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DEPARTMENT OF CLINICAL MICROBIOLOGY

IMPACT OF SCHISTOSOMIASIS AND SOIL TRANSMITTED HELMINTH INFECTION AMONG PREGNANT WOMEN IN THE DANGME EAST DISTRICT, GREATER ACCRA REGION.

THESIS SUBMITTED TO THE DEPARTMENT OF CLINICAL MICROBIOLOGY
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MASTER OF PHILOSOPHY DEGREE IN CLINICAL MICROBIOLOGY

BY

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DECLARATION

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

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DEDICATION

This work is dedicated to my parents Mr. Christian Nani and Mrs. Peace Nani. I also dedicate this work to my dear wife Mrs. Evelyn Nani and to my beloved siblings: Mrs. Cecilia Agamah, Patrick Nani, Sena Nani and Senyo Nani for their encouragement, love, advice, prayers and support.

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ABSTRACT

Parasitic infections during pregnancy and their subsequent pathological effects such as anaemia are common in developing countries. This study determined the prevalence of schistosomiasis and soil transmitted helminth infections during pregnancy. A cross sectional survey was conducted on 375 pregnant women during their visits to antenatal clinic in three health centers at Sege, Anyamam, Bonikope within Dangme East District in Greater Accra from April-July, 2012.

Urine specimens were prepared using the filtration method, stool specimens were prepared using formol ether concentration method whilst blood film specimens were prepared and stained with Giemsa. All prepared specimens were examined microscopically. The overall prevalence of infection with urine, stool and blood parasite was 49.6% with the following results *Schistosoma haematobium* (4.5%), *Ascaris lumbricoides* (8.5%), *Schistosoma mansoni* (7.5%), *Trichuris trichiura* (5.9%), Hookworm (4.0%), *Strongyloides stercoralis* (1.9%) and *Taenia species* (0.8%) and Plasmodium species (16.5%). More than half (66.4%) of the pregnant women who were infected were anaemic with the highest level of anaemia (37.9%) occurring within 20-29 years age group. *Schistosoma haematobium, Schistosoma mansoni,* hookworm and *Plasmodium spp* infections caused anaemia and was statistically significant ($p < 0.05$). However, severe anaemia (Hb < 7.0 g/dl) caused by the overall parasitic infection was not statistically significant. It was also observed that multigravids and multiparous women were more susceptibility to the negative impact of infections.

The study suggests that infections with soil transmitted helminthes, schistosomiasis and malaria, may contribute to anaemia in pregnant women. Therefore preventive and intensive educational

programmes for the control of these parasites are recommended to reduce the disease burden and their accompanying pathological effects during pregnancy.

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CHAPTER ONE

1.0 INTRODUCTION

1.1 BACKGROUND

Schistosomiasis and soil transmitted helminthiasis (STHs) remain a public health problem in many parts of the continent including Africa, the Middle East, Asia and South America (WHO, 2008). Schistosome infections co-occur with STHs, and these helminthes among other infections are known to be a risk factor for anaemia, and other health consequences in the human communities (Fleming, 1989; Asobayire, 2001). Soil transmitted helminthes and schistosome infections are prevalent in people living in developing countries and morbidity are mostly associated with heavy intensity of infection (Crompton, 1999; Montresor *et al.*, 2002). Schistosomiasis is a chronic parasitic disease caused by blood flukes of the genus schistosome and particularly affects agricultural and fishing communities, as well as populations using infested water bodies for domestic chores (WHO, 2002; Ziegelbaurer *et al.*, 2012). Schistosomiasis is estimated to affect 207 million people worldwide with 652 million people at risk of infection in 74 endemic countries (WHO, 2010). More than 90% of schistosomiasis cases are found in sub-Saharan Africa and more than 200,000 deaths are attributed to this infection in Middle East and North Africa (Hotez and Kamath, 2009; WHO, 2010; Hotez *et al.*, 2012). The principal schistosome species known to infect man are *Schistosoma haematobium* responsible for urinogenital schistosomiasis, and *Schistosoma mansoni* and *Schistosoma japonicum*; responsible for intestinal schistosomiasis (Schmitt, 2006). In Ghana, *Schistosoma haematobium* and *Schistosoma mansoni* are endemic and their transmissions are facilitated by intermediate fresh water snails, *Bulinus spp. (Bulinus globosus* and *Bulinus truncatues)* and *Biomphalaria pfeifferi* respectively (Ayanda, 2009, Madsen, 2008).

Soil-transmitted helminthes are a group of parasitic nematode worms causing human infection through contact with parasite eggs or larvae found mostly in warm and moist soil of the world's tropical and subtropical countries (Bethony *et al.,* 2006). One common route of soil-transmitted helminthes is engagement in agricultural pursuits (Hotez, 2002). Of particular worldwide importance are the roundworms (*Ascaris lumbricoides*), whipworms (*Trichuris trichiura*) and hookworms (*Necator americanus or Ancylostoma duodenale*) (de Silva *et al.,* 2003). More than one billion people are infected with at least one of these species (WHO 2005). These are considered together because it is common for a single individual to be chronically infected with all three worms (WHO, 2005). Heavy helminth infection occurs in about 300 million people worldwide and causes severe morbidity that results in more than 10,000- 135,000 deaths annually (Lustigman *et al.,* 2012). Heavily infected individuals are more at risk from morbidity and mortality and also act as significant resevoirs (Crompton, 1999; Montresor *et al.,* 2002). Susceptibility of host to disease conditions such as tuberculosis, HIV and malaria can be increased due to infection by soil transmitted helminthes (Fincham et al., 2003; Le *et al.,* 2004).

Globally, about 800 women die every day while giving life, and for each of them at least another 20 women suffer injury, infection or disability (WHO, 2013). In Ghana, maternal mortality rate continues to be high with 378 per 100,000 live births between 2000 and 2005 (Ghana Statistical Service, 2009). An estimated 10 million pregnant women in Africa who are infected with schistosomiasis suffer from anaemia and almost 7 million pregnant women in sub-Saharan Africa are infected with hookworms and are also at risk of developing anaemia (King, 2004; WHO, 1994). *Schistosoma haematobium* causes urogenital schistosomiasis in about one third of infected women (Hotez, 2006) and this is considered a potential risk factor for sexually transmitted

infection including HIV (Lawan, 2004). Chronic inflammation and blood loss caused by urogenital schistosomiasis is thought to increase the risk for adverse outcome of pregnancy (Friedman *et al.,* 2007). Intestinal helmithiasis are known to aggravate pre-existing anaemia by decreasing appetite and thus food and iron intake (Baidoo *et al.*, 2010; Bondevik *et al.,* 2000). There is evidence to highlight the importance of hookworm as a threat to maternal and child health (Cook, 1994; Scholl *et al.,* 1992). Out of the estimated 148 million women of reproductive age living in sub-Saharan Africa, 37.7 million (25 per cent) were infected with hookworms. It is further estimated that between 25 and 33 per cent of pregnant women were infected with hookworm, putting them at a greater risk "of preventable hookworm-related anaemia" (Brooker *et al.,* 2008).

1.0.1 PREGNANCY

Pregnancy, childbirth and the challenges associated with newborn needs are inevitable aspect of most women life (Cunnigham *et al.,* 2010). Studies have reported that compared to prepregnancy conditions, physical performance of women and their perception of their level of health and well-being decrease during pregnancy (Haas *et al.,* 2005; Otchet *et al.,* 1999). It has also been documented that, experience of pains and fatigue can negatively affect quality of life during and after childbirth (Katz, 2008).

Being pregnant is a period when significant increase in iron is highly required as compared to a non-pregnant state (Zavaleta *et al.,* 2000). This is because there is steady rise in iron requirement of approximately 0.8 mg per day in the first month to approximately 10 mg per day during the last six weeks of pregnancy (Bothwell, 2000). Reasons for increased iron requirement is due to expansion of maternal red cell mass for increased oxygen transport which includes, transfer of iron to the developing foetus and placenta structures; which will in turn serve as a reserve for blood

loss and lochia at parturition (Beaton, 2000). Because of increased iron requirements, there is a high risk of developing anaemia and this could account for the high proportion of women becoming anaemic during pregnancy (Beaton, 2000). The health status of a woman even before pregnancy is a crucial determinant of gestational morbidity and pregnancy outcomes (Friedman *et al.*, 2007). Gravidity is the number of times that a woman has been pregnant and parity is the number of times she has given birth to a foetus with a gestational age of 24 weeks or more regardless of whether the child was born alive or was stillborn (Creinin and Sinmhan, 2009)

1.0.2 ANAEMIA IN PREGNANCY

Anaemia in pregnancy is defined as a hemoglobin concentration less than 11.0g/dl and it poses public health concerns in many developing countries (WHO, 1992). Prevalence of anaemia during pregnancy worldwide had been estimated to be at 41.8% which corresponds to about 56.4 million women (McLean *et al*., 2006). Approximately 30% of total global cases of anaemia during pregnancy are reported in sub-Saharan Africa (WHO, 2008).

In Ghana, the prevalence of anaemia increased from 65% to 70% between 2003 and 2008, whiles among non-pregnant women there was an increase from 48% to 58% within the same period (FANTA, 2014). The etiology of anaemia during pregnancy among women is dependent on some factors and also varies by geographic region (van den Brook, 1998). The primary cause of anaemia during pregnancy worldwide is iron deficiency, secondary to chronic inadequate dietary intake and menstruation and it is even heightened by the physiological demands of the foetus and subsequent maternal blood volume expansion (van den Brook, 1998; Gopalan, 1996). Infections including helminthes and malaria are also involved in the pathogenesis of anaemia in pregnancy (Glover *et al*., 2005). Helminthes such as hookworms suck blood out of tissues, but large amounts may be

lost by haemorrhage at the sites of attachment. Additional blood loss may be related to the tendency of the worms to migrate within the intestine leaving bleeding points at old sites of attachment, thus increasing the susceptibility of infected pregnant women to anaemia (Stephenson *et al*., 2000; Bondevik *et al*., 2000). The changes in the immune system associated with pregnancy coupled with severity of infection by malaria are also a cause of anaemia in pregnancy (Brabin *et al*., 1983; Fried *et al*., 1998).

Anaemia could be classified as mild (10.0- 10.9 g/dl), moderate (8.0- 9.9 g/dl) and severe (< 8.0g/dl) (Stoltzfus, 2003), amongst these categories, severe anaemia induces the most dramatic consequences which are increased risk of maternal morbidity and mortality, poor intrauterine growth, abortion, preterm and low birth weight (Stoltzfus, 2003; Bodeau-Livinec *et al*., 2011). The factors above result in higher perinatal morbidity, mortality and subsequently higher infant mortality (Brabin *et al*., 2001). Maternal anaemia may also lead to fetal anaemia and subsequently to infant anaemia as well as long-term childhood morbidities, including impaired neuro developmental outcomes (Koura *et al.,* 2012; Adedrian *et al.,* 2011; Rogawski *et al.,* 2012; Tamura *et al.,* 2002; Walker *et al.,* 2007)

Anaemia in pregnancy is defined as haemoglobin concentration less than 11.0 g/dl (WHO, 1992) and remains a major risk factor of maternal deaths in developing countries (Abou Zahr and Royston 1991). Women with anaemia from one cause or more causes (e.g. iron deficiency, malaria, intestinal helminth infection) are at risk of delivering low birth weight infants (Stephenson *et al.,* 2000; Steketee *et al.,* 2001; van den Broek , 2001; Shulman *et al.,* 2001).

Some other adverse maternal-fetal consequences are prematurity and impaired lactation which are associated with iron deficiency anaemia in pregnancy (Stoltzfus *et al.,* 1997).

1.2 PROBLEM STATEMENT

Soil transmitted helminthes and schistosomiasis among other parasitic infection continue to be a threat in pregnant women (Ordi *et al.,* 2009). The mass drug administration (MDA) for most control programs are school-based targeting only school-going children and excluding other infected individuals of the community such as non-school going children ,non-pregnant and pregnant women who may have high levels of infection. Out of the forty million women who are of childbearing age infected by schistosomes, (Friedman, 2007) about 10 million women in Africa have schistosomiasis during pregnancy and have possible adverse outcome. Prevalence of intestinal helminth infections is high in pregnant women compared with non-pregnant women (Adegnika *et al,* 2007; Godwin *et al,* 2010). It is therefore imperative to curb the occurrence of these infections and also find effective measures to prevent adverse outcome during pregnancy.

1.3 RATIONALE AND JUSTIFICATION

Schistosomiasis and soil transmitted helminthes continue to play a major role in the economic activities of the inhabitants prone to these infections (WHO, 2008). Currently, the Millenium Development Goal 5 which addresses issues of maternal health has not improved considerably as 1 in 3 pregnant women living in the rural areas do not have access to skilled professional care as stated in MDG 2010 report. Therefore; laboratory diagnostic evaluations are needed to determine disease prevalence in specific population, so that it can provide guidelines for radical treatment of treatable etiologic agents. Previous surveys of schistosomiasis and helminthes are often improvised

and pregnant women were not targeted (Montresor *et al.,* 2011; King *et al.,* 2009; Doumenge J *et al.,* 1987). It is therefore important to conduct this study among rural pregnant women to determine the impact of these infections on their health. Successful outcome of this study will add up to the management and care given to the pregnant women and also strengthen the preventive and control measures to avoid occurrence of such infections.

1.4 MAIN OBJECTIVE

The main objective of the study is to determine the impact of schistosomiasis and soil transmitted helminthes in pregnant women and to measure the relationship with their anaemia status.

1.4.1 SPECIFIC OBJECTIVES

1. Determine the prevalence of schistosomiasis and STH among pregnant women.
2. To compare the distribution of schistosomiasis and soil transmitted helminth infections with age of the pregnant women
3. To investigate relationship between schistosomiasis and anemia condition in pregnant women
4. To investigate relationship between STH and anemia condition in pregnant women
5. Determine the association between schistosomiasis and STH infections and the stages of gravidity of the pregnant women**.**

1.5 ETHICAL CLEARANCE

Ethical approval was obtained from the Committee on Human Research Publication and Ethics (CHRPE) of the School of Medical Sciences, Kwame Nkrumah University of Science and Technology. Permission to conduct the study at the clinics was sought from the District Director of Health Services and the pregnant women through written consent

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 SCHISTOSOMIASIS

Schistosomiasis, also known as, *bilharzia, bilharziasis*, or snail fever, affects approximately 200 million people worldwide and remains the third most devastating tropical disease, after malaria and intestinal helminthiasis, (WHO, 2010). In the year 2010; about 120 million people were symptomatic (WHO, 2010). According to the World Health Organization (WHO, 2010), approximately 652 million people are at risk with an estimated 200,000 deaths occurring annually with forty million women of childbearing age are infected (Friedman, 2007). Extensive researches have been conducted in schistosomiasis infections which includes the early discovery of schistosomiasis in 1851 by the German surgeon Theodor Bilharz, who worked in Cairo, (Nawal, 2010)

Similar to other trematodes, *Schistosoma* have complex life cycles consisting of both free-living and parasitic forms (Basch, 1991). There are 5 species of schistosomes (WHO, 2010), but the three most common are *Schistosoma mansoni, S. japonicum*, and *S. haematobium; and* the two other forms are *S. intercalatum* and *S. mekongi* (Gryseels *et al*., 2006). *Schistosoma mansoni* occurs in Africa, the Caribbean, South America, and the Eastern Mediterranean (de Silva *et al.*, 2003). *S. japonicum* and *S. mekongi* are found in Southeast Asia and the Western Pacific (de Silva *et al.,* 2003). *S. intercalatum* is endemic in central Africa and *S. haematobium* occurs throughout Africa and the Eastern Mediterranean (de Silva *et al.,* 2003). *S. haematobium* affects both the urinary and reproductive tract systems, whereas the other four species affects the hepatic and gastrointestinal systems (Schmitt, 2006). The geographical distribution of schistosomiasis is

known to be driven by climatic and environmental factors that have effect on the parasitic populations and those of the snail intermediate host (Brooker and Clements, 2009). A study conducted by Ricardo *et al.,* (2011) in Ghana, indicated a widespread of schistosomiasis.

2.1.1 *Schistosoma haematobium and Schistosoma mansoni*

Schistosoma haematobium is endemic in 54 countries mainly Africa and the eastern Mediterranean (Cheesbrough, 1999). In some areas the distribution of *Schistosoma haematobium* overlaps with *Schistosoma mansoni* causing co-infection (Cheesbrough, 1999). In Ghana the prevalence of schistosomiasis is as high as 90% in endemic communities mainly caused by the construction of hydroelectric dams and control of flood have greatly increased the prevalence of *Schistosoma haematobium* in particular (Gryseels *et al.,* 2006; McManus *et al.,* 2008).

Schistosoma mansoni is also widespread in Africa, the Eastern-Mediterranean, the Caribbean and South America (van der Werf *et al.,* 2003). Zoonotic transmission of *Schistosoma mansoni* has been reported in some studies and this implies its infection is not limited to man alone but wild rodents could be infected (Theron, 1984; Theron *et al.,* 2004).

2.1.2 Epidemiology *Schistosoma haematobium and Schistoma mansoni*

Human schistosomiasis is endemic in large areas of the tropics and subtropics (WHO, 2010). It has been estimated that, over 652 million people in 74 countries are exposed to the risk of schistosomal infection (WHO, 2010) and almost 200 million were estimated to be infected in 2003 (Fenwick, 2006), of which 85% was seen in sub-Saharan Africa (WHO, 2010). About 95% of the cases are due to *S. mansoni* and *S. haematobium* infections (Chitsulo *et al.,* 2000).

Schistosomiasis is largely an infection found in rural areas, but urban schistosomiasis is an increasing problem in many countries (Mott *et al.,* 1990). Natural streams, ponds and lakes are typical sources of infection, but over the past few decades, man-made reservoirs and irrigation systems, as well as population growth and migration, have contributed to the spread of schistosomiasis (Gryseels *et al.,* 2006; McManus *et al.,* 2008).

Within countries, regions and villages, the distribution of schistosomiasis can be very focal, depending on variations in snail populations and human–water contact behaviour (Gryseels *et al.,* 1998). The distribution of schistosomiasis can be highly uneven across individuals; the majority of the parasites are usually present in a small fraction of the infected individuals (Kabatereine *et al.,* 2004).

Studies have shown that women of childbearing age and pregnant women are at risk of schistosome's infection and schistosomiasis in pregnancy can have both iron deficiency and anaemia of chronic disease (Friedman *et al.,* 2007). Harmful effects of schistosomiasis have been shown in pregnant experimental mice (Amano *et al.,* 1990; el-Nahal *et al.,* 1998). *Schistosoma haematobium* has been reported to be associated with increased risk of premature birth and low birthweight among pregnant women (Siegrist, 1992). Prematurity, Intrauterine growth restriction (IUGR) are some other complications associated with schistosome infection in pregnancy (Holcberg *et al.,* 2001, Wang *et al.,* 2003). Increased risk for the development of acute subchorionitis at the maternal-fetal interface due to schistosomiasis and its associated elevation of pro-inflammatory cytokines has been observed in some studies (Kurtis *et al.,* 2011).

2.1.3 Life Cycle of Schistosomes

Schistosomes have a typical trematode vertebrate-invertebrate life cycle, with snails being the intermediate host and humans being the definitive host (Ross *et al.,* 2002).

2.1.3.1 In Snail

The life cycles of all five human schistosomes (*Schistosoma haematobium, Schistosoma mansoni, Schistosoma japonicum, Schistosoma mekongi* and *Schistosoma intercalatunm*) are broadly similar (WHO, 2011). Schistosome parasite eggs are released into the environment from infected individuals, hatching on contact with fresh water to release the free-swimming miracidium guided by light and chemical stimuli (Negrao-Correa *et al.,* 2007). Miracidia infect fresh-water snails by penetrating the snail's foot (Behrman, 2008). *Biomphalaria* spp including (*B. glabrata, B. pfeifferi, B. alexandrina, B. straminea* etc) are the intermediate host for *S. mansoni* (Morgan *et al.,* 2001) whiles *Bulinus spp (Bulinus globosus* and *Bulinus truncatues)* are the intermediate host for *S. haematobium* (Ayanda 2009, Madsen 2008). After infection, close to the site of penetration, the miracidium transforms into a primary (mother) sporocyst (Behrman, 2008). Germ cells within the primary sporocyst will then begin dividing asexually to produce secondary (daughter) sporocysts, which migrate to the snail's hepatopancreas (CDC 2010). Once in the hepatopancreas, germ cells within the secondary sporocyst begin to divide again, this time producing thousands of new parasites, known as cercariae, which are the larvae capable of infecting humans (Behrman, 2008). Cercariae emerge daily from the snail host in a circadian rhythm, depending on daily ambient temperature and light (Gryseels *et al.,* 2006; McManus *et al.,* 2008). Young cercariae are highly mobile, alternating between vigorous upward movements and sinking to maintain their position in the water (CDC, 2012). Cercarial activity is particularly stimulated by factors such as water turbulence, shadows and chemicals found on human skin (CDC, 2012). Figure 2.1.

2.1.3.2 In Humans

Penetration of the human skin occurs after the infective stage of the parasite which is cercaria have attached to and penetrate the skin (Ross, 2002). The parasite secretes enzymes that break down the skin's protein to enable penetration of the cercarial head through the skin (Behrman, 2008). As the cercaria penetrates the skin it transforms into a migrating schistosomulum stage (WHO, 2013).

The newly transformed schistosomulum may remain in the skin for 2 days after which it enters the host blood circulation as a schistosomula (Walker, 2011) from here the schistosomulum travels to the lungs where it undergoes further developmental changes necessary for subsequent migration to the liver (Gryseels *et al.,* 2006). Eight to ten days after penetration of the skin, the parasite migrates to the liver sinusoids (Gryseels *et al.,* 2006). *S. japonicum* migrates more quickly than *S. mansoni*, and usually reaches the liver within 8 days of penetration. Juvenile *S. mansoni* worms develop an oral sucker after arriving at the liver, and it is during this period that the parasite begins to feed on red blood cells (Inal, 1999). The nearly-mature worms pair; with the longer female worm residing in the gynaecophoric channel of the shorter male (Inal, 1999). Worm pairs of *S. mansoni* relocate to the mesenteric or rectal veins (Abdul-Ghani and Hassan, 2010). *S. haematobium* schistosomula ultimately migrate from the liver to the perivesical venous plexus of the urinary bladder, ureters, and kidneys through the hemorrhoidal plexus (Abdul-
Ghani and Hassan, 2010).

Parasites reach maturity in six to eight weeks, at which time they begin to produce between 20 and 200 eggs daily (CDC, 2009). *Schistosoma haematobium* eggs pass through the ureteral or bladder wall and into the urine (Abdul-Ghani and Hassan, 2010). Only mature eggs are capable of crossing into the digestive tract, possibly through the release of proteolytic enzymes, but also as a function

of host immune response, which fosters local tissue ulceration (Inal, 1999). Up to half the eggs released by the worm pairs become trapped in the mesenteric veins, or will be washed back into the liver, where they will become lodged (Strandgaard *et al.*, 2001). The lifespan of an adult schistosome averages 3-5 years but can be as long as 30 years (Gryseels *et al.*, 2006).

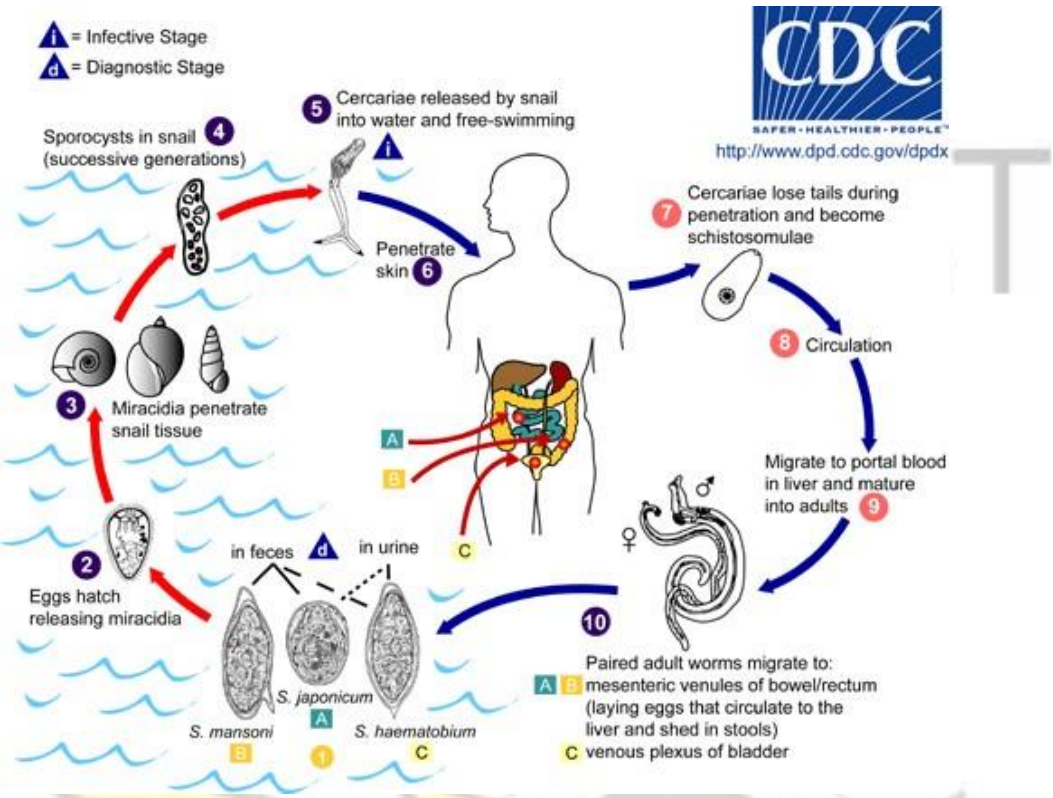

Figure 2.1 Life cycles of *schistosomes*

Source: www.cdc.gov/dpdx/schistosomiasis

2.1.4 Clinical Manifestation of Schistosomes Infection

Within 24 hours of infection an intense irritation and skin rash referred to as "swimmers itch" occur at the site of cercarial penetration (Bottieau et al., 2006). Haematuria caused by eggs penetrating through the bladder wall is a cardinal clinical symptom associated with *Schistosoma haematobium* infection (WHO, 2013). A urinary tract infection is usually characterized by painful urination, damage to the bladder, ureters, and kidneys, and in severe cases can even result in fatal bladder cancer (Chitsulo, 2002). The urogenital form may present with genital lesions (e.g. vulvar nodules), vaginal bleeding, dyspareunia, and fallopian tube damage in females (Ross et al., 2002). Katayama syndrome which is a serum like-sickness do occur as a result of immune complexes which are induced by the products of the worms and egg metabolism as mature female lay eggs

(Ross et al., 2002). Formations of granuloma due to the trapping of eggs in tissue surrounding the bladder can also occur. Prolonged infection coupled with immune response may cause the bladder to thicken (Neal, 2004). Microabcess, bowel lesions such as ulceration and pseudopolyps in the gastrointestinal tract can also be caused by *Schistosoma mansoni* and *Schistosoma japonicum*; these can manifest clinically as blood in stools and abdominal pains (Ross *et al.,* 2002). In advance cases, clinical signs such as spleen enlargement, bleeding-prone esophageal varices have also been documented (Ross *et al.,* 2002).

Anaemia associated with *Schistosomiasis* especially in those with low dietary iron intake in addition to other parasitic infections is common (Cheesbrough, 1999 p. 236). Burden of anaemia is worsened when there is co-infection with geohelminths (Yatich *et al.,* 2009). Damage to seminal vesicles, prostate and other related organs in males may lead to irreversible infertility (WHO, 2010). Neuroschistosomiasis which have been noted in up to 5% of clinically diagnosed cases may be due to eggs, causing space-occupying lesions in the brain and spinal cord (Ross *et al.,* 2002).

2.1.5 Laboratory Diagnosis of Schistosomes

Diagnosis can be established by the detection of eggs in urine (Cheesbrough, 1999 p.236). Occasionally the miracidia of *Schistosoma haematobium* may be found in the urine (Cheesbrough, 1999 p.236). For urogenital schistosomiasis, a filtration technique using nylon, paper or polycarbonate filters is the standard and sensitive method for light infection (WHO, 2010). Kato-Katz method is an inexpensive and simple means of detecting intestinal schistosome in endemic areas (Doenhoff *et al.,* 2004). Serological tests such as Indirect Hemaglutination Assay (IHA) and Circumoval Precipitin Test (COPT) can be used to detect the presence of antibody against different schistosome stages (Yu *et al.,* 2007). Microscopic examination of tissues

obtained by biopsies of rectal mucosa has been used as a diagnostic method (Al-Sherbiny *et al.*, 1999). In cases of liver fibrosis and portal hypertension, the use of sophisticated imaging methods such as ultrasound, computed tomography scan (CT scan) and Magnetic Resonance Imaging (MRI) is required to make definitive diagnosis (Colley *et al.*, 2013). Occasionally there are mixed infections of both *Schistosoma haematobium* and *Schistosoma mansoni* in urine and less often *Schistosoma haematobium* eggs in faeces (Cheesbrough 1999 p.238).

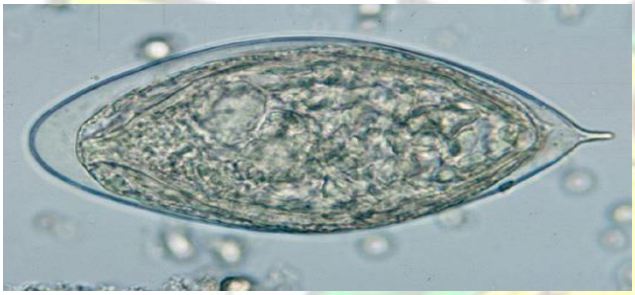

FIGURE 2.2 *S. haematobium* egg (Terminal spine)

Source: Centers of Disease Control and Prevention

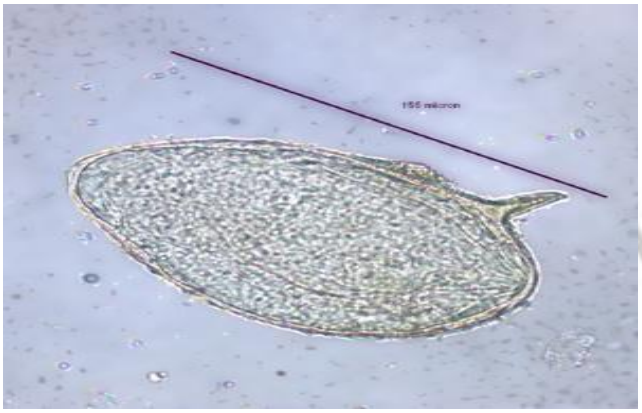

FIGURE 2.3 *S. mansoni* egg (Lateral spine)

Source: www.cdc.gov/dpdx/schistomiasis

2.1.6 Treatment of Schistosome Infection

Strategy for schistosomiasis control focuses on reducing disease through periodic, targeted treatment with single dosage of praziquantel tablet (WHO, 2002).

Groups targeted for treatment are:

- School-aged children in endemic areas;
- Adults considered to be at risk in endemic areas, e.g. pregnant and breastfeeding women, people with occupations involving contact with infested water – such as fishermen,

farmers, irrigation workers – and women whose domestic chores bring them into contact with infested water;

- Entire communities living in endemic areas (WHO 2010)

2.1.7 Prevention and Control of Schistosomes Infection

Prevention and control of schistosomiasis is based on preventive treatment, snail control with spraying and drip feeding, improved sanitation, safe source of water supply and health education (WHO, 2010).

2.2 Soil Transmitted Helminthiasis

2.2.1 Burden of Intestinal Helminth Parasites

Helminth infections are important cause of morbidity and mortality throughout the world, particularly in under developed countries and in persons with co-infection (Chan *et al.,* 1994; De Silva *et al.*, 1997; Guyatt and Bundy, 1991; Anderson and May, 1991). There are about 20 major helminth infections of humans, affecting more than one third of the world's population (Awasthi *et al.,* 2003). The major soil-transmitted helminth (STH) infections, namely ascariasis or roundworm infection (*A. lumbricoides*), trichuriasis or whipworm infection (*T. trichiura*), and hookworms (*N. americanus* and *A. duodenale*) (Table 1) and infection caused by schistosomes

account for most of the global helminth disease burden (Bethony *et al.*, 2006*;* Hotez *et al.*, 2006*;* De Silva *et al.*, 2003).

Table1. Major Soil-Transmitted Helminths (Da Silva *et al.*, 2003)

Parasite	**Disease**	**Prevalence**
Ascaris lumbricoides	Common roundworm infection, (ascariasis)	800 million to 1.4 billion
Trichuris trichiura	Whipworm infection, (trichuriasis)	600 million to 1 billion

Necator americanus and *Ancylostoma duodenale*	Hookworm infection	580 million to 1.2 billion
Strongyloides stercoralis	Threadworm infection, (strongyloidiasis)	30-300 million
Enterobius vermicularis	Pinworm infection	4 -28% of children

The global picture of the STHs shows widespread prevalences throughout sub-Saharan Africa, the Americas, India, China and East Asia (De Silva *et al.*, 2003). An estimated 4.5 billion individuals are at risk of the STHs. Two billion people are infected and more than 135,000 deaths have been reported to occur worldwide annually (De Silva et al., 2003).

2.2.2 Epidemiological Patterns of Helminthic Infections

Soil-transmitted helminth (STH) infections are common in communities with poor environment sanitation, often due to lack of adequate latrines (Kightlinger *et al.*, 1998; Appleton *et al.*, 1999; Beasley *et al.*, 2000).

Children as young as 6 months can be infected with helminths (Goodman *et al.*, 2007) which influence their nutritional status causing growth retardation (Adams *et al.*, 1994) and reduce learning ability with increased absenteeism from school (Beasley *et al.*, 2000; Nokes *et al.*, 1992) Women of childbearing age are also vulnerable to STH infections (Goodman *et al.*, 2007). *A. lumbricoides* and *T. trichiura* infections, convex age-intensity profiles, in which the incidence

peaks in childhood, are observed (Woolhouse, 1988). Infection prevalence and intensities rise with increasing age to a peak around 5 - 10 years old and decline in adults (Bachta *et al.,* 1990)

Unlike other STHs, hookworm infection appears to increase throughout childhood until it reaches a plateau in adulthood with highest prevalence occurring among the middle-aged or even individuals over the age of 60 years (Hotez, 2001; Behnke *et al.,* 2000; Brooker *et al.,* 2004) Strongyloidiasis has been shown to occur in all age groups, although acquisition is more common during childhood and has no predilection for either sex (Pearson, 2002).

On sex-dependency, helminth infections have been reported in some cases to infect males more than females (Anderson and May, 1991). Hookworm shows significantly higher prevalence in males than females (Behnke *et al.,* 2000) but prevalences of *A. lumbricoides* and *T. trichiura* show no significant difference between males and females (Guyatt and Bundy, 1991). Generally, intestinal helminthes infection could result in perinatal mortality, low birth weight and intrauterine growth retardation (Stephenson *et al.,* 2002; Khor, 2003).

2.2.3 Clinical Indications of Intestinal Helminth Infections

Majority of intestinal helminth infections are chronic and mild, and are usually asymptomatic or subclinical (Udonsi, 1984). However, several clinical signs and symptoms can occur in patients with moderate and heavy infections (Neva and Brown, 1994).

During the first 1-2 weeks after a cutaneous infection, helminthiasis can produce an intensely pruritic dermatitis at the site of infection termed ground itch, and larval invasion of the lungs may produce respiratory symptoms called Löeffler or Löeffler-like syndrome (Neva and Brown, 1994). This syndrome is characterized by pneumonitis which can be accompanied by paroxysmal attacks of cough, coughing with blood-tinged sputum, wheezing, dyspnea, pleurisy, low grade fever,

substernal pain, urticaria, asthma and eosinophilia (Coombs and Crompton, 1991). Adult worms in the intestine commonly cause abdominal pain (Spurchler, 1987). Other enteric symptoms reported include abdominal cramps/colic, intestinal blockage, nausea and/or vomiting (rarely), tenesmus, diarrhoea, constipation (occasionally) and dysentery (Spurchler, 1987; Vadlamudi *et al.,* 2006). Commonly observed complications in heavy helminthiasis include situations in which large and tangled worms of *Ascaris lumbricoides* may cause intestinal (usually ileal), common duct, pancreatic, or appendiceal obstruction (De Silva *et al.,* 1997). Less common features include ascending cholangitis, acute pancreatitis, and, rarely, obstructive jaundice (Bahu *et al.,* 2001; Khuroo, 2001).

In heavy *trichuriasis*, infected people may show mild anaemia, eosinophilia, bloody diarrhea (classic *Trichuris* dysentery syndrome, or chronic *Trichuris colitis*), prolapsed rectum (especially in children), and impaired physical and mental growth (Drake *et al.,* 2000).

The major manifestation of hookworm disease is iron deficiency anaemia, and patients with severe anaemia may have fatigue, syncope, or exertional dyspnea (Stoltzfus *et al.,* 1997; Albonico *et al.,* 1998). *Strongyloides stercoralis* infection produces burning or colicky abdominal pain, often epigastric, and is associated with diarrhea and the passage of mucus (Oduntan, 1974; Udonsi, 1984; Adams *et al.,* 1994; Drake *et al.,* 2000**)**. Non-specific clinical manifestations include restlessness, irritability, anorexia, chronic protein energy malnutrition, malabsorption, anasarca and weight loss (Spurchler, 1987).

2.3 *Ascaris lumbricoides*

2.3.1 Ascariasis

Ascariasis is one of the major causes of helminth diseases in developing countries (Ryan and Ray, 2004). The evidence of effectiveness of public health interventions to relieve its disease burden is worth attention (Adams *et al*, 2006). Ascariasis is an infection caused by *Ascaris lumbricoides*, the largest intestinal roundworm, (Berger and Marr, 2006). It lives in the small intestine and the female produces up to 200,000 eggs which pass with feces daily (Cheesbrough, 1992). It is one of the commonest nematode infestations of the small intestine (Cheesbrough, 1992). It does not appear to depend so much on the climate, although it is more perennial in the damp and humid areas of the tropics (Neva and Brown, 1994). This explains why ascariasis is common in all areas of Africa (Cheesbrough, 1992).

2.3.2 Epidemiology of *Ascaris lumbricoides*

Ascaris lumbricoides infects an estimated population of about 1.3 billion people worldwide and is a major cause of disease burden especially in developing countries, with an estimated loss of from 1.2 to 10.5 disability-adjusted life year per infected person (Bethony *et al*., 2006; Ryan and Ray, 2004). In 2002, WHO estimated that there were 1450 million persons infected with *A.lumbricoides* and annually 60000 deaths are recorded annually from ascariasis (Cheesbrough, 2005). Heavy *Ascaris* infections occur in children of 3 - 8years whose fingers become contaminated while playing on open ground (Cheesbrough, 2005). *Ascaris lumbricoides* can play a role in precipitating protein-energy malnutrition which may have adverse effect on pregnancy (Ananthakrishnan *et al.,* 1997). Pregnant women infected with *Ascaris* have shown lower levels of haemoglobin with marked eosinophilia (Alfonso *et al.,* 2006).

2.3.3 Life Cycle of *Ascaris lumbricoides*

Infection with the *Ascaris* parasite results when a person ingests contaminated food, water or soil containing eggs of this parasite (Walker *et. al.*, 2011). The eggs have to be embryonated in soil before they are infective for a period of 8-50 days (Peng *et. al.*, 2003). The soil must be loose, not too dry and oxygen must be available and the temperatures over 15 °C (Ryan and Ray, 2004). The embryonated eggs hatch and migrate to the small intestine (Cheesbrough, 1992) and the eggs can also pass through the gastrointestinal tract of animals and remain infective (Ryan and Ray, 2004). The other vehicle of transmission can be when fruit or other vegetables are eaten raw (Ryan and Ray, 2004). Unwashed hands and children picking up things from the floor or ground and putting them into the mouth are common ways of acquiring *Ascaris* (Ryan and Ray, 2004). In communities that use human faeces as organic manure, the possibility of ingesting the eggs from foodstuffs grown using this manure is very high (Walker *et. al.*, 2011). This is especially after consumption of vegetables which are sometimes eaten raw or half cooked (Geissler *et, al.*, 1998). Eggs resistant severe adverse environmental conditions and embryonated eggs can be carried away from the contaminated place into houses by feet, footwear or in the dust by wind (Ryan and Ray, 2004). Temperatures above 60°C are necessary to destroy the eggs (Cheesbrough, 1992).

To reach maturity the larvae need to pass through the lungs (Guyatt *et a.*, 1995). The larvae penetrate the intestinal wall and reach the liver via the portal system (Cheesbrough, 2005). From the liver they are carried in the blood through the right side of the heart into the lungs (Guyatt et al,. 1995). Here they penetrate into the airways and pass via the bronchiole, bronchi and trachea to the pharynx (Cheesbrough, 1992). Then they are coughed up and swallowed, return to the

gastrointestinal tract and settle in the jejunum where they develop into adult worms (Cheesbrough, 1992). During the lung phase, eosinophilia develops (Guyatt *et al.,* 1995). This eosinophilia is temporal if no new infestation occurs (Ryan and Ray, 2004). The migration phase may be associated with fever, cough, wheezing, shortness of breath and allergic dermatitis (Ryan and Ray, 2004). Lung migration may also cause pneumonia (Cheesbrough, 1992). The life span of an adult worm is about 1 to 2 years (Guyatt *et al.,* 1995).Figure 2.4.

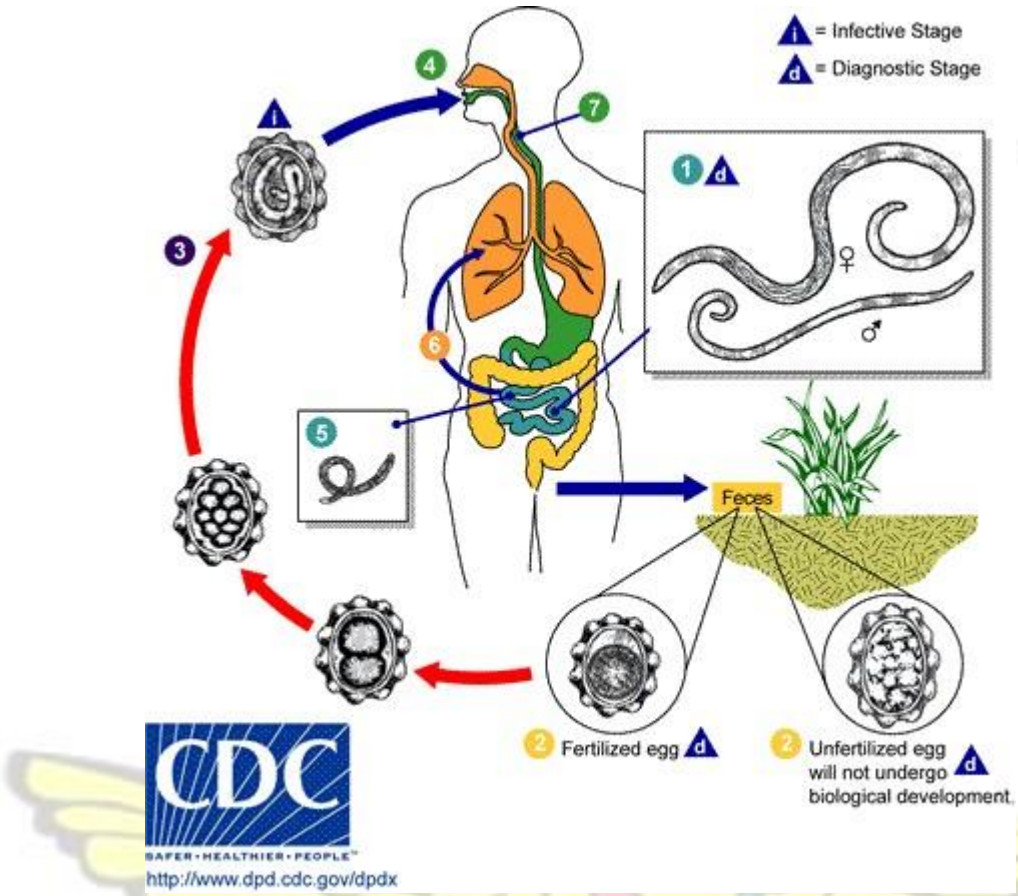

Figure 2.4 Life cycle of *Ascaris lumbricoides*

Source: www.cdc.gov/dpdx/ascariasis

2.3.4 Clinical Manisfestation of *Ascaris lumbricoides*

Except for the temporary symptoms experienced during larval migration through the lungs, infection with a few *Ascaris* is usually asymptomatic or if symptoms are present, they are not characteristic of *Ascaris* infection (Ryan and Ray, 2004). There may be vague abdominal discomfort (Ray and Ryan, 2004). Occasionally a worm may leave the body (in vomitus or stools) upsetting the patient and the family (Cheesbrough, 1992). Complications may occur in very heavy infections or due to wandering worms (Ryan and Ray, 2004). In some instances young *Ascaris*

larvae can migrate to the lungs via hepatic blood vessels to cause tissue destruction and if their numbers are large,it may lead to the enlargement and tenderness of the liver which may be short-lived. This hepatitis is usually short-lived (Ryan and Ray, 2004; Guyatt *et al.*, 1995). The diagnosis cannot be established until a few weeks later when the worms are mature and their eggs can be found in the host's faeces (Cheesbrough, 2005).

Intestinal obstructions may occur at the illio-caecal junction by a large ball of worms (Cheesbrough, 2005). Wandering worms may be provoked by tetra-chloroethylene, a drug used previously for hookworm treatment (Ryan and Ray, 2004). Wandering *Ascaris* may reach abnormal foci and cause acute symptoms (Ryan and Ray, 2004). For instance, if a person vomits worms, this may cause swelling of the glottis and larynx resulting in difficulties in breathing (Ryan and Ray, 2004). The wandering worms can also cause blockage of bile ducts resulting in obstructive jaundice (Cheesbrough, 2005). The worms can migrate into the liver tissue resulting in the formation of a liver abscess (Cheesbrough, 1992). The worms feed on the nutrients consumed by the host (Cheesbrough, 1992). Ascariasis may contribute to malnutrition states such as kwashiorkor and vitamin A deficiency (Cheesbrough, 2005).

2.3.5 Laboratory Diagnosis of *Ascaris lumbricoides*

Diagnosis is by stool microscopy which should show the characteristic *Ascaris* eggs (Cheesbrough, 2005). During the early lung phase, when eosinophilic pneumonia occurs, larvae can be found in sputum or gastric aspirates before diagnostic eggs appear in the stool (Ryan and Ray, 2004). A plain abdominal radiograph may reveal masses of worms in gas filled loops of bowel in patients with intestinal obstruction (Ryan and Ray, 2004). Magnetic Resonance Cholangiopancreatography

(MRCP) is an alternative to abdominal ultrasonography when it is not feasible, as is the case in some pregnant women (Arya *et al.,* 2005).

2.3.6 Treatment of *Ascaris lumbricoides*

Ascariasis should always be treated to prevent potentially serious complications (Ryan and Ray, 2004). Both Mebendazole and Albendazole are effective, but are contraindicated in pregnancy and in heavy infections in which they may provoke ectopic migration (Ryan and Ray, 2004). Piperazine is safe in pregnancy (Ryan and Ray, 2004). Mebendazole is the drug most commonly used because it is a broad-spectrum antihelminthic (Steinmann *et al*., 2008). It is given in a dose of 100 mg twice daily for 3 days. Alternatively, Levamisole at 5mg/kg can be given as a single dose or Albendazole 400 mg start (Steinmann *et al*., 2008). Piperazine citrate 150mg/kg can be given as a single dose and should not exceed 4g. Pyrantel pamoate is also highly effective (Steinmann *et al*., 2008).

2.4 HOOKWORM

2.4.1 HOOKWORM DISEASE

Hookworm infection in humans is caused by two types of worms: *Necator americanus* and *Ancylostoma duodenale* (Hotez *et al.,* 2004). The two species overlap in many tropical regions (Ryan and Ray, 2004). In most areas, incidence and intensity of hookworm infection increase with age (Ye *et. al.,* 1994). Hookworms need a hot humid climate for their development (Cheesbrough,

1992). A minimum temperature of 18°C is required and a soil temperature of 2032°C is optimal (Ryan and Ray, 2004). Hookworm infection is therefore, most common in the hot, humid areas of Africa (Brooker *et. al.*, 2002).

Infection with hookworm disease may vary from asymptomatic infection to a chronic debilitating disease caused by severe iron deficiency anaemia, and in some cases loss of protein from the bowel leading to oedema (John *et. al.*, 2006). Heavy worm burden, a prolonged duration of infection and poor iron intake all contribute to the development of hookworm disease (Cheesbrough, 1992). Many people (hookworm carriers) harbour the worms without any ill effects (Ryan and Ray, 2004). Nutrition, daily iron intake and total worm burden determine whether a carrier develops anaemia (Bethony *et. al.*, 2006).

2.4.2 Epidemiology of Hookworm

An estimated population of 740 million is affected (Hotez *et. al.*, 2004). *N. americanus* is the common hookworm infecting man in the Far East, South Asia, Pacific Islands, Tropical Africa, Central and South America (Cheesbrough, 1992). *A. duodenale* is found in the Middle East, in countries around the Mediterranean, and North China but can also be found with *N. americanus* in Africa, South East Asia, the Pacific Islands and South America (Cheesbrough, 1992). As of 1990, an estimated 7% of the world's pre-schoolchildren (41 million), 26% of school age children (239million), and 44.3 million of the developing world are infected with Hookworm (WHO, 1996 and Michael *et al.*, 1997). One hundred and twenty four point three million pregnant women harbored hookworm infection (Holland and Kennedy, 2002). Hookworm

infections induce deficiencies of iron, total energy, protein, folate and zinc due to gastrointestinal blood loss, malabsorption and appetite inhibition may aggravate anaemia in pregnancy (Nurdia *et*

al., 2001; Stephenson *et al.*, 2002). Studies have shown that hookworm infection could result in higher perinatal mortality rates, low birth weight (LBW) and intrauterine growth retardation (IUGR) in pregnant women (Stephenson *et al*., 2002; Allen 2001; Khor,2003).

2.4.3 Life Cycle of Hookworm

The eggs are already embryonated when passed out with faeces (Hawdon and Hotez, 1996) Figure 2.5. The eggs hatch into *rhabditiform* larvae which leave the faeces and bury themselves in moist damp soil where they change into the infective sheathed *filariform* stage (Kayser *et al*., 2001). The development from the non-infective *rhabditiform* larvae to the infective *filariform* stage occurs over a period of 1-week (Cheesbrough, 1992). The *filariform* larvae may attach themselves to grass or hide in the soil (Ryan and Ray, 2004).

An infective *filariform* larva then penetrates the skin and reaches the lungs through the venous system and the right side of the heart (Hotez, 2005). There, they invade the alveoli and ascend the airways before being swallowed back and reaching the small intestine 3-5 days after penetrating the skin (Kayser *et al*., 2001). The larvae develop into adults in the small intestine and stay attached onto the mucosa by hooks in their buccal cavity (Cheesbrogh, 1992). The period from skin penetration to appearance of eggs in the faeces is about 6 to 8 weeks, but it may be longer with *Ancylostoma duodenale* (Ryan and Ray, 2004). Larvae of *Ancylostoma duodenale*, if swallowed, can survive and develop directly in the intestinal mucosa into adults (Cheesbrough, 1992). Adult hookworms may survive over 10 years but usually live 6 to 8 years for *A.duodenale* and 2 to 5 years for *Necator americanus* (Ryan and Ray, 2004).

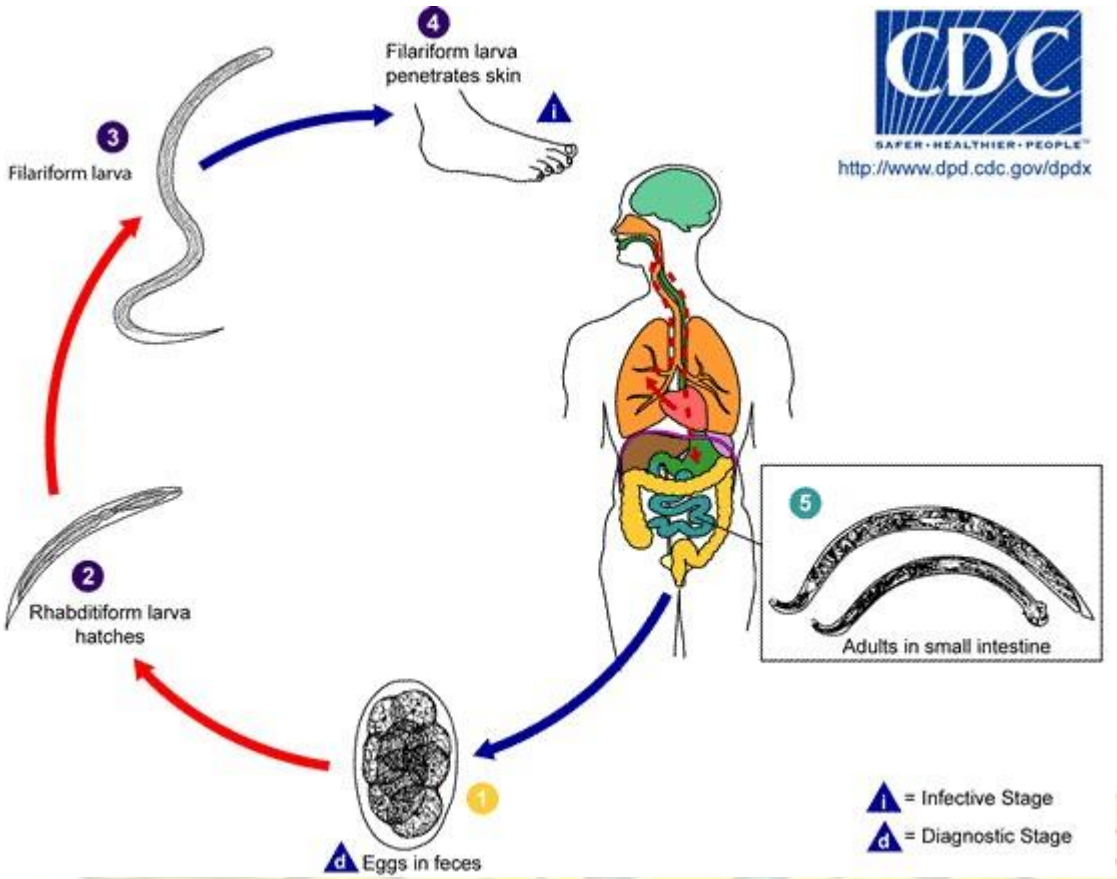

Figure 2.5: Life cycle of Hookworm

Source: www.cdc.gov/dpdx/hookworm

2.4.4 Pathogenesis and Clinical Manisfestation of Hookworm

Hookworm infestation is asymptomatic in the vast majority of cases (Ryan and Ray, 2004). Infective larvae may provoke an itch "ground itch" at the site of skin penetration (Markell et. al., 2006). Itchy erythematous papules appear at the site as well as tracts of subcutaneous migration (similar to *cutaneous larva migrans*) (Blackwell and Vega-Lopez, 2001). This is most common between the toes and on the sole of the feet (Holland and Kennedy, 2002). The lung passage is also affected, and there may be some coughing, wheezing, eosinophilia and occasionally mild transient pneumonia, but this condition develops less frequently in hookworm infection than in ascariasis (Cheesbrough, 1992).

In the digestive tract, there is dyspepsia, abdominal pain, distension, sometimes diarrhoea (Cheesbrough, 1992). In heavy infections, diarrhoea is mixed with blood (Holland and Kennedy, 2002). The symptoms may be mistaken for those of duodenal or gastric ulcers (Ryan and Ray, 2004). Iron deficiency anaemia however, develops when a heavy hookworm load overtaxes the iron reserves (Holland and Kennedy, 2002). The hookworm suck blood from the intestines and this leads to loss of red blood cells, iron and protein, especially albumin, from the host (Bethony, 2006). When the host's iron stores are depleted, iron deficiency anaemia sets in and symptoms related to anaemia appear (Holland and Kennedy, 2002). Symptoms are minimal if iron intake is adequate (Ryan and Ray, 2004). The evolution of this anaemia is slow and because of the physiological adjustments it evokes, the patient can continue to be up and about with a surprisingly low haemoglobin level, the so called "walking anaemia of hookworm" (Ryan and Ray, 2004).

2.4.5 Laboratory Diagnosis of Hookworm

Diagnosis is established by the finding of the typical oval hookworm eggs in the stool in wet mount (Markell *et al.*, 2000). Differentiation of the two species from the eggs is not possible, but the adult worms can be distinguished (Ukaga *et. al.*, 2002). Stool concentration techniques may be required to detect light infections (Cheesbrough, 1992). In a stool sample that is not fresh, the eggs may hatch to release *rhabdititform* larvae which need to be differentiated from those of *Strongyloides* (Ryan and Ray, 2004). Features of iron deficiency anaemia (hypochromic microcytic anaemia), occasionally with eosinophilia or hypoalbuminemia are common in blood profile (Holland and Kennedy, 2002).

2.4.6 Treatment of Hookworm

Re-infection is very likely if the community as a whole does not improve its methods of faecal disposal (Ryan and Ray, 2004). Where anaemia is present, treatment should aim at eliminating the worms and correcting the anaemia (Markell *et al*, 2006). Iron deficiency anaemia is treated with oral iron for at least 3 months (Holland and Kennedy, 2002). A high protein diet is necessary to replace protein loss (Holland and Kennedy, 2002). Even when the anaemia is severe, patients respond quickly and well to iron therapy (Holland and Kennedy, 2002). Folate deficiency may occur as a result of increased bone marrow activity when the iron deficiency is being corrected (Holland and Kennedy, 2002).

According to Steinmann *et al*. (2008**)** hookworms can be eradicated by the use of several safe and highly effective antihelminthic drugs including:

- Mebendazole 100mg twice daily for 3 days;
- Pyrantel Pamoate 10mg/kg body weight daily for 3 days;

- Levamisole 3 tablets stat. This can be used in mixed infections. The disadvantage with it is that it is expensive and not very effective against *Necator americanus*, the more common hookworm;
- Bephenium – This drug is more expensive than Levamisole and less effective with *Necator americanus.*

2.4.7 PREVENTION

Prevention of hookworm infection includes;

- Avoid the use of human excreta or raw sewage or untreated night soil as manure/fertilizer in agriculture should be sterilized first
- Avoid walking barefooted in known infected areas
- Avoid open-defecation in places other than latrines and toilets
- Provision of effective sewage disposal systems and proper and adequate public and private latrines

2.5 *Strongyloides stercoralis*

2.5.1 Strongyloidiasis

Strongyloidiasis is an infection caused by *Strongyloides stercoralis*, a nematode which is distinguished by its ability to replicate in the human host (Glinz *et. al.*, 2010). This unusual behaviour enables ongoing cycles of autoinfection due to the internal production of infective larvae (Grove, 1996). The infection can persist for ages without further exposure of the host to external infective larvae in immune-suppress and HIV/AIDS patients (Cheesbrough, 2005).

Strongyloides stercoralis is distributed in tropical areas and other hot humid regions and is particularly common in sub-Sahara Africa (Peters and Pasvol 2005).

The adult female worms live in the mucosa of the duodenum and jejunum (Cheesbrough, 2005). Most infections are asymptomatic (Cheesbrough, 1992). *Strongyloides* infection may remain quiescent for many years and become reactivated during immunosuppressive chemotherapy or when the host is immunocompromised (Ryan and Ray, 2004). This can cause severe and even fatal disease (Vadlamudi and Krishnaswamy, 2006).

S. stercoralis has been shown to be endemic in developing countries, where it is associated with situations that lead to immunodeficiency including HIV infections (Gomez *et al*., 1995). Men have a greater chance of being and/or re-infected by having sodomy relations with partners of both sexes (Dias *et al*., 1992).

2.5.2 Epidemiology of *Strongyloides stercoralis*

It is known that tens of millions of people are infected with *Strongyloides* worldwide (Keiser and Nutman, 2004) and mostly found in the Tropical and Sub tropical region but cases have been reported in the temperate zones (Walzer *et al*., 1982). Occupations that increase contact with soil contaminated waste which may include farming and coal mining depending on local practices, increase the risk of infections (Walzer *et al*., 1982). Different prevalences among ethnic groups may simply reflect behavioral or socioeconomic factors but some have suggested that different skin types may be more or less resistant to larva penetration (Keiser and Nutman, 2004).

Hyperinfection syndrome of *Strongyloides* has a mortality rate ranging from 15% to as high as 87% (Marcos *et al*., 2008).

2.5.3 Life Cycle of *Strongyloides stercoralis*

Eggs hatch in the small intestinal mucosa, releasing rhabditiform larvae that migrate to the lumen and pass with the stool into the soil (Mansfield *et al.*, 1996) Figure 2.6. *Rhabditiform* larvae in the bowel lumen can also develop directly into *filariform* larvae that penetrate the colonic wall or perianal skin and enter the circulation to repeat the migration that establishes ongoing internal reinfection (Ramdial *et al.*, 2006). This autoinfection cycle allows the infection to persist long after the host has left the endemic area (Ryan and Ray, 2004).

Rhabditiform larvae passed onto the soil with faeces transform into infective *filariform* larvae directly which penetrates the skin or may develop into free-living adults which continue to reproduce outside the body (Cheesbrough, 2005). The larvae then travel through the venous circulation to the lungs where they break into the alveolar spaces, ascend the bronchial tree, are coughed up and swallowed and thereby reach the small intestines (Segarra-Newnham, 2007). Here the larva matures into the adult worm that penetrates the mucosa and they can then hatch eggs to perpetuate the cycle of infection (Cheesbrough, 2005).

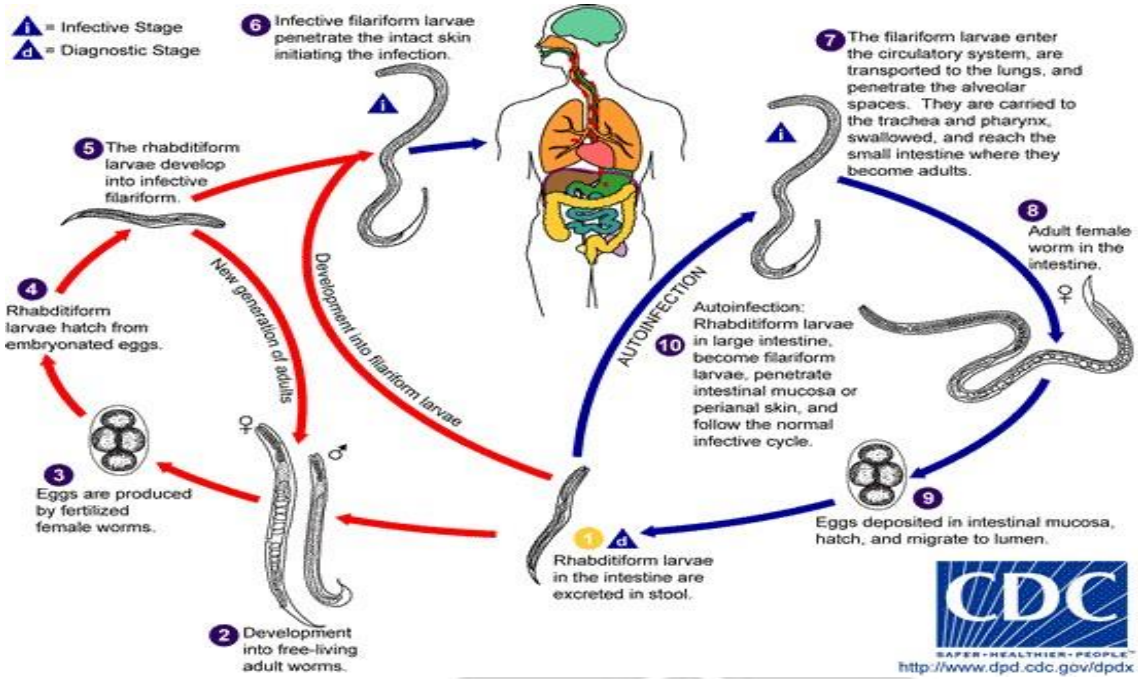

Figure 2.6: Life cycle of *Strongyloides stercoralis*

Source: www.cdc.gov/dpdx/strongyloidiasis

2.5.4 Clinical Manifestation of *Strongyloides stercoralis*

Usually the strongyloidiasis infection is asymptomatic, or has mild cutaneous and/or abdominal symptoms (Segarra-Newnham, 2007). Recurrent urticaria, often involving the buttocks and wrists is the most common cutaneous manifestation (Liu and Weller, 1993). Migrating larvae can elicit a pruritic raised erythematous lesion that advances rapidly along the course of larval migration (Ryan and Ray, 2004). This is known as l*arva currens* (running larva) (Ryan and Ray, 2004). Pulmonary symptoms are rare in light infections (Ruy *et. al.,* 2010). Adult worms residing in the small intestine mucosa can cause abdominal pain which is worsened with ingestion of food (Ryan and Ray, 2004). Nausea, diarrhoea, gastrointestinal bleeding and weight loss can occur (Segarra-Newnham, 2007). In disseminated infection, apart from the gastrointestinal and lung tissues, larvae may also invade the central nervous system, peritoneum, liver and kidney (Cheesbrough, 2005). Bacteremia may also develop due to the entry of enteric flora through disrupted mucosal barriers (Grove, 1996). In immunocompromised patients, transformation to filariform stage occurs within the gut itself, producing marked autoinfections and hyperinfection (Ryan and Ray, 2004)

2.5.5 Laboratory Diagnosis of *Strongyloides stercoralis*

Diagnosis is by observing the larvae in a fresh stool specimen. The eggs are almost never detectable because they hatch in the intestines (Cheesbrough, 2005). Single stool examination can help detect about one-third of uncomplicated infections, in which there are usually few larvae passed (Ryan and Ray, 2004). Serial stool examination is thus encouraged or advocated (Cheesbrough, 1992). An ELISA for antibodies to excretory-secretory antigens of *Strongyloides* is a sensitive method of diagnosing uncomplicated infections (Lindo, 1994).

2.5.6 Prevention and Treatment of *Strongyloides stercoralis*

Even in the asymptomatic state, treatment must be given because of the potential for fatal hyper infection (Steinmann *et al*., 2008). The drugs of choice are Mebendazole 100mg twice daily for 3 days or Thiabendazole 25mg/kg body weight twice daily for 2 days (Steinmann *et al*., 2008). However, in disseminated disease, treatment should be extended for 5-7 days (Steinmann *et al*., 2008). Albendazole 400mg/kg for 3 days is also effective. Levamisole is only effective in about 50% of the cases and is therefore not the drug of choice (Steinmann *et al*., 2008).

2.6 *Trichiuris trichura*

2.6.1 Trichuriasis

The adult whipworm is 30 to 50 mm in length (Ryan and Ray, 2004). The anterior two thirds is thin and threadlike, whereas the posterior end is bulbulous, giving the worm the appearance of a tiny whip (Baron, 1996). The tail of the male is coiled; that of the female is straight (Garcia, 2007). The female produces 3000 to 10000 oval eggs each a day (Ryan and Ray, 2004). They are of the same size as pinworm eggs but have a distinctive thick brown shell with translucent knobs on both ends (Garcia, 2007).

2.6.2 Epidemiology of *Trichiuris trichura*

Although it is less widespread than pinworm, the worm is cosmopolitan parasite, infecting approximately 500 million people throughout the world (Hunter and Mckay, 2004). It is concentrated in areas where indiscriminate defaecations in humid environments support the development of the larvae (Donkor, 2009). In tropical climates, infection rates may be as high as

80% (Ryan and Ray, 2004). Though the intensity of infection is generally low, adult worms may live 4 to 8 years (Ryan and Ray, 2004).

Attachment of the worms to the colonic mucosa and their subsequent feeding activities produce localized ulceration and haemorrhage (0.005ml blood per worm per day) (Baron, 1996). The ulcers provide enteric bacteria with a portal of entry to the blood stream, and occasionally a sustained bacteremia results (Ryan and Ray, 2004). A decrease in the prevalence of *trichuriasis* in the post-adolescent period and demonstration of acquired immunity in experimental animal infections suggest that immunity may develop in naturally occurred human infections (Ryan and Ray, 2004). An IgE-mediated immune mucosal response is demonstrated in humans, but is insufficient to cause appreciable parasite expulsion **(**Ryan and Ray, 2004).

Heavy infection of *Trichuris trichiura* may be associated with decreased food intake and blood loss which can result in anaemia in pregnancy (Ramdath *et al.,* 1995; Robertson *et al.*, 1992). The cause of anaemia by *Trichuris trichiura* infection during pregnancy can be described as additive (Ndomugyenyi *et al.,* 2002) and can worsen in pregnancy by having adverse birth outcomes (Nelly *et al.,* 2009).

2.6.3 Life Cycle of *Trichiuris trichura*

Trichuris trichura has a life cycle that differs from that of the pinworm only in its external phase (Ryan and Ray, 2004) Figure 2.7. The adults live attached to the colonic mucosa by their thin anterior end (Garcia, 2007). While retaining its position in the caecum, the gravid female releases its eggs into the lumen of the human gut (Ryan and Ray, 2004). These pass out of the body with the faeces and, in poorly sanitized areas of the world; they are deposited on soil (Ryan and Ray, 2004). The eggs are immature at the time of passage and must incubate for at least 10 days

before they become fully embryonated and infectious (Ekpo *et. al.*, 2008). Once mature, they are picked up on the hands of children at play or of agricultural workers and passed to the mouth (Ryan and Ray, 2004). In areas where human faeces are used as organic fertilizer, raw fruits and vegetables may be contaminated and later ingested (Ryan and Ray, 2004). Following ingestion, the eggs hatch in the duodenum, and the released larvae mature for approximately one month in the small bowel before migrating to their adult habitat in the caecum (Donkor, 2009).

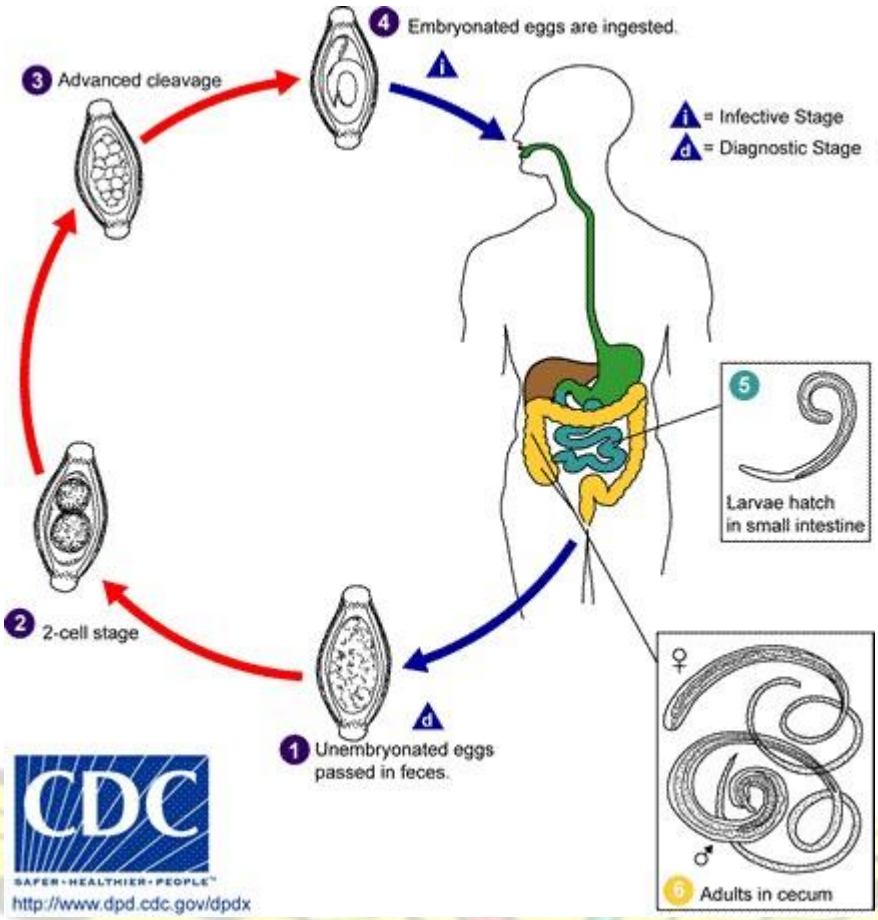

Figure 2.7: Life cycle of *Trichuris trichiura*

Source: www.cdc.gov/dpdx/trichuriasis

2.6.4 Clinical Manifestations of *Trichiuris trichura*

Light infections are often asymptomatic, however, moderate worm loads can damage to the intestinal mucosa by inducing nausea, abdominal pain, bloody mucoid diarrhoea, and stunting growth (Baron, 1996; Ryan and Ray, 2004). Occasionally, a child may harbor 800 worms or more and in these situations, the entire colonic mucosa is parasitized, with significant mucosal damage, blood loss, and anaemia (Ryan and Ray, 2004). The sheer force of the faecal stream on the bodies of the worms may produce prolapsed of the colonic or rectal mucosa through the anus, particularly when the host is straining at defaecation or during child birth (Elliot, 2006).

2.6.5 Laboratory Diagnosis of *Trichiuris trichura*

In light infections, stool concentration methods may be required to recover the eggs (Levinson, 2008). Such procedures are almost never necessary in symptomatic infections, as they inevitably produce more than 10000 eggs per gram of faeces, a density readily detected by examining 1 to 2 mg of emulsified stool with the lower-power lens of a microscope (Ryan and Ray, 2004). A moderate eosinophilia is common in such infections (Baron, 1996). Rectal prolapsed can be diagnosed easily using defecating proctogram and is one of many methods for imaging parasitic infection (Hunter *et al.*, 2004).

2.6.6 Treatment of *Trichiuris trichura*

Infections should not be treated unless they are symptomatic (Ryan and Ray, 2004), and mebendazole and albendazole are the drugs of choice (Hall and Nahar, 1994). Although the cure rate is only 60 to 70%, more than 90% of the adult worms are usually expelled, rendering the patient asymptomatic (Ryan and Ray, 2004).

2.6.7 Prevention of *Trichiuris trichura*

Infection can be avoided by proper disposal of human faeces, avoiding fecal contamination of food, not eating dirt, and avoiding crops fertilized with human faeces.

Simple and effective proper hygiene such as washing hands and fruits are recommended for control.

2.7 *Taenia species (T. saginata* and *T. solium)*

2.7.1 Taeniasis

Human taeniasis is parasitic infection caused by the three tapeworms species namely *Taenia solium* (pork tapeworm), *Taenia saginata* (beef tapeworm) and *Taenia asiatica* (Asian tapeworm) (Cheesbrough, 2005). They are relatively common in Africa, some parts of Eastern Europe, Southeast Asia and Latin America (Carabin *et al.*, 2009). Humans are the only definitive hosts for *Taenia species* and become infected by ingesting raw or undercooked infected beef or pork (Ukaga *et al.*, 2002). Both species are worldwide in distribution with *T. solium*, more prevalent in poorer communities where humans live in close contact with pigs and eat undercooked pork (Cox, 1998).

2.7.2 Epidemiology of *Taenia species*

Approximately 50 million people worldwide are infected with *Taenia spp.*and about 80% of those infected live in low-income countries (Sarti *et al.*, 1992)

Taenia solium are more prevalent in some under-developed parts of the world such as subSaharan Africa, India, Latin America and Eastern Europe among people with poor sanitation and where people consume raw or under cooked pork (Garcia and Del, 2000)

Taenia saginata infection occurs when contaminated raw beef is eaten (Cheesbrough, 2005). Infection is commonly found in Russia, Latin America and eastern Africa.

Studies on intestinal helminthic infections in Ghana have indicated relatively low prevalence of Taenia species infection (Annan et al., 1986; PCD, 1998).

2.7.3 Life Cycle of *Taenia species*

Humans are the definitive host for *Taenia spp.* Cattle and pigs become infected after feeding on vegetation contaminated with eggs or gravid proglottids (Carpio, 2002) Figure 2.8. Oncospheres from the eggs hatch within the intestines of infected animals and invade the intestinal wall and further migrate to the striated muscles and eventually develop into cysticerci. Survival of a cysticercus can take several years in infected animal (Cheesbrough, 2005).

Humans become infected by ingesting raw or undercooked infected meat (Cheesbrough, 2005, Garcia and Del, 2000). Cysticercus develops in the human intestine and this can be up to two months to reach adult tapeworm which can survive for several years (Allan *et al.*, 1996). Adult tapeworms attach to the small intestine with the aid of scolex (CDC, 2002). Length of adult worm is usually 5 meters or less for *Taenia saginata* and 2 - 7 meters for *Taenia solium* (Garcia *et al.*, 2003). The adults produce proglottids which mature to become gravid and eventually detach from the tapeworm. It migrates to the anus or passed in stool (Spurchler, 1987). *Taenia solium* adults have an average of 1000 proglottids whiles *Taenia saginata* have between 1000 – 2000 proglottids (CDC 2002). Finally, eggs in the gravid proglottids are released after the proglottids are passed with the faeces. *Taenia solium* may produce up to 50,000 eggs and *Taenia saginata* may produce 100,000 eggs per proglottid.

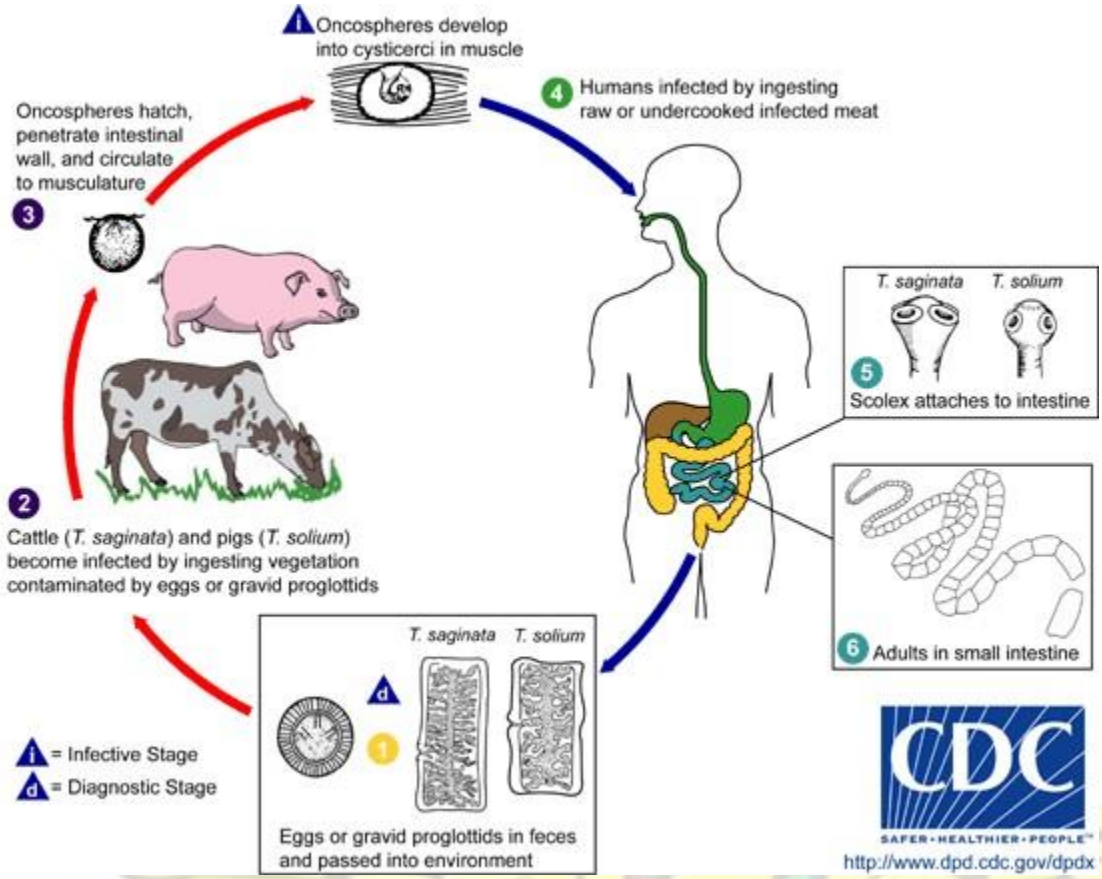

FIGURE 2.8: LIFE CYCLE OF *TAENIA SPECIES*

SOURCE: www.cdc.gov/dpdx/taeniasis

2.7.4 Clinical Manifestation of *Taenia species*

Infection with the adult stage of the tapeworm which is acquired by eating raw or undercooked meat contaminated by the larval stage (cysticerci) may be asymptomatic in many carriers (Flisser *et al.,* 2011, Ito *et al.,* 2003).

Infection with *Taenia saginata* is more symptomatic compared to *Taenia solium* due to the large size of *Taenia saginata* thus up to 10 meters and *Taenia solium* 3 meters. Abdominal pains, loss of appetite, weight loss and stomach upset are some of the digestive problems *Taenia spp.* can cause to infected individuals. *Taenia solium* infection can result in human cysticercosis thus infection of various tissues with the larval stage of the tapeworm which can eventually cause neurocysticercosis (Dorny *et al.,* 2009, Molyneux *et al.,* 2011). The consequences can be seizures and muscle or eye damage.

2.7.5 Laboratory Diagnosis of *Taenia species*

Concentration techniques and examination of several specimens may be necessary to detect *Taenia* eggs in faeces because eggs are not often discharged from tapeworm in the intestines (Cheesbrough, 2005). Adhesive tape technique can be used to recover eggs from perianal skin of infected individuals.Ziehl-Neelsen staining technique can be used to differentiate Taenia eggs. Whiles *Taenia saginata* is acid fast, *Taenia solium* is not acid fast. Enzyme-linked immunosorbent assay (ELISA) has been used to detect *Taenia spp* coproamtigens mostly for neurocysticercosis infections (Allan *et al.,* 1996).

Molecular detection of Taeniasis is highly reliable for differentiation of *Taenia spp* (Ito *et al.,* 2003 Schantz *et al.,* 1998 Flisser *et al.,* 2011 Dorny *et al.,* 2009).

2.7.6 Treatment of *Taenia species*

Praziquantel 5-10 mg/kg single administration for infected individuals. Niclosamide for adults and children over six years single administration after breakfast followed after 2 hours by a laxative. Neurocysticercosis treatment is tailored to the individual case since destruction of cysts may lead to an inflammatory response.

2.7.7 Prevention of *Taenia species*

1. Intensive educational intervention
2. Avoiding access to human faeces by practicing proper animal husbandry
3. Effective inspection of meat before consumption
4. Improved sanitation
5. Access to preventive chemotherapy

2.8 Malaria

2.8.1 Malaria Infection

Malaria is one of the most prevalent and serious infectious disease problems throughout the tropical and subtropical areas of the world (WHO, 2013). The disease infects more than 216 million inhabitants of more than 99 countries worldwide (WHO, 2011). Global malaria mortality was 1.2 million in 2010 (Murray *et al.*, 2010). Pregnant women comprise one of two groups particularly vulnerable to malaria, the other being children (Allen, 2000). Malaria during pregnancy can have devastating consequences to both mother and developing foetus and is associated with mild to severe maternal illness, maternal anaemia, spontaneous miscarriage, stillbirth, preterm delivery and foetal growth retardation (Desai *et al.*, 2007). Malaria is the most important preventable cause of low birth weight in malaria-endemic areas in sub-Saharan Africa, which in turn is associated with increased susceptibility to illness and death in early life (Desai *et al.*, 2007; Steketee *et al.*, 2001). *P. falciparum* is the only human malaria parasite that is more common in pregnant than in non-pregnant women and is the only human parasite with a clear and substantial adverse effect on pregnancy, nutrition during pregnancy and pregnancy outcome (Steketee *et al.*, 2001).

Pregnant women are 3 times more likely to suffer from severe disease as a result of malarial infection compared with their non-pregnant counterparts, and have a mortality rate from severe disease that approaches 50% **(** Monif and Baker, 2004; WHO, 2006). The clinical presentation of malaria in most persons is dependent on host immunity and the infecting parasite species. In malaria-endemic areas, pregnancy is associated with a significant decrease in the level of acquired immunity against malaria, which is evidenced by the greater frequency of clinical

symptoms and a higher degree of parasitemia (Desai *et al.*, 2007). This effect is particularly apparent during the second half of pregnancy, but the factors responsible for potentiation of parasitemia remain unclear. In many endemic areas, malaria is a leading cause of maternal mortality (Yatich *et al.*, 2009).

The anaemia in pregnancy is potentiated during malarial infection secondary to hypersplenism, direct lysis of parasitized erythrocytes, and autoimmune hemolysis (BouyouAkotet *et al.*, 2003). This rapid turnover of blood cells can produce serious folic acid deficiency and general hypochromic microcytic haemolytic iron deficiency anaemia. In placental pathology is often altered in heavy malarial infections transportation of nutrients, and oxygen to the fetus is markedly diminished (Julianna *et al.*, 2009). The anaemia produced by *P. falciparum* infection usually is seen after 20 weeks of pregnancy, and may induce congestive heart failure because of the reduced red cell mass (White *et al.*, 2008). When associated with pregnancy, the cerebral form of malaria that is caused by *P. falciparum* may be mistaken for eclampsia, especially if the patient is comatose.

The syndrome of acute renal insufficiency is a complication of *P. falciparum* malaria that can be superimposed on other diseases, such as toxemia of pregnancy. Familiarity with the diagnosis, complications, and treatment of malaria is essential because it is a potentially fatal infection for which prophylaxis and treatment are readily available.

2.8.2 Laboratory Diagnosis of Malaria

Diagnosis is based on clinical suspicion and confirmed by the finding of malaria parasites in thick and thin blood smears.

The diagnosis of malaria essentially rests on the finding of parasites in stained peripheral blood smears (WHO, 2009). Although a higher density of parasites appears in circulation during the paroxysms as schizonts burst and release merozoites, timing in obtaining smears is less important than obtaining the smears several times daily for several days. Giemsa stain is preferred for speciation, which is necessary for treatment protocols, but routine Wright's stain is adequate for identification of the parasites (Gillet *et al.*, 2009). Thick smears may be used for concentrating the parasites in persons with low parasitemia (Bell and Perkins, 2008). However, artifacts are numerous, and correct interpretation of these tests requires experience (Wongsrichanalai *et al.*, 2007). Once parasites are detected, Giemsa-stained thin blood smears should be examined to determine which species is present (Gillet *et al.*, 2009). Since most physicians are not experienced in the morphologic differentiation of *Plasmodium* species, an expert opinion should be sought as soon as possible because therapy varies from species to species (WHO, 2010). The most important distinction is to determine whether *P. falciparum* is present because drug resistance is known with this species, and it is associated with an increased mortality rate (WHO, 2010). Several rapid diagnostic tests (RDTs) have been developed recently avoiding the need for light microscopy in remote settings and potentially improving fever management in resource limited settings (Moody, 2002).

Quantification of parasitemia may be performed and then followed over the subsequent days of treatment to determine the effectiveness of therapy (Bell and Perkins, 2008).

2.8.3 Treatment of Malaria

After the diagnosis and speciation of malaria, treatment should be instituted immediately. Treatment of uncomplicated malaria in pregnancy is a balance between potential fetal adverse effects from drug toxicity and improved clinical status with clearance of the parasite.

2.8.4 Prevention of Malaria

In areas of the world where malaria is endemic, the use of limited residual insecticides and chemoprophylaxis for pregnant women and children is recommended. It is now recommended that all pregnant women living in areas of high or intermittent (stable) *P. falciparum* transmission should receive intermittent presumptive treatment (IPT) after quickening during antenatal visits (Peters *et al.,* 2007). Ideally this involves two and possibly three IPT doses with sulphadoxine-pyrimethamine (500 mg/25 mg orally). This strategy is not recommended for areas of low or unstable transmission.

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 STUDY DESIGN

This cross sectional study was conducted on 400 (participants) who accepted to be enrolled during their visit to the health centres between April and July, 2012.The participants aged between 15 and 49 years visiting the antenatal clinic for the first time of their current pregnancy were asked to volunteer for this study and to provide urine and stool sample. For each client, data regarding age, gravida and parity were documented.

All participants who tested positive for parasites or other abnormalities in the stool, urine and blood that suggested a disease condition were treated based on consultation with the midwife at the health center. The drug of choice included; praziquantel, albendazole and iron supplements depending on the type of parasitic infection.

3.2 PARASITOLOGICAL SURVEY

A pre-survey visit was made to prospective study sites (Hospital and Health Centres) within the Dangme East District during which consultations and discussions were held with the medical superintendent and the heads of the Laboratories of the Hospital and Health Centres to help in mobilization of patients for sample collection. Written permission was sought from the heads of the selected Hospital and Health Centres.

3.3 STUDY TYPE

This study was designed to determine the patterns of schistosomiasis and soil transmitted helminth infections in pregnant women. Hence a cross sectional study was conducted in selected Hospital and Health Centers in Dangme East District in the Greater Accra Region of Ghana from April – July, 2012.

3.4 THE STUDY AREA

The population of Dangme East district, the study site was 130,795 and the population under study was made up of pregnant clients visiting Sege Health Centre, Bonikope Health Centre and

Anyamam Health Centre all within the Dangme East district. The selected sites were chosen based on their demographic characteristics. Figure 3.1.

Dangme East District is situated in the eastern part of the Greater Accra Region and has AdaFoah as its capital. It shares common boundaries with North Tongu district at the North, South Tongu district and Dangme West at the East and West respectively. At the south is the Gulf of Guinea, which stretches over 45 kilometres (27.9 miles). The district lies within latitudes 5°45 south and 6°00 north and from longitude 0°20 west to 0°35 east. The district forms part of the south-eastern coast plains of Ghana, which is one of the hottest part of the country. The average rainfall is about 750mm. According to Local Service Delivery and Governance Program (2010), temperatures are high throughout the year and range between 23^{oC}-33^{oC} during the hot season. Humidity is very high; about 60% due to the proximity of the sea, the Volta River and other water bodies (LSDGP, 2010). Daily evaporation rates ranges from 5.4mm to 6.8mm. The vegetation is generally coastal savannah, with most characterised by short savannah grass interspersed with shrubs and short trees (LSDGP, 2010). Along the stretch of the sea shores are few mangrove trees and coconut groves (LSDGP, 2010). The savannah also provides extensive land for grazing livestock. Majority of the populace are engaged in livestock production, fishing, cash crop farming and trading (DPCU, 2012).

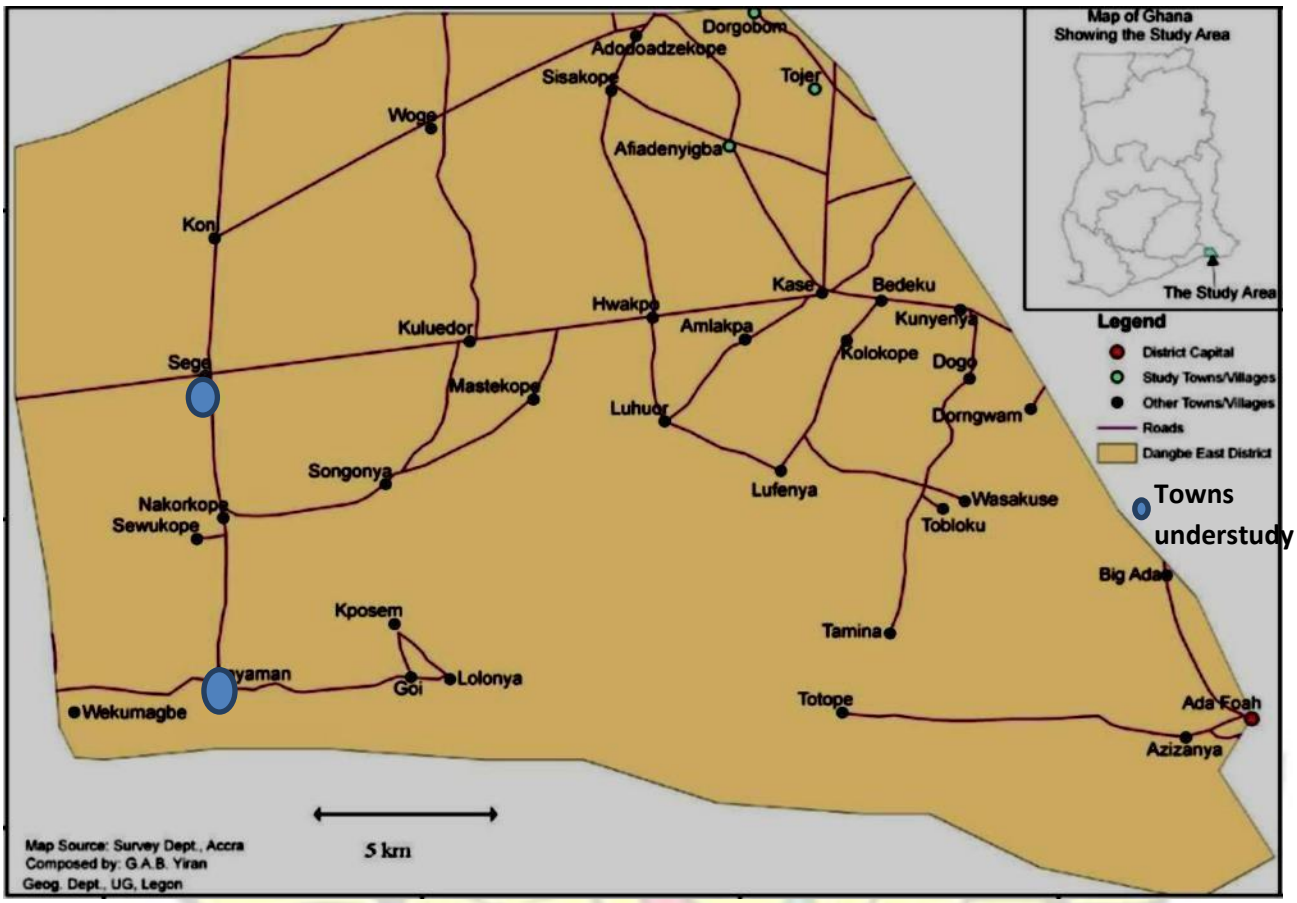

FIGURE 3.1 MAP OF DANGME EAST DISTRICT SHOWING STUDY AREA.

SOURCE: District Planning and Coordination Unit, Dangme East District Ada

3.5 SAMPLE SIZE:

A sample size of 375 participants were recuited for data collection among pregnant women using an overall prevalence of 59% anaemia cases observed in a national demographic survey in Ghana among pregnant women (Ghana Demographic Survey, 2008). The sample size was determined using the formula:

$$N=z^2 (p*q)/d^2$$

Where N = sample size, z = Statistical certainty chosen = (1.96), p= Estimated prevalence of anaemia among pregnant women, q = 1- p, d = precision desired.

The following assumptions were made: Z (Statistical certainty chosen) = 1.96 d (precision desired) = 0.05 p (estimated prevalence) = Prevalence of anaemia among pregnant women 59% (0.59)(Ghana

Demographic Survey, 2008)

Substituting into the formula:

$N = (1.96)^2(0.59)\ (0.41)/\ (0.05)^2$

$= 0.92928304/0.0025$

$=371.71$

Assuming a response rate of 99%

Sample size = 371.71/0.99

=375.46

=375 subjects to the nearest whole number

3.6 ETHICAL ISSUES:

The study was conducted with the approval of the Committee on Human Research Publication and Ethics (CHRPE) of the School of the Medical Sciences, Kwame Nkrumah University of Science and Technology. An informed written consent of each participant prior to inclusion in the study was also obtained. Subsequently proxy consent forms for participants of seventeen (17) years and below were administered seeking parental consent. Participants were also informed that they were free to withdraw consent anytime and their medical records and specimens were examined and treated with strict confidentiality. Study participants who had parasites in their samples were treated free of charge based on the Ghana Health Service treatment guidelines. The drugs were administered by qualified midwife prescribers working at the study sites. The full cost of treatment of each participant was absorbed by the research team.

3.7 SAMPLE COLLECTION:

Stool samples voided in the morning by participants were collected in a clean, wide mouth, and well capped container. Urine samples of about 20 - 30 mls were collected alongside in a clean wide mouth, and well capped container. Blood volumes of 4 mls were collected in EDTA anticoagulant tubes and were used to estimate the haemoglobin levels and presence of malaria parasite of participants.

3.8 LABORATORY TESTS:

3.8.1 STOOL EXAMINATION: Formalin-Ether Concentration

With the aid of an applicator stick, about 1g of stool sample was emulsified in 3 - 4 mls of 10% formalin and the contents transferred into 10mls centrifuge tube. The contents were mixed by shaking for 20 seconds and then sieved into a beaker. The sieved suspensions were poured back into the centrifuge tube and the debris discarded. About 3 mls of diethyl-ether was added to the suspension in the tube and was well stoppered, mixed well and the contents centrifuged at 3000 rpm for 1 minute. The supernatant was decanted in a single movement into a bowl containing disinfectant; allowing the last few drops of residual fluid to flow back onto the sediment and the tubes placed in a rack. The sediment was re-suspended with a disposable pastuer pipette and a few drops transferred onto a microscope slide, and covered with a cover slip. The specimen was examined microscopically using the lower power (x10) objective, in a systematic manner ensuring observation of the entire coverslip area. A higher magnification (x40) objective was used to observe the detailed morphology of ova or larvae found under the light microscope (Olympus CX21FSI).

3.8.2 URINE EXAMINATION

Midstream urine samples were collected in wide-mouthed sample containers. Each specimen was well mixed and 10mls aliquot of urine sample was withdrawn into a syringe. Filter holder fitted with 25mm millipore filter membrane of pore size 8um was attached to the syringe. The urine sample was then filtered through the filter, membrane. Again 10mls of physiological saline was withdrawn into the syringe also expressed through the filter membrane fitted in the filter holder a second time. Following this, the syringe was then filled with air instead of saline and expressed through the filter. Filter holder was removed from the syringe and disassembled to expose the filter

paper. With the aid of a pair of forceps, each membrane was carefully removed and placed upside down onto a microscope slide. A drop of saline was added to moisten the filter. Slides were examined under the microscope using 10x objective and reporting by systematically examining the entire field of the filter paper for *Schistosoma haematobium* eggs. The number of eggs were counted and was reported per 10ml of urine.

3.8.3 BLOOD EXAMINATION

3.8.3.1 HAEMOGLOBIN ESTIMATION

Haemoglobin levels of participants were measured using Sysmex haemoglobin analyzer (Sysmex KX-21N (Sysmex Corporation KOBE Japan). Blood obtained in EDTA anticoagulant tube was well mixed and inserted into a probe on the analyzer which subsequently aspirated the required quantity of blood for the estimation of the haemoglobin. Values were printed out in about 10 - 15 seconds and were immediately documented.

3.8.3.2 MALARIA PARASITE COUNT AND IDENTIFICATION

Blood was obtained from participants into an EDTA anticoagulant tube and malaria parasitaemia determined by the microscopic examination of giemsa-stained thick and thin blood films. Thick and thin smears were prepared on clean, dry microscope glass slides and allowed to dry. The thin smears were fixed in absolute methanol and both smears were stained with 2% Giemsa (BDH Laboratory Supplies, Poole BH15 ITD, England). Asexual stages of *Plasmodium falciparum* were counted to estimate parasitaemia.

3.9 DATA ANALYSIS:

Data entry and validation was performed in excel, and statistical analysis was done using Statistical Package for Social Sciences (SPSS) version 16.0. Values were considered statistically significant when P-values were less than 0.05 ($P<0.05$). Chi-square and correlations were used to determine the association between hemoglobin concentrations and infections, age group and infections, gravidity and infections, and parity and infections as indicators of anaemia. Binary logistic regression (odds ratio) was used to determine the odds at which an infection could cause anaemia. Cross tabulations was used to determine the frequencies and percentages between variables.

CHAPTER FOUR

4.0 RESULT

4.1 Participants and Non- infectious risk factors

A total of four hundred (400) pregnant women visiting three health centres in three communities: Sege, Anyamam and Bonikope in the Dangbe East district were enrolled in the study. Samples of stool, urine and blood were collected and examined for infections of soil transmitted helminths, intestinal and urogenital schistosome parasites and malaria parasites;(out of the 400 pregnant women enrolled in the study, 384 (96.0%) provided urine samples and 375 (93.8%) stool samples). The statistical analysis using SPSS was thus limited to 375 cases for which all three samples were taken. Various statistical tools such as frequency distributions cross tabulations, chi sq test of goodness fit, binary logistic regression and odds ratios were used to derive significance and impact of the anaemia in the presence or absence of each parasiteamia under study. The mean age of the pregnant women was 25.83 within a range of 15 – 45 years.

4.2 Percentage distributions of anaemia within the population under study.

Table 1 shows the categories of the non-infectious characteristics under study. Frequency distributions were used to derive frequencies of the sub-categories of each major category. The sub-categories were then couple with anaemia using cross-tabulations to derive the percentages of anaemia as occurring in each sub-category. In summary, there were 216 (57.6%) of pregnant women within the age group 15 – 19 years and 37.9% of them were anaemic. Pregnant women within age group 40 – 49 years were least represented with 10 people and aneamia was 1.6%.

With respect to gravidity, multigravids were 213 (56.8%) and 35.5% anaemic whilst primagravids were 93 (24.8%) and 17.6% of them were anaemic. With groupings based on parity, multiparous women were 195 (52.0%) and 32.5% anaemic.

TABLE 1: PERCENTAGE DISTRIBUTION OF ANAEMIA AMONG THE PREGNANT WOMEN

	Total		Anaemia	
characteristic	N	Percent	Absent (%)	Present (%)
Age Group (years)				
15-19	62	16.5	3.7	12.8
20-29	216	57.6	19.7	37.9
30-39	87	23.2	9.1	14.1
40-49	10	2.7	1.1	1.6
Gravidity				
Primagravid	93	24.8	7.2	17.6
Secondagravid	69	18.4	5.1	13.3
Multigravid	213	56.8	21.3	35.5
Parity				
Nulliparous	88	23.5	7.2	16.3
Primiparous	92	24.5	6.9	17.6
Multiparous	195	52.0	19.5	32.5

4.3 Frequency distributions of infections with soil transmitted helminths and schistosomes

Single and co-infections were identified in the examination of parasites causing anaemia in this study. In all, 180 cases of infections were observed, of which 165 (44.0%) were single isolated infections whilst 15 (4.0%) were co-infections. The following parasites were thus identified under the study, malaria 62 (16.5%), Hookworm 15 (4.0%), *Schistosoma haematobium* 17

(4.5%), *Ascaris lumbricoides* 32 (8.5%), *Trichuris trichiuria* 22 (5.9%), *Schistosoma mansoni* 28 (7.5%), *Strogyloides stercoralis* 7 (1.9%) and *Taenia species* 3 (0.8%). The parasites were then couple with anaemia using cross tabulations and subsequently tested for its significance to impact on anaemia using Pearson's Chi-sq statistic. *P* values less than 0.05 were considered significant whilst *P* values greater than 0.05 were considered non-significant.

It is evident from Table 2 that 16.9% of the malaria cases resulted in the pregnant women being anaemic and it was significant (χ^2 = 21.999 df (1), P< 0.05 95% CI). Also, the presence of *Schistosoma haematobium* and hookworm were both significant to cause anaemia in pregnancy. The Pearson chi-sq test statistic was respectively χ^2 = 5.661, df (1) P < 0.05 and χ^2 = 9.638 df (1) P < 0.05 at 95% CI. The rest of the infections were not significant to cause anaemia in pregnancy except for the presence of co-infections which was significant (χ^2 = 9.638 df (1), P < 0.05 at 95%CI). In all there were 151 cases of anaemia that was as the result of the presence of parasitic infections and 98 of the anaemic cases were not due to parasitic infections.

TABLE 2: FREQUENCY DISTRIBUTION OF MALARIA, SOIL TRANSMITTED HELMINTHS AND SCHICTOSOMES AMONG THE STUDY POPULATION.

Infection	**Total**		**Anaemia**			
	N	Percent	Absent (%)	Present (%)	$\chi 2$	Sig (2tailed)
Malaria					21.999	P < 0.05
Present	62	16.5	4.1	16.9		
Absent	313	83.5	38.6	40.3		
S.haematobium					5.661	P < 0.05
Present	17	4.5	0.8	3.7		
Absent	358	95.5	32.8	62.7		
Hookworm					9.638	P < 0.05
Present	15	4	0.3	3.7		
Absent	360	96	33.3	62.7		
Ascaris lumbricoides					0.931	P > 0.05
Present	32	8.5	3.2	5.3		
Absent	343	91.5	30.4	61.1		
Trichuris trichuria					0	P > 0.05
Present	22	5.9	2.7	3.2		
Absent	353	94.1	30.9	63.2		
S.mansoni					3.595	P > 0.05
Present	28	7.5	2.1	5.3		
Absent	347	92.5	31.5	61.1		
Strogyloides S					0.829	P > 0.05
Present	7	1.9	0.5	1.3		
Absent	368	98.1	33.1	65.1		
Taenia spp.					0.181	P > 0.05
Present	3	0.8	0.3	0.5		
Absent	372	99.2	33.3	65.9		
Co-Infections					9.638	P < 0.05
Present	15	4	0.3	3.7		
Absent	360	96	33.3	62.7		

4.4 Binary logistic regression of non- infectious characteristics.

Table 3 shows the proportionate change in odds of the non -infectious characteristic under study with the impact of predicting anaemia. Binary logistic regression was used to derive these results. The various categories were analysed as a composite to predict the trend of the impact of anaemia in their sub-categories and also derive the equation that exist between the characteristic and anaemia. The table reveals that anaemia was significant when the pregnant women were grouped into ages (Wald chi –sq = 4.004 df (1), $P < 0.05$ at 95% CI). The change in odds that a pregnant woman of a particular age to be anaemic was Exp (B) = 0.73. Thus for a unit change in age the odds for a pregnant woman to be anaemic is related by the equation: In (odds Anaemia) = 1.356 – 0.315 (Age). In this study the change in odds Exp (B) = 0.73 being lower than 1 suggest that with increase in age of a pregnant woman, the odds of being anaemic decreases. However, the unit change in odds for both gravidity and parity to cause anaemia were both non-significant $P > 0.05$ at 95% CI. The change in odds were respectively Exp (B) = 0.834 and 0.804 for parity and gravidity. The values also depict a decreasing trend to cause anaemia for a unit change in parity and gravidity respectively.

TABLE 3: BINARY LOGISTIC REGRESSION OF THE PARASITIC INFECTIONS AMONG THE PREGNANT WOMEN

Characteristic	B	S.E.	Wald	df	Sig.	Exp(B)	95.0% C.I.for EXP(B)	
							Lower	Upper
Age	-0.315	0.157	4.004	1	0.045	0.73	0.536	0.994
constant	1.356	0.358	14.334	1	0	3.88		
Parity	-0.182	0.136	1.786	1	0.181	0.834	0.639	1.088
constant	1.1	0.335	10.808	1	0.001	3.003		
Gravidity	-0.218	0.133	2.678	1	0.102	0.804	0.62	1.044
constant	1.192	0.334	12.705	1	0	3.294		

4.5 Binary logistic regression of the sub-categories of non-infectious characteristics

Table 4 elaborates the decreasing odds of the non-infectious characteristics to cause anaemia as predicted in Table 3. In this statistic binary logistic regression was used but analysis was done to reveal the trends in each sub category. The change in odds for a unit change in age group to effect anaemia decreased from Exp (B) = 3.429 to 0.438. That is from age group 15 -19 years to 40 -49 years. The change in odds was significant for age group 30 – 39 years (Wald chi-sq = 4.421 df(1) P< 0.05 at 95% CI. The other sub categories of parity and gravidity were all nonsignificant and revealed the decreasing odds as predicted in Table 3.

TABLE 4: BINARY LOGISTIC REGRESSION SHOWING THE SUB-CATEGORIES OF NON-INFECTIONS AMONG THE PREGNANT WOMEN UNDER STUDY

Characteristic	B	S.E.	Wald	df	Sig.	Exp(B)	95.0% C.I.for EXP(B)	
							Lower	Upper
Age			4.668	3	0.198			
Age(1)	-0.58	0.713	2.986	1	0.084	0.56	0.29	1.081
Age(2)	-0.788	0.661	4.421	1	0.036	0.455	0.218	0.948
Age(3)	-0.827	0.682	1.343	1	0.247	0.438	0.108	1.771
Constant	1.232	0.645	16.455	1	0	3.429		
Gravidity			3.484	2	0.175			
Gravidity(1)	0.074	0.353	0.044	1	0.835	1.077	0.539	2.152
Gravidity(2)	-0.385	0.269	2.058	1	0.151	0.68	0.402	1.152
Constant	0.894	0.228	15.308	1	0	2.444		
Parity			0.621	2	0.733			
Parity(1)	-0.161	0.335	0.232	1	0.63	0.851	0.442	1.64
Parity(2)	-0.215	0.273	0.62	1	0.431	0.806	0.472	1.378
Constant	0.815	0.231	12.433	1	0	2.259		

4.6 Binary logistic regression for infectious organisms causing anaemia

The table shows the various odds at which an infection under study could cause anaemia. From the table it is evident that the odds EXP (B) to cause anaemia was >1 which meant that for a unit change in the number of parasite, the odds for a parasite to cause anaemia increases. With respect to malaria, *Schistosoma haematobium* and Hookworm the odds at which the parasite could cause anaemia were 4.703, 4.292 and 12.744 respectively and found to be significant (P<0.05) at 95% CI. *Ascaris lumbricoides, Trichuris trichiura, Schistosoma mansoni, Strongyloides stercoralis* and *Taenia species* even though had varied odds to cause anaemia but were not significant (P>0.05) 95% CI. Co-infection of the parasites could cause anaemia at odds of 12.744 and was significant (P<0.05) 95% CI.

TABLE 5: BINARY LOGISTIC REGRESSION FOR MALARIA, SOIL TRANSMITTED HELMINTHS AND SCHISTOSOMIASIS CAUSING ANAEMIA IN PREGNANT WOMEN

	B (S.E)	Wald	df	Sig.	Exp(B)	95.0% C.I.for	
						Lower	Upper
Malaria(1)	1.548	19.646	1	0	4.703	2.372	9.326
Constant	-1.21	0.785	1	0.376	0.886		
S.haematobium(1)	1.433	4.885	1	0.027	4.292	1.176	14.927
Constant	0.108	0.753	1	0.385	1.114		
Hookworm(1)	2.547	5.971	1	0.015	12.774	1.656	98.551
Constant	0.092	0.549	1	0.459	1.096		
Ascaris lumbricoides(1)	0.372	0.923	1	0.337	1.45	0.679	3.096
Constant	0.139	1.178	1	0.278	1.149		
Trichuris trichuria(1)	0.001	0	1	0.997	1.001	0.418	2.401
Constant	0.181	2.069	1	0.15	1.198		
S.mansoni(1)	0.812	3.448	1	0.063	2.252	0.956	5.305
Constant	0.105	0.678	1	0.41	1.11		
Strongyloides s(1)	0.753	0.793	1	0.373	2.123	0.405	11.136
Constant	0.163	1.789	1	0.181	1.177		
Taenia spp.(1)	0.518	0.177	1	0.674	1.678	0.15	18.722
Constant	0.176	2.097	1	0.148	1.192		
Co-infections(1)	2.547	5.971	1	0.015	12.774	1.656	98.551
Constant	0.092	0.549	1	0.459	1.096		

4.7 Schistosomiasis, Helminthes and Malaria Infections within Age, Gravidity and Parity

As shown in Table 6, most of the infections (schistosomes, STH and malaria) recorded the highest prevalence among pregnant women in age group 20 - 29 years. A prevalence of 4 (1.1%), 10 (2.7%), and 3 (0.8%) was observed in age groups 15 - 19, 20 - 29 and 30 - 39 years respectively *S. haematobium*. There was no occurrence of *S.haematobium* among pregnant women aged 40 - 49 years. The prevalence of *S. haematobium* was statistically marginally nonsignificant (χ^2=5.059, df (2), P=0.08, 95% CI). There was 1(0.3%) case of hookworm infection occurring within age group 15-19 and 40-49 years respectively. However, 10 (2.7%) and 3 (0.8%) of *hookworm* infections occurred within age group 20 - 29 and 30 - 39 years respectively (Table 2). With exceptions to

Strongyloides and *Taenia spp.*infections, all other single infections were found to be statistically significant at 95%CI (P<0.05).In this study (Table 2), co-infections were however, non-significant across all age groups (χ^2=4.80, df (1), P=0.091). The 62 cases of malaria were distributed across the age groups as 18(4.8%) occurring within pregnant women aged 15-19 years, 32(8.5%) within 20-29 years, 11(2.9%) within 30-39 years and 1(0.3%) within

40-49 years. The prevalence of malaria was highly significant across age group (χ^2=32.839, df (3), P=0.00, 95% CI).

The distribution of *Ascaris* within gravidity was in the ratio 4(1.1%):7(1.9%):21(5.6%) in primigravid, secondagravid and multigravid respectively. *Trichuris* prevalence was 4(1.1%) primigravid, 2 (0.5%) secondagravid and 21(5.6%) multigravid. Both *Ascaris* and *Trichuris* were statistically strongly significant within gravidity (P<0.001, 95% CI). All others- *Strongyloides, Taenia spp, Schistosoma haematobium, Schistosoma mansoni,* hookworm including coinfections were found to be statistically non-significant within the gravidity of the pregnant women (P>0.05 at 95% CI). Similar to gravidity, malaria, *Ascaris* and *Trichuris* were statistically significant within parity (P<0.05, 0.02 and 0.02, 95%CI respectively). Malaria was again statistically significant within gravidity (P<0.05, 95% CI) and represented as follows, 24 (6.4%) primigravid, 7 (1.9%) secondagravid and 31(8.8%) multigravid.

TABLE 6: SINGLE, CO-INFECTIONS WITHIN AGE, GRAVIDITY AND PARITY AMONG THE PREGNANT WOMEN

AGE GROUPS	15-19	20-29	30-39	40-49	χ_2	***P*** value
Malaria	18 (4.8%)	32 (8.5%)	11 (2.9%)	1 (0.3%)	32.839	P<0.05
S.haematobium	4 (1.1%)	10 (2.7%)	3 (0.8%)	0 (0%)	5.059	P<0.05
Intestinal helminths	9 (2.4%)	70 (18.7%)	16 (4.3%)	6 (1.6%)	107.800	P<0.05
Hookworm	1 (0.3%)	10 (2.7%)	3 (0.8%)	1 (0.3%)	14.600	P<0.05
Ascaris	2 (0.5%)	24 (6.4%)	6 (1.6%)	1 (0.3%)	25.750	P<0.05
Trichuris	2 (0.5%)	12 (3.2%)	6 (1.6%)	2 (0.5%)	12.182	P<0.05
S.mansoni	3 (0.8%)	23 (6.1%)	1 (0.3%)	1 (0.3%)	49.143	P>0.05
Strongyloides	1 (0.3%)	4 (1.1%)	0 (0%)	2 (0.5%)	2.000	P>0.05
Taenia sp.	0 (0%)	3 (0.8%)	0 (0%)	0 (0%)	0.000	P>0.05
Co-infection	3 (0.8%)	9 (2.4%)	3 (0.8%)	0 (0%)	4.800	P>0.05

GRAVIDITY	Primagravid	Secondagravid	Multigravid		
Malaria	24 (6.4%)	7 (1.9%)	31 (8.8%)	14.742	P<0.05
S.haematobium	5 (1.3%)	4 (1.1%)	8 (2.1%)	1.529	P>0.05
Intestinal helminths	21 (5.6%)	19 (5.1%)	61 (16.3%)	33.347	P<0.05
Hookworm	4 (1.1%)	2 (0.5%)	9 (2.4%)	5.200	P>0.05
Ascaris	4 (1.1%)	7 (1.9%)	21 (5.6%)	15.438	P<0.05
Trichuris	4 (1.1%)	2 (0.5%)	16 (4.3%)	15.636	P<0.05
S.mansoni	7 (1.9%)	7 (1.9%)	14 (3.7%)	3.500	P>0.05
Strongyloides	1 (0.3%)	3 (0.8%)	3 (0.8%)	1.143	P>0.05
Taenia sp.	1 (0.3%)	0 (0%)	2 (0.5%)	0.333	P>0.05
Co-infection	4 (1.1%)	4 (1.1%)	7 (1.9%)	1.200	P>0.05

PARITY	Nulliparous	Primiparous	Multiparous		
Malaria	23 (6.1%)	8 (2.1%)	31 (8.3%)	13.194	P<0.05
S.haematobium	4 (1.1%)	7 (1.9%)	6 (1.6%)	1.529	P>0.05
Intestinal helminths	21 (5.6%)	22 (5.9%)	58 (15.5%)	26.396	P<0.05
Hookworm	4 (1.1%)	2 (0.5%)	9 (2.4%)	5.200	P>0.05
Ascaris	4 (1.1%)	8 (2.1%)	20 (5.3%)	13.000	P<0.05
Trichuris	4 (1.1%)	3 (0.8%)	15 (4.0%)	12.091	P<0.05
S.mansoni	7 (1.9%)	7 (1.9%)	14 (3.7%)	3.500	P>0.05
Strongyloides	1 (0.3%)	3 (0.8%)	3 (0.8%)	1.143	P>0.05
Taenia sp.	1 (0.3%)	1 (0.3%)	1 (0.3%)	0.000	P>0.05
Co-infection	4 (1.1%)	4 (1.1%)	7 (1.9%)	1.200	P>0.05

CHAPTER FIVE

5.0 DISCUSSION

Schistosomiasis and helminthiasis and their morbidity have been rated the most predominant infection in developing countries especially among pregnant women (Ordi *et al.*, 2009; King *et al.*, 2004; Montressor *et al.*, 2002). This has prompted several research studies into parasitic

helminthes infection and associated risk factors in various countries with particular attention to pregnant women. It is upon these concerns that a study on prevalence of schistosomiasis and helminthiasis and associated risk among pregnant women was conducted.

The overall prevalence of intestinal parasitic infections (25.07%) observed among our studied population was consistent with results obtained in a study conducted among pregnant women by Yatich et al., (2009) who reported 25.70%. However, this current study had a higher prevalence of intestinal parasites than similar studies conducted by Fuseni *et al.* (2010) in Northern Ghana (23.0%) and Amengor *et al.,* (2005) in Sekyere East district in Ashanti region of Ghana (17.6%). These differences could be due to the various environmental and socio-economic activities engaged in by the pregnant women within their locality. The prevalence of co-infection among the studied pregnant women was 4.00%. This prevalence was lower than a study by Yatich *et al.,* (2009) in Ghana which reported a prevalence of 16.60% and a similar study in Gabon by Ayola *et al.,* (2010) which reported a co-infection prevalence of 22.0%. The occurrence of schistosomiasis and helminth infection among pregnant women is indicative of faecal pollution of soil and domestic water supply around homes due to poor sanitation, ignorance of the mode of transmission of these worms and improper sewage disposal has been found to be predisposing factor to infection.

The overall prevalence of 4.5% for *Schistosoma haematobium* infection among the studied pregnant women was in consonance with a similar study in Bawku in northern Ghana by Siegrist (1992) which reported 4.5% prevalence of *Schistosoma haematobium* in a community-based study among pregnant women and outcome of their birth. The trend of infection among the age groups also agrees with earlier studies that reported peak intensity of infection among age groups 16 - 20

years whiles age group of 41- 45 years had the lowest mean intensity (Yirenya- Tawiah *et al.,* 2011; Eyo *et al.,* 2012). Young age with the highest prevalence in this present study could be attributed to the role played by young women in the rural sites such as fetching of water, washing of clothing and helping with agricultural tasks which predispose them to infective sites. Whilst acquired immunity might be the contributing factor to the no infection among 40-49 year age group, immunity to *Schistosoma haematobium* infection has been shown to affect mean egg output of infected persons in a study by Saviola *et al.,* (1990).

Prevalence of *Schistosoma haematobium* within gravidity of pregnant women was in agreement with a study by McClure *et al.,* (2014) in Kenya among pregnant women with multigravidae. With regards to *Schistosoma haematobium* infection within parity our study was in contrast with a study by Adegnika *et al.,* (2010) who reported a significance of (p=0.001) *Schistosoma haematobium* infection in a cohort study among pregnant women in Coastal Kenya. Unlike the study in Kenya which reported higher prevalence of *Schistosoma haematobium* among multiparous women than primiparous women, we observed in this study that, primiparous women had higher infection rate of *Schistosoma haematobium* than multiparous women. This might have been due to the differences in the age group activities with engagement in infected water bodies. Furthermore, the primaparous women in the current study might not have developed a strong immunity yet against *Schistosoma haematobium* infection.

Schistosoma haematobium caused significant level of anaemia among the pregnant women in this study ($p<0.05$) (Table 2). Findings in this study is in consonance with studies by Friedman *et al.,* (2005) and Allen (2000).This observation of anaemia and *Schistosoma haematobium* infection may have a deleterious consequence on the hematopoietic status of the women in the current study.

In contrast to our study, McClure *et al.,* (2014) has, reported no association between *Schistosoma haematobium* infection and anaemia among pregnant Kenyan women.

Ascaris lumbricoides was the most common intestinal helminth recorded in this present study. An overall prevalence of 8.5% for *Ascaris lumbricoides* infection among the studied pregnant women is in line within the range of prevalence of 1.9 - 67.7% compiled by Ojha *et al.,* (2014) for some randomly selected developing countries. The higher prevalence of *Ascaris lumbricoides* recorded as against the other helminthes might be attributed to the remarkable ability of a single worm to release up to twenty-seven million eggs during the course of infection and more importantly, the resistant nature of the eggs due to the presence of proteinaceous coat to extreme environment conditions for long periods of time (Ojha *et al*., 2014). The prevalence however falls below 12.30% reported by Yatich *et al.,* (2009) in a similar study in Kumasi in Ghana.This could be attributed to the larger sample size of the study by Yatich *et al.,* (2009).

In relation to the age groups, *Ascaris lumbricoides* infection was highest among the age groups 20-29 years; 6.4%, followed by 30-39 years; 1.6%, 15-19 years; 0.3% and 40-49 years. This trend observed is in consonance with work done by Obiezue *et al.,* (2013) in Nigeria. The trend observed was attributed to unhygienic practices among study population. According to a study by Woodburn *et al.,* (2009), young age and lack of education were risk factors for infection with helminthes thus including *Ascaris lumbricoides* infection as recorded in the present study.

A study conducted among pregnant Kenyan women by Eijk *et al.,* (2009) revealed *Ascaris lumbricodes* prevalence increased with gravidity as observed in the current study 1.1%, 1.9% and 5.6% for primigravidae, secondagravidae and multigravidae respectively. The same trend was observed in our study with regards to *Ascaris lumbricoides* infection with parity of pregnant

women who were studied which indicated a statistical significance ($p<0.05$) (Table 6). Pregnancy has been associated with an increase in *Ascaris lumbricoides* and *Trichuris trichiura* infections compared to non-pregnant women in a small study in Gabon by Adengnika *et al.,* (2007). In this present study, *Ascaris lumbricoides* caused 3.7% mild anaemia and 3.2% moderate anaemia, but it was not statistically significant ($p> 0.05$). This finding was consistent with report by Larocque *et al.,* (2005) in Peru who reported a similar trend of anaemia among pregnant women.

Schistosoma mansoni prevalence was 7.5% and the second highest infection in this study. This prevalence fell below a similar study conducted by Fuseni *et al.,* (2010) in Northern Ghana which reported 12.3% and another study by Betson *et al.,* (2010) which also recorded as high as 47.6% prevalence among women of reproductive age in Uganda. However, prevalence of 7.5% in this study is higher than a study in Nigeria by Egwunyenga *et al.,* (2001) which reported 3.4% and a study in Benin by Ouèdraogo *et al.,* (2012) which reported 0.2%; although the sample size in these two studies were 2104 and 1005 respectively. The prevalence of *S. mansoni* among the age groups were 0.8%, 6.1%, 0.3% and 0.3% in age groups 15-19 years, 20-29 years, 30-39 years and 40-49 years respectively (Table 6). There was no statistical significance with regards to infection among the various age groups ($p>0.05$). This trend of infection has been previously observed by Muhangi *et al.,* (2007) in Uganda. Anaemia due to *Schistosoma mansoni* infection was not statistically significant.

Although *Trichuris trichiura* infection is predominant in developing countries, the overall prevalence of infection obtained from this present study was 5.9%. The overall prevalence of *Trichuris trichiura* infection recorded is similar to 5.6% reported by Yatich *et al.,* (2010) in a study to determine the effect of malaria and intestinal helminth co-infection on birth-outcomes in

Kumasi, Ghana. However, the prevalence obtained in this study are much lower as compared to the results reported by Ayola *et al.,* (2010) in Gabon where they reported a prevalence rate of 24.0% and Alfonso *et al.,* (2006) study which reported a 36.0% among pregnant women in Venezuela. These differences could be attributed to personal hygiene as reported by Donkor (2009). *Trichuris trichiura* infection among the pregnant women were 0.5%, 3.2%, 1.6% and 0.5% in age groups 15 - 19 years, 20 - 29 years, 30 - 39 years and 40 - 49 years respectively (Table 6). There was a statistical significance among the age groups ($p<0.05$). The infection rate was highest for the age group 20 - 29 years and this declined in the age groups 30 - 39 years and 40 - 49 years. However, the age group 15 - 19 years had the same prevalence as that of 40-49 years. *Trichuris trichiura* is prevalent in warm humid tropics, soil pollution is a major factor in the transmission of the infection in a community as indicated in a study by Mordi and Ngwodo (2007), and they also noted transmission occured through poor sanitary habits of indiscriminate defecation.

Trichuris trichiura was highest among multigravid (4.3%), followed by primigravid (1.1%) and the lowest being secondagravid women (0.5%). The difference between *Trichuris trichiura* infection among the gravidity were statistically significant ($p< 0.05$) and in consonance with the work done by Boel *et al.,* (2010) along Thai- Burmese Border among pregnant women. This trend could be explained by observation made in determining the effect of anti-helminth usage in pregnant women by Woodburn (2009) in Uganda which showed no effect and also as observed by Stephenson *et al.,* (2000) which showed a tendency of *Trichuris trichiura* being resistant to single dose treatment. Similar trend was observed among the parity of the pregnant women; 1.1%, 0.8% and 4.0% in nulliparous, primiparous and multiparous women respectively. *Trichuris trichiura* caused 1.9% mild anaemia and 2.4% moderate anaemia but no severe anaemia was observed in this study due to its infection. This observation was made in a study by Larocque *et*

al., (2005) who concluded that, women having moderate and heavy *Trichuris trichiura* infection were just likely to have anaemia as women having no infection or light infection. This agrees with the current study as observed in Table 2.

Although hookworm infections are predominant in developing countries the overall prevalence obtained from the present study was 4.0% (Table 2). This study finding is similar to results obtained by Wekesa *et al.,* (2014) pregnant women in Kenya where they found a prevalence of 3.9%. Prevalence in the present study was lower than results obtained by Yatich *et al.,* (2009) in Ghana where they reported 7.9% and Ouèdrago *et al.,* (2012) also recorded 9.0% among similar study population in Benin, this trend is affirmed by a statistical significance within the age groups ($p<0.05$) (Table 6) in the present study. Poor sanitary disposal of human faeces and indiscriminate defecation are the principal factors in the aetiology of hookworm infections Mordi and Ngwodo (2007).The low prevalence recorded in the current study could be attributed to smaller sample size, increased use of protective footwear and improved sanitation. The prevalence of hookworm among the various age groups were in consonance with a study conducted by Wekesa *et al.,* (2014) which showed higher prevalence in pregnant women of ≤29 years as compared to their older counterparts..

Infection of hookworm classified under gravidity in this current study revealed a decline from primigravidae of 1.1% to 0.5% for secondagravidae and a rise to 2.4% for multigravidae was similar to the trend observed by Boel *et al.,* (2010) among pregnant women on the Thai- Burmese border. The same trend was observed within parity of the women in the present study. There was no statistical significance within the gravidity and parity of the study population with hookworm infection ($p>0.05$) (Table 4).

Anaemia caused by hookworm infection was 1.6% mild anaemia and 2.1% moderate anaemia. In a study by Stoltzfus *et al.,* (2004) in Zanzibar, treatment of geohelminth infections with mebendazole among young children was associated with an improvement of anthropometric indicators. The authors also suggested that geohelminth infection may stimulate inflammatory responses with adverse effects on protein metabolism and erythropoiesis during first infections which might have resulted in anaemia though not among the primagravid. Even though there was no severe anaemia reported as a result of hookworm infection, ability to cause anaemia was statistically significant ($p < 0.05$) (Table 2). A study by Amengor *et al*., (2005) also reported the same statistical significance of ($p < 0.05$) of haemoglobin in pregnant women in their last trimester and presence of hookworm in their stool. Studies by Melku *et al.,* (2014) and McClare *et al.*, (2014) in Ethiopia and Kenya respectively also reported similar findings.

Strongyloides stercoralis, one of the most common and globally distributed human pathogens of clinical importance infects 30-100 million people worldwide Olsen (2009). Globally, prevalence rates of strongyloidiasis are as high as 50% in certain areas where moist soil and improper disposal of human waste co-exist especially in West African, the Caribbean, Southeast Asia and temperate Glinz *et al*., (2010). Prevalence of *Strongyloides stercoralis* among the studied pregnant women was 1.9%. This prevalence rate is similar to a study by Kawai *et al*., (2009) in Tanzania among similar population which reported 1.6% and Fuseni *et al.,* (2010) in Northern Ghana which also reported 2.3%. The prevalence of *Strongyloides stercoralis* recorded fell below other studies conducted by Muhangi *et al.* (2007) and Verweij *et al.,* (2009) who reported 12.3% in Uganda and 17.9% in Ghana. The low prevalence reported in this present study is consistent with conclusion drawn by Puthiyakunnon *et al.,* (2014) who indicated low parasitic load and uncertain clinical manifestations might be reasons for under-diagnosis of *Strongyloides*

*stercoralis.*A study by Siddiqui and Berk (2001) suggests that a single stool examination by microscopic technique fails to detect larvae in up to 70% of cases, since larval output is low and intermittent.

The trend of prevalence among the age groups was similar to that reported by Yatich *et al.,* (2006) in Kumasi, Ghana with the infection rate peaking at the age group 20-29 years. There was no statistical significance among the age group ($p>0.05$) (Table 6). The infection of *Strongyloides stercoralis* within the gravidity and parity of the study population followed the same trend of 0.3%, 0.8% and 0.8% in primigravidae, secondagravidae and multigravidae women and nulliparous, primiparous and multiparous women.Immunological activation of previous infections might have accounted for this observation made. Even though there was a marginal increase across increasing gravidity and parity, it was not of statistical significance ($p>0.05$) (Table 6).The infection of *Strogyloides stercoralis* leading to anaemia was 1.1% and 0.8%, mild to moderate anaemia and no severe anaemia observed in this study. The ability to cause anaemia was not statistically significant ($p>0.05$) (Table 2). This means there was no association between anaemia and *Strogyloides stercoralis* infection and it is in agreement with studies made by Dreyfuss *et al.,* (2000); Larocque *et al.,* (2005) and Nurdia *et al.,* (2001) who reported no significance between *Strongyloides stercoralis* infection and anaemia.

In the present study, prevalence of Taenia species was 0.8%, this prevalence is similar to a study carried out in three rural communities in Cameroon by Nguekam *et al.,* (2003) who reported between 0.4% and 3.0%. It is also similar to a study by Garcia *et al.,* (2003) in Peru who reported 0 - 1.9% in a community based study. This study prevalence is however lower than Garcia-Noval *et al.,* (1996) study which reported a 2.8% in Guatamela.

Among the age groups, only 20-29 year had 0.8%, 15-19 year, 30-39 year and 40-49 year age groups had 0.0% and there was no statistical significance ($p > 0.05$) within the age groups and infection with *Taenia spp* (Table 2). Similarly, there was no statistical significance within the gravidity and parity of the pregnant women and *Taenia spp* infection ($p>0.05$). There was no statistical significance with the infection of *Taenia spp* and anaemia ($p>0.05$).

Prevalence of co-infection thus being infected with more than one parasite was 4.0%. The prevalence obtained was similar to a study made by Ivan *et al.,* (2013) who reported 6.6% coinfection whiles investigating prevalence of helminth and malaria infections in pregnant HIVpositive Rwandan women receiving anti-retroviral therapy (ART). The prevalence recorded in this study was below that of a study by Yatich *et al.,* (2009) who reported 16.6%.

Co-infection among the various age groups in this study were 0.8%, 2.4%, 0.8% and 0.0% for 15 - 19 years, 20 - 29 years, 30 - 39 years and 40 - 49 years respectively (Table 6). This trend was observed by Ivan *et al.* (2013). Though there was no statistical significance ($p > 0.05$) among the age groups, the subjects who were within 20 - 29 years showed the highest co-infection compared to the other age groups. Prevalence of co-infection related to gravidity and parity followed the same trend of 1.1%, 1.1% and 1.9% for primigravidae, secondagravidae and multigravidae women whiles the same trend was observed for nulliparous, primiparous and multiparous women. This is in contrast with observation made by Ivan *et al.,* (2013) who reported multigravidae women had a lower risk when compared to being primigravidae.

There was no association between co-infection and gravidity ($p>0.05$) and same for parity of study subjects ($p > 0.05$) (Table 6) even though parity and young has been indicated as factors associated with soil transmitted helminth and malaria Yatich *et al.,* (2009).

Co-infection anaemia in pregnancy may be aggravated by low nutritional status of subjects which may lack iron and folate needed during this period. Fuseni *et al.,* (2010) reported similar outcome of co-infection. A study in Nepal by Parul *et al.*, (2004) associated with anaemia caused by co-infection revealed a 3-fold increase risk to pregnancy. Anaemia among the present study population due to co-infection is statistically significant ($p>0.05$) (Table 2). Mechanisms by which helminthes and malaria affect haemoglobin are distinct but it can be suggested that their combined presence might enhance the risk of anaemia in preganant women.

Malaria is known to cause infection in approximately 216 million people globally WHO (2011) and also known to cause mortality of 1.2 million people in the year 2010 alone (Murray *et al*., 2010). Children and pregnant women are the most affected by the infection of malaria (Murray *et al.,* 2012).

This current study reported 16.5% of malaria infection among the pregnant women; this prevalence was much lower than studies by Yatich *et al*., (2006) and Amengor *et al.,* (2005) which reported a 36.3% and 35.1% respectively among pregnant women in Ashanti region in Ghana. The lower prevalence reported in the present study could be attributed to the fact that the study site falls within the low incidence zone of malaria in Ghana, Adams *et al.,* (2004) and Druilhe *et al.,* (2005) has previously speculated that geohelminths may trigger a T helper-2 response leading to the production of non-cytophylic clinically non-effective antibodies, which may delay the development of an effective immune response to malaria in pregnancy. Other studies by Leke *et al*., (1999) in Cameroon and Egwunyenga *et al.,* (2001) in Nigeria have also reported 21.9% and 38.8% respectively malaria infection among pregnant women.

Malaria infection was highest within the age group 20 - 29 years (8.5%), the second highest age group was 15 - 19 years (4.8%), then 30 - 39 years (2.9%) and the least 40-49 years (0.3%). There was a strong statistical significance within the age groups of the study population and malaria infection ($p<0.05$) (Table 6). This trend has been observed in studies by Yatich *et al.,* (2006) in Ghana and Bouyou-Akotet *et al*., (2003) in Gabon.

Prevalence of malaria infection with the gravidity of the pregnant women was highest among multigravidae women (8.8%), then primigravidae women (6.4%) and the least being secondagravidae women (1.9%). Even though there was a statistical significance within gravidity and malaria infection ($p< 0.05\%$) (Table 6), the trend observed in this current study was in contrast with studies by Shulman *et al.,* (1999) and Diagne *et al.,* (1997) who showed that primigravidae women are the most susceptible to malaria infection. However, Boel *et al.,* (2010) made an observation similar to the present study among women on Thai-Burmese Border. In the current study, the multigravids fell within the age group 20 - 29 years which may account for the contrast in the infection of malaria among this age group compared to other studies in Ghana.

Increased susceptibility of pregnant women to malaria occurs by the mechanism of sequestration in the placenta, which eventually escapes splenic clearance of the host (Fried and Duffy 1999). The ultimate consequence of this evasion is anaemia and low birth weight Brabin (1983).The observation of malaria infection and anaemia is in consonance with studies by Fuseini *et al.* (2010) and Saute *et al.,* (2002) in Ghana and Mozambique respectively.

CHAPTER 6

6.0 CONCLUSION AND RECOMMENDATION

6.1 CONCLUSION

The present study has revealed urinary schistosomiasis prevalence of (4.5%), stool helminth infection (29.0%) and co-infection of (4.0%) among the studied pregnant women sampled from Sege, Bonikope and Anyamam Health Centers all within the Dangme East District.

The predominant stool helminth was Ascaris *lumbricoides* (8.5%), followed by *Schistosoma mansoni* (7.5%), *Trichuris trichiura* (5.9%), Hookworm (4.0%), *Strongyloides stercoralis* (1.5%) with the least being. *Taenia spp*. (0.8%)

The differences in the rates of the Soil Transmitted Helminthes (*Ascaris lumbricoides, Trichuris trichiura*, Hookworm) and malaria infections, recorded among the studied pregnant women in the present study areas were statistically significant ($p < 0.05$) with risk factors such as age, gravidity and parity. Repercussions of parasitic infection on pregnant women and their foetus varies for asymptomatic infection to severe infection that may result in anaemia, malnutrition, retarded intrauterine growth and spontaneous abortion. In all, the effects of these infections depend on parasite load and immune status of the pregnant woman. An observation of the result shows the importance and potential negative impacts of these infections during pregnancy and the resultant outcomes of mild and moderate anaemia.

The studied pregnant women who were multigravid recorded the highest of all the parasitic infections (29.1%) and the least infected were secondagravid women (9.2%). Pregnant women in the age group 20 - 29 years were the most affected with schistosomiasis and helminth and also indicates a high number of the multigravids. The higher prevalence of schistosomiasis and

helminth at age range 20-29 years support the suggestions of some studies that all women of childbearing age including pregnant women in their 2nd and 3rd trimester could benefit from periodic antihelminthic treatment much as presumptive therapy for malaria infection is advised during late pregnancy. Multiparous women recorded the highest parasitic infection (27.3%) and they fell within the age group 20-29 years.

Anaemia recorded among the age groups, gravidity and parity was statistically significant ($p<0.05$) in the pregnant women in the present study. Personal hygiene and good nutrition plays a role in healthy pregnancy which should be highlighted in antenatal education to avoid infection and promote good health.

6.2 RECOMMENDATIONS

Due to the results obtained from the present study, it is recommended that the Ministry of Health in conjuction with other stakeholders should put measures in place to strengthen the screening women attending antenatal care for soil transmitted helminth at their first visit.

In order to prevent recurrence of parasitic infection, pregnant women should be advised to use footwear, improve sanitation and personal hygiene. Pregnant women should also be encouraged to have regular antenatal care follow up.Since it is likely many women enter pregnancies with these infections, control programmes should be considered well before pregnancy for preventive health education.

Use of mosquito insecticide nets should be intensified during pregnancy.

Deworming programmes during pregnancy should be considered aside the safety issues encountered to minimize infection. It is also recommended that, three stool samples should be obtained from each individual so as to effectively detect the parasites if present during future study.

Provision of proper adequate public (KVIPS) private toilets to prevent promiscuous open defecations in these communities**.** Future studies should be conducted using these parameters based upon the findings from the present study.

6.3 LIMITATIONS

Diagnosis of intestinal parasites was confirmed by the recovery of helminth eggs and larvae in the clinical parasitology laboratory. Due to the low density of parasites in the stool, the sensitivity, specificity and accurate microscopic identification of intestinal parasitic infections will depend on the methods used, the number of stools analysed and quantity of parasites excreted per sample. In the current study, formol-ether concentration method was used and the result was based on a single sample obtained from individual participants. If two or three samples from the study participants had been analysed, the sensitivity of the test results would have been improved.

Diethyl ether was used in place of ethyl acetate as feacal fat extraction solvent. This was due to challenges in obtaining ethyl acetate on the market. Ethyl acetate is a good feacal fat extractor as compared to diethyl ether because it provides clear sediment but may also end up extracting some of the parasites.

The present study did not take data on socioeconomic, behavioral or environmental factors for analysis.

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APPENDICES

Appendix 1.0 Assent form for the study

ASSENT TO PARTICIPATE IN A RESEARCH STUDY

PREVALENCE OF SCHISTOSOMIASIS AND SOIL TRANSMITTED HELMINTHIASIS AMONG PREGNANT WOMEN IN THE DANGME EAST DISTINCT, GREATER ACCRA REGION 15 – 18 YEAR OLD

My name is Emmanuel Agbeko Nani. I am a student in the School of Medical Sciences, Department of Clinical Microbiology at the Kwame Nkrumah University of Science and Technology. I am doing a research study. I would like to tell you about this study and ask if you will be take part in it.

What is a research study?

A research study is when people like me collect a lot of information about a certain thing to find out more about it. Before you decide if you want to be in this study, it is important for you to understand why I am doing the research and what is involved.

Please read this form carefully. You can discuss it with your parent or anyone else. If you have any questions about the research just ask me.

Why am I undertaking this study?

I am doing this study to find out more about parasitic infections during pregnancy and their effect on the pregnant person.

Why am I talking to you about this study?

To learn about parasitic infection during pregnancy, I would like to study about pregnant individuals around your age. I am inviting you to participate because you are pregnant.

What will happen if you are in this study?

Containers will be given to you for urine and stool samples. Blood sample will be obtained from your arm, the quantity will be about one (1) tea spoon. Collection of blood sample will be uncomfortable but very brief which will span between one minute to three minutes. In all the whole procedure will take about fifteen (15) minutes. All these will be done by a qualified laboratory technician.

If you agree to be in the study and your parents give permission, I will ask you to:

Answer questions

On the first day, you and your parents will come to our laboratory. We will ask you and your parent to answer some questions on what you to as profession, where you live, what water source you use for daily activities and place of convenience you use. If you do not want to answer any of the questions, you do not have to.

This part will take about 5 minutes.

Will you get healthier if you are in the study?

There will be an intervention in terms of drug supply in relation to the regulation with regards to your condition. In directly, the clinician or midwife will use the outcome of the laboratory test to determine your haemoglobin status which will benefit the unborn child.

Will any part of the study be uncomfortable or hurt?

Blood draining: getting your blood drained can hurt for a few seconds from the needle stick going in like when you get a shot at the doctor's office.

Afterwards, you might get a little bruise. Sometimes an infection can develop there but that hardly ever happens.

Who will know about your study participation?

Besides you and your parents / guidance, the researchers are the only ones who will know about your study participation. If I publish reports or give talks about this research, it will only discuss group results. I will not use your name or any personal information that would identify you. To help protect confidentiality, I will give your study data a code number and keep it in a file with a password that only the researchers know. The file will be on a computer that only the researchers are allowed to use.

We plan to keep this information for ten (10) years, in case we or other researchers want to use it later for other studies. But we will follow the same steps we just described to keep it as confidential as possible.

Will you get paid for being in the study?

You will not be paid for being in this study, you will receive a snack refreshment as a thank you for your time and effort to take part in this study. The snack will be given immediately you complete the questionnaire and your blood is drawn.

Do you have to be in this study?

No you don't. Research is something you do only if you want to. No one will get mad at you if you do not want to be in the study. And remember, you can always change your mind later if you decide you do not want to be in the study any more.

Do you have any questions?

You can ask questions about this study anytime, now or later. You can talk to me, or your parents or someone else at any time during the study. You can contact me Emmanuel Agbeko Nani at 0244-755979 or email kwame.king@yahoo.com or you can contact Dr. S. K. Tay at 0245670710.

Assent of adolescent (14 – 17 years old)

If you decide to participate and your parents agree, we will give you a copy of this form to keep for future reference. If you would like to be in this research study, please sign your name on the line below:

…… ……

Subject's name / signature (written by adolescent) Date

…… ……

Signature of investigator / person obtaining assent Date

Appendix 2.0 Materials used for the study

2.1. Equipments and Reagents

i. Binocular Microscope with X10, X40 and X100 objective lens ii. Centrifuges which can hold 15cm Falcon tubes and has a speed regulator and timer.

iii. Sodium Acetate acetic Formalin (SAF): To prepare 1liter of SAF solution, 15 g of sodium acetate was weighed into a 1L volumetric flask, 40 ml of Formalin, since we do not add water to acid but acid to water, 925ml of distilled water was added to the content in the volumetric flask and 20ml of glacial acetic acid was finally added and the mixture thoroughly mixed.

iv. Diethyl ether

v. Physiological saline (0.85% NaCl) vi. Absolute methanol

2.2. Other Materials used

i. Glass slides (26x76mm) ii.

Cover slips (22x 22mm) iii.

Wood applicators

iv. Gauze

v. Disposable pipettes

vi. Gloves vii. Test tube racks viii. Funnel ix. Disinfectants (70% ethanol)

Appendix 3.0.Direct Wet Mount Method

A small amount of the stool sample (about 2 mg) received was transferred onto a clean glass slide (26x76 mm) and about 1-2 drops of physiological saline was added and emulsified. The mixture on the glass slide was covered with a cover slip (22x22 mm) and examined microscopically for Ova, trophozoites, cysts and larva of intestinal parasites using X10 and X40 objective lens.

Appendix 4.0.Formol Ether concentration Method

A Falcon centrifuge tubes were prepared and labelled. A small stool sample with at least the size of a peanut was transferred to the labelled falcon tube. 7 ml of sodium acetate acetic formalin solution (SAF) was added to the tube falcon tube containing the stool sample. The tube was shaken thoroughly. Using a funnel, the stool sample in the labelled tube was poured through sieve with very small pore space into another labelled falcon centrifuge tube. The labelled falcon tubes containing the sieved stool samples were placed in a centrifuge well balanced. The tube containing the sample was centrifuge at 2000 rpm for a minute. The supernatant was discarded. 7 ml of 0.85% physiological saline solution and 2-3ml of diethyl ether was added to the filtrate.

The falcon tube containing the mixture was corked and shaken thoroughly for 2-3minutes. After shaken the cork was loosen to get rid of the gas in the tube. The tube containing the mixture was centrifuged at 2000 rpm for 3 minutes. Four layers was formed and the top three layers were discarded. A small portion of the sediment was transferred onto a clean glass slide (26x76mm) and

covered with a cover slip (22x22mm) and was examine microscopically for Ova, cyst of parasites using X10 and X40 objective lens.