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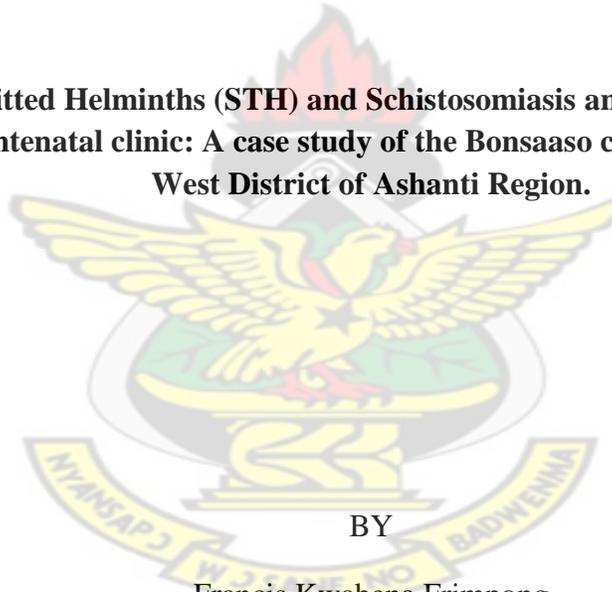
**DEPARTMENT OF THEORETICAL AND APPLIED BIOLOGY**

**(MSc. ENVIRONMENTAL SCIENCE)**

**INSTITUTE OF DISTANCE LEARNING**

**KNUST**

**Soil Transmitted Helminths (STH) and Schistosomiasis among Pregnant women  
attending antenatal clinic: A case study of the Bonsaaso cluster in the Amansie  
West District of Ashanti Region.**



BY

Francis Kwabena Frimpong

(BSc. Laboratory Technology)

December 2013

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A Thesis Submitted to the Department of Theoretical and Applied Biology, Kwame Nkrumah University of Science and Technology in partial fulfillment of the requirements for the degree of  
**MASTER OF SCIENCE (Environmental Science).**

**BY**

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**(BSc. Laboratory Technology)**

**December 2013**

**DECLARATION**

I hereby declare that this submission is my own work towards the MSc and that to the best of my knowledge, it contains no material previously published by another person nor material which has been accepted for award of any other degree of the University, except where due acknowledgement has been made in the text.

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Date

Dr. JOHN A. LARBI .....

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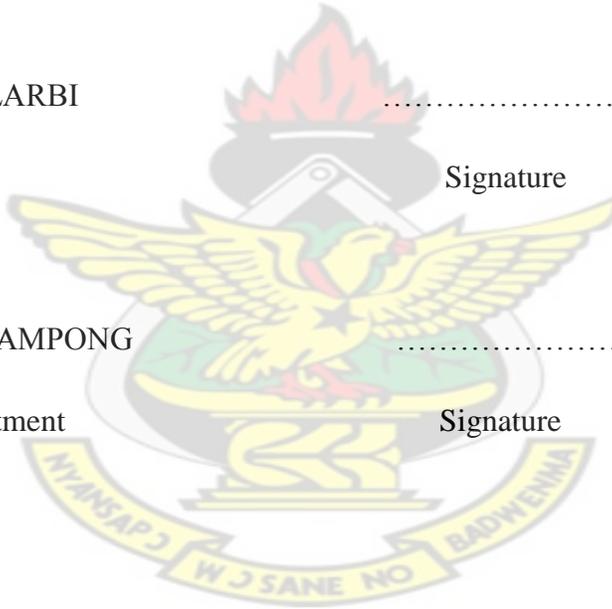
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I give thanks to God Almighty who provided the life, strength, and academic enthusiasm needed to undertake this project.

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And finally, thanks to my wife, parents and numerous friends who endured this long process with me, always offering support and love.

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## ABSTRACT

Soil Transmitted Helminths (STHs) and Schistosomiasis infections are known to cause high morbidity as well as reduce haemoglobin levels causing anaemia. These infections as well as anaemia during pregnancy are common in sub-Saharan Africa and are a major public health problem in developing countries. The study was carried out to determine STHs and Schistosomiasis among pregnant women in Bonsaaso Cluster in the Amansie West District. A total of 186 women attending antenatal clinic within the Bonsaaso cluster took part in the study after their consent was sought. Stool and urine samples were collected from the participants as well as some demographic information through a structured questionnaire. Sedimentation and formol ether methods were used for examination of the urine and stool samples respectively. The mean age of the women was 26 years from 15- 41 years. Most of the women were farmers (46%) and 50% in their second trimester. Most of the pregnant women (53%) were infected with at least one species of the soil transmitted helminths with hookworm being the most prevalent (23%) while eleven percent (11%) of all the urine examined had *S.haematobium* eggs. In all, 81% of the participants had their haemoglobin levels below the recommended value by WHO (11g/dl) for pregnant women. Infection with soil transmitted helminths is high among pregnant women in the cluster especially the farmers, and thus calls for appropriate control interventions.



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## CHAPTER ONE

### 1.0 INTRODUCTION

Soil Transmitted Helminth (STH) infection is found mainly in areas with warm and moist climates where sanitation and hygiene are poor (CDC, 2013). STH and Schistosomiasis (snail fever or Bilharziasis) are considered Neglected Tropical Diseases (NTDs) because they inflict tremendous disability and suffering although they can be controlled or eliminated. Whereas intestinal parasites often find their way into a host through eating infested food, drinking contaminated water and walking on infected soil bare footed, urinary schistosomiasis affects those who come into contact with infected water bodies. They are therefore of public health concern especially in sub-Saharan Africa. The World Health Organization (WHO) estimated that schistosomiasis and Soil Transmitted Helminthiasis represent more than 40% of the global disease burden caused by all tropical diseases excluding malaria (WHO, 2006a)

These diseases have long been recognized as an important cause of blood loss leading to iron deficiency and protein malnutrition (Baidoo *et al.*, 2010). Intestinal parasites and schistosomiasis cause loss of blood through gastrointestinal bleeding and haematuria respectively resulting in low haemoglobin level.

Intestinal worms occur throughout the developing world, but are most commonly seen in the poorest communities. The World Health Organization (WHO) has produced overwhelming evidence to show that the burden caused by many of the seventeen neglected tropical diseases including soil transmitted helminths and schistosomiasis that

affect one billion people worldwide can be effectively controlled and in many cases eliminated or even eradicated (WHO, 2012).

The aim of the Millennium Development Goals (MDGs) is to encourage development by improving the health, social and economic conditions in the world's poorest countries. One of the Millennium Development Goals (MDG 5) is to improve maternal health with a target of reducing maternal mortality ratio by three quarters by the year 2015. Maternal deaths result from a wide range of direct and indirect causes. Indirect causes represent 20% of the global total (WHO, 2005). They are caused by diseases (pre-existing or concurrent) that are not complications of pregnancy, but complicate pregnancy or are aggravated by it. These include malaria, anaemia, HIV/AIDS and cardiovascular disease. The commonest causes of anaemia are poor nutrition, iron and other micronutrient deficiencies, malaria, hookworm and schistosomiasis. There are seventeen diseases classified as Neglected Tropical Diseases (NTDs) which are especially endemic in low income populations in developing countries including Ghana (Hotez *et al.*, 2009). Since 2004 there has been an increased recognition of the importance of NTDs as impediments to development and that these diseases should be addressed as part of the MDG agenda where they are considered as "other diseases" of MDG 6 (Molyneux *et al.*, 2011).

Of the current estimates of two billion people infected with these worms (helminthiasis and schistosomiasis), about three hundred million suffer severe and permanent impairments as a result. (World Bank, 1993). While these figures are not reflected in huge number of deaths, the consequences for health and development are enormous. Apart from permanent organ damaged, worm infections cause anaemia, poor physical

growth, poor intellectual development and impaired cognitive function (Crompton and Nesheim, 2002).

Poor nutrition in general and anaemia in particular are the main underlying causes of poor pregnancy outcomes in the developing world. More than 44 million infected pregnant women suffer significant morbidity and contributory mortality due to hookworm-associated anaemia. Approximately 135,000 deaths occur per year, mainly due to infections with the hookworms, or the roundworm *Ascaris lumbricoides* (Awasthi *et al.*, 2003). In anaemic women, the risk of dying during pregnancy or child birth is up to three and half times higher than in non-anaemic women (Brabin *et al.*, 2001). Pregnant women who are also infected with STH especially hookworm could spontaneous abort (Brooker *et al.*, 2008).

It is estimated that half of the ten million pregnant women in Africa alone who are infected with Schistosomiasis suffer from anaemia (King, 2004). An estimated forty four million of the developing world's one hundred and twenty four million pregnant women harboured hookworm infection alone (Huddle *et al.*, 1999) of which more than ten percent suffer worm burdens heavy enough to adversely affect intrauterine the growth as well as the weight of the baby at birth. During pregnancy the fetus and placenta need their own supply of iron and this can be obtained from the mother. While plasma volume and red blood cell mass are both known to expand during pregnancy, plasma volume grows to a greater extent, therefore diluting the maternal haemoglobin concentration (Penelop, 2002).

Low birth weight can also be associated with maternal anaemia due to preterm labour induced by low Hb levels (Allen, 2000). Data shows that anaemia early in pregnancy is

associated with increased risk of preterm premature rupture of membranes, while anemia later in pregnancy is associated with spontaneous premature labour (Zhang *et al.*, 2009). Furthermore many short-term and long term outcomes associated with preterm infants born with a low birth weight, include cerebral palsy, blindness, deafness, hydrocephaly, diabetes, hypertension and heart disease (Rusia *et al.*, 1995). As a result the mother must have enough blood to nourish the growing fetus in the womb. Any situation that will facilitate the loss of blood therefore becomes inimical to the expectant mother.

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### **1.1 Justification**

Amansie West district has fertile lands and is one of the many districts in Ghana that abounds in minerals such as gold. The inhabitants are either farmers or engaged in illegal small scale mining popularly known as ‘Galamsey’ and are therefore in direct constant contact with soil. Though the nature of these two predominant occupations is challenging, female counterparts are not left out. They lack proper personal protective equipment coupled with poor hygiene, thus the pregnant woman who works on infested soil without observing proper hygienic practices is at a greater risk.

During pregnancy haemoglobin levels are reduced to a varying extent occasionally as low as 80 %. (Chandrakala *et al.*, 2013). With increasing knowledge on reproductive health, the significance of schistosomiasis and soil transmitted helminths has become important to reconsider among adults who also happen to be the reproductive and productive component of society. The low level of sanitation practices, nature of

vegetation and presence of dams and rivers make them susceptible to helminthiasis and schistosomiasis. However, there is no available data on these two diseases in the cluster.

This project seeks to find the prevalence of Soil Transmitted Helminths and Schistosomiasis among pregnant women attending antenatal clinic.

### **1.2.1 Main Objective**

The overall objective of this study is to determine the prevalence of soil transmitted helminthes and schistosomiasis among pregnant women attending antenatal clinic.

### **1.2.2 Specific objectives**

**These are to determine;**

- i. The types and prevalence of STHs and urinary schistosomiasis among pregnant women attending antenatal clinic.
- ii. The intensity of infection of STHs and urinary schistosomiasis.
- iii. The haemoglobin level of the pregnant women.

## CHAPTER TWO

### LITERATURE REVIEW

#### 2.1 Helminths

Helminths are worms causing a wide variety of diseases globally called helminthiasis. They are multicellular worms and do not normally multiply in the human host. There is increasing evidence that humans may be predisposed to heavy or light infection with variety of helminths including *A. lumbricoides*. Helminthiasis almost only occurs in developing countries particularly in areas where sanitation is low. Helminth eggs are discharged to the environment in faeces and oral faecal route is the main dissemination pathway of the disease.

#### 2.2 Classification of Helminths

There are three different kinds of helminths. These are Nematelminths, Platyhelminths and Annelid. Those infecting humans through wastewater, sludge or faecal sludge belong only to the first two groups. (Bird and Bird, 1991)

#### 2.3 Nematelminths (Nematodes)

Nematodes commonly referred to as roundworms can generally be grouped into two. The tissue nematodes and intestinal nematodes. The intestinal nematodes include *Enterobius vermicularis* (threadworm) *Ascaris lumbricoides* (large roundworm),

*Strongyloides stercoralis*, hookworm) and *Trichuris trichiura* (whipworm) [Cheesbrough, 2006].

They have a body cavity and cuticle which may smooth, spined or ridged. The adults of some species are very long. In some species example hookworm the lips open into a buccal cavity which has cutting or tooth-like plates. The digestive system is a simple tube which ends in anus. There is an excretory system and a nervous system .Most of the medically important intestinal nematodes is geohelminths (soil transmitted) spread by faecal contamination of the soil. A person becomes infected by swallowing infective eggs.

#### **General Life Cycle of Soil Transmitted Helminths.**

The dynamic process involved in STH transmission (free-living infective stages of development and survival) depend on the prevailing environmental conditions, climate is an important determinant of transmission of STH infections with adequate moisture and warm temperature essential for larval development in the soil (Brooker, 2006). The *A.lumbricoides* is a roundworm that infests the entire small intestine; the adult hookworm of the *Necator* and *Ancylostoma* genera parasite the upper part of the human small intestine and the adult *T.trichiura* lives in the large intestine, especially in the cecum. The STH vary greatly in size and female worms are larger than males (Despommier *et al*, 2005). The STH infection life cycle follows a general pattern, the parasites in adult stages inhabit part of the host intestine (*A.lumbricoides* and hookworm inhabit the small intestine, *T.trichiura* the colon), reproduce sexually and produce eggs which are passed in human faeces and deposited in the external environment. Adult

worms survive for several years and produce large numbers of eggs. Eggs can remain viable in the soil for several months (*A.lumbricoides* and *T.trichiura*) and larvae can survive for several weeks (hookworms), depending on the prevailing environmental conditions. Hookworm larvae can undergo hypobiosis (arrested development at a specific point in the nematode life cycle) in the human body under certain environmental conditions for several months. Infection occurs through accidental ingestion of eggs (*A.lumbricoides* and *T.trichiura*) or penetration of the skin by hookworm larvae.

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### **2.3.1 *Ascaris lumbricoides***

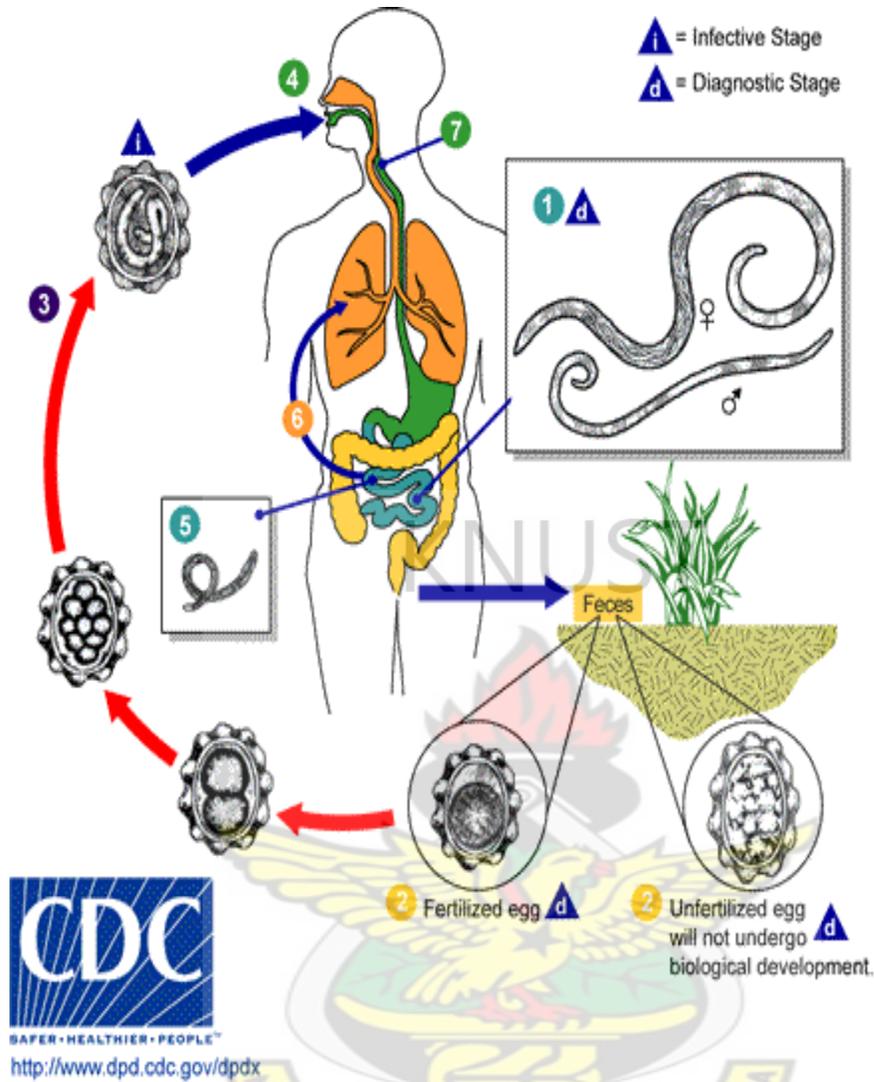
The human roundworm *Ascaris lumbricoides* is one of the most common parasites in the world infecting 1.2 billion people generally (de silva *et al*,2003).Disease associated with *A. lumbricoides* infection is known as Ascariasis and is caused by ingesting the eggs. This can happen when hands or fingers that have contaminated dirt on them are put in the mouth or by consuming vegetables or fruits that have not been carefully cooked, washed or peeled. Ascariasis is considered neglected tropical disease (Chan, 1997) with morbidity assessed as disability-adjusted life years (DALYs) of approximately 10.5 million (Hotez *et al*; 2008). People infected with *Ascaris lumbricoides* often show no symptoms. If symptoms do occur they can be light and include abdominal discomfort. Heavy infections can cause intestinal blockage and impair growth in children. Other symptoms such as cough are due to migration of the worms through the body. Adult *Ascaris lumbricoides* live in the human intestines principally in the lumen of the small intestine. Although the infection is most frequent in moist tropical areas, it can occur wherever poor sanitation provides opportunities for

faecal oral transmission. The white or pink worm is identified by large size, smooth finely striated cuticle, conical anterior and posterior extremities, ventrally curved papillated posterior extremity of male with two spicules, terminal mouth with three oval lips with sensory papillae and paired reproductive organs in posterior two thirds of female and a single long tortuous tubule in male. There is an outer coarsely mammillated, albuminous covering that serves as an auxiliary barrier to permeability but may be absent. The larvae develop inside the eggs and when these have been ingested they hatch out in the host's intestine. Eggs are excreted in the faeces and their thick walls enable them to survive for many months or years in cold and dry conditions. The proper egg has a thick, transparent, hyaline shell with a relatively thick outer layer that acts as a supporting structure and a delicate vitelline, lopoidal, inner membrane that is highly impermeable. At oviposition the shell contains an ovoid mass of impregnated with lecithin granules. Bizarrely shaped eggs without albuminous coating or with abnormally extensive and irregular coating are also found. The infertile eggs are difficult to identify and may be missed by the unwary and untutored. They are found not only in the absence of males but in about two fifths of all infections since repeated copulations are necessary for the continuous production of fertile eggs.

#### **2.3.1.1 Life cycle of *Ascaris lumbricoides***

Hosts contract *Ascaris* infection via the faecal-oral route. It is known that when infective eggs are ingested and hatch, *Ascaris* larvae develop in host parenteral tissue (Dold and Holland, 2010). The adult worms normally live in the lumen of the small intestine. They obtain their nourishment from the semi digested food of the host. Male and female adult worms measure 15-25cm and 20-35cm respectively. Estimates of daily

*Ascaris* female egg production generally are in the range of 200,000 eggs (Sinniah *et al.*,1982) but the number of eggs a female produces decreases with worm load (Sinniah *et al.*,2009). Unembryonated ova enter the environment via the faeces and can remain viable in the soil for up to 15 years (WHO, 1967). During embryonation, larvae undergo two moults in the egg (Roepstorff, 2003).Male or female worms are found alone in very lightly infected persons. The eggs are unsegmented when they leave the host in the faeces. Under favourable environmental conditions in the soil, infective second stage larvae after the first molt are formed within the egg shell in about three weeks. The optimal temperature for development is about 25 degrees ranging from 21 degrees to 30 degrees. Lower temperatures retard development but favour survival (Strauss and Blumenthal, 1990). Temperatures above 60 degrees are necessary to destroy *Ascaris* eggs (Vinneras *et al.*, 2003). When human faeces are used as fertilizer, they should first be composted but although the temperature may exceed 60 degrees Celsius in the centre of a well made heap, it is unlikely to do so in the outer layers or in poorly made heaps (Schönning *et al.*, 2007).



**Figure1: Life cycle of *Ascaris lumbricoides***

Adult worms [1] live in the lumen of the small intestine. A female may produce approximately 200,000 eggs per day, which are passed with the faeces [2]. Unfertilized eggs may be ingested but are not infective. Fertile eggs embryonate and become infective after 18 days to several weeks [3], depending on the environmental conditions (optimum: moist, warm, shaded soil). After infective eggs are swallowed [4] the larvae hatch [5], invade the intestinal mucosa, and are carried via the portal, then systemic circulation to the lungs [6]. The larvae mature further in the lungs (10 to 14 days),

penetrate the alveolar walls, ascend the bronchial tree to the throat, and are swallowed [7]. Upon reaching the small intestine, they develop into adult worms [1]. Between 2 and 3 months are required from ingestion of the infective eggs to oviposition by the adult female. Adult worms can live 1 to 2 years.

### **2.3.1.2 Epidemiology**

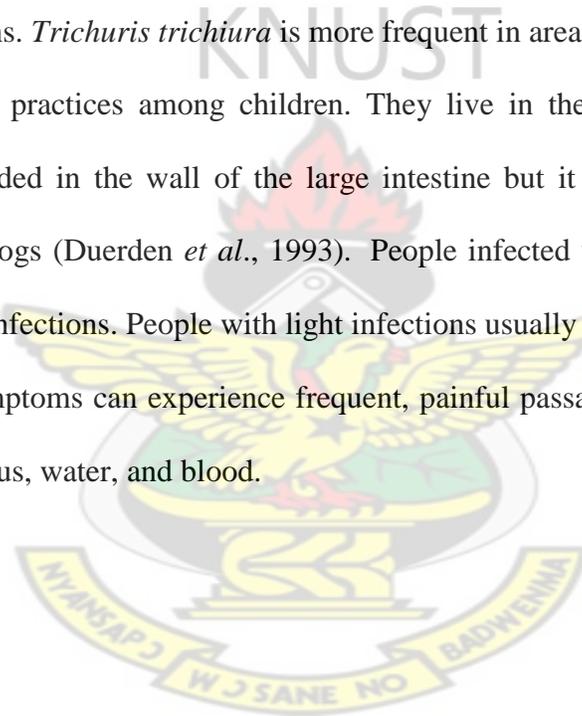
*A. lumbricoides* is a prominent parasite in the both temperate and tropical zones, but it is more common in warm countries and is most prevalent where sanitation is poor. More than one fourth of the earth's population is estimated to harbor *Ascaris* (Pawlowski and Davis, 1989). The poorer urban and the rural classes, because of heavy soil pollution and unsatisfactory hygiene are most afflicted. Infection is a household affair, the family being the unit of dissemination (Chan *et al.*, 1994). Infected small children provide the chief source of soil contamination by their promiscuous defecation in dooryards and earthen floored houses where the resistant eggs remain viable for long periods. The infective eggs are chief transmitted hand to mouth by children who have come in contact with contaminated soil directly, through playthings or through dirt eating. Infection in all ages is also derived from vegetables. Drinking water is rarely a source of infection. A moist, loose soil with moderate shade provides a suitable environmental. The eggs are destroyed by direct sunlight within 15 hours and are killed at temperatures above 40 degrees perishing within an hour at 50 degrees (Vinneras *et al.*, 2003). Eggs are resistant to chemical disinfectants and can withstand temporary immersion in strong chemicals. They survive for months in sewage or night soil (Schönning *et al.*, 2007).

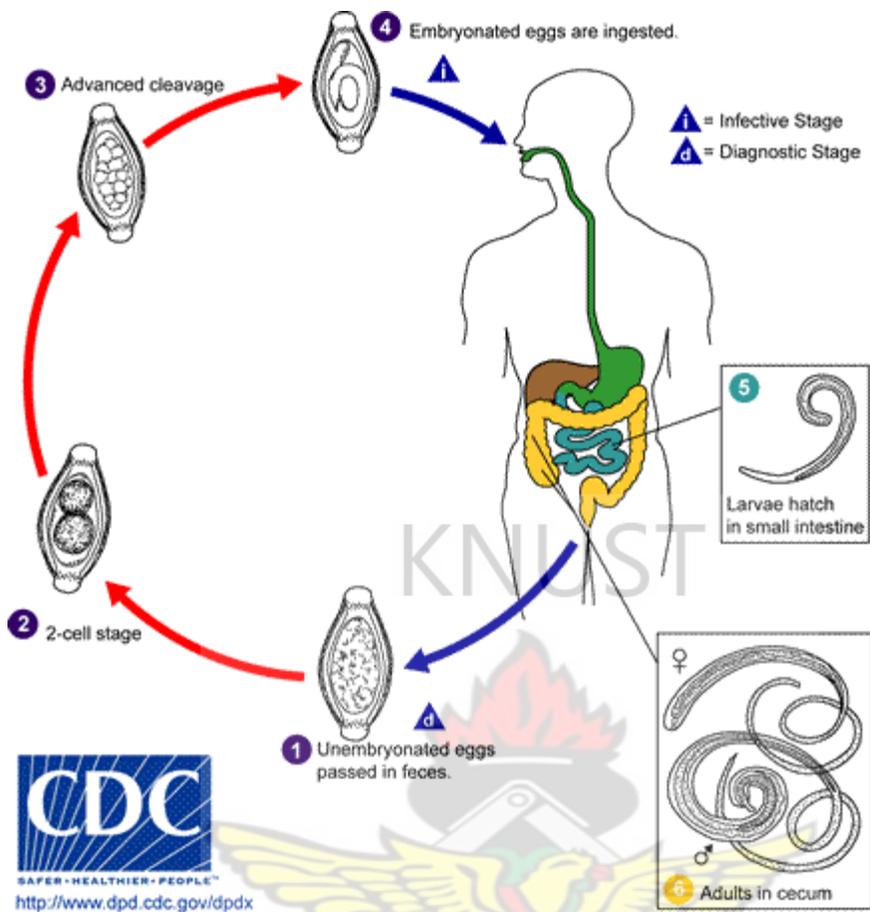
### 2.3.1.3 Diagnosis

Diagnosis is made by finding the eggs in the faeces. The numerous eggs are detected in the direct cover glass mount. If direct examination is negative, concentration technique may be employed (Cheesbrough, 2006).

### 2.3.2 *Trichuris Trichiura* (Whipworm)

It is estimated that 800 million people are infected worldwide and most common round worm of humans. *Trichuris trichiura* is more frequent in areas with tropical weather and poor sanitation practices among children. They live in the human intestine with its anterior embedded in the wall of the large intestine but it has also been reported in monkeys and hogs (Duerden *et al.*, 1993). People infected with whipworm can suffer light or heavy infections. People with light infections usually have no symptoms. People with heavy symptoms can experience frequent, painful passage of stool that contains a mixture of mucus, water, and blood.





**Figure2: Life cycle of *Trichuris trichiura***

The unembryonated eggs are passed with the stool [1]. In the soil, the eggs develop into a 2-cell stage [2], an advanced cleavage stage [3], and then the embryonate [4]; eggs become infective in 15 to 30 days. The eggs are less resistant to desiccation, heat and cold than are those of the *Ascaris lumbricoides*. After ingestion (soil-contaminated hands or food), the eggs hatch in the small intestine, and release larvae [5] that mature and establish themselves as adults in the colon [6]. The adult worms (approximately 4 cm in length) live in the cecum and ascending colon. The adult worms are fixed in that location with a spear like projection at its anterior extremity which enables the worm to penetrate into and embed its whip like anterior portion in the intestinal mucosa of the

host, whence it derives its nourishment. Its secretions possibly may liquefy the adjacent mucosal cell. The females begin to oviposit 60 to 70 days after infection. Female worms in the cecum shed between 3,000 and 20,000 eggs per day. The life span of the adults is about 1 year.

### **2.3.2.1 Epidemiology**

The prevalence of whipworm infection is high but its intensity is usually light. Hundreds of millions of people throughout the world are infected, the prevalence ranging as high as 80 percent in certain tropical countries (Neva and Brown, 1994). Its distribution is coextensive with that of *Ascaris lumbricoides*. The highest incidence is found in the regions of heavy rainfall, subtropical climate and highly polluted soil. Children are more frequently infected than adults. (Neva and Brown, 1994). The heaviest infections are in young children, who live largely at ground level, habitually contaminated the soil, and pick up infection from polluted dooryards. Infection results from the ingestion of embryonated eggs via hands food or drink that have been contaminated directly by infested soil.

### **2.3.2.2 Pathology**

*Trichuris trichiura* lives primarily in the human cecum but it is also found in the appendix and lower ileum. Light infections produce few symptoms. The worms apparently suck some blood of their host but the haemorrhage that may occur at their attachment sites is probably a greater source of blood loss. Approximately 0.005 ml of blood is lost per day per each *T. trichiura* (Duerden *et al.*, 1993). The white blood cell

count and differential are usually normal. Eosinophilia is encountered only rarely in uncomplicated *Trichuris* infections.

### **2.3.2.2 Diagnosis**

*Trichuriasis* cannot be differentiated clinically from infections with other intestinal nematodes. Diagnosis is made by finding the characteristic yellow-brown barrel shaped eggs, 50-56 $\mu$ m by 20-22 $\mu$ m in the faeces with double shell and translucent knobs at each of the two poles. Light infections may necessitate the use of concentration methods (Cheesbrough, 2006).

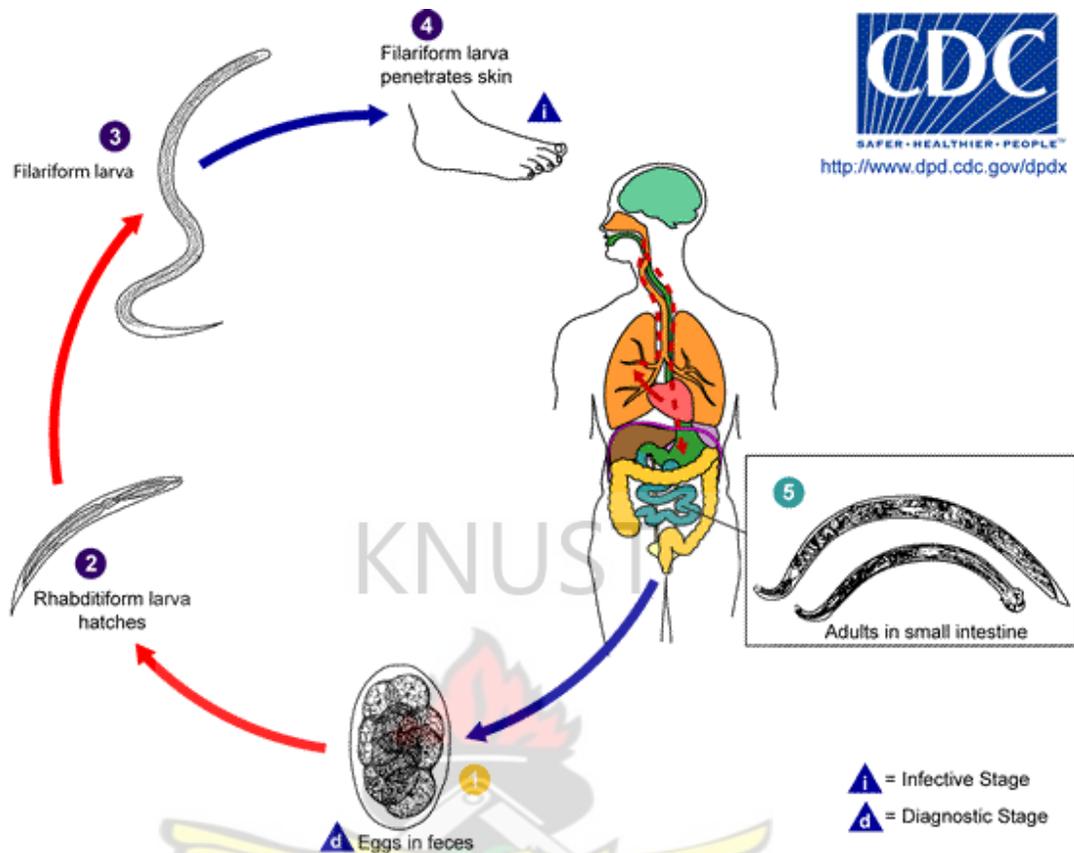
### **2.3.3 Hookworm**

There are multiple species of hookworm, but almost all cases of human hookworm infection are caused by the nematode parasites *Necator americanus* and *Ancylostoma duodenale*, with the former predominating in most of the world (Hotez *et al.*, 2004). An estimated 740 million people in the world are infected with hookworm (de Silva *et al.*, 2003). The key environmental components for ensuring hookworm transmission and endemicity are temperature, soil moisture and soil type (Brooker *et al.*, 2004). The World Health Organization defines moderate intensity infections as those with 2,000–3,999 eggs per gram of feces, and heavy-intensity infections as those with 4,000 or more eggs per gram (Montresor *et al.*, 2002).

#### **2.3.3.1 Morphology**

*A. duodenale* worms are grayish white or pinkish with the head slightly bent in relation to the rest of the body. This bend forms a definitive hook shape at the anterior end for

which hookworms are named. They possess well developed mouths with two pairs of teeth. While males measure approximately one centimeter by 0.5 millimeter, the females are often longer and stouter. Additionally, males can be distinguished from females based on the presence of a prominent posterior copulatory bursa. (Markell *et al.*, 2006). *N. americanus* is very similar in morphology to *A. duodenale*. *N. americanus* is generally smaller than *A. duodenale* with males usually 5 to 9 mm long and females about 1 cm long. Whereas *A. duodenale* possess two pairs of teeth, *N. americanus* possesses a pair of cutting plates in the buccal capsule. Additionally, the hook shape is much more defined in *Necator* than in *Ancylostoma* (Markell *et al.*, 2006). *A. duodenale* is larger than *N.americanus* (Neva *et al.*, 1994). The worm has a relatively thick cuticle. There are single male and pairs female reproductive organs. The posterior end of the male has a broad, translucent, membranous caudal bursa with rib like rays, which is used for attachment to the female during copulation. The chief morphological differences in the species are in the shape, buccal capsule and amle bursa. The vulva is located anterior to the middle of the body in *Necator* and posterior in *Ancylosstoma*. In the buccal capsule *N.americanus* has a conspicuous dorsal pair of semilunar cutting plates, a concave dorsal median tooth and a deep pair of triangular subventral lancets.



**Figure 3: Life cycle of Hookworm**

Eggs are passed in the stool [1], and under favorable conditions (moisture, warmth, shade), larvae hatch in 1 to 2 days. The released rhabditiform larvae grow in the feces and/or the soil [2], and after 5 to 10 days (and two molts) they become filariform (third-stage) larvae that are infective [3]. These infective larvae can survive 3 to 4 weeks in favorable environmental conditions. On contact with the human host, the larvae penetrate the skin and are carried through the blood vessels to the heart and then to the lungs. They penetrate into the pulmonary alveoli, ascend the bronchial tree to the pharynx, and are swallowed [4]. The larvae reach the small intestine, where they reside and mature into adults. Adult worms live in the lumen of the small intestine, where they attach to the intestinal wall with resultant blood loss by the host [5]. Most adult

worms are eliminated in 1 to 2 years, but the longevity may reach several years. Some *A. duodenale* larvae, following penetration of the host skin, can become dormant (in the intestine or muscle). In addition, infection by *A. duodenale* may probably also occur by the oral and trans-mammary route. *N. americanus*, however, requires a trans pulmonary migration phase.

### **2.3.3.3 Epidemiology**

The present distribution has been brought about by the migration of people and extends in the tropical and sub-tropical zones between 45 degrees north and 30 degrees south latitude (Neva *et al.*, 1994). It is estimated that throughout the world the hookworms harboured by 500 million persons cause a daily blood loss of more than one million liters (Neva *et al.*, 1994).

Factors that facilitate the maintenance and dispersal of hookworm infection include defecation on the soil by infected individuals in areas where others come for work or play, presence of shaded sandy soil or loam instead of tightly packed lay soil, a warm climate, sufficient but not excessive moisture to prevent desiccation of eggs and larvae and a population that goes bare foot or wears only simple sandals (Neva *et al.*, 1994).

Hookworm infection is virtually limited to the tropics and is a rural disease. It is particularly common in plantations of cocoa, banana, sugar or coffee where the crops provide privacy for defecation where dense shade, high humidity and good fertile soil provide ideal conditions for larval developments and where agricultural work is often carried out by hand. These are the settings and circumstances that provide ideal conditions for transmission of hookworm. Even though promiscuous defecation is

common in the arid tropics, the dry climate and the hot sun tend to inhibit larval development and survival. Infective larvae can penetrate unbroken skin when any part of the body example an unprotected foot, buttock or hand comes into contact with ground in which they are present.

#### **2.3.3.4 Pathology**

Adult hookworms cause morbidity in the host by producing intestinal hemorrhage (Hotez *et al.*, 2004). The adult hookworms then ingest the blood, rupture the erythrocytes and degrade the haemoglobin (Williamson *et al.*, 2003). Therefore, the disease attributed to hookworm is silent blood loss leading to iron deficiency anemia and protein malnutrition. There is a correlation between parasite intensity and host intestinal blood loss (Stoltzfus *et al.*, 1997) in children, women of reproductive age, and other populations with low iron stores. There is often a correlation between parasite intensity and reductions in host hemoglobin (Bundy *et al.*, 1995). Adult hookworms cause chronic blood loss. It has been estimated that a single *A. duodenale* worm ingest about 0.15ml of blood per day and *N. americanus* worm about 0.03ml (Cheesbrough, 2006). In pregnant women, they are associated with adverse maternal–fetal outcomes (Bundy *et al.*, 1995). Hookworm infection is acquired by invasion of the infective larval stages through the skin (*A. duodenale* larvae are also orally infective). Following host entry, the larvae undergo a journey through the vasculature, then the lungs and other tissues, before they enter the gastrointestinal tract and molt twice to become one-centimeter-long adult male and female worms (Hotez *et al.*, 2004). Because hookworms do not replicate in humans, the morbidity of hookworm is highest among patients that harbour large numbers of adult parasites. Estimates of the intensity of hookworm

infection are typically obtained by using quantitative fecal egg counts as a surrogate marker for worm burden.

#### **2.3.4.5 Diagnosis**

Hookworm infection is readily diagnosed by microscopy of freshly passed faeces for the presence of the characteristic eggs those of the two species are indistinguishable (Cheesbrough, 2006) but if faeces are kept at average day temperatures in the tropics then it is possible to distinguish *Ancylostoma* larvae from those of *Necator* by examining the mouth parts. The egg has bluntly rounded ends and a single thin transparent hyaline shell. It is unsegmented at oviposition and in two to eight cell stages of division in fresh faeces. Eggs are found in direct faecal films but in light infections concentration methods may required.

#### **2.3.4 *Strongyloides stercoralis*.**

*Strongyloides stercoralis* causes the disease of strongyloidiasis. *S. stercoralis* has a very low prevalence in societies where faecal contamination of soil or water is rare. Hence, it is a very rare infection in developed economies. In developing countries it is less prevalent in urban areas than in rural areas where sanitation standards are poor (Segarra-Newnham, 2007).

#### **2.3.4.1 Morphology**

Whereas males grow to only about 0.9 mm in length, females can be anywhere from 2.0 to 2.5 mm. Both genders also possess a tiny buccal capsule and cylindrical esophagus without a posterior bulb (Roberts and Janovy, 2005). In the free-living stage, the esophagi of both sexes are rhabditiform. Males can be distinguished from their female counterparts by two structures; the spicules and gubernaculum.

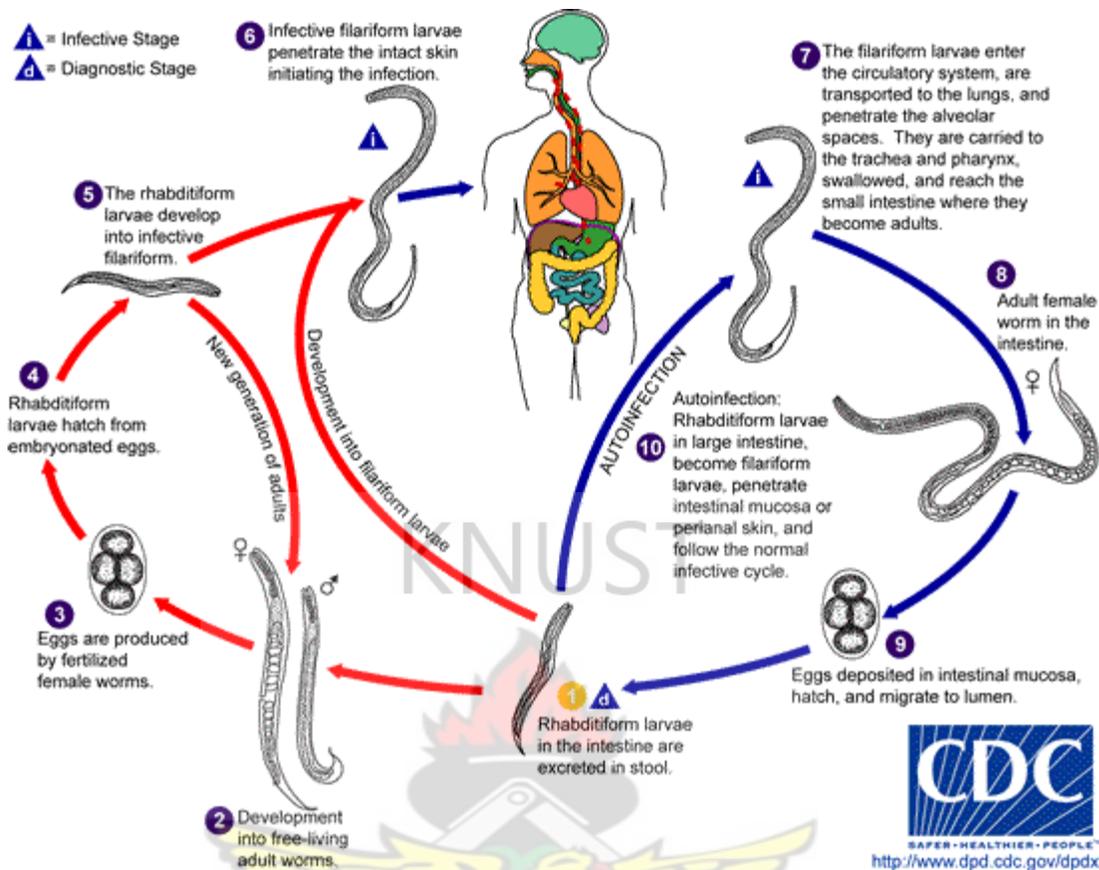


Figure 4: Life cycle of *Strongyloides stercoralis*.

#### 2.3.4.2 Life cycle of *strongyloides stercoralis*.

The *Strongyloides* life cycle is more complex than that of most nematodes with its alternation between free-living and parasitic cycles, and its potential for autoinfection and multiplication within the host. Two types of cycles exist:

**Free-living cycle:** The rhabditiform larvae passed in the stool [1](see "Parasitic cycle" below) can either molt twice and become infective filariform larvae (direct development) [6] or molt four times and become free living adult males and females [2] that mate and produce eggs [3] from which rhabditiform larvae hatch [4]. The latter in turn can either develop [5] into a new generation of free-living adults (as represented in

[2], or into infective filariform larvae [6]. The filariform larvae penetrate the human host skin to initiate the parasitic cycle [6].

**Parasitic cycle:** Filariform larvae in contaminated soil penetrate the human skin [6], and are transported to the lungs where they penetrate the alveolar spaces; they are carried through the bronchial tree to the pharynx, are swallowed and then reach the small intestine [7]. In the small intestine they molt twice and become adult female worms [8]. The females live threaded in the epithelium of the small intestine and by parthenogenesis produce eggs [9], which yield rhabditiform larvae. The rhabditiform larvae can either be passed in the stool [1] (see "Free-living cycle" above), or can cause autoinfection [10]. In autoinfection, the rhabditiform larvae become infective filariform larvae, which can penetrate either the intestinal mucosa (internal autoinfection) or the skin of the perianal area (external autoinfection); in either case, the filariform larvae may follow the previously described route, being carried successively to the lungs, the bronchial tree, the pharynx, and the small intestine where they mature into adults; or they may disseminate widely in the body. To date, occurrence of autoinfection in humans with helminthic infections is recognized only in *Strongyloides stercoralis* and *Capillaria philippinensis* infections. In the case of *Strongyloides*, autoinfection may explain the possibility of persistent infections for many years in persons who have not been in an endemic area and of hyper infections in immunodepressed individuals.

### 2.3.4.3 Diagnosis

There is no one ideal screening or diagnostic test, making strongyloidiasis a difficult infection to detect in humans (Gill *et al.*, 2004). The most important laboratory finding seen in patients with strongyloidiasis is eosinophilia (Gill *et al.*, 2004). Eosinophilia is shown to be 93.5% sensitive with a specificity of 93.1% in high risk populations (Roman-Sanchez *et al.*, 2003). But it is also shown that the eosinophil count if used alone is not sufficiently sensitive to screen for strongyloidiasis (Sudarshi *et al.*, 2003) especially in patients with chronic infection who can have low or normal eosinophil counts (Gill *et al.*, 2004), in people returning from developing countries (Schonning *et al.*, 2002), and in some cases of hyperinfection syndrome and disseminated strongyloidiasis. However, increased peripheral eosinophilia in the case of hyperinfection syndrome could be considered as a good prognostic factor (Keiser and Nutman, 2004). Eosinophilia is not a cost effective strategy compared to the stool examinations with agar plate culture method and serological testing in detecting strongyloides infection in humans (Libman *et al.*, 1993). The diagnosis of strongyloidiasis can also be made very reliably by observing strongyloid larvae in stool or sputum specimens. But these tests are not accurate as there is fluctuation in the rate of larval excretion especially in stools (Uparanukraw *et al.*, 1999) decreasing the efficacy and accuracy of these tests (Dreyer *et al.*, 1996). Repeated multiple stool specimens should be analyzed to increase the efficacy of the test in the presence of strong suspicion of strongyloidiasis (Sato *et al.*, 1995). Sudharshi and colleagues in their study have noted stool examinations by formalin-ether concentration method for larvae to be less sensitive in detecting the disease when used alone, especially in non-

endemic regions (Sudarshi *et al.*, 2003). Microscopic examination of stool specimens is done for strongyloid larvae. This can be done in the following different ways:

- 1) Simple direct smear with a sensitivity of 0–52% (Uparanukraw *et al.*, 1999),
- 2) Formalin-ether concentration method with a sensitivity of 13–55% (Uparanukraw *et al.*, 1999),
- 3) Harada-Mori filter paper culture method with almost equal sensitivity to the above 2 methods (Marchi and Cantons, 2003), and
- 4) Agar plate culture technique with higher sensitivity of 78–100% (Uparanukraw *et al.*, 1999).

#### **2.4 Platyhelminths**

Platyhelminths (flatworms) lack a true body cavity and are characteristically flat in dorsoventral section. All medically important species belong to the class Cestoda (tapeworms) and Trematoda (flukes) [Cheesbrough, 2006, Jawetz *et al.*, 1980]. Schistosome belongs to class trematoda and are hermaphrodite and produce eggs that are operculated. To develop the eggs must reach water. Snails serve as the intermediate host (Cheesbrough, 2006). Adult schistosoma species live in the blood venules around the gut or bladder.

### **2.4.1 Schistosomiasis**

Schistosomiasis is caused by infection with *Schistosoma haematobium*, *S. mansoni*, *S. japonicum*, *S. mekongi*, or *S. intercalatum*. The various species are associated with different clinical complications.

### **2.4.2 Epidemiology**

Schistosomiasis is endemic in 74 countries with a bulk of the global cases (90%) residing in sub-Saharan Africa (Steinmann *et al.*, 2006, WHO, 2002). In 2007, the World Health Organization estimated 235 million cases of schistosomiasis world-wide, with 732 million people at risk of infection in known transmission areas (WHO, 2009). In 2000, it was estimated that 70 million people had hematuria, 32 million had dysuria associated with *Schistosoma haematobium*, 18 million had major bladder wall pathology, 10 million people had *S. haematobium* related renal failure; and schistosomiasis related bladder cancer, resulting in an estimated mortality of 150 000 people per year in sub-Saharan Africa (Van der Werf *et al.*, 2003).

### **2.4.3 Pathology**

Schistosome eggs, which may become lodged within the hosts tissues, are the major cause of pathology in schistosomiasis. Some of the deposited eggs reach the outside environment by passing through the wall of the intestine; the rest are swept into the circulation and are filtered out in the periportal tracts of the liver resulting in periportal fibrosis. Onset of egg laying in humans is sometimes associated with an onset of fever (Katayama fever). This "acute schistosomiasis" is not, however, as important as the chronic forms of the disease. For *S. mansoni* and *S. japonicum* these are "intestinal" and

"hepatic schistosomiasis", associated with formation of granulomas around trapped eggs lodged in the intestinal wall or in the liver, respectively. The hepatic form of the disease is the most important, granulomas here giving rise to fibrosis of the liver and hepatosplenomegaly in severe cases. Symptoms and signs depend on the number and location of eggs trapped in the tissues. Initially, the inflammatory reaction is readily reversible. In the latter stages of the disease, the pathology is associated with collagen deposition and fibrosis resulting in organ damage that may be only partially reversible.

Granuloma formation is initiated by antigens secreted by the miracidium through microscopic pores within the rigid egg shell, and there is strong evidence that the vigorous granulomatous response, rather than the direct action of parasite egg antigens, is responsible for the pathologic tissue manifestations in schistosomiasis. The granulomas formed around the eggs impair blood flow in the liver and consequently induce portal hypertension. With time, collateral circulation is formed and the eggs disseminate into the lungs, where they cause more granulomas, pulmonary arteritis and, later, cor pulmonale. A contributory factor to portal hypertension is Symmers' fibrosis, which develops around branches of the portal veins. This fibrosis occur only many years after the infection and apparently is caused in part by soluble egg antigens and various immune cells which react to them.

#### **2.4.4 Transmission of schistosomiasis**

It's mainly transmitted through contact with water infested with intermediate host snail cercariae including through activities such as bathing, washing laundry, and fetching water (Jordan and Webbe, 1993). In Ghana man is the only known definitive host of any

importance and therefore his habits in the use of water are of the utmost significance in the epidemiology of schistosomiasis (Odei, 1983).

#### 2.4.5 Life Cycle of Schistosoma

Eggs are eliminated with feces or urine [1]. Under optimal conditions the eggs hatch and release miracidia [2], which swim and penetrate specific snail intermediate hosts [3]. The stages in the snail include 2 generations of sporocysts [4] and the production of cercariae [5]. Upon release from the snail, the infective cercariae swim, penetrate the skin of the human host [6], and shed their forked tail, becoming schistosomulae [7]. The schistosomulae migrate through several tissues and stages to their residence in the veins [8, 9]. Adult worms in humans reside in the mesenteric venules in various locations, which at times seem to be specific for each species [10]. For instance, *S. japonicum* is more frequently found in the superior mesenteric veins draining the small intestine A, and *S. mansoni* occurs more often in the superior mesenteric veins draining the large intestine B. However, both species can occupy either location, and they are capable of moving between sites, so it is not possible to state unequivocally that one species only occurs in one location. *S. haematobium* most often occurs in the venous plexus of bladder C, but it can also be found in the rectal venules. The females (size 7 to 20 mm; males slightly smaller) deposit eggs in the small venules of the portal and perivesical systems. The eggs are moved progressively toward the lumen of the intestine (*S. mansoni* and *S. japonicum*) and of the bladder and ureters (*S. haematobium*), and are eliminated with feces or urine, respectively [1]. Pathology of *S. mansoni* and *S. japonicum* schistosomiasis includes: Katayama fever, hepatic perisinusoidal egg granulomas, Symmers' pipe stem periportal fibrosis, portal hypertension, and

occasional embolic egg granulomas in brain or spinal cord. Pathology of *S. haematobium* schistosomiasis includes: hematuria, scarring, calcification, squamous cell carcinoma, and occasional embolic egg granulomas in brain or spinal cord.

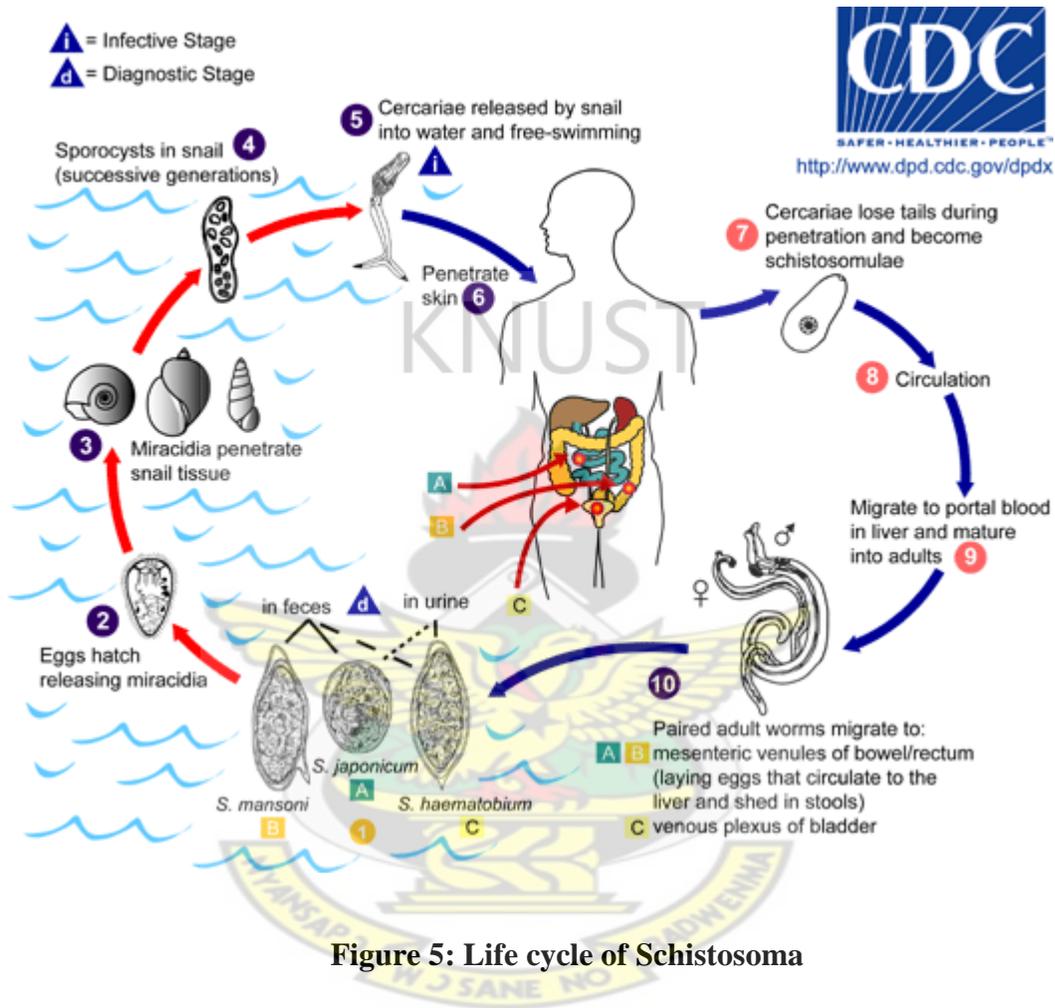


Figure 5: Life cycle of Schistosoma

#### 2.4.6 Diagnosis

Diagnosis of schistosomiasis infection in laboratory is mainly done by confirming the presence of eggs in urine (*S. haematobium*) and in stool (*S. mansoni* and *S. japonicum*). The eggs of *Schistosoma japonicum* are large and more rounded than other species, measuring 70-100  $\mu\text{m}$  long by 55-64  $\mu\text{m}$  wide. The spine on *S. japonicum* eggs is smaller and less conspicuous than other species. Eggs are shed in stool. *Schistosoma*

*mansoni* eggs are large (114 to 180  $\mu\text{m}$  long by 45-70  $\mu\text{m}$  wide) and have a characteristic shape, with a prominent lateral spine near the posterior end. The anterior end is tapered and slightly curved. The eggs of *Schistosoma haematobium* are large (110-170  $\mu\text{m}$  long by 40-70  $\mu\text{m}$  wide) and bear a conspicuous terminal spine.

## **2.5 Burden of schistosomiasis and STHs**

The economic effects and health implications of schistosomiasis and STHs are extensive. Schistosomiasis is second to malaria only in terms of socio-economic and public health importance in subtropical and tropical countries including Ghana (Klumpp, 1983). Chan (1997) described a model in which the worm burdens were separated into two threshold levels based on the disabilities experienced by the human hosts. The model facilitated calculation of the disability-adjusted life years (DALYs), which translates disabilities experienced into years of healthy life lost. As calculated by Chan (1997), the DALYs lost due to *Ascaris* are 10.5 million, while the combined DALYs for the STHs, (*Ascaris lumbricoides*, *Trichuris trichiura* and hookworms) are 39.0 million, which is higher than the DALYs estimated for malaria (35.7 million). Again it has also been established there is also association between helminths infection and a reduction in host cognitive abilities (Kvalsvig *et al.*, 2002).

## **2.6 Prevalence of Schistosomiasis and STHs**

Over 650 million people globally are at risk of infection, with more than 200 million people infected. Of these, 120 million are estimated to have symptoms, with 20 million people experiencing serious consequences (Gryseels *et al.*, 2006). Higher disease rates

occur in children with infection frequently found in those less than 14 years in many risk areas (WHO Fact sheet No 115).

Recent prevalence data suggest that approximately 1.3 billion people are infected (Dold and Holland, 2010). Prevalence of *S. haematobium* mono-infection was 14.4%, hookworm mono-infection was 3.2%, and *S. haematobium* and hookworm co-infection was 0.7% from a study to map helminth co infection and co intensity in Ghana (Soares *et al.*, 2011).

Ghana is highly endemic for the disease (Okoh, 1994) but most of the schistosomiasis studies are carried out in communities along the Volta Lake. This is because of the construction of the Akosombo dam which serves as breeding grounds for the intermediate host. In a study from the Ashanti region, the central tropical forest of Ghana, two adjacent villages endemic for the disease showed only urinary schistosomiasis. Kyereyase about 1200 inhabitants and prevalence more than 70% and Nyamebikyere about 300 inhabitants and prevalence more than 65% (Klumpp and Chu, 1977).

## **2.7 Helminths and Pregnancy**

Children and pregnant women are particularly vulnerable to STH which decreases work capacity (Montresor, 1998). Many women in developing countries spend most of their reproductive years either pregnant or lactating. Helminths infection during pregnancy could compromise maternal-perinatal health and nutritional status. (Weigel *et al.*, 1996). Pregnant women frequently suffer from soil transmitted helminths (*Ascaris lumbricoides*, *Necator americanus* and *Trichuris trichiura* ) and cause high morbidity

by leading to malnutrition, iron-deficiency anaemia, malabsorption, intestinal obstruction, mental and physical growth retardation in children (Allen and Maizels, 1996; Bethony *et al.*, 2006). *Schistosoma haematobium* causes substantial morbidity in affected populations and the considerable burden of genito-urinary pathology in women has recently been acknowledged (Ramarakoto *et al.*, 2008; Kjetland *et al.*, 2008). Intestinal Helminth infections and schistosomiasis are highly prevalent in Ghana (Okoh, 1994) but despite their potential clinical interaction, the burden of such co-infections has not yet been evaluated in pregnant women.

## **2.8 Pregnancy and Haemoglobin Level**

It has been established by studies that haemoglobin and haematocrit concentrations typically decrease during the first trimester, reach the lowest levels at the end of the second trimester, and increase again during the third trimester of pregnancy (CDC, 1989). Iron is a major component of blood. Each pregnancy requires a transfer from mother to fetus of about 300 mg of iron during the third trimester, the mother requires an additional 500 mg of iron to cope with the increased red blood cell mass needed for a successful pregnancy and each day of lactation involves a transfer of about 0.75 mg of iron from mother to child (WHO, 1994). Anaemia during pregnancy is associated with premature delivery, low birth weight, maternal ill health and maternal death (Seshadri, 1997). Blood loss caused by hookworm infections will put mother, fetus and child at risk of iron deficiency leading to anaemia. The extent to which this occurs will depend on host iron status, the infecting species and the intensity and duration of infection. In developing countries, the iron status of women at conception is frequently poor as a result of inadequate dietary iron intake, concurrent infections and frequent closely

spaced pregnancies (King, 2003). Anaemia is a qualitative or quantitative deficiency of Hb or red blood cells (RBC) in circulation resulting in reduced oxygen (O<sub>2</sub>)-carrying capacity of the blood to organs and tissues. Anaemia in pregnancy is defined as an Hb concentration of less than 11g/dl or a haematocrit less than 0.33 in first and third trimesters, while in the second trimester a fall of 0.5g/dl is adjusted for an increase in plasma volume and a value of 10.5g/dl is used (Sharma, 2003). Anaemia in pregnancy can also be classified as mild, moderate or severe, with WHO classifying mild anaemia as Hb level of 10.0-10.9g/dl, moderate anaemia as 7-9.9g/dl and less than 7g/dl as severe anaemia (Idowu *et al.*, 2005). The concentration of plasma adrenomedullin, a potent vasodilating peptide, rises during pregnancy and correlates significantly with blood volume (Hayashi *et al.*, 2005). RBC volume decreases during the first 8 weeks, increases to the pre-pregnancy level by 16 weeks and undergoes a further rise to 30% above the pre-pregnancy volume at term (Chestnut *et al.*, 2009). Elevated erythropoietin concentration and the erythropoietin effects of progesterone, prolactin and placental lactogen result in an increase in RBC volume (Chestnut *et al.*; 2009). Hence the plasma volume expansion increase exceeds the rise in RBC volume, resulting in haemodilution and consequent physiological anaemia of pregnancy (Chestnut *et al.*, 2009).

## 2.9 Dewormers and Helminths

Helminth infections during pregnancy may be associated with adverse outcomes, including maternal anaemia, low birth weight and perinatal mortality. A single dose oral anthelmintic treatment can be given to pregnant and lactating women but as a general rule drugs should not be given in the first trimester of pregnancy (WHO, 1994). Deworming during pregnancy has therefore been strongly advocated, but its benefits have not been rigorously evaluated. Studies involving the provision of anthelmintic treatment and subsequent observation of intensity of re-infection have shown that individuals tend to re-acquire similar worm burdens to those harboured before treatment. This phenomenon which is termed predisposition has been demonstrated in longitudinal studies for in humans (Elkins, 1986) and can be detected over multiple rounds of chemotherapy (Holland, 1989). Since STH infections are coedemic, deworming programmes are targeted at all the three helminths infections. World Health Organization in 2002, endorsed the combine approach of integrated control of both Schistomiasis and STHs. Anthelmintic drug treatment programmes are aimed to reduce morbidity as opposed to eradicate helminths, which is not feasible goal (WHO, 2002). Regular systematic treatment is necessary due to high re infection rates in endemic regions. Universal drug administration is recommended for communities where STH prevalence exceeds 20% and those found to have prevalence in excess of 50% are considered high risk (WHO, 2006b).

## 2.10 Intensity of Schistosomiasis and STHs infections.

The thresholds proposed for use by a WHO Expert Committee in 1987 for the classes of intensity for each helminth are the following;

Parasite	Light Intensity Infection(epg)	Moderate Intensity Infections(epg)	Heavy Intensity Infections(epg)
<i>A.lumbricoides</i>	1-4999	5000-49999	50000
<i>Trichuris trichiura</i>	1-999	1000-9999	10000
Hookworm	1-1999	2000-3999	4000
<i>S.mansoni</i>	1-99	100-399	400
<i>S.japonicum</i>	1-99	100-399	400
<i>S.haematobium</i>	Less than 50eggs/10ml		50eggs/10ml

Intensity of schistosoma and STHs are associated with blood loss. Light intensity infections are related to a loss of less than 2mg of haemoglobin per gram of faeces while heavy intensity infections corresponds to a loss of more than 5mg of haemoglobin per gram of faeces (Stoltzfus *et al.*, 1997). Much focus has been placed on investigating whether variation in infection intensity is a result of differences in environmental exposure to infection or susceptibility. Determinants of infection intensity may be divided into two categories-long term effects that operate on the time scale of the host life expectancy(host genetics ,host socio economic status) and short term effects that operate on the time scale of the parasite life expectancy(host acquired immune response). In attempt to estimate the relative importance of long and short term

effects of infection intensity, McCallum (1990) used probability theory and concluded that both categories have an approximately equal contribution to the observed heterogeneity. Differential exposure to infection in humans is difficult to quantify as there are many factors to consider (O’Lorcain and Holland, 2000; Bundy and Blumenthal, 1990). Results suggested that intensity of *A. lumbricoides* infection is influenced by gender-related behavioural and environmental factors that contribute to exposure (Knightlinger *et al.*, 1998).

Adults are also known to harbour *A. lumbricoides* worms but generally at a lower intensity than children (Thein-Hlaing, 1984). This has led to the suggestion that less marked aggregation in older age cohorts reflects a slow build up of specific immunity or variation in susceptibility to infection over time. However, over dispersed worm frequency distributions are also recorded within age classes as age is not the only source of variation. Coupled with this, as Bundy (1988) discussed, hosts with the greatest prior experience of infection are subsequently re infected, indicating that immunity cannot be the only primary determinant of variability in infection intensity. Morbidity and mortality increases with worm burden (Pawłowski and Davis, 1989) and those who harbour light infections tend to be asymptomatic. Aggregation leads to relatively few individuals harbouring sufficient worms to precipitate life threatening or severe morbidity (Anderson and May, 1995).

## CHAPTER THREE

### 3.0 Materials and Methods

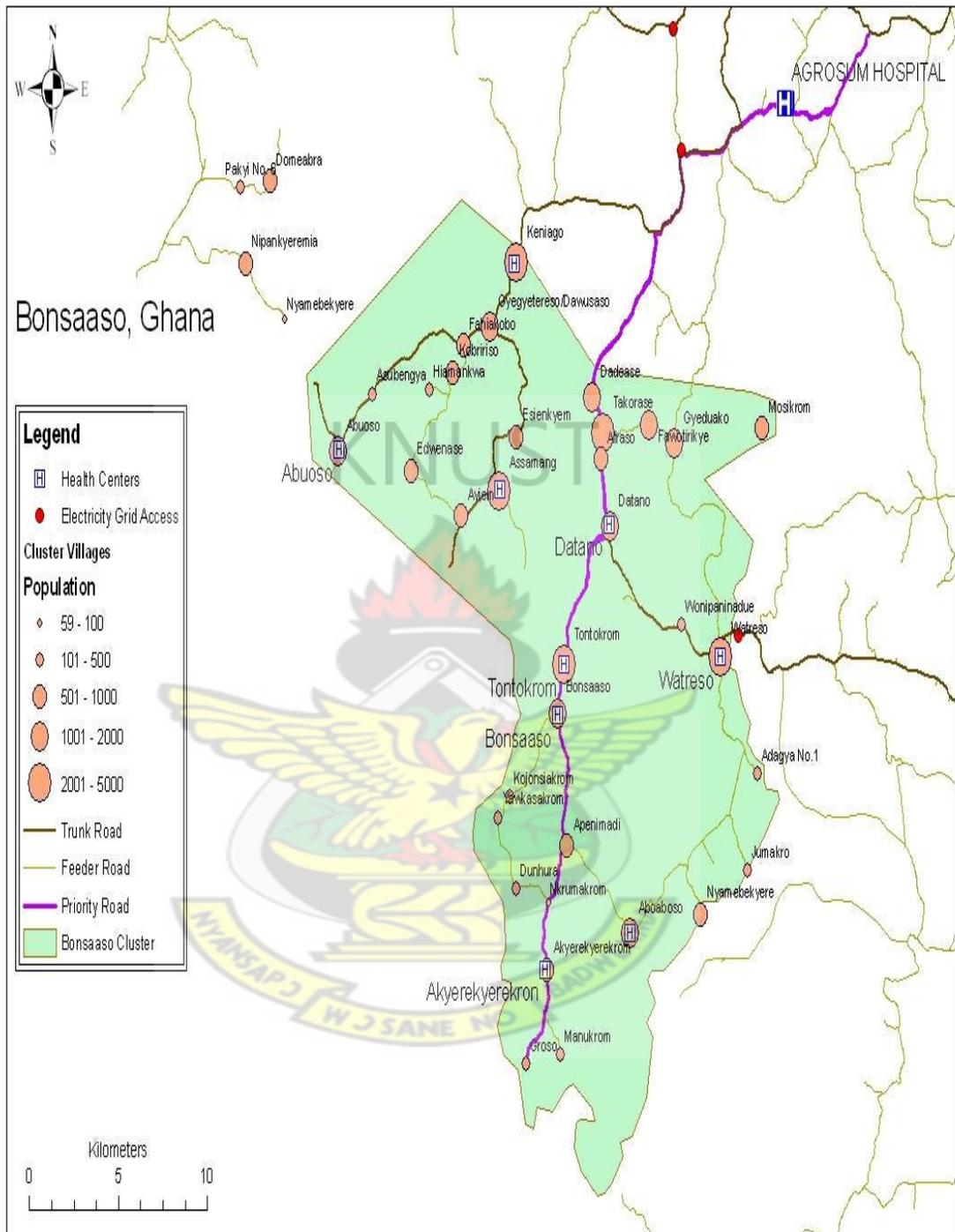
#### 3.1 Study Area

This study was conducted in Bonsaaso Cluster within the Amansie West District of the Ashanti Region of Ghana. The area is characterized by hot, humid and tropical climate conditions. The Villages in this cluster are spread out and separated from one another by thick rainforest and diverse vegetation.

The land area is 389 km<sup>2</sup> (29% of the total land area of Amansie West District). It lies between Latitudes 6° 5' and 6° 15' North and Longitudes 1° 7' and 2° 2' West.

This is the area that has been adopted by the Millennium Villages Project (the implementers of the Millennium Development Goals MDGs) in Ghana called the Bonsaaso Cluster. The project area covers about 30 contiguous rural communities with estimated population of 31000 (Ghana Statistical Service, 2010). Farming is the primary occupation of the inhabitants but almost all the youth are engaged in illegal small scale gold mining (galamsey).

Like most of the rural communities in Ghana, Bonsaaso lacks good drinking water and sanitation is poor. Although almost all the communities have public latrines, open defecation is rampant. There are seven clinics within the thirty one communities of the Millennium Villages Project and one central medical laboratory. The farthest community from a clinic is about six kilometers.



Source: Millennium Villages Project

Figure 6: MAP OF BONSAASO CLUSTER IN AMANSIE WEST DISTRICT OF ASHANTI REGION.

### **3.2 Study Population**

This study was conducted among pregnant women from any of the thirty one communities attending antenatal clinic in one of the seven clinics within the Bonsaaso cluster between December 2011 and June 2012. Informed consent was obtained and a total of 186 women took part in the study. The pregnancy of those who consented after the purpose of the project had been explained was confirmed by HCG using urine samples.

### **3.3 Sample Collection**

#### **3.3.1 Urine**

Ten to fifteen ml of urine specimen were obtained from pregnant women involved in the study in clean container and labeled. Each urine specimen was collected between 11.00 and 14.00 hours, the time of optimum egg passage (Cheesbrough, 2006). Urine specimen were kept cool in an insulated ice box and processed in the laboratory within four hours of passing.

#### **3.3.2 Blood**

Four ml of venous blood samples were collected directly into Vacutainer tubes (Becton Dickinson) containing potassium EDTA as anticoagulant after the collection site is cleaned with isopropyl alcohol. To avoid hemoconcentration, a tourniquet was placed on the arm at least 7 cm above the venipuncture site and left in place for less than 1 minute. The Vacutainer tube was inverted to ensure adequate mixture with the anticoagulant. All analyses were conducted within 60 minutes after sample collection.

### **3.3.3 Stool**

About 4-8 g fresh stool specimen was collected into clean, dry and leak proof container. It was then examined macroscopically for consistency. Two methods, the wet mount and the formol-saline concentration were used. If the number of parasites in the stool specimens is low, examination of a direct wet mount may not detect them, hence the stool should be concentrated. Eggs, cysts and larvae are recovered after concentration procedures whereas trophozoites get destroyed during the procedure. This makes direct wet mount examination obligatory as the initial phase of microscopic examination. (Rayapu *et al*, 2012)

### **3.4 Laboratory Analysis**

Parasitological diagnosis of soil transmitted helminths and schistosomiasis is made by analyzing stool and urine samples for presence of eggs.

#### **3.4.1 Stool**

##### **3.4.1.1 The wet-mount method**

This method was used to detect the presence of motile parasites such as larvae of *S. stercoralis* and trophozoites (Cheesbrough, 2006). A gram of the stool specimen was emulsified with 3-4mls of physiological saline depend on the consistency of the sample. A drop of the emulsified sample was placed on a clean grease free slide and gently covered with a cover slip. The slide was examined under microscope using 10x objective lens, then 40x for detailed identification of parasite under low light intensity.

##### **3.4.1.2 The formol-ether concentration method**

Even in moderate or severe schistosoma infection, only a few eggs are produced in the stool and therefore the concentration method becomes necessary (Cheesbrough, 2006).

Again the parasites need to be concentrated for quantification. A gram of stool sample was emulsified with 4 ml of 10% formol saline. An additional 3 ml of 10% formol saline was added and this was mixed thoroughly. The emulsified sample was sieve through a mesh size of 400µg and the filtrate collected in a beaker. The filtrate was then transfer into a centrifuge tube and 3 ml of diethyl ether added. The content was mixed by inverting and intermittent shaking for and centrifuged at 3,000 revolutions per minute for one minute. After centrifugation, the supernatant (layers of ether, debris, and formol saline) was discarded and the sediment (containing the parasites at the bottom of the test tube) was re-suspended in formol saline. With the aid of Pasteur pipette, sediment was placed on a clean grease free slide and covered with a cover slip. It was examined under microscope using under 10x and 40x magnification. The results were classified as negative or positive if schistosoma were found to be either absent or present respectively. Positive specimens were ranked by the level of infection as light, mild and severe. To ensure accurate identification of parasite species, bench aids for the diagnosis of intestinal parasites (Ash, 1994) and diagrams of various parasite ova and larvae from parasitological text were reviewed.

### **3.4.2 Urine**

Haematuria is an important sign of urinary schistosomiasis in endemic areas (Montresor *et al.*, 2002). Haematuria (presence of blood in urine) was detected by the use of a standard hemastix, with any positive reaction being designated positive for urinary schistosomiasis (Multistix; Bayer Diagnostics, Leverkusen, Germany).The reagent strip was dipped into the urine sample and waited for a little less than one minute and the reagent strip compared with the colorimetric scale. 10 ml of well mixed urine sample

was then centrifuged at 290 g for five minutes to sediment the eggs and the supernatant discarded. The sediment was re-suspended and a clean Pasteur pipette used to transfer all the sediment to a clean slide and cover with a cover glass. The entire slide is examined under microscope by using the x10 objective for schistosoma eggs. The intensity of schistosoma infection was determined by parasite egg count and reported as parasite egg count per 10ml of urine.

### **3.4.3 Haemoglobin level (Hb)**

The haemoglobin level was determined using haematology auto analyzer according to the manufactures manual. The haematology analyzer was calibrated with commercially available standard haemoglobin (SYSMEX EIGHTCHECK-3WP-H, Japan).

### **3.5 Data Collection**

The relevant data was obtained from participants medical records and through the use of a pretested questionnaire. The variables studied were demographic factors such as age, gestation period, gravid, occupation and dewormer use.

### **3.6 Quality Control**

1. To ensure quality control, all the laboratory procedures including collection and handling of specimens were carried out in accordance with standard protocols.
2. All negative slides were re-checked for confirmation by supervising clinical parasitologist to ensure consistency.
3. The microscope for this study was calibrated and the objectives and oculars used for the calibration. The calibration features for the 10x and 40x objectives were posted on the microscope for easy access.

### 3.7 Statistical Analysis

Analysis was done using STATA (Version 11.0) software. Descriptive statistics included frequencies and means (standard deviation). Frequencies of demographic variables were also presented in subgroups (occupation and age, gestation and gravidity). Association of haemoglobin levels with haematuria as well as schistosomiasis, dewormer use with parasite infection and schistosomiasis and haematuria were studied using Pearson's correlation. The level of significance was set at  $P \leq 0.05$ .



## CHAPTER FOUR

### 4.0 RESULTS

#### 4.1 Demographic Characteristics

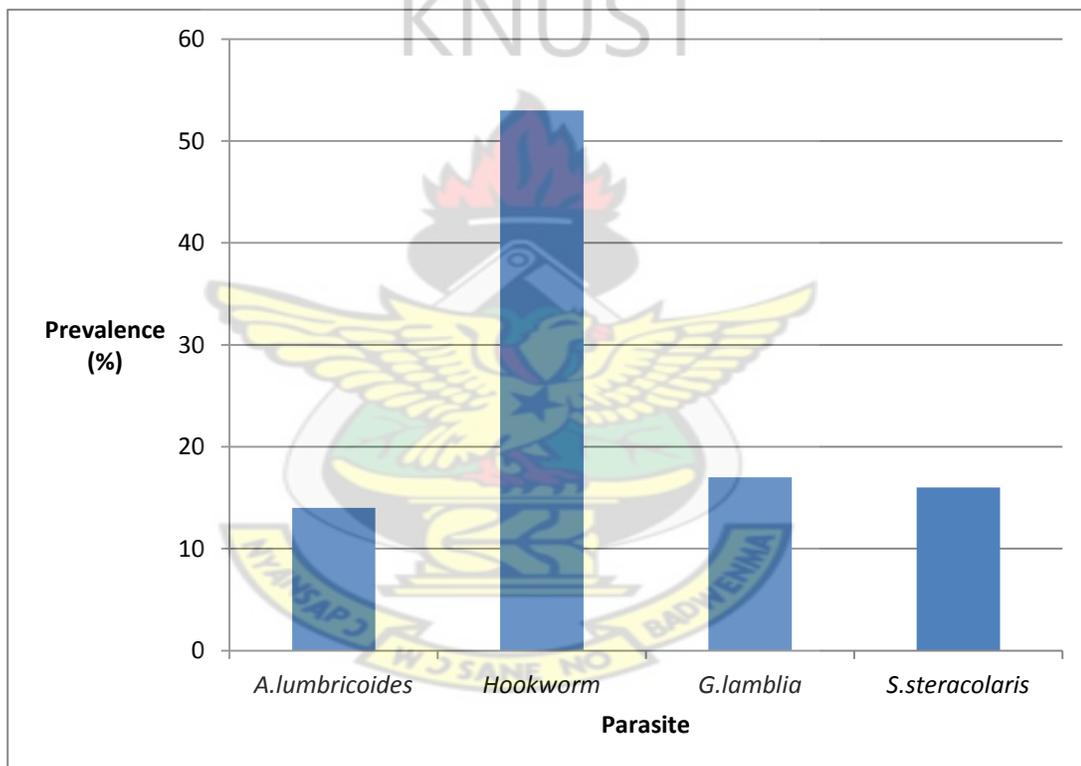
A total of one hundred and eighty six (186) participants took part in the study and consisted of pregnant women who were in their various stages of pregnancy and attending antenatal clinics in all one of the seven clinics within the Bonsaaso cluster between December 2011 to June 2012. The age of the study subject ranges from 15 years to 41 years with a mean age of 26 years. Majority (31%) of the pregnant women were between the ages of 20-24 years [Table 4.1]. Fifty eight percent of the women had between 2-5 children (multigravida) followed by those with a child (29%), with those with more than 6 children (grandmultigravida) being 13% [Table 4.1]. Fifty percent (50%) of the pregnant women were in their second trimester being the majority while 41% were in their third trimester with just 9% in their first trimester. Cumulatively, approximately 91% were in their second and third trimesters. Most of the pregnant women (46%) were farmers followed by the unemployed (23%) with professionals (1%) being the least. Small-scale miners (galamsey) were (8%), semi-skilled workers were 22% [Table 4.1].

**Table 4.1: Demographic Characteristics of study subjects.**

<b>Demographic Characteristics</b>	<b>(Total Number =186)</b>	<b>Percentage (%)</b>
	<b>N</b>	
<b>Age group(years)</b>		
15-19	32	17.20
20-24	57	30.65
25-29	40	21.51
30-34	35	18.82
35-39	20	10.75
40-44	2	1.08
<b>Gestation(Trimester)</b>		
First (less than 12 wks)	16	8.60
Second (12-28wks)	93	50.00
Third (more than 28 wks)	77	41.40
<b>Gravidity</b>		
Primigravida (1)	54	29.03
Multigravida (2-5)	108	58.06
Grandmultigravida (≥6)	24	12.90
<b>Occupation</b>		
Farming	85	45.70
Small scale Mining (Galamsey)	15	8.06
Semi-Skilled worker	41	22.04
Professional	2	1.08
Unemployed	43	23.19

## 4.2 General Prevalence of Intestinal Parasites in Pregnant Women

In all the stools, four intestinal parasites were found namely, *Ascaris lumbricoides*, Hookworm, *Giardia lamblia* and *Strongyloides stercoralis*. The overall prevalence of Soil Transmitted Helminths (STHs) was 67%. Hookworm infection had the highest (53%) prevalence among the pregnant women who took part in the study. *Ascaris lumbricoides* accounted for 14%, *Giardia lamblia* 17% and *Strongyloides stercoralis* 16% [Fig: 7]

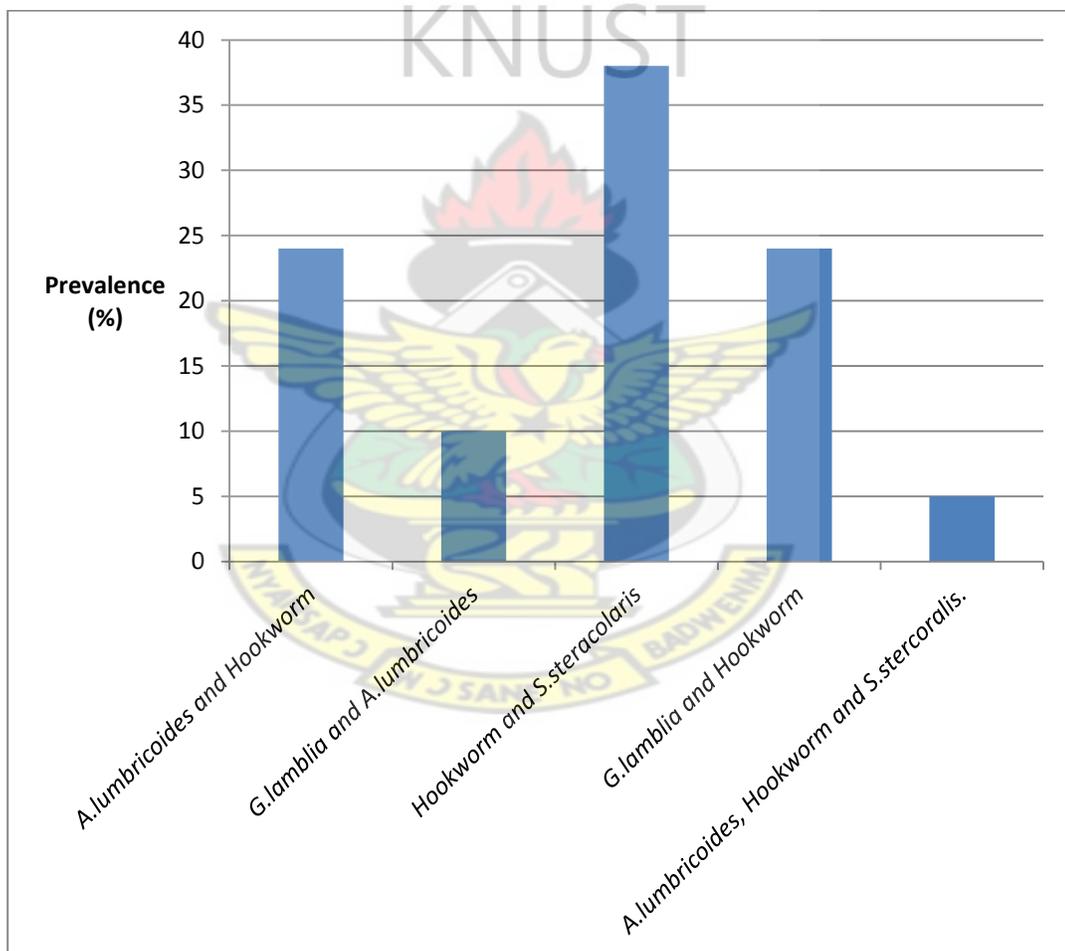


**Figure 7: General Prevalence of Parasite infection**

### 4.3 Prevalence of co infection.

Co-infection of *A. lumbricoides* with Hookworm was 23% and Hookworm with *S. stercoralis* 38%. Out of the one hundred and eighty six participants, only one percent of the study participants was infected with three parasites and represents 5%.

Though *Giardia lamblia* is not STH, 8% of the participants were infected. It was also co infected with other STHs, *A. lumbricoides* 10% and Hookworm 24%.



**Figure 8: Prevalence of co-infection.**

#### 4.4 Intensity of Soil Transmitted Helminths (*A. lumbricoides* and Hookworm)

Intensity of the two parasites (*A. lumbricoides* and Hookworm) that are soil transmitted was mainly of light infection. In the case of *Ascaris lumbricoides* ninety four percent of the total number had light infection with only one moderate infection. None of them was heavily infected. Hookworm ranges from light, moderate and heavy. Eighty four percent had light infection. [Table 4.2].

**Table 4.2: Intensity of Parasitic Infection among the pregnant women.**

<i>Ascaris lumbricoides</i>	Frequency	Percentage (%)
Light infection (1-4999) epg	16	94
Moderate infection (5000-49999) epg	1	6
Heavy infection (50000) epg	0	0
<b>Hookworm</b>		
Light infection (1-999) epg	48	84
Moderate infection (1000-3999) epg	7	12
Heavy infection (4000) epg	2	4

#### 4.5 Distribution of parasite type by gestation

Of all the participants who took part in the study, majority (47%) of those who were infected with different species of STHs were in the second trimester. This was followed closely by those who were in their third trimester (42%). Those who were least infected (11%) were in their first trimester. Of all the cases of infection, Hookworm was predominant. [Table 4.3]

**Table 4.3: Distribution of parasite type by gestation**

	First trimester (n=16)	Second trimester (n=93)	Third trimester (n=77)
<i>G.lamblia</i>	0	2	12
Hookworm	6	22	16
<i>Strongyloides stercoralis</i>	1	6	3
<i>Ascaris</i> and Hookworm	0	4	1
<i>Ascaris lumbricoides</i>	1	7	1
<i>S.stercoralis</i> and Hookworm	3	3	2
<i>A.lumbricoides</i> and <i>G.lamblia</i>	0	1	1
<i>G.lamblia</i> and Hookworm	0	0	5
<i>A.lumbricoides</i> , <i>S.stercoralis</i> and Hookworm	0	1	0
No parasite	11	45	32

#### 4.6 Urinary Schistosomiasis among the participants.

Out of the one hundred and eighty six pregnant women, haematuria was detected in twenty four of them and this represents thirteen percent (13%). Eleven percent (11%) of all the urine samples examined had *S. haematobium* eggs while in the majority (89%) of the urine samples there was no *S. haematobium* egg. [Table 4.4]

Out of the twenty four (24) pregnant women who had blood in their urine, twenty one (21) had *S. haematobium* and this represents 87%. [Table 4.4]

**Table 4. 4: Dipstick and Microscopy examination of urine examination.**

Urine Examination	(Total=186) N	Percentage (%)
<b>Haematuria (Dipstick)</b>		
Positive	24	13
Negative	162	87
<b>Microscopy for Schistosoma Ova</b>		
Positive	21	11
Negative	165	89

From Table 4.5, a total of twenty four had blood in their urine, twenty one (87%) were infected with schistosoma. With a p value of zero ( $P \leq 0.001$ ), this means that there was a strong relation between haematuria and schistosoma (Appendix 2).

**Table4. 5: *Schistosoma haematobium***

	<b>Blood in urine</b>	<b>No blood in urine</b>
<b>Infected</b>	<b>21</b>	<b>0</b>
<b>Non infected</b>	<b>3</b>	<b>162</b>
<b>Total</b>	<b>24</b>	<b>162</b>
<b>Intensity</b>		
	<b>Frequency</b>	
<b>Light infection (less than 50eggs/10ml)</b>	<b>21</b>	
<b>Heavy infection(more than 50eggs/10ml)</b>	<b>0</b>	

#### **4.7 Intensity of schistosomiasis infection.**

There were twenty one of the pregnant women with schistosomiasis. All of them were light infection with egg count of less than 50 per 10ml of urine. The schistosomiasis infection among the pregnant women was of light intensity.

#### 4.8 Haemoglobin levels.

Most (45%) of the pregnant women had mild anaemia (7.0-9.9 g/dl) and only (1%) of the total pregnant women in the study had severe anaemia (< 7 g/dl). 35% had moderate anaemia (10.0-10.9)g/dl and 19% had normal haemoglobin level (> 11g/dl) [Table 4.7].

Out of the 186 respondents only one of them had haemoglobin level below 7g/dl (severe anaemia) and was in her second trimester. Majority (45%) of them had haemoglobin level between 7.0 and 9.9 g/dl (moderate anaemia). This was followed by 35% who had their haemoglobin level 10.0-10.9(moderate anaemia). The haemoglobin level of 19% of all the respondents was more than 11g/dl (normal).

**Table 4.6: Distribution of Haemoglobin levels among the Participants.**

Haemoglobin level(g/dl)	Frequency	Percentage	Cumulative Frequency
Less than 7	2	1.08	1.08
7.0-9.9	84	45.16	46.24
10.0-10.9	65	34.95	81.18
More than 11	35	18.82	100
Total	186	100	

From Fig. 4.8, majority (74.3%) with multigravida had Haemoglobin level more than 11, followed by 57.1% between 7-9.9g/dl and 52.3% between 10-10.9 with none less than 7. This shows that, multigravida had majority across the various haemoglobin levels with the exception of less than 7. Again, multigravida had more than half (58.06%) out of the total samples studied.

From Table 4.10, out of the total of 97 between the ages of 20-29, approximately 61% had haemoglobin level between 7-9.9g/dl, followed by 57.1% for more than 11 and 40% had haemoglobin level between 10-10.9g/dl with not less than 7. This means that, majority of the respondents between 20-29 years had haemoglobin level between 7-9.9g/dl. Also majority (43.19%) of the respondent between the ages of 30-39 years had haemoglobin level between 10-10.9g/dl.

**Table 4.7: Distribution haemoglobin Level with respect to Age, Gestation, Gravida.**

<b>Gestation(Trimester)</b>	Haemoglobin level(g/dl)			
	Less than 7	7.0-9.9	10.0-10.9	More than 11
First	0	9	4	3
Second	1	47	32	13
Third	0	32	27	18
<b>Age(Years)</b>				
15-19	2	18	11	1
20-24	0	35	14	8
25-29	0	16	12	12
30-34	0	7	19	9
35-39	0	7	9	4
40-44	0	1	0	1
<b>Gravidity</b>				
Primigravida	2	30	17	5
Multigravida	0	48	34	26
Grandmultigravida	0	6	14	4

#### 4.9 Blood in urine and Schistosoma.

From Table 4.8, of all the respondents who tested negative only two had haemoglobin level less than 7g/dl. Also, for haemoglobin level between 10-10.9, majority (89%) were tested negative while 10.8% were tested positive. Majority (87) of the respondent tested negative.

Again from Table 4.9, out of 165 respondents with no Schistosoma, only two of them had Haemoglobin level less than 7g/dl. 37% of them had their haemoglobin level between 10-10.9. Again, out of the 21 respondents with Schistosoma, 17.1% had haemoglobin level more than 11.

**Table 4.8: Distribution of Haemoglobin level to haematuria and Schistosoma.**

Urine Dipstick	Haemoglobin level(g/dl)			
	Less than 7	7.0-9.9	10.0-10.9	More than 11
Negative	2 (100%)	73 (87%)	58 (89%)	29 (83%)
Positive	0 (0)	11 (13%)	7 (11%)	6 (7%)
<b>Schistosoma</b>				
No Schistosoma	2 (100%)	73 (87%)	61 (94%)	29 (83%)
Schistosoma	0 (0)	11 (13%)	4 (6%)	6 (83%)

There was weak positive relation between haemoglobin level and schistosomiasis infection (P=0.096).

#### 4.10 Haemoglobin level with co infection of hookworm and schistosomiasis

A total of eight of the participants were infected with hookworm as well as *S. haematobium*. Both the *S. haematobium* and hookworm infections were light. (in the ranges of 1-999epg for hookworm and 50eggs/10ml of urine in the case of *S. haematobium*). From table 4.9, two of the participants who were infected with the two parasites had their haemoglobin levels above 11g/dl. Though none of them had haemoglobin level below 7g/dl, six of them representing almost 75% had their haemoglobin levels below the WHO recommended value of 11g/dl.

**Table 4. 9: Co infection of Hookworm and *S. haematobium* in relation to Hb.**

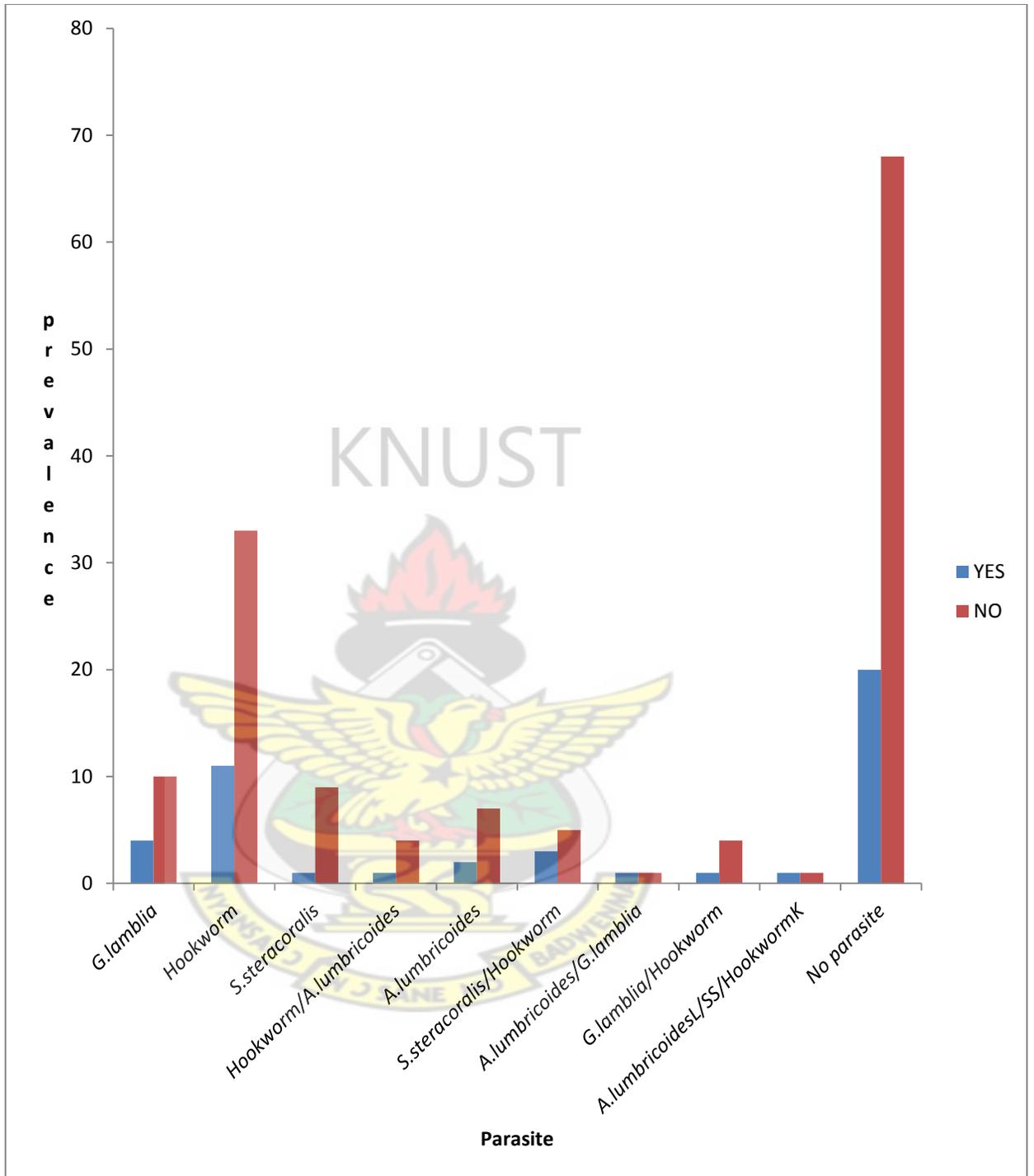
Haemoglobin level(g/dl)	Frequency	Hookworm (Epg)	<i>S.haematobium</i> (Eggs/10ml urine)
Less than 7	0	-	-
7.0-9.9	5	200-3050	2-9
10.0-10.9	1	0-700	0-2
More than 11	2	1250-1400	4-10
Total	8		

#### 4.11 USE OF DEWORMERS

From table 4.10, a total of forty seven (25%) of the participants had used dewormer within the past three months, whereas the remaining one hundred and thirty nine had not taken any dewormer representing 75%. Of the forty seven who had taken dewormer twenty one of them were infected and twenty six non infected. From the number of those who had not taken dewormer, seventy three were infected while the remaining sixty six were not infected.

**Table 4.10: Dewormer use within the last three months.**

	<b>Infected</b> (n=94)	<b>Non Infected</b> (n=92)
<b>Dewormer</b>	21(22%)	26(28%)
<b>No Dewormer</b>	73(78%)	66(72%)



**Figure 9: A graph showing the distribution of parasite in relation to dewormer usage.**

## CHAPTER FIVE

### 5.0 DISCUSSION

Soil Transmitted Helminths was common among the pregnant women in the Bonsaaso Cluster. Hookworm was the most common but no *T. trichiura* was encountered. However, the intensity of infection was low in the both cases of schistosomiasis and helminthiasis.

#### 5.1 Demography

The average age for the respondents was 26 years with 15 years being the youngest and 41 years the oldest. The age of a female in relation to pregnancy is very important. In Ghana the legal age for a female to get married is 16 years. It is therefore expected that an expectant mother must be 16 or more years. This however is not always the case especially in the rural areas like Bonsaaso. Pregnancy before the age of 18 or after the age of 35 increases the health risks for the mother and her baby (UNICEF, 2010). Majority of the respondents (52%) were between the ages of 20-29 years.

Agriculture is the backbone of the country's economy with the majority of the workforce especially in the rural areas in the agricultural sector. Majority (44%) of the respondents were farmers which is agreeable for a rural Ghanaian setting, but with the gold find in the district more and more of the inhabitants are shifting to the illegal gold mining (galamsey) since their lands are turning into goldfields. Despite the risk associated with galamsey women also participate although it may be assumed that women will refrain from the galamsey activities but perhaps its so because the money from galamsey comes readily and in bulk.

The study shows an average gravidity of two (2) with multigravida of two to five (2-5) being the highest. Generally, the age at which a woman gives birth for the first time is directly related to the number of children she will bear because it affects the length of time she will have the chances of becoming pregnant. People in rural areas start giving birth at an early age. In anticipation to getting support and to compensate for those who will die, they tend to give birth to many children.

Most rural mothers are ignorant of the importance of early Antenatal Clinic visits and therefore do so when the pregnancy has reached an advanced stage. The study showed that most of the pregnant women were in their second (50%) and third (41%) trimesters. This could be attributed to the fact that many women do not attend antenatal clinic until after the first trimester. At that time, the decision of whether the fetus will stay or otherwise has been determined. Again abortions of pregnancies usually take place within the first trimester.

## **5.2. Parasites**

### **5.2.1 Soil Transmitted Helminths**

Infection of STH's occurs worldwide in warm and humid climates where sanitation and hygiene are poor. Persons in these areas are at risk if soil contaminated with human feces enters their mouths or if they eat vegetables or fruits that have not been carefully washed, peeled or cooked. The Soil Transmitted Helminths encountered were *A. lumbricoides* and Hookworm however; they were of very low intensities.

In all, four different parasites were found in the stools examined. These were *Ascaris lumbricoides*, Hookworm (*A.duodenale* and *N.americanus*) *Giardia lamblia* and *Strongyloides stercoralis*. The Soil Transmitted Helminths are *Ascaris lumbricoides*,

Hookworm (*A. duodenale* and *N. americanus*) and *Trichuris trichiura*. The percentage of the study population who had *A. lumbricoides* was five. Its co infection with the other STH (Hookworm) was 3%. Hookworm infection alone among the study population accounted for 23% which was the highest. This observation is in line with a cohort study carried out in the Sekyere East district of Ashanti Region by Baidoo *et al* on pregnant women. In their study, hookworm was the commonest intestinal helminths encountered. Out of 17.6% of those who were infected, 13.9% had hookworm (Baidoo *et al.*, 2010). Interestingly *Giardia lamblia* and *Strongyloides stercoralis* (non STH) infections accounted for 33% of all the infection among the pregnant women with *G. lamblia* being the most prevalent with a percentage of seventeen from figure 7. *G. lamblia* and its co-infection with other parasites was 34% among all the pregnant women who took part in the study. Their co-infection with other STHs accounted for 11%. Most of the pregnant women (53%) were infected with at least one species of the STHs from figure 7.

All in all, the prevalence of hookworm was 59% among the study group but the highest (50%) prevalence occurred among those who were in their second trimester. This was followed by those in their third and first trimesters with the percentages of 36 and 14 respectively. None of the pregnant women was infected with *Trichuris trichiura*. In a study conducted in Nigeria, it was revealed that, those engaged in high energy task in farms are more likely to be exposed to infective hookworm larvae while those who eat or drink in the farm harbour more *Ascaris lumbricoides* and *Trichuris Trichiura* (Holland *et al*, 1989).

### 5.2.2 Schistosoma

Schistosomiasis is among the first top ten diseases in Amansie West District. (AWDHD, 2012). In Ghana the disease is attributed to *S. haematobium*, *S. japonicum* and *S. mansoni*, with snail as the intermediate host. Out of the total number of pregnant women who took part in the study 11% were infected with *S. haematobium*. Co-infection with Hookworm was 4.3% but mono infection of hookworm was 53%. In 2011 a study by Soares and colleagues in the country came out with 14.4, 3.2 and 07 percents for *S. haematobium* mono-infection, Hookworm mono-infection and co infection of the two diseases respectively. Neither the *S. mansoni* nor *S. japonicum* was seen in the stool or urine examined. Available data, which dates back to 1970's indicated that urinary schistosomiasis is widespread in all parts of the country, the same data shows that intestinal schistosomiasis is restricted and patchy in its distribution occurring mainly in the Volta basin. This could be supported by this study since the species that cause intestinal schistosomiasis were not encountered. Though the presence of blood in urine (haematuria) could be the cause of urinary schistosomiasis in areas where the disease is endemic, the study shows that not all of them had schistosoma ova. Out of the number of respondents who had haematuria, 87% had ova of *S. haematobium* when the urine was examined under microscope. No ova were seen in the rest representing 13%. The haematuria could be as a result of lesions in the bladder or other urinary tract infection. Another possibility could be just general haematuria. During pregnancy, the pressure from uterus on the bladder can cause a bit of bleeding. The study showed that not all cases of haematuria are schistosomiasis.

### 5.3 Use of Dewormers

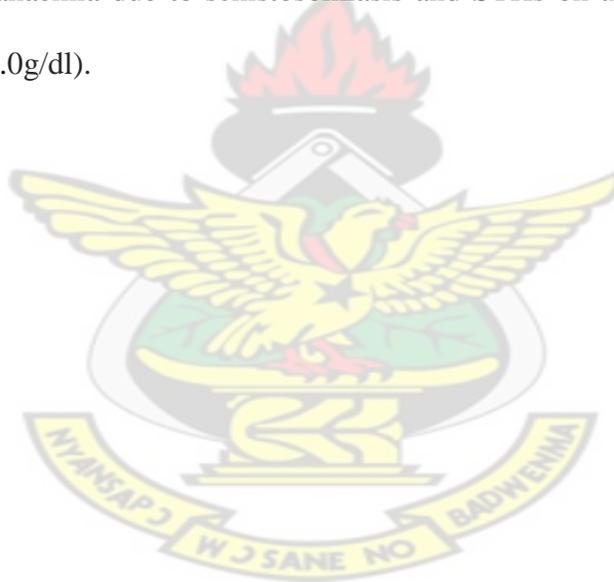
It is recommended that anthelmintic treatment should be selective and given at yearly intervals preferably with albendazole, due to its cost-effectiveness (Zani *et al*, 2004). The adult population very often is left out even in mass deworming programmes. Dewormers are contraindicative during the first trimester of pregnancy. WHO and UNICEF recommend that albendazole or mebendazole be given to pregnant women after the first trimester of pregnancy. This means any women who did not take dewormer prior to conception will have to wait after the first three months. Majority of the respondents (139), 75% had not used any dewormer within the last three months. From table 4.10, most of the pregnant women (53%) who said they had not used dewormer within the last three months had soil transmitted helminths infection with hookworm being the prevalent (46%) as seen from figure 9. There was no significant relation between the use of dewormer and parasitic infection ( $p=0.352$ ) at 95% confidence interval. Among those who had taken dewormer in the last three months, 44% of them still had soil transmitted helminths. This figure is on the higher side when compare to a study in Thailand (Liabsuetrakul *et al*, 2009) which looked at the effect of treatment of soil transmitted helminthiasis in pregnant women. In that study, soil transmitted helminths were detected in 17.9% pregnant after they had been treated with anthelmintic. This discrepancy could be attributed to the generic and the efficacy of the anthelmintic used in both cases.

#### 5.4 Haemoglobin level with Soil Transmitted Helminths and Schistosomiasis

Anaemia is defined as reduction in circulating haemoglobin mass below the critical level. The normal haemoglobin (Hb) concentration in the body is between 12-14g/dl. Pregnancy causes a state of hydraemic plethora. There is disproportionate increase of plasma volume during pregnancy leading to apparent reduction of RBC, haemoglobin and haematocrit value. Hb is consequently reduced to a varying extent occasionally as low as 80%. WHO has accepted up to 11g/dl as the normal haemoglobin level in pregnancy? Therefore any haemoglobin level below 11g/dl in pregnancy should be considered as anaemia. Apart from obstetrical factors such as gravidity, parity, history of previous preterm or small-for gestational-age deliveries, which cause acute or chronic blood loss, schistosomiasis and or STHs also reduce haemoglobin levels. Out of a total of 186 samples, the maximum haemoglobin level was 13.5 g/dl with a minimum of 6.2 g/dl (severe anaemia) given a range of 7.3 g/dl with an average of 9.95 g/dl (mild anaemia) (SD +/- 1.147692), which lies between 9.788 -10.120 (CI) at 95%. The respondent with the minimum haemoglobin level was 16 years and had no STHs or schistosoma and that was her first pregnancy. Interestingly the one with the highest haemoglobin of 13.5g/dl who was 26years had conceived four times and had hookworm as well as schistosoma. Perhaps this could be due to the fact that the parasites encountered were of low in intensities and their mere presence do not necessarily mean decrease in the haemoglobin level. In determining anaemia among pregnant women (Agyiri, 2011), found out that low intensities of parasites has no significant impact on haemoglobin levels. The overall mean haemoglobin level was 9.9g/dl (moderate anaemia). The mean haemoglobin for the infected and non infected with *Schistosoma*

*haematobium* was the same (9.9g/dl) but the standard deviation varied. (1.3 for those infected and 1.1 for non infected). This could be attributed to the light infections of *Schistosoma* (less than 100 eggs/10ml of urine). Those infected with STH had a mean haemoglobin level of 9.9g/dl and the non infected was 10.0g/dl.

This is consistent with a study conducted in the northern part of the country by Fuseni *et al*, (2010) which concluded that the mean haemoglobin of mothers without any parasite was within the normal range, mothers with co infections on the other hand were within the moderately anaemic range. Hookworm infections alone cause moderate anaemia. However, the anaemia due to schistosomiasis and STHs on the whole was not severe (Hb less than 7.0g/dl).

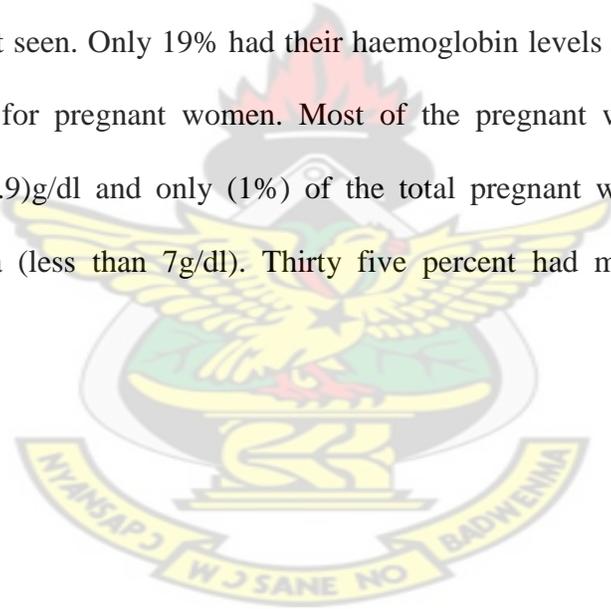


## CHAPTER SIX

### 6.0 CONCLUSION AND RECOMMENDATION

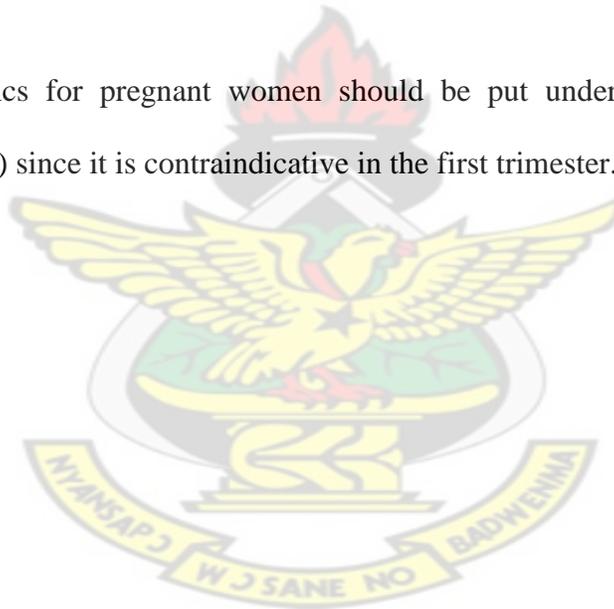
#### 6.1 Conclusion

Most of the pregnant women (53%) were infected with at least one species of the soil transmitted helminths with hookworm (23%) being the prevalent. In the study area, the prevalence of STHs was high but infection intensity was low as seen in Tables 4.2 and 4.4. Eleven percent (11%) of all the urine examined had *S.haematobium* eggs. The other species of (*S.mansoni* and *S.japonicum*) that are also known to cause schistosomiasis in Ghana were not seen. Only 19% had their haemoglobin levels above the recommended value (11g/dl) for pregnant women. Most of the pregnant women (45%) had mild anaemia (7.0-9.9)g/dl and only (1%) of the total pregnant women in the study had severe anaemia (less than 7g/dl). Thirty five percent had moderate anaemia (10.0-10.9)g/dl.



## 6.2 Recommendations

1. Pregnant woman should have their stool and urine examined at least three times before they deliver as in the case of haemoglobin level determination.
2. Apart from children of school going age, all women of child bearing age and adults who are at risk (pregnant women because of the role they play in homes; preparing food, fetching water from streams and rivers) should be part of the mass deworming exercise.
3. The women must be taught the proper way of hand washing with soap under running water.
4. Anthelmintics for pregnant women should be put under the Directly Observed Therapy (DOT) since it is contraindicative in the first trimester.



## REFERENCES

- Agyiri, J. (2011).** Determinants of anaemia in pregnant women in Peri-urban Kumasi in the Ashanti Region. (<http://dspace.knust.edu.gh:8080/jspui/>)  
Date visited: 15th Feb.2013.
- Allen, L.H. (2000).** Anaemia and iron deficiency; effects on pregnancy outcome. *The American Journal of clinical Nutrition.* (supp):1280s-1284
- Anderson, R. and May, R. (1995).** *Infectious Diseases of Humans, Dynamics and Control*, Oxford University Press, p 16.
- Allen, J.E. and Maizels, R.M. (1996).** Immunology of human helminth infection. *International Archives of Allergy and Immunology*; 109, 3–10.
- Ash, L.R., Orihel, T.C., and Savioli, L. (1994).** *Bench Aids for the diagnosis of intestinal parasites.* Geneva, World Health Organization.
- Awasthi, S., Bundy, D.A. and Savioli, L. (2003).** Helminth infections. *British Medical Journal* 327:431-433.
- AWDHD (2012). Amansie West District Health Directorate. Annual Performance Review Report for 2011.
- Baidoo, S.E., Tay, S.C.K., Obiri Danso, K. and Abruquah, H.H. (2010).** Intestinal Helminth infection and anaemia during pregnancy: A community based study in Ghana. *Journal of Bacteriology Research* Vol.2 (2) pp 9-13.
- Bethony, J., Brooker, S. and Albonico, M. (2006).** Soil-transmitted helminth infections: Ascariasis, trichuriasis, and hookworm. *Lancet* 367: 1521–1532.
- Bird, A.F. and Bird, J. (1991).** *The structure of Nematodes.* 2<sup>nd</sup> edition San Diego, California: Academic Press, Inc.
- Brabin, B.J., Hakimi, M., Pelletier, D. (2001).** An analysis of anemia and pregnancy-related maternal mortality. *J. Nut.* 131: 604s-615s.
- Brooker, S., Hotez, P.J., Bundy, D.A.P. (2008).** Hookworm-Related Anaemia among Pregnant Women: A Systematic Review. *PLoS Negl Trop Dis* 2(9): e291.  
doi:10.1371/journal.pntd.0000291
- Brooker, S., Bethony, J. and Hotez, P.J. (2004).** Human hookworm infection in the 21st century. *Advances in Parasitology* 58: 197–288.
- Brooker, S., Clements A. and Bundy, D.A.P. (2006).** Global epidemiology, ecology and control of soil transmitted helminth infections. *Advance in Parasitology*, 62 pp 223-65.

**Bundy, D.A.P., Chan, M.S. and Saviola, L. (1995).** Hookworm infection in pregnancy. *Trans R Soc Trop. Med. Hyg.* 89: 521–522. Center for Disease Control, author. "Current Trends CDC Criteria for Anemia in Children and Childbearing-Aged Women." *Morbidity and Mortality Weekly Report.* 1989 Jun 9; 38 (22):pp 400–404.

**Bundy, D.A.P, and Blumenthal, U.J. (1990)** Human behaviour and the epidemiology of helminth infections: the role of behaviour in exposure to infection, in: C.J. Barnard, J.M. Behnke (Eds.), *Parasitism and Human Behaviour*, Taylor and Francis, London. pp. 264-289.

**Bundy, D.A.P. (1988).** Population ecology of intestinal helminth infections in human Communities, *Philos. T. Roy. Soc. B* 321 pp 405-420

**Centre for Diseases Control. (2013).** Parasites-Soil Transmitted Helminths (2013) Available online at: [http:// www.cdc.gov/parasites/sth](http://www.cdc.gov/parasites/sth)

**Centre for Disease Control (1989).** "Current Trends CDC criteria for anaemia in children and Childbearing-Aged Women". *Morbidity and Mortality Weekly Report.* 1989 Jun.

**Chan, L., Bundy, D.A.P. and Kan, S.P. (1994).** Genetic relatedness as a determinant of predisposition to *Ascaris lumbricoides* and *Trichuris trichiura* infection, *Parasitology* 108 pp77-80.

**Chan, M.S. (1997).** The global burden of intestinal nematode infections—fifty years on, *Parasitology. Today* 13 pp 438-443.

**Chandrakala, K., Zaheda, B and Meenakumari, A. (2013).** *ISOR Journal of Dental and Medical Sciences* Volume 9 Issue 1 pp 09-13

**Cheesbrough, M. (2006).** *District Laboratory Practice in Tropical Countries.* 2nd edition. Cambridge University Press.

**Chestnut, D.H., Polley, L.S., Tsen, L.C. and Wong, C.A. (2009).** *Physiologic Changes of Pregnancy* 4th ed. USA: Mosby Elsevier. pp. 21–23.

**Crompton, D.W.T and Nesheim, M.C. (2002).** Nutritional impact of intestinalhelminthiasis during the human life cycle, *Ann. Rev. Nut.* 22: 35-59.

**de Silva N.R., Brooker, S. Hotez, P.J. Montresor, A. and Engels, D. (2003)** Soil-transmitted helminth infections: Updating the global picture. *Trends Parasitology* 19: 547–551.

**Despommier, D., Gwadz, R.W. Hotez, P.J. and Knirsch, C.A. (2005).** *Parasitic Disease*, 5th edition New York. Apple Tree Production.

**Dreyer, G., Fernandes-Silva. E., Alves,S., Rocha, A., Albuquerque, R. and Addis, D (1996).** Patterns of detection of *Strongyloides stercoralis* in stool specimens: implications for diagnosis and clinical trials. *J Clin Microbiol.* 34:2569–2571.

**Duerden, B.I., Reid, T.M.S., and Jewsbury, J.M. (1993).** *Microbial and Parasitic infection*, 7<sup>th</sup> Edition Great Britain Edward Arnold.

**Dold, C. and Holland, C.V. (2010).** *Ascaris* and ascariasis, *Microbes and Infection* (2010),doi: 10.1016/j.micinf.2010.09.012

**Elkins D.B., Haswell-Elkins, M. and Anderson, R.M. (1986).** The epidemiology and control of intestinal helminths in the Pulicat Lake region of Southern India. Study design and pre- and post-treatment observations on *Ascaris lumbricoides* infection. *T. Roy. Soc. Trop. Med. H.* 80 pp 774-792.

**Fuseini,G., Edoh, D.,Kalifa, B.G.H., Abdul-Wahab and Knight, D. (2010).** Parasitic infections and anaemia during pregnancy in the Kassena-Nankana district of Northern Ghana. *Journal of Public Health and Epidemiology* Vol. 2(3), pp. 48-52.

**Ghana Statistical Service: (2010).** Population and housing census 2010. available online at <http://www.statsghana.gov.gh>

**Gill G.V., Welch, E., Bailey, J.W., Bell, D.R.B. and Beeching, N.J.B. (2004).** Chronic *Strongyloides stercoralis* infection in former British Far East prisoners of war. *QJM.* 2004; 97:789–795. doi: 10.1093/qjmed/hch133.

**Gryseels, B., Polman, K. and Clerinx, J. (2006).** Human Schistosomiasis. *Lancet:* 368:110618.

**Hayashi Y, Ueyama, H., Mashimo, T., Kangawa, K. and Minamino, N. (2005).** Circulating mature adrenomedullin is related to blood volume in full-term pregnancy. *Anesth Analg.* 101:1816–20.

**Højbye, L.and Carlsen, A. (2007).** Microbial risk assessment of local handling and use of human faeces. *Journal of Water and Health* 05.1, 117-128

**Holland, C.V., Asaolu S.O., Crompton, D.W.T., Stoddart, R.C., MacDonald, R. and Torimiro, S.E.A. (1989).** The epidemiology of *Ascaris lumbricoides* and other soil-transmitted helminths in primary school children from Ile-Ife, Nigeria, *Parasitology* 99 (1989) 275-285.

**Hotez, P.J., Brindley, P.J., Bethony, J.M., King, C.H., Pearce, E.J. and Jacobson, J. (2008).** Helminth infections: the great neglected tropical diseases, *J. Clin. Invest.* 118 pp 1311-1321.

**Hotez, P.J., Brooker, S. and Bethony, S.J. (2004).** Hookworm infection. *New England Journal of Medicine* 351:pp799–807.

**Hotez, P.J and Kamath, A. (2009)** Neglected Tropical Diseases in sub-saharan Africa: Review of their prevalence, distribution and disease burden. *Plos Neglected Tropical Disease* 3(8):e412 doi; 10.1371/journals.pntd.0000412

**Huddle, M., Gibson, S., Cullinan, R. (1999).** The impact of malaria infection and diet on the anaemia status of rural pregnant Malawian women. *European J Clin Nutr* 1999, **53**:792-801.

**Idowu, O.A.,Mafiana, C.F. and Sotilove, D. (2005).** Anaemia in pregnancy: A survey of pregnant women in Abeokuta, Nigeria. *Afr Health Sci.*5:295–9.

**Jawetz, E., Melnick, J.L. and Adelberg, A.E. (1980).** Review of Medical Microbiology, 14th edition. Lange.

**Jordan, P. and Webbe, G. (1993).** Human Schistosomiasis:Epidermiology University Press,Cambrige; United Kingdom.

**Keiser, P.B. and Nutman, T.B. (2004)** *Strongyloides stercoralis* in the Immunocompromised Population. *Clin Microbiol Rev.* 2004; 17:208–217.

**King, J.C. (2003).** The Risk of Maternal Nutritional Depletion and Poor Outcomes Increases in Early or Closely Spaced Pregnancies *J. Nutr.* May 1, 2003 vol. 133 no. 5

**King, C. (2004).** Re-gauging the cost of chronic helminthic infection: meta-analysis of disability- related outcomes in endemic schistosomiasis. *Lancet* 368: 1106-1118

**Knightlinger, L.K. Seed, J.R. and Knightlinger, M.B. (1998)** *Ascaris lumbricoides* intensity inrelation to environmental, socioeconomic, and behavioral determinants of exposure toinfection in children from southeast Madagascar, *J. Parasitology.* 84 pp 480-484.

**Kjetland, E.F, Kurewa, E.N. and Ndhlovu, P.D.N. (2008)** Female genital schistosomiasis – a differential diagnosis to sexually transmitted disease: genital itch and vaginal discharge as indicators of genital *Schistosoma haematobium* morbidity in a cross-sectional study in endemic rural Zimbabwe. *Tropical Medicine and International Health* 13, 1509–1517.

**Klumpp, R.K and Chu, K.Y. (1977).** Ecological studies of *Bulinus rohlfsi*, the intermediate host of *Schistosomiasis haematobium* in the Volta Lake. *Bulletin of the WHO* 55, pp715-730.

**Klumpp, R.K. (1983).** A study of the transmission of *Schistosomiasis haematobium* in volta Lake,Ghana. University of London

**Kvalsvig, J.: Intestinal nematodes and cognitive development, in: Holland, C.V. and Kennedy, M.W.(Eds.). (2002).** The Geohelminths: Ascaris, Trichuris and Hookworm, Kluwer Academic, London. pp. 62-73

**Liabsuetrakul, T., Chaikongkeit, P. and Korviwattanagarn, S. (2009).** Epidemiology and effect of treatment of soil transmitted helminthiasis in pregnant women in southeast Thailand. Southeast Asian J Trop Med Public Health 2009 Mar; 40 (2):211-22

**Libman, M.D., MacLean J.D. and Gyorkos ,T.W. (1993).** Screening for schistosomiasis, filariasis, and strongyloidiasis among expatriates returning from the tropics. Clin Infect Dis.17:353–359.

**Marchi, B.J. and Cantons G.A.C. (2003).** Evaluation of techniques for the diagnosis of Strongyloides stercoralis in human immunodeficiency virus (HIV) positive and HIV negative individuals in the city of Itajai, Brazil. J Infect Dis.7:402–408.

**Markell, Edward, John, K., David, C., Petri, and William H. (2006).** *Markell and Voge's medical parasitology* (9th ed.). St. Louis, Mo: Elsevier Saunders. ISBN 0-7216-4793-6.

**McCallum, H.I. (1990)** Covariance in parasite burdens: the effect of predisposition to infection. Parasitology 100 pp 153-159

**Molyneux, D.H and Malecala, M.N. (2011)** Neglected Tropical Diseases and the MDGs Why the other diseases matter: reality versus rhetoric. Parasite vectors vol: 4

**Montresor, A. (1998).** Schistosomiasis and intestinal parasites unit, WHO. Child Health Dialogue. 1998;10:9.

**Montresor, A., Crompton, D.W.T., Gyorkos, T.W. and Savioli, L. (2002).** Helminth control in school-age children: A guide for managers of control programmes. Geneva: World Health Organization.  
Available:[http://www.who.int/wormcontrol/documents/helminth\\_control/en/](http://www.who.int/wormcontrol/documents/helminth_control/en/)

**Neva, F.A and Brown, H.W. (1994)** Basic Clinical Parasitology, 6<sup>th</sup> edition, Appleton and Lange Paramount publishing and business professional group.

**Odei, M.A. (1983).** The effect of the Volta dams at Akosombo and Kpong on the ecology of schistosomiasis transmission in the lower Volta and its estuary in Ghana. Bulletin of IFAN (A) 45: 195-207; 1983.

**O'Lorcain, P., and Holland, C.V. (2000)** The public health importance of *Ascaris lumbricoides*, Parasitology 121 pp51-71.

**Okoh, V. O. (1994).** Case study of the Volta Lake; the health aspect. Report by lakeside Health Unit. Volta River

**PLoS Negl Trop Dis. 2011 June;** 5(6): e1200. Published online 2011 June 7. doi: 10.1371/journal.pntd.0001200

**Pawlowski .S. and Davis, A. (1989).** Morbidity and mortality in ascariasis. In: Crompton D W T, Nesheim M C, Pawlowski Z S, editors. Ascariasis and its prevention and control. London, England: Taylor and Francis; 1989. pp. 71–86.

**Penelope, N. (2002).** Adjusting Haemoglobin Values in Program Surveys. International Nutritional Anaemia Consultative Group. 2002 Jun.

**Ramarakoto, C.E., Leutscher, P.D., and van Dam G. (2008)** Ultrasonographical findings in the urogenital organs in women and men infected with *Schistosoma haematobium* in northern Madagascar. Transactions of the Royal Society of Tropical Medicine and Hygiene 102, 767–773.

**Rayapu, V., Senthil P. D., Ivvala A.S., Sandeep, K. (2012).** Prevalence of Intestinal Helminthic Parasites in School Going Children in Rural Area of Kuppam, Andhra Pradesh. International Journal of Basic Medical Sciences and Pharmacy Vol. 2, No. 2, pp. 76-79.

**Roberts, L. and Janovy Jr., J. (2005).** Foundations of Parasitology. 2005. p 412

**Roepstorff, A. (2003).** Ascaris suum in pigs: population biology and epidemiology, Danish Centre for Experimental Parasitology, The Royal Veterinary and Agricultural University, Copenhagen. p.113

**Roman-Sanchez P., Pastor-Guzman, A. Moreno-Guillen, S. Igual-Adell, R. Suner-Genroso, S. and Tornero Estebanez, C. (2003).** High prevalence of *Strongyloides stercoralis* among farm workers on the Mediterranean coast of Spain: analysis of the predictive factors of infection in developed countries. Am J Trop Med Hyg. 69:336–340.

**Rusia, U., Madan, N., Agarwal, N., Sikka, M., and Sood, S. (1995).** Effect of maternal iron deficiency anemia on foetal outcome. Indian Journal of Pathology and Microbiology: 38 pp 273-279

**Sato, Y., Kobayashi, J., Toma, H. and Shiroma, Y. (1995).** Efficacy of stool examination for detection of *Strongyloides* infection. Am J Trop Med Hyg. 53:pp 248–250.

**Segarra-Newnham, M. (2007).** Manifestations, diagnosis, and treatment of *Strongyloides stercoralis* infection. Ann Pharmacother. 41(12): 1992-2001.

**Schonning, C., Westrell, T., Stenstrom, T.A., Ambjerg-Nelson, K., Hasling, A.B., Hoibye, L., Carlsen, A. (2007).** Microbial risk assessment of local handling and use of human faeces. *Journal of Water and Health* 05.1,117-128.

**Sharma, J.B. (2003).** Nutritional anaemia during pregnancy in non-industrialized countries: Progress in obstetrics and gynaecology. 15th ed. Spain: Churchill Livingstone; 2003. p.103-11

**Sinniah, B. (1982).** Daily egg production of *Ascaris lumbricoides*: the distribution of eggs in the faeces and the variability of egg counts, *Parasitology* 84 pp 167-175.

**Sinniah, B. and Subramaniam, K. (2009).** Factors influencing the egg production of *Ascaris lumbricoides*: relationship to weight, length and diameter of worms, *J. Helminthol* 65 pp 141-147.

**Soares Magalhães, R.J., Biritwum, N.K., Gyapong, J.O., Brooker, S., Zhang, Y., Blair, L., Fenwick, A., and Clements, A.C.A. (2011).** Mapping Helminth Co-Infection and Co-Intensity: Geostatistical Prediction in Ghana *PLoS Negl Trop Dis* 5(6): e1200.doi:10.1371/journal.pntd.0001200

**Steinmann, P., Keiser, J., Bos, R., Tanner, M. and Utzinger J. (2006).** Schistosomiasis and water resources development: systematic review, meta-analysis, and estimates of people at risk. *Lancet Infect Dis* 6:411–425.

**Stoltzfus, R.J., Dreyfuss, M.L. Chwaya H.M. and Albonico, M. (1997)** Hookworm control as a strategy to prevent iron deficiency. *Nutr Rev* 55: 223–232.

**Strauss, M. and Blumenthal, U.J. (1990).** Human Waste Use in Agriculture and Aquaculture - Utilization Practices and Health Perspectives. International Reference Centre for Waste Disposal (now SANDEC), Duebendorf, Switzerland. Report no. 08/90 (main report).

**Sudarshi, S., Stumpf, R., Armstrong, M., Ellman, T., Parton, S., Krishnan, P., Gbedema iodini P.L.Ch and Whitty, C.J. (2003).** Clinical presentation and diagnostic sensitivity of laboratory tests for *Strongyloides stercoralis* in travellers compared with immigrants in a non-endemic country. *Trop Med Int Health*. 8:pp 728–732. doi: 10.1046/j.1365-3156.2003.01069

**Thein-Hlaing, T.S. (1984).** Epidemiology and transmission dynamics of *Ascaris lumbricoides* in Okpo village, rural Burma, T. Roy. Soc. Trop. Med. H. 78 pp 497–504.

**UNICEF: (2010).** Facts for life. available online at <http://www.factsforlifeglobal.org/01/1.html>

**Uparanukraw, P., Phongsri S. and Morakote, N. (1999).** Fluctuations of larval excretion in *Strongyloides stercoralis* infection. *Am J Trop Med Hyg.* 60 pp 967–973.

**Van der Werf, M.J., de Vlas, S.J., Brooker, S., Looman, C.W., Nagelkerke, N.J., Habbema, J.D. and Engels, D (2003).** Quantification of clinical morbidity associated with schistosome infection in sub-Saharan Africa. *Acta Trop* 2003,86:125–139.

**Vinneras, B., Björklund, A. and Jönsson, H. (2003).** Thermal composting of faecal matter as treatment and possible disinfection method - laboratory-scale and pilot-scale studies. *Bioresource Technology* 88, 47-54

**Weigel, M., Calle, A., Armijos, R.X., Vega, I.P., Bayas, B.V., Montenegro, C.E. (1996).** The effect of chronic intestinal parasitic infection on maternal and perinatal outcome. *Int J Gynecol Obstet* 1996; 52: -17.

**World Bank (1993).** World Development Report: Investing in Health. Oxford University Press.

**WHO: Schistosomiasis and soil transmitted Helminth infection (2006a).** Preliminary estimates of the number of children treated with albendazole or mebendazole. *Weekly epidemiology Rec.* 2006; 81; pp 145-464

**WHO: Preventative Chemotherapy in Human Helminthiasis (2006b).** Coordinated Use of Anthelmintic Drugs in Control Interventions: A Manual for Health Professionals and programme Managers, WHO, Geneva.

**WHO: Prevention and Control of Schistosomiasis and Soil-Transmitted Helminthiasis. (2002)** WHO Technical Report Series 912. 1-57

**WHO: Control of Ascariasis (report of WHO committee),** Technical Report Series 379 (1967) 1-39.

**Williamson, A.L., Brindley, P.J. Knox, D.P. Hotez, P.J. and Loukas, A. (2003)** Digestive proteases of blood-feeding nematodes. *Trends Parasitol* 19: 417–423.

**World Health Organization. Schistosomiasis.** Fact Sheet No 115. July, Geneva.

**WHO: Prevention and control of schistosomiasis and soil-transmitted helminthiasis: Report of a WHO expert committee.** In WHO Technical Report Series No. 912. pp. 1–57. Geneva: World Health Organization; 2002.

**WHO: (2005).** Risking Death to Give Life. World Health Report. Available online at [www.who.int/whr/2005/chapter\\_4/en/index1.htm1#10](http://www.who.int/whr/2005/chapter_4/en/index1.htm1#10)

**WHO: (1994).** Report of the WHO informal consultation on hookworm infection and anaemia in girls and women. WHO/CTD/SIP/96.1. World Health Organization, Geneva

**WHO: (2012).** Accelerating work to overcome the global impact of neglected tropical diseases: A roadmap for implementation (2012) available online at [http://www.who.int/neglected\\_diseases/en](http://www.who.int/neglected_diseases/en)

**WHO: Preventive Chemotherapy Data bank. 2009.** Available online at: [http://www.who.int/neglected\\_diseases/preventive\\_chemotherapy/databank/en/index.html](http://www.who.int/neglected_diseases/preventive_chemotherapy/databank/en/index.html).

**Zani, L.C., Favre, T.C., Pieri, O.S., Barbosa, C.S. (2004).** Impact of anthelmintic treatment on infection *Ascaris lumbricoides*, *Trichuris trichiura* and Hookworms in Covas, A Rural Community of Pernambuco, Brazil. *Rev. Inst. Med. trop. S. Paulo* 46(2):63-71, March-April, 2004

**Zhang, Q., Ananthe, C.V. Li Z., and Smulian J.C. (2009).** Maternal Anaemia and Preterm birth, a prospective cohort study. *International Journal of Epidemiology*; 38:1380-1389



**APPENDIX 1-QUESTIONNAIRE**

Name .....Code.....

Age.....

Gravida.....

Gestation.....

Occupation.....

Have you taken dewormer in the last three months? ( ) YES ( ) NO

**LABORATORY USE**

Haemoglobin level.....g/dl

**Stool**

Microscopy.....  
.....  
.....

**Urine**

Dipstick ( ) Positive ( ) Negative

Microscopy.....  
.....  
.....

## APPENDIX 2-TEST STATISTICS

Table 4.5: Haematuria and Schistosomiasis.

The Chi-Square test of independence was used to test statistic at (2-1) (2-1) degree of freedom.

	Blood in urine	No Blood in Urine	Total
Infected	21	0	21
Non infected	3	162	165
Total	24	162	186

Expected frequency between Infected and Blood in Urine 2.710

Expected frequency between Infected and No Blood in Urine 18.290

Expected frequency between non Infected and Blood in Urine 21.290

Expected frequency between in Infected and No Blood in Urine 143.710

$$\chi^2 = \frac{\sum_{i=1}^2 \sum_{j=1}^2 (O_{ij} - e_{ij})^2}{e_{ij}} \sim \chi_{1,1}^2$$

Where:  $O_{ij}$ = Observed frequencies and  $e_{ij}$ = Expected frequencies.

$$\chi^2 = \frac{(21 - 2.710)^2}{2.710} + \frac{(0 - 18.290)^2}{18.290} + \frac{(3 - 21.290)^2}{21.290} + \frac{(162 - 143.710)^2}{143.710}$$

$$= 159.771$$

Critical Region

$$P_r(\chi^2 : \chi_{value}^2) < 0.05$$

$$P_r(\chi_{value}^2 = 159.771) = 0.000$$

Table 4.11: Dewormer use within the last three months.

The Chi-Square test of independence was used as the test statistic at (2-1)(2-1) degree of freedom.

	Dewormer	No Dewormer	Total
Infected	21	73	94
Non infected	26	66	92
Total	47	139	186

Expected frequency between Infected and Blood in Urine	23.753
Expected frequency between Infected and No Blood in Urine	70.247
Expected frequency between non Infected and Blood in Urine	23.247
Expected frequency between in Infected and No Blood in Urine	68.753

$$\chi^2 = \frac{\sum_{i=1}^2 \sum_{j=1}^2 (O_{ij} - e_{ij})^2}{e_{ij}} \sim \chi_{1,1}^2$$

Where:  $O_{ij}$  = Observed frequencies and  $e_{ij}$  = Expected frequencies.

$$\chi^2 = \frac{(21 - 23.753)^2}{23.753} + \frac{(73 - 70.247)^2}{70.247} + \frac{(26 - 23.247)^2}{23.247} + \frac{(66 - 68.753)^2}{68.753}$$

$$= 0.863$$

Critical Region

$$P_r(\chi^2 : \chi_{value}^2) < 0.05$$

$$P_r(\chi_{value}^2 = 0.863) = 0.352$$