribution of *Plasmodium* parasite in three hospitals

A total of 1,016 malaria positive thin blood smeared slides were examined to identify the various *Plasmodium* species and their stages found in malaria patients. This was done over 12 months, January-December 2006. The specimens were obtained from the Cape Coast District Hospital (CCDH), University of Cape Coast Hospital (UCCH) and Saint Francis Xavier Hospital

(SFXH) respectively. Our Lady of Grace Hospital (OLGH) could not provide smeared slides due to under staffing problem at the laboratory. All slides were stained and examined at the Cape Coast District Hospital because of the availability of space, reagents, other working materials and equipment.

At the CCDH, out of 433 positive slides examined, 97.9% was *P.falciparum*, 0.9% *P. malariae*, 0.2% *P.ovale* and 0.9% mixed infection of *P. falciparum* and *P. malariae*. The 285 slides examined from UCCH had 99.3% *P. falciparum*, 0.7% *P.malariae*. None of the other species was encountered. At SFXH 294 slides examined had 94.9% *P.falciparum*, 3.0% *P.malariae*, 1.0% *P.ovale* and 1.0% mixed infection of *P. falciparum* and *P. malariae*. The predominant malaria parasite encountered was *Plasmodium falciparum* (97.4%), followed by *P. malariae* (1.5%), *P. ovale* (0.4%) and mixed infection from *P. falciparum* and *P. malariae* (0.7%) (Table 2). The overall representation of the different species in selected areas of Central Region is illustrated using a pie chart in Figure 12. *Plasmodium vivax* was not encountered during the study.

Monthly occurrence of *Plasmodium* species in the three hospitals.

Monthly occurrence of the *Plasmodium* species in the 3 hospitals has been tabulated in Tables 3-5. At all the three hospitals, *P. falciparum* dominated throughout the period of study. At CCDH, *P. malariae* was encountered in January, May and November. *P. ovale* was seen only in January while mixed infection occurred in January, July and September. At UCCH, *P. falciparum* also dominated throughout the months with only *P. malariae* occurring in May

and September. Although *P. falciparum* still showed dominance at SFXH, *P. malariae* occurred in more months as compared to CCDH and UCCH. *P. malariae* was encountered continuously from January-April and October – November (Table 5). *P. ovale* was seen in January and March with mixed infection occurring in October and November.

Table 2: Spatial distribution of *Plasmodium* species in patients during 12 months in three hospitals

Hospital	Species				
	<i>P. falciparum</i>	<i>P. malariae</i>	<i>P. ovale</i>	<i>P. vivax</i>	Mixed infection
CCDH	426 (98.40%)	2 (0.50%)	1 (0.23%)	0 (0.00%)	4 (0.92%)
UCCH	283 (99.30%)	2 (0.70%)	0 (0.00%)	0 (0.00%)	0 (0.00%)
SFXH	280 (95.23%)	9 (3.04%)	3 (1.01%)	0 (0.00%)	2 (0.71%)
Total	989 (97.74%)	13 (1.31%)	4 (0.39%)	0 (0.00%)	6 (0.60%)

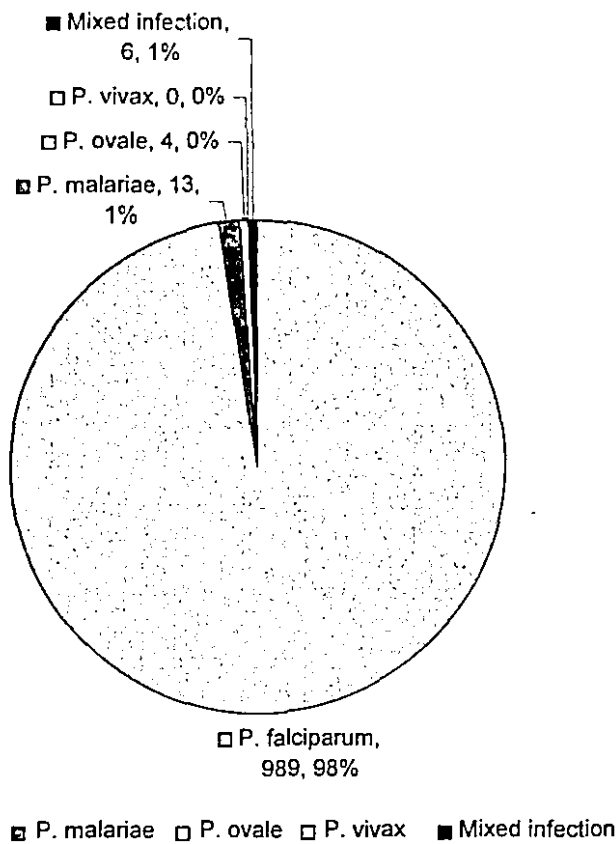


Fig. 12: The combined spatial distribution of malaria parasites in the study areas

Stages of malaria parasites in human blood encountered during the study

Erythrocytic stages of the various *Plasmodium* parasites identified have been presented in table 6. A total of 1016 slides were examined from three of the hospitals, namely CCDH, UCCH and SFXH. At CCDH, 433 slides were microscopically examined. *P. falciparum* trophozoites occurred on 408 slides, 5 slides had trophozoites + schizonts, 13 had trophozoites + gametocytes totaling

426 *P. falciparum* infected slides. *P. malariae* occurred on 2 slides with all stages, ie. trophozoites + schizonts + gametocytes. Only the trophozoites stage of *P. ovale* occurred on 1 slide. Mixed infection of *P. falciparum* + *P. malariae* had only trophozoites on 4 slides. UCCH had 285 slides. *P. falciparum* trophozoites occurred on 265 slides. Two slides had trophozoites + schizonts, and 16 slides had trophozoites + gametocytes making a total of 283 *P. falciparum* slides. There was 1 *P. malariae* trophozoites slide and 1 slide having all stages of the same species. No *P. ovale* species and mixed infection was recorded. Two hundred and ninety four slides were observed at SFXH. *P. falciparum* occurred on 280 slides. Only *P. falciparum* trophozoites occurred on 246 slides, trophozoites + schizonts on 9, trophozoites + schizonts + gametocytes on 24 and 1 with pigment. *P. malariae* had 5 trophozoites only, and 4 trophozoite + schizonts + gametocytes. *P. ovale* had 3 slides presenting all the stages. There were 2 mixed infections of *P. falciparum* + *P. malariae* having only 1 trophozoites.

Table 3: Monthly occurrence of *Plasmodium* Species at Cape Coast District Hospital in 2006

Month	Species				
	<i>P. falciparum</i>	<i>P. malariae</i>	<i>P. ovale</i>	<i>P. vivax</i>	Mixed infection
Jan.	33	1	1	0	1
Feb.	35	0	0	0	0
Mar.	35	0	0	0	0
April	37	0	0	0	0
May	47	1	0	0	0
Jun.	31	0	0	0	0
July	34	0	0	0	2
Aug.	38	0	0	0	0
Sept.	44	0	0	0	1
Oct.	36	0	0	0	0
Nov.	32	2	0	0	0
Dec.	24	0	0	0	0

Table 4: Monthly occurrence of *Plasmodium* Species at University of Cape Coast Hospital in 2006

Month	Species				
	<i>P. falciparum</i>	<i>P. malariae</i>	<i>P. ovale</i>	<i>P. vivax</i>	Mixed infection
Jan.	24	0	0	0	0
Feb.	29	0	0	0	0
Mar.	21	0	0	0	0
April	26	0	0	0	0
May	24	1	0	0	0
Jun.	18	0	0	0	0
July	23	0	0	0	0
Aug.	23	0	0	0	0
Sept.	25	1	0	0	0
Oct.	23	0	0	0	0
Nov.	21	0	0	0	0
Dec.	26	0	0	0	0

Table 5: Monthly occurrence of *Plasmodium* species at St. Francis Xavier Hospital in 2006

Month	Species				
	<i>P. falciparum</i>	<i>P. malariae</i>	<i>P. ovale</i>	<i>P. vivax</i>	Mixed infection
Jan.	21	1	1	0	0
Feb.	21	2	0	0	0
Mar.	20	1	2	0	0
April	21	2	0	0	0
May	23	0	0	0	0
Jun.	23	0	0	0	0
July	23	0	0	0	0
Aug.	23	0	0	0	0
Sept.	23	0	0	0	0
Oct.	23	1	0	0	2
Nov.	32	2	0	0	1
Dec.	28	0	0	0	0

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Table 6: Erythrocytic stages of *Plasmodium* parasites on slides in three hospitals

	Tropho- zoites (T)	Trophozoites + Schizonts (S)	Trophozoites + Gametocytes (G)	T + S+ G	Pigmen t	Species Total
CCDH						
<i>P.f</i>	408	5	13	-	-	426
<i>P. m</i>	-	-	-	2	-	2
<i>P. o</i>	1	-	-	-	-	1
<i>P.f</i>	4	-	-	-	-	4
+ <i>P.m</i>						
Total	413	5	13	2	0	433
UCCH						
<i>P.f</i>	265	2	16	-	-	283
<i>P. m</i>	1	-	-	1	-	2
<i>P. o</i>	-	-	-	-	-	0
<i>P.f</i>	-	-	-	-	-	0
+ <i>P.m</i>						
Total	266	2	16	1	0	285
SFXH						
<i>P.f</i>	246	9	-	24	1	280
<i>P. m</i>	5	-	-	4	-	9
<i>P. o</i>	-	-	-	3	-	3
<i>P.f</i>	2	-	-	-	-	2
+ <i>P.m</i>						
Total	253	9	0	31	1	294

Haemoglobin levels in malaria patients

The levels of haemoglobin in the malaria patients are presented below in relation to parasite density as well as sex.

Haemoglobin levels in relation to parasite density in the four hospitals.

Haemoglobin analysis was carried out on about 90 malaria patients from each of the four hospitals and related to the parasite density of the patient (Appendix 2). The results from the various hospitals have been illustrated in Figs.13, 14, 15 and 16. At CCDH, mean haemoglobin levels reduced from 9.9 g/dL to 5.9 g/dL as parasite density increased from 1+ to heavy infection of 4+ (Fig. 13). UCCH recorded the lowest 9.5g/dL as haemoglobin level at 3+ but slightly increased to 11.4g/dL at 4+ (Fig. 14). SFXH recorded 10.1g/dL as the highest haemoglobin level at 2+ and reduced to a minimum level of 7.0g/dl at 4+ (Fig. 15). At the OLGH, the haemoglobin level was highest at parasite density of 1+ (8.8g/dl) and lowest at 3+ with a value of 7.2g/dL; no heavy parasite density (4+) was observed (Fig.16). In the four hospitals, pooled data had haemoglobin levels reducing as parasite density increased (Fig. 17). Haemoglobin level was highest at parasite density of 1+ (10.0g/dL) and lowest at 4+ with a value of 6.1g/dL. Haemoglobin levels of 9.7g/dL and 8.4g/dl occurred at parasite densities 2+ and 3+, respectively.

Mean haemoglobin levels in male and female malaria patients

Haemoglobin levels of males and females from the various hospitals are shown in figure 18 and compared using Students t-test statistical model. CCDH recorded haemoglobin level of 8.8g/dL for males and 7.8g/dL for females. Patients from SFXH recorded levels of 9.2g/dL and 8.9g/dL in males and females respectively. OLGH also recorded a higher level of 8.3g/dL for males and 8.0g/dL for females. On the whole haemoglobin levels reduced from 10g/dL at low infection of 1+ to 6g/dL at high infection of 4+. At UCCH, referred cases used identity numbers instead of personal information on laboratory forms. Hence sexes could not be obtained from the laboratory forms. The students't-test analysis showed that the differences in the haemoglobin levels by sex of the malaria patients in all three hospitals were significant (P= 5.938, 9.200, 5.256 for CCDH, SFXH and OLGH respectively as compared to 4.30 at 5% level.

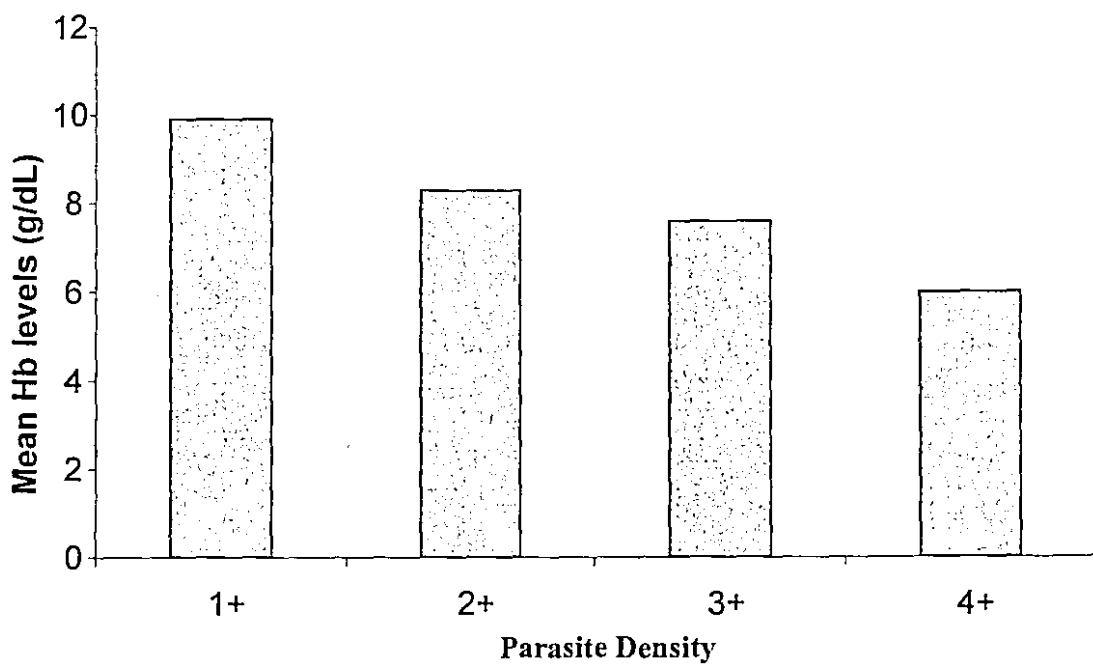


Fig.13: Mean haemoglobin levels relative to parasite density at CCDH

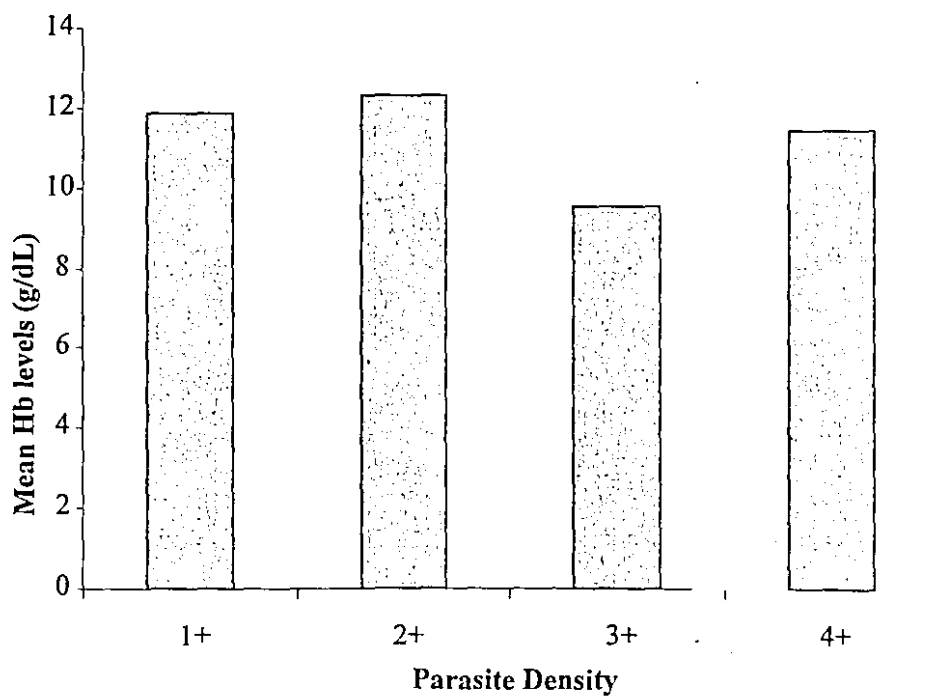


Fig.14: Mean haemoglobin levels relative to parasite density at UCCH

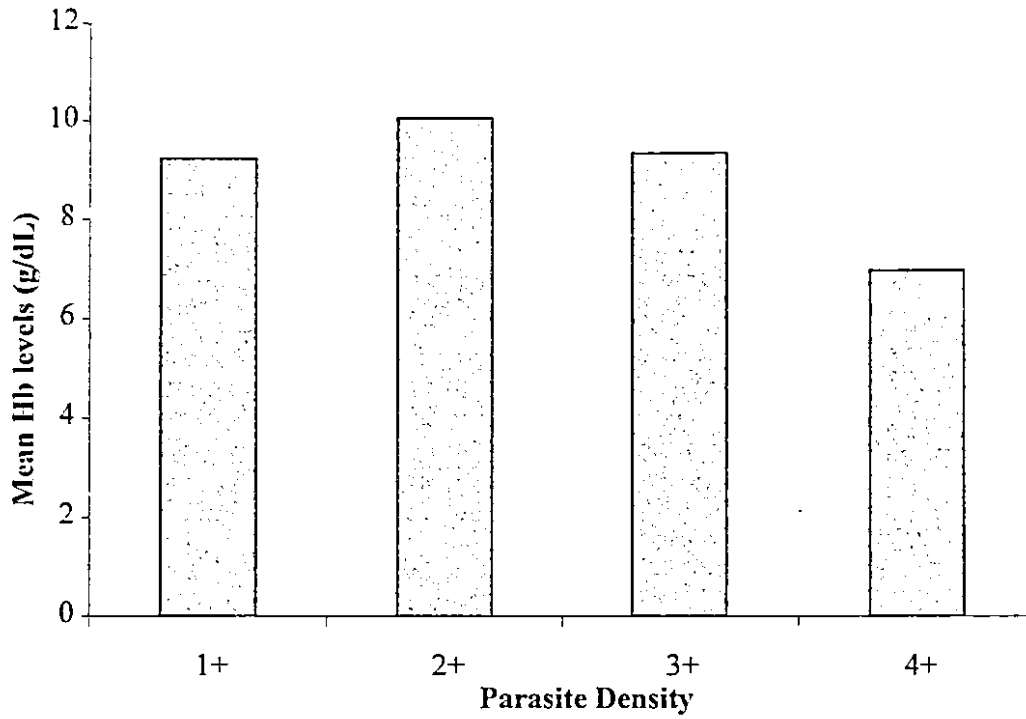


Fig.15: Mean haemoglobin levels relative to parasite density at SFXH

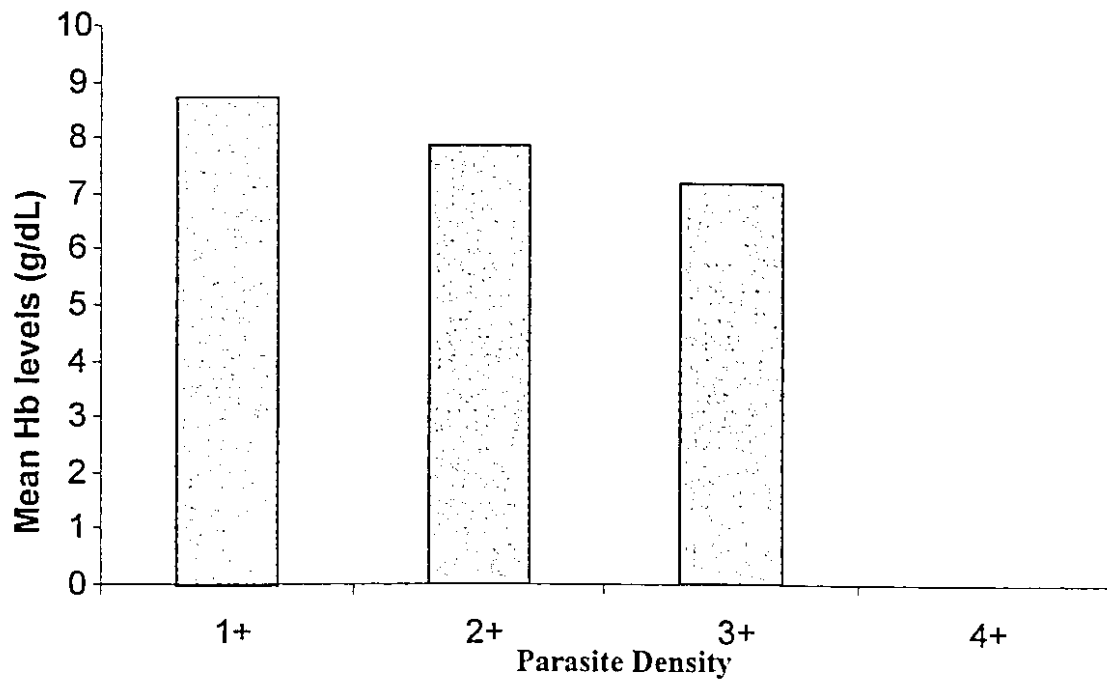


Fig. 16: Mean haemoglobin levels relative to parasite density at OLGH

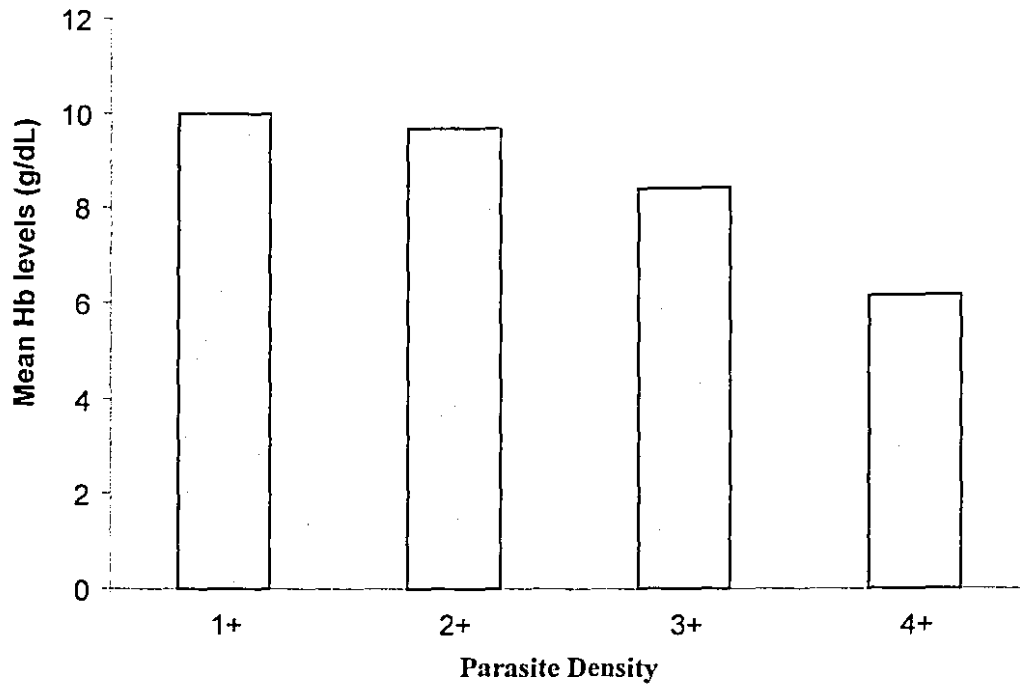


Fig.17: Mean haemoglobin levels relative to parasite density in the four hospitals combined

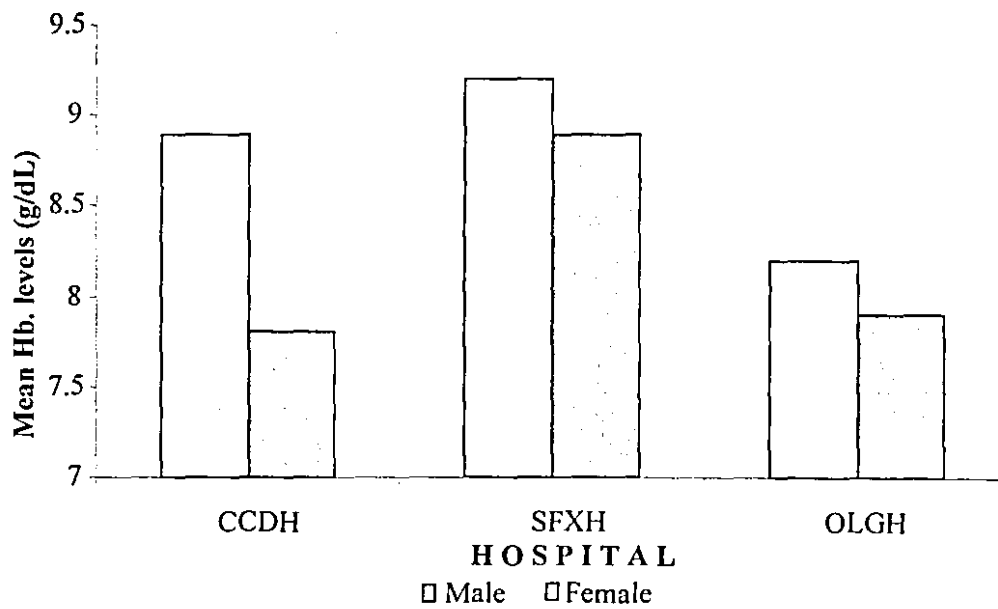


Fig. 18: Mean haemoglobin levels by sex of patients in three hospitals

Types, distribution and status of plant species used as anti-malarials

Breakdown of respondents to questionnaire

Respondents totaling 150 were interviewed using a questionnaire (Appendix 3) and were distributed as follows:

Herbal practitioners/Vendors – 8%, Traditional birth attendants (TBAs) -6%, Chiefs/Opinion leaders – 2%, general public-84%. The distribution according to gender was: Females (54%), Males (46%). Respondents who have had some level of education were considered literate and constituted 56% while 44% were considered illiterate with no formal education. On the average, respondents may have known the plants and used them for not less than 2 years. Information gathered indicated that knowledge of these species was passed on orally from relatives (parents/grandparents) who practiced traditional medicine.

Symptoms such as headache, chills, body pains, joint pains, loss of appetite, parlor, wasting, cough, convulsions in children, blurred vision, vomiting, constipation, backache, frequent urination, diarrhea, tiredness, general weakness and fatigue were mentioned as disease indicators of malaria fever.

Species sampled from the study area and which are used as antimalarial remedies

Eighty nine plant species belonging to 41 families were recorded as useful in treating malaria in the study areas (Table 7). *Euphorbiaceae* recorded the highest number of species (8) while families *Apocynaceae*, *Asteraceae* and

Meliaceae had 5 each, *Caesalpinaceae*, *Fabaceae* and *Poaceae* 4 each, *Combretaceae*, *Rubiaceae*, *Rutaceae*, and *Solanaceae* recorded 3 each. The following families recorded 2 species each: *Anacardaceae*, *Bignoniaceae*, *Compositae*, *Labiatae*, *Mimosaceae*, *Myrtaceae*, *Palmaceae*, *Sapotaceae*, *Ulmaceae* and *Verbanaceae* while 19 others: *Agavaceae*, *Amaranthaceae*, *Annonaceae*, *Bromeliaceae*, *Caricaceae*, *Connaraceae*, *Convolvulaceae*, *Cucurbitaceae*, *Lauraceae*, *Lecythidaceae*, *Lauranthaceae*, *Malvaceae*, *Maranthaceae*, *Musaceae*, *Nymphaeaceae*, *Passifloraceae*, *Periplocaceae*, *Sapindaceae* and *Sterculiaceae* recorded 1 species each.

Anti-malaria plants mentioned for the first time

At least 60 plant species are recorded for the first time as having anti-malarial properties (Table 8), (Agbovi *et al.* 2002)

Parts of plant species used, mode of preparation and administration of drug

The information gathered from the respondents to the questionnaire indicated that Leaves are the most part used (i.e. 49.5% of the total number of plants), Stem bark constituted (21.2%) followed by roots (14.1%). Fruits followed with (7.1%), seeds and whole plant constituted (3.0%) each while inflorescence was (2.0%). Ninety five percent of respondents prepared their drugs by boiling while 5% powder the plant material. Most of the respondents (68.2%) administer their preparation orally; steam bathing is employed by 21.2% of respondents while 9.4% use enema as mode of treatment. Other

approaches used are smearing and cold bath each of which is used by 1.2% of the respondents. The exact dosage of the drug could not be indicated by most respondents (86%) who at the same time recommended the use of herbal medicine to all.

A total of 89 species distributed in 41 families were sampled as plant materials used in treating malaria fever. A list of the species together with their other uses is presented in Table 7.

The plants are also used for a wide range of purposes such as fuel wood, carpentry, food, tool handles, fodder and many others.

Table 7: Plants used as remedies for malaria in three selected areas of Central Region, Ghana

Family	Species	Local Name	Plant form	Parts used	Other uses
1. Agavaceae	<i>Sansevieria longiflora</i> Thunb.	Twiton (Twi)	-	-	-
2. Amaranthaceae	<i>Pupalia lappacea</i> (Adans) Mut. Juss.	Mpupua (Twi)	H	Lvs	Blood purifier; Anti-snake bite.
3. Anacardaceae	<i>Mangifera indica</i> Linn.	Mango (English)	T	Lvs., Sb.	Anti-syphilitic; piles; diarrhea; dysentery; gonorrhoea; diuretic; fever; anti-helminthic.
	<i>Spondias mombin</i> Linn.	Atoa (Twi)	T	Lvs.	Febrifuge; diuretic; purgative; cough; laxative; fever; gonorrhoea; stomachic; astringent; paralysis.
4. Annonaceae	<i>Annona muricata</i> Linn.	Apple (Thorny) (English)	T	Lvs., Sb.	Chicken pox; tumours; cuts.
5. Apocynaceae	<i>Alstonia boonei</i> Linn.	Nyamedua (Twi)	T	Lvs.,S b.	Antipyretic; anti-helminthic; fever; malaria; sore; anti-rheumatic; ulcers; asthma; gonorrhoea; filarial infection; purgative; eye medicine; Antimicrobial.
	<i>Picralima nitida</i> Pierre.	Akuama (twi)	T	Lvs., Ft.	Anti-fever; stomachic; pneumonia; venereal diseases; vermifuge; anti-malarial.
	<i>Pleiocarpa mutica</i> Benth.	Onwenma (Twi)	S	Sb., Ft.	Anti-fever; carvings.
	<i>Rauvolfia vomitoria</i> Linn.	Kakapenpen (Twi)	T	Fl., Sb.,Rt.,Rb.	Sedative; tranquilizer in psychiatric condition; hypotensive; skin diseases; purgative; jaundice; gastro-intestinal problems; anti-convulsant; antispasmodic; aphrodisiac; eye drop for vertigo; anti-snake bite; abortifacient; anti-leprosy; cerebral discharges; anti-fever.

Table 7 continued

	<i>Theselia peruviana</i> (Lam.) Juss & Benth.	Milkthistle/eye no. nervene (Twt)	II	I, vii	
	<i>Fourcraea affricana</i> Thunb.	Ammonium/Nipentem/ cheds (Twt)	II	RI	
	<i>Acanthospermum</i> <i>hispidum</i> (Lam.) Ageratum conyzoides Lam.	Patakmandani/ patakumani (Fante) Mumumun (Fante)	II	Acrid pnt Acrid	
	<i>Aspida africana</i> Thunb.	Micobedev (Twt)	II	All pntb.	
	<i>Lactuca</i> <i>taraxacifolia</i> Thunb.	Dandelion (Ugulu)	II	I, vii	Wounds, ulcers, and leprosy and scabies.
	<i>Tridax procumbens</i> Lam.	Fonlezylbe (Twe)	II	I, vii	
	<i>Kipido affricana</i> Engl.	Mudim/ Mudim (Twt)	T	I, vii	Piles; dysentery; kidney problems; syphilis; gonorrhoea; conjunctivitis; tapeworm; anti snake bite
	<i>Neochondria laevis</i> Juss & Benth.	Chamomilla (Twt)	III/II	RI, 3b	Hypochondria; aphrodisiac; dysentery; rheumatic swellings; round worms; syphilis; head ache; obstetrics; convulsions in children; ear and tooth aches; malaria; constipation; post partum haemorrhage; leaves in soup for easy delivery. Anti fever, emollient; skin ointment; emmenagogue.
	<i>Bombax</i> <i>baobabense</i> P.Beatty.	Alaba (Twt)	T	I, vii	
					oil

Table 7 continued

	<i>Bombax brevicuspe</i> Sprague	Kuntunkuri (Twi)	T	Sb., Lvs.	Anti-rheumatic; dysentery; venereal diseases.
9. Bromeliaceae	<i>Ananas sativa</i> (Linn.)Merrill.	Pineapple		Ft.	-
10. Caesalpinaceae	<i>Cassia alata</i> Linn.	Nsempii (Twi)	S	Lvs.	Purgative; Fungicide; dysentery; gonorrhoea; anti-helminthic; abortifacient.
	<i>Cassia occidentalis</i> Linn.	Mofrabrode (Fante)	H	Lvs., Fl., Sds.	Febrifuge; Diuretic; Anti-microbial; Anti-hypertensive; Purgative; Vermifuge; Skin infection; stomachic; ascites; sore throats; non-septic swellings; coughs, quinine subst. for fever; eye wash in tetanus.
	<i>Senna siamea</i> Lam.	Senna	T	Lvs., Sb., Rt.	-
	<i>Dalium guineense</i> Willd.	Akyitockyi (Ewe)	T	Ft.	Stomach problems; mouth sores; tooth ache; tumours; ease labour in women; palpitations; fevers; refreshing drink.
11. Caricaceae	<i>Carica papaya</i> Linn.	Pawpaw (English)	T	Sds.,L vs., mInfl.,	Amoebicide; anti-helminthic; Stomachic; carminative and digestive; diuretic; wounds
12. Combretaceae	<i>Combretum grandiflorum</i> G.Don.	Whiremnini (Twi)	S	Lvs.	Anti-rheumatic; fresh cuts; Vermifuge, guinea worm expellant; Antimicrobial.

Table 7 continued

	<i>Terminalia ivorensis</i> Linn.	Emire (Twi)	T	Sb., Rt	Sore; ulcerated wound; rheumatism.
13. Compositae	<i>Vernonia amygdalina</i> Del.	Awonwene (Twi)	S	Lvs.	Analgesic; Antipyretic; Upper respiratory tract infection; skin diseases; Antihelminthic; gonorrhoea; fever with diarrhoea; febrifuge; diuretic; purgative; vomitive; tonic; anti-scorbutic; pneumonia
	<i>Vernonia conferta</i> Schreb.	Wudifookete (Twi)	S/T	Lvs., Sb.	Aphrodisiac; laxative; anti-diarrhoea; vermifuge.
14. Connaraceae	<i>Spiropetalum heterophyllum</i> (Bak)Gilg	Ahomakyem/Ahomabosom (Twi)	T	Lvs., Sb.	-
15. Convolvulaceae	<i>Ipomoea batata</i> All.	Santom (Fante)	H	Lvs.	-
16. Cucurbitaceae	<i>Mormordica charantia</i> Linn.	Nyanya (Twi)	H	Lvs.	Anti-stomach ache; anti rashes; Febrifuge; antimicrobial; anti-diarrhoeal; anti-diabetic; antihelminthic; anti-pyretic; wound; cough; fertility.
17. Euphorbiaceae	<i>Alchornea cordifolia</i> (Shun Ex Thonn) Murr. Arg.	Ogyama (Twi)	T/S	Lvs., Rt	Anti-diarrhoeal; dye; leprosy; venereal diseases; anti-snake bite; Antispasmodic; Anti-rheumatic; Antimicrobial; Wound and yaws; Ringworms; Antiprotozoal.
	<i>Bridelia atroviridis</i> Willd. Cor. Spreng	Opamkotokurodu (Twi)	T	Lvs., Sb	Antidiabetic; Mouthwash; Antihypertensive; Antihelminthic; Diuretic; aphrodisiac; gonorrhoea; laxative Body thrush.etc.

Table 7 continued

	<i>Jatropha curcas</i> Linn.	Akancadua (Twi)	T	Lvs	Dysentery; gonorrhoea; sores; wounds; astringent; diuretic; yelloc fever (jaundice); round worms; convulsions; fever; guinea worm sores; purgative.
	<i>Jatropha gossypifolia</i> Linn.	Yesu mogya (Twi)	S	Lvs.	Vermifuge; fever; purgative.
	<i>Mallotus oppositifolius</i> Lour.	Satadua (Twi)	S	Lvs.	Dysentery, vermifuge, anemia, fatigue, lumbago; syphilis; tape worm; aphrodisiac.
	<i>Manihot esculenta</i> Crantz.	Cassava	S	Lvs.	Lactogenic; insect repellent; tumours; cuts
	<i>Phyllanthus amarus</i> Murr.	Abomaguckyir (Fante)	H	Wp.	-
	<i>Securinega virosa</i> Comm. Ex Juss.	Akansa (Twi)	S	Rt.	Analgesic; lumbar/intercostals pain; pleurisy; bronchitis; fever; astringent; gonorrhoea; aphrodisiac; venereal disease; malaria; pneumonia; blood-tinged diarrhea; abscesses.
18. Fabaceae	<i>Cassia nigrican</i> Linn.	Osempe (Twi)	T	Sb., Rt.	Malaria; purgative; vermifuge; appetizer; fever; sore throat.
	<i>Erythrophleum ivorense</i> DC.	Potrodom (Twi)	T	Sb	-
	<i>Parkia biglobosa</i> Benth.	Osokronsroma (Twi)	T	Sb.	Tooth ache; febrifuge; diuretic; leprosy.
	<i>Tamarindus indica</i> Linn.	Borofo (Twi)	T S	Ft.Sb. Lvs	Anti-leprosy; sleeping sickness; febrifuge; eye inflammation sores; swellings; laxative; fever.
19. Labiatae	<i>Hoslundia opposita</i> Vahl.	Abrewaninkwan/nun umerewa (Twi)		Lvs., Rt.	Fever; cold; wound dressing; cough; jaundice; purgative; sore throat; diuretic; cholagogue; stomach troubles; gonorrhoea; anti-convulsant; anti-snake bite; sores; Body itch; anti-epileptic.

Table 7 continued

20. Lauraceae	<i>Persia Americana</i> Mill.	Nunum (Twi) Pear (English)	S T	Lvs. Lvs., Sb.	Anti-bacterial; Febrifuge; diaphoretic; Laxative. -
21. Lecythidaceae	<i>Petersianthus</i> Merr.	Esiaa (Twi)	T	Lvs	-
22. Loranthaceae	<i>Tapinanthus</i> <i>bangwensis</i> Engler & K Krause	Akrampan/Gyankuru du/Okurodu (Twi)	S	Lvs.	-
23. Malvaceae	<i>Gossypium</i> <i>arboretum</i> Linn.	Asaawa (Twi)	S	Lvs.,	Emmenagogue; abortifacient; dysentery; sores; swellings; headache.
24. Marantaceae	<i>Ataenidia conferta</i> Benth.	Abuabua (Twi)		Ft.	-
25. Meliaceae	<i>Azadirachta indica</i> Juss.	Neem (English)	T	Lvs., S b., Rt.	Anti-microbial; anti-inflammatory; anti-pruritic; anti-diabetic; anti-fungal; insecticide; galactagogue; antiseptic; vermifuge
	<i>Carapa procera</i> Aubl.	Kwakuobese (Twi)	T	Sd.	Vermifuge; syphilitic; eye wash; leprosy.
	<i>Guera cedrata</i> Adans Ex Juss.	Kwabohoro (Twi)	T	Sb.	-
	<i>Khaya senegalensis</i> A. Chev.	Mahogany (English)	T	Lvs., Sb.	Haematinic; dysentery; analgesic; febrifuge; anti- convulsant; anti-microbial; anti-pyretic; anti- helminthic; emetic; anti-rheumatic; skin diseases.
	<i>Trichilia</i> <i>monadelpha</i> R. Br.	Tanuro (Twi)	T	Lvs., Sb.	-
	<i>Tetrapleura</i> <i>tetraptera</i> Benth.	Prekese (Twi)	T	Sb., Ft.	Emetic; tonic; anti-fever.

Table 7 continued

27. Musaceae	<i>Musa paradisiaca</i> Linn.	Plantain sucker (English)	H	Lvs.	-
28. Myrtaceae	<i>Psidium gujava</i> Linn.	Guava (English)	T	Lvs.,S b	Anti-microbial; anti-diarrheal; coughs; tooth-ache; astringent.
	<i>Syzygium guineense</i> Willd.	Atena (Twi)	T	Sb.	-
29. Nymphaeaceae	<i>Nymphaea lotus</i> Linn.	Asukooko (Twi)	H	Lvs	Anti-malarial; Insomnia; food; Eczema; Prevent miscarriage, Diuretic; Sedative; eye lotion; Anti-cough; Bronchitis Emollient.
30. Palmaceae	<i>Cocos nucifera</i> Linn.	Kube (Twi)	T	Sb.,Rt. , Ft	Tooth ache and ear ache.
	<i>Elaeis guineensis</i> Linn.	Abe (Twi)	T	Lvs.	Head ache; menorrhagea; anti-trypanosomiasis.
31. Passifloraceae	<i>Adenia lobata</i> Jacq.	Nsurogya (Twi)		St., Lvs	Nose cancer; piles aphrodisiac; diuretic; gonorrhea; purgative; feverish pains; anti-rheumatic; intercostals pains; stomachic; coughs; bronchitis; fever
32. Periplocaceae	<i>Cryptolepis sanguinolenta</i> (Wall) R. Br.	Nibima (Twi)	S		Anti-inflammatory; Antimicrobial; Antihypertensive.
33. Poaceae	<i>Cymbopogon citrates</i> Spreng.	Lemon grass (English)	G	Lvs.	Febrifuge; diuretic; anti-rheumatic; anti-diarrheal;
	<i>Cynodon dactylon</i> (Linn.) Pers.	Aponkyeabodwese (Twi)	G	Sb.	-
	<i>Sorghum vulgare</i> Moench.	Millet (English)	G	Lvs	-

Table 7 continued

34. Rubiaceae	<i>Zea mays</i> Linn.	Aburoo (Twi)	G	Peels.	-
	<i>Canthium glabriflorum</i> Hiern.	Teteadupon (Twi)	T	Lvs.	-
	<i>Morinda lucida</i> (Benth)	Okonkroma (Twi)	T	Lvs.,S b.,Rt	Anti-fever; constipation; piles; gonorrhoea; flavoring; blood purifier; chest problems; abortifacient; yellow fever with haemoglobinuria and haematuria; dysentery; leprosy; astringent.
	<i>Nauclea latifolia</i> Korth.	Adesekankye (Fante)	S	Lvs.,S b.,Rt.	Tonic; anti-pyretic; diuretic; cytotoxic; anti-bacterial; febrifuge.
35. Rutaceae	<i>Citrus aurantiifolia</i> (Christm)Swingle	Lime (English)	T	Lvs., Ft.	Febrifuge; jaundice; laxative; carminative; anti-scorbutic; agent
	<i>Citrus medica</i> Linn.	Ankaatwaree (Twi)	T	Lvs.	Dysentery;
	<i>Clausena anisata</i> Burm.	Samanobiri (Twi)	S	Lvs.Sb Rt.	Anti-rheumatic; Anti-helminthic; Parasiticide; Mosquito repellent
36. Sapindaceae	<i>Paullinia pinnata</i> Linn.	Toantini (Twi)	T	Lvs., Rt.	Febrifuge; Cardiotonic; Dysentery; Anti-protozoal; Haemostatic.
37. Sapotaceae	<i>Synsepalum dulcificum</i> P.&K	Asaa (Twi)	T	Lvs., Rt.	-
	<i>Tieghemella heckelti</i> Piere Ex A. Chev.	Beko (Twi)	T	Sb.	mouth wash.
38. Solanaceae	<i>Lycopersicum esculentum</i> Hill.	Tomato (English)	H	Lvs	-
	<i>Solanum melogena</i> Linn.	Nsusua (Twi)	H	Ft.	-
	<i>Solanum torvum</i> Linn.	Bedrui (Twi)	S	Lvs., Ft.	Haemostat; anti-cough; haematinic; sedative; diuretic; digestive

Table 7 continued

39. Sterculiaceae	<i>Theobroma cacao</i> (Linn)	Cocoa (English)	T	Lvs., Sb	Stimulant to heart, kidney and muscle.
40. Ulmaceae	<i>Celtis adolfi- fidrichi</i> Linn.	Yiso Nkesua (Twi)	T	Sb., Lvs.	Fever; headache; general malaise.
	<i>Celtis Africana</i> Linn.	Esakosua (Twi)	T	Sb	Fever; headache; general malaise; sore eyes.
41. Verbanaceae	<i>Lantana camara</i> Linn.	Ananse dokono (Twi)	H	Aerial part.	-
	<i>Lippia multiflora</i> Moldenke	Saanunum (Twi)	S	Lvs.	Febrifuge; anti-hypertensive; muscle relaxant; laxative; anti-microbial; sudorific; cosmetic adjuvant; kitchen salt; anti-fever; coughs; colds; gastro-intestinal problems; enteritis; fumigation; ear treatment; venereal diseases; abortifacient.

Key:

T- Tree; S- Shrub; H- Herb; Sb.- Stem bark; Lvs.- Leaves; Ft.-Fruit; Rt.- Root; Sds.-Seeds; mInfl.-Male inflorescence; Fl.- Flower;
Rb.-Root bark; Wp.- Whole plant.

Table 8: Plants mentioned for the first time as remedies for malaria in three selected areas of the Central Region, Ghana.

Family	Species	Local Name	Plant form	Parts used
1. Agavaceae	<i>Sansevieria longiflora</i> Thunb.	Twiton (Twi)		
2. Amaranthaceae	<i>Pupalia lappacea</i> (Adans) Mut. Juss.	Mpupua (Twi)	H	Lvs
3. Annonaceae	<i>Annona muricata</i> Linn.	Apple (Thorny) (English)	T	Lvs., Sb.
4. Apocynaceae	<i>Voacanga africana</i> Thou.	Amanafoa/Mpente m/Obede (Twi)	S	Rt.
5. Asteraceae	<i>Acanthospermum hispidum</i> Schrank	Patakunsakum/patakunsoe (Fante)	H	Aerial part
	<i>Aspilia africana</i> Thou.	Mfofobedee (Twi)	H	All parts.
	<i>Lactuca taraxasifolia</i> Thunb.	Dandelion (English)	H	Lvs
	<i>Tridax procumbens</i> Linn.	Fomizegbe (Ewe)	H	Lvs.
6. Bignoniaceae	<i>Kigelia africana</i> Engl.	Nufuten/Nufusuo (Twi)	T	Lvs.
7. Bombacaceae	<i>Bombax brevicuspe</i> Sprague	Kuntunkuri (Twi)	T	Sb., Lvs.
8. Bromeliaceae	<i>Ananas sativa</i> (Linn.)Merrill.	Pineapple		Ft.
9. Caesalpiniaceae	<i>Cassia alata</i> Linn.	Nsempii (Twi)	S	Lvs.
	<i>Senna siamea</i> Lam.	Senna	T	Lvs., Sb., Rt.
11. Combretaceae	<i>Combretum grandiflorum</i> G. Don.	Whiremnini (Twi)	S	Lvs.
	<i>Terminalia catapa</i> Forst.	Abrofonkatee (Twi)	T	Wp.
	<i>Terminalia ivorensis</i> Linn.	Emire (Twi)	T	Sb., Rt.
12. Compositae	<i>Vernonia conferta</i> Schreb.	Wudifookete (Twi)	S/T	Lvs., Sb.

Table 8 continued

13. Connaraceae	<i>Spiropetalum heterophyllum</i> (Bak) Gilg	Ahomakyem/Ahomabosom (Twi)	T	Lvs., Sb.
14. Convolvulaceae	<i>Ipomea batata</i> All.	Santom (Fante)	H	Lvs.
15. Cucurbitaceae	<i>Mormordica charantia</i> Linn.	Nyanya (Twi)	H	Lvs.
16. Euphorbiaceae	<i>Bridelia atroviridis</i> Willd. Cor. Spreng	Opamkotokurodu (Twi)	T	Lvs., Sb.
	<i>Mallotus oppositifolius</i> Lour.	Satadua (Twi)	S	Lvs.
	<i>Manihot esculenta</i> Crantz.	Cassava	S	Lvs.
17. Fabaceae	<i>Erythrophleum ivorense</i> DC.	Potrodom (Twi)	T	Sb.
	<i>Parkia biglobosa</i> Benth.	Osokronsroma (Twi)	T	Sb.
18. Labiatae	<i>Ocimum viride</i> Willd.	Nunum (Twi)	S	Lvs.
19. Lauraceae	<i>Persia americana</i> Mill.	Pear (English)	T	Lvs., Sb.
20. Lecythidaceae	<i>Petersianthus</i> Merr.	Esiaa (Twi)	T	Lvs.
21. Loranthaceae	<i>Tapinanthus bangwensis</i> Engler & Krause	Akrampan/Gyankurudu/Okurodu (Twi)	S	Lvs.
22. Marantaceae	<i>Ataenidia conferta</i> Benth.	Abuabua (Twi)		Ft.
23. Meliaceae	<i>Carapa procera</i> Aubl.	Kwakuobese (Twi)	T	Sd.
	<i>Guera cedrata</i> Adans Ex Juss.	Kwabohoro (Twi)	T	Sb.
	<i>Khaya senegalensis</i> A. Chev.	Mahogany (English)	T	Lvs., Sb.
	<i>Trichilia monadelpha</i> R. Br.	Tanuro (Twi)	T	Lvs., Sb.

Table 8 continued

24. Mimosaceae	<i>Dichrostacys glomerata</i> Forsk	Dundu (Twi)	S/ T	Rt., Lvs.
25. Musaceae	<i>Musa paradisiaca</i> Linn.	Plantain sucker (English)	H	Lvs.
26. Myrtaceae	<i>Syzygium guineense</i> Willd.	Atena (Twi)	T	Sb.
27. Palmaceae	<i>Cocos nucifera</i> Linn.	Kube (Twi)	T	Sb., Rt., Ft
	<i>Elaeis guineensis</i> Linn.	Abe (Twi)	T	Lvs.
28. Poaceae	<i>Cymbopogon citratus</i> Spreng.	Lemon grass (English)	G	Lvs.
	<i>Cynodon dactylon</i> (Linn.) Pers.	Aponkyeabodwese (Twi)	G	Sb.
	<i>Sorghum vulgare</i> Moench.	Millet (English)	G	Lvs
	<i>Zea mays</i> Linn.	Aburoo (Twi)		Peels.
29. Rubiaceae	<i>Canthium glabriflorum</i> Hiern.	Teteadupon (Twi)	T	Lvs.
30. Rutaceae	<i>Citrus medica</i> Linn.	Ankaatwaree (Twi)	T	Lvs.
31. Sapotaceae	<i>Synsepalum dulcificum</i> P.&K	Asaa (Twi)	T	Lvs., Rt.
	<i>Tieghemella heckelii</i> Piere Ex A. Chev.	Beko (Twi)	T	Sb.
33. Solanaceae	<i>Lycopersicum esculentum</i> Hill.	Tomato (English)	H	Lvs
	<i>Solanum melogena</i> Linn.	Nsusua (Twi)	H	Ft.
33. Sterculiaceae	<i>Theobroma cacao</i> Linn	Cocoa (English)	T	Lvs., Sb

Key: T- Tree; S- Shrub; H- Herb; Sb.- Stem bark; Lvs.- Leaves; Ft.-Fruit; Rt.-
Root; Sds.-Seeds; mInfl.-Male inflorescence; Fl.- Flower; Rb.-Root bark; Wp.-
Whole plant

Status of plant species used as anti-malarial drug

Information gathered on the status of the species indicated that the plants have always been readily available on farmlands, river banks, waste places and on play grounds. There were herbal gardens around homes of most respondents. With exception of a few species which required quite long distances to the forest areas, none of them required traveling from one village or town to another for harvest. *Spiropetalum heterophyllum* (Connaraceae) required a special ritual in the deep forest in the night before harvesting. All others could be picked at any time of the day. Most plant species were found to be in all the three study areas (See Appendix 4).

CHAPTER FOUR

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

Discussion

The main objective of this study was to gather information on malaria in the Central Region of Ghana that will contribute to enhanced understanding and control of the disease. This was approached by looking at the incidence of the disease, spatial and temporal distribution of the parasite species involved the commonly encountered stages of the parasite in the blood of patients, the haemoglobin levels of patients and the herbs used in treating the disease in the region.

Incidence of malaria

The gross percentage incidence of malaria disease ranged between 19.5% and 36.3% in the study areas with a mean of 28.6%. Even though these figures might be lower than the national annual average incidence of 42.8%, Afari *et al.* (1993). The observed annual average incidence of 28.6% is nonetheless high indicating that Central Region is malaria endemic area. People who attended the various hospitals and were diagnosed as having the disease included all age groups under 1 year old to above 70 years.

The range of incidence rates <20% to 36.3% in the four study areas shows non-uniformity in the occurrence of the disease in the region. This could be

accounted for by the unique characteristics of each zone. Conditions prevailing in the coastal zone such as availability of health facilities, good roads and better income probably enable more people to report to the hospital promptly. It may also be argued that people from the coastal zone who in general are better educated, are more knowledgeable of the disease and therefore most likely report to healthcare providers when sick. The people in the forest zones lack many facilities and unlike those in the coastal zones, the sick people resorted to self medication confirming the documented findings (Swartout, 1951; Commey, 1989; Adasi, 2005). They may also harbour the parasite but showed no symptoms (asymptomatic) maintaining protective immunity against clinical attacks (Kurtzhals *et al.* 1999). According to Wagner *et al.* (1998), due to the use of protective measures against the disease, individuals tend to have lowered immunity towards it and would easily be infected by the bite of the vector due to less continual exposure. A related question that needs further attention is whether there are less breeding places for mosquitoes in the rural/ forest areas studied. At least in the Cape Coast area where the two urban hospitals sampled are cited, the Fosu lagoon provides a permanent year round breeding site for mosquitoes.

The results also indicated that the disease was prevalent in the region throughout the study period as shown to be consistently higher than 25% monthly incidence rates (Fig.6). However, the apparently low incidence of the disease recorded at CCDH in April and June 2006 (Fig.5a) is not easy to explain considering the fact that June is generally the wettest month in the Central Region

and that mosquitoes breed in pools of water. On the other hand, water collected into pools may be too turbid for the mosquito larvae to breed in or there were floods that flowed continually thus preventing collection of water into pools. The virtually year-round prevalence of the disease suggests that conditions for the propagation of the parasite are favourable continually and hence its endemicity in the region.

In all the study areas, variable age dependent malaria infections were observed and children (1-10 years) were found to be most susceptible to malaria infections (Fig.10). However, children under 1yr like those in 50-60yrs were least infected. This could be attributed to the fact that children under 1yr possess partial immunity from their parents by which they are able to fight some infections. Adult within the 50-60 years group are assumed to be very responsible. They are very conscious about their health and would immediately report of any health disorders for medications. They also could be described as the well dressed group hence they have minimal contact with the vector. It is generally claimed that children and pregnant women are more vulnerable to malaria (WHO, 2000a; Kakkilaya, 2006). The results of the present study show that children under 10 years (including <1yr) had the highest malaria infections in three of the hospitals except UCCH. The infants and children probably had not developed natural immunity against the disease. The highest infection rate (36.8%) at UCCH is among the 21-30 year group. This could be regarded as anomalous, and possibly highly influenced by the ages of the university students who are the major users of that

facility for greater part of the year. The prevalence rate by sex in the various hospitals showed that female patients were more affected than their male counterparts. This could be due to a number of reasons:

- ❖ Complete protection provided by the dresses that males wear i.e trousers and long/short sleeve shirts, pair of socks and shoes. This reduces the mosquito bites.
- ❖ Partial protection provided by the dresses females wear. They tend to expose greater parts of their body to mosquito bites.
- ❖ Differential activity of males and females may also account for the differences in their vulnerability and exposure to infection. Young boys usually spend more time playing outdoors and hence may be more prone to mosquito bites than the young girls who are usually indoors assisting parents with house chores.
- ❖ Among the adults, women are mostly traders who tend to stay out till very late in the night. Some female travelling traders mostly sleep in vehicles and by the roadside when night falls on the way thereby exposing themselves to mosquito bites.
- ❖ Pregnancy with its attendant stress and strain lowers the immunity of women and makes them more vulnerable to malaria (Kakkilaya, 2006).

Distribution of malaria parasite species

Of the four species of the known *Plasmodium* parasites that cause malaria in humans, three of them were encountered during the study. These were: *P. falciparum*, *P. malariae*, and *P. ovale*. *P. falciparum* was the predominant species encountered (97.7%). This supports the various studies conducted by Commey (1989); Meima (1989); Afari *et al.* (1993); Ayirebi-Akomea (1993); Safoa-Appenteng (1994). High *P. falciparum* prevalence has also been recorded in rural areas in Ashanti region (Browne *et al.* 2000) and in Northern Ghana (Koram *et al.* 2003). According to Commey (1989), the biological characteristics of *P. falciparum* are sufficiently different from the other *Plasmodium* species. *P. falciparum* successfully invade erythrocytes, enlarge and multiply. The parasite has the ability to exhibit sub-tertian cycle of 36 hours to facilitate the production of more merozoites (8-24) which have preference for all types of erythrocytes in a short possible time. The merozoites have surface proteins (MSP-1) which enable them to invade erythrocytes. The parasite also possesses var genes that enhance antigenic variation to evade the host's immune system (Connotea, 2008). The sexual forms of the parasite are found to resist chemotherapy and all these attributes make the *Plasmodium falciparum* more virulent thereby causing morbidity and mortality. *P. malariae* and *P. ovale* followed the trend with 1.3% and 0.4% prevalence respectively. Merozoites of *P. malariae* and *P. ovale* have preferences for the matured red blood cells which spend quite a little period in

circulation resulting in their minimal encounter (Commeey, 1989). The efficiency of *P. falciparum* transmission has been attributed to high relative humidity and higher probability of survival of female Anopheles (Afari *et al.* 1993). The authors report that *P. falciparum* infection might possibly suppress *P. malariae* infection when transmission in the former becomes more intense and efficient apparently in the wet season. It was noted that although predominance of *P. falciparum* has been recorded by other workers, the infections recorded for *P. malariae* and *P. ovale* in the present study are much lower than those recorded by Afari *et al.* (1993), ie 20.4% and 2.7% respectively for the two species in the dry season.

The absence of *P. vivax* in the study area corroborated the findings of other workers who have attributed it to the lack of genes expressing Duffy antigens on red blood cells in indigenous people of West and East Africa. Duffy antigen on red blood cells are required for attachment and subsequent invasion by merozoites of *P. vivax*, hence RBCs are refractory to *P. vivax* invasion (Miller *et al.* 1976, 1979, 1986; Centers for Disease Control and Prevention, 2004).

Trophozoites were the most frequently observed erythrocytic stage of the *Plasmodium* species encountered. It could be explained that the rings are sometimes invisible even when using powerful microscopes. Besides, the schizonts of *Plasmodium* species rupture during the evenings and that accounts for the chills and temperature most patients experience. These patients visit the

hospitals in the morning and by the time they get to the laboratory for their blood to be taken for the test, the ring forms might have grown to become trophozoites. Evidence by Trubowitz and Masek (1968) indicated that the polymorphonuclear leucocytes could recognize and phagocytose unprotected merozoites leading to their destruction. As a result of this the merozoites escape from one host erythrocyte much rapidly to invade fresh cells to repeat the erythrocytic schizogonic cycle. The moderate number of gametocytes encountered could be attributed to the fact that most patients treat malaria at home and only go to hospital when they are not fully cured. The disease is thus suppressed and the delay in seeking early treatment may cause several schizogonic cycles for merozoites to transform into drug resistant gametocytes. Some drugs, eg. Chloroquine and quinine when used, quickly transform most ring forms to become gametocytes (Miller *et al.* 1971).

Haemoglobin levels in malaria patients

Generally, the entire population of the patients sampled during the period might be said to be anaemic. The observed reduction of haemoglobin levels with corresponding increased parasitaemia could be attributed to lysis of enormous numbers of red blood cells, phagocytosis of infected red cells, sequestration of infected red cells and suppression of red cell formation (Newton *et al.* 1997). It could be speculated that the anaemia is attributable to malnutrition and helminthic infections or other diseases, but generally the findings agree with what has been

postulated for *falciparum* malaria by Newton *et al.* (1997), Dugbartey *et al.* (1998), Kakkilaya, (2006). There is a significant difference (P values; 5.938, 9.200, 5.256) in haemoglobin levels of malaria patients in terms of sex. Naturally, the normal haemoglobin levels for females (12-14g/dL) are lower as compared to males (14-16 g/dL) and this trend is probably carried through infection. Adult females pass an amount of blood every month as a result of menstruation. The inability to eat properly as a result of busy schedules such as farming and trading to replenish the lost blood may result in anaemic conditions. Pregnant women undergoing stress may suffer anaemia due to improper food intake.

Anti-malarial plants encountered during the study

The total reliance of herbal preparations for curing malaria in the communities of the study areas could be attributed to the non-availability of health facilities, bad roads, poverty and ignorance and this explains why the people have made gardens consisting of the desired plants around their houses. Also the people do not weed the plants on river banks, waste places and on play grounds because they obtain their herbs from these areas. With exception of a few species which required quite long distances to the forest areas, none of them required traveling from one village or town to another for harvest. *Spiropetalum heterophyllum* (Connaraceae) required quite a distance and a special ritual in the deep forest at night before harvesting. It is speculated that the plant is very potent against the malaria disease and that it is harvested on a large scale and this might have caused it to be going extinct. Collectors consider the plant to be sacred and would strip

naked, perform some rituals before harvesting. However, the reasons for doing so were not disclosed.

Plant parts used in the preparation of anti-malarial drugs.

Different parts of the same plant species were used by different people at different locations and in different formulations. Also different parts of different plant species could be used by an individual in the preparation of the decoction, infusion, powder or in the poultice to treat any symptom of general body malaise which they suspected to be "fever". In all leaves were the part mostly used by respondents (49.5%). Reasons could be that leaves are easy to process (Agbovi *et al.* 2002). For example, respondents were fully aware that the disease could manifest itself in many diverse ways. To overcome the complex manifestations of the disease, many plant species and parts were combined in preparing decoctions commonly used for treatment. This explains why in spite of the numerous symptoms such as headache, chills, body pains, joint pains, loss of appetite, parlor, wasting, cough, convulsions in children, blurred vision, vomiting, constipation, backache, frequent urination, diarrhea, tiredness, general weakness and fatigue common plant species were used for treatment. It is strikingly interesting that the concept has also been used in preparation of allopathic drugs as combined therapy to benefit from their additive or synergistic effects. Gurib-Fakim (2006) perfectly confirms the findings of the present study.

The use of decoction by respondents could be attributed to the easy nature by which ingredients are extracted. Besides, it is regularly boiled to prevent it from decaying. These reasons probably explain the preference for decoction to other dosage forms. To almost all respondents, such decoctions are taken 3 times a day, i.e. morning, afternoon and evening after meals. The preparation is left in the pot and heated up daily before use and as a result the extract becomes concentrated. The drug is discarded and a new one prepared when the aqueous extract is seen to be diluted.

The respondents used the decoction routinely as prophylaxis so as to prevent the occurrence of the disease. However, investigation should be conducted to ascertain whether prolong use of decoctions would not impair their immunity thereby making them susceptible to infection. Children and pregnant women mostly had enema from a single plant or a combination of few plant species. This practice among pregnant women and children is disturbing. In the case of the pregnant women, it is more dangerous because the teretogenic effects of the decoctions have not been ascertained. Another reason for the children is that since the immune system is not strong, they might suffer side effects. To reduce the effects of the decoction, some respondents practiced "vapour bath" and "extract bath". This could be explained that the extractive is just diffused through the skin and would not accumulate any toxic substance in the body. All the above stated procedures are employed by the respondents in the treatment of malaria (fever) and the various preparations when administered, enhance their mood and give

them a sense of well-being to enable them carry out their farming and other daily activities.

Most of the respondents could not indicate the exact dose of the herbal preparations that one could administer. However, the herbal practitioners could state the exact dose, expiry date etc because they have been trained by their predecessors. The practitioners otherwise could not tell exactly how much ingredients of particular plants are present and the exact dose that can treat the disease. This and the period within which ones' sickness is treated (maximum curative period) become imaginative. It would have to depend on the patient's immune status and how constant the drug would be administered. The observed inability to state clearly the exact dose, expiry date could be attributed to lack of education of some of the respondents. The literate respondents had some knowledge and used drugs with caution. They never exceeded the dose and would stop decoction intake when they realized total health gain.

The inability of the respondents to attribute untoward effects to any of the herbs could stem from their lack of knowledge of the possible signs and symptoms of adverse reaction in response to a specific herb. Nonetheless, the herbs could not cause any adverse response because small quantities of active ingredients present in herbs act in concert to produce therapeutic effects.

New anti-malaria plants encountered in the study

The popularity of ethnoherbal recipes is attributed partly to the inaccessibility to modern healthcare facilities which are expensive for the poor villagers (Sofowora, 1993; Britwum *et al.* 2000; Gurib-Fakim, 2006; Evans, 2006). In contrast, herbal medicines are cheaper, acceptable to local people and readily available (Sofowora, 1993; Gurib-Fakim, 2006). The respondents considered some herbs new because those plants were not previously used to treat malaria; however, antimalaria herbs of different origin have been hyped by the media in recent times such that most respondents could now use them. The respondents confirm the suitability or otherwise of the herbs when domestic animals fed with the herbs and did not show any adverse effects. The perception was that those herbs did not contain toxic substances that could be harmful and therefore could be used as medicine. The instances cited above might be true to the discovery of new anti-malaria plant species in the Central Region. In furtherance, most of these plant species were not peculiar to one locality; therefore it is believed that they might be true anti-malaria plants known very well to the people of Central Region which would require further research and documentation.

Conclusions

The following conclusions can be made from the results of the study:

1. The incidence of malaria during the study period was found to be higher in the coastal zone than in the forest zone. The mean incidence rate for the coastal zone was 34.4% where as the forest zone recorded 26.9%.
2. There was a more or less uniform occurrence of the malaria disease throughout the year in the study areas.
3. The younger population of up to 10 years (including <1yr) was found to be the most affected group in the region.
4. Females were found to be more vulnerable to the disease than their male counterparts in three of the four hospitals namely; CCDH, SFXH and OLGH. UCCH showed no significant difference in incidence of the disease among the sexes.
5. *Plasmodium falciparum* was found to be the most prevalent malaria parasite which causes the disease in the Central Region all year round. It maintained a predominance rate of over 90% among the different parasite species identified throughout the year.
6. The erythrocytic trophozoites dominated the life cycle stages encountered in the blood of malaria patients examined.
7. Malaria patients became more anaemic as parasite density increased.
8. Eighty nine plant species belonging to 41 families were found to be used in the treatment of malaria in the Central Region. Sixty of the species are being mentioned for the first time as anti-malarial plants in Central Reion.
9. Leaves, stem bark and roots are the main parts of plant species used in the treatment of malaria.
10. Boiling is the main process used in the processing of plant parts as anti-malaria drug.

11. Drug administration is mostly oral, steam bath and enema.
12. Various parts of 2 or more plants species are usually used in combination in treating malaria.

Recommendations

1. Efforts at controlling malaria in the region and the whole country should be stepped up since the youth, women and the working age group are more prone to the disease.
2. Inclusion of anaemia corrective measures in routine malaria management should be considered.
3. Further research should be conducted on the anti-malarial plants throughout the country to produce a national document on the subject paying attention to the plants that have been recorded as anti-malarias for the first time in this study.
4. Further research work should be conducted to identify the active ingredients in the various plant species to enable appropriate drug formulations.
5. Efforts should be made to adopt conservational measures to prevent the plant species from becoming endangered.
6. Practitioners of Herbal Medicine should be given recognition and training to enhance their capacity to supplement orthodox medical delivery.

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APPENDIX 1 (A, B, C & D): OPD Records on Incidence of Malaria in the Four Hospitals

A (CCDH) NOV. DEC. JAN FEB MAR APR MAY JUN JUL AUG SEPT OCT
 05 05 06 06 06 06 06 06 06 06 06 06

MALE

Age Grp.(Yrs.)

	NOV. 05	DEC. 05	JAN 06	FEB 06	MAR 06	APR 06	MAY 06	JUN 06	JUL 06	AUG 06	SEPT 06	OCT 06	TOTAL
<1	30	25	35	25	25	20	30	20	40	30	40	37	357, 5%
1 - 10	90	100	121	71	85	51	100	65	125	148	110	92	1158,17%
11 - 20	150	130	150	104	125	50	122	90	160	198	170	113	1562,23%
21-30	118	110	96	100	110	40	95	81	132	144	151	107	1284,19%
31-40	66	52	64	65	65	32	62	49	90	85	99	68	797, 12%
41-50	41	39	52	55	55	28	58	37	63	69	67	58	622, 9%
51-60	27	32	40	35	40	20	40	23	40	53	36	40	426, 6%
61-70+	32	40	75	45	40	21	30	25	50	50	35	55	498, 7%
TOTAL	554	528	633	500	545	262	537	390	700	777	708	570	6704

FEMALE

<1	40	20	35	35	40	20	35	15	35	40	33	35	383, 5%
1 - 10	135	102	110	100	120	55	130	70	135	170	111	107	1345,19%
11 - 20	130	125	170	125	150	65	135	102	157	224	215	136	1734,24%
21-30	114	91	137	80	107	47	103	76	126	165	185	126	1357,19%
31-40	75	77	84	47	65	25	79	46	95	100	117	79	889,12%
41-50	51	59	55	33	45	19	56	33	75	70	78	62	636, 9%
51-60	34	36	40	25	28	14	38	26	50	50	30	50	421, 6%
61-70+	35	40	50	40	30	15	45	20	62	50	35	58	480, 7%
TOTAL	614	550	681	485	585	260	621	388	735	869	804	653	7245

B (UCCII) MALE Age Grp.(Yrs)	NOV.											TOTAL	
	05	DEC. 05	JAN 06	FEB 06	MAR 06	APR 06	MAY 06	JUN 06	JUL 06	AUG 06	SEPT 06		OCT 06
<1	38	19	16	14	21	20	25	12	23	26	25	29	268, 3%
1 - 10	120	83	76	139	129	112	97	48	84	87	79	81	1135, 11%
11 - 20	127	107	68	137	107	77	132	299	78	125	98	101	1456, 14%
21-30	419	273	244	329	339	340	420	276	295	266	380	349	3930, 37%
31-40	255	163	145	185	193	195	183	154	141	154	200	228	2196, 20%
41-50	134	79	60	77	84	96	99	32	59	93	102	143	1058, 10%
51-60	49	18	19	36	38	47	41	8	40	30	44	80	450, 4%
61-70+	32	9	14	15	15	24	17	9	23	32	28	38	256, 2%
TOTAL	1174	751	642	932	926	911	1014	838	743	813	956	1049	10749

FEMALE

<1	36	15	18	19	25	28	22	9	25	30	22	24	273, 3%
1 -10	116	76	74	139	118	103	101	90	92	85	73	77	1144, 11%
11 - 20	130	103	52	100	103	85	116	186	87	118	89	97	1266, 12%
21-30	419	270	266	315	374	234	395	511	309	252	387	379	4111, 39%
31-40	246	152	141	196	199	152	208	163	145	129	208	250	2189, 21%
41-50	134	71	57	91	92	86	113	11	64	76	98	135	1028, 10%
51-60	40	15	14	32	30	34	40	10	34	34	40	40	363, 3%
61-70+	32	7	8	9	12	20	15	12	29	29	25	30	228, 2%
TOTAL	1153	709	630	901	953	742	1010	992	785	753	942	1032	10602

C (SFXII)	NOV. 05	DEC. 05	JAN 06	FEB 06	MAR 06	APR 06	MAY 06	JUN 06	JUL 06	AUG 06	SEPT 06	OCT 06	TOTAL
MALE													
Age Grp.(Yrs.)													
<1	79	130	83	73	54	83	78	39	25	50	22	36	752, 12%
1 - 10	151	360	165	146	200	179	228	219	146	140	117	133	2184,35%
11 - 20	53	76	59	38	50	51	95	78	36	39	28	59	662, 11%
21-30	59	86	52	45	63	65	86	85	32	41	39	100	753, 12%
31-40	41	59	44	33	42	58	62	73	35	40	28	94	609, 10%
41-50	25	64	31	24	20	39	40	42	30	30	27	53	425, 7%
51-60	21	46	30	21	12	25	38	38	34	22	23	36	346, 6%
61-70+	32	63	42	26	39	28	49	42	43	27	33	70	494, 8%
TOTAL	461	884	506	406	480	528	676	616	381	389	317	581	6225
FEMALE													
<1	56	137	63	46	57	67	66	39	28	22	25	44	650, 9%
1 - 10	160	330	187	146	154	167	138	192	129	127	109	115	1954,26%
11 - 20	68	129	74	46	66	74	109	76	49	65	43	41	840, 11%
21-30	89	126	84	68	100	91	115	104	80	92	70	31	1050,14%
31-40	79	118	80	56	68	89	109	97	83	85	69	29	962, 13%
41-50	58	87	71	42	45	55	90	74	74	79	58	26	759, 10%
51-60	50	35	48	35	40	32	70	63	35	60	40	19	527, 7%
61-70+	46	90	75	46	61	73	74	103	89	90	69	20	836, 11%
TOTAL	606	1052	682	485	591	648	771	748	567	620	483	325	7578

	NOV. 05	DEC. 05	JAN 06	FEB 06	MAR 06	APR 06	MAY 06	JUN 06	JUL 06	AUG 06	SEPT 06	OCT 06	TOTAL
D (OLGH)													
MALE													
Age Grp.(Yrs.)													
<1	48	25	29	32	35	27	22	20	25	25	19	28	335, 9%
1 - 10	85	47	98	50	60	65	52	56	63	60	63	77	776, 21%
11 - 20	70	40	43	57	57	71	63	66	59	70	76	82	754, 20%
21-30	69	29	42	46	52	61	44	45	45	59	51	55	598, 16%
31-40	46	23	33	28	39	44	34	33	37	34	29	40	420, 11%
41-50	30	18	26	22	27	35	27	29	31	27	28	33	333, 9%
51-60	16	14	18	18	15	22	23	23	27	17	26	25	244, 6%
61-70+	33	18	17	28	22	34	31	30	24	27	27	24	315, 8%
TOTAL	397	214	306	281	307	359	296	302	311	319	319	364	3775
FEMALE													
<1	55	26	17	30	37	32	22	24	23	30	37	35	368, 9%
1 - 10	74	50	52	69	62	63	60	66	68	80	74	71	789, 19%
11 - 20	75	46	48	64	77	84	67	68	69	96	76	88	858, 20%
21-30	84	37	33	45	63	61	51	53	58	72	62	63	682, 16%
31-40	46	22	20	28	43	47	36	36	39	47	45	45	454, 11%
41-50	28	15	16	21	33	45	36	36	32	30	43	43	378, 10%
51-60	25	14	11	14	20	35	31	32	22	22	30	21	277, 7%
61-70+	49	17	35	28	24	25	52	50	36	27	39	39	421, 10%
TOTAL	436	227	232	299	359	392	355	365	347	404	406	405	4227

**APPENDIX 2: Raw data on malaria patients relating haemoglobin levels to parasitaemia in the four hospitals.
A (CCDH)**

Age	Sex	Hb	Para. (+)	Age	Sex	Hb	Para. (2+)	Age	Sex	Hb	Para. (3+)	Age	Sex	Hb	Para. (4+)
32	f	9.6	+	55	f	10.3	2+	3	m	8.2	3+		m	2.7	4+
40	f	9.6	+	35	f	12.0	2+	1y5m	f	6.2	3+	1y9m	f	5.1	4+
35	f	14.4	+	27	m	14.7	2+	6	m	7.2	3+	4	m	5.5	4+
27	m	14.7	+	3	f	8.2	2+	14	m	12.7	3+	1	m	6.1	4+
5	m	9.9	+	1.5	m	2.7	2+	1	f	6.2	3+	7	m	10.3	4+
2	f	5.5	+	1y3m	f	5.5	2+	1	f	10.3	3+				
19	f	7.2	+	3.5	f	3.8	2+	2	m	7.9	3+				
9.5	f	8.2	+	45	m	7.5	2+	26	f	8.9	3+				
2	m	12.7	+	1y5m	m	8.9	2+	4m	f	3.4	3+				
1	f	2.7	+	7	m	11.3	2+	4	m	4.1	3+				
4	m	12.0	+	10	m	11.3	2+	3m	m	4.1	3+				
23	f	6.2	+	1y2m	M	4.1	2+	1.5	m	4.1	3+				
66	f	12.3	+	2y3m	f	5.8	2+	65	f	10.9	3+				
1.5	f	3.1	+	1	f	7.5	2+	2	f	4.4	3+				
20	m	14.1	+	19	f	7.9	2+	2	f	6.5	3+				
4	m	9.9	+	5	f	4.8	2+	9	f	12.0	3+				
2	m	6.8	+	1y9m	f	3.1	2+	3	m	12.7	3+				
31	m	14.7	+	45	f	10.4	2+	18	f	9.9	3+				
40	m	12.3	+	2y.3m	f	5.8	2+	17	m	12.3	3+				
10m	m	4.8	+	1.5	m	9.4	2+	4	F	7.5	3+				
8m	f	4.4	+	3	m	8.2	2+	2	f	4.4	3+				
19	m	15.7	+	5	f	8.6	2+	1y3m	f	3.1	3+				
35	f	13.8	+	2	m	9.9	2+	11m	f	7.5	3+				

Appendix 2 continued

37	f	13.7	+	28	f	9.9	2+	13m	f	5.1	3+				
6.5	m	11.6	+	23	f	10.6	2+	1	m	8.6	3+				
1y4m	f	12.0	+	20	f	8.6	2+	5	m	6.5	3+				
8m	f	4.4	+	22	f	12.3	2+	13	f	9.2	3+				

B (UCCH)

Age	Sex	Hb	Para. (+)	Age	Sex	Hb	Para. (2+)	Age	Sex	Hb	Para. (3+)	Age	Sex	Hb	Para. (4+)
		11.4				14.1				7.2				11.4	
		15.2				7.2				8.0					
		15.9				12.5				11.7					
		12.5				14.4				7.6					
		12.1				10.6				6.8					
		10.6				13.7				14.1					
		10.3				13.6				11.4					
		10.6				15.8									
		10.6				14.1									
		11.4				8.0									
		14.9				12.1									
		12.4				11.0									
		11.6				13.3									
		13.7													
		14.8													
		10.3													
		6.5													

Appendix 2 continued

		8.8													
		9.9													
		14.4													
		14.4													
		9.4													
		8.3													
		15.2													
		14.1													
		9.1													
		11.6													

C (SFXH)

Age	Sex	Hb	Para. (+)	Age	Sex	Hb	Para. (2+)	Age	Sex	Hb	Para. (3+)	Age	Sex	Hb	Para. (4+)
25	m	16.4	+	7	m	9.7	2+	2	m	5.1	3+	2.5	m	6.6	4+
3	f	9.0	+	20	f	11.3	2+	3	f	5.8	3+	2	m	12.2	4+
13	m	13.3	+	1	m	12.0	2+	5	f	10.5	3+	1	m	4.7	4+
4	m	15.7	+	30	f	9.7	2+	2	f	14.7	3+	8m	f	6.6	4+
8m	m	8.6	+	40	f	14.4	2+	2	f	14.7	3+	1	m	8.2	4+
3	m	13.0	+	63	f	11.7	2+	8	f	11.7	3+	1	m	1.7	4+
2	m	3.1	+	19	m	12.0	2+	10m	f	10.5	3+	10m	m	8.3	4+
42	f	12.9	+	75	f	9.0	2+	2	f	7.0	3+	3	f	7.4	4+
3	f	8.6	+	1	m	10.9	2+	6m	f	10.7	3+				
1	f	7.0	+	9	m	13.0	2+	9	m	4.3	3+				
9	m	2.7	+	5	m	10.5	2+	11m	m	11.0	3+				

Appendix 2 continued

42	f	7.0	+	3	f	10.5	2+	27	f	7.8	3+				
4	m	7.0	+	2	m	7.2	2+	4.5	f	5.8	3+				
8m	f	8.4	+	3	m	12.3	2+	9m	f	5.1	3+				
35	m	5.1	+	5	f	8.3	2+	7m	m	11.7	3+				
19	m	12.0	+	6	f	10.5	2+	1.5	m	12.5	3+				
4	f	13.3	+	4.5	m	12.5	2+	5	m	11.7	3+				
34	f	12.0	+	64	f	13.5	2+	10	f	13.3	3+				
16	f	10.9	+	7m	m	8.7	2+	3	f	9.0	3+				
22	f	9.0	+	.5	f	6.9	2+	10w	f	7.0	3+				
3.5	f	7.8	+	1	m	3.7	2+	15m	f	11.5	3+				
38	f	12.0	+	6	f	9.2	2+	14m	f	7.4	3+				
1	m	9.7	+	26	f	14.0	2+	1	f	10.9	3+				
6	f	11.0	+	4	f	11.7	2+	9m	f	8.1	3+				
29	f	9.7	+	3	m	5.8	2+	1.5	f	10.5	3+				
9m	f	3.4	+	4.5	m	12.5	2+	3	f	13.6	3+				
1	f	4.6	+	2.5	m	2.6	2+	6 dys	m	10.5	3+				

D (OLGH)

Age	Sex	Hb	Para. (+)	Age	Sex	Hb	Para. (2+)	Age	Sex	Hb	Para. (3+)	Age	Sex	Hb	Para. (4+)
2	m	8.3	+	40	f	10.7	2+	11	m	9.9	3+				
19	f	9.0	+	30	f	10.8	2+	26	f	13.6	3+				
25	f	8.5	+	6	f	5.6	2+	4m	f	10.6	3+				
20	m	11.2	+	3	m	9.3	2+	6m	f	6.8	3+				
24	f	8.0	+	3	m	4.8	2+	4	f	7.9	3+				
31	f	10.1	+	7	f	7.5	2+	3	f	5.4	3+				
25	f	10.7	+	1	f	6.0	2+	4.5	f	10.6	3+				
34	f	9.6	+	2	f	9.2	2+	11	f	9.0	3+				
30	f	9.9	+	4	m	9.1	2+	8	f	6.3	3+				
42	f	11.4	+	8	f	7.7	2+	9m	m	2.6	3+				
6m	f	12.1	+	2	f	3.5	2+	6	f	8.2	3+				
4	f	8.9	+	36	f	9.9	2+	26	f	8.0	3+				
69	m	6.2	+	3	m	5.6	2+	11m	m	3.8	3+				
9	f	7.0	+	2	f	8.0	2+	3	f	9.0	3+				
2	m	7.0	+	2.5	f	6.1	2+	5	m	8.3	3+				
20	m	12.4	+	1	m	5.6	2+	1	m	5.1	3+				
16	f	7.0	+	1.5	m	5.0	2+	1	f	4.5	3+				
1	f	1.5	+	2	f	6.8	2+	2	f	4.9	3+				
10	f	5.0	+	15	m	10.5	2+	9	m	12.4	3+				
78	m	12.9	+	12	f	7.5	2+	9m	m	1.9	3+				
10m	f	4.8	+	1.5	m	10.0	2+	12	f	9.8	3+				
32	m	14.2	+	32	f	7.7	2+								
10	f	5.0	+	2	m	5.0	2+								
14	m	10.1	+	8	m	8.8	2+								

Appendix 2 continued

56	m	8.6	+	4	f	10.0	2+												
99	m	12.3	+	9	f	10.1	2+												
33	f	5.0	+	21	f	11.4	2+												

APPENDIX 3

**MALARIA RESEARCH
QUESTIONNAIRE FOR HERBALISTS/GENERAL PUBLIC**

I. PERSONAL INFORMATION

Name: Male Female

Age: Town:

Occupation:

Marital Status: Married Devoiced Widow

Number of Children: House Number:

Education (Level):

II. TREATMENT RECORD

1. Have you used herbs to treat any malaria patient? Yes No

2. If yes, for how long?

3. (a) About how many patients come to you with malaria per year?

(b) Do they all get cured by you? Yes No

(c) About how many die while receiving treatment from you?

(d) Any possible reason why they die?

.....
.....
.....
.....

4. How do you know they have malaria disease?

Symptoms:
.....
.....
.....

III. PLANT SPECIES IN USE

5. What is (are) the name(s) of the plants you use?

(a) Local Name

(b) Scientific Name

- | | |
|-----------|-----------|
| i. | i. |
| ii. | ii. |
| iii. | iii. |
| iv. | iv. |
| v. | v. |

6. Which of them do you consider to be most effective?
.....

7. How did you get to know about the plant(s)?
.....

IV. PREPARATION AND EFFICACY

8. Which part of plant do you use?

- (i) Fruit (ii) Leaves (iii) Bark (iv) Root

9. How do you prepare it for use as medicine?

10. (a) How does one apply it?

(b) Has there been any modification in preparation and usage with time?

Yes No

(c) If yes why?

11. (a) How do you call your preparation?

(b) What is the dosage?

12. How long does one use it get cured?

Less than 1 week 1 to 2 weeks More than 2 weeks

13. Do patients come back to report of recurrence of the disease after the period of usage? Yes No

14. If yes, what do they complain about this time?
.....
.....

V. DISTRIBUTION AND STATUS OF PLANTS USED

15.(a) Where do you get the plant(s) from? Back yard
On Farmlands Another Village Deep Forest

(b) Do you travel to other parts of the country for it? Yes No

(c) If yes where?
.....

16. Are they readily available? Yes No

17. If no, were they abundant in the past? Yes No

18. Do you think there is the need to conserve this (these) plants?
.....

VI. GENERAL COMMENT

19. What do you think about herbal medicine?
.....
.....
.....
.....

Notes:

.....
.....
.....
.....
.....

APPENDIX 4: Distribution of plant species used as anti- malaria drugs in the study areas of the Central Region

Plants that were encountered in all the three districts

<u>Local name</u>	<u>Scientific name</u>
1. Nsempii	<i>Cassia alata (Linn)</i>
2. Coconut	<i>Cocos nucifera(Linn)</i>
3. Awonwene	<i>Vernonia amygdalina(Del)</i>
4. Prekese	<i>Tetrapleura tetraptera</i>
5. Pawpaw	<i>Carica papaya (Linn)</i>
6. Pear	<i>Persia Americana (Mill)</i>
7. Mango	<i>Mangifera indica (Linn)</i>
8. Mahogany	<i>Khaya ivorensis (A.Chev)</i>
9. Nunum	<i>Ocimum viride (Willd)</i>
10. Ananse dokono	<i>Lantana camara (Linn)</i>
11. Mofrabrode	<i>Cassia Ocidentalis (Linn)</i>
12. Lemon grass	<i>Cymbopogon citratus</i>
13. Nyanya	<i>Mormordica charantia</i>
14. Bedrui	<i>Solanum torvum</i>
15. Plantain sucker	<i>Musa paradisiaca</i>
16. Abomguekyir	<i>Phyllanthus amarus</i>
17. Cocoa	<i>Theobroma cacao</i>
18. Patakunsakum/ patakunsoe	<i>Acanthospermum hispidum</i>
19. Cassia	<i>Cassia siamea (lam)</i>
20. Neem	<i>Azadirachta indica (Juss)</i>
21. Okonkroma	<i>Morinda lucida (Benth)</i>
22. Ogyama	<i>Alchonia cordifolia</i>
23. Nyamedua	<i>Alstonia boonei</i>
24. Ahomakyem/ Ahomabosom	<i>Spiropetalum heterophyllum (Bak)</i>
25. Guava	<i>Psidium gujava (Linn)</i>
26. Lime	<i>Citrus aurantium (Linn)</i>
27. Pineapple	<i>Anana sativa</i>
28. Cassava	<i>Manihot esculenta (Crantz)</i>
29. Akaneadua/ Adaadze	<i>Jatropha curcas (Linn)</i>
30. Cotton	<i>Gossypium arboretum (Linn)</i>

Plants that were encountered in Foso and surroundings

Local Name	Scientific Name
1. Emire	<i>Terminalia ivorensis</i>
2. Toantini	<i>Paullinia pinata</i>
3. Samanobi	<i>Clausena anisata</i>
4. Nufuten/Nufusuo	<i>Kigelia Africana(Engl)</i>
5. Tanuro	<i>Trichilia monadelph</i>
6. Abuabua	<i>Ataenidia conferta (Benth)</i>
7. Whiremnini	<i>Combretum grandiflorum (G.Don)</i>
8. Nsurogya	<i>Adenia lobata (Jacq)</i>
9. Nsusua	<i>Solanum melogena</i>
10. Mpupua	<i>Pupalia lappacea</i>
11. Kwabohoro	<i>Guera cedrata</i>
12. Osempe	<i>Cassia nigrican</i>
13. Potrodom	<i>Erythrophleum ivorense (DC)</i>
14. Kwakuobese	<i>Carapa procera</i>
15. Esakosua	<i>Celtis Africana</i>
16. Nibima	<i>Cryptolepis sanguinolenta</i>
17. Osokronsroma	<i>Parkia biglobosa(Benth)</i>
18. Sasanemasa	<i>Newbouldia laevis</i>
19. Atena	<i>Syzygium guineense (willd)</i>
20. Twiton	<i>Sansevieria longiflora</i>
21. Akansa	<i>Securinega virosa</i>
22. Borofo	<i>Tamarindus indica (Linn)</i>
23. Kuntunkuri	<i>Bombax brevicuspe (Sprague)</i>
24. Dundu	<i>Dichrostacys glomerata (Forsk)</i>
25. Fomizegbe (Ewe)	<i>Tridax procumbens</i>

Plants encountered only in Asikuma and surroundings.

Local Name	Scientific Name
1. Amanafoa/Mpentem/Obede	<i>Voacanga africana</i>
2. Oil Palm	<i>Elaeis guineensis</i>
3. Asukooko	<i>Nymphaea lotus</i>
4. Abrofonkatee	<i>Terminalia catapa</i> (Forst)
5. Aponkyeabodwese	<i>Cynodon dactylon</i>
6. Esiaa	<i>Petersianthus</i>
7. Millet	<i>Sorghum vulgare</i>
8. Tomato	<i>Lycopersicum esculentum</i>
9. Maize	<i>Zea mays</i>
10. Teteadupon	<i>Canthium glabriflorum</i> (Hiern)
11. Sinuro	<i>Alstonia boonei</i> (De Willd)
12. Milkbush/nye me nkyereme	<i>Thevetia peruviana</i>
13. Dandelion	<i>Lactuca taraxisifolia</i>
14. Satadua	<i>Mallotus oppositifolius</i>
15. Beko	<i>Tieghmella heckelii</i> (Piere ex A. Chev)
16. Atoa	<i>Spodias mombin</i> (Linn)
17. Yesu mogya	<i>Jatropha gossypifolia</i>

Plants from both Foso and Asikuma

Local Name	Scientific Name
1. Akrampan/gyankrudu/ Okurodu	<i>Tapinanthus bangwensis</i> (Engler & K Krause)
2. Mfobedee	<i>Aspilia africana</i>
3. Onwenma	<i>Pleiocarpa mutica</i>
4. Abrewanikankye / Nunumerewa	<i>Hoslundia opposita</i>
5. Sweet Potato	<i>Ipomea batata</i>

APPENDIX 5: Preparation of Drabkin's Solution for Haemoglobin Analysis.

The Drabkin's solution used in mixing the blood sample was prepared as follows:

Potassium Cyanide weighing 0.05grams was added to 250 milliliters of distilled water. Potassium Ferro cyanide and Sodium Hydrogen Carbonate weighing 0.20grams and 2.0 grams respectively were added to the solution. Three drops of non ionic detergent (Noidet P40, Saponin 218 and Triton X-1000) were added and the solution was finally made up to one liter with distilled water. The prepared solution was poured into a dark bottle and kept at room temperature for storage. This method was adopted at all the four hospitals.