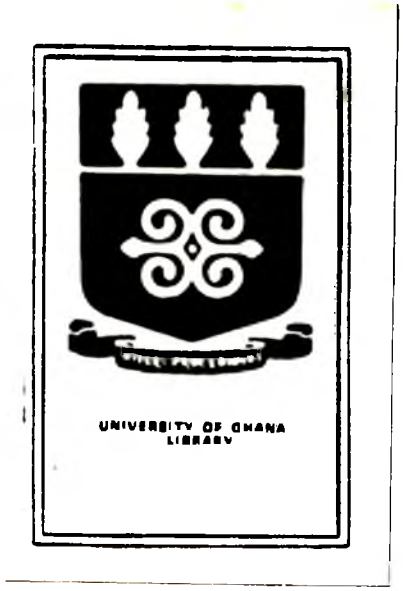

A CASE STUDY ON PARASITIC INFECTIOUS DISEASES IN PREGNANT WOMEN IN FOUR HOSPITALS IN GHANA

BY

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DECLARATION

I do hereby declare that for other people's work cited which have been duly acknowledged, the experimental work described in this thesis was done by me at the Zoology Department under the supervision of Professor Dominic D. Edoh and that this Thesis either in whole or part has not been prescribed elsewhere for any other degree.

.....
Student

.....
Prof. Dominic Edoh
Supervisor

.....
Dr. D. Attuquayefio
Head of Zoology Dept

DEDICATION

To God Almighty
and my parents for making me what I am today

ACKNOWLEDGEMENT

My greatest and sincerest thanks goes to God Almighty who by His mercies has brought me this far in life.

I wish to express my heartfelt gratitude to the following health persons and their staff at the various hospitals for their assistances and also allowing me to use their facilities for my project work.

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ABSTRACT

The prevalence of parasitic infectious diseases among pregnant women in some communities within rural and urban settlements in Ghana was investigated. The study was conducted in Adabraka and Mamobi Polyclinics in the Greater Accra Region, Nsawam and St. Martin's Hospital in the Eastern Region. The aim of this study was to compare incidence of parasitic diseases in pregnant women, and to find out how these infections are related to the age of expectant mothers from these hospitals, also whether there had been an increase or decrease in the incidence of these parasitic diseases over the past few years. Parasitological examinations were carried out on blood urine and stool samples from pregnant women within ages 11-45 years. These samples were collected from 98, 515, 280 and 400 women from Adabraka, Mamobi Polyclinics, St. Martin's and Nsawam Hospitals respectively.

Results show that many pregnant women within ages 16 – 25 years were positive for parasitic infections. Intestinal flagellates infestation was high at Adabraka (90%), Mamobi (77%), and *Ascaris* infestation (87%) at Nsawam Hospital. *Candidiasis* and *Trichomonas vaginalis* infections were also high (80% and 20% respectively).

Malaria infection was significantly higher at Mamobi Polyclinic (64%), followed by St. Martins Hospital (55%), and Adabraka Polyclinic (27%). Incidence of parasitic diseases also decreased slightly over the years whilst *T. vaginalis* and *Candidiasis* infections increased for ages 16 –25.

This evidence shows that parasitic diseases may be a public health problem and effective control measures must be employed to control it. Improvements in water supply, sewerage disposal and general environmental hygiene through human behavioural changes and health education may be used.

CHAPTER ONE

1.0 INTRODUCTION

Parasitic infections have become a constant threat but human beings pay very little attention to it. Parasites are believed to be responsible for about 60% of human ailments. Thus, they constitute a powerful group of pathogens. Although parasites derive maximum benefit from an intimate association with their hosts, they unwillingly also inflict harm and cause diseases to them. They weaken their victims and cause an open avenue(s) for serious health complications (Obeng, 1997).

Living conditions, including ready access to basic domestic needs and systems of inoculations against parasitic infections have improved over the years. This has resulted in better protection, whilst infection rates and occurrence of some parasites in many regions of the world have witness declines. However, the fact still remains that all human beings are potential victims of parasites. If conditions change or people are exposed to parasites in endemic areas, they would become vulnerable to infections (Larry and Janovy, 1996).

Parasites as compared to the host are very small. Their infective stages(-spores, cysts, eggs and larvae) are so small that they are hardly visible without proper scientific tests. It is virtually impossible to detect them in water or food. This makes it easy for parasites to sneak surreptitiously into their host. It is very easy for parasites to find their way into human beings and initiate infection (Obeng, 1997).

Some parasitic infections result from certain human habits such as the practice of eating fish, crustaceans, meat and vegetables raw or insufficiently cooked, and this is responsible for the continued spread of some parasitic worms (Obeng, 1997). In addition,

man made faecal contaminated environments, lack of adequate and safe domestic water supply may lead to the spread of parasitic infections. Over crowded urban areas, lack of shelter, and education and inadequate information on the prevention of infections all contribute to the creation of situations that support parasitic infections at both the personal and community levels. For instance, in malaria infection, the breeding habits of the mosquito vector are supported by existing environmental conditions. Uncovered water-holding cistern, discarded tins and bottles as well as temporary pools serve as suitable places for laying of eggs for the female flies. In parasitic worm infections, under favourable conditions, their infective eggs, larvae, spores and cysts need only minute smears of dirt to conceal them under fingernails, drinking and eating implements, vegetables and some types of fruits. (Larry and Janovy, 1996).

More than 20% of the populations in developing countries are women in their reproductive years. Pregnancy brings high risks of sickness, complications during delivery, disability and death. Pregnant women are normally infected with many of these parasitic diseases, since pregnancy decreases a woman's immunity and makes her more susceptible to numerous complications. The greater number of women with malaria was within the age group 16-25 years (Duerden et al, 1987). According to Steketee et al., (1996), women in highly endemic areas in their first and second pregnancies are more likely to experience malaria as well as high parasite densities. Other studies have shown that with successive pregnancies, both incidence and severity of infection decline with successive pregnancies (Desowitz et al., 1992).

The foetus is then greatly affected because it has no immunity to these parasitic diseases. The main factors of maternal health problems are poverty, poor nutrition and

illiteracy (Duerden et al, 1987). It is also estimated that about 30% of the world's population are anaemic owing to parasitic infections, and most of them are pregnant women (Demaeyer,1981). According to Hercberg *et al.*, (1987), one of the many important factors, which have been found to have an effect on the balance of iron in pregnant women, is pathological blood losses owing to parasitic infections.

Worm infestations and protozoan infections are very common in tropical countries. They are responsible for many cases of anaemia and gastro intestinal infections (Beard, 2000). Parasitic worms, including flatworms, roundworms, which are endoparasites live usually in the intestines, lungs and liver or other internal organs. These have developed adaptations that enable them to avoid the host immune response. During the parasites developmental stages, they are protected by a cyst wall or an outer surface that constantly changes, thus making it more difficult for the host immune system to target the parasite for attack (McGregor,1984).

Malnutrition or anemia caused by intestinal worms can bring complications in pregnancy. Not uncommonly, the diagnosis of nutritional anemia in women with intestinal worms has been indicated in pregnancy. Lack of folic acid and other micronutrients, which may result from intestinal worms infestation or chronic malaria, has been associated with premature placental separation (Fleming, 1989).

Some of the parasitic infections under investigation include *Ascaris lumbricoides*, *Strongyliodes stercoralis*, Intestinal flagellates, Malaria, *Trichomonas vaginalis*, *Candidiasis*, *Ancyslosstoma duodenale*, *Trichuris trichura*, *Entoameoba histolytica*, *Schistosoma mansoni* and *Hymenolepis nana*, *Escherichia coli*, *Schistosoma*

haematobium among others. Studies have shown that all parasitic infections have the potential of reducing fertility, cause anemia and malnutrition (Dion, 1995).

Malaria's effect on pregnancy can be so severe that preventive treatment with antimalarial drugs during pregnancy is recommended in areas where the disease is endemic. Prophylactic treatment in pregnant women is important because pregnancy decreases a woman's immunity and makes her more susceptible to numerous complications from malaria, which includes cerebral malaria, renal failure, and hypoglycemia (Cook, 1996). According to WHO (1994), some tropical parasitic diseases are also known to reduce fertility. For example, schistosomiasis has been associated with infection to the upper genital tract and chronic pelvic inflammatory diseases. Partial fallopian tube blockage that allows sperms to reach and fertilize an egg but does not allow the egg to move to the uterus can result in life threatening ectopic pregnancy (Simarro et al, 1993).

The WHO Health Report (1996) warns that, "Much progress achieved in the recent decades towards improving human health is now at risk. We stand on the brink of a global crisis in infectious disease. No country is "safe". Based on the burden of parasitic infections and its effect on humanity, there is the need for more research to be conducted and implementation of health education especially in developing countries to be increased.

Malaria and other parasitic infections caused by helminth parasites have an enormous toll on human health and well being. In tropical regions including Africa, South of the Sahara, the burden has increased further in recent years. The costs of parasitic infections are enormous when measured in economic terms. Countries with

high rate of Malaria infection are the poorest in the world, and typically have very low rate of economic growth. Many have also experienced declines in living standards over the past thirty years.

There is lost of work time, economic losses associated with infant and child mortality and morbidity, cost of treatment and prevention are typically estimated to be higher than one percent of a country's gross national product (Gottlieb, et al).

1.1 OBJECTIVES OF STUDY

In this work, the prevalence of parasitic diseases in pregnant women in cross-sectional and longitudinal study was done in four areas. The results were compared among the areas.

The objectives are:

(1) To compare the incidence of parasitic diseases in pregnant women from four different hospitals.

(2) To investigate how these infections are related to the age of expectant mothers from these hospitals.

(3) To find out whether there has been an increase or decrease in the incidence over the past few years.

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Prevalence and Distribution of Parasitic Infections

Parasitism may be defined as an association between two species in which one species, the parasite live in or on the host for a significant period of its life and obtains nourishment from it. Parasites live at the expense of its host usually a larger organism. Some parasites of man are normally harmless commensals, notable among these are several intestinal amoeba and flagellates. On the other hand, a great majority of the animal parasites called pathogens are harmful, causing local and systemic damage to its host (Allen, 2000).

There is a worldwide prevalence of parasitic infections. Studies carried out on pregnant women in the rural plains of Nepal revealed that; parasitic infections were extremely common, the prevalence of hookworm and *A. lumbricoides* infection was 74.2 and 58.9% respectively. 88.9% of the women were infected with at least one of the three helminthes assessed. *T. trichrura* was rare (5.3%) *Plasmodium vivax* parasitaemia was present in 19.8% of women (Nzeako, 1992).

In Nigeria, out of the 2,104 pregnant women examined, 38.8% were infected with malaria, 48.3% with intestinal helminthes. The prevalence of hookworm was 14.3% *ascaris lumbricordes* 19.1%, *T. trichiara* (7%), *S. mansoni* (3.4%) Hookworm and *Ascaris lumbricordes* were the predominant helminthes infections accounting for over 69% of all helminthes recorded. The total prevalence of helminthes infections in the area of residence was 55.3% in Bauchi, 44.8% in Jos and 45.4% in Eku. Studies carried out in

Manchiça district in southern Mozambique revealed that overall prevalence of parasitaemia was 23%.(Egwunyenga et al., 2001).

There are various categories of the classification of parasites, according to Boyd (1995); animal parasites, are classified into four divisions or kingdoms;

(i) The protozoa – single celled animal parasites;

(ii) Nemathelminthes – the roundworm parasites;

(iii) Platyhelminthes – flatworm parasites;

(iv) Arthropods – invertebrate animals with jointed appendages.

Generally, microparasites include bacteria, viruses, fungi, and protozoa, whereas arthropods and helminthes are called macroparasites (WHO, 1996). Parasites that live inside of the host's body are known as endoparasites while those that live on the outer surface of their host are known as ectoparasites. (Larry and Janovy, 1996)

Parasites are so unrelenting in their quest to cause infection and perpetuate their existence such that it seems as though they lie in wait for victims (Obeng, 1997). People in both rural and urban homes where environmental and social conditions suit the life cycles of parasites are quite vulnerable to parasitism (Boyd, 1995). Lack of essential facilities resulting in inadequate domestic water supply, unsafe disposal of human waste together with fecal contaminated environments, bad housing, illiteracy, and inadequate information on parasites in particular, encourages the spread of parasites.

The harmful consequences of other tropical diseases to a pregnant woman or her foetus largely depend on the severity of the infection, as well as the stage of pregnancy (WHO,1996). For instance, malaria's effect on pregnancy can be so severe that preventive treatment with antimalarial drugs during pregnancy is recommended in areas

where the disease is endemic (WHO, 1994). Prophylactic treatment in pregnant women is important because pregnancy decreases a woman's immunity and makes her more susceptible to numerous complications from malaria, which includes cerebral malaria, renal failure, hypoglycemia, pulmonary edema, and circulatory collapse. In addition, animal studies have shown that schistosomiasis inhibits reproductive hormonal function because of an immune response to schistosomal eggs (WHO1996).

In order to survive from one generation to the next, parasites have a series of distinct and complex developmental stages and hosts collectively known as a life cycle (Allen, 2000). To understand the various parasites under investigation, one has to take a look at their life cycles.

2.2 Description of Some Parasitic Infections

Ascaris lumbricoides

Ascaris Lumbricoides, it's a large roundworm comparable to the earthworm, the males are 15-30cm long and females are 15–30cm long. It is one of the commonest and most widespread human infections world wide. The infection is found where there are unhygienic environmental conditions. They live in the small intestines and obtain nourishment from the intestinal contents, however they also suck blood and damage the mucosa significantly. (Dion, 1995).

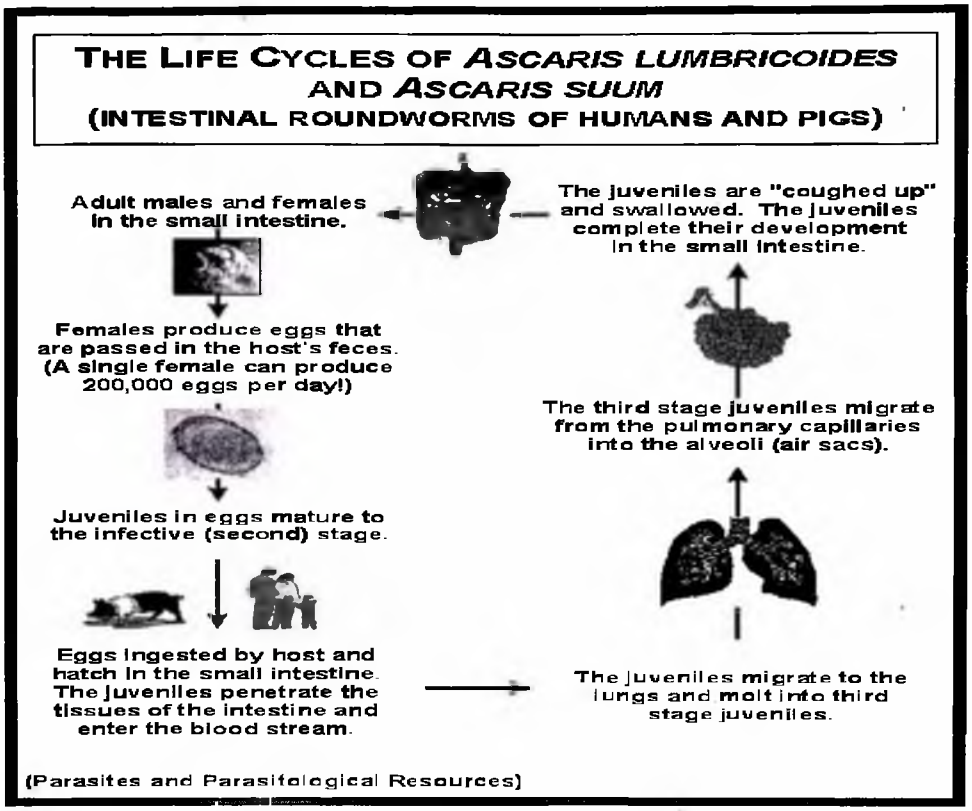
About 1 in 4 of the world's population is infected with Acaris. In China and South-East Asia, it is highly prevalent and common in humid areas in the Central Asian republics, whilst the average rate of infection in Central and South America is 45%. The prevalence is low in Europe and Southern U. S. A, and very high in parts of Africa, about 95% (Cook 1996). Ascaris which is an intestinal nematode, affect maternal-child health.

These retard the intellectual and physical growth of children. Many women with underlying dietary iron-deficiency anemia, habour one of the intestinal worms. Malnutrition and anemia caused by intestinal worms can bring complications in pregnancy. Lack of folic acid and other micronutrients, which may result from intestinal worms or chronic malaria, has also been associated with premature placental separation (Gillies, 1985)

2.2.1 Aetiology And Life Cycle

The fertilized females of Ascaris lay about 200,000 eggs a day which are passed out in stool, and in favourable conditions, develop into the infective embryo. The embryo develops in moist soil in 2–4 months at 36–40°C. It then undergoes one moult, and hatches into an infective second stage. When the egg is swallowed, the rhabditiform larva enters the small intestines, penetrates the mucous, enters the blood stream and reaches the lungs via the right heart (Cook, 1996). Since it cannot pass through the lung capillaries, it burrows through the alveolar wall, enters the respiratory tract, and is carried up to the larynx through the trachea. It moves over the epiglottis and enters the esophagus and is swallowed a second time to reach the small intestine. This whole process takes 10–14 days, during which the larva moults twice, a fourth moulting takes place between 25 and 29 days. In man, the period of infection to the first passage ova in the stool is 60 to 70 days (Cook, 1996).

LIFECYCLE



The effects of worm infection depend on the worm load, the numerous the worms the greater the severerity of the disease. When these worms find their way into the small bowel in the appendix, it may cause appendicitis, when in the biliary tree, causes obstructive jaundice and intrahepatic abscesses. In addition, when worms find their way into the pancreatic duct; pancreatitis results and in the larynx, worms vomited up may occasionally cause asphyxia Ascariditiasis which results in vitamin A deficiency, and malnutrition (Dion, 1995).

In treatment of Ascariasis Piperazine salts is most widely used, other drugs of choice are Pyrantel embonate, Mebendazole, Levamisole, and Albendazole (Dion, 1996). Albendazole and Mebendazole are now widely used because of their broad spectrum activity, low cost and high efficacy. Control of the disease is by periodic mass treatment. Transmission is normally interrupted when chemotherapy is combined with health education and high levels of environmental sanitation (Bradley et al, 1993).

2.2.2 *Hymenolepis Nana*

Hymenolepis nana is referred to as the dwarf tapeworm. Humans and other animals become infected when they intentionally or unintentionally eat food contaminated by insects (Cook, 1996). *H. nana* is the only cestode that parasitizes humans without requiring an intermediate host (Obeng, 1997). The worm's life cycle can be completed in the bowel, therefore infection can persist for years. *H. nana* is a very common parasite in warm climates where sanitation is poor particularly in children amongst whom prevalence often exceeds 10%. Eggs generally measure 30-70 microns in diameter, round to oval and contain six hooked oncosphere (Gilles, 1985).

Infection is usually acquired by the ingestion of *H. nana* eggs primarily from human stool. The ingested ova are activated in the gut and invade the small intestinal mucosa where they encyst within a villus (Cook, 1996). Within 3–4 days the protoscolex of this cercocyst evaginates to become the scolex of an adult worm, which attaches to the intestinal wall. Detached proglottids degenerate during passage through the intestine, releasing their numerous ova from the stool. Autoinfection occurs when eggs spontaneously hatch in the small intestine and initiate a new life cycle (Hotez et al., 1996a).

It causes no symptoms even with a heavy worm burden, some patients complain of headache, dizziness, anorexia, abdominal pain, diarrhea or possibly irritability. Persistent diffuse abdominal pains seem to be the most common symptoms. The infection can be diagnosed by stool or faecal microscopy. (Gillies, 1985)

2.2.3 Ancylostomiasis (Hookworm)

Hookworm infection is one of the most prevalent in tropical and temperate climates, which causes devastating infections of human. Some tropical and clinical investigators rank hookworm as the second most important parasitic human infections next to malaria (Udonski, 1980). Human hookworm infection is usually caused by *Ancyclostoma duodenale* or *Necator americanus.*

This helminthic infection affects an estimated 1 billion people worldwide particularly in the tropics and sub tropics (Udonski, 1980). Attention is focused mainly on Hookworms (*Ancylostoma duodenale* and *Necatur americanus*), because they are the main causes of iron-deficiency anemia. *A. duodenale* is essentially a parasite of southern Europe, the north coast of Africa, northern India, north China and Japan. Whiles *N. Americanus* is the predominant in Western, Central and Southern Africa, and Southern Asia. It is widely distributed in the Southern U. S. A., the Island of the Caribbean. Most of these infections are caused by *Necatur americanus.* Its preferred habitat is the jejunum, where it attaches to the mucous tissue to feed, and secretes an anticoagulant enzyme that causes bleeding (Cook, 1996).

It is estimated that one *Necatur americanus* can cause the loss of 0.031-0.015ml of blood per day, and one Ancylostoma duodenale 0.08-0.02ml/day (Stolzfus et al, 1997).

In Itagua Paraguay, an overall hookworm infection of 59% Age specfic prevalency ranged from 43% (> 40age group) to 79% (19-29 age group) (Ajayi et al, 2001).

Hookworms may enter the body through the skin in early childhood and reach their highest number at the end of adolescence and young adulthood. The degree of anemia they produce is linked to their number. An estimated 30–40 million pregnant women worldwide are infested with hookworms. Estimates of hookworm infection in pregnancy conducted jointly by the Welcome Centre for the Epidemiology of infectious Disease, Oxford and the WHO indicated that some 44 million women are simultaneously pregnant and infected with hookworm. An estimated 3-5 million of these pregnant women harbour heavy hookworm infection that adversely influence intrauterine growth rates, premature and low birth weight (Stolzfus et al, 1997).

A. duodenale have a buccal capsule which contains two pairs of teeth for attaching to the small intestinal mucosa. The eggs are deposited into the lumen of the intestine and are passed out in the faeces and, if deposited in damp soil with temperature range of 23–33°C, would hatch into rhabditiform larvae (Cook, 1996). The larva moults again on the third day, then elongates and fully develops then moves into soil, moults into the filariform larva, which is the infective form.

The larva are most numerous in the upper soil but can move to deeper layers and can survive in damp soil for 2 years. Infection in human host occurs when infective filariform larval penetrates the skin (Cook, 1996). The presence of worm within the skin can cause burning sensation, which may lead to pryritus and pappulovesicular rash persisting for 1–2 weeks (Hotez, 1989). Upon entry into a blood or lymph vessel, the juveniles are carried into the heart and subsequently the lungs, where they penetrate the

air spaces of the alveolar sacs then carried to the glottis, which may lead to pharyngeal itching, nausea and vomiting.

When in the intestine, the larva attach to the mucosal layer and begin suction feeding on the blood and tissue fluids (Gilles, 1985). Besides being infected through the skin from filariform, infection may be by larvae in the soil contaminated by human faeces, or orally via the ingestion of contaminated food, and also be infected through eating uncooked meat containing larvae of *A. duodenale* and also through infected milk (Cook, 1996).

Most patients are asymtomatic, however hookworm infection remains a major cause of hyprochronic microcytic anemia and hypoproteinemia. Pneumonia, cough and sore throat resulting from migrating worm is mild unless infection is heavy. The development of clinical symptoms depends on several factors including worms, species involved and the nutritional status of the host (Hotez,1989).

The diagnosis is made by finding eggs in the stool. A single dose of Mebendazole combined with weekly iron doses is recommended for present infection. Personal precaution against infection is to protect the skin and the feet, ankles and legs from coming into direct contact with infective lavae at infected sites. Wearing suitable footwear is a simple preventive measure. Communities should be educated on the causes of the hookworm infection, also on the safe disposal of human waste as well as be cautioned against consumption of uncooked vegetables (Cook, 1996).

2.2.4 Malaria

Malaria is found throughout the tropics, it is the one of the most important of all tropical diseases, and is of great public health concern, it causes many deaths and much

morbidity. It is widely distributed in the tropical and subtropical zones. Roughly, 110 million cases of malaria develop annually. Some 270 million people are infected, carrying malaria parasites without developing clinical symptoms.

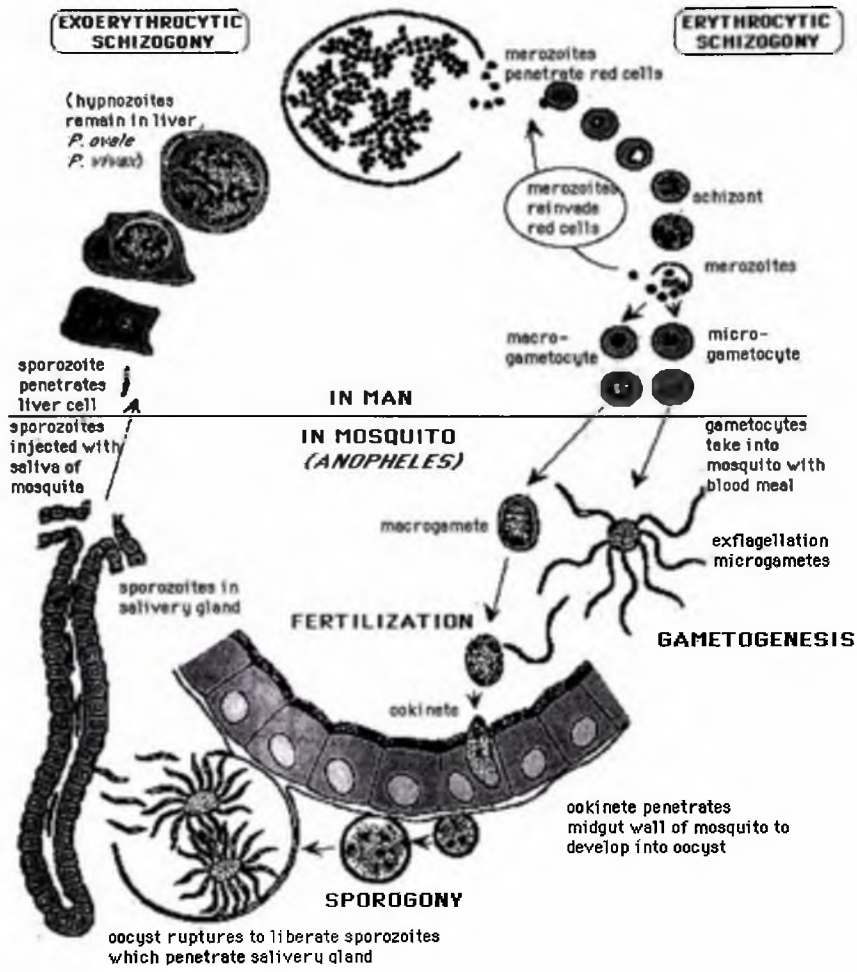
There is an estimated 400 million to 600 million cases of malaria each year. There are four parasite species that cause human malaria belonging to the genus *Plasmodium*, these are *P. falciparum* (causes malignant tertian malaria), *P. vivax* (benign tertian malaria), *P. ovale* (ovale tertian malaria) and *P. malariae* (quartan malaria) (Smith, 1985). In Africa *P. faciparum* predominates. *P. vivax* is more common in central parts of South America, North Africa and Middle East and in the Indian sub continent. *P. Ovale* is rare in sub Saharan Africa whilst *P malariae* is found in most areas but is relatively uncommon outside Africa (Murphy *et al.,* 2001).

In the tropics, malaria remains a major cause of maternal death, abortion, still birth premature delivery and low birth weight. In some holoendemic areas the clinical symptoms and parasitemia are more severe in primiparous than multiparous women, but in many parts of Africa the only definite consequences of malaria in pregnancy seems to reduced birth weights of the infant (Gilles et al., 1993). In non-immune people, cerebral and other forms of *falciparum* malaria are more common in pregnancy and the mortality is higher than in other patients. Malaria contributes to severe anaemia, which complicates pregnancy.

In many tropical countries, studies have shown that progressive anaemia resulting from chronic hookworm infection, malnutrition and repeated attacks of malaria may result in cardiac failure and generalized oedema in late pregnancy (Gilles et al., 1993).

Malaria is usually transmitted by the bite of an infected female anopheline mosquito. When an infected mosquito bites man, numerous sporozoites are injected directly into the blood stream and enter the hepatocytes of the liver. Asexual development takes place in the liver cells or the hepatocytes and develops into pre-erythrocytic schizont, which contains thousands of merozoites. The merozoites develop and differentiate into infective female and male gametocytes, which are ingested into the mosquito during a blood meal. Sexual reproduction of the gametocytes takes place in the mid gut of the mosquito, and through a process of sporogony, sporozoites are released to continue the cycle. (Matteelli *et al.,* 1994).

The life-cycle of *Plasmodium vivax* in man & the mosquito. (after Vickerman and Cox, 1967)



Almost all deaths and severe form of the disease are caused by *P. falciparum*. In pregnant women, all types of malaria can lead to abortion. In *P. falciparum* infection, even in women who are normally immune, pregnancy is associated with an increase likelihood of developing parasitemia and with higher parasite densities especially in the first pregnancy (Dion, 1995).

Anaemia is a common consequence and many women enter labour with dangerously low haemoglobin level. Organ complications such as coma and renal failure are rare in pregnant women living in endemic areas, but among the non-immune pregnant women are liable to the same complications as other adults (Murphy et al, 2001).

If pregnant woman develops severe malaria, fatal loss is common and the maternal mortality may be high. Acute pulmonary oedema and hypoglycaemia are particular complications. The mortality of cerebral malaria in pregnancy is approximately 50%, compared with approximately 20% in non-pregnant adults (Cook, 1996). *P. falciparum* in endemic areas is an important cause of low birth weight, especially in first pregnancy babies who are then at increased risk of dying in infancy, from any of variety of causes. Low birth weight owing to maternal malaria presumably results from the fact that the placenta becomes packed with late stage parasites especially in the first pregnancy (Steketee *et al.*, 2001).

Pregnancy increases susceptibility to malaria, this may be caused by a suppression of systemic and placental cell-mediated immune responses. There is also intense sequestration of *P falciparum* infected erythrocytes in the placenta and maternal anaemia. This normally leads to placental insufficiency and retarded fatal growth. In addition, there is low birth weight and increased possibility of stillbirth in areas of intense transmission. Placental malaria, in which maternal parasites replicate in the placenta and block the exchange of oxygen and nutrients to the fetus, is particularly frequent and severe during the first pregnancies. Placental malaria can result in low birth-weight, mainly by retarding intrauterine growth, which increases the risk of infant death and diseases during the first year of life (Steketee *et al.*, 2001).

The common features of malaria infection are irregular fever, the pattern of regularly periodic fever often does not occur until the illness has continued for a week or more. During the severe form of malaria infection, the organs of the body that may be affected are the liver, spleen and the kidney. They may be enlarged or darkened owing to malaria pigment (Cook, 1996).

Infections with all the four different malaria species have many clinical features in common. These are related to the release of fever producing substances especially during schizogony. Anaemia also occurs most severely in *P. falciparum* malaria because in this infection, cells of all ages can be invaded and even unparasitized red cells may undergo haemolysis. In addition, parasitemia in this infection can be much higher than in other malarias (Smith, 1985).

The early use of chemoprophylaxis has been recommended to prevent fetal infection and the subsequent anemia, which promotes stunted intrauterine growth and low birth weight. There are studies which prove that the anti-malarial prophylaxis have higher weights than the newborns of mothers who did not receive chemoprophylaxis during pregnancy (Viteri *et al.,* 1994). Chloroquine, Proguanil, Mefloquine, Pyrimethamine or Sulfadoxine are a safe and effective drug for antimalarial prophylaxis, although some of these have their side effects. Chloroquine is a drug of choice in areas where resistance to it has not developed, especially by *P. falciparum* (WHO Report 1996).

The use of bed nets normally impregnated with mosquito repellant chemicals, mosquito repellants on the body, environmental sanitation measures, such as education

on preventive actions against malaria for the main purpose of attempting to avoid contact with mosquitoes, are also control measures recommended (McGregor, 1984).

2.2.5 *Strongyloides Stercoralis*

S. Stercoralis is the smallest nematode of the parasitic and free-living forms of nematode that causes the disease strongyloidiasis. These are parasites primarily of tropical regions but extend well into temperate zones in several continents. It is highly prevalent in parts of tropical Brazil, Colombia and South East Asia (Cook, 1996). The disease is contracted by contact with contaminated soil or water. Adult *strogyloides* live in small intestines of humans only, the females live in the mucosa (Dion, 1995). Like most filth borne diseases, strongyloidiasis is most prevalent under conditions of low sanitation standards. This disease is said to be traditionally a disease of the poor uneducated in depressed areas of the world It is estimated that over 90 million people worldwide are infected (Simarro et al, 1993).

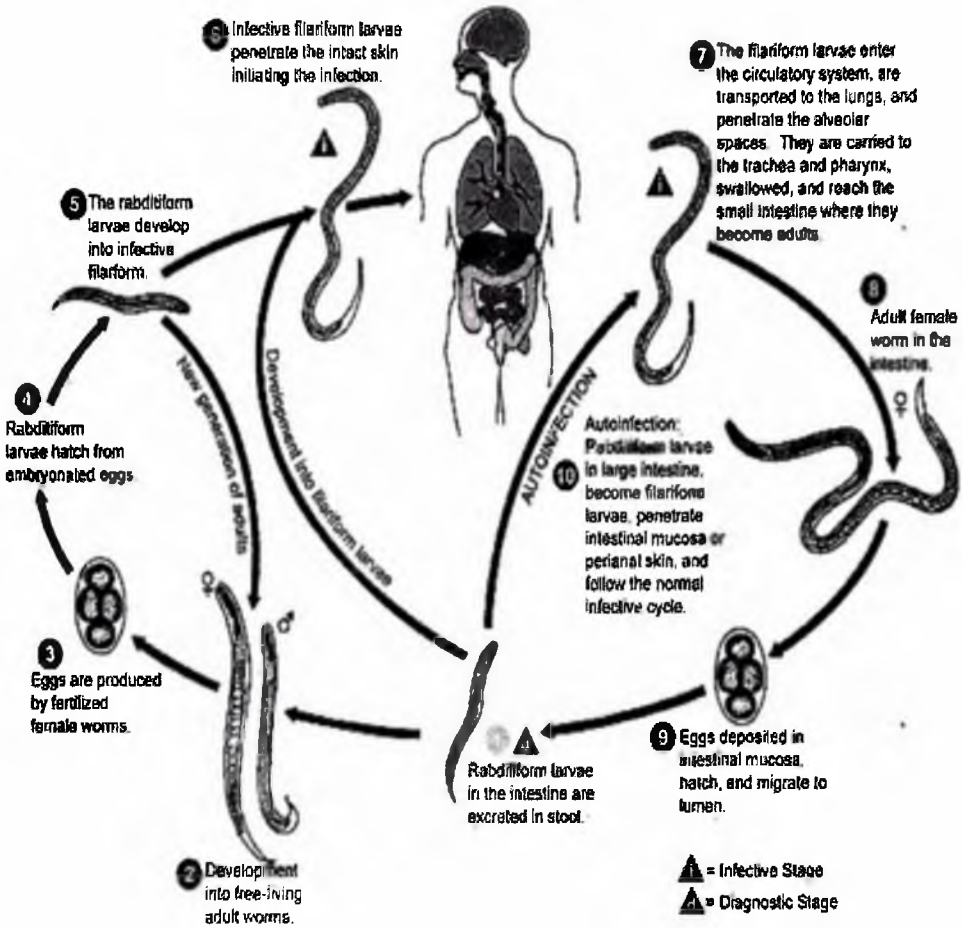
The life cycle consists of two stages, an internal sexual cycle, which involves parasitic worms, and the external sexual cycle, which involves free-living worms. The eggs in the female hatch immediately in the bowel into male and female rhabditiform larvae, which pass in the faeces to continue the external sexual cycle. This free-living rhabditiform develop into adult, which copulate in the soil and produce eggs.

The rhabditiform larvae produced by both parasitic and free living forms are indistinguishable and develop into the infective filariform larvae. This can remain alive in the soil for many weeks. Under unsuitable conditions, the external sexual cycle may be omitted and the filariform larvae infect the definitive host via the skin or buccal mucosa.

The larvae then travel up to the lungs, enter the bronchi, crossover the glottis and pass to the small intestine where they mature into parasitic adults (Nzeako,1992).

Autoinfection arises in one of two ways; the filariform larvae do not pass out in the stools but reinvade the bowel or skin. The other way is when the filariform larvae lodge in the bronchial epithelium and produces further progeny. Autoinfection leads to a build-up in the body of the population so that worms can maintain themselves in the absence of any further infection from an external source. This normally results in the intermittent recurrence of symptomatic episodes. In the case of any breakdown in the immune defenses a rapid increase in the worm burden results in hyperinfection (Cook, 1996).

2.2.5.1 Life Cycle of *Strongyloides stercoralis*



Slight hemorrhage, swellings with intense itching results when juveniles penetrate the skin. A burning sensation in the chest, a cough, and other symptoms of bronchial pneumonia may accompany this phase. The worms migrate randomly through the mucosa, deposited eggs resulting in an intense localized burning sensation or aching pain in the abdomen (Larry and Janovy, 1996).

The infection may be diagnosed when the adults or rhabditiform larvae are seen in the stool. Other recently developed methods used are modified agar plate method, Beesman techniques ELISA and serum IgG reactivity to larval protein. Control of the infection is by shielding the feet and hands by wearing shoes and gloves when farming. Health education on how infection takes place and the ways larvae gets through to unsuspected victims, as well as good personal hygiene is important. Thiabendazole and Albendazole may be used for treatment. (Cook, 1996)

2.2.6 Amoebiasis

Amoebiasis, is an infection caused by the parasitic protozoan *Entamoeba histolytica*. The disease occurs in two forms; acute amoebiasis, characterized by an intense dysentery with blood mucus-filled stools and chronic or latent amoebiasis, which is described by vague intestinal disturbances, muscular aching, loss of weight even constipation or no manifestation at all (WHO, 1995). *E. histolytica* is endemic and has a worldwide distribution, usually found in countries with low socioeconomic conditions. It is the third leading parasitic disease after malaria and schistosomiasis (Cook, 1996).

Approximately 480 million people which are 12% of the world's population are infected and the annual mortality is about 40,000–110,000 persons. In Equtorial Guinea which is located in the Western Africa between Cameroon and Gabon with an estimated

population of 400,000 has a death rate of 14.36%/1,000 individuals owing to amoebiasis (UNICEF,1997).High risk groups including travelers, immigrants, migrant workers, immuno-compromised individual's homosexuals, individuals in mental institutions and children in day care centers (Cook, 1996).

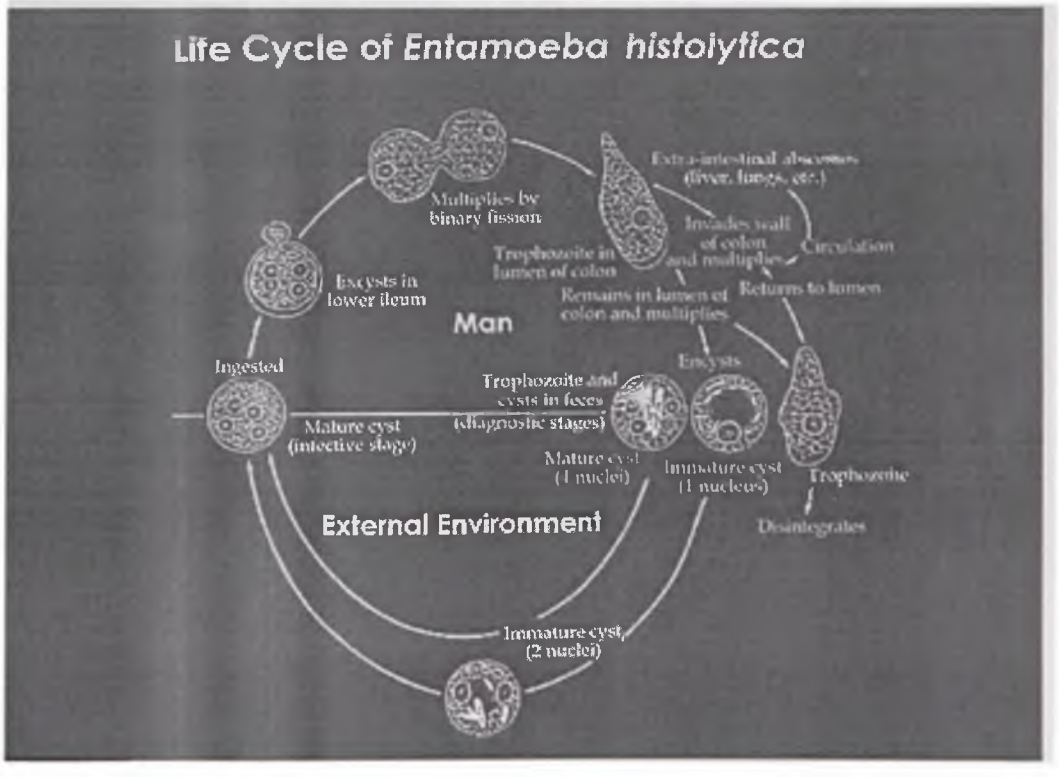
Severe infections occur in very young children, pregnant women, the malnourished and individuals taking corticosteroids. The most important single source of infection is the food handler with chronic amebiasis, especially the one preparing uncooked foods. Other sources of infection are vegetables fertilized with human excreta and drinking water contaminated with sewage. Flies and other insects may spread the cyst mechanically and spread may come from direct hand to mouth contact. In the majority of 85% to 95% of cases, there seems to be a state of balance between the amoebas and the host resulting in mild disturbances, and ulcers heal almost as fast as they are formed (Agi, 1995).

When the amoeba becomes invasive, they breach the mucosa with the aid of cytolytic enzymes, change their diet by consuming the erythrocytes escaping from damaged capillaries and cause ulcer (Dion, 1995). There is increasing ulceration, and violent dysentery in which the stool consists entirely of blood and mucus, this is referred to as acute amebiasis or amoebic dysentery. *E. histolytica* has the capacity to destroy almost all tissues of human body. The intestinal mucosa, the liver and to a lesser extent, the brain and skin are most commonly affected. The cartilage and bone can be eroded by *E. histolytica* trophozoites (Ogunsanya *et al.,* 1994).

The life cycle begins when humans swallow the four-nucleated cyst in food or water contaminated by human faeces. The cyst is digested in the gut to release several

small amoebae (Dion, 1995). The cyst is resistant to gastric acid, and in the ingested form is passed into the small intestine, the amoeba becomes active in the alkaline medium of the small intestine, rapid cell division takes place (Cook, 1996). A precyst form as the cysts migrates through the colon, then evacuated in stool and discharged into the environment. The cysts remain variable and infective for several days in faeces and water but are easily killed by desiccation (Nzeako, 1992).

2.2.6.1 ***LIFE CYLE of E. histolytica***



Diagnosis is by examination of freshly prepared stool for the cysts and trophozoites, culture preparations of amoebae, and ELISA. Drugs used for the treatment

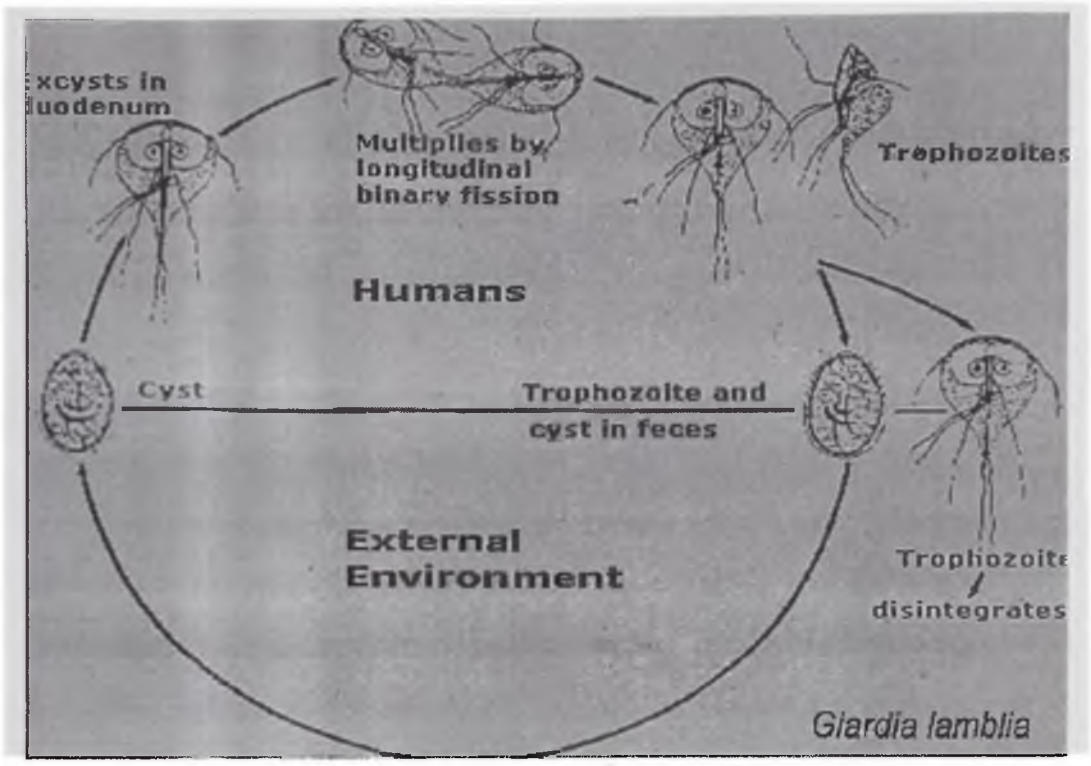
of amoebic infections are diloxanide furoate and iodoquinol. These act on organisms in the intestinal lumen whereas metronidazole, dehydroemetine and chloroquine are effective in the treatment on invasive amoebiasis. The improvement of living standards and establishment of adequate sanitary conditions may control the disease. (Cook, 1996).

2.2.7 *Giardia lamblia*

Giardiasis is the most commonly reported pathogenic protozoan disease in the United States. *Giardia* exists as a trophozoite, which colonizes the proximal small intestine and is responsible for the production of diarrhoea and malabsorption. The cyst is able to exist outside the host in a suitable environment where gardiasis is usually transmitted (Cook, 1996).

Surveys show a worldwide prevalence of *giardia* infection between 2.4% to 67.5%. In 1984, 26,560 cases of gardiasis were reported in United States. Although, Giadiasis is found worldwide, there is a high prevalence in the developing world (20-30%). Age appears to be a risk factor for susceptibility to giardiasis, the infection is more common in infants and young children. Undernutrition may increase susceptibility to infection. Infection is spread from person to person primarily by the fecal-oral route. (Cook, 1996).

2.2.7.1 LIFE CYCLE



Cysts are ingested in contaminated food or water, and in the duodenum trophozoites emerge from matured cyst. The alkaline in the jejunum favours trophozoite growth. Infection of gardiasis results in intense irritation of the lining of the intestine, which makes other types of inflammation worse leading to chronic diarrhoea. *Giardia* cysts and trophozoites are detected in faecal specimens by light microscopy. Drugs commonly used to treat giardiasis are metronidazole, trinidazole, mepacrine, furazolidone and albendazole. (Obeng, 1997).

2.2.8 *Trichuris trichiura* (WHIPWORM)

Trichuris trichiura is a nematode parasite, which inhabits the caecum region of the intestine. It occurs worldwide, it is estimated that 800 million people in the world are infected. Although whipworm infection is common throughout the world, moist climate of the tropics favours the life cycle most (Obeng, 1997). Since soil plays a major role in the transmission of the parasite, children from age 5 – 15, especially in poor communities tend to be the most heavily infected. An estimated 2.2 million people are believed to be infected in the USA, and UK has 13% prevalence. Global estimated rate of whipworm infection is 500–800 million people infected (Dion, 1995).

Trichuris trichiura lives in the caecum and appendix, infection with whipworm is through direct ingestion of infective egg from state faeces. The egg hatches after being swallowed in the intestine, the shell is digested by intestinal juices and the larva emerges in the small intestine. It penetrates the villi and develops, then attaches itself to the mucosa (Cook, 1996). Heavy infections lead to the spread throughout the colon to the rectum. The whipworm does not feed on blood but it absorbs 80% of the hosts juices and nutrients that heavily infected people seem to waste away (Obeng, 1997). In acute cases, there is inflammation of the mucosa, which leads to diarrhea and dysentery and opens up the intestine to secondary infections (Bundy et al., 1987).

Trichuris is frequently associated with Ascaris, hookworm and a secondary infection with *Entamoeba histolytica* causes further ulceration (Cook, 1996). The diagnosis is made by finding the characteristic eggs in the stool by direct smear. An egg count reveals the degree of infection. Drugs of choice are Membedazole and Albendazole. It can be prevented when human waste is effectively managed to make

disposal safe so that there is no contact between people and infective eggs. Hookworm and Trichuriasis causes systemic secondary effects related to iron deficiency, anemia and malnutrition. (Cook, 1996).

2.2.9 *Escherichia coli*

Escherichia coli are rod shaped bacteria, which inhabit the mucosa of the small intestine and colon. *E. coli* is the major aerobic component of the normal intestinal flora but it is also the major cause of diarrhoeal diseases. It is the commonest cause of pyelonephritis and urinary tract infections and an epidemic diarrhea in nurseries of newborn infants (Smith, 1985).

E. coli have a worldwide distribution and are a major health hazard in adults and children in developing countries as well as a major cause of travelers diarrhoea. In community-based studies in developing countries *E. coli* infection are responsible of 15-20% cases of diarrhea. The infection occurs throughout the year but is most common in the wet season and it is as a result of the ingestion of spores in fecal contaminated food and also from dirty fingers and utensils. Shellfish from fecal contaminated water is a major source of infection, if not sufficiently cooked (Cook, 1996).

The harm is largely due to the toxins the E. coli produce and the destruction of the mucosa of the intestine. In addition, it is responsible for infantile gastroenteritis, which is frequently accompanied by bloody stools. The severity of infection is often determined by the state of health of the victim. The main treatment is the assessment of dehydration and replacement of body fluids and electrolytes. Control of infection is achieved by giving good attention to personal hygiene and the prevention of the fecal contamination of food and water are best ways of avoiding infections (Cook, 1996).

2.2.10 Schistosomiasis

Schistosomiasis also known as bilharziasis is the second most important tropical disease in terms of public health importance (Simarro et al., 1992). Schistosomiasis is endemic in 74 developing countries and approximately 200 million people are infected. Some 20,000 people die each year (WHO Report, 1998).

The four types of *schistosoma* that infects man are *Schistosoma haematobium*, which is found in 53 countries in the Middle East, Africa, Island of Madagascar, Mauritius and India. *S. mansoni* is found in 54 countries including Arabia Penisula, Egypt, Libya, Suddan, and Sub Sahara Africa (Rosenblatt, 1999). *S. japonicum* is endemic in China, Indonesia and the Philippines. *S. mekongi* is found in Cambodia and Laos along the Mekong River. The changes in the environment, which, is linked to water resource development, increasing population and population movements have led to the spread of the disease to previously low or non-endemic areas particularly in Sub-Saharan Africa. Examples are the building of the Diama on the Senegal River, Kpong dam in Ghana (WHO Report 1998).

Studies carried out at the Base-Uele district of the Democratic Republic of Congo (formerly Zaire) showed the world's lowest recorded fertility rate during the 1960's. About 50% of women between ages 30 to 34 years were childless. Among conditions thought to have contributed to these are malaria, shistosomiasis, sexually transmitted disease, and nutritional deficiencies. This shows the degree to which tropical diseases can affect fertility. In addition, animal studies have shown that schistosomiasis inhibits reproductive hormonal function as a result of an immune response to schistosomal eggs (WHO Report 1998). In Kenya, those infected with *S. mansoni* had their egg excretion

reduced as CD4 cell count decreased. In women, genital schistosomiasis occurs in 60% individuals infected with *S. haematobiom* (Simarro *et al.*, 1993)

In Africa, it has been estimated that 9–13 million women are afflicted by genital schistosomiasis *S. mansoni* could be diagnosed by the presence of ova in feaces or tissue, whiles *S. haematobium* is by the presence of ova in urine or tissue (Behrman, 2002). Praziquantel, Oxamniquine and metrifonate could be administered to an infected person.

2.2.11 Trichomonal Infection

Trichomonas vaginalis is a pathogenic protozoan infection of the genital tract. Studies from Africa have suggested that *T. vaginalis* infection may increase the rate of HIV transmission (Cotch, 1990). *T. vaginalis* has been found in the vagina of up to 30% of antenatal clinic attendants in certain African centers. In USA, studies revealed that prevalence is higher among women with many partners. Three studies from Nigeria revealed the prevalence of infection in West Africa. In a higher institution prevalence among women was 74% and in men 26%. At Jos, infection rates of 37.6% and 24.8% were recorded in groups of urban and rural women respectively. (Wilkinson et al, 1999).

At Dar-Es-Salaam, Tanzania, investigation revealed that those infected with *T. vaginalis* had an almost threefold higher risk of being infected with the human immunodeficiency virus. The organism is frequently co-existent with another infection such as candidiasis, gonorrhoea, syphilis or HIV infection (Cook, 1996). Humans are the only natural host for *T. vaginalis* and the trophozoites are transmitted directly from one person to another usually by sexual intercourse (Cook, 1996).

A study carried out in Denver hospital, revealed that 14.7% of pregnant women screened were found to be infected with *T. vaginalis* infection. Research carried out in

Northern Kwa Zulu Natal revealed that women within ages 15-49 had at least one sexually transmitted infection (STI), of which *T. vaginalis* infection was one of them (Bulletin of WHO, 1998).

It is estimated that more than 170 million cases worldwide and annually in the United States. There are 2-3 million symptomatic cases. Incidence varies with age, sexual activity, number of sexual partners, presence of other STD's, phase of the menstrual cycle, personal hygiene, number of contacts socioeconomic status, methods of specimen collection and examination as well as sensitivity of diagnosis (Duane,2001).

Pregnant women infected with *T. vaginalis* are more likely to give birth prematurely to children of low birth weight. In addition, children born to mothers with trichomoniasis are more than twice likely to be still born than those who are born to women who do not have the disease. Diagnosis of the disease is by microscopic examination of vaginal discharge on a wet mount. Drugs used for the treatment of trichomonas infection are metronidazde, clotrimazole, clindamycin. The use of condom is very effective in the prevention of the infection (Cook, 1996).

2.2.12 Candidiasis

Candidiasis infection is normally found in the mouth, gastrointestinal tract and vagina (Cook, 1996). Two infections of *C. albicans* exisits, one on the mucous membrane of the mouth (thrush) and the other involving the mucous membrane of the female genitalia, is *Vulvo vaginitis* or *vaginal thrush* (Smith, 1985). Thrush is an especially troublesome infection in newborn infants in hospital nurseries. Most infections are of the endogenous type that is disease caused by one's own microbial flora. Transmission from mother to fetus and venereal transmission are also possible. (Boyd,1995). Maternal

infection is the primary source of organisms that the baby acquires during the birth process, and infection may spread from person to person by contaminated finger, utensils and nipples (Smith, 1985)

Research carried out at Ghent University, Belgium revealed that about 75% of women have candidiasis in their life time. Also at Ghent University at Belgium, studies revealed that 6.3% of women had candidiasis (Bauter et al, 2000).

The sugar content of the urine in pregnancy and uncontrolled diabetes may be a contributing factor to candidiasis. Candida easily gains the ascendancy when dosage with a broad spectrum antibiotic is prolonged. Other pathogenic conditions over which the threat of candidiasis hangs are diabetes, chronic alcoholism, endocrine disorders, malnutrition, and certain kinds of cancers. Diagnosis is by making a wet preparation from the vaginal discharge and adding 10% potassium hydroxide to increase sensitivity. Typical mycelis and yeast cells are seen under the miscroscope.

CHAPTER THREE

3.0 MATERIALS AND METHOD

3.1 STUDY AREA AND STUDY POPULATION

Studies were conducted in four hospitals namely; Mamobi and Adabraka Polyclinics all in the Greater Accra Region; Nsawam General Hospital and Agormenya St. Martins Hospital, in the Eastern Region. The number of pregnant women on whom these parasitological examination were carried on were 515, 98, 400, 280 the hospitals respectively. The studies were carried out from June 2002 to June 2003. Data on the prevalence of parasitic diseases and other infections in pregnant women were also collected from hospital records from 1999 to 2001. Laboratory tests were conducted on samples from pregnant women that reported to the hospital for the presence of parasitic diseases.

3.2 STUDY PROCEDURES

The laboratory techniques used in preparing samples of blood, stool and urine from pregnant women for examination in this study are detailed below:

3.2.1 Blood Film For Malaria Parasite Examination

Patients were pierced on the thumb and a drop of well-mixed blood was placed in the centre of a clean glass slide. Using the edge of another slide, the blood was spread over an area of 10-12 mm. The correct thickness of the film was determined by placing the slide on a piece of newspaper and making sure that small prints were just visible through the blood. This was dried at room temperature and then stained with Giemsa stock (1 in 10 dilutions) for 10 minutes, after which it was washed gently in buffer and

left in upright position to dry at room temperature. This slide was then mounted in glycerin and covered with slip, this was placed under a microscope and observed with x 40 objective for the presence of parasites in the blood.

3.2.2 Stool Examination

Faecal material (1 gram) was emulsified in saline and brought to a suspension using an applicator stick. One drop of stool suspension was placed on the slide and 1 drop of Lugol's iodine was added to a second drop, mixed and covered with a cover slip. This slide preparation was for cyst detection and then observed under a microscope. Both preparations were examined systematically with x10 objective and x40 objective for detailed morphology of parasites.

3.2.3 Urine Examination

Urine sample from patients was poured into a 5ml labeled tube to half-full and spun at 3000 rpm for 3-5 minutes. The supernatant was decanted and the deposite resuspended in a residual fluid. A drop of urine deposit was then placed on a glass slide and a cover slip applied gently and examined using a x40 objective for detailed morphology.

3.2.4 High Vaginal Swab (HVS) Routine Examination

The laboratory techniques for vaginal infections in pregnant women, was done by putting a few drops of saline in a tube to cover the cotton bud of the vaginal swab from patients. The saline was then mixed with the sample thoroughly. A grease-free slide was labeled and a wet mount was made from the contents in the tube by putting about two drops of the contents on the slide and a cover slip applied. The wet mount was examined under the microscope by first scanning with X10 objective and using X40 objective for

detailed morphology. This was used to look for *Candida* species and motile trophozoites of *Trichomonas vaginalis*.

The abundance of parasites in the samples were determined using the Mansa-Bahr & Bell scale shown below;

+	-	1 – 10 parasites per field view
++	-	10 – 50 parasites per field view
+++	-	50 – 100 parasites per field view
++++		over 100 parasites per field view

3.2.5 Data Analysis

The study used Statistical Package for Social Scientist (SPSS) for the analysis of data. The frequency with the corresponding percentages of the various age groups of the pregnant women were tabulated. t-test was used to test the significance of the various levels of parasitic infection on the pregnant women under study.

ANOVA (analysis of variance) was also used to test the significance of the parasitic infection between and within the various age groups. Finally, Pearson correlation coefficient was used to determine the correlation between the age of the pregnant women and the various parasitic infections. In all, 5% level of significanee was used to accept or reject a statistic.

CHAPTER 4

4.0 RESULTS

4.1 MALARIA INFECTIONS

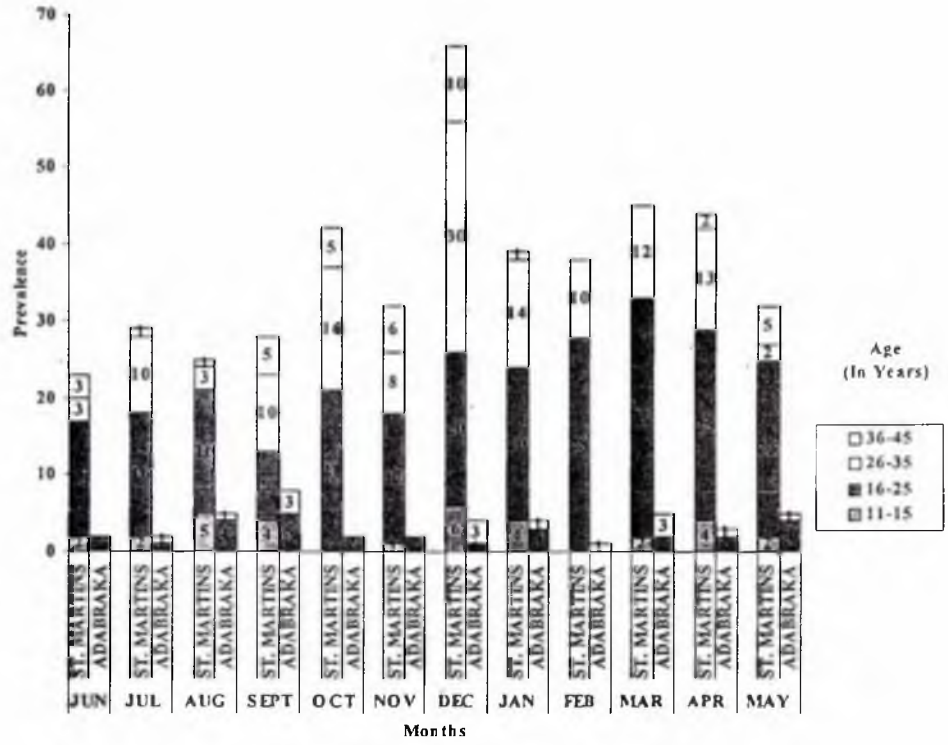
There was a seasonal trend in the prevalence of malaria in pregnant women at St. Martins and Adabraka hospital between June 1999 to May 2000. The highest prevalence of infection was in December, (70%) followed by March (50%) and April (48%) at St Martins hospital. However, June to September recorded relatively lower prevalence. At Adabraka Polyclinic, a relatively lower prevalence was recorded throughout the months (Figure 4.1).

The study also revealed that the highest number of pregnant women infected with malaria were within the age group of 16–25 in both hospitals, with the exception of December at St Martins Hospital where most of them fell within ages 26–35 (Figure 4.1).

Similarly, a high prevalence was observed in June 2000-May 2001 at the St. Martins Hospital (Appendix1: Fig. 4.2). However, June had a higher prevalence (30%) as compared with the previous year. The trend at Adabraka Polyclinic was similar to the previous year (23%). It was observed that there were no malaria infection in the months of December, March and April.

The age group with highest number of pregnant women infected with malaria at Adabraka Polyclinic in June 2000 – May 2001 was within ages16-25, which is similar to that of June 1999 – May 2000 with the exception of the months of January and February, where the highest numbers of women were within the age group of 26 -35 (Appendix 1: Fig 4.2).

Figure 4.1



Prevalence of malaria in pregnant women from two different hospitals; ST. Martins and Adabraka Hospital (June 1999-May 2000)

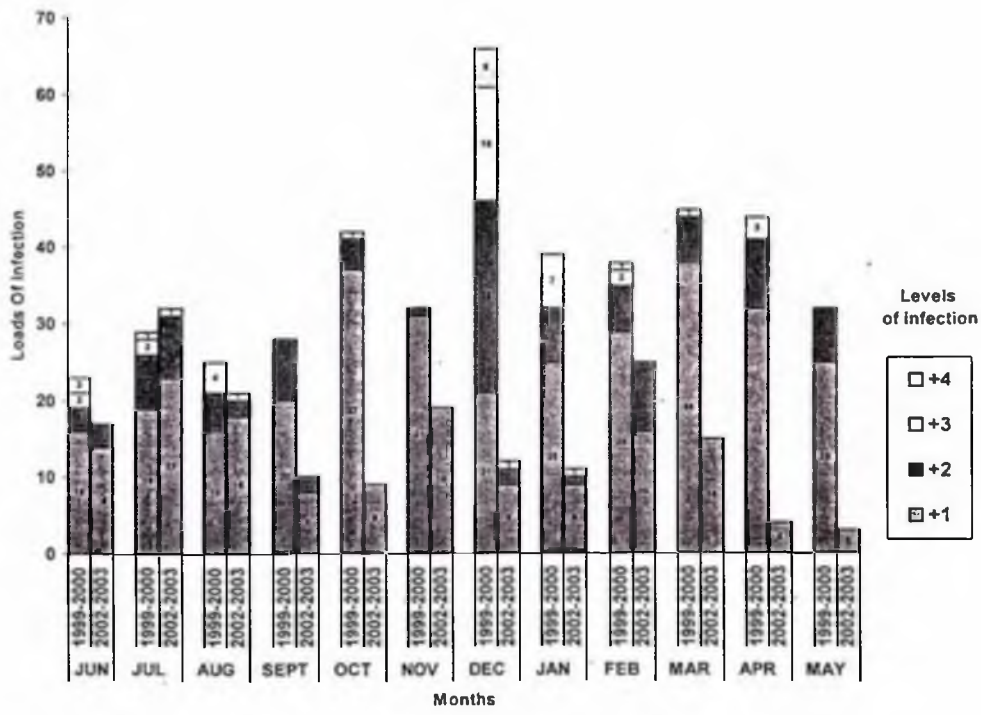
In June 2001–May 2002, the highest prevalence at St. Martin's Hospital was recorded in July and August, and the least in December and May. The prevalence at Adabraka Polyclinic remained low with no malaria infection recorded in October, December and April. Mamobi Polyclinic recorded high malaria prevalence throughout the year (Appendix 1: Fig 4.3).

Many pregnant women with malaria infection between June 2001-May 2002 at Mamobi, Adabraka and St. Martin's hospital were within the age group of 16–25 whereas many of the pregnant women at St. Martins Hospital fell within ages 26-35. Cross sectional studies done into the prevalence in June 2002-May 2003 revealed that, the trend

of infection was low throughout the year in St. Martins and Adabraka Hospital. However, Mamobi recorded higher malaria prevalence, with the highest in October, January and November and the lowest in February. In June 2002–May 2003, many of the pregnant women with malaria infection in the three Hospitals were within ages 16–25 (Appendix1: Fig 4.4)

In assessing the highest and lowest parasitemia levels over the two-year period for St Martin's Hospital, it was realized that June 1999-May 2000 had the highest load of infection with the exception of July (1999 – 2000). The lowest load of infection was observed in July 2002–2003 (Figure 4.5).

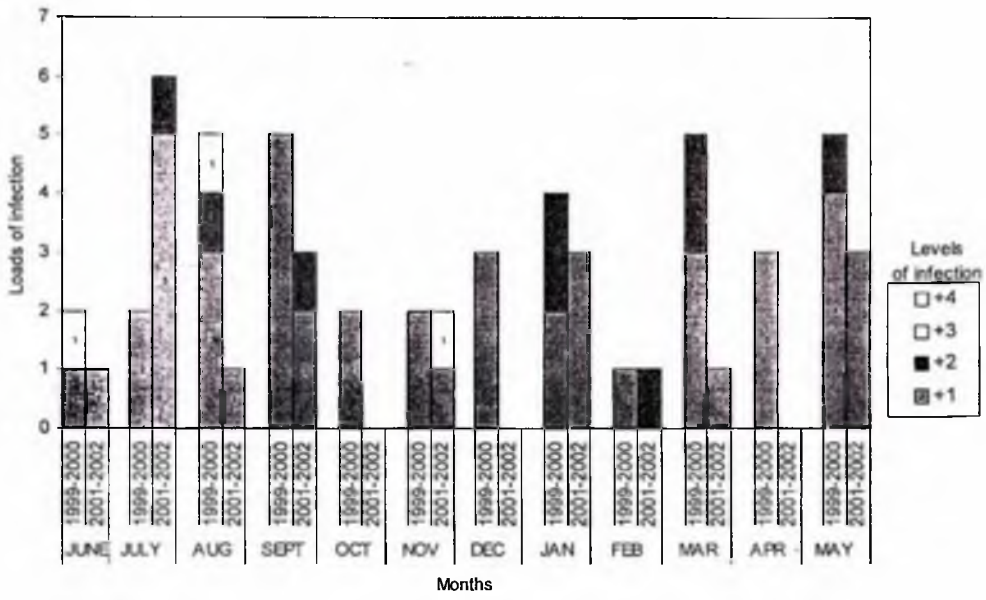
Figure 4.5



Presenting Highest and Lowest Parasitemia Levels Over 2years for St Martins Hospital St. Martins Hospital, June 1999–May 2000 (Highest), June 2002–May 2003 (lowest)

A similar trend was observed at Adabraka Polyclinic except for the month of July, where the highest load of infection was recorded in 2001–2002. In November and February the same loads of infection was recorded for the two years (Figure 4.6).

Figure 4. 6



Presenting highest and lowest parasitemia levels over 2years at Adabraka Polyclinic, June 1999–May 2000 (Highest), June 2002 – May 2003 (lowest).

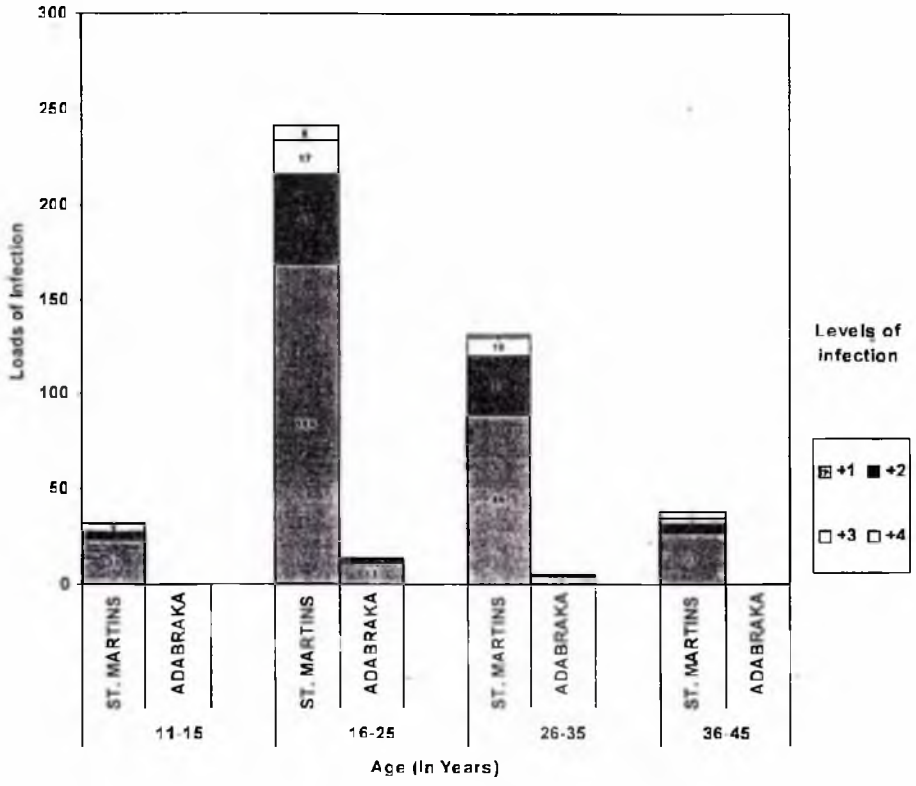
At Mamobi Polyclinic, a similar trend was also observed except for the following months July, October, November, December and January where the highest loads of infection were in the year 2003 (Appendix 1: Fig 4.7).

Parasitemia levels

In the three hospitals, Adabraka and Mamobi polyclinics and St Martin's Hospital many of the pregnant women had malaria infection density of +1 level of infection and few had +2 and +3 levels of infection. In investigating the relative parasitemia levels in the various age groups at the three different hospitals, it was revealed that between June

1999-May 2000, pregnant women within the age group of 16-25 at St Martins had the highest load of infection and those with least was within the age group of 11-15 (Fig 4.8). A similar trend was observed throughout the other years (Appendix1: Fig 4.9- 4.11).

Figure 4. 8

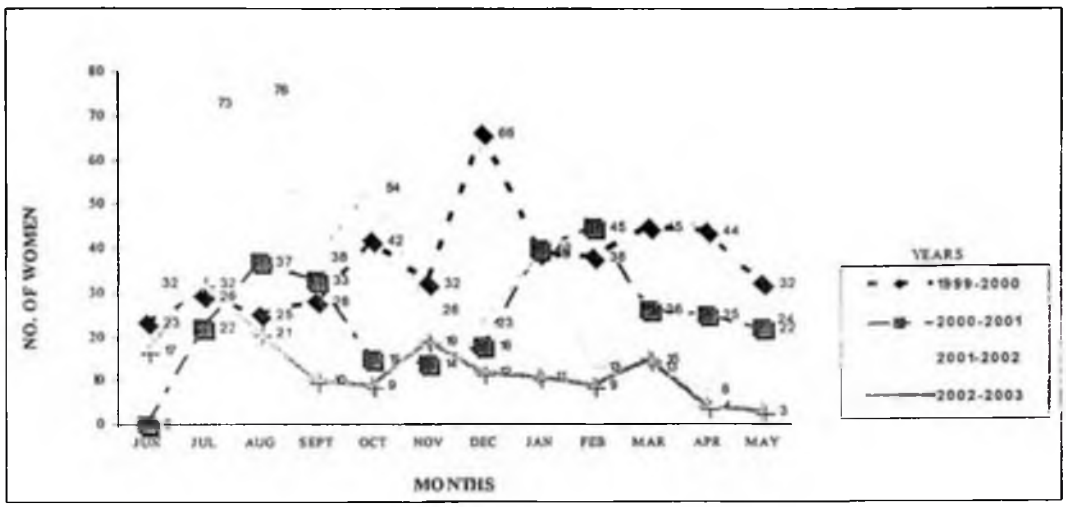


Presenting the relative parasitemia levels in pregnant women at St. Martins Hospital and Adabraka Hospital (June 1999 – May 2000).

The study of the malaria infection among pregnant women in (June 1999- May 2000), at St. Martin's Hospital, revealed the highest peak season in December and the minor peak season in June. Whilst in June 2000- May 2001, the major peak season was observed in August and the minor peak in October. In June 2001 to May 2002, the trend was similar to the previous year, in which the major peak season was observed in August

and the minor peak season in April. In 2002 to 2003, the major peak season was in July and the minor peak season was in May (Fig 4.12).

Figure 4.12



Longitudinal survey of malaria infection among pregnant women in St. Martins Hospital (June 1999-May 2003).

The major peak malaria season at the Adabraka Polyclinic (June 1999-May 2000), was in September and the minor peak season in February, whiles in June 2000-May 2001, the major malaria peak season was in August and the minor in December. The monthly malaria trend in June 2001-May 2002, showed the major peak season in July and the minor peak season in October, December and April. The trend was different in June 2002-May 2003, in which the major malaria peak seasons were in January and March and the minor peak season was in May (Appendix 1: Fig 4.13). At the Mamobi Polyclinic, 2001-2002 showed a major peak season in malaria infection in January, and a minor peak in July, whiles the subsequent year (2002-2003) revealed major peak season in November and January and a minor malaria season in February (Appendix 1: Fig 4.14).

At Mamobi polyclinic, the women who reported at the antenatal clinic between June 2001-May 2002. Within age group of 16-25 had the highest number of women infected with malaria (61.1%) followed by age group of 26-35 (34.2%). A similar trend was observed in June 2002- May 2003. At St. Martins Hospital between June 1999-May 2000, many women with malaria were within ages 16-25 (53.8%) followed by 26-35 age group (30.0%).The same trend was observed as the years progressed. At Adabraka polyclinic, a similar trend was observed where ages 16-25 had the highest number of infections (65.1%) in June 1999-May 2000. This increased to 80% in June 2000- May 2001 (Appendix 2: Table 4.14- 4.23).

4.2 URINARY INFECTIONS

A cross-sectional study of urinary infection among pregnant women who reported at Adabraka Polyclinic from June 1999-May 2000 revealed that a greater percentage had *Candidiasis* infection as compared to *T. vaginalis* infection. The age group which were most infected were within 16–25 years. 77.3% of pregnant women in this age group had *Candidiasis* while 22.7% had *T. vaginalis* infection. Within the age group of 26 – 35, 81.0% had *Candidiasis* infection while 19.0% had *T. vaginalis* infection and age group of 36 – 45 years, 75% had *Candidiasis* infection while 25.0% had *T. vaginalis* infection (Table 4.1). This trend was similar in all the years for the other hospitals (Table 4.2–4.5 at Appendix 2).

Table 4.1

AGE	URINE INFECTIONS	Statistics	Months											Total
			Jun	Jul	Aug	Sep	Nov	Dec	Jan	Feb	Mar	Apr	May	
16-25	URINE INFECTIONS	*Candidiasis* Count	2	2	2	2	2	1	2	2	1		1	17
		Candidiasis % of Total	9.1%	9.1%	9.1%	9.1%	9.1%	4.5%	9.1%	9.1%	4.5%		4.5%	77.3%
		T. vaginalis Count	1	3	1									5
		T. vaginalis % of Total	4.5%	13.6%	4.5%									22.7%
	Total	Count	3	5	3	2	2	1	2	2	1		1	22
		% of Total	13.6%	22.7%	13.6%	9.1%	9.1%	4.5%	9.1%	9.1%	4.5%		4.5%	100.0%
26-35	URINE INFECTIONS	*Candidiasis* Count	3		3		2	2	2			2	3	17
		Candidiasis % of Total	14.3%		14.3%		9.5%	9.5%	9.5%			9.5%	14.3%	81.0%
		T. vaginalis Count	2		2									4
		T. vaginalis % of Total	9.5%		9.5%									19.0%
	Total	Count	5		5		2	2	2			2	3	21
		% of Total	23.8%		23.8%		9.5%	9.5%	9.5%			9.5%	14.3%	100.0%
36-45	URINE INFECTIONS	*Candidiasis* Count	1						1				1	3
		Candidiasis % of Total	25.0%						25.0%				25.0%	75.0%
		T. vaginalis Count	1											1
		T. vaginalis % of Total	25.0%											25.0%
	Total	Count	2						1				1	4
		% of Total	50.0%						25.0%				25.0%	100.0%

Table 1: Adabraka Polyclinic (Jun 1999 - [a]2000)

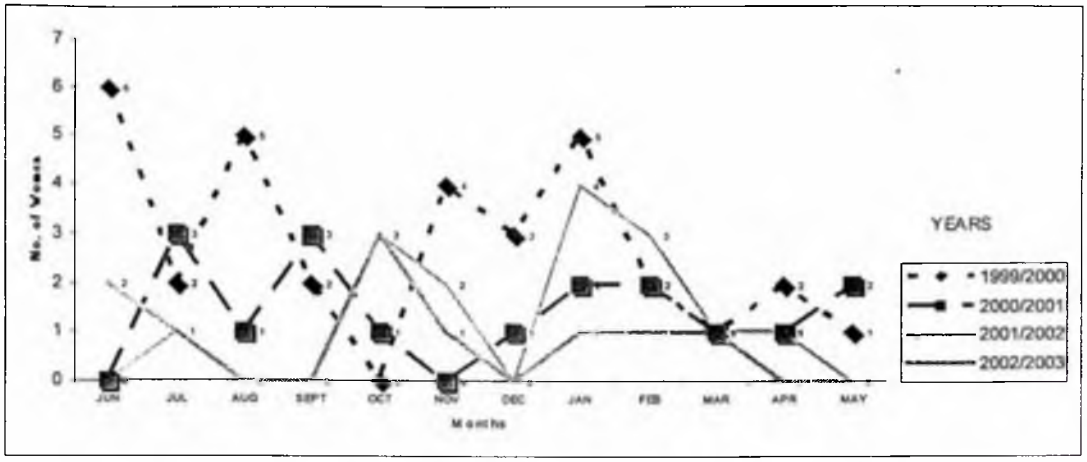
[a]. No recordings for October

Prevalence of urinary infection among pregnant women at Adabraka Polyclinic.

Investigations into the urinary infections among pregnant women at Adabraka hospital also revealed a decrease in the number of women with candidiasis infection within the various age groups in the subsequent years. In the case of *T. vaginalis*, no infection was recorded as the years progressed (Appendix 1: Fig. 4.15 and Fig. 4.16). At Mamobi Polyclinic there was a little decrease over the years except within age group of 16–25, where there was an increase in the number of women infected with *candidiasis* in the year 2002-2003 (Appendix 1: Fig 4.17). In the case of *T. vaginalis* infection, with the exception of the age group of 11–15, there had been an increase in the infection among the various age groups as the years progressed at Mamobi Polyclinic (Appendix 1: Fig 4.18).

The overall longitudinal survey on Candidiasis infection revealed a trend that indicates a decrease across the years (Fig 4.19).

Figure 4.19



Longitudinal survey of urinary infection at Adabraka Polyclinic (June 1999 – May 2003 For Candidiasis)

In assessing the highest and lowest levels of urinary infections among pregnant women at the Adabraka Polyclinic, it revealed a decrease in infection from 1999 to 2003 was observed (Appendix 1: Fig 4.20).

4.3 WORM INFESTATIONS IN STOOL

The hookworm eggs observed measured between 56 – 60µ by approximately 30 40µ and barrel shaped. *Stronglyliodes stercolaris* larvae measured between 180 - 380µm by 14-20µm whilst the ova measured between 50-58µ by 30-35µ and oval in shape. *Ascaris* ova which is ovoid in shaped measured between 47-75µ by 30-50µ in size *E. coli* is also rod shaped. *H. nana* eggs were round and measured between 30-46µ, *S. mansoni* with a prominent lateral spine measuring 114-175µm by 45-70µm. *Trichuris trichura* egg with bipotarplugs measured 50-55µm by 22-24µm.

Parasitological studies carried out on worm infestation among pregnant women in the three hospitals; Adabraka and Mamobi Polyclinic and Nsawam Hospital, revealed

that some of the pregnant women were infested with Intestinal flagellates, Hookworm, Strongyloides, Ascaris, *Trichuris*, *S. mansoni*, *E. histolytica* and *H. nana* and protozoan *E. coli*,. The most common intestinal flagellate observed in the hospitals was *Giardia lamblia*. At Adabraka Polyclinic the study revealed that the age group of 26-35 had the highest number of worm infested women for 1999 – 2000 and 2000 - 2002 (52.6% and 48.5% respectively) (Table 4.6a and 4.6b).

Worm infestation in pregnant women at Adabraka Polyclinic (June 1999-May 2000)
Table 4.6a

Age (in years)	Statistics	TYPE OF WORM			Total
		Intestinal flagellates	*Hookworm*	*Strongyloids*	
11-15	Count	1			1
	% of Total	.9%			.9%
16-25	Count	44	6	2	52
	% of Total	37.9%	5.2%	1.7%	44.8%
26-35	Count	51	4	6	61
	% of Total	44.0%	3.4%	5.2%	52.6%
36-45	Count	2			2
	% of Total	1.7%			1.7%
Total	Count	98	10	8	116
	% of Total	84.5%	8.6%	6.9%	100.0%

Table : Adabraka Polyclinic (Jun 1999 - May 2000)

Worm and protozoan infestation in pregnant women at Adabraka Polyclinic (June 2000-May 2001)

Table 4.6b

Age (in years)	Statistics	Type of worm					Total
		Intestinal flagellates	*Hookworm*	*Strongyloids*	*Ascaris*	*E. coli*	
16-25	Count	39		4	1		44
	% of Total	40.2%		4.1%	1.0%		45.3%
26-35	Count	39	4	3		1	47
	% of Total	40.2%	4.1%	3.1%		1.0%	48.5%
36-45	Count	5		1			6
	% of Total	5.2%		1.0%			6.2%
Total	Count	83	4	8	1	1	97
	% of Total	85.6%	4.1%	8.2%	1.0%	1.0%	100.0%

Table : Adabraka Polyclinic (Jun 2000 - May 2001)

Many of the women between age groups of 16–25 (37.9%) and 26–35 (44.0%) were infested with intestinal flagellates throughout the months with the exception of October and September for age groups of 16–25, 26–35 respectively (Table 4.6). In the

year (1999-2000), out of the total number of pregnant women infested with intestinal worms, 84.5% had intestinal flagellates, 8.6% had Hookworm and 6.9% were infested with strongyliodes (Table 4.6). In 2001-2002, age group of 16–25 had the highest number of women infected (42.9%). In 2000-2001, a similar trend was observed with many infections within the ages 16–25 and 26–30 and being mostly intestinal flagellate whereas very few were infected with the other worms (Appendix 2: Table 4.7). During the study period of May 2002 – June 2003, it was realized that there had been an increase in the intestinal worm infection, 90.5% and 93.1% respectively (Appendix 2: Table 4.7).

In assessing the highest and lowest levels of worm infection over a two-year period, the highest level of worm infection was in 1999-2000 as compared to 2001-2002. At the Nsawam General Hospital, in the year 1999-2001, many of the pregnant women between ages 16–25 (46.9%) and 26–35 (28.5%) were infected with Ascaris worm (Table 4.8).

Table 4.8

Age	Type of worm	Statistics	Months												Total
			Jan	Jul	Aug	Sept	Oct	Nov	Dec	Jun	Feb	Mar	Apr	May	
11-15	*Ascaris*	Count			2	1				3					6
		% of Total			28.6%	14.3%				42.9%					85.7%
	Strongyloides	Count								1					1
		% of Total								14.3%					14.3%
	Total	Count			2	1				4					7
		% of Total			28.6%	14.3%				57.1%					100.0%
16-25	*Ascaris*	Count	25	26	22	20	34	40	29	23	30	29	32	40	350
		% of Total	6.0%	6.3%	5.3%	4.8%	8.2%	9.6%	7.0%	5.5%	7.2%	7.0%	7.7%	9.6%	84.1%
	Intestinal flagellates	Count		2	3		5	1	4	2	4	1	6	4	32
		% of Total		.5%	.7%		1.2%	.2%	1.0%	.5%	1.0%	.2%	1.4%	1.0%	7.7%
	Hookworm	Count	1	5		2	2	1	1		4		1	5	22
		% of Total	.2%	1.2%		.5%	.5%	.2%	.2%		1.0%		.2%	1.2%	5.3%
	Strongyloides	Count				1	2		1	1	1			4	10
		% of Total				.2%	.5%		.2%	.2%	.2%			1.0%	2.4%
	E coli	Count									2				2
		% of Total									.5%				.5%
	Total	Count	26	33	25	23	43	42	35	26	41	30	39	53	416
		% of Total	6.3%	7.9%	6.0%	5.5%	10.3%	10.1%	8.4%	6.3%	9.9%	7.2%	9.4%	12.7%	100.0%
26-35	*Ascaris*	Count	13	24	23	24	14	8	13	16	24	19	16	18	213
		% of Total	5.7%	9.8%	9.4%	9.8%	5.7%	3.3%	5.3%	6.6%	9.8%	7.8%	6.6%	7.4%	87.3%
	Intestinal flagellates	Count		1	3		2			1		4	2	3	16
		% of Total		.4%	1.2%		.8%			.4%		1.6%	.8%	1.2%	6.6%
	Hookworm	Count		1	2	1	1	1	1	1		2			10
		% of Total		.4%	.8%	.4%	.4%	.4%	.4%	.4%		.8%			4.1%
	Strongyloides	Count		1				1			1			1	4
		% of Total		.4%				.4%			.4%			.4%	1.6%
	E coli	Count					1								1
		% of Total					.4%								.4%
	Total	Count	14	27	28	25	18	10	14	18	25	25	18	22	244
		% of Total	5.7%	11.1%	11.5%	10.2%	7.4%	4.1%	5.7%	7.4%	10.2%	10.2%	7.4%	9.0%	100.0%
36-45	*Ascaris*	Count	7	10	2	14			12	13	2	6	3		69
		% of Total	8.9%	12.7%	2.5%	17.7%			15.2%	16.5%	2.5%	7.6%	3.8%		87.3%
	Intestinal flagellates	Count		1	1					1			1		4
		% of Total		1.3%	1.3%					1.3%			1.3%		5.1%
	Hookworm	Count	1	1						2	1				5
		% of Total	1.3%	1.3%						2.5%	1.3%				6.3%
	Strongyloides	Count								1					1
		% of Total								1.3%					1.3%
	Total	Count	8	12	3	14			12	17	3	6	4		79
		% of Total	10.1%	15.2%	3.8%	17.7%			15.2%	21.5%	3.8%	7.6%	5.1%		100.0%
46-50	*Ascaris*	Count				1									1
		% of Total				100.0%									100.0%
	Total	Count				1									1
		% of Total				100.0%									100.0%

Nsawam Hospital (June 1999-May 2000)

Worm Infestation in Pregnant Women from Nsawam Hospital for 1999 - 2000

Ascaris infection was also common throughout most months in the year 1999-2000. Pregnant women within the various year groups who had a high percentage of infection; 11–15 (85.7%); 16–25 (84.1%); 26–35 (87.3%) and 36–45 (87.3%) (Table 4.8). In the whole year of June 1999 – May 2000, out of the total number of pregnant women, 85.5% had *Ascaris*, 7.0% had intestinal flagellates, 5.0% had hookworm 2.1%

had *strongyliodes* and 0.4% had *E. coli* (Table 4.8). A similar trend was observed over the years - June 2001 to May 2003 (Appendix 2: Table 4.9- 4.11).

At the Mamobi Polyclinic, studies revealed that in May 2001- June 2002 many of the pregnant women were infected with intestinal flagellates (61.4%). A few were infected with other worms; Hookworm (11.8%); Strongyloides (8.8%); *Trichuris* (1.3%); *S. mansoni* (1.8%); *E. histolytic* (0.9%); H. nana (1.3%); *S. haematobium* (7.9%) (Table 4.12).

Table 4.12

Age	Statistics	Type of worm									Total
		Ascaris	*Intestinal flagellates*	*Hookworm*	*Strongyloides*	*Trichuris*	*S. mansoni*	*E. histolytica*	*H. nana*	*S. haematobium*	
16-25	Count	6	84	16	11	3	2	1	2	16	141
	% of Total	2.6%	36.8%	7.0%	4.8%	1.3%	.9%	.4%	.9%	7.0%	61.8%
26-35	Count	5	46	11	8		1	1	1	2	75
	% of Total	2.2%	20.2%	4.8%	3.5%		.4%	.4%	.4%	.9%	32.9%
36-45	Count		10		1		1				12
	% of Total		4.4%		.4%		.4%				5.3%
Total	Count	11	140	27	20	3	4	2	3	18	228
	% of Total	4.8%	61.4%	11.8%	8.8%	1.3%	1.8%	.9%	1.3%	7.9%	100.0%

Mamobi polyclinic (Jun2001-2002)

Worm infestation in pregnant women from Mamobi Polyclinic (June 2001-2002)

The age group 16–25 had the highest number of pregnant women with worm infection (61.8%) for year June 2001 to May 2002 (Table 4.12). Intestinal flagellates infection occurred almost throughout all the months of the year, followed by hookworm infection. In May 2002 to June 2003, there was an increase in the intestinal worm infection (77%) and a slight decrease in the other worm infections (Appendix 2: Table 4.13). The age group 16–25 still had the highest number of pregnant women with worm infection (54.0%) followed by 26 – 35 age group (37.9%) (Appendix 2: Table 4.13).

Correlation between the age of pregnant women and parasitic infection is significant ($p = 0.01$). This shows that as the age of the pregnant women increased the infection reduces. There was also significant difference between age and *A. lumbricoides* ova infection in the subsequent years (Appendix 2: Table 4.25).

Similarly, there was negative correlation between intestinal flagellate infestation an the age of pregnant women ($p = 0.05$) showing that as the age of the pregnant women increased the infection reduces (Appendix 2: Table 4.24). With the subsequent years (2000-2001 and 2002-2003), there was no significant difference between age and intestinal flagellates infestation (Table 4.27). The correlation between infestation and age of pregnant women was not significant ($p < 0.05$). Hookworm ova and Strongyloides infection were not associated with age for all the years.

From the table 4.28, *A. lumbricoides* infection on the average reduced from 17.6667 in June 1999 – May 2000 to 12.9706 in June 2001 – May 2002, and later increased to 14.333 in June 2002–May 2003. Intestinal flagellates infection increased from 2.4783 in June1999 – May 2000 to 4.5714 in June 2001 – May 2002, but further reduced to 14.333 in June2002 – May 2003. Hookworm ova infection reduced over the years but increased slightly in June 2002 – May 2003. Strongyloides infection initially reduced but rose sharply in June 2001 – May 2002, then reduced again.

CHAPTER 5

5.0 DISCUSSION

From the study it can be inferred that parasitic infections may be a public health problem at Mamobi, Adabraka, Nsawam and Agormenya. Informal discussions held with the pregnant women who visited the hospitals revealed that many of the infected were within ages 16-25 and 26-35. They had either given birth for the first or it was the second time birth. A few of them had more than two children.

At Nsawam hospital the following worms were found: *Ascaris, Strongyliodes*, Intestinal flagellates, Hookworm, *Trichuris trichiura, E. histolytica, S. mansoni* and *H. nana*. The worms found at the Adabraka polyclinic were *Trichuris, E. histolytica, S. mansoni* and *H. nana.* At Mamobi polyclinic, were *Ascaris*, Intestinal flagellates, Hookworm, *Strongyloides, Trichuris, S. mansoni, E. histolytica, H. nana, S. haematobium*.

The highest prevalence of malaria observed in June 1999–May 2000 at the St. Martins Hospital (Figure 4.1) could be due to the fact that it was the wet season. Also, health care services may not be good. However, the low prevalence of malaria observed in June to September may be attributed to improved living standards, through education, or better health care with orthodox drugs and the use of herbs. The study also revealed that the highest number of pregnant women infected with malaria were within age group of 16–25 and in their active reproductive age. At Mambobi Polyclinc and St. Martins Hospital, with the exception of December at St Martins Hospital where most of the patients were within ages 26–35 (Figure 4.1). Verbal discussions with the laboratory technicians revealed that most the pregnant women had given birth for the first or second time. The high prevalence of malaria among pregnant women in both Mamobi Polyclinic (though Mamobi is a reference hospital), and St. Martins Hospitals could be related to

poor environmental sanitation in those communities, which had a number of favourable breeding areas for mosquito and lack of adequate health education.

At St. Matins' Hospital a large number of pregnant women were found infected in December (1999-2000, 2000- 2001) though it was not the peak season for malaria. This could be explained by the fact that many of them reported at the hospital at that time since it was out of the farming season. Many were at home and had time to attend to their health needs. Also the high malaria prevalence in June (2000-01) was normal since that is the peak season for malaria. However, a decrease in number reporting in the previous year could also be that many treated themselves.

The high level of malaria infection in the pregnant women reporting at St. Martins and Adabraka Hospitals could be an indication that there is low immunity during this period of pregnancy. This may be confirmed by studies that showed that pregnancy decreases a woman's immunity and makes her more susceptible to numerous parasitic infections. The main factors of maternal health problems are poverty, poor nutrition and illiteracy (Duerden *et al.*, 1987). A study of the parasitemia levels over the two-year period at St Martin's Hospital and Adabraka, revealed that the load of infection decreased from June 1999-May 2000 to 2002–2003 (Figure 4.5 and 4.6). This suggested that malaria may be controlled with time if proper health practices are adhered to. Some of these practices include the taking of prophylactics, use of mosquito insecticide spray, mosquito treated bed nets, and wearing of protective clothing. However, at Mamobi Polyclinic, a similar trend was also observed, except that most of the months had the highest loads of infection in the year 2003 (Appendix 1: Fig 4.7). This suggests a likelihood of malaria becoming a serious health problem in this community. The seasonal/monthly distribution of malaria, showed that the normal major peak season between September and October. This study revealed that most of the major and minor

malaria seasons fell outside the normal peak season. This could be attributed to the fact that most of these pregnant women were either fish mongers or peasant farmers, they normally attend to their work at that time, and would not go to the hospital in the rural areas. However, in the urban areas there could be a possibility of these women treating themselves because of the high cost of medical bills.

The greater number of women with malaria were within the age group 16-25 years. According to Steketee et al.,(1996), women in highly endemic areas in their first and second pregnancies are more likely than multigravidae to experience malaria as well as high parasite densities. Also studies have shown that with successive pregnancies, both incidence and severity of infection decline with successive pregnancies (Desowitz et al., 1992). This may explain the trend observed in the hospitals. A related study on Gabon into *P. falciparum* infection in pregnant women showed a malaria prevalence of 57%. The prevalence of parasitaemia did not vary with season (dry or rainy overall, the parasite prevalence was 52% in the dry season and 59% in the rainy season (Bouyou-Akotet et al, 2003).

In relation to age, comparing the prevalence of malaria infection between teenagers, young and older women in Gabon, parasitaemia was significantly more common in teenagers (63.9%) than young (47.7%) and older women (55.5%). Studies done by other authors in the same epidemiological context were similar, 65% in Dielmo, Senegal, 62% in Tanzania, 65% in Malawi 41% in Kenya (Bouyou-Akotet et al, 2003). This study compares with what was found in Ghana, in which many of those with malaria are in their reproductive ages 16-35 years and also the level of parasitaemia did not vary within the peak malaria season and the minor one. Studies done at the Presbyterian Mission District Hospital in Agogo, Ghana revealed out of 530 women, 63% (n =336) were infected with malaria parasites. Also plasmodia infections were slightly more

frequent in women who originated from the rural villages (68%, 133/195) than those found in Agogo (61% 203/335). In women of rural and urban residence both parasite densities and the proportion of submicroscopic parasitaemia among infected individuals (53% us 46%) were similar (Mockenhaupt, et al., 2000). In addition, studies showed that women in the oldest age group had a lower risk of harbouring malaria parasites than the youngest women. The risk of parasitaemia was twice as high during rainy season as during dry season. The rainy season was associated with high malaria infection compared with the dry season (Roche et al , 2003).

A longitudinal study of urinary infection among pregnant women from June 1999-May 2000 revealed a greater percentage of *candidiasis* infection as compared to *T. vaginals* infection. This reveals the fact that during malaria treatment, many are treated with antibiotics. The taking in of these antibiotics may offset the balance between bacteria and fungus in the urinary tract hence increasing the fungal infection.

Most pregnant women with candidiasis (77.3%) infection and 22.7% had *T. vaginalis* infection are within ages, 16–25 years Within age group 26–35 years, 81.0% had candidiasis infection while 19.0% had *T. vaginalis* infection (Table 4.1). The increase in the candidiasis infection over the years could also be a result of the indiscriminate use of antibiotics. This trend was similar in all the years for the other hospitals (Table 4.2– 4.5 Appendix 2). However, a decrease in *T. vaginalis* infection at Adabraka Polyclinic (Table 4.1 and 4.2 – 4.5 Appendix 2) could be a result of health education and self-medication, as well as many cases may not have been reported at the hospital. In Mamobi however, *T. vaginalis* seems to be on the increase, thus this could be as a result of their lifestyle for example in their sexually active age, age of curiosity in which sexual activity may be high. Also, many of the women with the infection are between ages 16–25 and 26–35 within the sexually active years. Adolescents are at

higher risk of *T. vaginalis* and *Candidiasis* infection because of frequent unprotected sex, they also engage in partnership often for limited duration (Duane, A 2001). This could also be a possible explanation for the high rate of infection in the hospitals where the studies were carried out.

Studies carried out on worm infestation on pregnant women revealed an increase in Intestinal flagellates at Adabraka Polyclinic and Mamobi Polyclinic (52.6% and 48.5% respectively). This is contrary to Nsawam General Hospital where Ascaris infection is highly prevalent, many of the pregnant women between ages 16–25 (46.9%) 26–35 (28.5%) were infected with *Ascaris lumbricoides* (Table 4.8). This could be possible due to the fact that there are important differences in the biology and epidemiology of these parasites, and it is possible that suitable conditions for transmission of both parasites do not exist in all communities.

Studies carried out in November and December 1998 in a village at Fagnampleu in Cote d' Ivoire revealed the prevalence of Hookworm infection (60%), *T. trichiura* and *A. lumbricoides* were rare about 3.4 and 2.2%, Escherichia coli was the most prevalent intestinal protozoa (60%). These findings contradict what was observed in this study in which *Ascaris* infection was high (46.9%). In Kenya, intestinal helminthes are ubiquitous in low-income communities with prevalence of 50–80% for ascariasis, trichuriasis and hookworm infections in many populations. The ascariasis prevalence in Kenya was similar to what was observed at Nsawam hospital during this study period (Bund, 1988).

Research carried out at Sanliurfa province, a developing region in South Eastern Turkey revealed the helminthes infection to be 77.1% in Shantytown, 53.2% in apartment district and 53.1% in rural areas. *Ascaris lumbricoides* was most prevalent in this study area. These findings confirm what was observed at Nsawam General Hospital, also a

rural area (Seyrek et al, 2003). Studies carried out in China showed that vegetable garden farmers have the highest prevalence of hookworm infection 31.0% as well as high rates of *Ascaris* 36.7% and *Trichuris* 47.9% infections. School children have the highest prevalence and intensity of ascariasis 48.5% (Hotez, et al., 1989). *Ascaris* and *Trichuris* eggs are able to withstand greater extremes of moisture and temperature and other harsh environments, which are unfavourable for other worm transmissions. Since majority of the people in Nsawam are farmers, engaged in small-scale vegetable farming, this could probably explain why ascariasis is endemic in rural settings of Nsawam and its surrounding villages. Also, common agricultural practices of using un-decomposed human manure for fertilizers, unclean water from the drainage systems to water most vegetables in the urban settings could possibly be the source of high prevalence of intestinal flagellates observed at Adabraka and Mamobi Polyclinics. This explanation also agrees with findings made in the Far East in which farmers use undecomposed human manures for fertilizers (Duerden, *et al.*, 1987).

Studies have revealed water as the most common means of transmission of cyst of intestinal flagellates (Boyd, 1995). This could possibly account for the high prevalence of intestinal flagellates around Mamobi and Nima, where sanitation is poor. Infection is spread from person to person primarily by the fecal oral route (Boyd, 1995).

Studies have revealed that approximately 66 million women between 15 to 49 years of age, many with underlying dietary iron deficiency anemia habour intestinal worms (Hotez, *et al.*, 1989). This probably explains why most pregnant women between ages 16–25 and 26–35 years of age have the worm infection in all hospitals that the studies were carried out (i.e. 38.1% and 19.0% in Adabraka Hospital, 54.0%, and 37.9% in Mamobi Polyclinic; 84% and 87.1% in Nsawam Hospital).

The extremely high prevalence of these infections in this study may be a function of unprotected water supply and unsanitary practices of the villages and parts of the urban areas. Defaecation in the surrounding fields and the stools are exposed to scavenging animals and the drying effect of the sun and wind. Animals and wind are known to be sources of water supply, contamination and direct infection. The prevalence of intestinal worm infection was assessed in 1993 -1995 among two different groups in Guinea (rural dwellers and in and out patients in a hospital). It was found out that the average prevalence of *Entomoeba histolytica* (14.9% and 32.7% respectively), *Gardia Lamblia* (7.2% and 8.6%), *Ascaris limbricoides* (45.8% and 31.4%) and *Trichuris trichiura* (25.7% and 36.4%) (Roche et al. 2003).

CHAPTER SIX

6.0 CONCLUSION AND RECOMENDATION

In comparing the parasitic diseases at the four different hospitals, the following findings were revealed; Intestinal flagellate infestation was significantly higher at Adabraka polyclinic and Mamobi polyclinic (90.5% and 77.0% respectively). Whiles *Ascaris* infestation was significantly higher (87.0%) at Nsawam general hospital. Studies on urinary infection showed that there is a significantly higher *Candidiasis* and *T. vaginalis* infection (80% and 20%) at Mamobi polyclinic for ages 16-25 in the year 2002-2003 as compared to Adabraka polyclinic(77% and17%). Malaria infection at Mamobi polyclinic was significantly higher followed by St. Martins and Adabraka polyclinic. The statistical analysis also showed that the infections decreased with age in all four hospitals: Adabraka and Mamobi Polyclinics and Nsawam and St. Martin Hospitals where studies were conducted.

In conclusion, this study carried out in four different hospitals in some parts of eastern and greater Accra regions showed that parasitic and other infections represent a major health problem among pregnant women. Some parasitic infections like Hookworm, Ascariadiasis, and Trichuriasis are mainly associated with poverty and the environmental and educational conditions that accompany deprivation. However, even in these conditions, short-term measures can play an effective role in reducing their transmission through simple community improvements in water supply, excreta disposal, and general environmental hygiene and through changes in personal behaviour of individuals as well as health education.

In controlling parasitic infections, some of the ways by which it could be done is by effective use of human disposal facilities, high standards of personal and domestic hygiene must be maintained. For instance, general cleanliness, frequent washing of hands

after use of toilet must be done to prevent fecal contamination within the home, which could result in worm infestation. In addition, human excreta should be well treated before used as manure. Vegetables, fruits and food must be well washed, meat should also be well cooked before consumption. The use of bed nets impregnated with mosquito repellant chemicals, mosquito spray and coil and wearing of protective clothing is also recommended with reference to malaria infection. Environmental sanitation measures must be adhered to in order to control parasitic diseases. Examples, are wearing of protective foot ware, avoiding damp warm soils around the home, which may act as transmission sites for parasites especially strongyloides.

Also clearing and spraying of water banks of rivers, streams, and lakes to destroy aquatic plants, which serve as habitats for intermediate host of some parasites in areas such as Nsawam and Odumase-Krobo. In the case of *T. vaginalis* and *Candidiasis* the youth should abstain from premarital sex. Also, married couples should remain faithful to partners. Finally, Education on prevention and control of parasitic diseases, chemotherapy as well as mass deworming is also recommended in the study area and Ghana as a whole.

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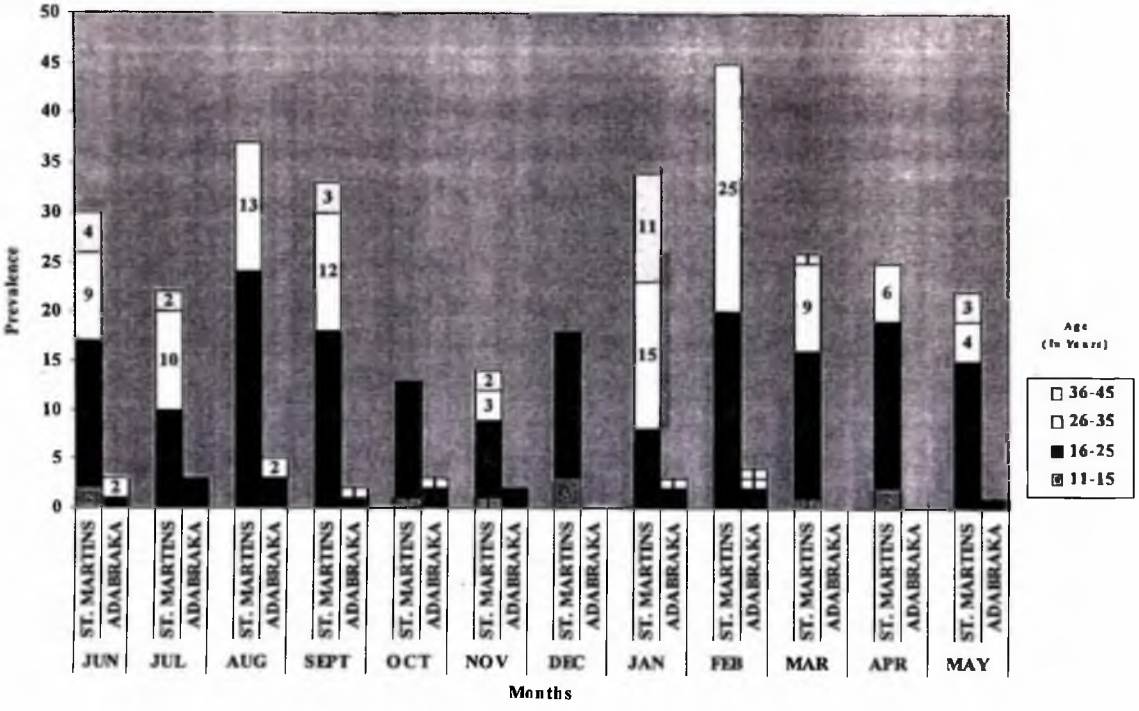
APPENDIX 1:

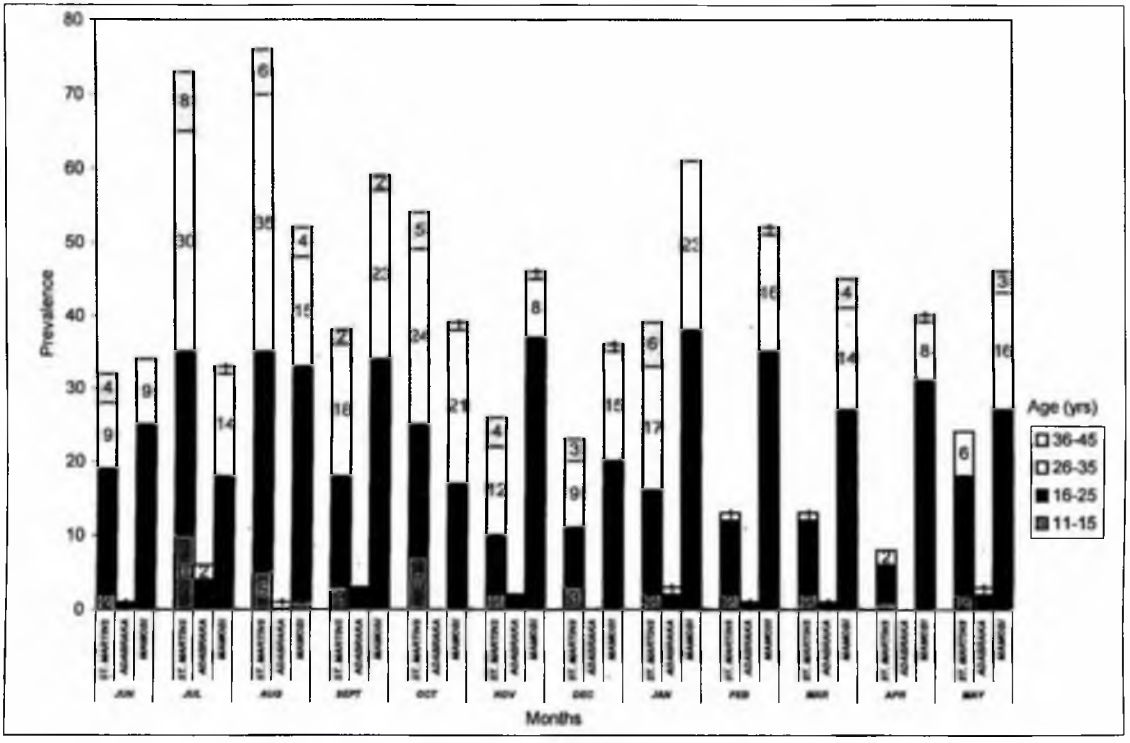
Fig. 4.2 **Comparing Prevalence of Malaria in Pregnant Women From St. Martins Hospital and Adabraka Polyclinic**



ST. Martins and Adabraka Hospital

(Jun 2000-May 2001)

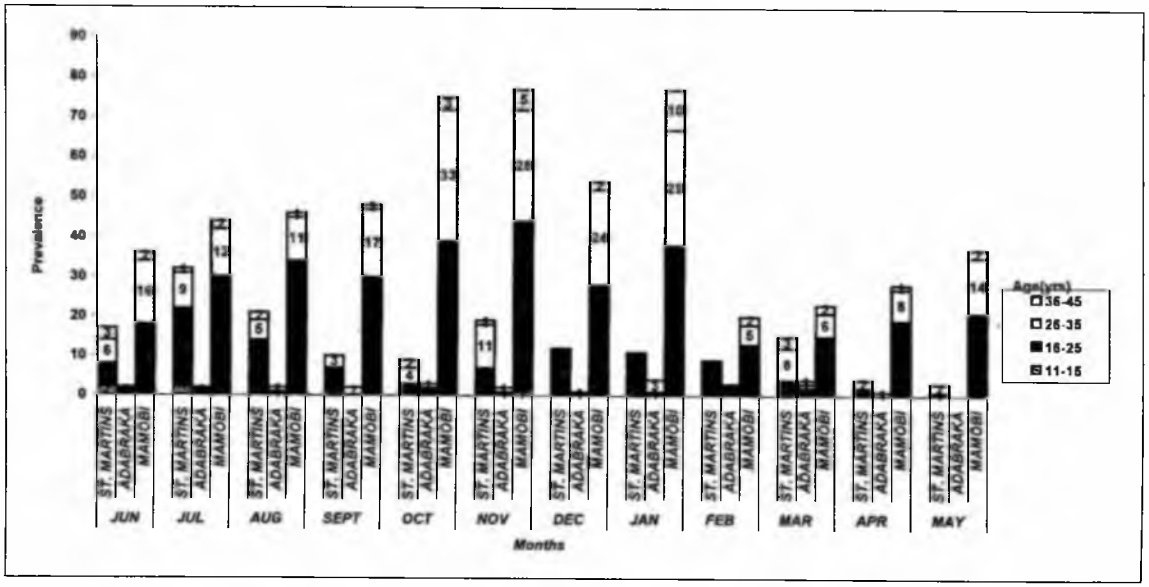
Fig. 4.3 **Comparing Prevalence of Malaria in Pregnant Women from Three Different Hospitals**



ST. Martins, Adabraka and Mamobi Polyclinic

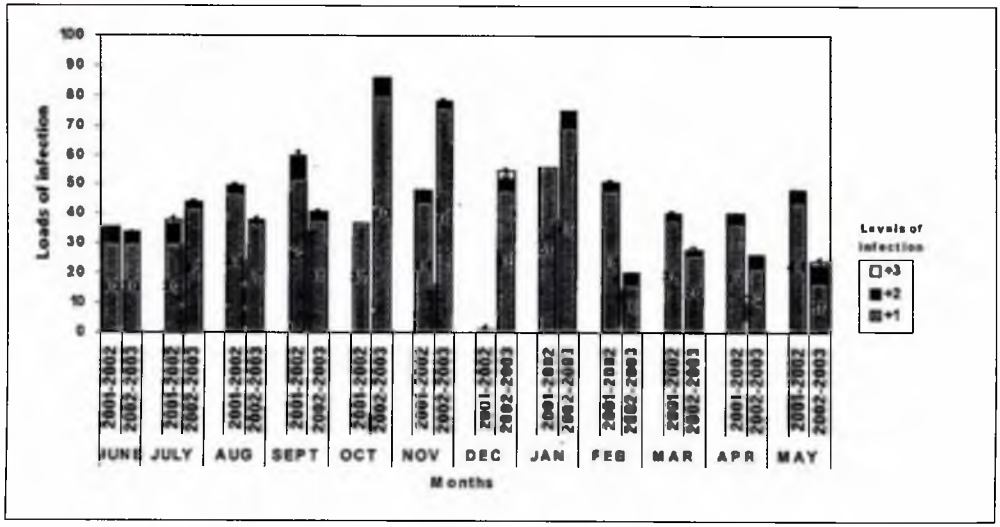
(Jun 2001-May 2002)

Fig. 4.4 **Comparing Prevalence of Malaria in Pregnant Women From Three Different Hospitals**



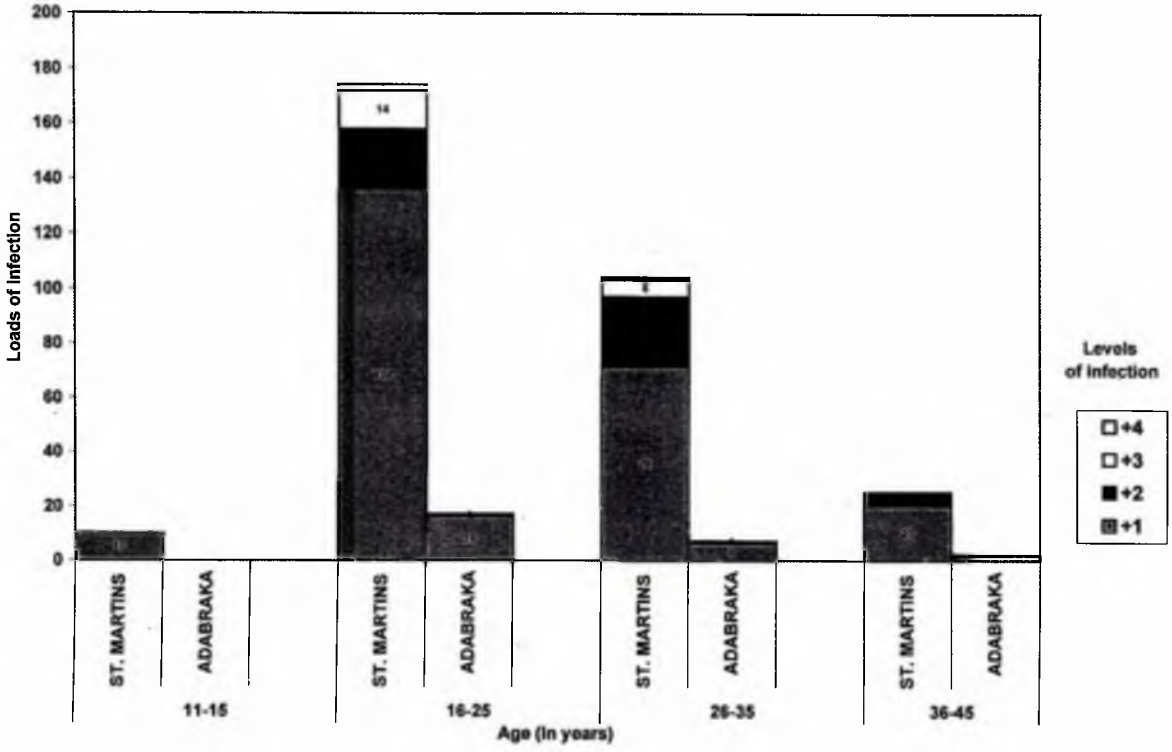
ST. Martins, Adabraka and Mamobi Polyclinic from June 2002-May 2003

Fig. 4.7 Presenting Highest and Lowest Parasitemia Levels over 2years in Mamobi Polyclinic



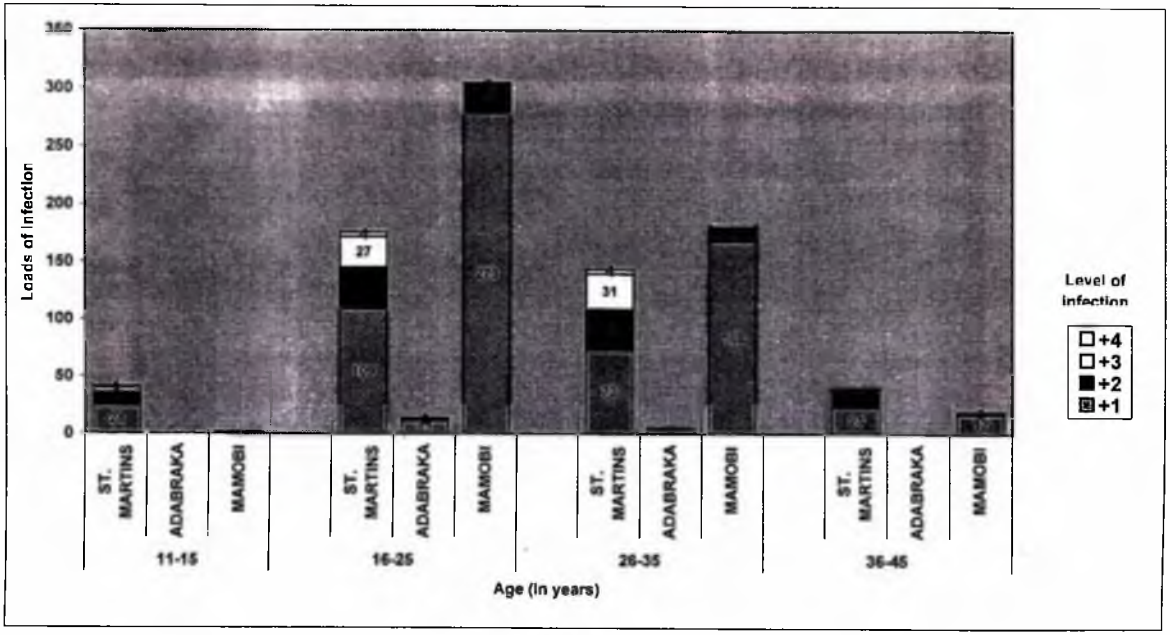
Mamobi Polyclinic, June 2001 – May 2002 (Highest), June 2002 – May 2003 (lowest)

Fig. 4.9 Presenting the Relative Parasitemia Levels in Pregnant Women



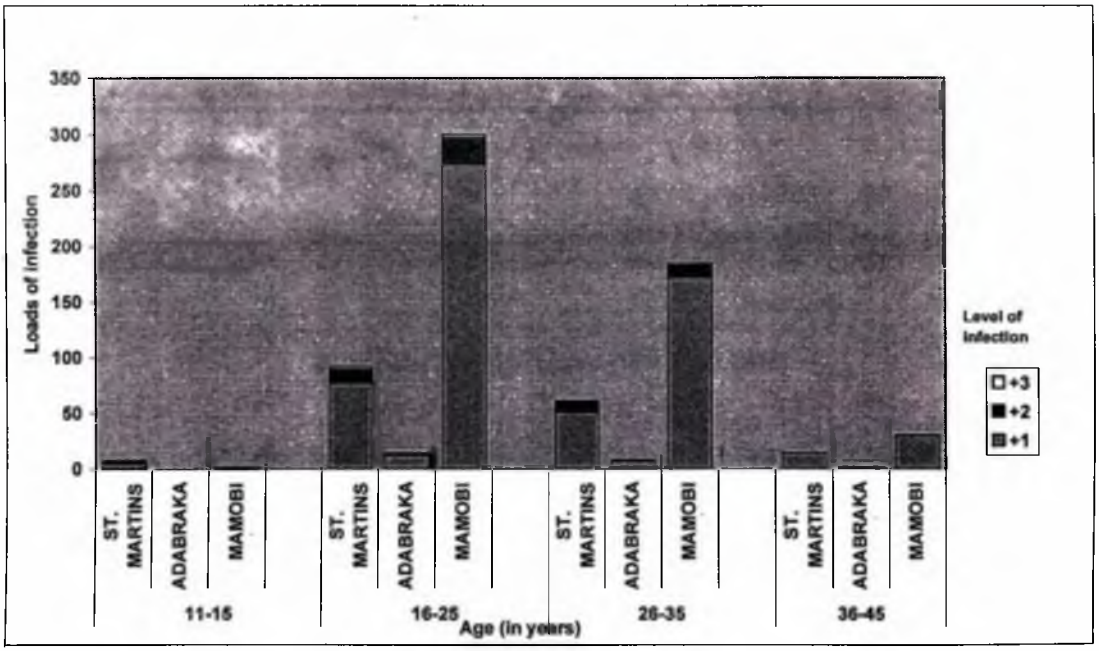
St. Martins Hospital and Adabraka Hospital
(June 2000 – May 2001)

Fig. 4.10 **Presenting the Relative Parasitemia Levels in Pregnant Women**



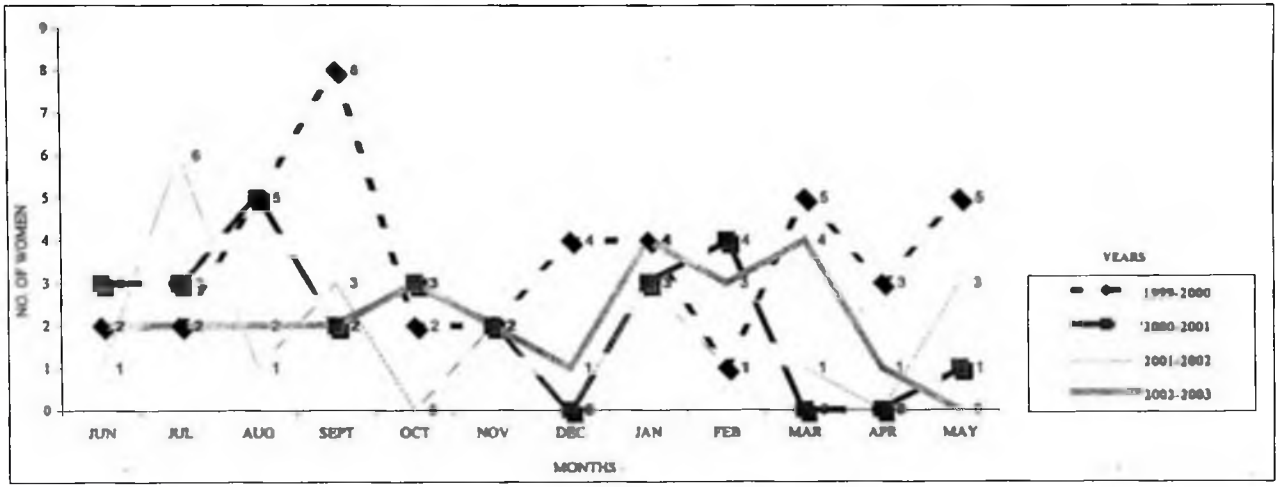
St. Martins, Adabraka and Mamobi Hospital
(June 2001 – May 2002)

Fig. 4.11 **Presenting the Relative Parasitemia Levels in Pregnant Women**



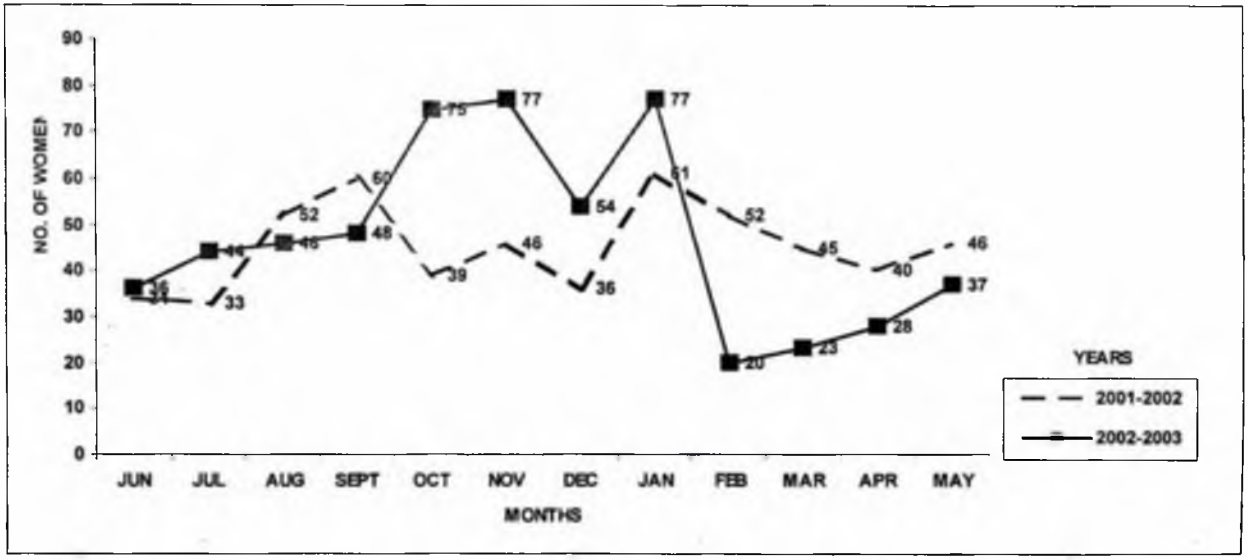
St. Martins, Adabraka and Mamobi Hospital
(June 2002 – May 2003)

Fig. 4.13 Presenting Seasonal/Monthly Distribution of Malaria Infection in Pregnant Women



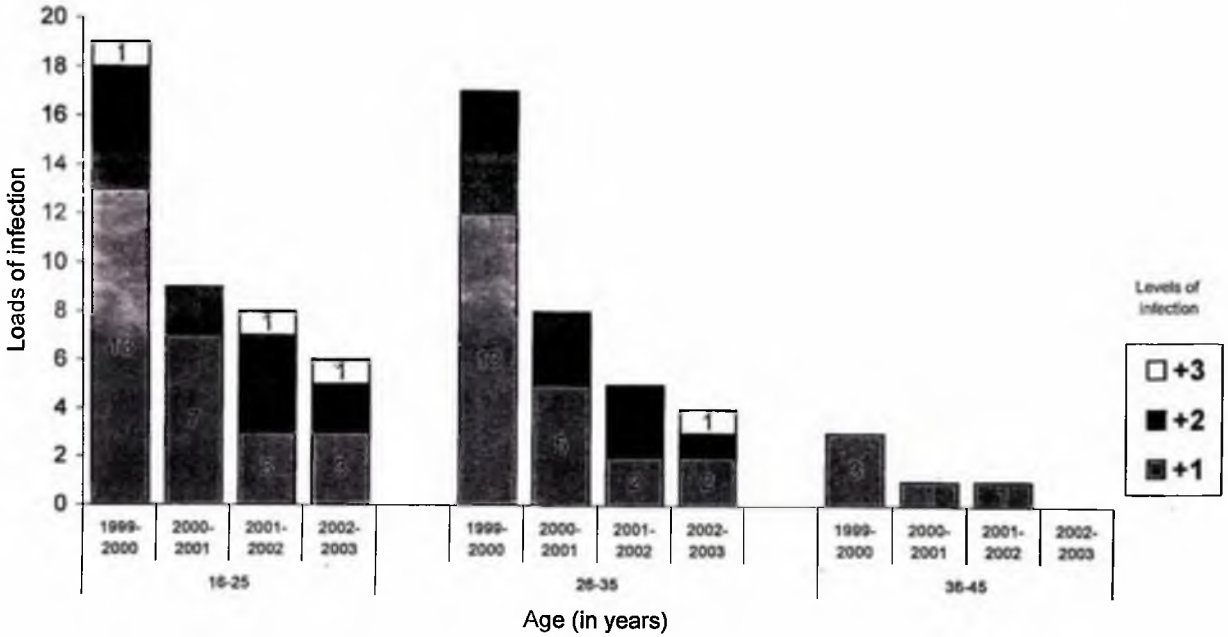
Adabraka Hospital (June 1999-May 2003)

Fig. 4.14 Presenting Seasonal/Monthly Distribution of Malaria Infection in Pregnant Women



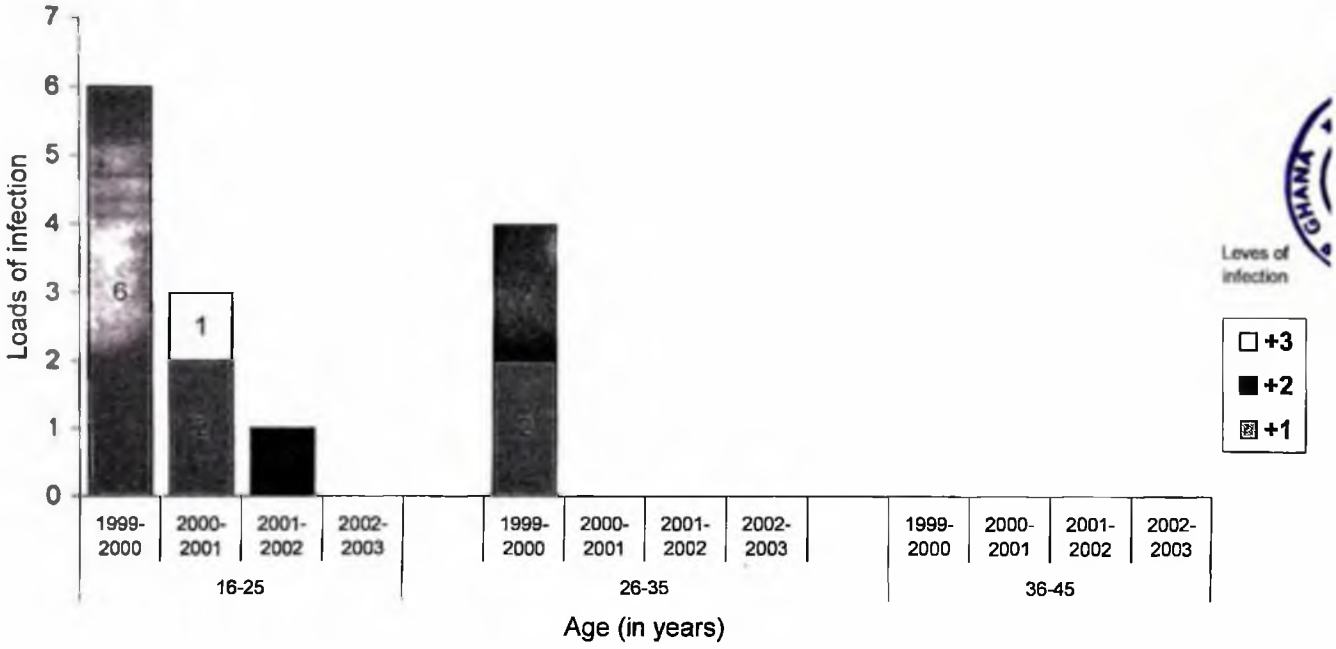
Mamobi Hospital (June 2001-May 2003)

Fig. 4.15 **Presenting the Relative Urine Infection Levels among Pregnant Women in Adabraka Polyclinic**



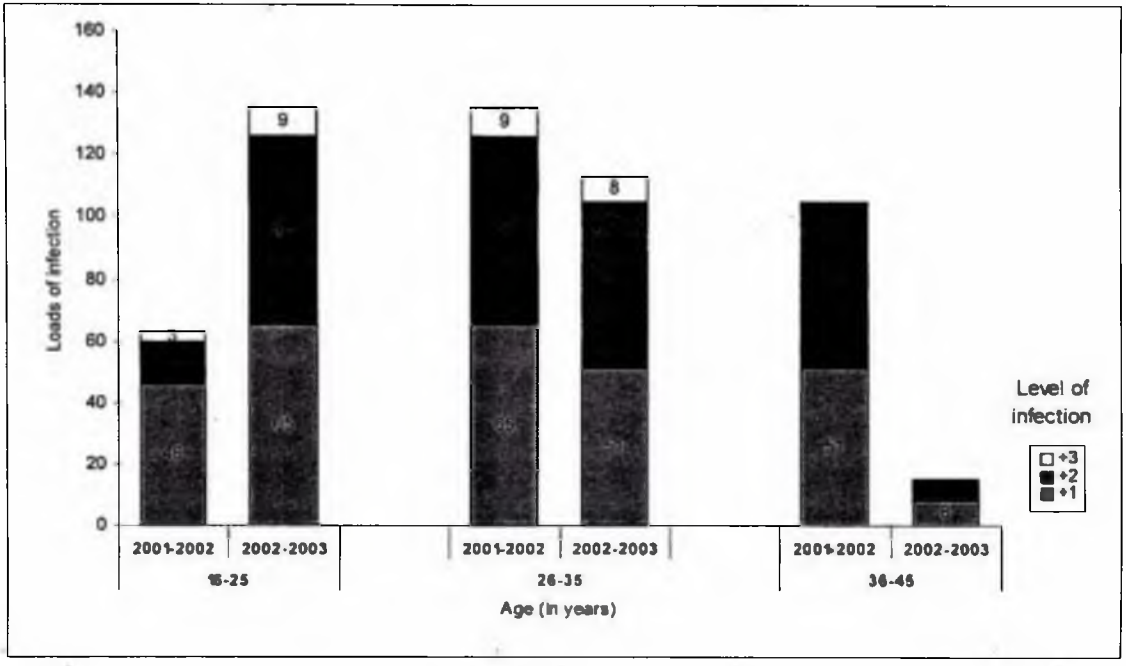
For Candidiasis (from Adabraka June 1999- May 2003)

Fig. 4.16 **Presenting the Relative Urine Infection Levels among Pregnant Women in Adabraka Polyclinic**



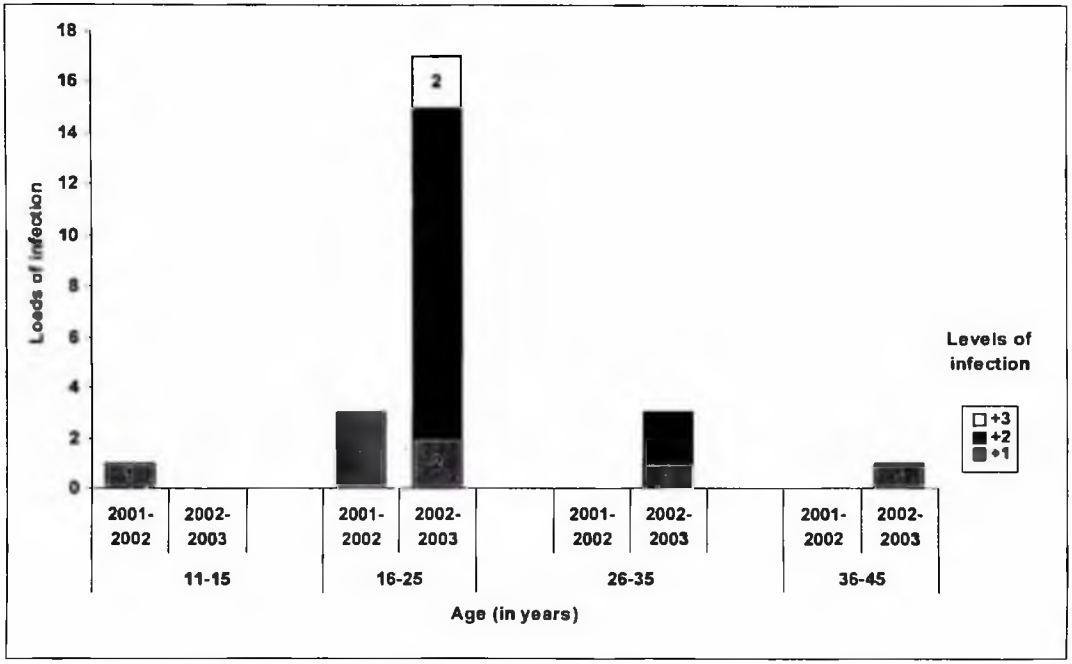
for *T. vaginalis* (Adabraka from June 1999-May 2003)

Fig. 4.17 Presenting the Urinary Infection levels across the years in Mamobi Polyclinic



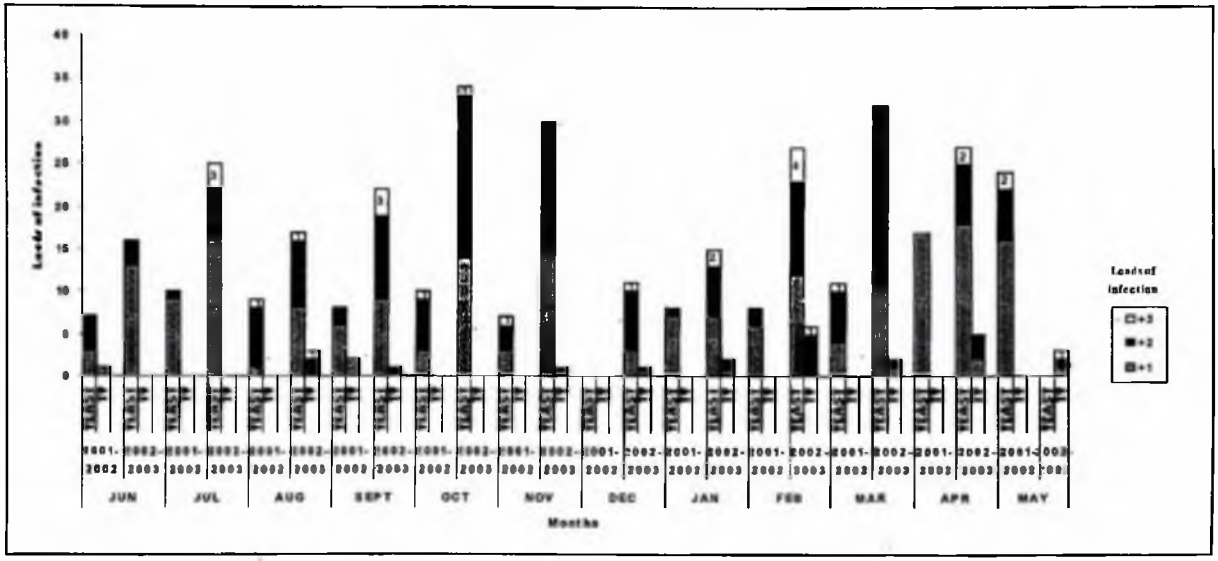
Candidiasis Infection (Mamobi Polyclinic from 2001-2003)

Fig. 4.18 Presenting the Urinary Infection levels across the years in Mamobi Polyclinic



T. vaginalis **(Mamobi from 2001-2003)**

Fig. 4.20 **Highest and Lowest Level of Urinary Infection among Pregnant Women**
At Mamobi Polyclinic



Mamobi Polyclinic Highest (Jun 2002-2003) and Lowest (Jun 2001-May2002)

APPENDIX 2:

Table 4.2: Cross sectional Study of Urinary Infection among Pregnant Women in Adabraka Polyclinic

AGE	URINE INFECTIONS		Statistics	Months										Total
				Jul	Aug	Sept	Oct	Dec	Jan	Feb	Mar	Apr	May	
16-25	URINE INFECTIONS	*Candidiasis*	Count	1		2	1		1	1	1	1		8
			% of Total	11.1%		22.2%	11.1%		11.1%	11.1%	11.1%	11.1%		88.9%
		T. vaginalis	Count		1									1
			% of Total		11.1%									11.1%
	Total		Count	1	1	2	1		1	1	1	1		9
			% of Total	11.1%	11.1%	22.2%	11.1%		11.1%	11.1%	11.1%	11.1%		100.0%
26-35	URINE INFECTIONS	*Candidiasis*	Count	2	1			1	1				2	7
			% of Total	25.0%	12.5%			12.5%	12.5%				25.0%	87.5%
		T. vaginalis	Count			1								1
			% of Total			12.5%								12.5%
	Total		Count	2	1	1		1	1				2	8
			% of Total	25.0%	12.5%	12.5%		12.5%	12.5%				25.0%	100.0%
36-45	URINE INFECTIONS	*Candidiasis*	Count							1				1
			% of Total							100.0%				100.0%
	Total		Count							1				1
			% of Total							100.0%				100.0%

Table 2 : Adabraka Polyclinic (Jun 2000 - May 2001) [a]

a. **No recordings for June and November**

Table 4.3 Cross sectional Study of Urinary Infection among Pregnant Women in Adabraka Polyclinic

AGE	URINE INFECTIONS		Statistics	Months								Total
				Jul	Aug	Oct	Nov	Jan	Feb	Mar	Apr	
16-25	URINE INFECTIONS	*Candidiasis*	Count	1		1		2	2		1	7
			% of Total	12.5%		12.5%		25.0%	25.0%		12.5%	87.5%
		T. vaginalis	Count		1							1
			% of Total		12.5%							12.5%
	Total		Count	1	1	1		2	2		1	8
			% of Total	12.5%	12.5%	12.5%		25.0%	25.0%		12.5%	100.0%
26-35	URINE INFECTIONS	*Candidiasis*	Count				2	1	1	1		5
			% of Total				40.0%	20.0%	20.0%	20.0%		100.0%
	Total		Count				2	1	1	1		5
			% of Total				40.0%	20.0%	20.0%	20.0%		100.0%
36-45	URINE INFECTIONS	*Candidiasis*	Count					1				1
			% of Total					100.0%				100.0%
	Total		Count					1				1
			% of Total					100.0%				100.0%

Table 3 : Adabraka Polyclinic (Jun 2001 - May 2002)

a. **No recordings for June, September, December and May**

Table 4.3 Cross Sectional Study of Urinary Infection among Pregnant Women in Adabraka Polyclinic

AGE	URINE INFECTIONS	Statistics	Months							Total
			Jun	Jul	Oct	Nov	Jan	Feb	Mar	
16-25	URINE INFECTIONS *Candidiasis*	Count		1	2		1	1	1	6
		% of Total		16.7%	33.3%		16.7%	16.7%	16.7%	100.0%
	Total	Count		1	2		1	1	1	6
		% of Total		16.7%	33.3%		16.7%	16.7%	16.7%	100.0%
26-35	URINE INFECTIONS *Candidiasis*	Count	2		1	1				4
		% of Total	50.0%		25.0%	25.0%				100.0%
	Total	Count	2		1	1				4
		% of Total	50.0%		25.0%	25.0%				100.0%

Table 4 : Adabraka Polyclinic (Jun 2002 - May[a] 2003)

a. No recordings for August, September, December, April and May

Table 4.5 Presenting the Highest and Lowest Levels of Urinary Infection among Pregnant Women in Adabraka Polyclinic over a Two year Period

	YEAR	LEVEL	URINE INFECTIONS		Months												Total
					Jun	Jul	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	
Count	1999-2000 (HIGHEST)	+1	URINE INFECTIONS	*Candidiasis*	4	1	4	1		4	2	4	2		1	4	27
				T. vaginalis	2	3	2				1						8
				Total	6	4	6	1		4	3	4	2		1	4	35
		+2	URINE INFECTIONS	*Candidiasis*	2	1	1	1			1	1			1	1	8
				T. vaginalis	2		1										3
				Total	4	1	2	1			1	1			1	1	11
		+3	URINE INFECTIONS	*Candidiasis*										1			1
				Total										1			1
	2002-2003 (LOWEST)	+1	URINE INFECTIONS	*Candidiasis*		1			2	1			1				5
				Total		1			2	1			1				5
		+2	URINE INFECTIONS	*Candidiasis*	1				1			1					3
				Total	1				1			1					3
		+3	URINE INFECTIONS	*Candidiasis*	1									1			2
				Total	1									1			2
% of Total	1999-2000 (HIGHEST)	+1	URINE INFECTIONS	*Candidiasis*	11.4%	2.9%	11.4%	2.9%		11.4%	5.7%	11.4%	5.7%		2.9%	11.4%	77.1%
				T. vaginalis	5.7%	8.6%	5.7%				2.9%						22.9%
				Total	17.1%	11.4%	17.1%	2.9%		11.4%	8.6%	11.4%	5.7%		2.9%	11.4%	100.0%
		+2	URINE INFECTIONS	*Candidiasis*	16.7%	8.3%	8.3%	8.3%			8.3%	8.3%			8.3%	8.3%	75.0%
				T. vaginalis	16.7%		8.3%										25.0%
				Total	33.3%	8.3%	16.7%	8.3%			8.3%	8.3%			8.3%	8.3%	100.0%
		+3	URINE INFECTIONS	*Candidiasis*										100.0%			100.0%
				Total										100.0%			100.0%
	2002-2003 (LOWEST)	+1	URINE INFECTIONS	*Candidiasis*		20.0%			40.0%	20.0%			20.0%				100.0%
				Total		20.0%			40.0%	20.0%			20.0%				100.0%
		+2	URINE INFECTIONS	*Candidiasis*	33.3%				33.3%			33.3%					100.0%
				Total	33.3%				33.3%			33.3%					100.0%
		+3	URINE INFECTIONS	*Candidiasis*	50.0%									50.0%			100.0%
				Total	50.0%									50.0%			100.0%

Adabraka Polyclinic (1999-2000 and 2002-2003

Table 4.7 Presenting worm infestation in Adabraka Polyclinic

Age (in years)	Statistics	Type of worm: *Intestinal flagellates*	*Hookworm*	*Ascaris*	Total
16-25	Count	7	1	1	9
	% of Total	33.3%	4.8%	4.8%	42.9%
26-35	Count	8			8
	% of Total	38.1%			38.1%
36-45	Count	4			4
	% of Total	19.0%			19.0%
Total	Count	19	1	1	21
	% of Total	90.5%	4.8%	4.8%	100.0%

Table : Adabraka Polyclinic (Jun 2001 - May 2002)

Adabraka Polyclinic (June 2001-May 2002)

Table 4.9 Presenting worm infestation in Nsawam Polyclinic

Age	Statistics	Type of worm: *Ascaris*	*Intestinal flagellates*	*Hookworm*	*Strongylaides*	*E. coli*	Total
11-15	Count	6			1		7
	% of Total	.8%			.1%		.9%
16-25	Count	350	32	22	10	2	416
	% of Total	46.9%	4.3%	2.9%	1.3%	.3%	55.7%
26-35	Count	213	16	10	4	1	244
	% of Total	28.5%	2.1%	1.3%	.5%	.1%	32.7%
36-45	Count	69	4	5	1		79
	% of Total	9.2%	.5%	.7%	.1%		10.6%
46-50	Count	1					1
	% of Total	.1%					.1%
Total	Count	639	52	37	16	3	747
	% of Total	85.5%	7.0%	5.0%	2.1%	.4%	100.0%

Nsawan Hospital (June 1999-May 2000)

Nsawam Hospital: June 1999-May 2000

Table 4.10 Presenting Worm Infestation in Nsawam Hospital

Age		Statistics	Months													Total
			Jun	Jul	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	55	
11-15	Ascaris	Count		2	3					2	5	6	11			29
		% of Total		4.2%	6.3%					4.2%	10.4%	12.5%	22.9%			60.4%
	Intestinal flagellates	Count		1							3	2	3			9
		% of Total		2.1%							6.3%	4 2%	6.3%			18.8%
	Hookworm	Count		1							1	2	2			6
		% of Total		2.1%							2.1%	4.2%	4 2%			12.5%
	Strongyloides	Count									1	2	1			4
		% of Total									2.1%	4.2%	2 1%			8.3%
	Total	Count		4	3					2	10	12	17			48
		% of Total		8.3%	6.3%					4.2%	20.8%	25.0%	35.4%			100.0%
16-25	Ascaris	Count	40	36	45	13	27	36	25	34	30	30	20	26	1	363
		% of Total	8.7%	7.8%	9.8%	2.8%	5.9%	7.8%	5.4%	7.4%	6.5%	6.5%	4.3%	5.7%	.2%	78.9%
	Intestinal flagellates	Count	9	2	3	1	1		1	2	6	3	4	8		40
		% of Total	2.0%	.4%	7%	2%	.2%		.2%	.4%	1.3%	.7%	9%	1.7%		8 7%
	Hookworm	Count	5	4	4	2	3	2		3	5	8	5	3		44
		% of Total	1.1%	.9%	9%	.4%	.7%	.4%		.7%	1.1%	1.7%	1.1%	.7%		9.6%
	Strongyloides	Count	1							1	3	5	2			12
		% of Total	.2%							.2%	.7%	1.1%	.4%			2.6%
	Trichuris	Count	1													1
		% of Total	.2%													.2%
	Total	Count	56	42	52	16	31	38	26	40	44	46	31	37	1	460
		% of Total	12.2%	9.1%	11.3%	3.5%	6.7%	8.3%	5.7%	8.7%	9.6%	10.0%	6.7%	8.0%	.2%	100.0%
26-35	Ascaris	Count	40	13	17	14	10	15	7	24	28	27	30	6		231
		% of Total	13.2%	4.3%	5.6%	4.6%	3.3%	5.0%	2.3%	7.9%	9.3%	8.9%	9.9%	2.0%		76.5%
	Intestinal flagellates	Count	3	1	1		1			1	5	10	10			32
		% of Total	1.0%	.3%	.3%		.3%			.3%	1.7%	3 3%	3.3%			10.6%
	Hookworm	Count	2					2		1	3	7	6			21
		% of Total	.7%					7%		.3%	1.0%	2.3%	2.0%			7.0%
	Strongyloides	Count							1	1	2	4	10			18
		% of Total							.3%	.3%	.7%	1.3%	3.3%			6.0%
	Total	Count	45	14	18	14	11	17	8	27	38	48	56	6		302
		% of Total	14.9%	4.6%	6.0%	4.6%	3.6%	5.6%	2.6%	8.9%	12.6%	15.9%	18.5%	2.0%		100.0%
36-45	Ascaris	Count	2	2	3	4	6	2		7	8	6	9			49
		% of Total	2.8%	2.8%	4.2%	5.6%	8.3%	2.8%		9.7%	11.1%	8.3%	12.5%			68.1%
	Intestinal flagellates	Count									2	3	2			7
		% of Total									2.8%	4.2%	2.8%			9.7%
	Hookworm	Count		3			1				3	2	2			11
		% of Total		4.2%			1.4%				4.2%	2.8%	2.8%			15.3%
	Strongyloides	Count									2	2	1			5
		% of Total									2.8%	2.8%	1.4%			6.9%
	Total	Count	2	5	3	4	7	2		7	15	13	14			72
		% of Total	2.8%	6.9%	4.2%	5.6%	9.7%	2.8%		9.7%	20.8%	18.1%	19.4%			100.0%

Nsawan Hospital (June 2000-May 2001)

Nsawam Hospital: June 1999-May 2000

Table 4.11: Nsawam Hospital: June 2002- May 2003

Age	Type of Worm		Jun	Jul	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Total
11-15	Ascaris	Count				1									1
		% of Total				100.0%									100.0%
	Total	Count				1									1
		% of Total				100.0%									100.0%
16-25	Ascaris	Count	21	19	18	21	24	34	37	38	27	44	49	25	357
		% of Total	4.8%	4.3%	4.1%	4.8%	5.5%	7.8%	8.5%	8.7%	6.2%	10.1%	11.2%	5.7%	81.7%
	Intestinal flagellates	Count	1		2	1		1	1	5	2	6	4	11	34
		% of Total	.2%		.5%	.2%		.2%	.2%	1.1%	.5%	1.4%	.9%	2.5%	7.8%
	Hookworm	Count		1					2	2	3	3	4	2	17
		% of Total		.2%					.5%	.5%	.7%	.7%	.9%	.5%	3.9%
	Strongyloides	Count				2	4		2	2	2	3	9		24
		% of Total				.5%	.9%		.5%	.5%	.5%	.7%	2.1%		5.5%
	Trichuris	Count					1								1
		% of Total					.2%								.2%
	S. mansoni	Count						1							1
		% of Total						.2%							.2%
	E. histolytica	Count		2											2
		% of Total		.5%											.5%
	H. nana	Count					1								1
		% of Total					.2%								.2%
	Total	Count	22	22	20	24	30	36	42	47	34	56	66	38	437
		% of Total	5.0%	5.0%	4.6%	5.5%	6.9%	8.2%	9.6%	10.8%	7.8%	12.8%	15.1%	8.7%	100.0%
26-35	Ascaris	Count	8	10	14	13	16	12	2	24	16	9	8	16	148
		% of Total	4.7%	5.9%	8.2%	7.6%	9.4%	7.1%	1.2%	14.1%	9.4%	5.3%	4.7%	9.4%	87.1%
	Intestinal flagellates	Count	1		1		1					1			4
		% of Total	6%		.6%		.6%					.6%			2.4%
	Hookworm	Count				2		1			2	1	1		7
		% of Total				1.2%		.6%			1.2%	.6%	.6%		4.1%
	Strongyloides	Count		1		4	2			2	1				10
		% of Total		.6%		2.4%	1.2%			1.2%	.6%				5.9%
	E. histolytica	Count									1				1
		% of Total									.6%				.6%
	Total	Count	9	11	15	19	19	13	2	26	20	11	9	16	170
		% of Total	5.3%	6.5%	8.8%	11.2%	11.2%	7.6%	1.2%	15.3%	11.8%	6.5%	5.3%	9.4%	100.0%
36-45	Ascaris	Count	1	4	2	4	5	4		8	1	1		2	32
		% of Total	2.7%	10.8%	5.4%	10.8%	13.5%	10.8%		21.6%	2.7%	2.7%		5.4%	86.5%
	Intestinal flagellates	Count				1		1							2
		% of Total				2.7%		2.7%							5.4%
	Hookworm	Count				1								1	2
		% of Total				2.7%								2.7%	5.4%
	Strongyloides	Count								1					1
		% of Total								2.7%					2.7%
	Total	Count	1	4	2	6	5	5		9	1	1		3	37
		% of Total	2.7%	10.8%	5.4%	16.2%	13.5%	13.5%		24.3%	2.7%	2.7%		8.1%	100.0%

Nsawan Hospital (June 2002- May 2003)

Table 4.13 Presenting Worm Infestation in Pregnant Women from Mamobi Polyclinic

Age	Statistics	Type of worm							Total
		Ascaris	*Intestinal flagellates*	*Hookworm*	*Strongyloides*	*S. mansoni*	*H. nana*	*S. haemotobium*	
11-15	Count			1					*1*
	% of Total			.6%					*.6%*
16-25	Count	1	59	9	2	1	3	12	*87*
	% of Total	.6%	36.6%	5.6%	1.2%	.6%	1.9%	7.5%	*54.0%*
26-35	Count		54	4				3	*61*
	% of Total		33.5%	2.5%				1.9%	*37.9%*
36-45	Count		11						*11*
	% of Total		6.8%						*6.8%*
46-50	Count						1		*1*
	% of Total						.6%		*.6%*
Total	Count	*1*	*124*	*14*	*2*	*1*	*4*	*15*	*161*
	% of Total	*.6%*	*77.0%*	*8.7%*	*1.2%*	*.6%*	*2.5%*	*9.3%*	*100.0%*

Mamobi (2002-2003)

Mamobi Hospital: June 2002-May 2003

Table 4.14 Presenting Malaria infection in Pregnant Women at Mamobi Polyclinic

			Disease	
			Malaria	Total
age	11 - 15	Count	3	3
		% of Total	.6%	.6%
	16 - 25	Count	314	314
		% of Total	61.1%	61.1%
	26 - 35	Count	176	176
		% of Total	34.2%	34.2%
	36 - 45	Count	20	20
		% of Total	3.9%	3.9%
	46 - 50	Count	1	1
		% of Total	.2%	.2%
Total		Count	514	514
		% of Total	100.0%	100.0%

June 2001 – May 2002

Table 4.15 Presenting Malaria infection in Pregnant women at Mamobi Polyclinic

			Disease	
			Malaria	Total
age	11 - 15	Count	1	1
		% of Total	.2%	.2%
	16 - 25	Count	328	328
		% of Total	63.7%	63.7%
	26 - 35	Count	156	156
		% of Total	30.3%	30.3%
	36 - 45	Count	30	30
		% of Total	5.8%	5.8%
Total		Count	515	515
		% of Total	100.0%	100.0%

June 2002 – May 2003

Table 4.16 Presenting Malaria infection in Pregnant women at St. Martins Hospital

			Disease	
			Malaria	Total
age	11 - 15	Count	33	33
		% of Total	7.1%	7.1%
	16 - 25	Count	249	249
		% of Total	53.8%	53.8%
	26 - 35	Count	139	139
		% of Total	30.0%	30.0%
	36 - 45	Count	42	42
		% of Total	9.1%	9.1%
Total		Count	463	463
		% of Total	100.0%	100.0%

June 1999 – May 2000

Table 4.17 Presenting Malaria infection in Pregnant women at St. Martins Hospital

			Disease	
			Malaria	Total
age	11 - 15	Count	8	8
		% of Total	2.7%	2.7%
	16 - 25	Count	171	171
		% of Total	56.8%	56.8%
	26 - 35	Count	100	100
		% of Total	33.2%	33.2%
	36 - 45	Count	22	22
		% of Total	7.3%	7.3%
Total		Count	301	301
		% of Total	100.0%	100.0%

June 2000 – May 2001

Table 4.18 Presenting Malaria infection in Pregnant women at St. Martins Hospital

			Disease	
			Malaria	Total
age	11 - 15	Count	41	41
		% of Total	9.9%	9.9%
	16 - 25	Count	176	176
		% of Total	42.3%	42.3%
	26 - 35	Count	159	159
		% of Total	38.2%	38.2%
	36 - 45	Count	40	40
		% of Total	9.6%	9.6%
Total		Count	416	416
		% of Total	100.0%	100.0%

June 2001 – May 2002

Table 4.19 Presenting Malaria infection in Pregnant women at St. Martins Hospital

			Disease	
			Malaria	Total
age	11 - 15	Count	14	14
		% of Total	5.2%	5.2%
	16 - 25	Count	148	148
		% of Total	55.4%	55.4%
	26 - 35	Count	88	88
		% of Total	33.0%	33.0%
	36 - 45	Count	17	17
		% of Total	6.4%	6.4%
Total		Count	267	267
		% of Total	100.0%	100.0%

June 2002 – May 2003

Table 4.20: Presenting Malaria infection in Pregnant women at Adabraka Polyclinic

			Disease	
			Malaria	Total
age	16 - 25	Count	28	28
		% of Total	65.1%	65.1%
	26 - 35	Count	15	15
		% of Total	34.9%	34.9%
Total		Count	43	43
		% of Total	100.0%	100.0%

June 1999 – May 2000

Table 4.21: Presenting Malaria infection in Pregnant women at Adabraka Polyclinic

			Disease	
			Malaria	Total
age	16 - 25	Count	17	17
		% of Total	65.4%	65.4%
	26 - 35	Count	7	7
		% of Total	26.9%	26.9%
	36 - 45	Count	2	2
		% of Total	7.7%	7.7%
Total		Count	26	26
		% of Total	100.0%	100.0%

June 2000 – May 2001

Table 4.22: Presenting Malaria infection in Pregnant women at Adabraka Polyclinic

			Disease	
			Malaria	Total
age	16 - 25	Count	16	16
		% of Total	80.0%	80.0%
	26 - 35	Count	4	4
		% of Total	20.0%	20.0%
Total		Count	20	20
		% of Total	100.0%	100.0%

June 2001 – May 2002

Table 4.23: Presenting Malaria infection in Pregnant women at Adabraka Polyclinic

			Disease	
			Malaria	Total
age	16 - 25	Count	16	16
		% of Total	80.0%	80.0%
	26 - 35	Count	4	4
		% of Total	20.0%	20.0%
Total		Count	20	20
		% of Total	100.0%	100.0%

June 2002 – May 2003

Table 4.24: Correlations between age and infections in Pregnant women at Nsawam

		age of women	*A. lumbricoides ova*	*Intestinal flagellates*	*Hookworm ova*	*Strongyloides*	*E. coli*
age of women	Pearson Correlation	1.000	-.478**	-.454*	-.401	-.215	-1.000**
	Sig. (2-tailed)		.003	.029	089	.550	
	N	60	36	23	19	10	2
A. lumbricoides ova	Pearson Correlation	-.478**	1.000	.506*	.306	.392	1.000**
	Sig. (2-tailed)	.003		.014	.217	.297	
	N	36	36	23	18	9	2
Intestinal flagellates	Pearson Correlation	-.454*	506*	1.000	.078	.158	1.000**
	Sig. (2-tailed)	029	.014		800	.765	
	N	23	23	23	13	6	2
Hookworm ova	Pearson Correlation	-.401	.306	.078	1.000	.704	1.000**
	Sig (2-tailed)	089	.217	.800		.119	
	N	19	18	13	19	6	2
Strongyloides	Pearson Correlation	-.215	.392	.158	.704	1.000	[a]
	Sig. (2-tailed)	.550	.297	.765	.119		
	N	10	9	6	6	10	1
E. coli	Pearson Correlation	-1.000**	1.000**	1.000**	1.000**	[a]	1.000
	Sig. (2-tailed)						
	N	2	2	2	2	1	2

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

a. Cannot be computed because at least one of the variables is constant.

June 1999 – May 2000

Table 4.25: **Correlations between age and infections in pregnant women at Nsawam**

		age of women	*A. lumbricoides*	*Intestinal flagellates*	*Hookworm ova*	*E. coli*
age of women	Pearson Correlation	1.000	-.495*	-.221	-.294	[a]
	Sig. (2-tailed)		.023	.490	.410	
	N	40	21	12	10	1
A. lumbricoides	Pearson Correlation	-.495*	1.000	467	457	[a]
	Sig. (2-tailed)	.023		.126	.185	
	N	21	21	12	10	0
Intestinal flagellates	Pearson Correlation	-.221	467	1.000	.502	[a]
	Sig. (2-tailed)	490	.126		.205	
	N	12	12	12	8	0
Hookworm ova	Pearson Correlation	-.294	.457	.502	1.000	[a]
	Sig. (2-tailed)	.410	.185	.205		
	N	10	10	8	10	0
E. coli	Pearson Correlation	[a]	[a]	[a]	[a]	[a]
	Sig. (2-tailed)					
	N	1	0	0	0	1

*. Correlation is significant at the 0.05 level (2-tailed).

a. Cannot be computed because at least one of the variables is constant.

June 2000 –May 2001

Table4. 4.26: Correlations between age and infections in Pregnant women at Nsawam

		age of women	*A. lumbricoides*	*Intestinal flagellates*	*Hookworm ova*	*Strongyloides*	*E. coli*
age of women	Pearson Correlation	1.000	-.579**	-.170	-.033	a	a
	Sig. (2-tailed)		.000	.578	.924		
	N	60	39	13	11	1	0
A. lumbricoides	Pearson Correlation	-.579**	1.000	-.403	-.094	a	a
	Sig. (2-tailed)	.000		.194	.796		
	N	39	39	12	10	0	0
Intestinal flagellates	Pearson Correlation	-.170	-.403	1.000	-.304	a	a
	Sig. (2-tailed)	.578	.194		.558		
	N	13	12	13	6	1	0
Hookworm ova	Pearson Correlation	-.033	-.094	-.304	1.000	a	a
	Sig. (2-tailed)	.924	.796	.558			
	N	11	10	6	11	1	0
Strongyloides	Pearson Correlation	a	a	a	a	a	a
	Sig. (2-tailed)						
	N	1	0	1	1	1	0
E. coli	Pearson Correlation	a	a	a	a	a	a
	Sig. (2-tailed)						
	N	0	0	0	0	0	0

**. Correlation is significant at the 0.01 level (2-tailed).

a. Cannot be computed because at least one of the variables is constant.

June 2001-May 2002

Table 4.27: Correlations between age and infections in Pregnant women at Nsawam

		age of women	*A. lumbricoides*	*Intestinal flagellates*	*Hookworm*	*Strongyloides*	*E. coli*
age of women	Pearson Correlation	1.000	-.727**	-.558*	-.443	-.417	[a]
	Sig. (2-tailed)	.	.000	.025	.113	.264	.
	N	60	35	16	14	9	0
A. lumbricoides	Pearson Correlation	-.727**	1.000	.697**	.612*	.661	[a]
	Sig. (2-tailed)	.000	.	.003	.020	.053	
	N	35	35	16	14	9	0
Intestinal flagellates	Pearson Correlation	-.558*	.697**	1.000	.425	.188	[a]
	Sig. (2-tailed)	.025	.003	.	.254	.721	
	N	16	16	16	9	6	0
Hookworm	Pearson Correlation	-.443	.612*	.425	1.000	.592	[a]
	Sig. (2-tailed)	.113	.020	.254		.293	
	N	14	14	9	14	5	0
Strongyloides	Pearson Correlation	-.417	.661	.188	.592	1.000	[a]
	Sig. (2-tailed)	.264	.053	.721	.293	.	.
	N	9	9	6	5	9	0
E. coli	Pearson Correlation	[a]	[a]	[a]	[a]	[a]	[a]
	Sig. (2-tailed)	.	.	.	.	.	.
	N	0	0	0	0	0	0

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

a. Cannot be computed because at least one of the variables is constant.

June 2002 – May 2003

Table 4.28: **Comparing means of the infections in Pregnant women over the years (1999 - 2003) at Nsawam**

Years of infectio		*A. lumbricoides*	*Intestinal flagellates*	*Hookworm*	*Strongyloides*	*E. coli*	*E. histolytica*	*H. nana*
1999-2000	Mean	17.6667	2.4783	1.8421	1.4000	1.5000		
	N	36	23	19	10	2		
	Std. Deviatio	10.8838	1.4419	1.3443	.9661	.7071		
2000-2001	Mean	16.7619	2.5000	2.6000	1.0000			
	N	21	12	10	1			
	Std. Deviatio	14.7442	2.4680	1.3499	.			
2001-2002	Mean	12.9706	4.5714	2.2500	6.0000			
	N	34	14	12	2			
	Std. Deviatio	12.5057	8.2993	1.7645	4.2426			
2002-2003	Mean	14.3333	1.6667	2.2000	2.2500		3.0000	1.0000
	N	30	12	10	8		2	1
	Std. Deviatio	11.9376	1.2309	1.4757	.8864		1.4142	.
Total	Mean	15.3636	2.8033	2.1569	2.1429	1.5000	3.0000	1.0000
	N	121	61	51	21	2	2	1
	Std. Deviatio	12.3309	4.2576	1.4611	1.8516	.7071	1.4142	.

June 1999- May 2003

Table 4.29

Age (in years)			Statistics	Months								Total
				Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	
16-25	Type of worm	*Intestinal flagellates*	Count		2	2	2	1	1		1	9
			% of Total		22.2%	22.2%	22.2%	11.1%	11.1%		11.1%	100.0%
		Total	Count		2	2	2	1	1		1	9
			% of Total		22.2%	22.2%	22.2%	11.1%	11.1%		11.1%	100.0%
26-35	Type of worm	*Intestinal flagellates*	Count	2	1	2	2	3	1	2	1	14
			% of Total	12.5%	6.3%	12.5%	12.5%	18.8%	6.3%	12.5%	6.3%	87.5%
		Hookworm	Count				1					1
			% of Total				6.3%					6.3%
		Strongyloids	Count						1			1
			% of Total						6.3%			6.3%
		Total	Count	2	1	2	3	3	2	2	1	16
			% of Total	12.5%	6.3%	12.5%	18.8%	18.8%	12.5%	12.5%	6.3%	100.0%
36-45	Type of worm	*Intestinal flagellates*	Count	1				2			1	4
			% of Total	25.0%				50.0%			25.0%	100.0%
		Total	Count	1				2			1	4
			% of Total	25.0%				50.0%			25.0%	100.0%

Table : Adabraka Polyclinic (Jun 2002 - May 2003)

a. No recordings for June, July and August

Table 4:30

Age	Statistics	Type of worm							Total
		Ascaris	*Intestinal flagellates*	*Hookworm*	*Strongyloides*	*S. mansoni*	*H. nana*	*S. haemotobium*	
11-15	Count			1					1
	% of Total			.6%					.6%
16-25	Count	1	59	9	2	1	3	12	87
	% of Total	.6%	36.6%	5.6%	1.2%	.6%	1.9%	7.5%	54.0%
26-35	Count		54	4				3	61
	% of Total		33.5%	2.5%				1.9%	37.9%
36-45	Count		11						11
	% of Total		6.8%						6.8%
46-50	Count						1		1
	% of Total						.6%		.6%
Total	Count	*1*	*124*	*14*	*2*	*1*	*4*	*15*	*161*
	% of Total	*.6%*	*77.0%*	*8.7%*	*1.2%*	*.6%*	*2.5%*	*9.3%*	*100.0%*

Mamobi (2002-2003)