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DEPARTMENT OF CLINICAL MICROBIOLOGY

A COMPARATIVE STUDY OF INTESTINAL PARASITIC INFECTION AND ASSOCIATED RISK FACTORS AMONG PRIMARY SCHOOL CHILDREN IN SIX NEIGHBOURING COMMUNITIES IN KUMASI, GHANA: AYIGYA, KENTINKRONO, ABOABO, MANHYIA, GYINYASE AND KYIRAPATRE

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MASTER OF SCIENCE DEGREE (MSC.) IN CLINICAL MICROBIOLOGY

BY

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DECLARATION

I hereby declare that this submission is my own work towards the M.Sc. 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. Paul Tetteh and Mrs. Vida Tetteh. I also dedicate this work to my beloved siblings: Eunice Tetteh, Michael Tetteh, Esther Darley Tetteh and Elizabeth Amponsah Tetteh for their encouragement, love, advice, prayers and support.

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My ultimate thanks go to the Almighty God for giving me strength and seeing me through this research work successfully.

The completion of this work was as a result of the enormous effort and contributions of some following persons.

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ABSTRACT

Intestinal parasitic infection remains one of the conditions that have serious consequences on child health development especially in developing countries. The aim of this study was to compare the intestinal parasitic infections and associated risk factors among primary school children (5-12 years) within the Kumasi metropolitan area. A cross sectional survey was conducted in six communities (Ayigya, Kentinkrono, Aboabo, Manhyia, Gyinyase and Kyirapatre). Fresh stool samples were obtained from primary school children within these communities from January to September, 2011. The stool samples were analysed using direct wet mount preparation and formol ether concentration methods for identification of protozoan trophozoites and cysts, helminth ova and larva. Modified Kinyoun staining method was performed on a smear prepared from the fresh stool samples for the identification of oocysts of *Cryptosporidium parvum*. Out of 2400 questionnaires administered, 1162 (48.42%) were administered to males and 1238 (51.58%) to females. The overall intestinal parasitic infections rate recorded in the present study was 49.18%. A total of 8.20% of the primary school children were infected with more than one intestinal parasite. Of the overall prevalence rate of intestinal parasitic infections 12.17% had *Giardia lamblia*, 0.21% had *Entamoeba histolytica*, 8.50% had *Cryptosporidium parvum*, 10.33% had *Entamoeba coli*, 7.04% had *Endolimax nana*, 3.17% had *Chilomastix mesnil*i, 1.46% had *Iodamoeba butschlii*, 1.54% had Hookworm, 3.88% had *Ascaris lumbricoides* and 0.88% had *Strongyloides stercolaris* were recorded among children sampled in the six communities. *G. lamblia* (12.17%) and *A. lumbricoides* (3.88%) infections were the predominant intestinal protozoan and helminth parasites respectively. The pathogenic protozoa (especially *Giardia lamblia* and *Cryptosporidium parvum*) and helminths (Hookworm and *Ascaris lumbricoides*) recorded

among the studied children in the present study areas were statistically significant ($p < 0.05$) to risk factors such as gender, age, poor environmental sanitation, improper personal hygiene practices, and socioeconomic activities (especially studied children whose parents are farmers and unemployed). Primary school children from Kentinkrono recorded the highest of all intestinal parasitic infections (74.75%) in the present study. Males (65.66%) recorded the highest of intestinal parasitic infections than females (34.34%) among the studied population. The age groups most affected were 5-6years, followed by 7-8years, 9-10years and 11-12years among the studied children in all the communities.

The age groups most affected are 5-6years, followed by 7-8years, 9-10years and 11-12years among the studied children in all the communities. The Questionnaire assessment revealed that studied children who lack sanitary facilities, lack of knowledge of personal hygiene practices, lack of access to good drinking water, buy food from food vendors around their school compounds and those whose parents were farmers and unemployed in the six communities were most infested with intestinal parasitic infections.

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CHAPTER ONE

1.0. INTRODUCTION

1.1. BACKGROUND INFORMATION TO THE STUDY

Intestinal parasitic infections (IPIs) are endemic worldwide and have been described as constituting the greatest single worldwide cause of illness and disease (Quihui *et al.*, 2006; Sehgal *et al.*, 2010). Poverty, illiteracy, poor hygiene, lack of access to potable water and warm and humid tropical climate are some factors associated with intestinal parasitic infections (Olstein, 2001; Adamu *et al.*, 2006; Noor *et al.*, 2007; Sehgal *et al.*, 2010). Parasitic protozoa and helminths are responsible for some of the most devastating and prevalent diseases such as intestinal or biliary obstruction, growth retardation and iron deficiency anaemia that affect humans (Ahmed *et al.*, 2003; Quihui *et al.*, 2006). Intestinal parasitic infections (IPIs) constitute a global health burden causing clinical morbidity in 450 million people; many of these are women of reproductive age and children in developing countries (Quihui *et al.*, 2006). Intestinal parasitic infection is one of the major health problems in developing countries (WHO, 2000). It has been estimated to affect some 3.5 billion people globally and 450 million are thought to be ill as a result of such infections, the majority being children (WHO, 2000). Intestinal parasitic infections, mostly helminths, have been linked with an increased risk for nutritional anaemia, protein-energy malnutrition and growth deficits in children, loss of weight in pregnancy and intrauterine and growth retardation followed by low birth weight (Sackey *et al.*, 2003; Rodriguez-Morales *et al.*, 2006).

Approximately 3 billion people globally are infected with helminths (Maizels et al., 2003).

Epidemiological surveys have revealed that, poor sanitation and inappropriate environmental conditions coupled with indiscriminate defaecation, geophagy and contamination of water

bodies are the most important predisposing factors to intestinal worm infestation (Brooker *et al*., 2008). The prevalence and intensity of infection is especially high in developing countries, particularly among populations with poor environmental sanitation (van Eijk *et al*., 2009). Other practices such as improper hand washing, disposal of refuse, personal hygiene and not wearing shoes may contribute to the infection (Stoltzfus *et al*., 1997). *Entamoeba histolytica, Giardia lamblia* and *Cryptosporidium parvum* are the three of the most common intestinal protozoan parasites infecting humans worldwide. They are known to be the most important diarrhoea-causing protozoa (Marshall *et al.*, 1997).

It is estimated that 10% of the world's population are infected with *E. histolytica* with the highest prevalence in developing countries (Vandenberg *et al.*, 2006). Global statistics on the prevalence of *E. histolytica* infection indicates that 90% of infected individuals remain asymptomatic carriers while the other 10% develop clinically apparent disease (Ayeh-Kumi *et al.*, 2001). This results in 50-100 million cases of colitis or liver abscesses per year and up to 100,000 deaths annually (Ayeh-Kumi *et al.*, 2001). Rates of 20-40% of the global burden *of G. lamblia* are reported in developing countries, especially in children (Vandenberg *et al.*, 2006). Earlier studies conducted by Annan *et al*. (1986) among pre-school children in Ghana however revealed up to 18.2% *Giardia* infection although Verweij *et al*. (2003) reported 21.5% among children in Northern Ghana. Prevalence of cryptosporidiosis in Asia and Africa ranges from 5-10% (Vandenberg *et al.*, 2006). Recent global estimates indicate that more than a quarter of the world's population are infected with one or more of these parasites; the roundworm, *Ascaris lumbricoides*; the hookworms, *Necator americanus* and *Ancylostoma duodenale*; and the whipworm, *Trichuris trichiura* (Chan *et al.,* 1994).

For many years, the need to control these infections has been uncontested, and with the advent of broad-spectrum anthelminthic drugs that are cheap, safe and simple to deliver,

control has at last become a viable option for many communities (Drake *et al.*, 2001). The World Health Organization reports a 35 percent infection rate for roundworm, which is a common parasitic worm (WHO, 2000). Several studies have reported the association of intestinal parasitic infections with other sets of risk factors (WHO, 2000). A study in Kenya identified risk factors associated with intestinal helminths infections using a combined qualitative method (Olsen *et al.*, 2001). It revealed that absence of latrine and household without soap were significant predictors of these worm infections (Olsen *et al.*, 2001). The results of the qualitative part of the study suggested that latrines and personal hygiene were important preventive measures (Olsen *et al.*, 2001). In southern Thailand, the statistically significant protective factor for hookworm infection was wearing of shoes (Chongsuvivatwong *et al.*, 1996). Infections with *A. lumbricoides* and *T. trichiura* were found to be associated with socio-demographic variables in Honduran communities (Jolly, 2001). Furthermore, for the high risk group, i.e. school-aged children, a guide for managers of control programs for soil-transmitted helminthiasis and schistosomiasis emphasized the importance of intensive learning, awareness of good personal hygiene, and adequate nutrition (Montresor, 2002).

1.2. STATEMENT OF PROBLEM

The morbidity of intestinal parasitic infections is greatest among children of school going age and may have adverse effect on their growth (Nematian *et al.*, 2008 and Brooker *et al*, 1999). Intestinal parasitic infections are a major problem in children from developing countries. It causes nutritional deficiencies and anaemia in children, especially when hookworm infestation is present (Ahmed *et al.*, 2003 and Ananthakrishnan *et al.*, 1997). The most common effect of intestinal parasitic infection on the health of children affect the normal physical development, resulting in children failing to achieve their genetic potential for

growth and having the clinical consequences of iron deficiency anaemia and other nutritional deficiencies (Brooker *et al*., 2008). Intense whipworm infection in children may result in Trichuris dysentery syndrome, the classic signs of which include growth retardation and anaemia, (Bundy *et al.,* 1989) and heavy burdens of both roundworm and whipworm are associated with protein-energy malnutrition (Stephenson *et al.*, 1993).

1.3. JUSTIFICATION OF THE STUDY

Due to high morbidity associated with intestinal parasitic infections in children, it will be very prudent to undertake this research work so as to determine rate of parasitic infections among these primary school children in the said communities and the associated risk factors. Most children who are asymptomatic may serve as a source of infection to other children. This research will help determine children who are asymptomatic but are carriers of parasitic infections so that they can be treated immediately to prevent the spread of the infection. The results which would be obtained from the research work will help the Ministry of Health, the Town Development Council and stakeholders to provide the necessary interventions such as school deworming programmes, health education and other preventive measures so as to minimize the burden of parasitic infection within the study areas.

1.4. HYPOTHESIS

Younger children are more likely to suffer from intestinal parasitic infections compared to older children because they easily come in contact with contaminated water and food, faeces and other source of infection through play and other behaviour.

1.5. GENERAL OBJECTIVE OF THE STUDY

The general objective of this research work was to compare the rate of intestinal parasitic infection and associated risk factors among primary school children in six neighbouring communities in Kumasi: Ayigya, Kentinkrono, Aboabo, Manhyia, Gyinyase and Kyirapatre.

1.5.1. Specific objectives of the study

The specific objectives of this study were:

1. To identify and determine the various intestinal parasitic infections in the faecal samples of primary school children within Ayigya, Kentinkrono, Aboabo, Manhyia, Gyinyase and Kyirapatre communities in Kumasi.
2. To determine the association between intestinal parasitic infections and risk factors among primary school children in the above mentioned communities.
3. To compare the distribution of intestinal parasitic infections with age and sex among primary school children in the above mentioned communities.

1.6. ETHICAL CLEARANCE

Ethical approval was obtained from the Ethics and Research committee of Komfo Anokye Teaching Hospital. Permission to conduct the study in the schools was sought from the primary school head teachers and a parent through written consent.

CHAPTER TWO

2.0. LITERATURE REVIEW

2.1. INTESTINAL PARASITIC INFECTIONS

Intestinal parasitic infections (IPIs) enjoy a wide global distribution. They are estimated to affect an estimated 3.5 billion people, most of whom are children residing in developing countries (WHO, 2000). The major intestinal parasitic infections of global public health concern are the protozoan species *Entamoeba histolytica* and *Giardia lamblia* and soil transmitted helminths *Ascaris lumbricoides, Trichuris trichiura* and hookworm(*Necator americanus* and *Ancylostoma duodenale*) (WHO, 1999; WHO, 2000). The incidence and prevalence of these parasitic pathogens vary both between and within countries. The majority of intestinal parasitic infections are associated with poverty conditions such as reduced access to safe drinking water, poor sanitation and hygiene, housing and inadequate access to health care (Mata, 1982; Montresor *et al.*, 1998). They are also influenced by poor family and community hygiene as well as prevailing climatic and environmental conditions (Jemaneh, 1998). These conditions lay the stage for the continuous transmission of the IPIs (Mata, 1982; Montresor *et al.*, 1998; Crompton 1999).

2.2. INTESTINAL PROTOZOAN INFECTIONS

Protozoans are extremely diverse group of unicellular organisms occurring in almost all ecological niches known to humans, including the bottom of hot spring and the edges of ice flows (Melhorn, 1988; Katz *et al.*, 1989). Even though the majority of protozoa occur as free-living organisms in the soil, moist, marine or fresh water environments, a substantial number also exist as mutualist, commensals or parasites (Mehlhorn, 1988; Katz *et al.*, 1989). Protozoan parasites are known to affect all species of vertebrates and many invertebrates. They are able to adapt to life in virtually all body sites of their hosts. Their characteristic

high infectivity enhances their pathogenicity within the host (Katz *et al.*, 1989; Neva and Brown, 1994).

2.3. INTESTINAL AMOEBAE

The amoebae which inhabit the intestinal tract of humans belong to the *Entamoeba, Iodamoeba* and *Endolimax* genera. The non-pathogenic members of these groups include *Entamoeba dispar, Entamoeba gingivalis, Entamoeba coli, Entamoeba hartmani, Endolimax nana,* and *Iodamoeba butschlii* (Mahon and Manuselis 2000; Washington *et al*., 2006). The pathogenic member is *Entamoeba histolytica* (Mahon and Manuselis 2000; Washington *et al*., 2006).

2.4. ENTAMOEBA HISTOLYTICA INFECTION

2.4.1. Epidemiology of *Entamoeba histolytica*

Entamoeba histolytica is estimated to infect about 50 million people worldwide (Ryan and Ray, 2004). An estimated 10% of the world's population is infected with *E. histolytica*, with higher rate occurring in developing countries where sanitation is low (Chacon-cruz, 2009). This results in 50-100 million cases of colitis or liver abscesses per year and up to 100,000 deaths annually resulting in a mortality rate of 1 in 500-1000 diagnosed cases (Ayeh-Kumi *et al.*, 2001; Chacon-cruz, 2009).

The prevalence of *Entamoeba* infection is as high as 50% in areas of Central and South America, Africa, and Asia. In Egypt, 38% of individuals presenting with acute diarrhoea to an outpatient clinic were found to have amoebic colitis (Stanley, 2003). *E. histolytica* sero prevalence studies in Mexico revealed that more than 8% of the population were positive (Caballero-Salcedo *et al.*, 1994). Asymptomatic *E. histolytica* infections seem to be region-dependent and may be as high as 11% in Brazil. However, the introduction of molecular

techniques, it is estimated that 500 million individuals with *Entamoeba* infection are colonized by *E. dispar* (Fotedar *et al.*, 2007*).*

Global statistics on the prevalence of *E. histolytica* infection indicates that 90% of infected individuals are asymptomatic carriers while the remaining 10% develop clinically apparent disease (Li *et al.*, 1996; Stanley, 2003; Fotedar *et al.*, 2007). Amebiasis is second only to malaria in terms of protozoa-associated mortality (Li and Stanley, 1996; Stanley, 2003; Fotedar *et al.*, 2007). The combined prevalence of amoebic colitis and amebic liver abscess is estimated at 40-50 million cases annually worldwide, resulting in 40,000-100,000 deaths (Li and Stanley, 1996; Stanley, 2003; Fotedar *et al.*, 2007).

Amoebic colitis evolves to fulminant necrotizing colitis or rupture in approximately 0.5% of cases. The mortality rate due to amoebic liver abscess has fallen to 1-3% in the last century following the introduction of effective medical treatment (Stanley, 2003). Nevertheless, amoebic liver abscess is complicated by sudden intraperitoneal rupture in 2-7% of patients, leading to a higher mortality rate (Stanley, 2003).

2.4.2. Morphology and Life cycle of *Entamoeba histolytica*

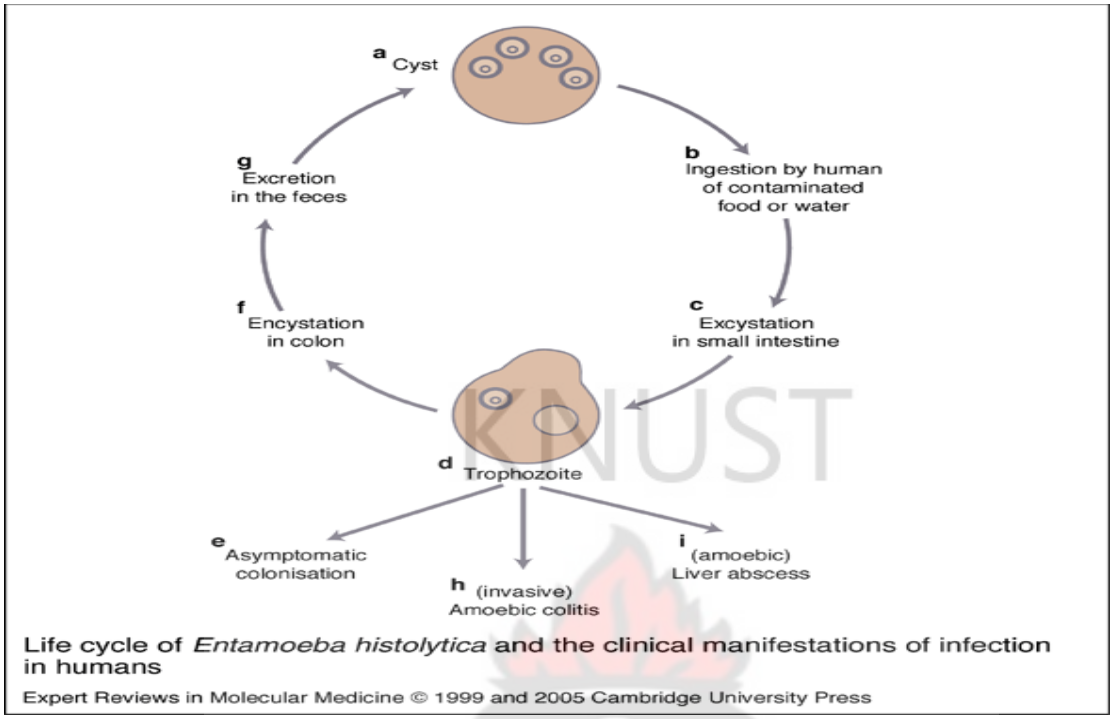


Figure 2.1: Lifecycle of *Entamoeba histolytica*

Huston *et al.* (1999) have described the life cycle of *E. histolytica* (Fig 2.1).

It consists of two stages: cysts and trophozoites (Huston *et al.*, 1999). Fig 2.1 shows that cysts measure 10–15 mm in diameter and typically contain four nuclei; (b) they are spread via the ingestion of faecally contaminated food or water (Huston *et al.*, 1999); (c) During excystation within the lumen of the small intestine, nuclear division is followed by cytoplasmic division, giving rise to eight trophozoites (Huston *et al.*, 1999); (d) Trophozoites, which measure 10–50 mm in diameter and contain a single nucleus with a central karyosome, reside in the lumen of the caecum and large intestine, where they adhere to the colonic mucus and epithelial layers (Huston *et al.*, 1999); (e) Approximately 90% of individuals infected with *E. histolytica* are asymptomatically colonised; (f) re-encystation of the trophozoites occurs within the lumen of the colon, resulting in (g) the excretion of cysts in the faeces and continuation of the life cycle (Huston *et al.*, 1999).(h) Alternatively, the trophozoites can

invade the colonic epithelium, causing amoebic colitis (in approximately 10% of infected people). Amoebic dysentery usually occurs gradually, with symptoms [such as abdominal pain and tenderness, and painful sudden bowel evacuation (tenesmus) and diarrhoea] developing over a period of one to several weeks, often followed by weight loss (Huston *et al.,* 1999). *E. histolytica* can spread in the bloodstream (haematogenously) after it has penetrated the colonic epithelium and can establish persistent extraintestinal infections, most commonly (i) amoebic liver abscess. Liver abscess is overwhelmingly the most common extraintestinal manifestation of amoebiasis (Huston *et al,* 1999). This complication is ten times more common in adult men than in adult women (Huston *et al.,* 1999).

2.4.3. Pathogenesis of *Entamoeba histolytica*

Entamoeba histolytica is a pseudopod-forming, nonflagellated protozoa parasite that causes proteolysis and tissue lyses (hence its name) and can induce host-cell apoptosis. Humans and perhaps nonhuman primates are the only natural hosts (Haque *et al.,* 2006). Ingestion of *E histolytica* cysts from the environment is followed by excystation in the terminal ileum or colon to form highly motile trophozoites (Haque *et al.,* 2006). Upon colonization of the colonic mucosa, the trophozoites may encyst and is then excreted in the faeces or may invade the intestinal mucosal barrier and gain access to the blood stream and disseminate to the liver, lung, and other sites. Excreted cysts reach the environment to complete the cycle (Haque *et al.,* 2006).

Disease may be caused by only a small number of cysts, but the processes of encystation and excystation are poorly understood. The adherence of trophozoites to colonic epithelial cells seems to be mediated by a galactose/*N* -acetylgalactosamine (GAL/GalNAc)–specific lectin (Stanley, 2003; Ravdin *et al.*, 1989). A mucosal immunoglobulin A (IgA) response against

this lectin can result in fewer recurrent infections (Haque *et al.,* 2006). Both lytic and apoptotic pathways have been described. Cytolysis can be undertaken by amoebapores, a family of peptides capable of forming pores in lipid bilayers (Stanley, 2003). Furthermore, in animal models of liver abscess, trophozoites induced apoptosis via a non-Fas and non–tumour necrosis factor-α1 receptor pathway (Seydel *et al.,* 1998). The amoebapores, at sublytic concentrations, can also induce apoptosis (Seydel *et al.,* 1998). Cysteine proteinases have been directly implicated in invasion and inflammation of the gut and may amplify interleukin (IL)–1–mediated inflammation by mimicking the action of human IL-1–converting enzyme, cleaving IL-1 precursor to its active form (Stanley, 2003; Que and Reed 2000).

Epithelial cells also produce various inflammatory mediators, including IL-1B, IL-8, and cyclooxygenase-2, leading to the attraction of neutrophils and macrophages (Seydel *et al.,* 1998; Stensen *et al.,* 2001). Corticosteroid therapy is known to worsen the clinical outcome, possibly because of its blunting effect on this innate immune response (Braga *et al.,* 1992). Additional host defences, including the complement system, could be inhibited directly by the trophozoites, suggested by the finding that a region of the GAL/GalNAc–specific lectin showed antigenic cross reactivity with CD59, a membrane inhibitor of the C5b-9 attack complex in human red blood cells (Braga *et al.,*1992). Trophozoites that reach the liver create unique abscesses with well-circumscribed regions of dead hepatocytes surrounded by few inflammatory cells and trophozoites and unaffected hepatocytes, suggesting that *E histolytica* are able to kill hepatocytes without direct contact (Stanley, 2003).

E. dispar and *E. histolytica* cannot be differentiated by direct examination, but recent molecular techniques established them as two different species, with *E dispar* being

commensal (including in patients with HIV infection) and *E. histolytica* pathogenic (Fotedar *et al.,* 2007). In fact, it is now estimated that many individuals with *Entamoeba* infections are colonized with *E. dispar*, which appears to be 10 times more common than *E. histolytica* (Fotedar *et al.,* 2007). However, in certain regions (eg, Brazil, Egypt), asymptomatic *E. dispar* and *E. histolytica* infections are equally prevalent (Stanley, 2003). In Western countries, approximately 20%-30% of men who have sex with men are colonized with *E. dispar* (Fotedar *et al.,* 2007).

2.4.4. Clinical Manifestation of *Entamoeba histolytica*

Disease presentations range from mild to severe or toxic and the course of the infection may vary in different communities based on disparities in development, standards of living, sanitation, and the nutritional status of infected persons (Petri *et al.,* 1993). Amoebic colitis may masquerade as shigellosis, inflammatory bowel disease, ischemic colitis, diverticulitis, arteriovenous malformations or carcinoma of the colon (Petri *et al.,* 1993).

Amoebic Colitis often mimics other enteric infections. Manifestations may include abdominal pain, dysentery, bloody/mucoid stools, diarrhoea, colonic lesions/perforations, ameboma (resulting from intra-luminal granulation of tissue), toxic megacolon, peritonitis, amebic appendicitis and cecitis, cutaneous amebiasis, rectovaginal amoebic cutis, hemorrhage, and rectovesicular fistulas (de Villiers *et al.,* 1998). Unlike bacterial dysentery, amoebic colitis has a gradual onset that ranges from 1 to several weeks of varied symptoms, including fever in a minority of cases (de Villiers *et al.,* 1998). It is often accompanied by weight loss. Colonic lesions may manifest as thickening of the mucosal wall, flask-shaped ulcerations, or necrosis of the intestinal wall depending on the degree of invasion (de Villiers *et al.,* 1998).

The most severe form of amoebic colitis is acute necrotizing colitis with toxic megacolon, which often requires surgical intervention. It occurs in 0.5% of cases and is suspected to be associated with more than 40% of the deaths from complications of amoebic colitis (de Villiers *et al.*, 1998). It is important to note, however, that some of the cases of toxic megacolon may have been caused by treatment with corticosteroids as a result of misdiagnosis (de Villiers *et al.*, 1998).

2.4.5. Intestinal Complications

Intestinal infection with *E. histolytica* can be classified either as invasive amoebic colitis or as asymptomatic colonization without pathophysiologic consequences. About 90% of intestinal colonization is with the non-pathogenic species, *E. dispar*, and invasive amoebiasis tends to develop in only 10% of persons colonized with *E histolytica* (de Villiers *et al.*, 1998).

A significant proportion of patients with liver abscess often present with no signs of dysentery, diarrhoea, or colitis (Archampong *et al.*, 1973). Helpful clues include elevated leukocyte counts and alkaline phosphatase levels and in some case an elevated right hemidiaphragm (Archampong *et al.*, 1973).

2.4.6. Extraintestinal Complications

Extraintestinal complications of amoebic infection include amoebic liver abscess, splenic and brain abscess, empyema, and pericarditis (Petri *et al.*, 1993; Lushbaugh *et al.*, 1988). Of these presentations, hepatic involvement in the form of amoebic liver abscess (ALA) is the most common. In areas where amoebiasis is endemic, ALA is much more common in men than in women or children (Nazir and Moazam, 1993).

Clinical presentations may be acute or subacute and often reflect the size, site, number of lesions, and involvement of adjacent tissues (Adams *et al.,* 1977; Barnes *et al.,* 1987; Seeto and Rockey, 1999). Acute presentations often manifest after 1 to 2 weeks as fever, cough, unlocalized abdominal pain and tenderness, and pain in the right upper quadrant. Abdominal pain, exquisite tenderness, enlarged liver, weight loss, and less prominent fever may be hallmarks of superficial or subacute presentation with or without capsular involvement (Adams *et al.,* 1977; Barnes *et al.,* 1987; Seeto and Rockey, 1999). On average, about 80% of cases of amoebic liver abscesses affect the right lobe and could easily be mistaken for pyogenic abscess on the basis of location. Abscesses located in the left lobe may go undetected because of atypical manifestations (Archampong *et al.,* 1973; Petri *et al.,* 1993). Abscesses occur most often in patients older than 50 years; diabetes mellitus is a risk factor, and patients may have jaundice, pruritus, sepsis, or shock with a palpable mass (Petri *et al.,* 1993).

Evidence pointing to an amoebic abscess would include history of immigration from or travel to developing countries and presence of circulating amebic antigen (Archampong *et al.,* 1973). Many patients with amoebic liver abscess have a history of dysentery and alcoholism. Differential diagnosis of amoebic abscess should of necessity include pyogenic liver abscesses, necrotic hepatoma, and echinococcal cyst (hydatid cyst, which often is an incidental occurrence) (Archampong *et al.,* 1973). The most commonly encountered complications of amoebic liver abscesses are rupture, pleuropulmonary involvement, and secondary bacterial infections (Archampong *et al.,* 1973). Rupture into the peritoneum occurs in between 2% and 10% of cases, leading to intraperitoneal spillage of pus (Archampong *et al.,* 1973).

2.4.7. Laboratory Diagnosis of *Entamoeba histolytica*

Microscopic identification remains the most common routine means of diagnosis used in areas where amoebiasis is endemic (Zhang *et al.,* 1994; Haque *et al.,* 1998). However, it is both insensitive and nonspecific (*E histolytica* cannot be distinguished from *E dispar* morphologically (Zhang *et al.,* 1994; Haque *et al.,* 1998). Motile trophozoites with engulfed red blood cells in emulsified fresh stools, rectal smears or rectosigmoidoscopy materials, and pus from liver abscesses or colonic biopsy samples are relatively specific but not sensitive findings for the identification of *E histolytica (*Gonzalez-Ruiz *et al.,* 1994.)

Several different enzyme-linked immunosorbent assays (ELISAs) kit for antigen detection in specimens has been developed to serve as adjuncts to microscopy (Haque *et al.,* 2000). Of the available kits, the *E histolytica* II stool antigen detection test (TechLab, Blacksburg, Va), which distinguishes *E histolytica* from *E dispar*, is currently the only test that meets the World Health Organization recommendation for *E histolytica*-specific diagnosis (Haque *et al.,*2000). Other available antigen detection kits are not selective for *E histolytica (*Mirelman *et al*, 1997). The TechLab kit is only effective on fresh or frozen stool specimens but not formalin-preserved specimens (Haque *et al.,* 2000). It is recommended that patients who present with dysentery and are at risk of acquiring amoebic infection should initially be screened with a combination *E histolytica* ELISA test if available(Huston *et al.,*1999).

Serological testing is useful for the diagnosis of invasive Amebiasis. The indirect haemagglutination test is usually positive in patients with invasive disease but is frequently negative in asymptomatic individuals who are passing cyst (Levinson, 2008).

Polymerase Chain Reaction (PCR) assay are powerful, highly sensitive, and useful for the differentiation of *E histolytica* and *E dispar* and for genetic typing of isolates. A number of PCR-based methods have been developed (Tannich *et al.*, 1989; Tachibana *et al.*, 1992; Clark and Diamond, 1992; Clark and Diamond, 1993). However, this method is time-consuming and expensive and requires special skills and practical experience. Hence, it is not yet practical for clinical diagnosis in regions of endemic amoebiasis that have limited resources (Tannich *et al.*, 1989; Tachibana *et al.*, 1992; Clark and Diamond, 1992; Clark and Diamond, 1993).

Colonoscopy or sigmoidoscopy may be used for the diagnosis of amoebic colitis. Colonoscopy is preferable because the infection may be focal and localized in the caecum or ascending colon and therefore may go undetected by sigmoidoscopy (Petri *et al.*, 1993). Biopsy specimens or aspirated material could be examined by microscopy for motile trophozoites (Petri *et al.*, 1993). The use of cathartics and enemas in the preparation of patients for examination should be avoided, since these procedures often interfere with morphologic identification of the parasite (Petri *et al.*, 1993).

2.4.8. Treatment of *Entamoeba histolytica*

Treatment may require multiple drugs and different regimens to eradicate the parasite. Antiamoebic drugs may be divided into 2 groups: luminal and tissue agents (Petri *et al.*, 1993). Asymptomatic intestinal colonization with *E. histolytica* can be treated with luminal agents alone, and asymptomatic infection with *E dispar* does not require any treatment (Petri *et al.*, 1993). Drugs used for the treatment of luminal infections and generally prescribed for asymptomatic cyst passers include iodoquinol, diloxanide furoate, and paromomycin (Petri *et al*, 1993; Huston *et al.*, 2001).

Metronidazole is the drug of choice for the treatment of tissue-invasive disease (colitis and liver abscess) and is highly effective (greater than 90% cure rate) (Ayeh-Kumi *et al.*, 2001). To confirm eradication of the parasite, all patients, regardless of clinical manifestations of amoebiasis, should be screened with the stool antigen test after treatment (Ayeh-Kumi *et al.*, 2001).

2.4.11. Prevention and Control of *Entamoeba histolytica*

The prevention and control of amoebic infections, as in programmes to control other water and food borne diseases, can be affected through a multifaceted approach that requires both individual and corporate involvement (Ayeh-Kumi *et al.*, 2001). Education on public and personal hygiene, proper disposal of human faeces should be sustained. There should be provision of good water supply devoid of faecal contamination. Education of high-risk groups on sexual and other habitual practices that promote faecal-oral transmission should be promoted (Ayeh-Kumi *et al.*, 2001).

2.5. INTESTINAL FLAGELLATES

Chilomastix mesnili, *Retortamonas intestinalis*, *Enteromonas hominis* and *Trichomonas hominis* are some of the intestinal flagellates which infect man. *Giardia lamblia* is the only intestinal flagellate that is considered to be pathogenic (Washington *et al.*, 2006).

2.6. GIARDIA LAMBLIA

2.6.1. Epidemiology of *Giardia lamblia*

According to World Health Organisation (WHO) estimation, globally there are 200 million cases of Giardiasis (Showkat *et al.*, 2010). The organism has a worldwide distribution and is a major cause of epidemic childhood diarrhoea in developing countries. Prevalence rates

vary from 4 to 42%. It is the most commonly isolated intestinal parasite throughout the world. Rates 20-40% are reported in developing countries, especially in children (Chacon-cruz, 2009). In the United States, rate of prevalence varies from 3-13%. Giardiasis is one of several causes of traveller's diarrhoea. Person-to-person transmission is associated with groups that exercise poor faecal-oral hygiene, such as children in childcare centers (Chacon-cruz, 2009).

Giardia lamblia is endemic in childcare centers and can be detected in approximately 20% of asymptomatic children. The attack rates of *G. lamblia* during outbreaks in childcare centers range from 20 to 50% (Chacon-cruz, 2009). A study conducted in Bangkok among children with diarrhoea and those without gastrointestinal symptoms recorded 2% and 1.3% *G. lamblia* infection respectively (Showkat *et al*., 2010). A study conducted in Kampongcham, Cambodia with 251 faecal specimens from primary school children and examined by formalin-ether sedimentation technique revealed that 3.2% had *G. lamblia* (Kyu-Jae *et al*., 2002). A study conducted in Kaski District, Western Nepal with 2091 stool samples collected from school children selected from eleven (11) rural and eight(8) urban schools revealed a prevalence rate of intestinal parasite to be 21.3%, and that of *G. lamblia* 13.2% (Chandrashekhar *et al*., 2005). A study conducted in northern Ghana by Klaus *et al*. (2007), revealed that *Giardia lamblia* were prevalent in asymptomatic individuals (9.7%) than in symptomatic individuals (3.7%).

2.6.2. Morphology of *Giardia lamblia*

The cysts are non-motile and egg-shaped. They measure 8–14 μm by 7–10 μm (John *et al.,* 2006). The cysts are encased in a smooth and colourless, thick and refractile wall (John and Petri, 2006). However, each organelle duplicates so that in permanently stained mature cysts,

four prominent nuclei and four median bodies are observed (John and Petri, 2006). Compared to trophozoites, cysts also have twice the number of intracytoplasmic flagella structures (John and Petri, 2006). The cysts are the infective form of the parasite and each cyst gives rise to two trophozoites (John and Petri, 2006).

Trophozoites are motile and non-infectious because they cannot survive long outside the host body. The trophozoites are pear shaped with a broad anterior end and a narrow posterior end (John *et al.*, 2006). It is 9–21 μm long and 5–15 μm wide (John and Petri, 2006). The trophozoite is bilaterally symmetrical and dorsoventrally flattened (John and Petri, 2006). A large sucking disk, which allows the parasite to attach to the surface of the intestinal mucosa of the host, takes up most of the ventral surface of the parasite (John and Petri, 2006). Four pairs of flagella are located anterior, lateral, ventral, and posterior on the body of the organism (John and Petri, 2006). The pair of anterior flagella, known as axome, is straight, closely approximated and parallel to each other, dividing the body of the organism into two halves longitudinally (John and Petri, 2006). Motility brought by the four pairs of flagella is essential for virulence of the parasite. The two spherical or ovoid nuclei, containing a large, central karyosome, can be found on each side of the axonemes (John and Petri, 2006). The parasite does not have peripheral chromatin (John and Petri, 2006).

2.6.3. Life cycle of *Giardia lamblia*

The life cycle of *Giardia lamblia* (also known as *Giardia intestinalis*) comprises of two stages: cyst and trophozoites (Wolfe, 1992). The cysts are approximately 7-10 μm in length and are oval in shape. The mature cyst contains four nuclei. They are environmentally resistant and responsible for disease transmission (Wolfe, 1992). Cysts may remain viable for several months in cool, moist conditions, and have been detected in natural surface waters

(Farthing, 1996). They are also able to survive standard concentrations of chlorine used in water purification systems (Jones *et al.*, 1988). Infection occurs after cysts are ingested. This marks the beginning of the life cycle. After ingestion, mature cysts in the small intestine release trophozoites through a process called excystation (Jones *et al.*, 1988). Cysts are able to survive exposure to gastric acid; gastric acid may actually trigger encystations (Farthing, 1996).

The trophozoites stage is responsible for producing clinical disease in humans. Trophozoites have two distinct nuclei and four pairs of flagellae. They resemble teardrops when viewed from the top and they are spoon shaped from the side. They are 12-15 µm in length. Trophozoites colonize the small intestine, attaching to the mucosa of the bowel using ventral sucking disks. The trophozoites then multiply by longitudinal binary fission (Hill, 1993). As the *Giardia* trophozoites move toward the colon, they retreat into the cyst stage (known as encystations) and the new cysts are excreted in the faeces. Bile salts and intestinal mucus boost trophozoites multiplication and encystations (Adrabbo and Peura, 2002).

2.6.4. Pathophysiology of *Giardia lamblia*

The pathogenesis of *Giardia* is not completely understood due to the extensive variation seen in disease expression. Clinical presentation ranges from asymptomatic cyst passage to chronic diarrhoea, malabsorption, severe weight loss, and malnutrition (Adrobbo *et al.*, 2002). In asymptomatic patients, histological examination of the duodenum often shows minimal changes or no abnormal representations (Farthing, 1996). However, the major structural and functional changes associated with giardiasis when symptoms are present are usually found in the small intestine (Farthing, 1996). Factors that influence the clinical presentation of the disease range from the host's immune response to the parasite, the parasite

load in the small intestine, and the virulence of the infecting strain of Giardia (Farthing, 1996).

There are several proposed mechanisms of disease that involve both mucosal and luminal factors in patients who exhibit abdominal pain, diarrhoea, and malabsorption symptoms (Farthing, 1996). Actual invasion of the mucosa by organisms is a rare finding. The intestinal mucosa may be damaged by the trophozoites itself disrupting the epithelial brush border during attachment, or less likely by direct invasion (Farthing, 1996). In addition, release of toxic substances from the organism itself may damage intestinal epithelium (Wolfe, 1992). Absorptive activities may also be blocked due to the trophozoites "blanketing" the intestinal mucosa and causing functional mucosal obstruction (Wolfe, 1992). Some studies have shown that immunologic mechanisms may also play a role since individuals with decreased gamma globulin levels have a higher prevalence of infection and reinfection (Wolfe, 1992).

The luminal factors that could possibly explain the pathogenesis of symptoms are increased number of anaerobic and aerobic bacteria in the small intestine of the infected patient (Tandon *et al.*, 1977). Malabsorption may be due, in part, to bacterial overgrowth which leads to the deconjugation of bile salts. The bile salts are then taken up by the trophozoites, triggering encystations and stimulating parasite growth (Tandon *et al.*, 1977)

Studies have indicated that inflammatory mast cells may interfere with duodenal growth of *G. lamblia* trophozoites. Other inflammatory cells, as well as CD8+ T cells, contribute to villus-shortening and crypt hyperplasia (Meyer, 1990). Inflammation results in an increased

turnover rate of intestinal mucosal epithelium. The immature replacement cells have less functional surface area and less digestive and absorptive ability (Meyer, 1990).

2.6.5. Clinical Manifestations of *Giardia lamblia*

Giardia infection was initially regarded as non-pathogenic and often found in asymptomatic patients. However, there is now much evidence for the pathogenic nature of *Giardia lamblia* (Wolfe, 1992). The major symptom of acute giardiasis, mainly seen in travellers, is protracted diarrhoea. The incubation period for infection is generally 9-15 days. The acute stage usually begins with a feeling of intestinal uneasiness followed by nausea and anorexia (Wolfe, 1992). Low grade fever and chills may occur. These symptoms are followed by watery, foul-smelling, explosive diarrhoea, abdominal pain, passage of foul gas and belching (Levinson, 2008). Malabsorption due to chronic *Giardia* infection has also been reported. Other common symptoms of giardiasis include abdominal pain, flatulence, bloating, vomiting and weight loss (Flanagan, 1992). Symptoms vary from person to person, often depending on the inoculum size, duration of infection, and individual host and parasite factors (Wolfe, 1992). The diarrhoea can be mild and produce semi-solid stools, or it can be intense and debilitating (Homan *et al*., 2001). Children generally become less ill than adults and frequently develop asymptomatic infection (Flanagan, 1992). It has been suggested that as many as 50% of infections are asymptomatic.

2.6.6. Laboratory Diagnosis

Diagnosis is made by finding trophozoites or cysts or both in diarrhoea stool. In formed stools only cysts are seen (Levinson, 2008). An Enzyme Linked Immunosorbent Assay (ELISA) test that detects a *Giardia* cyst wall antigen in the stool is also very useful (Levinson, 2008). However, in clinical practice, typically only a single stool exam is

performed (Adrobba and Peura, 2002). Stool microscopy is relatively inexpensive, but it does require a skilled technician and may be a time consuming process (Adrobba and Peura, 2002).

The introduction of *Giardia* stool antigen tests has improved giardiasis diagnostic capabilities (Wolfe, 1992; Adrobba *et al.*, 2002). Immunodiagnostic assays are available for the detection of *Giardia* and are both more sensitive and more specific (>90%) than the traditional ova and parasite (O+P) examinations (Wolfe, 1992; Adrobba *et al.*, 2002). A single antigen test is able to detect 50% more infections than O+P examinations. Antigen tests also require less time than an O+P exam and can be combined with antigen testing for diagnosing *Cryptosporidium*, making the antigen test more cost-effective (Wolfe, 1992; Adrobba and Peura, 2002). *Giardia* specific antigen may be detected in stool specimens even during an absence of cyst passage or visible signs of trophozoites (Adrobba and Peura, 2002; Wolfe, 1992). *Giardia* specific antigen in stool specimens are detected by several different methods: EIA, indirect and direct immunofluorescent assays using monoclonal antibodies and direct fluorescent assays (Wolfe, 1992; Adrobba and Peura, 2002).

2.6.7. Treatment of *Giardia lamblia*

Metronidazole and Tinidazole are drugs of choice for Giardiasis. According to the 2007 Medical Letter, Metronidazole is administered in doses of 250 mg 3 times a day for 5-7 days for adults and 15 mg/kg 3 times a day for 5-7 days in children (Ortiz *et al.*, 2001). Side effects associated with Metronidazole include metallic taste, dark urine, and gastrointestinal symptoms. As previously mentioned, Metronidazole is not approved by the FDA for the treatment of giardiasis (Ortiz *et al.*, 2001). Tinidazole is indicated for the treatment of giardiasis in adults with a single 2 g dose and with 50 mg/kg single dose in children over 3

years of age. In eight randomized comparative studies totalling 299 adult and paediatric patients, the average cure rate for Tinidazole in giardiasis was 90% (Ortiz *et al.,* 2001).

2.6.8. Prevention and Control of *Giardia lamblia Infestation*

Practicing of good hygiene in day care centers, retirement homes, and at homes would help to prevent the spread of infection (Yoder and Beach, 2007). Avoid contact with the faeces of an infected person. When travelling in areas where giardiasis is common, infection can be prevented by using only bottled water and consumption of raw fruits and vegetables should be avoided (Yoder and Beach, 2007). Untreated water in areas where the parasite might be present, such as lakes, rivers and streams should be avoided (Yoder and Beach, 2007). Water should be boiled for at least one minute before drinking. Public swimming pools that are not properly treated and maintained are another potential source of contamination (Yoder *et al.,* 2007). Swimmers are advised against swallowing water in swimming pools (Yoder and Beach, 2007).

2.7. THE COCCIDIA

Parasitic Coccidia are small protozoa which belong to the Phylum apicomplexa within the sub class sporozoa (Washington *et al.*, 2006). The important intestinal coccidia which infect man include *Cryptosporidium parvum, Isospora belli* and *Toxoplasma*. They are obligate tissue parasites with sexual and asexual stages in their life cycle (Washington *et al.*, 2006).

2.8. CRYPTOSPORIDIUM PARVUM

2.8.1. Epidemiology of *Cryptosporidium parvum*

Cryptosporidium parvum is an obligate intracellular parasite that infects the epithelial lining of luminal surfaces of gastrointestinal and respiratory tract in a wide variety of hosts (Armson

et al., 2003). The generally quoted prevalence of cryptosporidia in stool specimens is 1-3%. *C. parvum* is highly transmissible in the family setting, is one of many causes of travellers' diarrhoea, and is associated with diarrhoea outbreaks due to contaminated water supplies. In childcare centres the prevalence rate varies between 33% and 73% (Chacon-cruz, 2009).

Recent studies have provided more information regarding *C. parvum* transmission (Chacon-cruz, 2009). *Cryptosporidium* is transmitted through multiple routes. The infection may be transmitted by direct person-to-person contact and contact with infected animals or by ingestion of contaminated food or water (Khan *et al*., 2004). The oocysts are highly resistant to common household disinfectants and survive for long periods in the environment. *C. parvum* has major public health implication because infection can result from exposure to low doses of *Cryptosporidium* oocysts (Gatei *et al*., 2006). The major reservoir for *C. parvum* is domestic livestock, and direct contact with infected cattle is a major transmission pathway along with indirect transmission through drinking water (Chacon-cruz, 2009).

Cryptosporidium parvum is found in the stool of 10-20% of patients with HIV-AIDS associated diarrhoea (Chacon-cruz, 2009). The frequency with which *Cryptosporidium parvum* is identified in the stool of patients with AIDS is related to the CD4 count. Identification is more frequent when the CD4 count is less than 100 cells/ml and the presence of Gastrointestinal (GI) symptoms (Chacon-cruz, 2009). However higher rates of 27.8% and 15.6% from 227 children with diarrhoea and 77 children without diarrhoea respectively, aged less than 5 years have been reported in Ghana (Adjei *et al.*, 2004). In another study in Kumasi Ghana, analysis of 474 acute phase stool samples collected from children 2 months - 5 years of age found 61 (12.9%) rate of infection (Addy and Aikins-Bekoe, 1986).

2.8.2. Morphology of *Cryptosporidium parvum*

The oocysts are ovoid or spherical and measure 5 to 6 µm across. The Oocysts contains up to 4 sporozoites that are bow-shaped (Washington *et al.*, 2006). When stained with modified Ziehl Neelsen stain, the oocysts appear as small round or oval pink-stained bodies (Washington *et al.*, 2006).

2.8.3. Life cycle of *Cryptosporidium parvum*

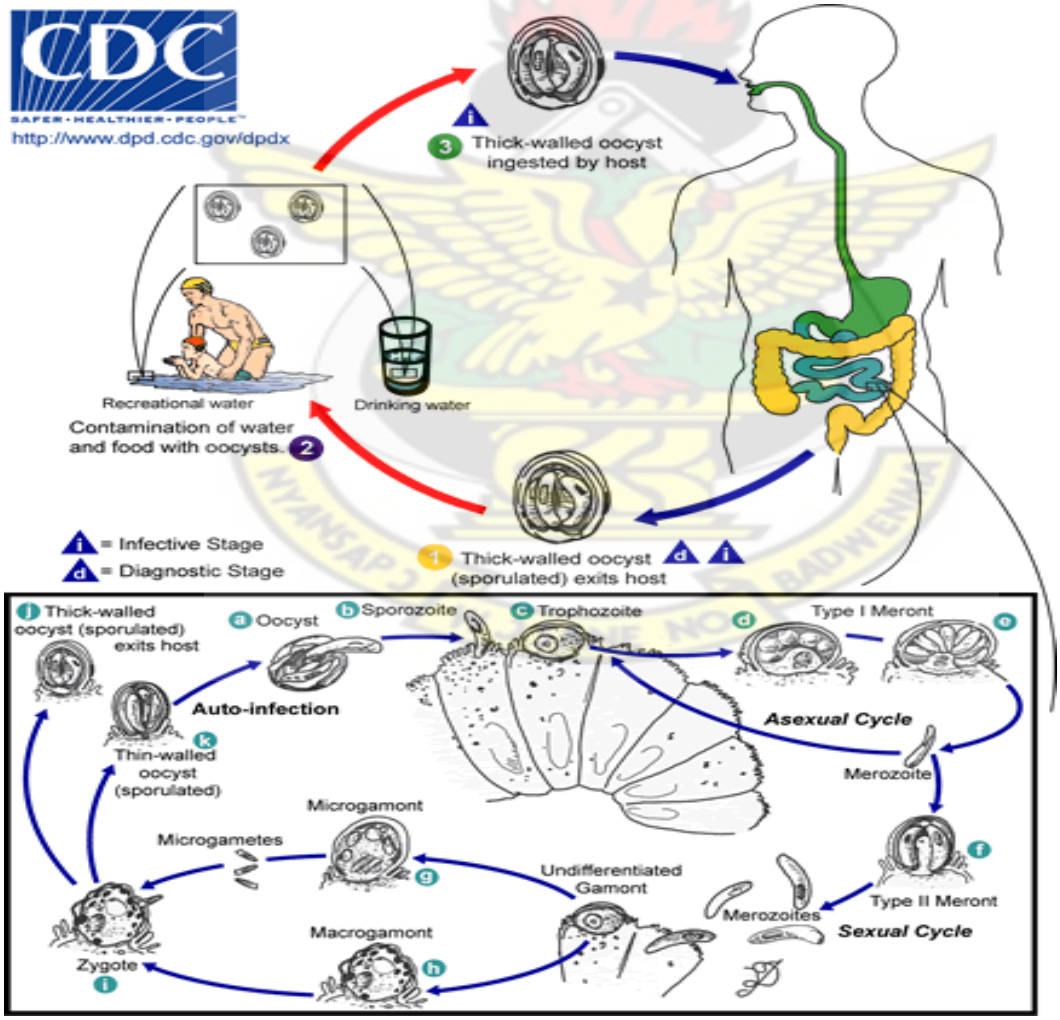


Figure 2.2: Lifecycle of *Cryptosporidium parvum*
Source: http://www.dpd.cdc.gov/dpdx/html/Cryptosporidiosis.htm

From Fig 2.2, sporulated oocysts, containing 4 sporozoites, are excreted by the infected host through faeces and possibly other routes such as respiratory secretions (CDC, 2010). Transmission of *Cryptosporidium parvum* and *C. hominis* occurs mainly through contact with contaminated water (e.g., drinking or recreational water). Occasionally food sources, such as chicken salad, may serve as vehicles for transmission (Fig 2.2) (CDC, 2010).

According to Fig 2.2, following ingestion (and possibly inhalation) by a suitable host (3), excystation (a) occurs. The sporozoites are released and parasitize epithelial cells (b, c) of the gastrointestinal tract or other tissues such as the respiratory tract (CDC, 2010). In these cells, the parasites undergo asexual multiplication (schizogony or merogony) (d, e, f) and then sexual multiplication (gametogony) producing microgamete (male) (g) and macrogamete (female) (h) (CDC, 2010). Upon fertilization of the macrogamete by the microgamete (i), oocysts (j, k) develop that sporulate in the lumen (Fig 2.2). Two different types of Oocysts are produced, the thick-walled, which is commonly excreted from the host (j), and the thin-walled oocysts (k) which is primarily involved in autoinfection. Oocysts are infective upon excretion, thus permitting direct and immediate fecal-oral transmission (CDC, 2010).

2.8.4. Pathogenesis of *Cryptosporidium parvum*

The oocysts are ovoid or spherical and measure 5 to 6 micrometers across. The oocysts contain up to 4 sporozoites that are bow-shaped (Washington *et al*., 2006). As few as 2 to 10 oocysts can initiate an infection (Chen *et al.,* 2003). The parasite is located in the brush border of the epithelial cells of the small intestine (Brooks *et al.,* 2004). They are mainly located in the jejunum (Wichro *et al.,* 2005). When the sporozoites attach the epithelial cells'

membrane envelops them. After invasion of the enterocytes, the epithelial cells release cytokines (Wichro *et al.*, 2005). These cytokines activate phagocytes and recruit new leukocytes, which, in turn, release soluble factors (resulting in intestinal secretion of chloride ions and water) and inhibit absorption (Wichro *et al.*, 2005). The parasite can cause damage to the microvilli where it attaches (Washington *et al*., 2006). The infected human excretes the most oocysts during the first week (Ryan and Ray, 2004). Oocysts can be excreted for weeks after the diarrhoea subsides (Centers for Disease Control and Prevention, 2009).

2.8.5. Clinical Manifestation of *Cryptosporidium parvum*

The intestinal tract is the primary site of cryptosporidiosis. Although infection can be asymptomatic, most patients have profuse watery diarrhoea containing mucus but rarely blood or leukocytes (Farthing *et al.*, 2000). The duration and severity of clinical symptoms depend largely on the immune status of the infected person. In immunocompetent persons, the disease is usually either asymptomatic or self-limiting (Farthing *et al.*, 2000). The three major clinical presentations in immunocompetent persons are asymptomatic carriage, acute diarrhoea, and persistent diarrhoea that may continue for several weeks (Farthing *et al.*, 2000). After an incubation period of 7 to 10 days, more than 90 percent of infected patients present with acute watery diarrhoea that lasts approximately 2 weeks, accompanied by nausea, vomiting, and cramp-like abdominal pain; 36 percent also have fever (Farthing *et al*, 2000). Acute and chronic diarrhoea due to *C. parvum* in children in the developing world is associated with malnutrition and high morbidity and mortality rates (Farthing *et al.*, 2000). The diarrhoea also has lasting adverse effects on weight and height (Checkley *et al.*, 1998; Guerrant *et al.*, 1999). The severity and duration of diarrhoea and the extraenteric manifestations of the infection differ in immunocompetent and immunocompromised patients (Farthing *et al.*, 2000).

Immunocompromised people, as well as very young or very old people, can develop a more severe form of cryptosporidiosis (Brooks *et al.*, 2004). There are 4 clinical presentations for patients with HIV-AIDS; 4% have no symptoms, 29% have a transient infection, 60% have chronic diarrhoea, and 8% have a severe, cholera-like infection (Chen *et al.*, 2002). With transient infections diarrhoea ends within 2 months and *Cryptosporidium* is no longer found in the faeces. Chronic diarrhoea is one that lasts for 2 or more months (Chen *et al.*, 2002). The most severe form results in the patients excreting at least 2 litres of watery diarrhoea per day (Chen *et al.*, 2002). They can lose up to 25 litres per day (Ryan *et al.*, 2004). AIDS patients may pass up to 10 stools per day. They experience severe malabsorption and can suffer 10% weight loss. Many of them never completely eliminate *Cryptosporidium* from their bodies (Chen *et al.*, 2002).

When *Cryptosporidium* spreads beyond the intestine, as it can, predominantly in patients with AIDS, it can reach the lungs, middle ear, pancreas, and stomach (Chen *et al.*, 2002). Thus, one symptom is pain in the right upper quadrant (Chen *et al.*, 2002). The parasite can infect the biliary tract, causing biliary cryptosporidiosis. This can result in cholecystitis and cholangitis (Ryan and Ray, 2004).

2.8.6. Laboratory Diagnosis of *Cryptosporidium parvum*

There are many diagnostic tests for *Cryptosporidium*. These include microscopy, staining, and detection of antibodies. Microscopy (Centers for Disease Control and Prevention, 2009) can help identify oocysts in faecal matter (Brooks *et al.*, 2004). To increase the chance of finding the oocysts, the diagnostician should inspect at least 3 stool samples (Murray *et al.*, 2005). There are several techniques to concentrate either the stool sample or the oocysts.

The modified formalin-ethyl acetate (FEA) concentration method concentrates the stool (Winn *et al,* 2006). Both the modified Zinc sulphate centrifugal flotation technique and the Sheather's sugar flotation procedure can concentrate the oocysts by causing them to float (Murray *et al.,* 2005).

Another form of microscopy is fluorescent microscopy done by staining with auramine (Brooks *et al.,* 2004). Other staining techniques include acid-fast staining (Chen *et al.,* 2003) which will stain the oocysts red (Washington *et al,* 2006). One type of acid-fast stain is the Kinyoun technique (Gideon, 2009). Giemsa staining can also be performed (Ryan *et al.,* 2004). Part of the small intestine can be stained with haematoxylin and eosin (H & E), which will show oocysts attached to the epithelial cells (Washington *et al,* 2006).

Detecting antigens is yet another way to diagnose the disease. This can be done with direct fluorescent antibody (DFA) techniques (Centers for Disease Control and Prevention, 2009). It can also be achieved through indirect immunofluorescence assay (Murray *et al.,* 2005). Enzyme-Linked Immunosorbent Assay (ELISA) also detects antigens (Brooks *et al.,* 2004). Polymerase chain reaction (PCR) is another way to diagnose cryptosporidiosis. It can even identify the specific species of *Cryptosporidium* (Centers for Disease Control and Prevention, 2009).

If the patient is thought to have biliary cryptosporidiosis, then an appropriate diagnostic technique is ultrasonography. If that returns normal results, the next step would be to perform endoscopic retrograde cholangiopancreatography (Chen *et al.,* 2002).

2.8.7. Treatment of *Cryptosporidium parvum*

The treatment of cryptosporidiosis is unsatisfactory. There is no antimicrobial chemotherapeutic agent that will reliably eradicate the organism. However, there are agents that appear to suppress infection (Farthing, 2000; Clark, 1999). When highly active antiretroviral therapy reduces the HIV load, symptoms may resolve in patients with cryptosporidium infection (Farthing, 2000; Clark, 1999). Paromomycin, azithro-mycin, and (most recently) nitazoxanide are commonly used for the treatment of cryptosporidium infection (White *et al.*, 1994; Rossignol *et al.*, 2001).

2.8.8. Prevention and Control of *Cryptosporidium parvum*

Cryptosporidium infection can be controlled by practising good hygiene such as washing of hands with soap and water after using the toilet, changing diapers, and before and after eating. Care should be taken when handling animal or human excreta (CDC, 2000). All vegetables and fruits should be thoroughly washed before eating and avoid drinking of untreated water from lakes, rivers, springs, ponds, or streams (CDC, 2000). The oocysts of the parasite can be eliminated from the drinking water by boiling for one minute or by filter sterilization with absolute 1.0 micron filters, reverse osmosis filters, or filters that meet NSF (National Sanitation Foundation) standards (CDC, 2000). Infected food handlers should be removed from work areas, and infected children should be excluded from day-care and recreational water facilities until the infection clears (CDC, 2000).

2.9. INTESTINAL HELMINTH INFECTION

The four most common soil-transmitted helminths (STHs) are roundworm (*Ascaris lumbricoides*), whipworm (*Trichuris trichiura*), and the anthropophilic hookworms (*Necator americanus* and *Ancylostoma duodenale*) (de Silva *et al.,* 2003). Recent estimates suggest

that *A. lumbricoides* infects 1.221 billion people, *T. trichiura* 795 million, and hookworms 740 million (de Silva *et al.,* 2003). The greatest numbers of STH infections occur in the Americas, China and East Asia, and Sub-Saharan Africa (de Silva *et al.,* 2003). Over one billion of the world's population is estimated to be infected with these parasites; two billion or more are said to be at risk (Montessor *et al.,* 1998). Children are reported to be at especially increased risk for severe infections and the mortality and morbidity associated with these infections (Chan *et al.,* 1994). Helminth infection has been linked with an increased risk for several nutritional anaemias, protein-energy malnutrition and reduced physical growth and development in infants and children (Stephenson *et al.,* 2000; Nokes and Bundy, 1994; Connolly and Kvalsvig, 1993).

2.10. *ASCARIS LUMBRICOIDES* (ROUNDWORM INFECTION)

2.10.1. Epidemiology and Prevalence of *Ascaris lumbricoides*

Human Ascariasis is a human disease caused by the parasitic roundworm *Ascaris lumbricoides* (Berger *et al.,* 2006). *Ascaris lumbricoides* is the giant roundworm of humans, belonging to the phylum Nematoda. Perhaps as many as one quarter of the world's people are infected, with rates of 45% in Latin America and 95% in parts of Africa (Berger *et al.,* 2006). Ascariasis is particularly prevalent in tropical regions and in areas of poor hygiene. Annual morbidity associated with *Ascaris lumbricoides* has been estimated by the WHO at 60,000 with another 250 million people said to be at risk for acquiring the infection (Montressor *et al.,* 1998). Both domestic and wild animals are common reservoirs for *A. lumbricoides*.

However, some controversy exists regarding whether *A. suum,* the pig roundworm, is genetically identical to *A. lumbricoides* (Anderson and May, 1995; Peng *et al.,* 1998). *Ascaris lumbricoides* is a robust parasite. This quality is due, in part, to the resilient nature of

its eggs, which are capable of surviving a wide range of hot and cold temperatures, chemicals, chemical disinfectants and other extreme conditions (Neva and Brown, 1994).

2.10.2. Morphology of *Ascaris lumbricoides*

The eggs of *Ascaris* are one of the most resilient of the helminth eggs and can remain infective for years in the soil (Crompton, 1999; Gilgen and Mascie-Taylor, 2000). *Ascaris lumbricoides* is characterized by its great size. Males are 2–4 mm in diameter and 15–31 cm long (Roberts *et al.,* 2009). The male's posterior end is curved ventrally and has a bluntly pointed tail. Females are 3–6 mm wide and 20–49 cm long (Roberts and Janovy, 2009). The vulva is located in the anterior end and accounts for about a third of its body length (Roberts and Janovy, 2009). Uterus may contain up to 27 million eggs at a time with 200,000 being laid per day (Roberts Janovy, 2009). Fertilized eggs are oval to round in shape and are 45-75 micrometers long and 35-50 micrometers wide with a thick outer shell. Unfertilized eggs measure 88-94 micrometers long and 44 micrometers wide (Roberts and Janovy, 2009).

2.10.3. Multiplication and Life cycle of *Ascaris lumbricoides*

Ascaris lumbricoides is found in the small intestine, particularly the jejunum (Guyatt *et al.,* 1995). Females produce as many as 240,000 eggs per day and as many as 65 million in a lifetime. The eggs are unsegmented and are passed in the feces. In moist, warm, shady soil, the eggs embryonate, and an infective larva develops within the egg in about 3 weeks (Guyatt *et al.,* 1995). After ingestion by a human, the eggs pass to the duodenum where they hatch; the released larvae penetrate the intestinal mucosa, enter the lymphatics and portal system, and are carried to the liver, heart, and lungs (Guyatt *et al.,* 1995). This migratory phase requires a few days (Guyatt *et al.,* 1995). The larvae then break out of the capillaries into the alveoli, pass up the respiratory tree, and are swallowed. They reach the intestines and

continue their development, and 8 to 12 weeks after infection, become sexually mature adults (Guyatt *et al.,* 1995). The adults live for about a year and are subsequently passed in the faeces (Guyatt *et al.,* 1995).

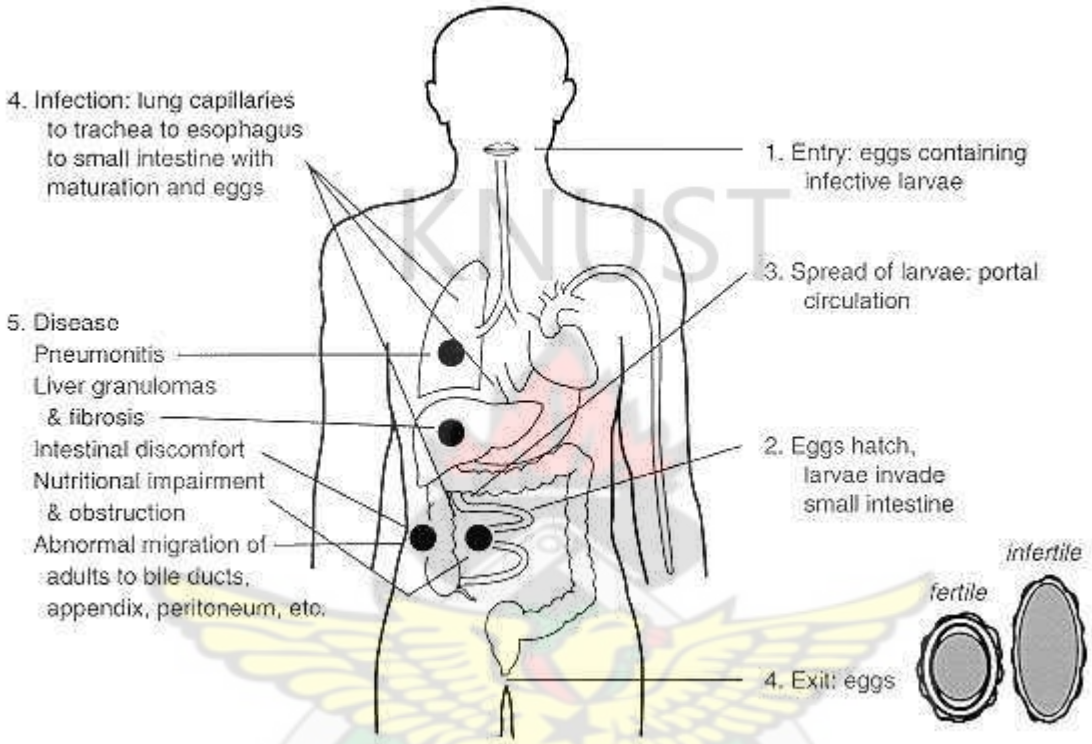


Figure 2.3 Lifecycle of *Ascaris lumbricoides*
Source: http://www.ncbi.nlm.nih.gov/books/NBK8374/#top

2.10.4. Pathogenesis and Clinical Manifestation of *Ascaris lumbricoides*

The initial pathology is associated with migrating larvae; the severity depends upon the number of invading organisms, the sensitivity of the host, and the host's nutritional status. Persons repeatedly infected become sensitized, and migrating larvae may cause tissue reactions in the liver and lungs, with eosinophilic infiltration and granuloma formation (Guyatt *et al.,* 1995). The reactions lead to pneumonitis and a condition known as Loeffler's syndrome (Guyatt *et al.,* 1995). Adult worms may cause blockage of the intestines, and

migrating adults may provoke severe pathology when they wander into other organs. Acute pancreatitis and biliary stones may occur. The rare fatalities usually result from intestinal obstruction or biliary ascariasis (Guyatt *et al.*, 1995).

2.10.5. Laboratory Diagnosis of *Ascaris lumbricoides*

Diagnosis is usually made microscopically by detecting *Ascaris lumbricoides* eggs in the stool. The egg is oval with irregular surface. Occasionally, the patient sees adult worms in the stool (Jawetz and Levinson, 1996). Abdominal ultrasonography is the study of choice for imaging *Ascaris* worms in the biliary tree (Sherman *et al.*, 2005). Worms may be single, multiple, in bundles, and moving during the examination. Abdominal CT scanning is an alternative study to abdominal ultrasonography (Sherman *et al.*, 2005). A thin line of dye is occasionally seen within the corpus of a worm that has swallowed dye. Within the biliary tree, the worm is more easily visualized on an unenhanced scan (Sherman *et al.*, 2005). Double-contrast abdominal CT scanning is an excellent study for the evaluation of a patient who presents with acute abdominal symptoms suggestive of intestinal obstruction or another surgical emergency (Sherman *et al.*, 2005). 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). It is good for a general evaluation of the pancreatobiliary organs. It is an alternative to abdominal CT scanning when radiation exposure is to be avoided (Arya *et al.*, 2005).

2.10.6. Treatment of *Ascaris lumbricoides*

Benzimidazoles are effective for the treatment of intestinal ascariasis, although some authors do not recommend their use in the first year of life and during pregnancy due to their teratogenic effects in animal studies (Xiao *et al.*, 2005; Steinmann *et al.*, 2008). The most commonly recommended agents are albendazole and mebendazole. Ivermectin and pyrantel

pamoate are alternatives, the latter having been suggested for pregnant patients in whom benzimidazoles are contraindicated (Xiao *et al.,* 2005; Steinmann *et al.,* 2008). An anthelmintic agent from China, tribendimidine (at a dose of 300 mg), has been shown to be as efficacious as Albendazole for adult (Xiao *et al.,* 2005; Steinmann *et al.,* 2008).

2.10.7. Prevention and Control of *Ascaris lumbricoides*

Ascaris lumbricoides can be prevented and controlled by providing adequate public and private toilets to avoid contamination of soil with infectious faeces (Cheesbrough, 2005). Proper disposal of human faeces, avoiding the use of untreated human faeces as fertilizer and washing of hands before eating helps to control *Ascaris* infection (Cheesbrough, 2005).

2.11. HOOKWORM

2.11.1. Epidemiology and Prevalence

Human hookworm infection is caused by blood-feeding nematode parasites of the Genera *Ancylostoma* and *Necator* (Hotez *et al.,* 2004). Worldwide, *N. americanus* is the predominant aetiology of human hookworm infection, whereas *A. duodenale* occurs in more scattered focal environments (Hotez *et al.,* 2004). Most infected individuals are concentrated in sub-Saharan Africa and East Asia/the Pacific Islands with each region having estimates of 198 million and 149 million infected individuals, respectively (Bethony *et al.,* 2006). Other affected regions include: South Asia (50 million persons), Latin America and the Caribbean (50 million persons), South Asia (59 million persons), Middle East/North Africa (10 million persons) (Bethony *et al.,* 2006). A majority of these infected individuals live in poverty-stricken areas with poor sanitation. Hookworm infection is most concentrated among the worlds poorest who live on less than $2 a day (Hotez *et al.,* 2005). A study conducted in Kintampo North District of the Brong Ahafo Region, revealed that hookworm is one of the

major soil-transmitted helminthes in Ghana but no definitive prevalence assessment has been done (Ghana Health Service, 2007-2008). *A. duodenale* worms are greyish white or pinkish with the head slightly bent in relation to the rest of the body (Markell *et al.,* 2006). This bend forms a definitive hook shape at the anterior end for which hookworms are named (Markell *et al.,* 2006). They possess well developed mouths with two pairs of teeth. While males measure approximately one centimetre by 0.5 millimeter, the females are often longer and stouter (Markell *et al*., 2006). Additionally, males can be distinguished from females based on the presence of a prominent posterior copulatory bursa (Markell *et al.,* 2006).

2.11.2. Morphology of Hookworm

N. americanus is very similar in morphology to *A. duodenale*. *N. americanus* is generally smaller than *A. duodenale* with males usually 5 to 9 mm long and females about 1 cm long (Markell *et al.,* 2006). Whereas *A. duodenale* possesses two pairs of teeth, *N. americanus* possesses a pair of cutting plates in the buccal capsule. Additionally, the hook shape is much more defined in *Necator* than in *Ancylostoma* (Markell *et al.,* 2006). The eggs are oval or elliptical, measuring 60 µm by 40 µm, colourless, not bile stained and with a thin transparent hyaline shell membrane. When released by the worm in the intestine, the egg contains an unsegmented ovum (Markell *et al.*, 2006). During its passage down the intestine, the ovum develops and thus the eggs passed in feces have a segmented ovum, usually with 4 to 8 blastomeres (Markell *et al.*, 2006).

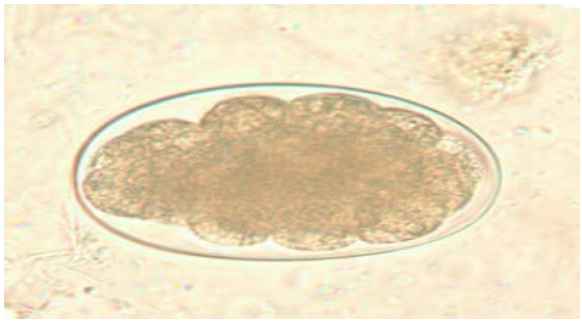


Figure 2.4: Morphology of Hookworm egg

Source: http://www.dpd.cdc.gov/dpdx/HTML/ImageLibrary/G-L/Hookworm/body_Hookworm_il1.htm

2.11.3. Multiplication and Life cycle of Hookworm

N. americanus and *A. duodenale* eggs can be found in warm, moist soil where they will eventually hatch into first stage larvae, or L1. L1, the feeding non-infective rhabditiform stage, will feed on soil microbes and eventually molt into second stage larvae, L2. L2, which is also in the rhabditiform stage, will feed for approximately 7 days and then molt into the third stage larvae, or L3 (Hawdon and Hotex, 1996; Hotez *et al.*, 2005; Bethony *et al.*, 2006). L3 is the filariform stage of the parasite, that is, the non-feeding infective form of the larvae. The L3 larvae are extremely motile and will seek higher ground to increase their chances of penetrating the skin of a human host (Hawdon and Hotex, 1996; Hotez *et al.*, 2005; Bethony *et al.*, 2006). The L3 larvae can survive up to 2 weeks without finding a host. While *N. americanus* larvae only infect through penetration of skin, *A. duodenale* can infect both through penetration as well as orally (Hawdon and Hotex, 1996; Hotez *et al.*, 2005; Bethony *et al.*, 2006). After the L3 larvae have successfully entered the host, the larvae then travel through the subcutaneous venules and lymphatic vessels of the human host (Hawdon and Hotex, 1996; Hotez *et al.*, 2005; Bethony *et al.*, 2006). Eventually, the L3 larvae enter the lungs through the pulmonary capillaries and break out into the alveoli. They will then travel up the trachea to be coughed and swallowed by the host. After being swallowed, the L3

larvae are then found in the small intestine where they molt into the L4, or adult worm stage (Hawdon and Hotex, 1996; Hotez *et al.*, 2005; Bethony *et al.*, 2006). The entire process from skin penetration to adult development takes about 5–9 weeks (Hawdon and Hotex, 1996; Hotez *et al.*, 2005; Bethony *et al.*, 2006). The female adult worms will release eggs (*N. Americanus* about 9,000-10,000 eggs/day and *A. duodenale* 25,000-30,000 eggs/day) which are passed in the feces of the human host (Hawdon and Hotex, 1996; Hotez *et al.*, 2005; Bethony *et al.*, 2006). These eggs will hatch in the environment within several days and the cycle with start anew (Hawdon and Hotex, 1996; Hotez *et al.*, 2005; Bethony *et al.*, 2006).

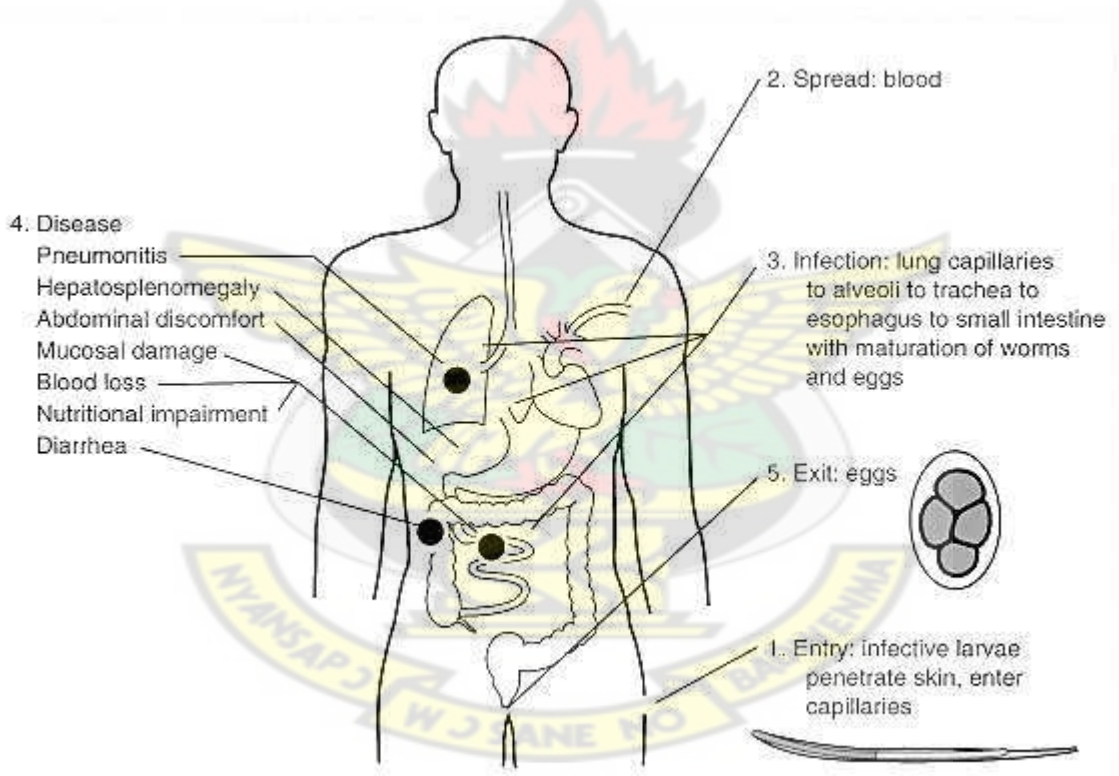


Figure 2.5: Lifecycle of *Hookworm*
Source: http://www.ncbi.nlm.nih.gov/books/NBK8027/

2.11.4. Pathogenesis and Clinical Manifestation of Hookworm

Hookworm infection is generally considered to be asymptomatic, but as Norman Stoll (1962) has affirmed, hookworm is an extremely dangerous infection because its damage is "silent and insidious". There are general symptoms that an individual may experience soon after infection. Ground-itch, which is an allergic reaction at the site of parasitic penetration and entry, is common in patients infected with *N. americanus* (Markell *et al.*, 2006). Additionally, cough and pneumonitis may result as the larvae begin to break into the alveoli and travel up the trachea (Markell *et al.*, 2006). Then once the larvae reach the small intestine of the host and begin to mature, the infected individual will suffer from diarrhoea and other gastrointestinal discomfort (Markell *et al.*, 2006). However, the "silent and insidious" symptoms referred to by Stoll are related to chronic, heavy-intensity hookworm infections. Major morbidity associated with hookworm is caused by intestinal blood loss, iron deficiency anemia, and protein malnutrition (Bethony *et al.*, 2006). They result mainly from adult hookworms in the small intestine ingesting blood, rupturing erythrocytes, and degrading haemoglobin in the host (Hotez *et al.*, 2005). This long-term blood loss can manifest itself physically through facial and peripheral oedema; eosinophilia and pica caused by iron deficiency anemia are also experienced by some hookworm-infected patients (Markell *et al.*, 2006). Recently, more attention has been given to other important outcomes of hookworm infection that play a large role in public health (Hotez *et al.*, 2005). It is now widely accepted that children who suffer from chronic hookworm infection can suffer from growth retardation as well as intellectual and cognitive impairments (Hotez *et al.*, 2005). Additionally, recent research has focused on the potential of adverse maternal-fetal outcomes when the mother is infected with hookworm during pregnancy (Hotez *et al.*, 2005).

2.11.5. Laboratory Diagnosis of Hookworm Infection

Diagnosis depends on finding characteristic worm eggs on microscopic examination of the stools, although this is not possible in early infection (Markell *et al.*, 2006). The eggs of both *Ancylostoma* and *Necator* (and most other hookworm species) are indistinguishable, to identify the genus, they must be cultured in the laboratory to allow larvae to hatch out (Markell *et al.*, 2006). If the faecal sample is left for a day or more under tropical conditions, the larvae would have hatched out, so eggs might no longer be evident (Markell *et al*, 2006). The larvae of the two hookworm species can also be distinguished microscopically, although this would not be done routinely, but usually for research purposes (Markell *et al.*, 2006). Adult worms are rarely seen (except via endoscopy, surgery or autopsy), but if found, would allow definitive identification of the species (Markell *et al.*, 2006). Classification can be performed based on the length of the buccal cavity, the space between the oral opening and the oesophagus: hookworm rhabditiform larvae have long buccal cavities whereas *Strongyloides* rhabditiform larvae have short buccal cavities (Markell *et al.*, 2006).

Recent research has focused on the development of DNA-based tools for diagnosis of infection, specific identification of hookworm and analysis of genetic variability within hookworm populations (Gasser *et al.*, 2009). Because hookworm eggs are often indistinguishable from other parasitic eggs, PCR assays could serve as a molecular approach for accurate diagnosis of hookworm in the feces (Yong *et al.*, 2007; Gasser *et al.*, 2009).

2.11.6. Treatment of Hookworm Infection

The most common treatment for Hookworm is Benzimidazoles (BZAs), specifically Albendazole and Mebendazole. BZAs kill adult worms by binding to the nematode's β-tubulin and subsequently inhibiting microtubule polymerization within the parasite (Bethony

et al., 2006). In certain circumstances, levamisole and Pyrantel pamoate may be used (Hotex *et al.*, 2005). The 2008 study by Keiser and Utzinger, Efficacy of Current Drugs Against Soil-Transmitted Helminth Infections: Systematic Review and Meta-analysis, examined the relative efficacies of different drug treatments. They found that the efficacy of single-dose treatments for Hookworm infections were as follows: 72% for Albendazole, 15% for mebendazole, and 31% for pyrantel pamoate (Keiser *et al.*, 2008). This substantiates prior claims that Albendazole is much more effective than mebendazole for Hookworm infections. Also of note is that the World Health Organization does recommend anthelmintic treatment in pregnant women after the first trimester (Bethony *et al.*, 2006). It is also recommended that if the patient also suffers from anaemia, ferrous (200 mg) be administered three times daily at the same time as anthelmintic treatment; this should be continued until hemoglobin values return to normal which could take up to 3 months (Markell *et al.*, 2006).

2.11.7. Prevention and Control of Hookworm Infection

Disposal of sewage properly and use of foot wear are effective means of prevention. Anthelminthic treatment of all infected individuals should be implemented to reduce to a minimum the sources of soil infestations (Jawetz *et al.*, 1996). Health education is also required (Cheesbrough, 2005). Evaluations of numerous public health interventions have generally shown that improvement in each individual component ordinarily attributed to poverty (for example, sanitation, health education, footwear and underlying nutrition status) often have minimal impact on transmission (Huttly, 1990). For example, one study found that the introduction of latrines into a resource-limited community only reduced the prevalence of hookworm by four percent (Huttly, 1990). Another study in Salvador, Brazil found that improved drainage and sewerage had minimal impact of the prevalence and no

impact at all on the intensity of hookworm (Moraes *et al.*, 2004). Disposing of sewage properly and wearing of shoes are effective means of prevention (Levinson, 2008).

2.12. TRICHURIS TRICHIURA INFECTION

2.12.1. Epidemiology of *Trichuris trichiura*

There is worldwide distribution of *Trichuris trichiura*, with about 500 million human infections (Hunter and Mckay, 2004). However, it is chiefly tropical especially in Asia and to a lesser extent in Africa and South America (Hunter and Mckay, 2004). Within the United States, infection is rare overall but may be common in the rural Southeast where 2.2 million people are thought to be infected (Hunter and Mckay, 2004). Studies carried out in Srinagar City, Kashmir, India with 514 school children, 4.9% had *Trichuris trichiura* parasite and attributed its frequency to water sources, defecation site, personal hygiene and the extent of maternal education (Showkat *et al*., 2007).

In a study of 352 school children from Gurez valley of Jammu and Kashmir State, India showed a prevalence rate of 26.42% of the infection with *Trichuris trichiura* (Showkat *et al*., 2010). *Trichuris trichiura* is more common in less-developed countries. This parasite is carried by nearly one quarter of the world's population (Donkor, 2009). Poor hygiene is associated with *trichuriasis* transmission, and children are especially vulnerable because of their high exposure risk (Donkor, 2009). Eggs are infective about 2–3 weeks after they are deposited in the soil under proper conditions of warmth and moisture, hence its tropical distribution (Hunter and Mckay, 2004). *Trichuris trichiura* is one of the soil-transmitted helminthes in Ghana but its assessment has not been done (Ghana Health Service, 2007-2008).

In a study conducted on non-school going children in Kintampo District in the Brong Ahafo Region (Ghana), 711 stool samples were examined. *Trichuris trichiura* infected children were six (6), with a frequency of 0.8% (Tay *et. al*, 2010).

2.12.2. Morphology of *Trichuris trichiura*

The worm is long, slender with a threadlike anterior portion and a thicker posterior portion; this gives an appearance of a whip handle (Baron, 1996). The female adult worm measures between 35 to 50 mm in length and has a bluntly rounded posterior end (Garcia, 2007; Heelan, 2004). The male adult worm measures 30 to 45 mm in length and has a 360^0 coil at the posterior end (Garcia, 2007; Heelan, 2004). The adult worms are rarely recovered from the stool since they attach to the wall of the host's intestine (Garcia, 2007; Heelan, 2004). The eggs are barrel-shaped with clear, mucoid appearing polar plugs. The eggs measure 50 to 54 um long and 22 to 23 um wide (Gracia, 2007).

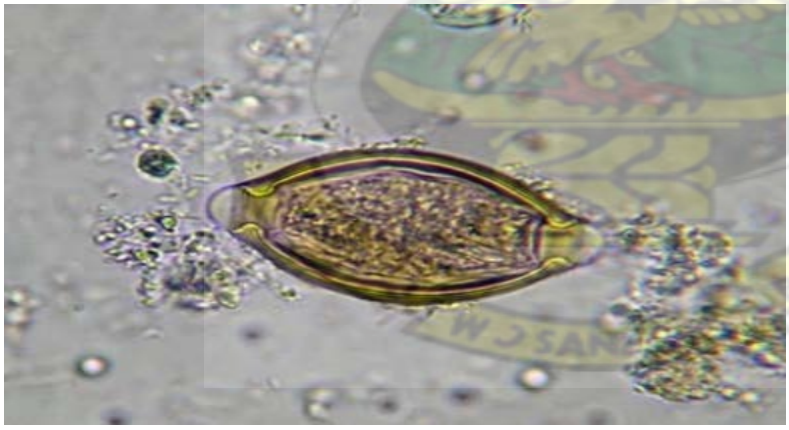


Figure 2.6: Morphorlogy of *Trichuris trichiura* egg

Source: http://www.wadsworth.org/parasitology/critiquesJun05.htm

2.12.3. Multiplication and Life cycle of *Trichuris trichiura.*

Females produce 2,000 to 10,000 single-celled eggs per day. These pass in the faeces and embryonate in the soil (Baron, 1996). Under favourable conditions, they become infective in about 3 weeks. After being ingested, embryonated infective eggs hatch in the small intestine (Baron, 1996). The infective larvae penetrate the villi and continue to develop. Young worms move to the cecum, penetrate the mucosa, and complete development. Females copulate to lay eggs about 3 months after infecting the host (Baron, 1996).

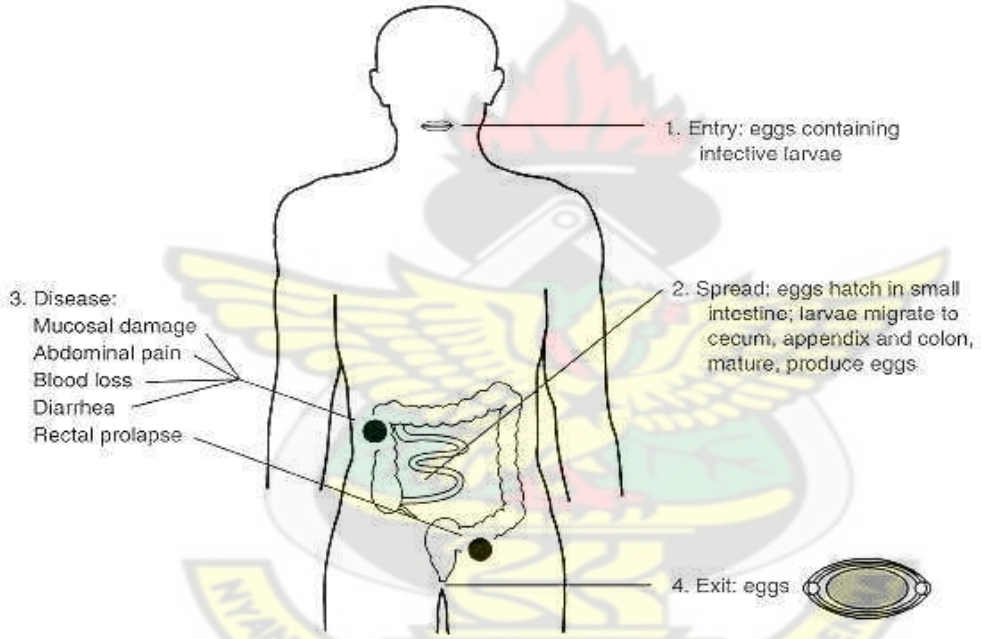


Figure 2.7: Lifecycle of *Trichuris trichiura*
Source: http://www.ncbi.nlm.nih.gov/books/NBK8089/

2.12.4. Pathogenesis and Clinical Manifestations of *Trichuris trichiura*

Trichuris, as with *Ascaris lumbricoides*, is spread via fecal-oral transmission. Eggs are deposited in soil through human feces. After 10-14 days in soil, eggs become infective (Ekpo *et al*., 2008). The caecum and the colon are the most commonly infected sites, although in heavily infected individuals, infection can be present in more distal segments of

gastrointestinal tract (GIT), such as the descending and rectum (Donkor, 2009). The time of ingestion of eggs to the development of mature worms is approximately three (3) months (Donkor, 2009). During this time, there may be no shedding of eggs and only limited evidence of infection in stool sample (Donkor, 2009).

Immunologically, cytokines such as Interleukin-25 (IL-25) mediate immunity and are required for the regulation of inflammation in the gastrointestinal tract (Donkor, 2009). Recent linkage analysis of a genome-wide scan revealed that two (2) quantitative traits loci on chromosome nine (9) and eighteen (18) may be responsible for the susceptibility to infection with *Trichuris trichiura* in some genetically predisposed individuals (Donkor, 2009).

Petechial hemorrhage, edema, inflammation, and mucosal bleeding develop, and heavy infections can cause rectal prolapse. Small amounts of blood (0.005 ml per worm) are lost each day by seepage at the attachment site (Baron, 1996). Colitis/proctitis, anemia, clubbing of fingers, and growth retardation are also reported to be associated with heavy *T trichiura* infections (Baron, 1996). The parasite *T. trichiura* lives primarily in the caecum and appendix but can also be found in large numbers in the colon and rectum (Baron, 1996). Light infections are asymptomatic but heavy infections may cause diarrhoea, at times containing mucus and blood (Baron, 1996). Anemia may develop along with weight loss, abdominal pain, nausea, vomiting, tenesmus and rectal prolapse. Nutritional changes can cause stunted growth and clubbing of fingers (Baron, 1996). Eosinophilia may also develop in response to worms embedded in the mucosa (Baron, 1996).

2.12.5. Laboratory Diagnosis of *Trichuris trichiura*

Trichuriasis can be diagnosed when *Trichuris trichiura* eggs are detected in stool examination (Levinson, 2008). Eggs will appear barrel shaped, unembryonated, have bipolar plugs and a smooth shell. Rectal prolapsed can be diagnosed easily using defecating proctogram and is one of many methods for imaging parasitic infection (Hunter *et al*., 2004).

2.12.6. Treatment and Control of *Trichuris trichiura* Infection

Metronidazole is 90% effective in the first dose, and Albendazole may also be offered as an anti-parasitic agent. Adding iron to the bloodstream helps solve the iron deficiency and rectal prolapsed (Summers *et al*., 2005). Infection can be avoided by proper disposal of human faeces, avoiding fecal contamination of food, not eating dirt, and avoiding crops fertilized with human faecal manure. Simple and effective proper hygiene such as washing hands and food is recommended for control (Summers *et al*., 2005).

2.13. STRONGYLOIDES STERCORALIS

2.13.1. Epidemiology and Prevalence of *Strongyloides stercoralis*

Strongyloides stercoralis is a nematode that can parasitize humans. The adult parasitic stage lives in tunnels in the mucosa of the small intestine (Speare, 1989; Skerratt, 1995). The genus *Strongyloides* contains 53 species (Speare, 1989; Skerratt, 1995) and *S. stercoralis* has been reported in other mammals, including cats and dogs. However, it seems that the species in dogs is typically not *S. stercoralis*, but the related species *S. canis*. Non-human primates are more commonly infected with *S. fuelleborni* and *S. cebus* although *S. stercoralis* has been reported in captive primates (Speare, 1989; Skerratt, 1995).

S. stercoralis has a very low prevalence in societies where fecal contamination of soil or water is rare. Hence, it is a very rare infection in developed economies (Segarra-Newnham, 2007). In developing countries it is less prevalent in urban areas than in rural areas (where sanitation standards are poor). *S. stercoralis* can be found in areas with tropical and subtropical climates (Segarra-Newnham, 2007).

S. stercoralis is thought to infect 30-100 million people in 70 different countries. Strongyloidiasis is endemic in Africa, but the prevalence is typically low (1% or less) (Johnston *et al.*, 2005). Pockets of strongyloidiasis have been reported from rural Italy, but current status is unknown (Johnston *et al.*, 2005). In the Pacific islands strongyloidiasis is rare although there have been reports of cases from Fiji (Johnston *et al.*, 2005). In tropical Australia, some rural and remote Australian aboriginal communities have very high prevalence of strongyloidiasis (Johnston *et al.*, 2005). In some African countries (e.g., Zaire) *S. fuelleborni* was more common than *S. stercoralis* in parasite surveys from the 1970s, but current status is unknown. In Papua New Guinea (PNG), *S. stercoralis* is endemic, but prevalence is low (Dorris *et al.*, 2002). However, in some areas another species, *S. kellyi* (Dorris *et al.*, 2002), is a very common parasite of children in the PNG highlands and Western Province (King *et al.*, 2004). It is endemic in the Appalachian United States, especially in eastern Tennessee, Kentucky, and West Virginia (Porto *et al.*, 2005). Population at risk also includes those who have recently travelled to or immigrated from endemic areas and Veterans of World War II and Vietnam (Porto *et al.*, 2005). Southeast Asian immigrants living in Washington, DC, were found to have a 38% incidence of infection (Porto *et al.*, 2005). Canadian epidemiology study of Southeast Asian immigrants to Canada demonstrated infection in 11.8% in the Vietnamese population and a 76.6% seroprevalence in Cambodian immigrants (Katz *et al.*, 2004). Sudanese Lost Boys and Girls

and Somali Bantu refugees demonstrated 46% and 23% respective seropositive rate. 5% of Vietnams Veterans reporting mild symptoms demonstrated *S. stercoralis* infection (Posey *et al*., 2007).

Strongyloidiasis causes diarrhoea and malnutrition in Sub-Saharan Africa, although there is little information on its distribution or disease burden, in part because of the difficulties in diagnosing this infection. In one study, Strongyloidiasis accounted for 5.3% of diarrhoea in malnourished Nigerian children (Dada-Adegbola and Bakare, 2004). *Strongyloides stercoralis* is known to be endemic 138 districts in the ten regions of the country, although no definitive prevalence assessment has been done (Ghana Health Service, 2007-2008).

2.13.2. Morphology of *Strongyloides stercoralis*

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

2.13.3. Multiplication and Lifecycle of *Strongyloides stercoralis*

The lifecycle of *Strongyloides* spp. is more complex than that of most nematodes, involving both parasitic and free-living stages (CDC, 2010).

Free-living cycle: The rhabditiform larvae passed in the stool can either moult twice and become infective filariform larvae, or moult four times and become free-living adult males

and females, which then mate and produce eggs from which rhabditiform larvae hatch (CDC, 2010). The filariform larvae either penetrate the human host skin to initiate the parasitic cycle or develop into free-living worms living in the soil independently of a human or mammalian host (CDC, 2010).

Parasitic cycle: Filariform larvae in contaminated soil penetrate the human skin and travel via the bloodstream to the lungs, from where they migrate through the bronchial tree to the pharynx, and are then swallowed and reach the small intestine (CDC, 2010). In the small intestine the filariform larvae moult twice and become adult female worms. The females live threaded in the epithelium of the small intestine and produce eggs, which yield rhabditiform larvae, which can either be passed in the stool or can cause autoinfection (CDC, 2010).

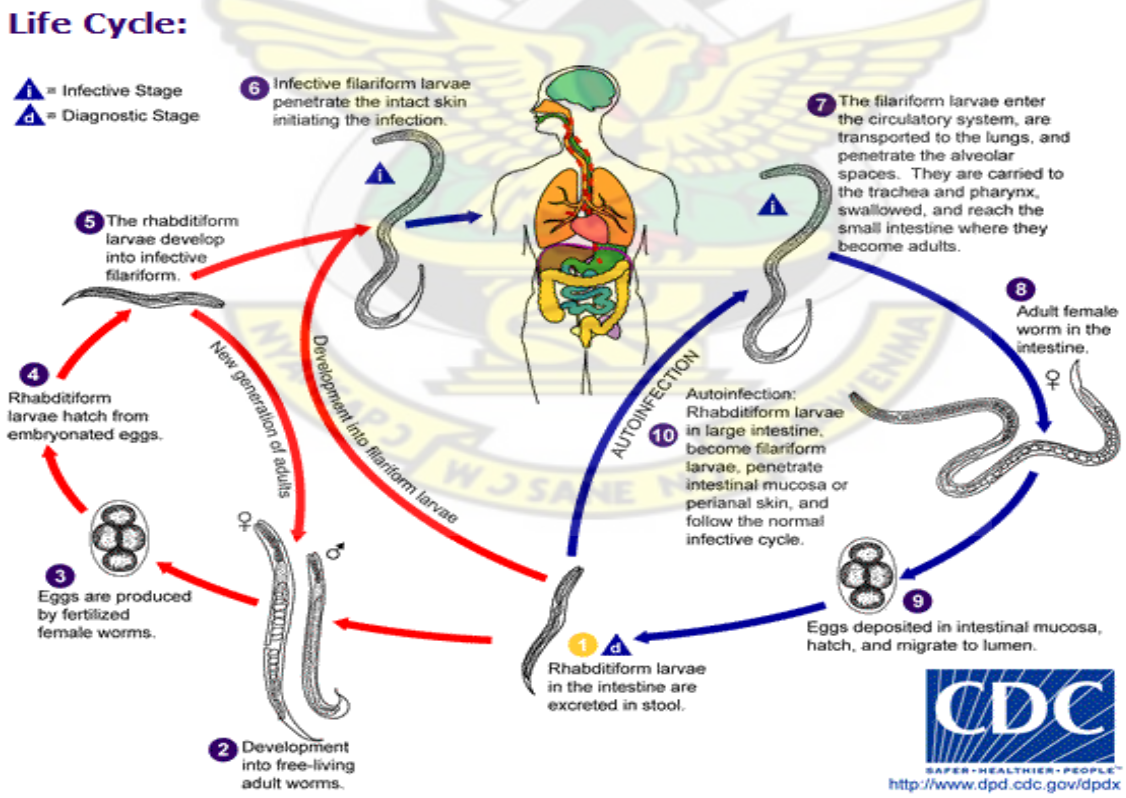


Figure 2.8: Life cycle of *Strongyloides stercoralis*

Source: http://www.dpd.cdc.gov/dpdx/html/Strongyloidiasis.htm

2.13.4. Pathogenesis and Clinical Manifestation

The females live threaded in the epithelium of the small intestine and produce eggs, which yield rhabditiform larvae, which can either be passed in the stool or can cause autoinfection (CDC, 2010). In autoinfection, the larvae become infective filariform larvae, which can penetrate either the intestinal mucosa or the skin of the perianal area. In either case, the filariform larvae travel via the blood stream to the lungs, through the bronchial tree, the pharynx and then the small intestine where they mature into adults, or they may disseminate widely in the body (CDC, 2010). Autoinfection may explain the persistence of infections for many years in people who have not been in an endemic area and hyperinfections in immuno-depressed individuals (CDC, 2010).

Many people infected are usually asymptomatic at first. Symptoms include dermatitis: swelling, itching, mild hemorrhage at the site where the skin has been penetrated (Roberts and Janovy, 2005). If the parasite reaches the lungs, the chest may feel as if it is burning, and wheezing and coughing may result, along with pneumonia-like symptoms (Löffler's syndrome) (Roberts and Janovy, 2005). Eventually, the intestines could be invaded, leading to burning pain, tissue damage, sepsis, and ulcers. In severe cases, edema may result in obstruction of the intestinal tract as well as loss of peristaltic contractions (Roberts and Janovy, 2005). Strongyloidiasis in immunocompetent individuals is usually an indolent disease (Igra-Siegman *et al.*, 1981). However, in immunocompromised individuals, Strongyloidiasis can cause a hyperinfective syndrome (also called disseminated Strongyloidiasis) due to the reproductive capacity of the parasite inside the host (Igra-Siegman *et al.*, 1981). This hyperinfective syndrome has a mortality rate of close to 90% (Igra-Siegman *et al.*, 1981).

2.13.5. Laboratory Diagnosis of *Strongyloides stercoralis*

Diagnosis rests on the microscopic identification of larvae (rhabditiform and occasionally filariform) in the stool or duodenal fluid (Siddiqui *et al.*, 2001). Examination of serial samples may be necessary, and not always sufficient, because stool examination is relatively insensitive (Siddiqui *et al.*, 2001). Larvae may be detected in sputum from patients with disseminated strongyloidiasis (Siddiqui *et al.*, 2001). Other techniques used include direct fecal smears, culturing fecal samples on agar plates, serodiagnosis through ELISA, and duodenal fumigation (Roberts and Janovy, 2005).

2.13.6. Prevention and Control of *Strongyloides stercoralis*

The ideal method of prevention is by improving sanitation (proper disposal of feces), practicing good hygiene (washing of hands), etc., before any drug regimen is administered. Ivermectin 200 micrograms/kg daily for 2 days is the most effective drug for strongyloidiasis (Zaha *et al.*, 2000).

2.14. ENTEROBIUS VERMICULARIS

2.14.1. Epidemiology and Prevalence

The pinworm (Genus *Enterobius*) is a type of roundworm and three species of pinworm have been identified with certainty (NCBI, 2009). Humans are hosts only to *Enterobius vermicularis* (formerly *Oxyuris vermicularis*). Chimpanzees are host to *Enterobius anthropopitheci*, which is morphologically distinguishable from the human pinworm (Nakano *et al.*, 2006). Its existence is controversial however; Totkova *et al*. (2003) consider the evidence to be insufficient (Totkova *et al.*, 2003) and Hasegawa *et al*. (1998) contend that *E. gregorii* is a younger stage of *E. vermicularis* (Hasegawa *et al.*, 1998; Nakano *et al.*, 2006).

The pinworm has a worldwide distribution (Gutiérrez, 2000), and is the most common helminth (i.e. parasitic worm) infection in the United States and Western Europe (Burkhart *et al.*, 2005*).* In the United States, a study by the Center of Disease Control reported an overall incidence rate of 11.4% among people of all ages (Burkhart and Burkhart, 2005). Pinworms are particularly common in children, with prevalence rates in this age group having been reported as high as 61% in India, 50% in England, 39% in Thailand, 37% in Sweden, and 29% in Denmark (Burkhart and Burkhart, 2005). Finger sucking has been shown to increase both incidence and relapse rates (Burkhart and Burkhart, 2005), and nail biting has been similarly associated. Because it spreads from host to host through contamination, pinworms are common among people living in close contact, and tend to occur in all people within a household (Gutiérrez, 2000). The prevalence of pinworms is not associated with gender (Gutiérrez, 2005), nor with any particular social class, race, or culture (Burkhart and Burkhart, 2005). Pinworms are an exception to the tenet that intestinal parasites are uncommon in affluent communities (Burkhart *et al*, 2005; Cook, 1994).

2.14.2. Morphology of *Enterobius vermicularis*

The pinworm appears as a white, small and delicate nematode (i.e. roundworm) (Gutiérrez, 2000*).* The adult female has a sharply pointed posterior end, is 8 to 13 millimetres long, and 0.5 millimeter thick (Gutiérrez, 2000*).* The adult male is considerably smaller, measuring 2 to 5 millimetres long and 0.2 millimeter thick, and has a curved posterior end (Gutiérrez, 2000*).* The eggs are translucent (Gutiérrez, 2000) and have a surface that adheres to environmental objects (Cook, 1994*).* The eggs measure 50 to 60 micrometers by 20 to 30 micrometers, and have a thick shell that is flattened on one side (Gutiérrez, 2000*).* The small size and transparency of the eggs make them invisible to the naked eye, except in barely visible clumps of thousands of eggs (Caldwell, 1982*).* Eggs may contain a developing

embryo or a fully developed pinworm larva (Gutiérrez, 2000). Inside the host, the larvae grow to 140–150 micrometers in length (Cook, 1994).

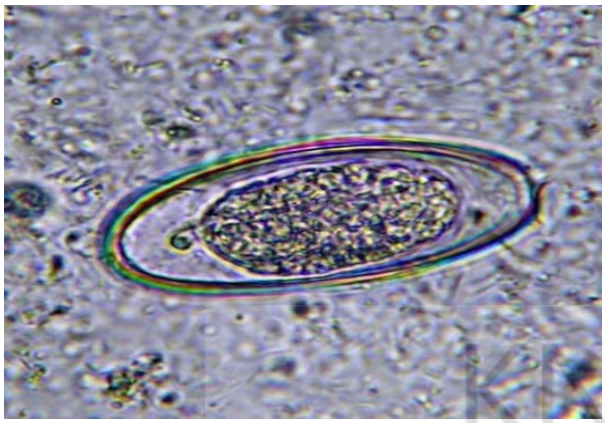


Figure 2.9: Morphology of *Enterobius vermicularis* egg

Source:
http://www.stanford.edu/class/humbio103/ParaSites2006/Enterobius/egg.gif&imgrefurl

2.14.3. Multiplication and Life cycle of *Enterobius vermicularis*

The entire life cycle, from egg to adult, takes place in the human gastrointestinal tract of a single human host (Gutiérrez, 2005; Cook, 1994). While Cook and Zumla (2009) state that the length of development from egg to adult is 2-4 weeks, Burkhart and Burkhart (2005) have put the length of period at 4-8 weeks. The life cycle begins with eggs being ingested (Cook, 1994). The eggs hatch in the duodenum (i.e. first part of the small intestine) (Garcia, 2009). The emerging pinworm larvae grow rapidly to a size of 140 to 150 micrometers in size (Cook *et al.*, 2009), and migrate through the small intestine towards the colon (Cook, 1994). During this migration they moult twice and become adults (Cook, 1994; Burkhart and Burkhart, 2005). Females survive for 5 to 13 weeks, and males about 7 weeks (Cook, 1994). The male and female pinworms mate in the ileum (i.e. last part of the small intestine) (Cook, 1994), where after the male pinworms usually die (Garcia, 2009), and are passed out with stool (Caldwell, 1982). The gravid female pinworms settle in the ileum, caecum (i.e. beginning of the large intestine), appendix and ascending colon (Cook, 1994), where they attach

themselves to the mucosa (Burkhart *et al.*, 2005) and ingest colonic contents (Gutiérrez, 2005). Almost the entire body of a gravid female becomes filled with eggs (Garcia, 2009). The estimations of the number of eggs in a gravid female pinworm is about 11,000 (Cook, 1994) to 16,000 (Burkhart *et al.*, 2005). The egg-laying process begins approximately five weeks after initial ingestion of pinworm eggs by the human host (*Cook*, 1994). The gravid female pinworms migrate through the colon towards the rectum at a rate of 12 to 14 centimetres per hour (Cook, 1994). They emerge from the anus, and while moving on the skin near the anus, the female pinworms deposit eggs either through (1) contracting and expelling the eggs, (2) dying and then disintegrating, or (3) bodily rupture due to the host scratching the worm (Garcia, 2009). After depositing the eggs, the female becomes opaque and dies (Caldwell, 1982). The reason the female emerges from the anus is to obtain the oxygen necessary for the maturation of the eggs (Caldwell, 1982).

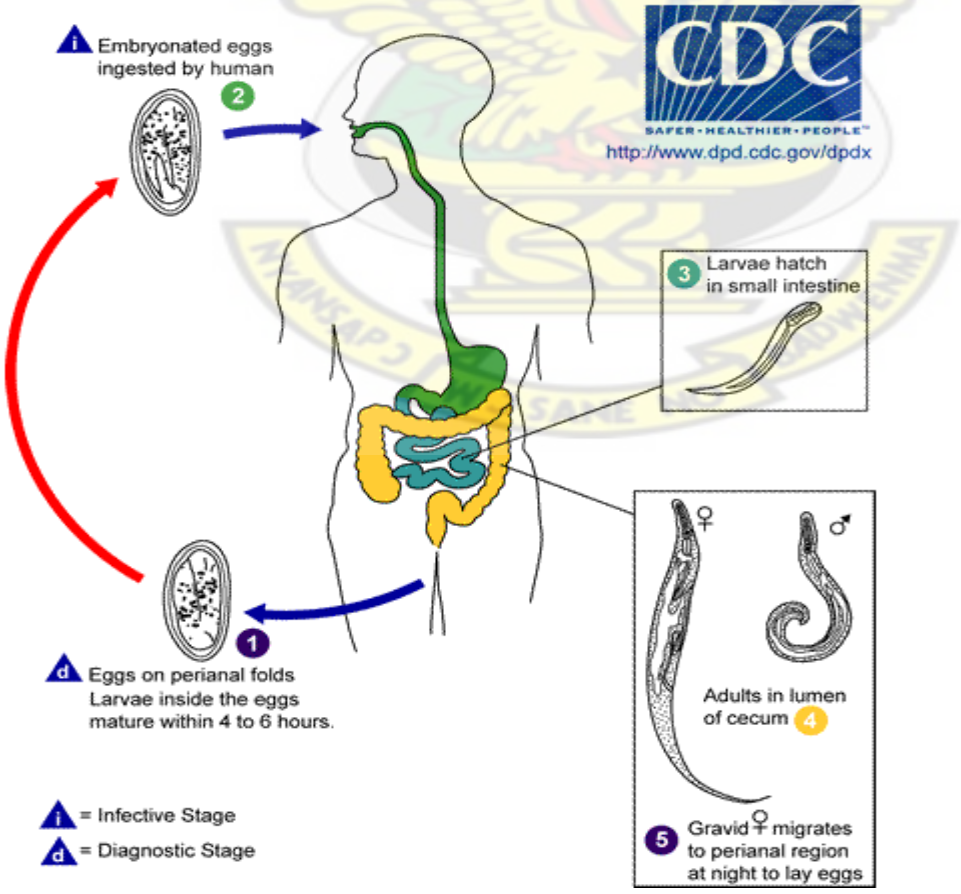


Figure 2.10: Life cycle of *Enterobius vermicularis*

Source: http://www.dpd.cdc.gov/dpdx/html/Enterobiasis.htm

2.14.4. Pathogenesis and Clinical Manifestation

One common symptom is intense pruritus ani that in some patients can lead to insomnia, restlessness and irritability (St Georgiev, 2001). Scratching may cause skin irritation, and in more serious cases, eczematous dermatitis, haemorrhage or secondary bacterial infections (St Georgiev, 2001). Ectopic migration of *E. vermicularis* often results in pinworm infestation of the female genital tract often causing granulomas of the uterus, ovary and the fallopian tubes and pelvic peritoneum (St Georgiev, 2001).

2.14.5. Laboratory Diagnosis of *Enterobius vermicularis* Infection

A person infected with pinworm is often asymptomatic, but itching around the anus is a common symptom (Center of Disease Control, 2010). Diagnosis of pinworm can be reached from three simple techniques. The first option is to look for the worms in the perianal region 2 to 3 hours after the infected person is asleep (Center of Disease Control, 2010). The second option is to touch the perianal skin with transparent tape to collect possible pinworm eggs around the anus first thing in the morning (Center of Disease Control, 2010). If a person is infected, the eggs on the tape will be visible under a microscope (Center of Disease Control, 2010). The tape method should be conducted on 3 consecutive mornings right after the infected person wakes up and before he/she does any washing. Since anal itching is a common symptom of pinworm, the third option for diagnosis is analyzing samples from under fingernails under a microscope (Center of Disease Control, 2010). An infected person who has scratched the anal area may have picked up some pinworm eggs under the nails that could be used for diagnosis (Center of Disease Control, 2010). Since pinworm eggs and

worms are often sparse in stool, examining stool samples is not recommended. Serologic tests are not available for diagnosing pinworm infections (Center of Disease Control, 2010).

2.14.6. Treatment of *Enterobius vermicularis* Infection

The medications used for the treatment of pinworm are mebendazole, pyrantel pamoate, and albendazole. All three of these drugs are to be given in 1 dose at first and then another single dose 2 weeks later (Center of Disease Control, 2010). Pyrantel pamoate is available without prescription. Health practitioners and parents should weigh the health risks and benefits of these drugs for patients under 2 years of age (Center of Disease Control, 2010). Repeated infections should be treated by the same method as the first infection (Center of Disease Control, 2010). In households where more than one member is infected or repeated symptomatic infections occurs, it is recommended that all be treated at the same time, as a group. In institutions, mass and simultaneous treatment repeated in 2 weeks, can be effective (Center of Disease Control, 2010).

2.14.7. Prevention and Control of *Enterobius vermicularis* Infection

Washing of hands with soap and warm water after using the toilet, changing diapers, and washing hand before handling food are the most successful way to prevent pinworm infection (CDC, 2010). In order to stop the spread of pinworm and possible re-infection, people who are infected should bathe every morning to help remove a large amount of the eggs on the skin (CDC, 2010). Showering is a better method than taking a bath, because showering avoids potentially contaminating the bath water with pinworm eggs. Infected people should not co-bathe with others during their time of infection (CDC, 2010). Also, infected people should comply with good hygiene practices such as washing their hands with soap and warm water after using the toilet, changing diapers, and before handling food (CDC, 2010). They

should also cut fingernails regularly, and avoid biting the nails and scratching around the anus. Frequent changing of underclothes and bed linens first thing in the morning is a great way to prevent possible transmission of eggs in the environment and risk of reinfection (CDC, 2010).

CHAPTER THREE

3.0. MATERIALS AND METHODS

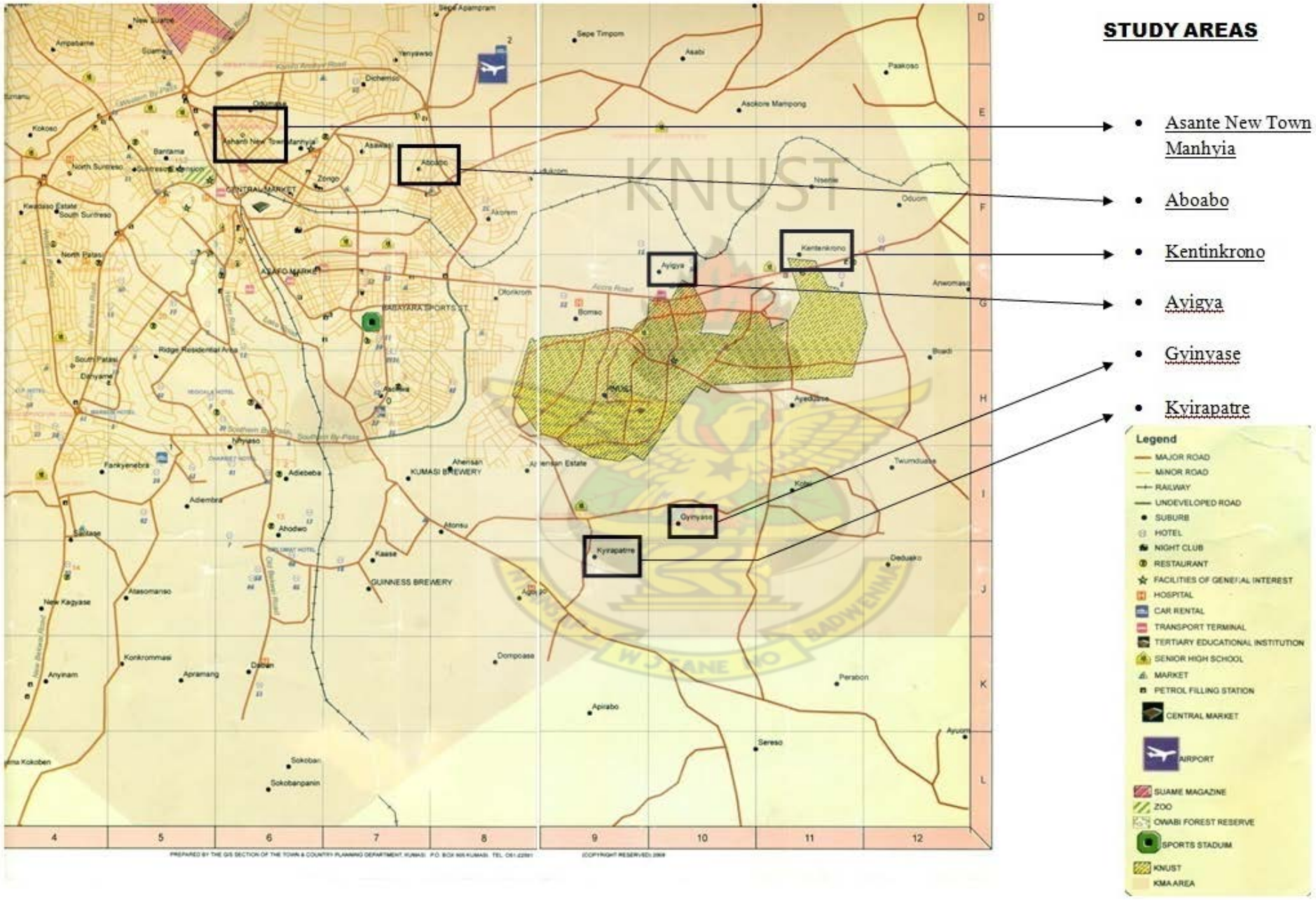
3.1. STUDY DESIGN AND STUDY AREA

A cross-sectional study was conducted within peri-urban (Kentinkrono, Gyinyase and Kyirapatre), urban-poor (Ayigya and Aboabo) and urban (Manhyia) communities in Kumasi Metropolitan Area from January to September (2011). Kumasi is the second largest city in Ghana located in the transitional forest zone of West Africa and is about 270 km north of the national capital, Accra. It is between latitude 6.35^{o} – 6.40^{o} and longitude 1.30^{o} – 1.35^{o}, an elevation which ranges between 250 – 300 metres above sea level with an area of about 254 square kilometres. The Kumasi metropolis is the most populous district in the Ashanti Region. During the 2000 Population Census it recorded a figure of 1,170,270 (Ghana Statistical Service, 2000-2002). It has been projected to have a population of 1,625,180 in 2006 based on a growth rate of 5.4% p.a and this accounts for just under a third (32.4%) of the region's population (Population Census Report, 2006). The age structure of the population in the metropolis is skewed towards the youth (2000 Population census). The city falls within the moist semi-deciduous South-East Ecological Zone of annual rainfall of around 1400mm with two distinct rainy seasons.

The peri-urban area of Kumasi had previously been defined by Natural Resources Institute, UK, as a radius of 40km around the city (Adam *et al.*, 2001). The peri-urban communities were chosen relatively close to the city. The urban boundary was adapted from Kumasi Metropolitan Assembly.

Three of the communities were peri-urban (Kentinkrono, Gyinyase and Kyirapatre), two were urban-poor (Ayigya and Aboabo) and an urban community (Manhyia) as shown on Fig. 3.1.

Figure 3.1: The Map of a Section of Kumasi



Source: Town and country planning department, Kumasi, 2008

3.2. STUDY POPULATION

The study population comprises children of school going age from age five (5) to twelve (12) within six selected communities in Kumasi metropolis: Ayigya, Kentinkrono, Aboabo, Manhyia, Gyinyase and Kyirapatre. A primary school each was selected in the six communities for the study. These included Ayigya M/A primary school, Kentinkrono M/A primary school, Aboabo M/A primary school, Afia Kobi Serwaa Ampem M/A primary school, Gyinyase M/A primary school and Kyirapatre R/C primary school. The sample size was achieved by using the Fisher's sampling formula:

$$N = \frac{Z^2 PQ}{d^2}$$

(Z= this is the critical value of the standard normal distribution (1.96 at 95% confidence interval); P=estimated prevalence of intestinal infection; Q= 100-P and d = absolute precision or sampling error tolerated =5%). The minimum calculated sample size that can be use for this study is 384 participants.

3.3. SAMPLING

A simple random sampling technique was used to select the study population. This was achieved by selecting pupils from age five to twelve. Pupils were allowed to randomly pick cards inscribed 'Yes' or 'No'. Those who picked 'Yes' were included in the study. The exclusive criteria covers pupil below 5 years and those above 12 years, as well as pupils who were sick at the time of the study. A labelled clean plastic container was given to each participant. The container bears the participants identity number, the age, sex and class. The teachers in the various schools helped to educate the children on how to collect the sample properly. The parents were educated to assist their children during stool collection.

3.4. DATA COLLECTION

A closed questionnaire was used to collect information from the parents and teachers concerning the child's household sociodemographic, personal hygiene, and water and sanitation characteristics.

3.5. SAMPLE PROCESSING AND MICROSCOPIC ANALYSIS

A fresh stool sample was obtained from each participant. These fresh stool samples were transported to the Diagnostic Microbiology laboratory for analysis. After a gross examination of the sample characteristics, a direct wet faecal smear was prepared from each of the fresh samples by emulsifying about 2 mg of the stool sample on a clean 26x76 mm glass slide in a drop of lugol's iodine (Appendix 2.0). The sample was covered with a 22x22 mm cover slip and observed using low power (x10) and high power (x40) objectives for the identification of protozoan trophozoites and cyst, helminth ova and larvae (Appendix 2.0). A smear was also made from the fresh stool samples on 26x76 mm glass slide and stained with Modified Ziehl Neelsen acid-fast stain (modified kinyoun acid-fast stain) as described by Garcia (2001) for the identification of the oocysts of *Cryptosporidium parvum* using the x40 and x100 objectives (Appendix 4.0). Formol ether concentration method was performed on each of the stool samples (Appendix 3.0). The sediment obtained was stained with Lugol's iodine and was mixed thoroughly. A drop of the iodine stained sediment was placed on a clean 26x76 mm glass slide and covered with a 22x22 mm cover slip. The protozoan cyst was identified using x40 objective lens. Morphological features used in the identification of the parasites microscopically was aided by pictures and colour atlases provided by Washington *et al*, 2006; WHO, 1998; CDC, 2010 and Cheesbrough, 2007.

3.6. STATISTICAL ANALYSIS

The data obtained from 2400 primary school children who participated in the current study was entered into SPSS 16.0 version statistical package. The software package was used to determine the frequency distribution of the studied children in the various communities with respect to the intestinal parasitic infection. Multinomial logistic regression model was used to determine the association between the response variables (intestinal parasitic infections) and risk factors and their levels of significance ($p < 0.05$).

CHAPTER FOUR

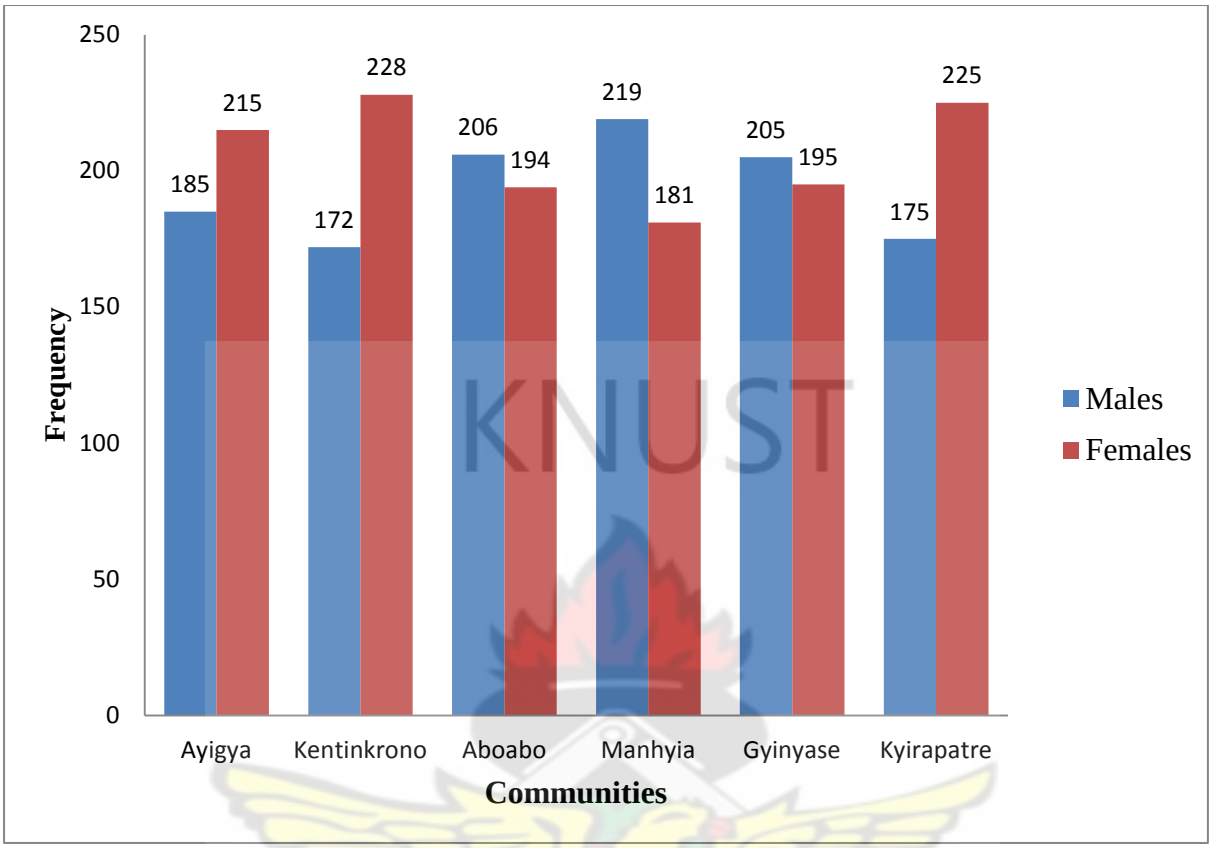
4.0. RESULTS

4.1. Total Number of Primary School Children Sampled.

A total of 2400 primary school children from six communities in Kumasi: Ayigya, Kentinkrono, Aboabo, Manhyia, Gyinyase and Kyirapatre were enrolled in the study.

Out of 2400 participants, 1162/2400 (48.42%) were males and 1238/2400 (51.58%) were females with the ages from 5-12 years (Fig. 4.1). In Ayigya M/A, 185/400 (46.25%) males and 215/400 (53.75%), Kentinkrono M/A 172/400 (43.00%) were males and 228/400 (57.00%) were females, Aboabo M/A 206/400 (54.75%) males, 194/400 (48.50%) females, Afia Kobi Serwaa Ampem M/A 219/400 (54.50%) were male and 181/400 (45.25%) were females, Gyinyase M/A 205/400 (51.25%) were males and 195/400 (48.75%) were females and Kyirapatre R/C 175/400 (43.75%) were males and 225/400(56.25%) were females (Fig. 4.1).

Figure 4.1. Total number of males and females participants in the various communities.



4.2. Demographic, Household Sanitary and Water Source of Children Studied.

The Primary school children who were enrolled to participate in the current research in Ayigya, Kentinkrono, Aboabo, Manhyia, Gyinyase and Kyirapatre were between the ages of 5-12years. Most of the children were within the ages of 11-12yrs, 840(35.00%), 625(26.04%) were between 9-10 years, 529(22.04%) were between 7-8 years and 406 (16.92%) were between 5-6 years (Table 4.1).

Most of these children are of the Akan ethnicity 1200/2400 (50.00%), 920/2400 (38.33%) from different ethnicities in Northern Ghana and 280/2400 (11.67%) from other ethnicities in

Ghana. 1410/2400 (58.75%) of the children were Christians and 990/2400 (41.25%) were from the Islamic religion (Table 4.1).

Table 4.1 also indicates that a majority, 1657/2400 (69.04%) of the studied school children relied mostly on pipe-borne water and 743/2400(30.96%) relied on borehole/Well as their source of water for drinking, cooking and other household chores in the six communities. A total of 400/2400 (16.67%) of the respondent relied on water closet (W.C), 618/2400 (25.75%) relied on Pit latrine and 1152/2400 (48.00%) relied on community KVIP (Kumasi Ventilated Improved Pits) as their sanitary source. 230/2400 (9.58%) of the children defecate in nearby bushes.

A majority, 835/2400(34.79%) of the respondent's parent were Traders, 589/2400 (24.54%) were businessmen, 504/2400(21.00%) were civil servants, 250/2400(10.42%) were unemployed, 170/2400 (7.08%) were farmers and 52/2400 (2.17%) were carpenters (Table 4.1).

Table 4.1 Demography, Household Sanitary and Water Source of Children Studied.

VARIABLE	TOTAL
Residence interviewed	
Ayigya	n=400
Kentinkrono	n=400
Aboabo	n=400
Manhyia	n=400
Gyinyase	n=400
Kyirapatre	n=400
Gender	
Male	1162/2400 (48.42%)
Female	1238/2400 (51.58%)
Age	
5-6yrs	406 (16.92%)
7-8yrs	529(22.04%)
9-10yrs	625(26.04%)
11-12yrs	840(35.00%)
Ethnicity	
Akan	1200/2400 (50.00%)
Northern	920/2400 (38.33%)
Others	280/2400 (11.67%)
Religion	
Christian	1410/2400 (58.75%)
Islam	990/2400 (41.25%)
Parent's occupation	
Farmer	160/2400 (6.67%)
Civil servant	489/2400(20.38%)
Trader	780/2400(32.0%)
carpenter	52/2400 (2.17%)
Unemployed	230/2400(9.58%)
Businessmen	574/2400 (23.92%)
Household water source	
Pipe-borne water	1657/2400 (69.04%)
Borehole/Well	743/2400(30.96%)
Household Sanitary facility	
No facility(Bush/.field)	230/2400 (9.58%)
W.C	400/2400 (16.67%)
Pit latrine	618/2400 (25.75%)
KVIP	1152/2400 (48.00%)

4.3. Macroscopic Examination of Stool Specimen.

The macroscopic examinations of the stool samples received from the participants were noted. In all, 950/2400 (39.58%) of the stool samples were formed (hard), 1215/2400 (50.63%) were semiformed (soft), 175 (7.29%) were loose, 35/2400 (1.46%) were watery and 25 (1.04%) were bloody-mucoid (Table 4.2).

Table 4.2. Relative frequencies of the types of consistency of 2400 stool samples collected from the studied primary school children.

Consistency Type	Number	Frequency %
Formed(hard)	950	39.58
Semiformed(soft)	1215	50.63
Loose	175	7.29
Watery	35	1.46
Bloody-mucoid	25	1.04
Total	**2400**	**100**

4.4. Intestinal Parasitic Infections identified by microscopy.

The results of 2400 stools analysed revealed ten species of intestinal parasites; seven protozoans and three species of helminth (Hookworm, *Ascaris lumbricoides* and *Strongyloides stercoralis*). Three of the intestinal protozoans were pathogenic (*Giardia lamblia, Entamoeba histolytica/dispar* and *Cryptosporidium parvum)* and four were non-pathogenic species (*Entamoeba coli, Endolimax nana, Chilomastix mesnili* and *Iodamoeba butschlii*). These intestinal parasites where differentiated based on their morphological characteristics and the measurement of their length and width (Appendix 5.0). The overall prevalence of intestinal parasites identified among the studied school children was 49.18%. Out of the overall prevalence of intestinal parasitic infections, 42.88% were intestinal protozoa and 6.30% were helminth. The studied children had 292/2400 (12.17%) *Giardia lamblia*, 5/2400 (0.21%) had Entamoeba *histolytica,* 204/2400 (8.50%) had *Cryptosporidium parvum*, 248/2400 (10.33%) had *Entamoeba coli,* 169/2400 (7.04%) had *Endolimax nana,*

76/2400 (3.17%) had *Chilomastix mesnili*, 34/2400 (1.46%) had *Iodamoeba butschlii*, 37/2400 (1.54%) had Hookworm, 93/2400 (3.88%) had *Ascaris lumbricoides* and 35/2400 (0.88%) had *Strongyloides stercoralis* infections in the six communities. The results obtained from individual communities are shown in the Table 4.3.

Table 4.3.Intestinal Parasites identified microscopically in the stool samples of the primary school children in the six communities.

	Ayigya	**Kentinkrono**	**Aboabo**	**Manhyia**	**Gyinyase**	**Kyirapatre**	**Total**
Intestinal Parasites	F(%,n=400)	F(%,n=400)	F(%,n=400)	F(%,n=400)	F(%,n=400)	F(%,n=400)	
G. lamblia	45(11.25)	72(18.00)	37(9.25)	20(5.00)	62(15.50)	56(14.00)	292(12.17)
E. histolytica/ dispar	1(0.25)	3(0.75)	1(0.25)	-	-	-	5(0.21)
C. parvum	30(7.50)	50(12.50)	25(6.25)	16(4.00)	38(9.50)	45(11.25)	204(8.50)
E.coli	32(8.00)	55(13.75)	32(8.00)	26(6.50	45(11.25)	58(14.00)	248(10.33)
E. nana	20(5.00)	35(8.75)	25(6.25)	18(4.50)	30(7.50)	41(10.25)	169(7.04)
I.butschlii	5(1.25)	10(2.50)	5(1.25)	3(0.75)	5(1.25)	7(1.75)	35(1.46)
C. mesnili	10(2.50)	20(5.00)	10(2.50)	2(0.50)	14(3.50)	20(5.00)	76(3.17)
Hookworm	5(1.25)	15(3.75)	2(0.50)	-	5(1.25)	10(2.50)	37(1.54)
Ascaris lumbricoides	10(2.50)	30(7.50)	10(2.50)	3(0.75)	20(5.00)	20(5.00)	93(3.88)
Strongyloides stercolaris	2(0.50)	9(2.25)	2(0.50)	-	3(0.75)	5(1.25)	21(0.88)
Total	178(44.5)	299(74.75)	149(37.25)	88(22.0)	222(55.50)	262(65.50)	1180(49.18)

NB: *G. lamblia = Giardia lamblia, E. histolytica= Entamoeba histolytica, E. coli= Entamoeba coli, C. parvum = Cryptosporidium parvum, E. nana = Endolimax nana, I. butschlii = Iodamoeba butschlii, C. mesnili = Chilomastix mesnil*

Table 4.4.Direct smear preparation of the stool samples.

Intestinal Parasites	Ayigya	Kentinkrono	Aboabo	Manhyia	Gyinyase	Kyirapatre	Total
	F(%,n=400)	F(%,n=400)	F(%,n=400)	F(%,n=400)	F(%,n=400)	F(%,n=400)	
G. lamblia cyst	15(3.75)	30(7.50)	15(3.75)	6(1.50)	18(4.50)	15(3.75)	99(4.13)
G. lamblia trophozoites	20(5.00)	15(3.75)	10(2.50)	6(1.50)	20(5.00)	14(3.50)	85(3.54)
E. histolytica /dispar cyst	-	-	-	-	-	-	-
E. histolytica trophozoites	-	2(0.50)	-	-	-	-	2(0.08)
Hookworm	3(0.75)	10(2.50)	-	-	2(0.25)	10(2.50)	25(1.04)
Ascaris. lumbricoides	5(1.25)	20(5.00)	3(1.50)	1(0.25)	10(2.50)	12(3.00)	51(2.13)
Strongyloides stercolaris	2(0.25)	6(1.50)	2(0.25)	-	3(1.50)	4(1.00)	17(0.71)
Total	45(11.25)	83(20.75)	30(7.50)	13(3.25)	53(13.25)	55(13.75)	279(11.6)

Giardia lamblia infection was the highest intestinal protozoan recorded from the current study (Table 4.3). Kentinkrono, Ayigya, Kyirapatre, Gyinyase, Aboabo and Manhyia recorded 18.00%, 11.25%, 14.00%, 15.50%, 9.25% and 5.00% respectively. *Cryptosporidium parvum* infection was the highest in Kentinkrono 12.50%, followed by Kyirapatre (11.25%), Gyinyase (9.50%), Ayigya (7.50%), Aboabo (6.25%) and the lowest in Manhyia (4.00%). *Entamoeba histolytica/dispar* was highest in Kentinkrono (1.5%), followed by Ayigya (0.5%) and Aboabo (0.5%). The prevalence of *Ascaris lumbricoides* infection was highest in Kentinkrono 7.50%, followed by Gyinyase 5.00%, Kyirapatre 5.00%, Ayigya 2.50%, Aboabo 2.50% and Manhyia 0.75%. Hookworm infection was 3.75% in Kentinkrono, 2.50% in Kyirapatre, 1.25% in Ayigya and Gyinyase, 0.50% in Aboabo. There was no hookworm infection in Manhyia. The prevalences of *Strongyloides stercolaris* infection were 2.25%, 1.25%, 0.75%, 0.50% and 0.50% in Kentinkrono, Kyirapatre,

Gyinyase, Ayigya and Aboabo respectively. All the intestinal parasites recorded were low using the direct wet smear method (Table 4.4).

4.5. Overall distribution of intestinal parasitic infection among the studied children by sex.

Out of 2400 stool samples analysed, the prevalence rate of intestinal parasites was higher in males than females. Of the 1162 males enrolled, 614 (65.66%) were infected while 404/1238 (34.34%) females were infected with intestinal parasitic infections. The highest parasitic infection was *Giardia lamblia* with the prevalence of 16.78% (195/1162) and 7.84 % (97/1238) in males and females respectively. The lowest infection was *Entamoeba histolytica/dispar* 0.34% (4/1162) and 0.08% (1/1238) in males and females respectively (Table 4.5).

Table 4.5. The overall distribution of parasitic infections by sex.

Intestinal Parasite	**Number infected (%)**		***P-value***
	Males = 1162	**Female =1238**	
G. lamblia	195(16.78)	97(7.84)	0.001
E. histolytica/dispar	4(0.34)	1(0.08)	0.15
C. parvum	129(11.10)	75(6.06)	0.001
E.coli	149(12.82)	99(7.99)	0.01
E. nana	105(9.04)	64(5.17)	0.001
I.butschlii	18(1.55)	17(1.37)	0.22
C. mesnili	48(4.13)	28(3.07)	0.06
Hookworm	30(2.58)	7(0.57)	0.001
Ascaris lumbricoides	68(5.85)	25(2.02)	0.01
Strongyloides stercoralis	17(1.46)	4(0.32)	0.001

4.6 Age distribution of Intestinal Parasitic infections among studied children in the six communities.

There were variations of intestinal parasitic infections with respect to age in the six communities (Table 4.6). In Ayigya, the prevalence of *Giardia lamblia* infection among the studied children between 5-6 years, 7-8 years, 9-10 years and 11-12 years were 33.87% (21/62), 11.76% (10/85), 8.00% (8/100) and 3.92% (6/153) respectively. *Entamoeba histolytica/dispar* was found only among 5-6 years with a prevalence of 1.61% (1/62). The prevalence of *Cryptosporidium parvum* infection between 5-6 years, 7-8 years, 9-10 years and 11-12 years in Ayigya were 24.17% (15/62), 12.97% (11/85), 3.00% (3/100) and 0.65% (1/153) respectively. The prevalence of the non- pathogenic parasitic infection (*Entamoeba coli, Endolimax nana, Chilomastix mesnili* and *Iodamoeba butschlii)* was higher in 5-6 age groups than in 7-8 years, 9-10 years and 11-12years. The prevalence of Hookworm among 7-8, 9-10 and 11-12 age groups were 2.35% (2/85), 1.00% (1/100) and 1.31% (2/153) respectively. None of the studied children between 5-6 years had hookworm infection. The prevalences of *Ascaris lumbricoides* infection recorded among the ages 5-6, 7-8, 9-10 and 11-12 years are 8.04% (5/62), 2.35% (2/85), 2.00% (2/100) and 0.65% (1/153) respectively. *Strongyloides stercolaris* infection was recorded in ages 5-6 and 7-8 years with prevalences of 1.61% (1/62) and 1.18% respectively (Table 4.6).

In Kentinkrono, the highest prevalence of *Giardia lamblia* infection was recorded in the 5-6years age group with 53.85% (42/78). Prevalences of 25.00% (16/64), 11.11% (10/90) and 2.38% (4/168) were recorded in age groups of 7-8years, 9-10 years and 11-12years respectively. The prevalence of *Entamoeba histolytica/dispar* infection was recorded only among the studied children between 5-6years and 7-8 years with prevalences of 2.56% (2/78) and 1.56% (1/64). The lowest prevalence was recorded in the 11-12 years age group. Hookworm infection was recorded only among primary school children in Kentinkrono. The

prevalences were 2.22% (1/45), 5.41% (2/37), 2.08% (1/48) and 4.29% (3/70) in the 5-6years, 7-8 years, 9-11 years and 11-12 years age groups. Also, 30.77% (24/78), 21.88% (14/64), 8.89% (8/90) and 2.38% (4/168) were the prevalences of *Cryptosporidium parvum* infection recorded among the studied children in 5-6, 7-8, 9-10 and 11-12 age groups respectively. The non pathogenic intestinal parasite also varies with age differences (Table 4.6).

From table 4.6, the prevalences of hookworm infection recorded among 5-6years, 7-8 years, 9-10 years and 11-12years age groups were 3.85% (3/78), 4.69% (3/64), 3.33% (3/90) and 3.57% (6/168) respectively. *Ascaris lumbricoides* infection was highest among children of 5-6 years with prevalence of 16.67% (13/78), followed by 7-8years 14.06% (9/64), 9-10 years 5.55% (5/90) and 11-12 years 1.79% (3/168). 6.41% (5/78), 3.13% (2/64), 1.11% (1/90) and 0.59% (1/168) were prevalences of *Strongyloides stercoralis* infection recorded among 5-6, 7-8, 9-10 and 11-12 age groups.

Studied children from Aboabo in the 5-6 age groups recorded the highest prevalence (20.59%) of *Giardia lamblia* infection in the community. The lowest prevalence was recorded among the 11-12 age groups (1.78%). Children between 7-8 years and 9-10years recorded 16.00% (12/78) and 9.09% (8/88) respectively. *Entamoeba histolytica /dispar* infection was recorded only among children in the 9-10 age groups with a prevalence of 1.14% (1/88). *Cryptosporidium parvum* infection was recorded among children in the 5-6, 7-8, 9-10 and 11-12 age groups with prevalences of 14.71% (10/68), 9.33% (7/75), 6.82% (6/88) and 1.18(2/169) respectively. The non pathogenic parasites were also high in the younger age (5-6 years and7-8 years) than the older age (9-10years and 11-12years). Hookworm infection was recorded among the studied children between the ages of 11-12 years (1.18%). 5.88% (4/68), 4.00% (3/75), 1.14% (1/88) and 1.18% (2/169) were the

prevalences of *Ascaris lumbricoides* infection among children in 5-6, 7-8, 9-10 and 11-12 age groups respectively. *Strongyloides stercolaris* infection was recorded among ages 5-6 years and 7-8 years with prevalences of 1.47% (1/68) and 1.33% (1/75) respectively.

Intestinal parasites recorded among the studied children in Manhyia were low. The prevalence of *Giardia lamblia* infection was highest among children in the 5-6years (16.67%) age group and lowest in 11-12 years (0.79%) age groups. Children between 7-8years and 9-10years recorded 3.64% (4/110) and 2.88% (3/104) prevalence of *Giardia lamblia* infection. None of the children were infected with *Entamoeba histolytica*/*dispar*, Hookworm and *Strongyloides stercolaris* infections. The prevalence *Cryptosporidium parvum* recorded among the 5-6, 7-8, 9-10 and 11-12 age groups are 16.67(10/60), 2.73% (3/110), 1.92% (2/104) and 0.79% (1/126) respectively. *Ascaris lumbricoides* infection was present among children between ages 5-6 years, 7-8 years and 9-10 years with prevalences of 1.67% (1/60), 0.91% (1/110) and 0.96% (1/104) respectively. The non pathogenic intestinal parasite also varies with age differences.

The prevalence of *Giardia lamblia* infection among studied children between 5-6 years, 7-8 years, 9-10 years and 11-12 years were 44.83% (26/58), 20.00% (20/100), 7.81% (10/128), and 5.26% (6/114) respectively in Gyinyase. The highest prevalence was 44.83% (5-6 years) and the lowest was 5.26% (11-12 years). None of the children were infected with *Entamoeba histolytica* and helminth infection. *Cryptosporidium parvum* infection was high among children in 5-6 age groups with prevalence of 29.31% (17/58). 8.00 (8/100), 5.47% (7/128), 5.26% (6/114) are the prevalences of *Cryptosporidium parvum* among 7-8, 9-10 and 11-12 age group. Hookworm infection was also recorded among the studied population in Gyinyase with prevalences of 1.72% (5-6 years), 1.00% (7-8 years), 0.78% (9-10years) and 1.75% (11-12 years). 17.24% (10/58), 5.00% (5/100), 2.34% (3/128) and 1.75% (2/114) were

the prevalences of *Ascaris lumbricoides* infection recorded among the 5-6, 7-8, 9-10 and 11-12 age group.

Strongyloides stercoralis infestation was recorded among 5-6, 7-8, and 11-12 age groups with prevalences of 1.72% (1/58), 1.00% (1/100) and 0.88% (1/114) respectively. The non-pathogenic intestinal parasite also varies with age differences.

In Kyirapatre, The highest prevalence of *Giardia lamblia* infection was recorded among the 5-6 age groups with a prevalence of 40.00% (32/80). The lowest prevalence was recorded among the 9-10 age groups with 6.36%. The prevalences of *Cryptosporidium parvum* infection recorded among 5-6, 7-8, 9-10 and 11-12 age groups are 32.50% (26/80), 10.53% (10/95), 3.48% (4/115) and 4.55% (4/110) respectively. None of the children were infected with *Entamoeba histolytica/ dispar*. Hookworm infection was highest among the 11-12 age group with a prevalence of 4.55% (5/110) and lowest among the 7-8 age group (1.05%). The prevalence rate of Hookworm infection among 5-6 years and 9-10 years age groups are 2.50% and 1.74% respectively. The prevalence of *Ascaris lumbricoides* infection recorded among 5-6, 7-8, 9-10 and 11-12 age groups were 13.75% (11/80), 5.26% (5/95), 2.61% (3/115) and 0.91% (1/110) respectively. The prevalence of *Strongyloides stercoralis* infection recorded among 5-6, 7-8, 9-10 and 11-12 age group are 2.50% (2/80), 2.11% (2/95), 0.87% (1/115) and 0.91% (1/110) (Table 4.6).

Table 4.6. Age distribution of Intestinal Parasitic infections among studied children in the six communities.

AYIGYA	**AGES**			
INFECTIONS	**5-6YRS(62)**	**7-8YRS(85)**	**9-10YRS(100)**	**11-12YRS(153)**
G.lamblia	21(33.87)	10(11.76)	8(8.00)	6(3.92)
E. histolytica/dispar	1(1.61)	-	-	-
E. coli	17(27.42)	8(9.41)	5(5.00)	2(1.31)
E.nana	6(9.68)	6(7.06)	2(2.00)	6(3.92)
I.butschlii	2(3.23)	1(1.18)	1(1.00)	1(0.65)
C. mesnili	7(11.29)	1(1.18)	1(1.00)	1(0.65)
C. parvum	15(24.19)	11(12.97)	3(3.00)	1(0.65)
Hookworm	-	2(2.35)	1(1.00)	2(1.31)
A. lumbricoides	5(8.04)	2(2.35)	2(2.00)	1(0.65)
S. stercolaris	1(1.61)	1(1.18)	-	-
KENTINKRONO	**5-6YRS(78)**	**7-8YRS(64)**	**9-10YRS(90)**	**11-12YRS(168)**
G.lamblia	42(53.85)	16(25.00)	10(11.11)	4(2.38)
E. histolytica/ dispar	2(2.56)	1(1.56)	-	-
E. coli	23(29.49)	18(28.13)	9(10.00)	5(2.98)
E.nana	5(6.41)	7(10.94)	6(6.67)	17(10.11)
I.butschlii	3(3.85)	2(3.13)	3(3.33)	2(1.19)
C. mesnili	4(5.13)	2(3.13)	1(1.11)	13(7.74)
C. parvum	24(30.77)	14(21.88)	8(8.89)	4(2.38)
Hookworm	3(3.85)	3(4.69)	3(3.33)	6(3.57)
A. lumbricoides	13(16.67)	9(14.06)	5(5.55)	3(1.79)
S. stercolaris	5(6.41)	2(3.13)	1(1.11)	1(0.59)
ABOABO	**5-6YRS(68)**	**7-8YRS(75)**	**9-10YRS(88)**	**11-12YRS(169)**
G.lamblia	14(20.59)	12(16.00)	8(9.09)	3(1.78)
E. histolytica /dispar	-	-	1(1.14)	-
E. coli	3(4.41)	1(1.33)	1(1.14)	2(1.18)
E.nana	13(19.12)	11(14.67)	5(5.68)	6(3.55)
I.butschlii	1(1.47)	1(1.33)	1(1.14)	1(0.59)
C. mesnili	6(8.82)	1(1.33)	2(2.27)	1(0.59)
C. parvum	10(14.71)	7(9.33)	6(6.82)	2(1.18)
Hookworm	-	-	-	2(1.18)
A. lumbricoides	4(5.88)	3(4.00)	1(1.14)	2(1.18)
S. stercolaris	1(1.47)	1(1.33)	-	-
MANHYIA	**5-6YRS(60)**	**7-8YRS(110)**	**9-10YRS(104)**	**11-12YRS(126)**
G.lamblia	10(16.67)	4(3.64)	3(2.88)	1(0.79)
E. histolytica/ dispar	-	-	-	-
E. coli	6(10.00)	6(5.45)	10(9.62)	4(3.17)
E.nana	8(13.33)	3(2.73)	2(1.92)	5(3.97)
I.butschlii	1(1.67)	1(0.19)	-	1(0.79)
C. mesnili	1(1.67)	-	-	1(0.79)
C. parvum	10(16.67)	3(2.73)	2(1.92)	1(0.79)

Hookworm	-	-	-	-
A. lumbricoides	1(1.67)	1(0.91)	1(0.96)	-
S. stercolaris	-	-	-	-
GYINYASE	**5-6YRS(58)**	**7-8YRS(100)**	**9-10YRS(128)**	**11-12YRS(114)**
G.lamblia	26(44.83)	20(20.00)	10(7.81)	6(5.26)
E. histolytica/ dispar	-	-	-	-
E. coli	10(17.24)	10(10.00)	5(3.91)	20(17.54)
E.nana	16(27.58)	9(9.00)	4(3.13)	2(1.75)
I.butschlii	1(1.72)	2(2.00)	1(0.78)	1(0.88)
C. mesnili	3(5.17)	4(4.00)	3(2.34)	1(0.88)
C. parvum	17(29.31)	8(8.00)	7(5.47)	6(5.26)
Hookworm	1(1.72)	1(1.00)	1(0.78)	2(1.75)
A. lumbricoides	10(17.24)	5(5.00)	3(2.34)	2(1.75)
S. stercolaris	1(1.72)	1(1.00)	-	1(0.88)
KYIRAPATRE	**5-6YRS(80)**	**7-8YRS(95)**	**9-10YRS(115)**	**11-12YRS(110)**
G.lamblia	32(40.00)	10(10.53)	7(6.09)	7(6.36)
E. histolytica /dispar	-	-	-	-
E. coli	20(25.00)	19(20.00)	6(5.22)	13(11.82)
E.nana	20(25.00)	7(7.37)	10(8.69)	4(3.64)
I.butschlii	2(2.50)	1(1.05)	3(2.61)	1(0.91)
C. mesnili	10(12.50)	3(3.16)	4(3.48)	3(2.73)
C. parvum	26(32.50)	10(10.53)	4(3.48)	5(4.55)
Hookworm	2(2.50)	1(1.05)	2(1.74)	5(4.55)
A. lumbricoides	11(13.75)	5(5.26)	3(2.61)	1(0.91)
S. stercolaris	2(2.50)	2(2.11)	1(0.87)	1(0.91)

NB: *E. coli= Entamoeba coli, G. lamblia= Giardia lamblia. E. histolytica=Entamoeba histolytica, C. parvum=Cryptosporidium parvum, I. butschlii =Iodamoeba butschlii* and *C. mesnili=Chilomastix mesnili*

4.7 Proportion and Association of *Giardia lamblia* and *Entamoeba histolytica* infections in Ayigya.

Giardia lamblia infection was statistically significant ($p < 0.05$) among males than females. *Giardia lamblia* infection was statistically significant ($p < 0.05$) among 5-6years age group compared to 7-8years, 9-10years and 11-12years age groups (Table 4.7). Studied children who lack sanitary facilities (bushes or field) had a prevalence rate of 26.67% as compared to those who use water closet (11.11%), pit latrine (15.29%) and KVIP (7.92%) (Table 4.7). *Giardia lamblia* infection was statistically significant ($p < 0.05$) among children who do not wash their hand before eating and after defecation. The prevalence rate of *Giardia lamblia* infection was statistically significant ($p < 0.05$) among children whose parents were farmers. From table 4.7, *Giardia lamblia* infection was high among children who depend on boreholes (14.71%) as their source of drinking water compared to those who use pipe-borne water (10.07%). The difference in the rate of *Giardia lamblia* infection was not significant ($p > 0.05$). Children who buy food from food vendors around their school compound had a prevalence of 17.60% and those who bring their food from their various homes had a prevalence of 17.33%. Their differences in the prevalence rate of *Giardia lamblia* infection was not significant ($p > 0.05$).

Entamoeba histolytica infection was not statistically significant ($p > 0.05$) among the studied children with respect to all the risk factors considered in Ayigya community as shown in Table 4.7.

Table 4.7 Proportion and Association of *Giardia lamblia* and *Entamoeba histolytica* infections in Ayigya.

	Giardia lamblia				*Entamoeba histolytica*		
				Odd Ratio			
Variable	N	**n (%)**	***P***	**(95% CI)**	**n(%)**	***P***	**(95% CI)**
Gender							
Male	185	30(16.22)	0.05	1.48(0.52-2.20)	1(0.54)	-	-
Female	215	15(6.98)	-	1	-	-	-
Age							
5-6yrs.	62	21(33.87)	0.03	2.14(1.20-4.66)	1(2.38)	-	-
7-8yrs	85	10(11.76)	0.12	1.25(0.78-2.77)	-	-	-
9-10yrs	100	8(8.00)	0.30	0.75(0.46-1.88)	-	-	-
11-12yrs	153	6(3.92)	-	1	-	-	-
Household Water Source							
Pipe-borne water	298	30(10.07)	0.23	0.95(0.45-2.50)	-	-	-
Borehole/Well	102	15(14.71)	-	1	1(0.98)	-	-
River/stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	30	8(26.67)	0.06	1.87(0.97-3.14)	1(3.33)	-	
W.C	45	5(11.11)	0.20	0.72(0.35-1.73)	-	-	-
Pit latrine	85	13(15.29)	0.19	0.92(0.54-2.34)	-	-	-
KVIP	240	19(7.92)	-	1	-	-	-
Hand washing after Defecation							
Yes	185	15(8.11)	0.02	0.72(0.32-2.86)	-	-	-
No	95	11(11.58)	0.34	1.47(0.44-3.23)	1(1.05)	-	-
Sometimes	120	19(15.83)	-	1	-	-	-
With what							
Soap and Water	96	10(10.42)	0.48	0.68(0.42-1.88)			
Water Only	209	24(11.48)	-	1	1(0.48)	-	-
Hand washing before eating							
Yes	209	15(7.18)	0.22	0.59(0.21-1.96)	-		
No	30	10(33.33)	0.02	2.62(1.06-4.77)	1(20.0)	-	-
Sometimes	161	20(12.42)	-	1	-	-	-
With what							
Soap and Water	100	10(10.00)	0.32	0.65(0.14-2.00)	-	-	-
Water Only	270	25(9.26)	-	1	1(0.37)	-	-

Purpose for Washing							
Known	230	17(7.39)	0.21	0.76(0.23-3.12)	-	-	-
Unknown	162	28(17.28)	-	1	1(0.62)	-	-
Source of Food							
From the House	150	13(17.33)	0.17	1.72(0.54-3.28)	1(0.67)	-	-
Around the school	250	22(17.6)	-	1	-	-	-
Parent's occupation							
Farmer	15	9(60.00)	0.001	6.25(2.22-10.7)	1(6.67)	-	-
Civil servant	100	15(15.00)	0.21	1.45(0.65-2.66)	-	-	-
Trader	130	10(7.69)	0.44	0.72(0.53-1.96)	-	-	-
carpenter	10	1(10.00)	0.29	1.15(0.62-2.74)	-	-	-
unemployed	30	5(16.67)	0.09	1.73(0.70-2.78)	-	-	-
businessman	115	5(4.35)	-	1	-	-	-

4.8 Proportion and Association of *Cryptosporidium parvum* and Hookworm infection in Ayigya.

From table 4.8, *Cryptosporidium parvum* infection was higher in males (10.81%) than females (4.65%), but the difference in the rate of prevalence was statistically significant ($p < 0.05$). *Cryptosporidium parvum* infection was statistically significant ($p < 0.05$) among 5-6years age group (24.17%, p = 0.04) than 7-8years age group (12.94%, p = 0.15), 9-10years age group (3.0%, p = 0.31) and 11-12 years age group (0.65%). There was no significant difference (p > 0.05) in the rate of *Cryptosporidium parvum* infection among children who depend on borehole (6.86%) as their source of drinking water than those who use pipe-borne water (7.72%). *Cryptosporidium parvum* infection was not statistically significant ($p > 0.05$) among children who depend on bushes or open space, water closet, pit latrine and KVIP as their sanitary source in Ayigya, but the infection was high among those who defecate in bushes or fields (16.67%). *Cryptosporidium parvum* infection was significant ($p < 0.05$)

among children who do not practice personal hygiene (those who do not wash their hand before eating and after defecation with soap and water). Children who buy food from food vendors around their school compound had a prevalence of 8.52% and those who bring their food from their various homes had a prevalence of 3.48%. *Cryptosporidium parvum* infection was not statistically significant ($p < 0.05$) among children whose parents were farmers (Table 4.8).

From table 4.8, hookworm infection was higher in males (2.16%) than females (0.47%). The difference in the prevalence rate of hookworm infection among the males and females were statistically significant ($p < 0.05$). Hookworm infection was not statistically significant ($p > 0.05$) among the age group studied (5-6years, 7-8 years, 9-10years and 11-12 years). The prevalence rate of hookworm infection was high among children who lack sanitary facility (bushes or field) (10.0%) as compared to those who relied on pit latrine (1.18%) and KVIP (0.42%). The difference in the prevalence rates were statistically significant ($p < 0.05$). Hookworm infection was statistically significant ($p < 0.05$) among children whose parents were farmers. Hookworm infection among children who sometimes wear footwear were high (Table 4.8).

Table 4.8 Proportion and Association of *Cryptosporidium parvum* and Hookworm infection in Ayigya.

	Cryptosporidium parvum				Hookworm		
				Odd Ratio			Odd Ratio
Variable	**N**	**n(%)**	***P***	**(95% CI)**	**n(%)**	***P***	**(95% CI)**
Gender							
Male	185	20(10.81)	0.23	1.89(0.28-4.21)	4(2.16)	0.33	0.45(0.17-1.22)
Female	215	10(4.65)	-	1	1(0.47)	-	1
Age							
5-6yrs.	62	15(24.19)	0.04	1.85(0.92-4.10)	-	-	-
7-8yrs	85	11(12.94)	0.15	0.69(0.22-1.92)	2(2.35)	0.36	0.67(0.26-1.93)
9-10yrs	100	3(3.00)	0.31	0.42(0.18-2.26)	1(1.00)	0.40	0.42(0.25-2.00)
11-12yrs	153	1(0.65)	-	1	2(1.31)	-	1
Household Water Source							
Pipe-borne water	298	23(7.72)	0.21	0.55(0.14-1.68)	-	-	-
Borehole/Well	102	7(6.86)	-	1	-	-	-
River/stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	30	5(16.67))	0.12	1.54(0.32-2.70)	3(10.00)	0.01	1.24(0.77-3.56)
W.C	45	4(8.89)	0.30	0.65(0.28-1.79)		-	-
Pit latrine	85	7(8.24)	0.40	0.51(0.14-1.68)	1(1.18)	0.44	0.52(0.27-1.88)
KVIP	240	14(5.83)	-	1	1(0.42)	-	1
Hand washing after Defecation							
Yes	185	11(5.95)	0.25	0.62(0.40-2.20)	-	-	-
No	95	6(6.32)	0.19	0.74(0.36-2.59)	-	-	-
Sometimes	120	13(10.83)	-	-	-	-	-
With what							
Soap and Water	96	7(7.29)	0.22	0.75(0.45-3.60)	-	-	-
Water Only	209	23(11.00)	-	1	-	-	-
Hand washing before eating							
Yes	209	9(4.31)	0.42	1.10(0.63-2.52)	-	-	
No	30	5(16.67)	0.04	1.46(0.96-3.41)	-	-	-
Sometimes	161	16(9.94)	-	1	-	-	-
With what							
Soap and Water	100	7(7.00)	0.40	0.62(0.44-2.47)	-	-	-
Water Only	270	23(8.52)	-		-	-	-

Purpose for Washing							
Known	230	8(3.48)	0.18	0.50(0.26-1.76)	-	-	-
Unknown	162	22(13.58)	-	1	-	-	1
Source of Food							
From the House	150	13(8.67)	0.22	1.33(0.54-3.23)	-	-	-
Around the school	250	17(6.80)	-	1	-	-	1
Parent's occupation							
Farmer	15	6(40.00)	0.02	1.66(0.45-2.85)	3(20.00)	0.03	1.98(1.20-4.62)
Civil servant	100	11(11.00)	0.20	0.70(0.28-1.89)	-	-	
Trader	130	8(6.15)	0.61	0.42(0.21-1.55)	1(0.77)	0.22	0.68(0.46-2.25)
carpenter	10	1(10.00)	0.31	0.62(0.15-2.63)	-	-	-
unemployed	30	3(10.00)	0.32	0.63(0.15-2.64)	1(3.33)	-	1
businessman	115	1(0.87)	-	1	-		
Foot wear at home and school							
Never	-	-	-	-	-		
Sometimes	25	-	-	-	4(16.00)	0.04	1.75(1.00-4.45)
Usually	375	-	-	-	1(0.27)	-	1

4.9 Proportion and Association of *Ascaris lumbricoides* and *Strongyloides stercolaris* infection in Ayigya.

The proportion and association of *Ascaris lumbricoides* and *Strongyloides stercolaris* infection are shown on table 4.9. From table 4.9, *Ascaris lumbricoides* infection was not statistically significant ($p > 0.05$) among males and females, but males recorded higher prevalence (3.78%) than females (1.39%). *Ascaris lumbricoides* infection was significant ($p > 0.05$) among the various age groups but was highest among children in 5-6years age group (8.04%), followed by 7-8years age group (2.35%), 9-10 years age group (2.0%) and 11-12years age group (0.65%). Children who relied on pipe-borne water as their source of drinking water had low prevalence of 1.01% *Ascaris lumbricoides* infection compared to those who relied on borehole (6.86%) (Table 4.9). *Ascaris lumbricoides* infection was

statistically significant ($p < 0.05$) among children who relied on bushes or field (16.67%) as their sanitary facilities as compared with those who relied on pit latrine (1.18%) and KVIP (1.60%) in Ayigya. *Ascaris lumbricoides* infection was statistically significant ($p < 0.05$) among children who do not wash their hands before and after eating and defecation respectively. Children who had no knowledge of personal hygiene practices had higher prevalence (4.94%) than those who had knowledge of personal hygiene practice (0.87%). The difference in the prevalence rate of *Ascaris lumbricoides* infection was statistically significant ($p < 0.05$) among children whose parents were farmers.

Strongyloides stercolaris infection was not statistically significant ($p > 0.05$) among the studied children with respect to all the risk factors analysed. Only the males (1.08%) had *S. stercolaris* infection in Ayigya. Children of the age group 5-8years and 7-8years had a prevalence rate of 1.61% and 1.18% *S. stercolaris* infection respectively (Table 4.9). Only children who relied on borehole as their source of drinking water had prevalence of 1.96%, and those who relied on bushes or open space as their sanitary facility had a prevalence rate of 6.67%. Children whose parents were farmers had a prevalence of 6.67% and those whose parents were unemployed had a prevalence of 3.33%.

Table 4.9 Proportion and Association of *Ascaris lumbricoides* and *Strongyloides stercolaris* infection in Ayigya.

	Ascaris lumbricoides				*Strongyloides stercolaris*		
				Odd Ratio			**Odd Ratio**
Variable	**N**	**n(%)**	***P***	**(95% CI)**	**n(%)**	***P***	**(95% CI)**
Gender							
Male	185	7(3.78)	0.42	1.20(0.72-3.10)	2(1.08)	-	-
Female	215	3(1.39)	-	1	-	-	-
Age							
5-6yrs.	62	5(8.04)	0.23	1.54(0.52-4.20)	1(1.61)	0.60	0.69(0.25-2.42)
7-8yrs	85	2(2.35)	0.36	1.20(0.36-2.77)	1(1.18)	-	1
9-10yrs	100	2(2.00)	0.33	0.87(0.42-2.18)	-	-	-
11-12yrs	153	1(0.65)	-	1	-	-	-
Household Water Source							
Pipe-borne water	298	3(1.01)	0.50	0.45(0.21-1.56)	-	-	-
Borehole/Well	102	7(6.86)	-	1	2(1.96)	-	-
River/stream	-			-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	30	5(16.67)	0.04	2.44(1.00-5.68)	2(6.67)	-	-
W.C	45	-	-	-	-	-	
Pit latrine	85	1(1.18)	0.45	0.82(0.44-2.60)	-	-	-
KVIP	240	4(1.60)	-	1	-	-	-
Hand washing after Defecation							
Yes	185	1(0.54)	0.70	0.42(0.12-1.58)	-	-	-
No	95	6(6.32)	0.19	1.00(0.58-2.66)	2(2.11)	-	-
Sometimes	120	3(2.50)	-	1	-	-	-
With what							
Soap and Water	96	-	-	-	-	-	-
Water Only	209	4(1.91)	-	-	-	-	-
Hand washing before eating							
Yes	209	1(0.48)	0.72	1.03(0.63-2.95)	-	-	-
No	30	6(20.00)	0.03	3.21(1.6-6.78)	1(3.33)	0.48	1.00(0.45-2.42)
Sometimes	161	3(1.86)	-	1	1(0.62)	-	1
With what							
Soap and Water	100	-	-	-	-	-	-
Water Only	270	4(1.48)	-	-	1(0.37)	-	-

Purpose for Washing							
Known	230	2(0.87)	0.63	0.82(0.47-1.88)	-	-	-
Unknown	162	8(4.94)	-	1	2(1.23)	-	-
Source of Food							
From the House	150	3(2.00)	0.42	0.78(0.45-2.11)	-	-	-
Around the school	250	7(2.80)	-	1	2(0.80)	-	-
Parent's occupation							
Farmer	15	5(33.33)	0.01	3.22(1.54-6.77)	1(6.67)	0.32	1.77(0.92-3.26)
Civil servant	100	1(1.00)	0.58	1.22(0.62-2.98)	-	-	-
Trader	130	2(1.54)	0.44	1.36(1.10-2.25)	-	-	-
Carpenter	10	-	-	-	-	-	-
Unemployed	30	2(6.67)	-	1	1(3.33)	-	1
Businessman	115	-	-	-	-	-	-

4.10 Proportion and Association of *Giardia lamblia* and *Entamoeba histolytica/dispar* in Kentinkrono.

From table 4.10, males had high prevalence of *Giardia lamblia* infection (29.07%) than female (9.65%) in Kentinkrono community. The difference in the prevalence rates was statistically significant ($p < 0.05$). *Giardia lamblia* infection was statistically significant ($p < 0.05$) among 5-6 years and 7-8years age groups than 9-10 years and 11-12years age groups. *Giardia lamblia* infection was high among children who relied on borehole (30.0%) as their source of drinking water than those who relied on pipe-borne water (10.80%). The difference in the prevalence rates was statistically significant ($p < 0.05$). *Giardia lamblia* infection was statistically significant ($p < 0.05$) with a prevalence of 41.29% among children who relied on bushes or fields as their sanitary facilities than those who relied on water closet (14.29%), pit latrine (16.83%) and KVIP (12.50%) as their sanitary facilities. *Giardia lamblia* infection was statistically significant ($p < 0.05\%$) among children who do not wash their hands before

and after defecation respectively (Table 4.10). Children who buy food from food vendors around their school compound had higher prevalence of 20.0% than those who bring food from their various house (13.33%). *Giardia lamblia* infection was statistically significant ($p < 0.05$) among children whose parents were farmers and unemployed.

From table 4.10, *Entamoeba histolytica* infection was not statistically significant ($p > 0.05$) among the studied children with respect to all the risk factors analysed in Kentinkrono community. *Entamoeba histolytica* infection was higher in males (1.16%) than females (0.44%). Only 5-6 years age group (2.56%) and 7-8years age group (1.56%) had *Entamoeba histolytica* infection in Kentinkrono community.

Table 4.10 Proportion and Association of *Giardia lamblia* and *Entamoeba histolytica/dispar* in Kentinkrono.

	Giardia lamblia				*Entamoeba histolytica/dispar*		
				Odd Ratio			**Odd Ratio**
	N	**n(%)**	***P***	**(95%CI)**	**n(%)**	***P***	**(95%CI)**
Variable							
Gender							
Male	172	50(29.07)	0.05	2.4(0.95-4.16)	2(1.16)	0.40	0.56(0.31-1.84)
Female	228	22(9.65)	-	1	1(0.44)	-	1
Age							
5-6yrs.	78	42(53.85)	0.001	10.77(3.78-19.6)	2(2.56)	0.32	0.68(0.19-2.53)
7-8yrs	64	16(25.00)	0.01	4.22(1.56-10.22)	1(1.56)	-	1
9-10yrs	90	10(11.11)	0.23	1.85(0.85-3.82)	-	-	-
11-12yrs	168	4(2.38)	-	1	-	-	-
Household Water Source							
Pipe-borne water	250	27(10.80)	0.30	0.88(0.52-2.23)	1(0.56)	0.51	0.45(0.27-1.67)
Borehole/Well	150	45(30.00)	-	1	2(1.33)	-	1
River/stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	60	25(41.67)	0.01	4.89(2.12-10.25)	1(1.67)	0.46	0.72(0.41-1.69)
W.C	35	5(14.29)	0.19	1.88(0.73-4.66)	-	-	-
Pit latrine	101	17(16.83)	0.13	2.10(0.95-5.52))	-	-	-
KVIP	200	25(12.50)	-	1	2(1.00)	-	1
Hand washing after Defecation							
Yes	150	18(12.00)	0.22	1.26(0.52-2.85)	-	-	-
No	86	32(37.21)	0.03	2.85(1.20-6.46)	2(2.33)	0.33	1.36(0.53-2.77)
Sometimes	164	22(13.41)	-	1	1(0.61)	-	-
With what						-	-
Soap and Water	87	10(11.49)	0.42	1.32(0.43-2.46)	-	-	-
Water Only	227	30(13.22)	-	1	1(0.44)	-	-
Hand washing before eating							
Yes	185	21(11.35)	0.24	0.78(0.52-2.86)	1(0.54)	0.60	0.48(0.14-2.36)
No	52	25(48.08)	0.001	8.66(3.56-16.65)	1(1.92)	0.43	0.69(0.44-2.12)
Sometimes	163	26(15.95)	-	1	1(0.61)	-	-
With what							
Soap and Water	100	12(12.00)	0.19	1.62(0.41-3.20)	-	-	-
Water Only	248	35(14.11)	-	1	2(0.81)	-	-

Purpose for Washing							
Known	225	19(8.44)	0.42	0.74(0.35-1.98)	1(0.44)	0.44	0.34(0.10-1.37)
Unknown	175	53(30.29)	-	1	2(1.14)	-	1
Source of Food							
From the House	120	16(13.33)	0.14	1.55(0.66-2.56)	-	-	-
Around the school Compound	280	56(20.00)	-	1	3(1.07)	-	-
Parent's occupation							
Farmer	45	20(44.44)	0.001	10.62(4.17-21.1)	1(2.22)	0.35	0.74(0.19-1.78)
Civil servant	98	12(12.24)	0.22	0.94(0.34-2.10)	-	-	-
Trader	130	21(16.15)	0.09	1.89(0.42-3.66)	2(1.54)	-	1
Carpenter	15	2(13.33)	0.19	1.20(0.50-3.26)	-	-	-
Unemployed	50	15(30.00)	0.01	4.45(1.64-10.84)	-	-	-
Businessmen	62	2(3.23)	-	1	-	-	-

4.11 Proportion and Association of *Cryptosporidium parvum* and Hookworm in Kentinkrono

C. parvum infection was high among males than females with prevalence rate of 18.60% and 7.89% respectively (Table 4.11). The difference in the prevalence rates was significant ($p < 0.05$). *C. parvum* infection was statistically significant among younger age groups (5-6 years and 7-8 years age groups) than the older age group (9-10years and 11-12 years age groups). *C. parvum* infection was high among children who relied on borehole (23.33%) as their source of drinking water than those who relied on pipe-borne water (6.00%). The difference in the prevalence rates was significant ($p < 0.05$). *C. parvum* infection was statistically significant ($p < 0.05$) among children who do not practise proper personal hygiene (those who do not wash their hand before and after eating and defecation respectively). There was

no significant difference in the prevalence rates of *C. parvum* infection among children who buy food from food vendors around the school compound (12.50%) and those who bring food from their various homes (12.50%). *C. parvum* infection was statistically significant ($p < 0.05$) among children whose parents were farmers.

From table 4.11, Hookworm infection was high in males (6.98%) than females (1.32%) in Kentinkrono. Children in the age group of 7-8years had the highest prevalence of 4.49%, followed by 11-12 years age group (3.57%), 5-6 years age group (3.85%) and 9-10years age group (3.33%). Children who relied on bushes or open space as their sanitary facilities had the highest prevalence of 13.33%, followed by those who use KVIP (2.50%) and those who relied on pit latrine (1.98%). Children who sometimes wear footwear had prevalence of 30.0% as compared with those who wear foot ware usually (1.62%). Hookworm infection was statistically significant ($p < 0.05$) among children whose parents were farmers (Table 4.11).

Table 4.11 Proportion and Association of *Cryptosporidium parvum* and Hookworm in Kentinkrono.

	Cryptosporidium parvum				Hookworm		
				Odd Ratio			Odd Ratio
Variable	**N**	**n(%)**	***P***	**(95% CI)**	**n(%)**	***P***	**(95% CI)**
Gender							
Male	172	32(18.60)	0.28	1.55(0.53-2.77)	12(6.98)	0.11	1.54(0.42-4.52)
Female	228	18(7.89)	-	1	3(1.32)	-	1
Age							
5-6yrs.	78	24(30.77)	0.01	3.58(1.10-6.85)	3(3.85)	0.22	0.82(0.32-2.65)
7-8yrs	64	14(21.88)	0.04	2.22(0.78-4.62)	3(4.46)	0.19	1.00(0.51-2.78)
9-10yrs	90	8(8.89)	0.42	0.86(0.33-2.54)	3(3.33)	0.25	0.54(0.33-2.10)
11-12yrs	168	4(2.38)	-	1	6(3.57)	-	1
Household Water Source							
Pipe-borne water	250	15(6.00)	0.19	0.77(0.46-3.25)	-	-	-
Borehole/ Well	150	35(23.33)	-	1	-	-	-
River/ Stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	60	14(23.33)	0.04	2.22(1.40-4.56)	8(13.33)	0.03	1.75(0.52-3.44)
W.C	35	2(5.71)	0.42	0.62(0.22-1.99)	-	-	-
Pit latrine	101	10(9.90)	0.30	1.00(0.54-4.12)	2(1.98)	0.38	0.58(0.11-2.65)
KVIP	200	24(12.00)	-	1	5(2.50)	-	1
Hand washing after Defecation							
Yes	150	10(6.67)	0.21	1.50(0.41-2.42)	-	-	-
No	86	25(29.07)	0.01	7.23(2.14-15.62)	-	-	-
Sometimes	164	15(9.15)	-	1	-	-	-
With what							
Soap and Water	87	8(9.19)	0.32	1.02(0.33-2.75)	-	-	-
Water Only	227	17(7.49)	-	1	-	-	-
Hand washing before eating							
Yes	185	21(11.35)	0.28	0.69(0.41-2.45)	-	-	-
No	52	9(17.31)	0.19	1.22(0.75-4.20)	-	-	-
Sometimes	163	20(12.27)	-	1			
With what							
Soap and	100	10(10.00)	0.21	1.32(0.39-2.86)	-	-	-

Water							
Water Only	248	31(12.50)	-	1	-	-	
Purpose for Washing							
Known	225	15(6.67)	0.16	0.87(0.43-2.55)	-	-	-
Unknown	175	35(20.00)	-	1	-	-	-
Source of Food							
From the House	120	15(12.50)	0.20	1.33(0.66-2.17)	-	-	-
Around the school Compound	280	35(12.50)	-	1	-	-	-
Footwear use							
Usually	370	-	-	-	6(1.62)	0.24	1.62(0.45-2.23)
Sometimes	30	-	-	-	9(30.00)	-	1
Never	-		-	-	-	-	-
Parent's occupation							
Farmer	45	14(31.11)	0.02	2.66(1.51-6.78)	10(22.22)	0.02	2.66(1.00-6.20)
Civil servant	98	15(15.31)	0.15	1.54(0.72-3.77)	-	-	-
Trader	130	10(7.69)	0.42	0.93(0.61-3.52)	2(1.54)	0.52	0.62(0.25-2.39)
Carpenter	15	1(6.67)	0.44	0.65(0.22-1.88)	-	-	-
Unemployed	50	5(10.00)	0.25	1.23(0.55-2.63)	3(6.00)	-	1
Businessmen	62	5(8.06)	-	1	-	-	-

4.12 Proportion and Association of *Ascaris lumbricoides* and *Strongyloides stercolaris* infection in Kentinkrono.

The prevalence of *A. lumbricoides* infection was high in males (13.37%) than females (3.07(Table 4.12). The prevalence rate of *A. lumbricoides* infection was high among children of 5-6 years age group (16.67%) followed by 7-8 years age group (14.06%), 9-10 years (5.55%) and 11-12years age group (1.79%). From table 4.7.6, *A. lumbricoides* infection was high among children who relied on borehole (15.55%) as their source of drinking water than those who relied on pipe-borne water (2.80%). The difference in the prevalence rates was statistically significant ($p < 0.05$). *A. lumbricoides* infection was statistically significant ($p <$

0.05) among children who relied on bushes or open space as their sanitary source as compared to those who relied on water closet, pit latrine and KVIP as their sanitary facilities. *A. lumbricoides* infection was statistically significant ($p < 0.05$) among children who do not wash their hand before eating and after defecation. Children who buy food from food vendors around their school compound had a prevalence of 8.93% *A. lumbricoides* infection and those who bring food from their various homes had a prevalence of 4.17%. *A. lumbricoides* infection was statistically significant ($p < 0.05$) among children whose parents were farmers.

Strongyloides stercolaris infection was not statistically significant ($p > 0.05$) among the studied children with respect to all the risk factors considered in Kentinkrono community as shown in table 4.12

Table 4.12 Proportion and Association of *Ascaris lumbricoides* and *Strongyloides stercolaris* infection in Kentinkrono.

	Ascaris lumbricoides				*Strongyloides stercolaris*		
				Odd Ratio			**Odd Ratio**
Variable	**N**	**n(%)**	***P***	**(95% CI)**	**n(%)**	***P***	**(95% CI)**
Gender							
Male	172	23(13.37)	0.10	1.20(0.62-3.20)	7(4.07)	0.20	0.58(0.24-2.00)
Female	228	7(3.07)	-	1	2(0.88)	-	1
Age							
5-6yrs.	78	13(16.67)	0.05	1.56(0.78-4.32)	5(6.41)	0.11	1.00(0.23-1.99)
7-8yrs	64	9(14.06)	0.16	1.10(0.44-2.70)	2(3.13)	0.28	0.52(0.12-1.73)
9-10yrs	90	5(5.55)	0.32	0.65(0.23-1.68)	1(1.11)	0.33	0.35(0.08-1.55)
11-12yrs	168	3(1.79)	-	1	1(0.59)	-	1
Household Water Source							
Pipe-borne water	250	7(2.80)	0.33	0.77(0.25-1.84)	2(0.80)	0.45	0.54(0.11-2.30)
Borehole/Well	150	23(15.33)	-	1	7(4.67)	-	1
River/stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	60	10(16.67)	0.04	2.10(0.87-4.52)	3(5.00)	0.30	0.95(0.42-2.40)
W.C	35	1(2.86)	0.40	1.0(0.21-1.99)	-	-	-
Pit latrine	101	7(6.93)	0.23	1.43(0.82-3.40)	2(1.98)	0.38	0.44(0.20-2.48)
KVIP	200	12(6.00)	-	1	4(2.00)	-	1
Hand washing after Defecation							
Yes	150	2(1.33)	0.44	0.43(0.22-1.58)	-	-	-
No	86	15(17.44)	0.10	1.15(0.68-3.00)	5(5.81)	0.11	1.02(0.43-2.50)
Sometimes	164	13(7.93)	-	1	4(2.44)	0.25	1
With what							
Soap and Water	87	2(2.29)	0.28	0.56(0.10-1.88)	-	-	-
Water Only	227	13(5.73)	-	1	4(1.76)	-	-
Hand washing before eating							

Yes	185	1(0.54)	0.53	0.84(0.44-2.22)	-	-	-
No	52	20(38.46)	0.01	4.33(1.42-10.52)	7(13.46)	0.04	1.24(0.57-2.56)
Sometimes	163	9(5.52)	-	1	2(1.23)	-	1
With what							
Soap and Water	100	-	-	-	-	-	-
Water Only	248	10(4.03)	-	-	2(0.81)	-	-
Purpose for Washing							
Known	225	5(2.22)	0.30	0.75(0.32-2.45)	2(0.89)	0.50	0.47(0.13-1.00)
Unknown	175	25(14.29)	-	1	7(4.00)	-	1
Source of Food							
From the House	120	5(4.17)	0.24	1.33(0.45-3.10)	1(0.83)	0.36	0.49(0.0.33-2.10)
Around the school	280	25(8.93)	-	1	8(2.86)	-	1
Parent's occupation							
Farmer	45	12(23.37)	0.02	3.25(1.18-8.44)	4(8.89)	0.19	1.53(0.72-4.36)
Civil servant	98	1(1.02)	0.46	0.54(0.22-1.84)	-	-	-
Trader	130	7(5.38)	0.22	1.00(0.56-3.85)	2(1.54)	0.38	0.84(0.42-2.56)
Carpenter	15	-	-	-	-	-	-
unemployed	50	8(16.00)	0.12	1.76(0.87-4.79)	2(4.00)	0.15	1.10(0.77-4.20)
businessman	62	2(3.33)	-	1	1(1.61)	-	1

4.13 Proportion and Association of *Giardia lamblia* and *Entamoeba histolytica/dispar* in Aboabo.

The prevalence of *Giardia lamblia* infection was high among males (13.11%) than females (5.15%) in Aboabo community (Table 4.13). *Giardia lamblia* infection was statistically significant ($p < 0.05$) among the 5-6 years age group as compared with 7-8 years, 9-10years and 11-12 years age groups (Table 4.13). *Giardia lamblia* infection was high among children who relied on borehole (17.0%) as their source of drinking water than those who relied on pipe-borne water (5.0%). The difference in the prevalence rates was statistically significant ($p < 0.05$). From table 4.13, the prevalence rate of *G. lamblia* infection was statistically significant ($p < 0.05$) among children who relied on bushes or open space (40.0%) as their sanitary source compared with those who relied on water closet (4.0%), pit latrine (9.47%) and KVIP (6.96%). *G. lamblia* infection was statistically significant ($p < 0.05$) among children who do not wash their hand before and after eating and defecation respectively. Children who buy food from food vendors around their school compound had a prevalence rate of 10.99% compared with those who bring food from their various homes (5.08%). *G. lamblia* infection was statistically significant ($p < 0.05$) among children whose parents were farmers and unemployed (Table 4.13).

From table 4.13, *Entamoeba histolytica* infection was not statistically significant ($p > 0.05$) among the studied children with respect to all the risk factors considered in Ayigya community.

Table 4.13 Proportion and Association of *Giardia lamblia* and *Entamoeba histolytica/dispar* in Aboabo.

	Giardia lamblia				*Entamoeba histolytica/dispar*		
				Odd Ratio			**Odd Ratio**
Variable	**N**	**n(%)**	***P***	**(95%CI)**	**n(%)**	***P***	**(95% CI)**
Gender							
Male	206	27(13.11)	0.19	1.66(0.52-4.21)	1(0.49)	-	-
Female	194	10(5.15)	-	1	-	-	-
Age							
5-6yrs.	68	14(20.59)	0.03	2.53(1.3-6.65)	-	-	-
7-8yrs	75	12(16.00)	0.15	1.56(0.55-3.75)	-	-	-
9-10yrs	88	8(9.09)	0.30	0.72(0.32-2.99)	1(1.14)	-	-
11-12yrs	169	3(1.78)	-	1		-	-
Household Water Source							
Pipe-borne water	300	15(5.00)	0.41	0.68(0.25-2.62)	-	-	-
Borehole/Well	100	17(17.00)	-	1	1(1.00)	-	-
River/stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	25	10(40.00)	0.001	10.5(3.14-21.7)	1(4.00)	-	-
W.C	50	2(4.00)	0.26	0.36(0.01-1.88)	-	-	-
Pit latrine	95	9(9.47)	0.20	1.42(0.59-4.24)	-	-	-
KVIP	230	16(6.96)	-	1	-	-	-
Hand washing after Defecation							
Yes	175	9(5.14)	0.19	0.52(0.16-2.20)	-	-	-
No	65	12(18.46)	0.04	2.59(1.23-6.52)	1(1.54)	-	-
Sometimes	160	16(10.00)	-	1	-	-	-
With what							
Soap and Water	106	4(3.77)	0.22	0.57(0.26-2.76)	-	-	-
Water Only	229	21(9.17)	-	1	1(0.44)	-	-
Hand washing before eating							
Yes	200	5(2.50)	0.25	0.86(0.41-3.66)	-	-	-
No	35	10(28.57)	0.04	2.11(0.68-4.75)	1(2.58)	-	-
Sometimes	165	22(13.33)	-	1	-	-	-
With what							
Soap and Water	100	4(4.00)	0.32	0.52(0.25-2.12)	-	-	-

Water Only	265	23(8.67)	-	1	1(0.38)	-	-
Purpose for Washing							
Known	265	10(3.77)	0.20	0.62(0.33-2.49)	-	-	-
Unknown	135	27(20.00)	-	1	1(0.74)	-	-
Source of Food							
From the House	118	6(5.08)	0.44	1.22(0.48-2.65)	-	-	-
Around the school Compound	282	31(10.99)	-	1	1(0.35)	-	-
Parent's occupation							
Farmer	15	8(53.33)	0.001	10.5(3.66-20.2)	-	-	-
Civil servant	82	7(8.54)	0.25	1.51(0.71-3.42)	-	-	-
Trader	120	10(8.33)	0.56	0.69(0.23-2.44)	1(0.83)	-	-
Carpenter	3	-	-	-	-	-	-
Unemployed	30	8(26.67)	0.01	4.15(1.76-6.85)	-	-	-
Businessmen	150	4(2.67)	-	1	-	-	-

4.14 Proportion and Association of *Cryptosporidium parvum* and Hookworm infection in Aboabo.

From table 4.14, males had high prevalence rate (6.79%) of *C. parvum* infection than females (5.67%). *C. parvum* infection was statistically significant ($p < 0.05$) among 5-6 years age group than 7-8 years, 9-10 years and 11-12 years age groups. Studied children who relied on borehole had a prevalence of 17.0% *C. parvum* infection compared with those who use pipe-borne water (2.67%) as their source of drinking water (Table 4.14). Studied children who relied on bushes or open space as their sanitary facility had a prevalence of 28.0% while those who relied on water closet had 2.0%, pit latrine had 4.20% and KVIP had 6.50% (Table 4.14). *C. parvum* infection was not significant ($p > 0.05$) among children who do not wash their hands before eating and after defecation (Table 4.7.8). Children who buy food from food vendors around their school compound had a prevalence of 6.74% *C. parvum* infection

while those who bring food from their various homes had a prevalence of 5.08%. *C. parvum* infection was statistically significant ($p < 0.05$) among children whose parents are farmers.

Hookworm infection was not significant ($p > 0.05$) among studied children with respect to the risk factors such as foot wear characteristics and sanitary facilities in Aboabo as shown in table 4.14.

Table 4.14 Proportion and Association of *Cryptosporidium parvum* and Hookworm infection in Aboabo.

	Cryptosporidium parvum				Hookworm		
				Odd Ratio			Odd Ratio
Variable	**N**	**n(%)**	***P***	**(95%CI)**	**n(%)**	***P***	**(95% CI)**
Gender							
Male	206	14(6.79)	0.28	1.26(0.7-4.82)	2(0.97)	-	-
Female	194	11(5.67)	-	1	-		
Age							
5-6yrs.	68	10(14.71)	0.02	1.89(1.23-4.85)	-	-	-
7-8yrs	75	7(9.33)	0.20	1.09(0.50-3.56)	-	-	-
9-10yrs	88	6(6.82)	0.28	0.65(0.22-3.82)	-	-	-
11-12yrs	169	2(1.18)	-	1	2(1.18)	-	-
Household Water Source							
Pipe-borne water	300	8(2.67)	0.44	0.58(0.35-2.44)	-	-	-
Borehole/ Well	100	17(17.00)	-	1	-	-	-
River/stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	25	7(28.00)	0.01	2.45(1.50-3.42)	1(4.00)	0.18	0.52(0.11-1.85)
W.C	50	1(2.00)	0.36	0.52(0.20-2.73)	-		
Pit latrine	95	4(4.20)	0.27	2.96(1.31-5.44)	-		
KVIP	230	15(6.50)	-	1	1(0.43)	-	1
Hand washing after Defecation							
Yes	175	4(2.29)	0.22	0.82(0.48-2.57)	-	-	-
No	65	10(15.38)	0.18	1.56(0.45-4.10)	-	-	-
Sometimes	160	11(6.88)	-	1	-	-	-
With what							
Soap and Water	106	2(1.89)	0.33	0.45(0.10-1.69)	-	-	-
Water Only	229	13(5.68)	-	1	-	-	-
Hand washing before eating							
Yes	200	5(3.00)	0.45	0.53(0.22-2.62)	-	-	-
No	35	6(17.14)	0.11	1.88(0.72-4.45)	-	-	-
Sometimes	165	14(8.48)	-	1	-	-	-

With what							
Soap and Water	100	3(3.00)	0.34	1.00(0.61-2.56)	-	-	-
Water Only	265	16(6.38)	-	1	-	-	-
Purpose for Washing							
Known	265	8(3.02)	0.28	0.57(0.36-3.66)	-	-	-
Unknown	135	19(14.07)	-	1	-	-	-
Source of Food							
From the House	118	6(5.08)	0.24	1.20(0.33-3.42)	-	-	-
Around the school Compound	282	19(6.74)	-	1	-	-	-
Parent's occupation							
Farmer	15	4(40.00)	0.01	2.50(0.42-3.40)	2(13.3)	-	-
Civil servant	82	8(13.79)	0.31	0.66(0.14-2.57)	-	-	-
Trader	120	5(8.06)	0.45	0.52(0.31-1.96)	-	-	-
Carpenter	3	-	-	-	-	-	-
unemployed	30	1(8.33)	0.42	0.45(0.09-1.5)	-	-	-
Businessmen	150	2(3.57)	-	1	-	-	-
Foot wear							
Usually	390	-	-	-	-	-	-
Sometimes	10	-	-	-	2(20.0)	-	-
Never	-	-	-	-	-	-	-

4.15 Proportion and Association of *Ascaris lumbricoides* and *Strongyloides stercolaris* infection in Aboabo.

A. lumbricoides infection was not significant ($p < 0.05$) among males and females. Studied children from Aboabo who relied on borehole as their source of drinking water had prevalence rate of 8.00% as compared to those who relied on pipe-borne water as their source of drinking water (0.67%). Children who lack sanitary facilities (bushes or open space) had prevalence rate of 20.0% *A. lumbricoides* infection as compared to those who relied on pit latrine (1.05%) and KVIP (1.74%) as their sanitary facilities. *A. lumbricoides* was not significant ($p < 0.05$) among children who do not wash their hands before eating and after defecation. Children who buy food from food vendors around their school compound had low

prevalence of 2.48% *A. lumbricoides* infection as compared to those who bring food from their various homes had a prevalence of 2.54 %. The difference in the prevalence rate was not significant. *C. parvum* infection was statistically significant ($p < 0.05$) among children whose parents are farmers (Table 4.15).

Strongyloides stercolaris infection was not statistically significant ($p > 0.05$) among the studied children with respect to all the risk factors considered in Kentinkrono community as shown on table 4.15.

Table 4.15 Proportion and Association of *Ascaris lumbricoides* and *Strongyloides stercolaris* infection in Aboabo.

	Ascaris lumbricoides				*Strongyloides stercolaris*		
				Odd Ratio			**Odd Ratio**
Variable	**N**	**n(%)**	***P***	**(95%CI)**	**n(%)**	***P***	**(95% CI)**
Gender							
Male	206	8(3.88)	0.18	0.59(0.25-2.11)	1(0.49)	0.47	0.53(0.18-2.02
Female	194	2(1.03)	-	1	1(0.52)	-	
Age							
5-6yrs.	68	4(5.88)	0.30	0.86(0.45-3.21)	1(1.47)	0.26	0.78(0.44-3.25)
7-8yrs	75	3(4.00)	0.38	0.52(0.22-2.45)	1(1.33)	-	1
9-10yrs	88	1(1.33)	0.45	0.41(0.10-1.76)	-	-	-
11-12yrs	169	2(1.18)	-	1	-	-	-
Household Water Source							
Pipe-borne water	300	2(0.67)	0.29	0.77(0.35-1.86)	-	-	-
Borehole/Well	100	8(8.00)	-	1	2(2.00)	-	-
River/stream	-				-	-	-
Household Sanitary Facility							
No facility (Bush/field)	25	5(20.0)	0.04	1.89(0.85-4.18)	1(4.00)	0.21	1.52(0.59-4.36)
W.C	50	-	-	-	-	-	-
Pit latrine	95	1(1.05)	0.24	0.65(0.24-1.75)	-	-	-
KVIP	230	4(1.74)	-	1	1(0.43)	-	1
Hand washing after Defecation							
Yes	175	-	-	-	-	-	
No	65	7(10.77)	0.15	1.00(0.45-2.55)	1(1.54)	0.39	0.58(0.32-2.57)
Sometimes	160	3(1.88)	-	1	1(0.63)	-	1
With what							
Soap and Water	106	-	-	-	-	-	-
Water Only	229	3(1.31)	-	-	1(0.44)	-	-
Hand washing before eating							
Yes	200	-	-	-	-	-	-
No	35	7(20.00)	0.05	1.82(1.00-4.84)	1(2.56)	0.20	1.03(0.59-2.88)

Sometimes	165	3(1.82)	-	1	1(0.61)	-	1
With what							
Soap and Water	100	-	-	-	-	-	-
Water Only	265	3(1.13)	-	-	1(0.38)	-	-
Purpose for Washing							
Known	265	2(0.75)	0.32	0.62(0.26-2.10)	-	-	-
Unknown	135	8(5.93)	-	1	2(1.48)	-	-
Source of Food							
From the House	118	3(2.54)	0.26	1.00(0.43-2.56)	-	-	-
Around the school Compound	282	7(2.48)	-	1	2(0.71)	-	-
Parent's occupation							
Farmer	15	5(33.33)	0.02	2.24(1.20-6.20)	1(6.67)	0.16	0.56(0.26-1.79)
Civil servant	82	-	-	-	-	-	-
Trader	120	3(2.50)	0.22	0.84(0.23-2.68)	-	-	-
Carpenter	3	-	-	-	-	-	-
unemployed	30	2(6.67)	-	1	1(3.33)	-	1
Businessmen	150	-	-	-	-	-	-

4.16 Proportion and Association of *Giardia lamblia* and *Cryptosporidium parvum* infection in Manhyia

The prevalence rates of *G. lamblia* and *C. parvum* infection were high among males (5.48% *G. lamblia*, 3.65% *C. parvum*) than females (4.42% *G. lamblia*, 4.41% *C. parvum*) in Manhyia (Table 4.16). *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among 5-6years age group than 7-8 years, 9-10 years and 11-12 years age groups (Table 4.7.11). Studied children from Manhyia who relied on borehole had high prevalence rates of 48% *G. lamblia* and 48% *C. parvum* infections as compared to those who relied on pipe-borne water as their source of drinking water (2.13% *G. lamblia*, 1.07% *C. parvum*). Studied children who relied on KVIP as their sanitary source had prevalence rates of 11.81% *G. lamblia* and 7.87% *C. parvum* while those who relied on water closet had prevalence rates of 1.03% *G. lamblia* and 0.51% *C. parvum* and Pit latrine had prevalence rates of 3.85% *G. lamblia* and 6.41% *C. parvum*. *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among studied children who do not wash their hands before eating and after defecation (Table 4.16). Children who buy food from food vendors around their school compound had prevalence rates of 5.36% *G. lamblia* and 5.0% *C. parvum* infections compared to those who bring food from their various homes had a prevalence rates of 4.17% *G. lamblia* and 1.67% *C. parvum* infections. *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among children whose parents were farmers and unemployed.

Table 4.16 Proportion and Association of *Giardia lamblia* and *Cryptosporidium parvum* in Manhyia.

	Giardia lamblia				*Cryptosporidium parvum*		
				Odd Ratio			**Odd Ratio**
Variable	**N**	**n(%)**	***P***	**(95% CI)**	**n(%)**	***P***	**(95% CI)**
Gender							
Male	219	12(5.48)	0.23	1.26(0.62-3.78)	8(3.65)	0.32	1.54(0.6-3.20)
Female	181	8(4.42)	-	1	8(4.41)	-	1
Age							
5-6yrs.	60	10(16.67)	0.02	2.47(1.0-6.44)	10(16.67)	0.04	3.12(1.58-6.56)
7-8yrs	110	4(3.64)	0.22	1.15(0.43-4.52)	3(2.73)	0.19	1.00(0.42-4.23)
9-10yrs	104	3(2.88)	0.20	0.66(0.12-2.09)	2(1.92)	0.26	0.58(0.06-2.22)
11-12yrs	126	1(0.79)	-	1	1(0.79)	-	1
Household Water Source							
Pipe-borne water	375	8(2.13)	0.18	0.55(0.2-1.76)	4(1.07)	0.21	0.90(0.45-2.54)
Borehole/Well	25	12(48.00)	-	1	12(48.0)	-	1
River/stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	-	-	-	-	-	-	-
W.C	195	2(1.03)	0.42	0.45(0.12-1.82)	1(0.51)	0.54	0.75(0.26-1.63)
Pit latrine	78	3(3.85)	0.20	1.0(0.38-3.50)	5(6.41)	0.24	1.25(0.38-2.56)
KVIP	127	15(11.81)	-	1	10(7.87)	-	1
Hand washing after Defecation							
Yes	300	4(1.33)	0.35	0.47(0.21-2.10)	4(1.33)	0.34	0.72(0.38-2.56)
No	14	8(57.14)	0.001	10.02(3.52-22.00)	4(28.57)	0.04	1.69(0.58-4.78)
Sometimes	86	6(6.98)	-	1	8(9.30)	-	1
With what							
Soap and Water	245	4(1.63)	0.46	0.52(0.18-1.66)	2(0.08)	0.66	0.42(0.11-1.62)
Water Only	141	8(5.67)	-	1	12(8.51)	-	1
Hand washing before eating							
Yes	310	8(2.58)	0.28	0.85(0.50-2.70)	8(2.58)	0.22	1.20(0.47-2.56)
No	-	-	-	-	-	-	-
Sometimes	90	12(13.33)	-	1	8(8.89)	-	1
With what							
Soap and Water	202	2(0.99)	0.49	0.35(0.05-1.28)	4(1.98)	0.29	0.63(0.26-2.23)

Water Only	198	18(9.09)	-	1	12(6.06)	-	1
Purpose for Washing							
Known	320	10(3.13)	0.28	0.66(0.19-2.00)	2(0.63)	0.53	0.77(0.26-2.88)
Unknown	80	10(12.50)	-	1	14(17.5)	-	1
Source of Food							
From the House	120	5(4.17)	0.41	1.25(0.52-4.23)	2(1.67)	0.39	0.53(0.11-1.86)
Around the school Compound	280	15(5.36)	-	1	14(5.00)	-	1
Parent's occupation							
Farmer	5	2(40.00)	0.01	3.89(1.88-8.45)	3(60.00)	0.001	8.55(4.22-16.89)
Civil servant	114	7(6.14)	0.25	0.92(0.40-2.23)	2(1.75)	0.72	0.45(0.20-2.10)
Trader	160	4(2.50)	0.32	0.52(0.12-1.89)	5(3.13)	0.49	0.58(0.44-1.86)
Carpenter	1	-	-	-	-	-	-
unemployed	20	2(10.00)	0.18	1.20(0.54-2.53)	4(20.00)	0.04	2.89(0.66-6.82)
Businessmen	100	5(5.00)	-	1	2(2.00)	-	1

4.17 Proportion and Association of *Ascaris lumbricoides* infection in Manhyia.

From table 4.17, *Ascaris lumbricoides* infection was not significant ($p < 0.05$) among males and females in Manhyia. The differences in the prevalence rates with respect to the risk factors considered were not significant ($p < 0.05$).

Table 4.17 Proportion and Association of *Ascaris lumbricoides* infection in Manhyia.

	Ascaris lumbricoides			
				Odd Ratio
Variable	**N**	**n(%)**	***P***	**(95% CI)**
Gender				
Male	219	1(0.46)	0.35	0.50(0.24-1.52)
Female	181	2(1.10)	-	1
Age				
5-6yrs.	60	1(1.67)	0.16	1.00(0.25-3.12)
7-8yrs	110	1(0.91)	0.24	0.85(0.43-2.45)
9-10yrs	104	1(0.96)	0.20	0.62(0.33-1.99)
11-12yrs	126	-	-	1
Household Water Source				
Pipe-borne water	375	-	-	-
Borehole/Well	25	3(12.00)	-	-
River/stream	-			
Household Sanitary Facility				
No facility (Bush/field)	-	-	-	-
W.C	195	-	-	-
Pit latrine	78	1(1.28)	0.38	0.65(0.22-2.18)
KVIP	127	2(1.57)	-	1
Hand washing after Defecation				
Yes	300	-	-	-
No	14	2(14.29)	0.18	1.26(0.77-4.25)
Sometimes	86	1(1.16)	-	1
With what				
Soap and Water	245	-	-	-
Water Only	141	1(0.71)	-	-
Hand washing before eating				
Yes	310	-	-	-
No	-	-	-	-
Sometimes	90	3(3.33)	-	-
With what				
Soap and Water	202	1(0.49)	0.36	0.39(0.11-1.50)
Water Only	198	2(1.01)	-	1
Purpose for				

Washing				
Known	320	1(0.31)	0.44	0.42(0.19-2.55)
Unknown	80	2(2.50)	-	1
Source of Food				
From the House	120	-	-	-
Around the school Compound	280	3(1.07)	-	-
Parent's occupation				
Farmer	5	1(20.00)	0.05	1.95(1.00-4.62)
Civil servant	114	-	-	-
Trader	160	1(0.63)	0.20	-
Carpenter	1	-	-	-
Unemployed	20	1(5.00)	0.15	1.23(0.85-5.00)
Businessmen	100	-	-	1

4.18 Proportion and Association of *Giardia lamblia* and *Cryptosporidium parvum* infection in Gyinyase

From table 4.18, the prevalence rates of *G. lamblia* and *C. parvum* infection were high among males (19.51% *G. lamblia*, 12.19% *C. parvum*) than females (11.28% *G. lamblia*, 6.67% *C. parvum*) in Manhyia. *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among 5-6years age and 7-8 years than 9-10 years and 11-12 years age groups (Table 4.18). Studied children from Manhyia who relied on borehole had high prevalence rates of 23.68% *G. lamblia* and 14.74% *C. parvum* infections compared to those who relied on pipe-borne water as their source of drinking water (8.09% *G. lamblia* and 4.79% *C. parvum*). Studied children who lack sanitary facilities (bushes or open space) had high prevalence rates of 36.36% *G. lamblia* and 27.27% *C. parvum* whiles those who relied on water closet had prevalence rates of 5.00% *G. lamblia* and 2.50% *C parvum*, those who relied on pit latrine had prevalence rates of 9.09% *G. lamblia* and 6.36% *C. parvum* and those who relied on KVIP had prevalence rates of 15.38% and 0.50% *C. parvum*. *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among studied children who do not wash their hands before eating and after defecation (Table 4.18). Children who buy food from food vendors around their school compound had high prevalence rates of 16.67% *G. lamblia* and 11.22% *C. parvum* infections as compared to those who bring food from their various homes had prevalence rates of 11.36% *G. lamblia* and 3.41% *C. parvum* infections. *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among children whose parents were farmers and unemployed.

Table 4.18 Proportion and Association of *Giardia lamblia* and *Cryptosporidium parvum* infection in Gyinyase.

	Giardia lamblia				*Cryptosporidium parvum*		
				Odd Ratio			**Odd Ratio**
Variable	**N**	**n (%)**	***P***	**(95% CI)**	**n(%)**	***P***	**(95%CI)**
Gender							
Male	205	40(19.51)	0.19	1.96(0.55-4.86)	25(12.19)	0.22	1.66(1.10-4.23)
Female	195	22(11.28)		1	13(6.67)	-	1
Age							
5-6yrs.	58	26(44.83)	0.001	8.92(4.25-16.77)	17(29.31)	0.01	3.22(1.55-6.80)
7-8yrs	100	20(20.00)	0.04	2.56(1.44-6.85)	8(8.00)	0.29	1.50(0.52-4.22)
9-10yrs	128	10(7.81)	0.20	1.00(0.27-4.33)	7(5.47)	0.33	0.89(0.54-5.26)
11-12yrs	114	6(5.26)	-	1	6(5.26)	-	1
Household Water							
Source							
Pipe-borne water	210	17(8.09)	0.21	1.23(0.62-3.20)	10(4.76)	0.20	1.06(0.23-3.14)
Borehole/Well	190	45(23.68)	-	1	28(14.74)	-	1
River/stream	-	-	-	-			
Household Sanitary Facility							
No facility (Bush/field)	55	20(36.36)	0.03	2.22(1.21-4.82)	15(27.27)	0.04	2.25(1.25-6.33)
W.C	40	2(5.00)	0.31	0.65(0.26-2.00)	1(2.50)	0.30	0.65(0.48-4.30)
Pit latrine	110	10(9.09)	0.20	1.3(0.32-3.41)	7(6.36)	0.19	1.42(0.85-3.23)
KVIP	195	30(15.38)	-	1	15(0.50)	-	1
Hand washing after							
Defecation							
Yes	190	10(5.26)	0.22	0.84(0.17-2.67)	8(4.21)	0.27	0.65(0.35-1.95)
No	110	40(36.36)	0.01	3.42(1.80-10.22)	12(10.91)	0.16	1.15(0.74-3.33)
Sometimes	100	12(12.00)	-	1	18(12.00)	-	1
With what							
Soap and Water	101	5(4.95)	0.35	1.25 (0.34-2.36)	3(2.97)	0.41	0.56(0.24-2.15)
Water Only	189	17(8.99)	-	1	23(12.17)	-	1
Hand washing before							
Eating							
Yes	186	20(10.75)	0.30	0.62(0.37-2.73)	5(2.68)	0.20	1.0(0.56-3.4)
No	39	20(51.28)	0.001	10.25(4.66-22.1)	15(38.45)	0.01	5.66(2.52-10.20)
Sometimes	175	22(12.57)	-	1	18(10.29)	-	1
With what							
Soap and Water	122	12(9.84)	0.24	1.22(0.52-3.58)	3(2.46)	0.19	0.52(0.24-1.82)
Water Only	239	30(12.55)	-	1	20(8.37)	-	1
Purpose for Washing							

Known	222	17(7.66)	0.17	1.26(0.66-4.50)	7(3.15)	0.33	0.57(0.18-2.76)
Unknown	178	45(25.28)	-	1	31(17.42)	-	1
Source of Food							
From the House	88	10(11.36)	0.11	1.56(0.69-4.54)	3(3.41)	0.42	0.72(0.41-1.96)
Around the school Compound	312	52(16.67)	-	1	35(11.22)	-	1
Parent's occupation							
Farmer	45	20(44.44)	0.001	7.44(3.25-16.80)	5(11.11)	0.10	1.02(0.54-2.79)
Civil servant	50	5(10.00)	0.13	1.69(0.52-4.28)	4(8.00)	0.22	0.85(0.42-2.56)
Trader	140	13(9.29)	0.20	1.23(0.58-2.03)	9(6.43)	0.35	0.65(0.31-2.17)
Carpenter	15	1(6.67)	0.34	0.75(0.45-2.44)	-	-	-
Unemployed	50	20(40.00)	0.01	5.22(2.14-12.66)	15(30.00)	0.04	2.10(0.77-4.10)
Businessmen	100	3(3.00)	-	1	5(5.00)	-	1

4.19 Proportion and Association of *Ascaris lumbricoides* and Hookworm infection in Gyinyase

A. lumbricoides infection was high in males than females with prevalence rates of 7.32% and 2.56% respectively (Table 4.19). *A. lumbricoides* infection was statistically significant ($p < 0.05$) among 5-6years age group compared with 7-8 years, 9-10years and 11-12years age groups. *A. lumbricoides* infection was high among children who use borehole as their source of drinking with prevalence of 8.42% as compared to those who relied on pipe-borne water as their source of drinking water (1.90%). *A. lumbricoides* was significant ($p < 0.05$) studied children who use bushes or fields as their sanitary facilities as compared with those who relied on water closet, pit latrine and KVIP as their sanitary facilities. *A. lumbricoides* infection was statistically significant ($p < 0.05$) among children who do not wash their hand before eating and after defecation. *A. lumbricoides* infection was high among children who do not have knowledge about the purpose of hand washing (10.11%) than those who had knowledge about the purpose of hand washing (0.90). Children who buy food from food vendors around their school compound had high prevalence of 5.77% *A. lumbricoides*

infection as compared with those who bring food from their various homes (2.27 %). *A. lumbricoides* infection was statistically significant ($p < 0.05$) among children whose parents were farmers.

From table 4.19, hookworm infection was high among males than females with prevalence rates of 1.51% and 0.51% respectively. Children of 11-12year age group had a prevalence rate of 1.75%, followed by 5-6 years age group with a prevalence rate of 1.72%, 7-8 years age group had a prevalence rate of 1.00% and 9-10years age group had a prevalence of 0.78% (Table 4.19). The prevalence rate of 5.45% hookworm was recorded among children who lack sanitary facilities (bushes or fields). None of the children who usually wear footwear were infected with hookworm. Children who sometimes wear footwear had a prevalence rate of 16.67%.

Table 4.19 Proportion and Association of *Ascaris lumbricoides* and Hookworm infection in Gyinyase.

	Ascaris lumbricoides				Hookworm		
				Odd Ratio			Odd Ratio
Variable	**N**	**n(%)**	***P***	**(95% CI)**	**n (%)**	***P***	**(95%CI)**
Gender							
Male	205	15(7.32)	0.18	1.10(0.45-2.48)	4(1.95)	0.45	0.54(0.23-1.59)
Female	195	5(2.56)	-	1	1(0.51)	-	1
Age							
5-6yrs.	58	10(17.24)	0.04	1.58(0.66-2.06)	1(1.72)	0.28	0.62(0.11-2.13)
7-8yrs	100	5(5.00)	0.21	1.08(0.42-3.50)	1(1.00)	0.34	0.50(0.09-1.88)
9-10yrs	128	3(2.34)	0.35	0.52(0.11-2.40)	1(0.78)	0.51	0.43(0.21-2.00)
11-12yrs	114	2(1.75)	-	1	2(1.75)	-	1
Household Water							
Source							
Pipe-borne water	210	4(1.90)	0.42	0.50(0.25-1.79)	-	-	-
Borehole/Well	190	16(8.42)	-	1	-	-	-
River/stream	-						
Household Sanitary Facility							
No facility (Bush/field)	55	12(21.82)	0.01	2.12(0.93-4.32)	3(5.45)	0.18	1.03(0.41-3.24)
W.C	40	-	-	-	-	-	-
Pit latrine	110	2(1.82)	0.27	0.48(0.22-1.86)	1(0.90)	0.53	1
KVIP	195	6(3.08)	-	1	1(0.51)	-	-
Hand washing after							
Defecation							
Yes	190	2(1.05)	0.22	0.46(0.12-2.12)	-	-	-
No	110	12(10.91)	0.09	1.17(0.66-3.25)	-	-	-
Sometimes	100	6(6.00)	-	1	-	-	-
With what							
Soap and Water	101	-	-	-	-	-	-
Water Only	189	8(4.23)	-	-	-	-	-
Hand washing before							
Eating							
Yes	186	1(0.54)	0.30	0.75(0.42-2.15)	-	-	-
No	39	10(25.64)	0.02	2.78(1.06-6.24)	-	-	-
Sometimes	175	9(5.14)	-	1	-	-	-
With what							
Soap and Water	122	-	-	-	-	-	-
Water Only	239	10(4.18)	-	-	-	-	-
Purpose for Washing							

Known	222	2(0.90)	0.29	0.39(0.09-1.55)	-	-	-
Unknown	178	18(10.11)	-	1		-	-
Source of Food							
From the House	88	2(2.27)	0.34	0.68(0.42-2.88)	-	-	-
Around the school Compound	312	18(5.77)	-	1	-	-	-
Parent's occupation							
Farmer	45	10(22.22)	0.04	2.32(1.00-4.12)	4(8.89)	0.10	1.48(0.77-4.20)
Civil servant	50	-	-	-	-	-	-
Trader	140	5(3.57)	0.26	1.17(0.76-3.45)	-	-	-
Carpenter	15	-	-	-	-	-	-
Unemployed	50	5(10.00)	-	1	1(2.00)	-	1
Businessmen	100	-	-	-	-	-	-
Foot wear							
Usually	370	-	-	-	-	-	-
Sometimes	30	-	-	-	5(16.67)	-	-
Never	-	-	-	-	-	-	-

4.20 Proportion and Association of *Strongyloides stercoralis* infection in Gyinyase

S. stercolaris infection was not statistically significant ($p > 0.05$) with all the risk factor considered in the Gyinyase communities (Table 4.20).

Table 4.20 Proportion and Association of *Strongyloides stercoralis* infection in Gyinyase.

	Strongyloides stercolaris			
				Odd Ratio
Variable	**N**	**n(%)**	***P***	**(95% CI)**
Gender				
Male	205	3(1.46)	-	-
Female	195	-	-	-
Age				
5-6yrs.	58	1(1.72)	0.16	0.65(0.27-1.76)
7-8yrs	100	1(1.00)	0.32	0.42(0.09-1.15)
9-10yrs	128	-	-	-
11-12yrs	114	1(0.88)	-	1
Household Water				
Source				
Pipe-borne water	210	-	-	-
Borehole/Well	190	3(1.58)	-	-
River/stream	-	-	-	-
Household Sanitary Facility				
No facility (Bush/field)	55	2(3.64)	0.12	0.75(0.56-3.22)
W.C	40	-	-	-
Pit latrine	110	-	-	-
KVIP	195	1(0.51)	-	1
Hand washing after				
Defecation				
Yes	190	-	-	-
No	110	2(1.82)	0.26	0.55(0.45-2.42)
Sometimes	100	1(1.00)	-	1
With what				
Soap and Water	101	-	-	-
Water Only	189	1(0.53)	-	-
Hand washing before				
Eating				
Yes	186	-	-	
No	39	2(5.13)	0.15	1.05(0.21-2.55)
Sometimes	175	1(0.57)	-	1
With what				
Soap and Water	122	-	-	-
Water Only	239	1(0.42)	-	-

Purpose for Washing				
Known	222	-	-	-
Unknown	178	3(1.69)	-	-
Source of Food				
From the House	88	-	-	-
Around the school Compound	312	3(0.96)	-	-
Parent's occupation				
Farmer	45	-	-	-
Civil servant	50	-	-	-
Trader	140	-	-	-
Carpenter	15	-	-	-
Unemployed	50	3(6.00)	-	-
Businessmen	100	-	-	-

4.21 Proportion and Association of *Giardia lamblia* and *Cryptosporidium parvum* infection in Kyirapatre

The prevalence rates of *G. lamblia* and *C. parvum* infection were high among males (20.57% *G. lamblia*, 17.14% *C. parvum*) than females (8.89% *G. lamblia*, 6.67% *C. parvum*) in Kyirapatre (Table 4.21). *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among 5-6years age and 7-8 years than 9-10 years and 11-12 years age groups (Table 4.21). From table 4.21, studied children from Gyinyase who relied on borehole had high prevalence rates of 21.02% *G. lamblia* and 19.88% *C. parvum* infection as compared to those who relied on pipe-borne water as their source of drinking water had prevalence rates of 8.48% *G. lamblia* and 4.46% *C. parvum*. *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among children who lack sanitary facilities (bushes or open space). *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among studied children who do not

wash their hands before eating and after defecation (Table 4.20). Children who buy food from food vendors around their school compound had high prevalence rate of 12.40% *G. lamblia* and low prevalence of 12.00% *C. parvum* infections as compared to those who bring food from their various homes had prevalence rates of 12.00% *G. lamblia* and 10.80% *C. parvum* infections. *Giardia lamblia* and *Cryptosporidium parvum* infections were statistically significant ($p < 0.05$) among children whose parents were farmers and unemployed.

Table 4.21 Proportion and Association of *Giardia lamblia* and *Cryptosporidium parvum* infection in Kyirapatre.

	Giardia lamblia				*Cryptosporidium parvum*		
				Odd Ratio			**Odd Ratio**
Variable	**N**	**n(%)**	***P***	**(95% CI)**	**n (%)**	***P***	**(95% CI)**
Gender							
Male	175	36(20.57)	0.01	2.48(1.33-6.84)	30(17.14)	0.106	1.55(0.53-4.39)
Female	225	20(8.89)	-	1	15(6.67)	-	1
Age							
5-6yrs.	80	32(40.00)	0.001	10.02(2.72-15.12)	26(32.50)	0.013	3.42(1.88-8.25)
7-8yrs	95	10(10.53)	0.179	1.63(0.45-3.40)	10(10.53)	0.18	1.86(0.68-3.48)
9-10yrs	115	7(6.09)	0.31	1.00(0.58-2.23)	4(3.48)	0.36	0.76(0.25-2.46)
11-12yrs	110	7(6.36)	-	1	5(4.55)	-	1
Household Water							
Source							
Pipe-borne water	224	19(8.48)	0.20	1.22(0.62-4.10)	10(4.46)	0.24	0.56(0.18-1.65)
Borehole/well	176	37(21.02)	-	1	35(19.88)	-	1
River/stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	60	20(33.33)	0.02	2.57(1.60-5.72)	15(25.00)	0.05	2.22(0.83-6.24)
W.C	35	4(16.0)	0.16	1.32(0.55-4.09)	1(2.56)	0.323	
Pit latrine	145	10(6.89)	0.26	0.92(0.22-5.26)	8(5.52)	0.164	1.35(0.74-3.52)
KVIP	160	22(13.75)	-	1	21(13.13)	-	1
Hand washing after Defecation							
Yes	168	5(2.98)	0.23	0.68(0.21-2.46)	5(2.98)	0.36	0.50(0.16-2.00)
No	100	30(30.00)	0.04	2.00(1.20-4.93)	25(25.00)	0.11	1.66(1.02-4.10)
Sometimes	132	21(15.91)	-	1	15(11.36)	-	1
With what							
Soap and Water	100	2(2.00)	0.35	0.62(0.24-3.12)	1(1.00)	0.50	0.45(0.21-1.75)
Water Only	200	24(12.00)	-	1	19(9.50)	-	1
Hand washing before Eating							
Yes	175	8(4.57)	0.209	1.02(0.52-3.56)	2(1.14)	0.40	0.66(0.32-1.95)
No	42	25(59.52)	0.001	5.66(1.5-10.52)	20(47.62)	0.01	2.77(1.42-5.42)
Sometimes	183	23(12.57)	-	1	23(12.57)	-	1
With what							

Soap and Water	92	2(2.17)	0.41	0.47(0.20-1.52)	2(2.17)	0.372	0.58(0.11-2.51)
Water Only	266	29(10.90)	-	1	23(8.65)	-	1
Purpose for Washing							
Known	240	15(6.25)	0.20	1.20(0.57-4.15)	15(6.25)	0.173	1.12(0.42-3.33)
Unknown	160	41(25.63)	-	1	35(21.88)	-	1
Source of Food							
From the House	150	15(10.00)	0.33	0.84(0.44-2.57)	18(12.00)	0.207	0.85(0.52-2.45)
Around the school compound	250	31(12.40)	-	1	27(10.80)	-	1
Parent's occupation							
Farmer	45	15(33.33)	0.01	3.23(1.10-6.30)	7(4.67)	0.43	0.55(0.19-3.16)
Civil servant	60	3(5.00)	0.35	0.75(0.28-3.85)	4(6.67)	0.29	0.78(0.22-2.17)
Trader	155	14(9.03)	0.29	1.20(0.75-4.88)	10(6.45)	0.25	0.86(0.35-2.23)
Carpenter	8	-	-	-	1(12.50)	0.12	1.44(0.52-4.00)
Unemployed	70	20(28.57)	0.10	2.10(1.00-4.50)	20(28.57)	0.03	2.26(1.00-4.27)
Businessmen	62	4(6.45)	-	1	3(4.84)	-	

4.22 Proportion and Association of *Ascaris lumbricoides* and Hookworm infection in Kyirapatre

A. lumbricoides infection was high in males than females with prevalence rates of 8.00% and 2.67% respectively (Table 4.22). The prevalence rate of *A. lumbricoides* infection was high among 5-6years age group (13.75%), followed by 7-8years age group (5.26%), 9-10 years age group (2.61%) and 11-12 years age group (0.91%). *A. lumbricoides* infection was high among children who use borehole as their source of drinking with a prevalence rate of 9.09% as compared to those who relied on pipe-borne water as their source of drinking water with a prevalence rate of 1.79%. Studied children who lack sanitary facilities (bushes or field) had a high prevalence rate of 16.67% *A. lumbricoides* infection as compared with those who relied on pit latrine (2.07%) and those who use KVIP (4.38%) as their sanitary facilities. *A. lumbricoides* infection was not statistically significant ($p > 0.05$) among children who do not

wash their hand before eating and after defecation. Children who buy food from food vendors around their school compound had prevalence of 5.60% *A. lumbricoides* as compared to those who bring food from their various homes with prevalence of 4.00 %. *A. lumbricoides* infection was statistically significant ($p < 0.05$) among children whose parents were farmers.

From table 4.22, Hookworm infection was high among males than females with prevalence rates of 4.57% and 0.88% respectively. Children form the 11-12year age group had a prevalence rate of 4.55%, followed by 5-6 years age group with a prevalence of 2.50%, 9-10years age group with a prevalence of 1.74% and 7-8 years age group with a prevalence of 1.05% (Table 4.22). The prevalence rate of 8.33% Hookworm was recorded among children who lack sanitary facilities (bushes or field) and prevalence rates of 1.33% and 1.88% were recorded among children who relied on pit latrine and KVIP respectively as their sanitary facilities (Table 4.22). None of the children who usually wear footwear were infected with Hookworm infection. Children who sometimes wear foot wares had a prevalence rate of 28.57%.

Table 4.22 Proportion and Association of *Ascaris lumbricoides* and Hookworm infection in Kyirapatre.

	Ascaris lumbricoides				Hookworm		
				Odd Ratio			Odd Ratio
Variable	**N**	**n(%)**	***P***	**(95% CI)**	**n (%)**	***P***	**(95% CI)**
Gender							
Male	175	14(8.00)	0.204	0.82(0.36-2.54)	8(4.57)	0.11	1.33(0.26-3.21)
Female	225	6(2.67)	-	1	2(0.88)	-	1
Age							
5-6yrs.	80	11(13.75)	0.163	1.28(0.93-3.27)	2(2.50)	0.32	0.65(0.18-1.96)
7-8yrs	95	5(5.26)	0.25	0.73(0.54-2.10)	1(1.05)	0.44	0.45(0.11-1.53)
9-10yrs	115	3(2.61)	0.28	0.51(0.16-1.39)	2(1.74)	0.35	0.50(0.26-2.10)
11-12yrs	110	1(0.91)	-	1	5(4.55)	-	1
Household Water							
Source							
Pipe-borne water	224	4(1.79)	0.50	0.48(0.18-1.67)	-	-	-
Borehole/well	176	16(9.09)	-	1	-	-	-
River/stream	-	-	-	-	-	-	-
Household Sanitary Facility							
No facility (Bush/field)	60	10(16.67)	0.16	1.26(0.76-4.21)	5(8.33)	0.11	1.13(0.52-3.46)
W.C	35	-	-	-	-	-	-
Pit latrine	145	3(2.07)	0.305	0.65(0.44-2.26)	2(1.38)	0.34	1
KVIP	160	7(4.38)	-	1	3(1.88)	-	-
Hand washing after Defecation							
Yes	168	-	-	-	-	-	-
No	100	15(15.00)	0.195	0.91(0.53-3.15)	-	-	-
Sometimes	132	5(3.79)	-	1	-	-	-
With what							
Soap and Water	100	-	-	-			
Water Only	200	5(2.50)	-	-	-	-	-
Hand washing before eating							
Yes	175	1(0.57)	0.49	0.62(0.42-1.66)	-	-	-
No	42	12(28.57)	0.04	2.43(1.22-4.85)	-	-	-
Sometimes	183	7(3.83)	-	1	-	-	-
With what							
Soap and	92	-	-	-	-	-	-

Water							
Water Only	266	8(3.01)	-	-	-	-	-
Purpose for Washing							
Known	240	5(2.08)	0.26	0.72(0.36-2.77)	-	-	-
Unknown	160	15(9.38)	-	1	-	-	-
Source of Food							
From the House	150	6(4.00)	0.38	1.00(0.52-3.15)	2(1.33)	-	-
Around the school compound	250	14(5.60)	-	1	8(3.20)	-	1
Parent's occupation							
Farmer	45	10(22.22)	0.05	1.88(1.50-4.23)	6(13.33)	0.04	2.11(1.58-5.227)
Civil servant	60	-	-	-	-	-	-
Trader	155	5(3.23)	0.207	0.96(0.55-3.17)	-	-	-
carpenter	8	-	-	-	-	-	-
unemployed	70	5(7.14)	0.146	1	4(5.71)	-	1
businessmen	62	-	-	-	-	-	-
Foot wear							
Usually	365	-	-	-	-	-	-
Sometimes	35	-	-	-	10(28.57)	-	-
Never	-	-	-	-	-	-	-

4.23 Proportion and Association of *Strongyloides stercolaris* infection in Kyirapatre

The prevalence rate of *Strongyloides stercolaris* infection was high in males (2.29%) than females (0.44%) (Table 4.23). Children from 5-6 years group had a prevalence of 2.50%, followed by 7-8 years age group with prevalence of 2.11%, 11-12 years age group with prevalence of 0.91% and 9-10 years age group with prevalence of 0.87%. Children who lack sanitary facilities (bushes or field) had a prevalence of 3.33% but those who relied on pit latrine and KVIP had prevalence rate of 1.38% and 1.88% respectively as shown on Table 4.22. Children whose parents were farmers and unemployed had prevalence rate of 2.22% and 5.71% respectively.

Table 4.23 Proportion and Association of *Strongyloides stercolaris* infection in Kyirapatre.

	Strongyloides stercolaris			
				Odd Ratio
Variable	**N**	**n(%)**	***P***	**(95% CI)**
Gender				
Male	175	4(2.29)	0.199	1.15(0.45-4.27)
Female	225	1(0.44)	-	1
Age				
5-6yrs.	80	2(2.50)	0.25	0.82(0.46-2.28)
7-8yrs	95	2(2.11)	0.314	0.64(0.51-1.89)
9-10yrs	115	1(0.87)	0.53	0.45(0.13-1.78)
11-12yrs	110	1(0.91)	-	
Household Water				
Source				
Pipe-borne water	224	-	-	-
Borehole/well	176	5(2.84)	-	-
River/stream		-	-	-
Household Sanitary Facility				
No facility (Bush/field)	60	2(3.33)	0.34	0.62(0.23-1.86)
W.C	35	-	-	-
Pit latrine	145	1(0.69)	0.54	0.54(0.36-1.77)
KVIP	160	2(1.25)	-	1
Hand washing after Defecation				
Yes	168	-	-	-
No	100	3(3.00)	0.229	0.79(0.48-2.66)
Sometimes	132	2(1.52)	-	1
With what				
Soap and Water	100	-	-	-
Water Only	200	2(1.00)	-	-
Hand washing before Eating				
Yes	175	-	-	-
No	42	4(9.52)	0.415	1.00(0.53-2.22)
Sometimes	183	1(0.55)	-	1
With what				
Soap and Water	92	-	-	-
Water Only	266	1(0.38)	-	-
Purpose for				

Washing				
Known	240	-	-	-
Unknown	160	5(3.13)	-	-
Source of Food				
From the House	150	1(0.67)	0.56	0.63(0.25-2.16)
Around the school compound	250	4(1.60)	-	1
Parent's occupation				
Farmer	45	1(2.22)	0.46	0.88(0.51-2.54)
Civil servant	60	-	-	-
Trader	155	-	-	-
Carpenter	8	-	-	-
Unemployed	70	4(5.71)	-	1
Businessmen	62	-	-	-

CHAPTER FIVE

5.0. DISCUSSION

Intestinal parasitic infections and their morbidity have been rated the most predominant infections in developing countries especially among children of school going age (Nematian *et al.*, 2008). This has prompted several research studies into intestinal parasitic infections and their associated risk factors in different parts of the world with great enthusiasm. It is with similar interest that a comparative study of intestinal parasitic infection and associated risk factors among primary school children was conducted.

In the present study, the overall prevalence of intestinal parasitic infections observed among the studied population was 49.18% which was similar to results obtained in a study conducted among school children by Adeyeba and Akinlabi (2002), which revealed that 50.40% of the population harbored intestinal parasites. This could be due to similarities in geographical location and socioeconomic activities of the people. This finding was also consistent with a study conducted by Awolaju and Morenikeji (2008), which recorded 48.40%. The studied children had two or more of the intestinal parasitic infections with a prevalence rate of 8.20%.

Giardia lamblia infection was the most important intestinal protozoan recorded in the present study. The overall prevalence of *Giardia lamblia* infection among the studied children was 12.17%. In relation to the individual communities, the prevalence rates of *Giardia lamblia* infection were 11.25%, 18.00%, 9.25%, 5.00%, 15.50% and 14.00% in Ayigya (urban-poor), Kentinkrono (peri-urban), Aboabo (urban-poor), Manhyia (urban), Gyinyase (peri-urban) and Kyirapatre (peri-urban) respectively (Table 4.3). These prevalences of *Giardia lamblia* infections in the various communities in the present study falls below the 20 - 40%

prevalence range for developing countries as reported by Vandenberg (2006). This may be attributed to the fact that most of the studied children had access to good water supply and adequate sanitary facilities at home and school. Earlier studies conducted by Annan *et al*. (1986) among children in Ghana however revealed up to 18.2% Giardia infection whilst Verweij *et al*. (2003) reported 21.5% among children in Northern Ghana. Higher prevalence have been reported in other developing countries such as among children in the urban slums of Karachi, Pakistan (23.9%), Iranian day-care children (26.2%) by Heidari *et al*. (2003), children in the aborigine community in Pahang, Malaysia (44.1%) by Noor *et al*. (2007), and children in Amman, Jordan (78%) by Shakkoury *et al*. (2005).

The lower prevalence in the current study could be attributed to the fact that most of the studied communities had adequate sanitary facilities, good water supply and practiced proper hygiene (hand washing before eating and after defaecation). The overall prevalence of *Giardia lamblia* infection contrasted with results obtained by Nkrumah and Nguah (2011) among children within the Ashanti Akim North Municipality, where they found 89.5% prevalence. This was probably due to the fact that their study duration was longer (2006 to 2011) as compared with the present study period (January to September, 2011). The results were also in contrast with the study conducted in Kampongcham, Cambodia by Kyu-Jae (2002) (3.2% had *G. Lamblia*). This was attributed to poor hygiene practices and inadequate sanitary facilities found in some of the peri-urban communities (especially Kentinkrono and Gyinyase).

Giardia lamblia infection was highest in Kentinkrono (peri-urban) 18.00%, followed by Gyinyase (peri-urban) 15.50%, Kyirapatre (peri-urban) 14.00% Ayigya (urban-poor) 11.25%, Aboabo (urban-poor) 9.25%, and Manhyia (urban) 5.00%. The differences of *Giardia*

lamblia infection in communities were not statistically significant ($p > 0.05$). However the infection was high among males (16.78%) compared to females (7.84%) in all the studied communities, and the statistical difference was significant ($p < 0.05$) and in consonance with the work done by Adeyeba and Akinlabi (2002) (20.19% males) and Baldo *et al*. (2004) (17.00%) in Nigeria. According to their study and work done by Ahmed *et al.*, 2003, males obtained higher prevalence because they get more freedom than females whose leisure hours are strictly controlled and restricted hence are less exposed to parasitic infections. According to work done by Adebote *et al*., 2004, the more aggressive and explorative behaviour of the boys consequently make them more prone to infection and re-infection girls. The level of association was high with respect to males in Ayigya and Kentinkrono, than in Aboabo, Manhyia, Gyinyase and Kyirapatre.

The difference in the rate of *Giardia lamblia* infections among younger age (5-6years and 7-8 years) compared to older age (9-10years and 11-12years) in all the six communities was statistically significant ($p < 0.05$). This could be due to the fact that younger children have very active playing habits in and out of school and may come into contact with soil and water contaminated with these parasites. Our findings was in agreement with studies done by Addy *et al.* (2004) and Ayeh-Kumi *et al.* (2009), who reported that younger children have high *Giardia lamblia* infection than older children because most of these children do not practice important hygiene practices such as washing of hands with soap and water before eating, after playing in the soil and after visiting the toilets. Their studies also revealed that these children buy a lot of food from food vendors some of whom do not practise proper personal hygiene and may be carriers of the parasite. According to the study conducted by Heresi *et al.* (1997), children acquire immunity after the initial infections in early life which results in

some protection in later life hence the high infection rate observed in this study among younger age.

A study conducted by Arora and Arora (2005) in New Delhi revealed that sources of drinking water and their levels of cleanliness are a big factor in the infectivity of humans by parasites. Water polluted with human excreta will contain viable cysts and eggs of parasites which when swallowed by humans become infective for them. In the present study, *Giardia lamblia* infection was not significant ($p > 0.05$) with the children source of drinking but the studied children whose source of drinking water was borehole or well had the high *Giardia lamblia* infection compared to those who relied on pipe borne water. This could be attributed to low level of contamination of the water by the infective cyst of the parasites.

Giardia lamblia infection was statistically significant ($p < 0.05$) among children who lack sanitary facilities (bush/field) in Kentinkrono, Gyinyase and Kyirapatre. This agrees with work done by WHO (1993) which stated that inadequate sanitary facilities play an important role in intestinal parasitic infections.

Giardia lamblia infection was statistically significant ($p < 0.05$) among children whose parents were farmers in Ayigya, Kentinkrono, Gyinyase, Kyirapatre and Aboabo. This is because most of the children help their parents on their farms after school and during holidays. Our finding was in agreement with studies done by Kaur *et al*. (2002), Wongjindanon *et al*. (2005) and Noor *et al*., 2007, who reported that transmission of most intestinal parasites, required well favoured condition such as warm temperatures and moist climate. According to Traub *et al*. (2005), domestic animals such as dogs and sheep which also serve as reservoir hosts for *Giardia lamblia* provide a high of the infection. Most households have domestic animals such as dogs, sheep and goats which are often allowed to

roam outdoors either unsupervised or in the company of children as detected during the observational studies.

The studied children whose parents were unemployed recorded significant ($p < 0.05$) *Giardia lamblia* infection in Ayigya, Kentinkrono, Aboabo, Gyinyase and Kyirapatre. Studies done by Noor *et al*., 2007, Wongjindanon *et al*. (2005) and Quihui *et al.* (2006) revealed that poverty plays a vital role in intestinal parasitic infection.

Entamoeba histolytica is estimated to infect about 50 million people worldwide by Ryan *et al*. (2004). An estimated 10% of the world's population is infected with *E. histolytica*, the highest in developing countries with the lowest levels of sanitation Chacon-cruz (2009). *Entamoeba histolytica* and *Entamoeba dispar* are morphologically identical species. Although *Entamoeba histolytica/dispar* infections are predominant in developing countries, the overall prevalence of *Entamoeba histolytica/dispar* infection obtained from the present study was 0.21% (Table 4.3). This overall prevalence of *Entamoeba histolytica*/*dispar* infection recorded was much lower than results obtained by conducted by Igbinosa *et al*. (1996) in Nigeria where they found a prevalence rate of 6.79% and Malla *et al*. (2004) also recorded a prevalence of 6.22% among primary school children in Nepal. This could be attributed to less transmission of the parasite due to good water supply and adequate sanitary facilities in most of the studied communities.

The prevalence of *Entamoeba histolytica/dispar* among the studied children in a closed neighboured urban poor and peri-urban commuinites (Ayigya, Kentinkrono and Aboabo) were 0.25%, 0.75% and 0.25% respectively (Table 4.3). These prevalences were in contrast with a study conducted by Awolaju and Morenikeji (2008) within five communities in South West Nigeria (9.29%) and a study conducted in northern Ghana by Verweij *et al.* (2003).

Entamoeba histolytica/dispar infection *was* the least frequent intestinal protozoa present among the studied children. The differences in the rate of the infection were not statistically significant ($p > 0.05$) and the level of association between the risk factors employed in the study was weak. The present study recorded children from age 5-6 yrs to be most infected with *Entamoeba histolytica/dispar* compared with ages 7-8 yrs, 9-10 yrs and 11-12 yrs (Table 4.5). According to studies done by Addy *et al.* (2004) and Ayeh-Kumi *et al.* (2009), younger children easily come into contact with contaminated water, dirt, soil and they do not often practice proper hygiene.

Cryptosporidiosis is mostly common and severe among immunocompromised people, as well as very young or very old people (Brooks *et al.,* 2004). According to Kosek *et al.* (2001), *Cryptosporidium* is recognized as a major cause of waterborne diarrhoeal illness and as a pathogen with long term effects on child growth and development.

In the present study, *Cryptosporidium parvum* infection was the third predominant intestinal parasite detected among the primary school children in the studied communities with the prevalence of 8.50%% (Table 4.3). *Cryptosporidium parvum* infections in the individual communities were 7.50%, Ayigya and 12.50%, Kentinkrono (close neighbours), 6.25%, Aboabo and 4.00%, Manhyia (close neighbours), and 9.50%, Gyinyase and 11.25%, Kyirapatre (close neighbours) (Table 4.3). These prevalences in the present study were in contrast with 26.90% *Cryptosporidium parvum* recorded during a cross sectional survey of children with diarrhoea conducted in Liberia by Hojlyng *et al.* (1984) probably because the present study is an epidemiological one and excludes hospitalized diarrhoea children. The present prevalence was in contrast with a study conducted by Nagwa *et al.* (2010) among children in the Kingdom of Saudi Arabia (0.6% *Cryptosporidium parvum*). This could be

attributed to the less transmission rate of the parasites due to availability of good water source and adequate sanitary facilities and good hygiene practises among children in their study as compared to the present study. *Cryptosporidium parvum* infection was highest among primary school children in Kentinkrono (12.5%), followed by Kyirapatre (11.25%), Gyinyase (9.50%), Ayigya (7.50%), Aboabo (6.25%) and Manhyia (4.00%). This could be attributed to the fact that the peri-urban communities (Kentinkrono, Gyinyase and Kyirapatre) do not have adequate sanitary facilities in school and at home, most of them depend on boreholes or wells as their source of drinking water which may be contaminated with infective *Cryptosporidium* oocysts. Most of the studied children do not practice good personal hygiene practices (hand washing with soap and water before eating and after defaecation) hence high *Cryptosporidium parvum* infections the peri-urban commuities.

The difference in the rates of *Cryptosporidium parvum* infection in the current study was not statistically significant ($p > 0.05$) with gender, but infection was high in males (17.41%) than in female (12.95%). Our findings was in agreement with studies done by Addy *et al.,* (2004) and Ayeh-Kumi *et al.* (2009), who reported that most males do not practice proper personal hygiene before eating, after defecation and after playing in soils. *Cryptosporidium parvum* infection was statistically significant ($p < 0.05$) among the younger age (5-6 years) and the strength of association was high in all the communities. A study conducted in Ghana among children aged less than 5 years by Adjei *et al.,* (2004) (in Korle Bu Teaching Hospital, Accra) reported 27.80% prevalence of *Cryptosporidium parvum* in children with diarrhoea and 15.60% in children without diarrhoea. This may indicate that *Cryptosporidium parvum* infection has a strong association with respect to lower ages. A study conducted by Addy and Aikins (1986) in Kumasi, Ghana among children between the ages of 2 months- 5 years also revealed a prevalence of 12.90% *Cryptosporidium parvum* and was in contrast with that

of infection recorded among children between the ages of 5-6years, 7-8yrs in some of the six communities. *Cryptosporidium parvum* infection was statistically significant ($p < 0.05$) and had a strong association with poor hygiene practices (especially those who do not wash their hands before eating and after defecation) in the urban-poor and peri-urban communities. Workdone by Khan *et al*. (2004) and Adjei *et al*. (2004) revealed that poor hygiene is one of the major modes of transmission of *Cryptosporidium* infection.

Cryptosporidium parvum infection among the primary school children in Manhyia (4.00%) was lowest as compared to Ayigya (7.50%), Kentinkrono (12.5%), Aboabo (6.25%), Gyinyase (9.50%) and Kyirapatre (11.25%). This is attributed to the fact that Manhyia is a middle class community with availability of good drinking water and adequate sanitary facilities in both the schools and the homes of the children as compared to the other communities. *Cryptosporidium parvum* infection was statistically significant among the studied children who lack sanitary facilities (bushes/field) in Ayigya, Kentinkrono, Gyinyase, Kyirapatre and Aboabo. This corroborates a study conducted by Adebote *et al.* (2004) among primary school children in both public and private schools in Nigeria. They recorded higher infection in the studied children who lack sanitary facilities.

According to Olstein, 2001and Quihui *et al.* (2006), poverty constitutes one of the major factors which enhance intestinal parasitic infection; hence the studied children whose parents were unemployed were prone to high parasitic infection rate.

The investigation also reveals a prevalence of 6.30% intestinal helminth infections which comprise Hookworm (1.54%), *Ascaris lumbricoides* (3.88%) and *Strongyloides stercolaris* (0.88%). The overall prevalence 6.30% was much lower than a study conducted by Chukwuma *et al*. (2009) among primary school children in Ebenebe Town, Anambra State, Nigeria. They recorded 53.6% prevalence of intestinal helminth infection. A study conducted

by Tadesse (2005) among children in Babile, eastern Ethiopia found a prevalence of 27.2% helminth infection. This was attributed to the fact that in their study, most pupils defecate in the nearby bush surrounding the school and their homes. This results in the eggs being washed into the school compound and their surrounding when it rains resulting in the environment of the school and surrounding area being highly contaminated with eggs/larvae of the parasites and also most of the school children go to school barefooted leading to the high prevalence of geohelminth infections especially hookworm infections and 70% of the pupils had never taken anti-helminthic drugs in their life, hence the high prevalence in their study. The low prevalence in the current study could be attributed to the presence of adequate sanitary facility and good water supply in some of the studied communities and deworming practices reflected in the current study. According to Onwuliri *et al*. (1993), some ecological factors such as temperature, relative humidity and rainfall could affect the prevalence of the parasites.

The prevalences of Hookworm infection recorded among the studied children in Kentinkrono, Kyirapatre, Gyinyase, Ayigya and Aboabo are 3.75%, 2.50%, 1.25%, 1.25% 0.5% and respectively. The overall prevalence (1.54%) and that of the prevalence recorded in various communities were in contrast with a study conducted by Tadesse (2005) among primary school children in Babile town, eastern Ethiopia and Waikagul (2002) in Northern Thailand with prevalence rates of 6.7% and 18.5% respectively. This was because in the current study about 90% of the studied children put on foot wear both at school and at home. The prevalences of the infection were in contrast with a study conducted by Tay *et al*. (2010) among non school going children and occupational risk groups in the Kintampo north district of the Brong Ahafo region of Ghana (62.31%) which recorded high prevalence in children (42.80%), farmers (25.30%) and traders (12.80%). Hookworm infection was not statistically

significant ($p > 0.05$) among males and females, but males had a prevalence of 2.58% while females had a prevalence of 0.57% in all the communities. Our finding was in agreement with work done by Tay *et al*. (2010), who reported that males were usually exposed to the hookworm infection due to their activities such as playing footballs without footwear. Hookworm infection was not significant ($p > 0.05$) among age groups studied. Primary school children of 11-12 age group recorded highest hookworm infection. This could be due to their engagement in activities like gardening and farming that could expose them to the infection (Ahmed *et al.,* 2003). Hookworm infection was high among children whose parents are farmers in Kentinkrono (22.22%), Kyirapatre (13.33%) and Ayigya (20.00%). According to studies done by Ahmed *et al.,* (2003), the farming environment are conducive for the survival of infective egg of the parasite since the parasite requires warm, moist and shady condition to moult into an infective larva.The difference in the rate of Hookworm infection was statistically significant ($p < 0.05$) among the studied children who use bushes or fields as their sanitary facilities in Kentinkrono (13.33%), Kyirapatre (8.33%) and Gyinyase (5.45%). This agrees with a study done by Chukwuma *et al*., (2009), in Nigeria which revealed that due to lack of sanitary facilities, children defaecate in bushes and open spaces which aid in the contamination of the surrounding with infective egg/larvae of the parasites which may serve as a source of infections to the people especially children, hence high infection in these areas.

Ascaris lumbricoides (3.88%) was the highest intestinal helminth infection recorded among the studied children. The prevalence of *Ascaris lumbricoides* in the various communities were 7.50% in Kentinkrono, 5.00% in Gyinyase, 5.00% in Kyirapatre, 2.50% in Ayigya, 2.50% in Aboabo and 0.75% in Manhyia. Kentinkrono recorded the highest prevalence of *Ascaris lumbricoides* infection (7.50%). The overall prevalence and the individual

prevalences among the studied children in the studied communities were in contrast to a study conducted by Adeyeba and Akinlabi (2002) (12.04%) and Malla *et al,* (2004) (14.22%) respectively. This could be attributed to the fact that their studies were conducted in a rural setting as compared to the current study (peri-urban and urban areas). *Ascaris lumbricoides* infection was higher in males (5.58%) than females (2.02%) and was highest in 5-6years (10.84%) followed by 7-6 years (4.73%), 9-10years (2.40%) and 11-12years (1.07%) in all the communities. This could be due to the fact that males and younger children are most likely to come into contact with contaminated soil (because of their active behaviour and less supervision at home), water, food, most of them do not practise proper hygiene (do not wash hand after defecation and before eating), these as a result make them prone to the infection (WHO, 1997). The differences in the rate of *Ascaris lumbricoides* infection was not statistically significant ($p > 0.05$) among the studied children who relied on pipe-borne (1.01% Ayigya, 2.80% Kentinkrono, 0.67% Aboabo, 1.90% Gyinyase and1.79% Kyirapatre) but high among those who relied on borehole (6.86% Ayigya, 15.33% Kentinkrono, 8.00% Aboabo, 12.00% Manhyia, 8.42% Gyinyase and 9.09% Kyirapatre) as their source of drinking water. This was because most of the communities especially the urban-poor and urban communities have access to good water supply and adequate sanitary facilities.

Ascaris lumbricoides was statistically significant ($p < 0.05$) among the studied children who do not practise proper hygiene, and are high in children who buy food from food vendors around their school compound as compared to those who bring their food from their various homes. According to Addy *et al.,* (2004) revealed that *A. lumbricoides* infection is transmitted by ingesting food, water and soil contaminated with the infective eggs of the parasites and by faeco-oral transmission. According to work done by Ayeh-Kumi *et al.,* (2009) among food vendors, most food vendors do not practice personal hygiene and may

serve as a source of infection to people through the food they sell, hence children who buy food from food vendors around their schools recorded high *A. lumbricoides* infection as compared to those who bring food from their homes.

Strongyloides stercolaris (0.88%) infection was the least of helminth infection recorded among the studied children in the six communities. The prevalence of *Strongyloides stercolaris* in Ayigya, Kentinkrono, Aboabo, Gyinyase and Kyirapatre are 0.50%, 2.25%, 0.50%, 0.75% and 1.25% respectively. None of the studied children in Manhyia had *Strongyloides stercolaris* infection. This is due to the fact that Manhyia is middle class community (urban) and has access to sanitary facilities, potable drinking water and most of the children wear shoes both at school and at home. The prevalence observed were in contrast with the study conducted by Chukwuma *et al*. (2009) (9.40%). This was because in their study about 70% of the studied population lack sanitary facilities both at school and homes, hence they defecate in the open space or fields which may result in high contamination of the soil with the parasite larvae as compared to the current study. The prevalence recorded in current study was in agreement with studies conducted by AL-Zain (2009) (2.00%) among children in Gaza, Pakistan and Adhikari *et al*. (2004) (2.00%) in Kathmandu Valley. *Strongyloides stercolaris* infection was not statistically significant ($p > 0.05$) to gender and age but was higher among males (1.46%) and younger ages (especially 5-6years, 2.56%) than females (0.32%) and older ages (0.36% 11-12years) in all the communities. This was attributed to the fact that males and young age children are very active in their playing life and may come into contact with contaminated soil containing the infective larvae since the parasite required moist and shady conditions for their moulting into infective larvae (CDC, 2010).

The differences in the rate of *Strongyloides stercolaris* infection was not statistically significant ($p > 0.05$) with sanitary facilities (water closet, pit latrine, KVIP and those who defecate in bushes/field) but was high among the studied children who defecate in bushes and fields. This agrees with a study conducted by Ogwurike *et al*. (2010) among primary school children in Nigeria, recorded high infections among children (82.52% private school and 47.83% public school) who defecate in open space or bushes.

CHAPTER SIX

6.0. CONCLUSION AND RECOMMENDATION

6.1. CONCLUSION

The present study has revealed high prevalence of intestinal protozoa (42.88%) and low intestinal helminth infection (6.30%) among the studied children in some communities in Kumasi metropolis: Ayigya (urban-poor), Kentinkrono (peri-urban), Aboabo (urban-poor), Manhyia (urban), Gyinyase (peri-urban) and Kyirapatre (peri-urban). *Giardia lamblia* (12.17%) was the predominant pathogen recorded with a lower percentage having 0.21% *Entamoeba histolytica*, 0.88% *Strongyloides stercolaris*, 1.54% Hookworm and 3.88% *Ascaris lumbricoides*. The differences in the rates of most of the pathogenic protozoa (*Giardia lamblia*, *Cryptosporidium parvum*) and helminths (Hookworm and *Ascaris lumbricoides*) infections recorded among the studied children in the present study areas were statistically significant ($p < 0.05$) with risk factors such as gender, age, poor environmental sanitation, improper personal hygiene practices (children who do not wash their hands before eating and after defecation) and socioeconomic activities (especially studied children whose parent were farmers and unemployed). The studied children from Manhyia recorded the lowest of all the intestinal parasitic infections recorded (22%) in the present study. Primary school children from Kentinkrono recorded the highest of all intestinal parasitic infections (74.75%) in the present study. Males (65.66%) recorded the highest of intestinal parasitic infections than females (34.34%) among the studied population. The age groups most affected were 5-6years, followed by 7-8years, 9-10years and 11-12years age groups in all the communities.

6.2 Limitations

Diagnosis of intestinal parasites is confirmed by the recovery of protozoan trophozoites and cysts, helminth eggs and larvae in the clinical Parasitology laboratory. Due to the low density of the parasites in the faeces, the sensitivity, specificity and accurate microscopic identification of intestinal parasitic infections will depend on the method used, the number of stools analysed, and the quantity of parasites excreted per sample. In the current study direct and formol ether concentration methods were used and the result was based on a single sample obtained from individual participants. If three stool samples from each participant had been analysed, the sensitivity of the results would have been enhanced.

Diethyl ether was used in place of ethyl acetate as faecal fat extraction solvent. This was because it was very difficult to obtain ethyl acetate on the market. Ethyl acetate is a good faecal fat extractor as compared to diethyl ether because it provides clear sediment but may also end up extracting some of the parasite.

6.3. RECOMMENDATIONS

Due to the results obtained from the present study, it is recommended that the Ministry of health in conjunction with other stakeholders and school authorities should conduct Health education in these communities and schools on the modes of transmission, prevention and control of the intestinal parasites, environmental sanitations, personal hygiene and the impact of these intestinal parasitic infections.

There should be adequate provision of sanitary facilities in schools and in the communities. Food sellers at schools should be periodically screened for protozoa and helminths to prevent the transmission of the infection through the food they sell.

School deworming programmes and health educations (especially on personal hygiene practices) should be conducted in the study areas so as to minimize the spread of intestinal parasitic infections.

It is recommended that three stool samples should be obtained from individual participants so as to effectively detect the parasites if present during future study.

REFERENCES

Adamu, H., Endeshaw, T., Teka, T., Kife, A. and Petros, B. (2006). Prevalence of intestinal parasite. *Ethiop J Health Div*, **20(1)**:39-47.

Adam, M. (2001). Definition and boundaries of the Peri-urban interface: Patterns in the patch Work: Drenchsel, P., Kunze (Eds.), Waste composting for Urban and Periurban Agriculture: Closing the Rural-Urban Nutrient cycle in sub-saharan Africa, CABI Publishing, Wallingford. pp. 193-208.

Adams, E.B., MacLeod I.N., (1977). Invasive Amebiasis. II. Amebic liver abscess and complications. Medicine (Baltimore); **56**:325-334.

Addy, P.A. and Aikins-Bekoe, P., (1986). Cryptosporidiosis in diarrhoeal children in Kumasi, Ghana. *Lancet* **1**:735.

Addy, P.A.K., Antepim, G. And Frimpong, E.H., (2004). Prevalence of pathogenic Escherichia coli and parasites in infants with diarrhoea in Kumasi, Ghana. *E Afr Med J.* **81(7)**:353-357.

Adeyeba, O.A. and Akinlabi, A.M., (2002). Intestinal parasitic infections among school children in a rural community, South West Nigeria. *Nig. J. Parasitol*. **23**: 11-18.

Adjei, A.A., Armah, H., Rodrigues, O., Renner, L., Borketey, P., Ayeh-Kumi, P., Adiku, T., Sifah, E. and Lartey, M., (2004). *Cryptosporidium* spp., a frequent cause of diarrhoea among children at the Korle-Bu Teaching Hospital, Accra, Ghana. *Jpn J Infect Dis.* **57**: 216-219.

Adhikari ,N., Bomjan, R., Khatri, D.B., Joshi, D.R., Dhakal, P. and Lekhak, B., (2004). Intestinal Helminthic Infections among School Children in Kathmandu Valley. *Journal of Nepal Health Research Council* Vol.**5** (**1**). Page 17-21

Adrabbo, K. and Peura, D., (2002). Giardiasis: A Review. Practical Gastroenterology; 300-302.

Ahmed, A.K., Malik, B., Shaheen, G., Yasmeen, J.B., Dar, A.K., Mona, S., Gulab, M., Ayub J. (2003). Frequency of intestinal parasitic infestation in children of 5-12 years of age in Abbottabad. *J Ayub Med Coll Abbottabad*. **15(2)**: 28-30.

Alzain, B. F., (2006). A study on the Status of Prevalence of *strongyloides stercoralis* infection among children in Agricultural Areas in beit lahia, gaza strip. The Islamic University Journal (Series of Natural Studies and Engineering).Vol.**14**(**2**). Pages 67-73

Anderson, R.M. and May, R.M., (1995). Helminth infections of humans: mathematical models, population dynamics and control. *Advance in Parasitology.* **24**:1-101.

Annan A, Crompton D.W.T., Walters D.E., Arnold S.E., (1986).An investigation on the prevalence of intestinal parasites in pre-school children in Ghana. Parasitology 92: 209–217

Archampong, E.Q. and Clark, C.G., (1973) Surgical problems in Amebiasis. *Ann R Coll Surg Engl.* **52**:36-48.

Armson, A., Thompson, R.C. and Reynoldson, J.A., (2003). A review of chemotherapeutic approaches to the treatment of cryptosporidiosis. Expert Rev. *Anti Infect. Ther*. **1(2)**: 297-305.

Aristizábal, H., Acevedo, J. and Botero M., (1991). Fulminant amoebic colitis. *World J Surg*.; **15(2)**:216-219.

Arora, D. R. and Arora, B. (2005). *Medical Parasitology*. Second Edition, CBS Publishers and Distributors, New Delhi and Bangalore, India: 243pp

Arya, P.K., Kukreti, R., Arya, M. And Gupta, S.N., (May 2005). Magnetic resonance appearance of gall bladder ascariasis. *Indian J Med Sci*. **59(5)**:208-10.

Asaolu, S.O., Holland, C.V., Jegede, J.O., Fraser, N.R., Stoddard, R.C., Crompton, D.W.T., (1992). The prevalence and intensity of soil-transmitted helminthiases in rural communities in southern Nigeria. *Annals of Tropical Medicine and Parasitology* **86**,

279-287.

Awolaju, B.A. and Morenikeji, O.A., (2008). Prevalence and intensity of intestinal parasites in five communities in south-west Nigeria. *African Journal of Biotechnology*. **8 (18)**: 4542-4546.

Ayeh-Kumi, P.F., Ali, I.M., Lockhart, L.A., Gilchrist, C.A., Petri, W.A., Jr. and Haque, R., (2001). Entamoeba histolytica: genetic diversity of clinical isolates from Bangladesh as demonstrated by polymorphisms in the serine-rich gene. *Exp Parasitol.* **99**: 80-88.

Ayeh-Kumi, P.F., Quarcoo, S., Kwakye-Nuako, G., Kretchy, J.P., Osafo-Kantanka, A. and Mortu, S., (2009). Prevalence of Intestinal Parasitic Infections among Food Vendors in Accra, Ghana. *J Trop Med Parasitol*. **32(1)**:1.

Baldo, E.T., Belizairo, V.Y., Deleon, W.U., Kong, H.H. and Chung, D.I, (2004). Infection Status of intestinal parasites in children living in residential institutions in Metro Manila, the Philippines. *Korean J. Parasitol*. **42 (2)**: 67-70.

Barnes, P.F., De Cock, K.M., Reynolds, T.N. and Ralls, P.W., (1987). A comparison of amebic and pyogenic abscess of the liver. Medicine (Baltimore). **66**:472-483.

Baron, S., (1996). Enteric Nematodes of Humans in Medical Microbiology. 4th ed. Galveston, Texas. Online: http:// www.ncbi.nlm.nih.gov/bookshelf/br.fcgi?

Bethony, J., Brooker, S., Albonico, M., (May 2006). Soil-transmitted helminth infections: ascariasis, trichuriasis and hookworm. *Lancet.* **367** (**9521**): 1521-1532

Berger, L. M., Hill ,J., Waldfolgel, J.,(2005). Maternity Leave, Early Maternal Employment and Child Health and Development in the US. "The Economic Journal, **115**, F29-F47.

Berger, S.A. and Marr, J.S., (2006). Human Parasitic Diseases Sourcebook. Jones and Bartlett Publishers: Sudbury, Massachusetts: 502-527

Braga, L.L., Ninomiya, H. and McCoy, J.J., (1992). Inhibition of the complement membrane attack complex by the galactose-specific adhesion of Entamoeba histolytica. *J Clin*

Invest. **90(3)**:1131-7.

Brooker, S., Peshu, N., Warm, P. A., Mosobo, M., Guyatt, H. L., Marsh, K. and Snow, R. W. (1999). The epidemiology of hookworm infection and its contribution to anaemia among pre-school children on the Kenyan Coast. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, **91**: 240-24.

Brooker, S., Hotez, P.J. and Bundy, D.A., (2008). Hookworm-Related Anaemia among Pregnant Women: A Systematic Review. Trop.Dis. **2**: e291

Brooks, G., Janet, F., Butel, A. and Stephen, A. M., (2004). Jawetz, Melnick, & Adelberg's Medical Microbiology, 23rd Edition. New York: Lange Medical Books/McGraw Hill: pp 684-685

Bundy, D.A.P. and Cooper, E.S., (1989). Trichuris and trichuriasis in human. *Adv Parasitol*. **28**: 107-73.

Burkhart, C.N. and Burkhart, C.G., (2005). Assessment of frequency, transmission, and genitourinary complications of enterobiasis (pinworms). *International Journal of Dermatology* **44 (10)**: 837–40.

Caballero-Salcedo, A., Viveros-Rogel, M., Salvatierra, B., (1994). Seroepidemiology of amebiasis in Mexico. *Am J Trop Med Hyg*. **50(4)**:412-9.

Caldwell, J.P. (1982) Pinworms (*Enterobius vermicularis*). Canadian Family Physician **28**: 306–309.

Centers for Disease Control and Prevention (2000). *Parasitic Pathways—Drinking Water.* Available at http://www.cdc.gov/ncidod/dpd/parasiticpathways/drinkingwater.htm

Centers for Disease Control and Prevention. 2009-02-05. http://www.cdc.gov/crypto/.

Centers for Disease Control and Prevention (CDC) (2010). "Enterobiasis" Global Health – Division of Parasitic Diseases and Malaria.

http://www.cdc.gov/parasites/pinworm/diagnosis.html

Chan, M.S., Medley, G.F. and Jamison, D.A., (1994). The evaluation of potential global morbidity attributable to intestinal nematode infections. *Parasitology* **109**; 373-87.

Chacon-cruz, E., (2009). Intestinal protozoa diseases. General hospital Tijuana, Mexico. Pp 23- 28.

Chardrahekhar, T.S., Joshi, H. S., Gurung, M., Subba, S.H., Rana, M.S. and Shivananda, P.G., (2005). Prevalence and distribution of intestinal parasitic infections among school children in Kaski district, western Nepal. *Journal of medicine and biomedical research*. **4:** 78-82.

Chen, W., Harp, J.A. and Harmsen, A.G., (April 2003). *Cryptosporidium parvum* infection in gene-targeted B cell-deficient mice. *J. Parasitol.* **89 (2)**: 391–393.

Chen, X.M., Keithly, J.S., Paya, C.V. and LaRusso, N.F., (2002). Cryptosporidiosis. *N. Engl. J. Med.* **346 (22)**: 1723–31.

Checkley, W., Epstein, L.D., Gilman, R.H., Black, R.E., Cabrera, L. and Sterling, C.R., (1998). Effects of *Cryptosporidium parvum* infection in Peruvian children: growth faltering and subsequent catch-up growth. *Am J Epidemiol*. **148**:497-506.

Chongsuvivatwong V., Pas-Ong S., Mcneil D., Geater A., Duerawee M., (1996). Predictors for the risk of hookworm infection: experience from endemic villages in southern Thailand. Trans R Soc. Trop. Med. Hyg. **90**: 630-633.

Chukwuma ,M.C., Ekejindu ,I.M., Agbakoba ,N.R., Ezeagwuna ,D.A., Anaghalu I.C. and Nwosu,D.C.,(2009). The Prevalence and Risk Factors of Geohelminth Infections among Primary School Children in Ebenebe Town, Anambra State, Nigeria. Middle-East Journal of Scientific Research **4** (**3**): 211-215

Clark, C.G. and Diamond, L.S., (1992). Differentiation of pathogenic *Entamoeba histolytica* from other intestinal protozoa by riboprinting. *Arch Med Res*. **23**:15-16.

Clark, C.G. and Diamond, L.S., (1993). *Entamoeba histolytica*: a method for isolate identification. *Exp Parasitol*. **77**:450-455.

Clark, D.P. (1999). New insights into human cryptosporidiosis. *Clin Microbiol Rev*. **12**:554- 63.

Connolly, K.J. and Kvalsvig, J.D., (1993). Infection, nutrition and cognitive performance in children. *Parasitology* 107, S187-S200.

Chongsuvivatwong, V., Pas-Ong, S., McNeil, D., Geater, A. And Duerawee, M., (1996). Predictors for the risk of hookworm infection: experience from endemic villages in southern Thailand. *Trans R Soc Trop Med Hyg*. **90**:630-3.

Cook, G.C. (1994) *Enterobius vermicularis infection. Gut* **35 (9)**: 1159–62.

Cook, G.C. and Zumla, A.I., (2009). Manson's tropical diseases. 22nd ed. Saunders Elsevier. pp. 1515–1519.

Crompton, D.W.T. (1999). How much helminthiasis is there in the world? *Journal of Parasitology* **85**: 397-403.

"Cryptosporidiosis" Gideon. 2009-02-23. http://web.gideononline.com/web/tox_diseases/index.php?disease=355&view=General.

Dada-Adegbola, H.O. and Bakare, R.A., (2004). Strongyloidiasis in children five years and below. *West Afr. J. Med*. **23(3)**: 194-197.

de Silva N. R., Brooker S., Hotez P.J., Montresor A., Engles D., and Savioli L. (2003). "Soil-Transmitted Helminth Infections: Updating the Global Picture." *Trends in Parasitology.* **19**: 547–51.

De Villiers, J.P. and Durra, G. (1998). Amoebic abscess of the brain (Case Report). *Clin. Radiol.* **53**: 307-309

Donkor, A-P. K., (2009). *Trichuris trichiura*. Department of emergency medicine. Olive view internal medicine. University of Califonia, Los Angeles medical center.321-333pp

Dorris M., Viney M.E. and Blaxter M.L. (2002). Molecular phylogenetic analysis of the genus *Strongyloides* and related nematodes. *Int J Parasitol*. **32(12)**:1507-17.

DPDx. Division of Parasitic Diseases, Centers for Disease Control and Prevention. "Enterobiasis".http://www.dpd.cdc.gov/DPDx/html/Enterobiasis.htm.retrieved 09/02/2011

DPDx; Strongyloidiasis - (No Authors Listed) Centers for Disease Control & Prevention, National Center for Infectious Diseases, Division of Parasitic Diseases.

Drake, L. J. and Bundy, D. A. P., (2001). Multiple helminth infections in children: impact and control. *Parasitology,* **122**, pp S73-S81.

Ekpo, U.F., Odoemene, S.N., Mafiana, C.F. and Sam-Wobo, S.O., (2008). Helminthiasis and hygiene conditions of schools in Ikenne. Ogun state, Nigeria. *PLoS Negl Trop Dis.* **2(1)**: 146.

Farthing, M.J., (1996). Giardiasis. *Gastroenterol Clin North Am.* **25(3)**:493-515.

Farthing, M.J., (2000). Clinical aspects of human cryptosporidiosis. *Contrib Microbiol*. **6**:50-74.

Flanagan, P.A., (1992). Giardia--diagnosis, clinical course and epidemiology. A review. *Epidemiol Infect*. **109(1)**:1-22.

Fotedar, R., Stark, D., Beebe, N., (2007). Laboratory diagnostic techniques for Entamoeba species. *Clin Microbiol Rev*. **20(3)**:511-32.

Garcia, L.S., (2007). Intestinal nematodes In: *Diagnostic Medical Parasitology.* 5th ed. Washington, DC: ASM Press: 249 -289.

Garcia, L.S., (2009). Practical guide to diagnostic Parasitology. *American Society for Microbiology*. pp. 246–247.

Garcia, L. S., (2001). *Diagnostic Medical Parasitology*, ed. 4., ASM Press, Washington, D.C., p. 723.

Gasser, R.B., Cantacessi, C. and Campbell, B.E., (2009). Improved molecular diagnostic tools for human hookworm. *Expert Rev. Mol. Diagn.* **9 (1)**: 17–21.

Gatei, W., Wamae, C.N., Mbae, C., Waruru, A., Mulinge, E., Waithera, T., Gatika, S.M., Kamwati, S.K., Revathi, G. and Hart, C.A., (2006). Cryptosporidiosis: prevalence, genotype analysis, and symptoms associated with infections in children in Kenya. *Am. J. Trop. Med. Hyg*. **75(1)**: 78-82.

Ghana Health Service (2007). Two year strategic plan for Integrated Neglected Tropical Disease Control in Ghana: 2007-2008.

Ghana Statistical Service (2002) 2000 Population and Housing census, Special Report on Urban localities.

Gilgen, D. and Mascie-Taylor, C.G.N., (2000). The effect anthelminthic treatment on helminth infection and anaemia. *Parasitology* **(122)**: 105-110.

Gonin, P. and Trudel, L., (2003). Detection and differentiation of *Entamoeba histolytica* and *Entamoeba dispar* isolates in clinical samples by PCR and enzyme-linked immunosorbent assay. *J Clin Microbiol*. **41**: 237-241.

Gonzalez-Ruiz, A., Haque, R. and Aguirre, A., (1994). Value of microscopy in the diagnosis of dysentery associated with invasive *Entamoeba histolytica*. J Clin Pathol. **47**:236-239.

Guerrant, D.I., Moore, S.R., Lima, A.A., Patrick, P.D., Schorling, J.B. and Guerrant, RL., (1999) Association of early childhood diarrhea and cryptosporidiosis with impaired physical fitness and cognitive function four–seven years later in a poor urban community in northeast Brazil. *Am J Trop Med Hyg*. **61**:707-13.

Gutiérrez, Y., (2000) Diagnostic pathology of parasitic infections with clinical correlations.

2nd ed. Oxford University Press. pp. 354–366.

Guyatt, H.L., Chan, M.S. and Medlet, G.F., (1995). Control of Ascaris infection by chemotherapy: which is the most cost effective option? *Tran Roy Soc Trop Med Hyg.* **89**:16.

Hasegawa, H., Takao, Y., Nakao, M., Fukuma, T., Tsuruta, O. and Ide, K., (1998). Is *Enterobius gregorii* Hugot (Nematoda: Oxyuridae) a distinct species? *The Journal of Parasitology* **84 (1)**: 131–134.

Haque, R., Mondal, D., Duggal, P., (2006). *Entamoeba histolytica* infection in children and protection from subsequent amebiasis. *Infect Immun*. **74(2)**:904-909.

Haque, R., Mollah, N.U., Ali, I.K.M., (2000). Diagnosis of amebic liver abscess and intestinal infection with the TechLab *Entamoeba histolytica* II antigen detection and antibody tests. *J Clin Microbiol*. **38**:3235-3239.

Hawdon, J.M. and Hotez, P.J., (1996). Hookworm: development biology of the infectious process. *Curr. Opin. Genet. Dev.* **6 (5)**: 618–23.

Heidari, A., Rokni, M., (2003). Prevalence of Intestinal Parasites among Children in Day-care Centers in Damghan-Iran. Iranian *J Publ Health*; **32(1)**: 31-34.

Heresi, G. and Cleary, T.G., (1997). Giardia. *Ped Rev*. **18(7)**:243

Hill, D.R., (1993). Giardiasis. Issues in diagnosis and management. *Infect Dis Clin North Am*. **7(3)**:503-25.

Hojlyng, N., Molbak, K., Jepsen, S. and Hansson, A.P., (1984). Cryptosporidiosis in Liberian children. *Lancet* 1: 734.

Hotez, P.J., Bethony, J., Bottazzi, M.E., Brooker, S. and Buss, P., (2005). Hookworm: the great infectious of mankind.*PLoS Med.* **2 (3)**: 67.

Hotez, P.J., Brooker, S., Bethony, J., Bottazzi, M.E., Loukas, A., (2004). Current concepts: Hookworm infection. *N Engl J Med.* **351**: 799–807.

Homan, W.L. and Mank, T.G., (2001). Human giardiasis: genotype linked differences in clinical symptomatology. *Int J Parasitol* 31 (**8**): 822-826

Hugot, J.P., (1983). *Enterobius gregorii* (Oxyuridae, Nematoda), a new human parasite (in French). Annales de Parasitologie Humaine et Comparée 58 (4): 403–404.

Hunter, M.M. and Mckay, D.M., (2004). Review Article: Helminths as therapeutic agents for inflammatory bowel threadworms. Veternary clinic north, Amsterdam. Veternary partners.com Amsterdam. pp 167.

Huston, C.D., Haque, R. and Petri, W.A. Jr., (1999). Molecular-based diagnosis of *Entamoeba histolytica* infection. *Expert Rev Mol*. 1-11.

Huston, C. and Petri, W.A. Jr., (2001). Amebiasis. In: Rakel R, Bope A, eds. Conn's Current Therapy. Philadelphia: WB Saunders Co; 50-54.

Huttly, S.R. (1990). The impact of inadequate sanitary conditions on health in developing countries. *World Health Stat Q*. **43 (3)**: 118–26.

Igbinosa, I.B. and Ogbunuju, T.C., Ubonoiko, U.S.A., (1996). The Pattern of Intestinal Parasites. Anambra State, *Nigeria. J. Med. Lab. Sci*. **5**: 27- 31.

Igra-Siegman, Y., Kapila, R., Sen, P., Kaminski, Z.C. and Louria, D.B., (1981). Syndrome of hyperinfection with Strongyloides stercoralis. *Rev Infect Dis*. **3**:397-407.

Jemaneh, L., (1998). Comparative prevalence of some common intestinal helminth infections in different altitudinal regions in Ethiopia. *Ethiopia Medical Journal.* **36**: 1-8.

John, D.T. and Petri, W.A., (2006). Markell and Voge's Medical Parasitology (9th Ed.). Philadelphia: W. B. Saunders. p. 49.

Johnston, F.H., Morris, P.S., Speare, R., McCarthy, J., Currie, B., Ewald, D., Page, W. and Dempsey, K., (2005). Strongyloidiasis: A review of the evidence for Australian

practitioners. *Australian Journal of Rural Health*. **13**:247-254.

Jolly, C. A., Fernandez ,R., Muthukumar, A. R., Fernandes, G., (2001). Calorie restriction modulates Th-1 and Th-2 cytokine-induced immunoglobulin secretion in young and old C57BL/6 cultured submandibular glands. Aging (Milan); 11:383–389.

Jones, J.E., Balows, A., Hausler, W.J., Ohashi, M. and Turano, A., (1988). Giardiasis, Laboratory diagnosis of infectious diseases. 2nd ed., vol. 1. Springer-Verlag, New York. 872-882

Katz, M., Despommier, D.D. and Gwadz, R.W., (1989). Parasitic Diseases, 2nd ed., New York Inc. Springer-Verlag. Pages 136- 143

Katz K., Lim S. and Krajden S., (2004). Complicated and fatal *Strongyloides* infection in Canadians: Risk factors, Diagnosis and management. *Canadian medical association Journal*. Pp.479-484.

Kaur, R., Rawat, D., Kakkar, M., Uppal, B. and Sharma, V.K., (2002). Intestinal parasites in children with diarrhea in Delhi, India. *Southeast Asian J Trop Med Publ Health*. **33(4)**:725-729.

Keiser, J. and Utzinger, J., (April 2008). Efficacy of current drugs against soil-transmitted helminth infections: systematic review and meta-analysis. .*JAMA.* **299 (16)**: 1937–48.

Khan, W.A., Rogers, K.A., Karim, M.M., Ahmed, S., Hibberd, P.L., Calderwood, S.B., Ryan, E.T. and Ward, H.D., (2004). Cryptosporidiosis among Bangladeshi children with diarrhoea: a prospective, matched, case control study of clinical features, epidemiology and systemic antibody responses. *Am. J. Trop. Med. Hyg*. **71(4)**: 412-419.

King, S.E. and Mascie-Taylor, C.G., (2004). *Strongyloides fuelleborni kellyi* and other intestinal helminths in children from Papua New Guinea: associations with nutritional status and socioeconomic factors. *P N G Med J*. **47(3-4)**:181-91

Klaus, R., Ignatius, R., Weitzel, T., Seidu-Korkor, A., Anyidoho, L., Saad, E., Djie-Maletz, A., Ziniel, P., Amoo-Sakyi, F., Danikuu, F., Danour, S., Otchwemah, R.N., Schreier, E., Bienzle, U., Stark, K. and Mockenhaupt, F.P., (2007). Acute childhood diarrhoea in northern Ghana: epidemiological, clinical and microbiological characteristics. *BMC Infectious Diseases.* **7**: 1471-2334.

Kyu-Jae, L., Yong-Tae, B., Dong-Heui, K., Yong-Kun, D., Yong-Suk, R., Hun-Joo, K., Kyung-II I. and Tai-Soon, Y., (2002). Status of intestinal parasite infection among primary school children in Kamponcham. Cambodia. *Korean Journal Parasitol*. pp. 153-158.

Levinson, W., (2008) Review of medical microbiology and immunology, 10th edition.U.S.A. McGraw-Hill companies, Inc. pp379, 384-391.

Li, E. and Stanley, S.L. Jr., (1996). Protozoa: Amebiasis. *Gastroenterol Clin North Am*. **25(3)**:471-92.

Mahon, R.C. and Manuselis, G. (2000). Text book of Diagnostic Microbiology, 2 ed. Pennsylvania: Saunders.

Maizels, R.M., and Yazdanbakhsh, M. (2003). Immune regulation by helminth parasites: cellular and molecular mechanism. *Nat. Rev. Immunol.* **3 (9)**: 733–44.

Malla, B., Sherchand, J.B., Ghimire, P., Rajendra, B.C. K. and Gauchan, P. (2004). Prevalence of Intestinal Parasitic Infections and Malnutrition among Children in a Rural Community of Sarlahi, Nepal. *JNepal.Health Res Council*,Vol. 2: 55-57

Markell, E.K., John, D. C., Petri, W.H., (2006). Markell and Voge's medical Parasitology . 9th ed. St. Louis, Mo: Elsevier Saunders. ISBN 0-7216-479.

Marshall M.M., Naumovitz D., Ortega Y. and Sterling C.R. (1997). Waterborne protozoan pathogens. *Clin Microbiol Rev* 10, 67-85

Mata, L.J., (1982). Sociocultural Factors in the Control and Prevention of Parasitic Diseases.

Review of Infectious Diseases. **4**:871-79.

Mehlhorn, H., (1988). Parasitology in focus: fact and trends. Berlin, Heidelberg: Springer-Verlag. Pages *115: 104–112*.

Mengistu, A., Gebre-Selassie, S. and Kassa, T. (2007). Prevalence of intestinal parasitic infections among urban dwellers in southwest Ethiopia. *Ethiop.J.Health Dev.***21**(1). 13(2):157-161.

Meyer, E.A. (1990). Taxonomy and nomenclature. In Meyer E.A (ed). *Giardiasis.* Elsevier science: The Netherlands; 51-60.

Montresor, A., Crompton, D.W.T., Hall, A., Bundy, D.A.P. and Savoli, L., (1998). Guidelines for the evaluation of soil-transmitted helminthiasis and schistosomiasis at community level. Division of Control of Tropical Parasites Unit, World Health Organization: Geneva, Switzerland, WHO/CTD/SIP/98.1

Montresor, A., (2002). Helminth control in schoolchildren: a guide for managers of control programme. Geneva: World Health Organization.

Moraes, L.R., Cancio, J.A., Cairncross, S., (2004). Impact of drainage and sewage on intestinal nematode infections in poor urban areas in Salvador, Brazil. *Trans. R. Soc. Trop. Med. Hyg.* **98 (4)**: 197–204.

Murray P. R., Ken S. R. and Michael, A. P .F., (2005). Medical Microbiology. 5th ed. Philadelphia: Elsevier Inc.: pp. 855-856.

Nagwa, S.M.A. and Magda, M.M.M., (2010). Intestinal Parasitic Infection among Children in the Kingdom of Saudi Arabia. *Australian Journal of Basic and Applied Sciences*. **4(9)**: 4200-4204.

Nakano, T., Okamoto, M., Ikeda, Y. and Hasegawa, H., (2006). Mitochondrial cytochrome c oxidase subunit 1 gene and nuclear rDNA regions of *Enterobius vermicularis* parasitic in captive chimpanzees with special reference to its relationship with pinworms in

humans. *Parasitology Research.* **100 (1)**: 51–7.

Nazir, Z., Moazam, F., (1993). Amebic liver abscess in children. *Pediatr Infect Dis J*.**12**:929-932.

National Center for Biotechnology Information (NCBI) taxonomy database (2009). *Enterobius*. National Center for Biotechnology Information, U.S. National Library of Medicine.

Nematian, J., Gholamrezanezhad, A. and Nematian, E., (2008). Giardiasis and other intestinal parasitic infections in relation to anthropometric indicators of malnutrition: a large, population-based survey of schoolchildren in Tehran. *Ann Trop Med Parasitol*. **102(3)**: 209-14.

Neva, F.A. and Brown, H.W., (1994). Basic Clinical Parasitology, 6th ed. Norwalk, Connecticut: Appleton and Lange. Pp 356-358

Nkrumah, B. and Nguah, S.B., (2011). Giardia lamblia: a major parasitic cause of childhood diarrhoea in patients attending a district hospital in Ghana. **4**:163 http://www.parasitesandvectors.com/content/4/1/163

Nokes C. & Bundy D.A.P. (1994). Does helminth infection affect mental processing and educational achievement? *Parasitology Today* **10**:14-18.

Noor, A. M.Y., San, Y.M., Gan, C.C., Yusri, M.Y., Nurulsyamzawaty, Y., Zuhaizam, A.H., Maslawaty, M.N., Norparina, I. and Vythilingam, I., (2007). Prevalence of intestinal protozoa in an aborigine community in Pahang, Malaysia. *Trop Biomed*. **24**: 55-62.

Ogwurike, B., Ajayi, J., Ajayi, O.,Olaniyan, O., Nangna, J., Oluwadare, A.,(2010). A Comparative Study of Helminthiasis among Pupils of Private and Public Primary Schools in Jos North Local Government Area of Plateau State, Nigeria. *.Annals of Natural Sciences.*10 (1): 28 – 47.

Olstein, A., Samuelsen, H. and Onyango-Ouma, W., (2001). A study of risk factors for

intestinal helminth infections using epidemiological and anthropological approaches. *J Bio Sci*. **33**:569-84.

Ortiz, J.J., Ayoub, A., Gargala, G., Chegne, N.L. and Favennec, L., (2001). Randomized clinical study of nitazoxanide compared to metronidazole in the treatment of symptomatic giardiasis in children from Northern Peru. *Aliment Pharmacol Ther*. **15(9)**:1409-1415.

Peng, W., Anderson, T.J.C., Zhou, X. and Kennedy, M.W., (1998) Genetic variation in sympatric *Ascaris* population from humans and pigs in China. *Parasitology.* **117**: 355-356.

Petri W.A., Jr. and Mann, B.J. (1993). Molecular mechanisms of invasion by *Entamoeba histolytica*. *Semin Cell Biol* 4, 305-313.

Porto, L.M., Aurelia, M.F and Alcantara, M.L., (2005). Atypical clinical presentation of Strongyloidiasis in a patient co-infected with human T-cell lymphotrophic virus Type 1. *American Journal tropical medicine hygiene*.Vol **3**. pp. 124-126.

Posey, D.L., Blackburn, B.G. and Weinberg, M., (2007). Prevalence of Strongyloidiaisis among Africa refugees. Clinical infectious diseases. *American Journal tropical medicine*. Vol **3**.pp. 45-46.

Que, X. and Reed, S.L., (2000). Cysteine proteinases and the pathogenesis of amebiasis. *Clin Microbiol.* **13(2)**:196-206.

Quihui, L., Valencia, M. E., Crompton, D. W., Phillips, S., Hagan, P., Morales, G. and Diaz-Camacho, S. P., (2006). Role of the employment status and education of mothers in the prevalence of intestinal parasitic infections in Mexican rural schoolchildren. *BMC Public Health*. **6**: 225.

Rajak, C.L., Gupta, S., Jain, S., (1998). Percutaneous treatment of liver abscesses: needle aspiration versus catheter drainage. *AJR*. **170**:1035-1039.

Ravdin, J.I., Stanley, P., Murphy, C.F., (1989). Characterization of cell surface carbohydrate receptors for Entamoeba histolytica adherence lectin. *Infect Immun*. **57(7)**:2179-86.

Roberts, L.S. and Janovy, J. Jr., (2009). Foundations of Parasitology, 8th Edition. United States: McGraw-Hill. 865pp.

Rodriguez-Morales, A. J., Barbella, R.A., Case, C., Arria, M., Ravelo, M., Perez, H., Urdaneta, O., Gervasio, G., Rubio, N., Maldonado, A., Aguilera, Y., Viloria, A., Blanco, J. J., Colina, M., Hernandez, E., Araujo, E., Cabaniel, G., Benitez, J. and Rifakis, P,. (2006). Intestinal parasitic infections among pregnant women inVenezuela. *Infect Dis Obstet Gynecol*. **23**:125.

Roberts, L., Janovy, Jr., J., (2005). Foundations of Parasitology, 5th Edition. United States: McGraw-Hill. pp 412-415.

Rossignol, J.F., Ayoub, A. and Ayers, M.S., (2001). Treatment of diarrhoea caused by *Cryptosporidium parvum:* a prospective randomized, double-blind, placebo-controlled study of nitazoxanide. *J Infect Dis*. **184**:103-6.

Ryan, K. J. and Ray, C.G., (2004). Sherris Medical Microbiology: An Introduction to Infectious Disease. 4th ed. New York: McGraw-Hill, pp 727-738.

Sackey, M.E., Weigel, M.M. and Armijos, R. X., (2003). Predictors and nutritional consequences of intestinal parasitic infections in rural Ecuadorian children. *J Trop Pediatr*. **49:** 17-23.

Seeto, R.K. and Rockey, D.C., (1999). Amebic liver abscess: epidemiology, clinical features, and outcome. *West J Med*. **170**:104-109.

Sehgal, R., Gogulamudi, V. R., Jaco, J. V. and Atluri, V. S. R. (2010). Prevalence of intestinal parasitic infections among school children and pregnant women in a low socio-economic area, Chandigarh, North India. Reviews of infection (RIF) **1**(2):100-103. ISSN: 1837-6746.

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

Seydel, K.B., Li, E., Zhang, Z., and Stanley, S.L., Jr. (1998). Epithelial cell-initiated inflammation plays a crucial role in early tissue damage in amebic infection of human intestine. *Gastroenterology* **115:** 1446–1453.

Showkat, A. W., Fayaz, A., Showkat, A.Z., Zubair, A.D., Pervaiz, A.D. and Ayesha, A., (2010). Intestinal Helminthiasis in children of Gurez Valley of Jammu and Kashmir State, India. *America Society of Parasitologist*. 2(**2**): 91-94.

Siddiqui, A.A. and Berk, S.L., (2001). Diagnosis of *Strongyloides stercoralis* infection. *Clin Infect Dis*. **33(7)**:1040-7.

Skerratt, L.F., (1995). *Strongyloides spearei* n. sp. (Nematoda: Strongyloididae) from the common wombat *Vombatus ursinus* (Marsupialia: Vombatidae). *Systematic Parasitology*; **32**:81-89.

Speare, R., (1989). Identification of species of *Strongyloides*. In: Grove DI. (ed) Strongyloidiasis: a major roundworm infection of man. Taylor & Francis: London. pp 11-83.

Stanley, S.L. Jr., (2003). Amebiasis. *Lancet*. **361(9362)**:1025-34.

Steinmann, P., Zhou, X.N., Du, Z.W., Jiang, J.Y., Xiao, S.H., Wu, Z.X., (2008). Tribendimidine and Albendazole for Treating Soil-Transmitted Helminths, Strongyloides stercoralis and Taenia spp.: Open-Label Randomized Trial. *PLoS Negl Trop Dis*. **2(10):** 322.

Stenson, W.F., Zhang, Z., Riehl, T., and Stanley, S.L., Jr (2001). Amebic infection in the human colon induces cyclooxygenase-2. *Infect Immun* **69:** 3382–3388.

Stephenson, L.S., Latham, M.C. and Ottesen, E.A., (2000). Malnutrition and parasitic helminth infection. *Parasitology* 121: 23-38.

Stephenson, L.S., Latham, M., Adams, E., Kinoti, S.N. and Pertiet, A., (1993). Physical fitness, growth and appetite of Kenyan schoolboys with hookworm, Trichuris trichiura and Ascaris lumbricoides infections are improved four months after a single dose of albendazole. *J Nutr*. **123**: 1036-46.

St Georgiev, V., (2001). Chemotherapy of enterobiasis (oxyuriasis). *Expert Opin* Pharmacother. 2(2): 267-75.

Stoll, N.R., (August 1962). "On endemic hookworm, where do we stand today?". *Exp. Parasitol.* **12** (4): 241–52

Stoltzfus, R.J., Chwaya, H.M., Tielsch, J.M., Schulze, K.J., Albonico, M. And Savioli, L., (1997). Epidemiology of iron deficiency anaemia in Zanzibari school children: the importance of hookworms. *Am. J. Clin. Nutr*. **65**:153-159.

Tachibana, H., Kobayashi, S., Okuzawa, E. and Masuda, G. (1992). Detection of pathogenic *Entamoeba histolytica* DNA in liver abscess fluid by polymerase chain reaction. *Int J Parasitol*. **22**:1193-1196.

Tadesse, G., (2005). The prevalence of intestinal helminthic infections and associated risk factors among school children in Babile town, eastern Ethiopia. *Ethiop.J.Health Dev.* **19**(2): 140-147.

Tannich, E., Horstmann, R.D., Knobloch, J. and Arnold, H.H., (1989). Genomic DNA difference between pathogenic and nonpathogenic *Entamoeba histolytica*. *Proc Natl Acad Sci*. **86**:5118-5122.

Tandon, B.N., Tandon, R.K, and Satpathy, B.K., (1977). Mechanism of malabsorption in giardiasis: a study of bacterial flora and bile salt deconjugation in upper jejunum. *Gut*. **18(3)**:8115-29

Tay, S.C.K., Twum, W.A., Bosompem, K.M., (2010). Epidemiological survey of soil-transmitted helminthes in occupational risk groups and non-school going children in

the Kintampo North district of Ghana. *International Journal of Pure and Applied Science*, volume **3**: 64-81.

Tigabu, E., Petros, B. and Endeshaw, T., (2010). Prevalence of Giardiasis and Cryptosporidiosis among children in relation to water sources in Selected Village of Pawi Special District in Benishangul-Gumuz Region, Northwestern Ethiopia *Ethiop. J. Health Dev.* **24 (3)**:205- 212.

Totkova, A., Klobusicky, M., Holkova, R. and Valent, M., (2003). *Enterobius gregorii*-reality or fiction? *Bratislavské Lekárske Listy*. **104 (3)**: 130–133.

Traub, R.J., Inpankaew, T., Reid, S.A., Sutthikornchai, C., Sukthana, Y., Robertson, I.D. and Thompson, R.C., (2004). Transmission cycles of Giardia duodenalis in dogs and humans in Temple communities in Bangkok–a critical evaluation of its prevalence using three diagnostic tests in the field in the absence of a gold standard. *Acta Tropica*. **111(2)**:125-132.

Vandenberg, O., Van Laethem, Y., Souayah, H., Kutane, W.T., Van Gool, T. and Dediste, A., (2006). Improvement of routine diagnosis of intestinal parasites with multiple sampling and SAF-fixative in the triple-faeces-test. *Acta Gastroenterol Belg.* **69**: 361-366.

Van Eijk, A.M., Lindblade, K.A. and Odhiambo, F., (2009). Infections among Pregnant Women in Rural Western Kenya; a Cross-Sectional Study. *PLoS Negl. Trop. Dis*. **3**: 370

Verweij J.J., Oostvogel F., Brienen E.A., Nang-Beifubah A., Ziem J. and Polderman A.M. (2003). Short communication: Prevalence of *Entamoeba histolytica* and *Entamoeba dispar* in northern Ghana. *Trop Med Int Health* 8, 1153-1156.

Waikagul, J., Krudsood, S., Radomyos, P., Radomyos, B., Chalemrut, K., Jonsuksuntigul, P., Kojima, S., Looareesuwan, S. and Thaineau, W., (2002). A Cross- sectional Study

of Intestinal Parasitic Infections among School Children in Nan Province, Northern Thailand. *Southeast Asian J Trop Med Public Health*, **33(2)**: 218-23.

Washington W., J., Allen, S., Janda, W., Koneman, E., Procop, G., Schreckenberger, P. and Woods, G., (2006). Koneman's colour atlas and text book of diagnostic microbiology, 6th ed. Lippincott Williams and Wilkins, Baltimore. **22**:1245-1321.

Watkins, W.E. and Pollitt E., (1997). Stupidity or Worms: do intestinal worms impair mental performance? *Psychol. Bull.* **121**:171-191.

Welch, T.R., (2004). Evidence-based medicine in the wilderness: The safety of back country water. *Wilderness and environment medicine*, **15**, pp. 235-237.

Wichro, E., Hoelzl, D., Krause, R., Bertha, G., Reinthaler, F. and Wenisch, C., (2005). Microsporidiosis in travel-associated chronic diarrhoea in immune-competent patients. *Am J Trop Med Hyg* **73**: 285-287.

White, A.C. Jr, Chappell, C.L., Hayat, C.S., Kimball, K.T., Flanigan, T.P., Goodgame, R.W., (1994). Paromomycin for cryptosporidiosis in AIDS: a prospective, double-blind trial. *J Infect Dis*.**170**:419-24.

WHO (1997). World Health Organization/Pan American Health Organization/UNESCO report of a consultation of experts on amoebiasis. Wkly. Epidemiol. Rec. 72, 97-99.

WHO (1999). Monitoring Helminth Control Programmes (WHO/CDC/SIP/99.3). Guidelines for monitoring the impact of control programmes aimed at reducing morbidity caused by soil-transmitted helminths and schistosomes, with particular reference to school-age children, WHO, Geneva, Switzerland.

WHO (2000). Conquering Suffering Enriching Humanity. Report of an Informal Consultation, WHO.CWS ,Geneva, Switzerland.

Wolfe, M.S., (1992). Giardiasis. *Clin Microbiol Rev*. **5(1)**: 93-100.

Xiao, S.H., Hui-Ming, W., Tanner, M., Utzinger, J. and Chong, W., (2005). Tribendimidine:

a promising, safe and broad-spectrum anthelmintic agent from China. *Acta Trop*. **94(1)**:1-14.

Yoder, J.S. and Beach, M.J., (2007). Centers for Disease Control and Prevention (CDC).Giardiasis surveillance--United States, 2003-2005. *MMWR Surveill Summ*. **56(7)**:11-18.

Yong, T.S., Lee, J.H., Sim, S., (2007). Differential diagnosis of Trichostrongylus and hookworm egg via PCR using ITS-1 sequence. *Korean J. Parasitol.* **45 (1)**: 69–74.

Zaha, O., Hirata, T., Kinjo, F., (2000). Strongyloidiasis--progress in diagnosis and treatment. *Intern Med*. **39(9)**: 695-700.

Zhang, T., Cieslak, P.R., Foster, L., (1994). Antibodies to the serine rich *Entamoeba histolytica* protein (SREHP) prevent amoebic liver abscess in severe combined immunodeficient (SCID) mice. *Parasite Immunol*. **6**:225-230.

APPENDICES

Appendix 1.0. Materials used for the study

1.1. 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. Lugol's iodine (1%)

vii. Kinyoun carbol fuchsin

viii. Absolute methanol

ix. 50% ethanol: Add 50 ml of absolute ethanol to 50 ml of distilled water.

x. 1% sulphuric acid: Add 1 ml of concentrated sulphuric acid to 99 ml of distilled water.

xi Methylene blue

1.2. 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)

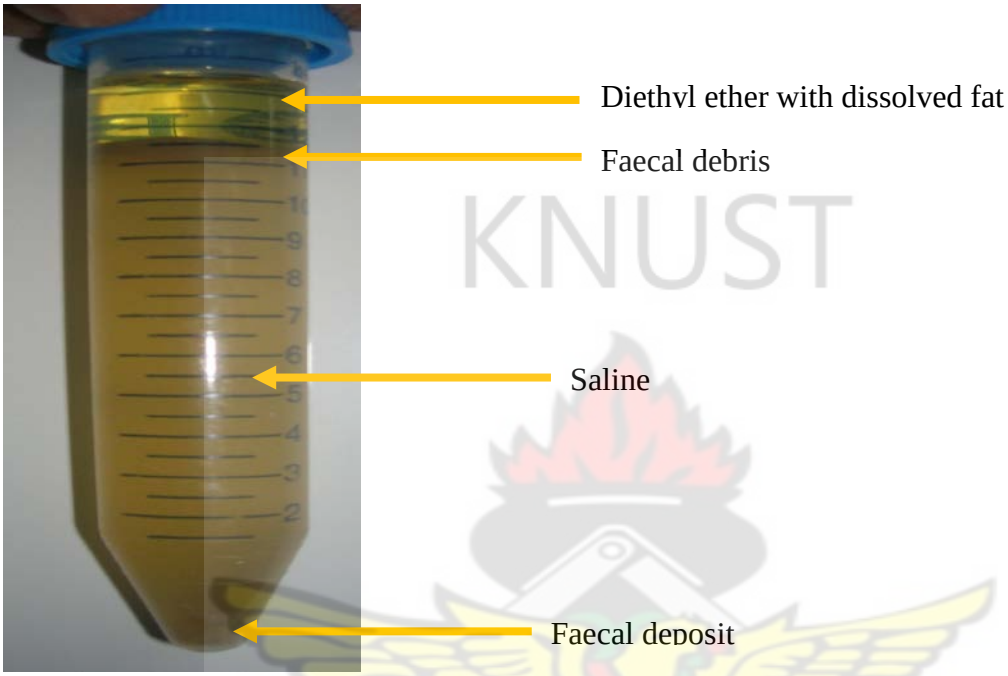
Appendix 2.0. Direct Wet Mount Method

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 3.0. 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

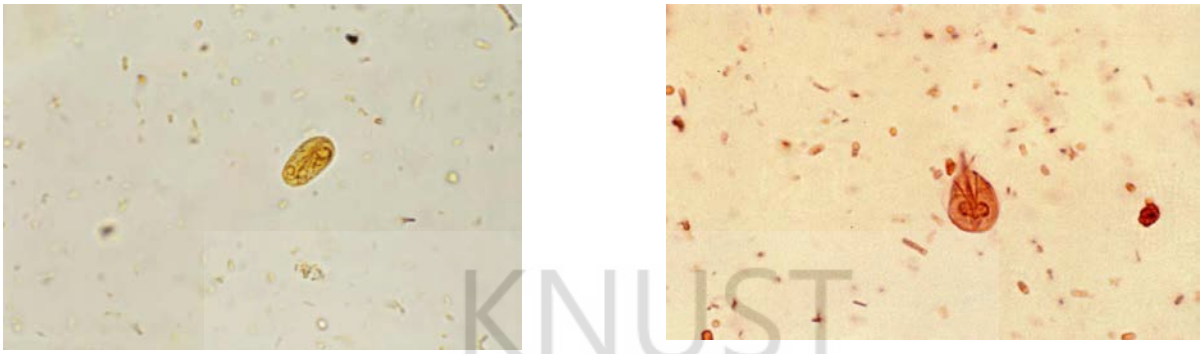
layers was form and the top three layers were discarded. A small portion of the sediment was transferred onto a clean glass slide (26x76mm) and covered with a cover slip (22x22mm) and was examine microscopically for Ova, cyst of parasites using X10 and X40 objective lens.



Appendix 4.0. Kinyoun Staining Method (*Cryptosporidium parvum Oocysts*)

A smear was made on a glass slide from a portion of fresh stool sample or formol ether concentrated sediment of the stool samples. The smear was allowed to air dry and it was fixed with absolute methanol for a minute. The smear on the slide was flooded with Kinyoun's carbol fuchsin and was stained for 5 minutes. The slide was rinsed briefly (3-5 seconds) with 50% ethanol after which it was thoroughly rinsed with water. The smear stained with Kinyoun's carbol fuchsin was decolourized with 1% sulphuric for 2 minutes. It was also rinsed with water and air dried. Methylene blue was used as counterstained for a minute. The prepared stained slide was examined microscopically using X40 and X100 (oil immersion) objective lens for the Oocysts of *Cryptosporidium parvum*.

Appendix 5.0. Diagnostic characteristic of some of the intestinal parasites which help in their identification in the current study.



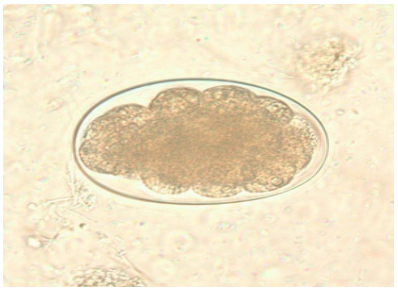
Giardia lamblia cyst and trophozoites iodine stained.

(SOURCE: PHIL 3741 – CDC/ Dr. Mae Melvin)

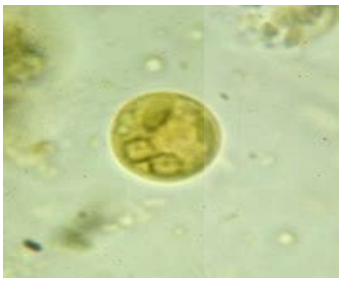
The cyst of *Giardia lamblia* is about 10-14µm in length, oval shaped, and has a thick cell-wall and contains four nuclei and various remnants of flagella. The trophozoite is pear-shaped, has four paired flagella, two nuclei and undulating membrane. Its length measures 10-18µm

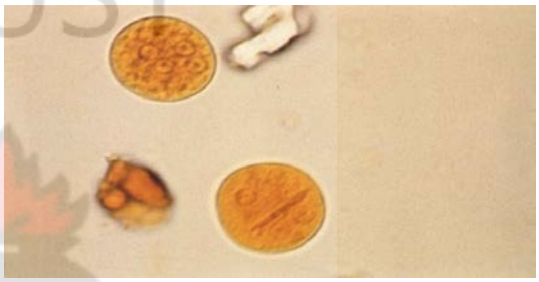
Iodine stain *Endolimax nana* cyst. (SOURCE: PHIL 530 - CDC/Dr. L.L.A. Moore, Jr.

Cysts of *E. nana* are 6-9µm in diameter. They can be spherical or ovoid in shape and contain four pinpoint nuclei, which are highlighted by the addition of iodine.



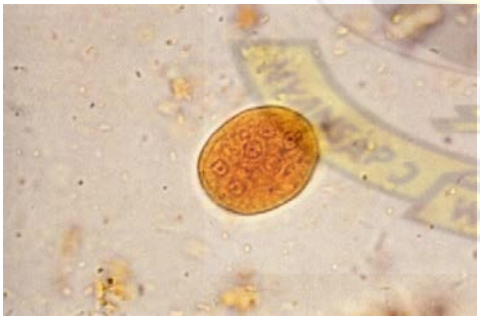
Hookworm eggs in iodine stained wet mounts SOURCE: (CDC, 2010: DPDx).





E. histolytica cyst in iodine with two visible nuclei and a chromatoid body (arrow). SOURCE: (CDC.2010: DPDx).

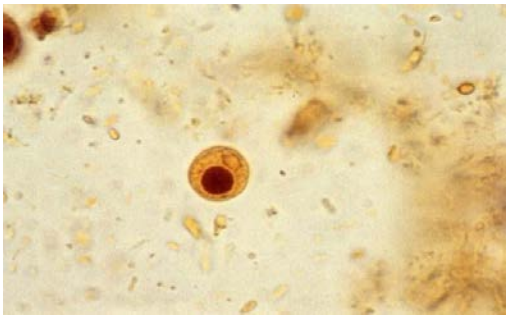
Cysts of *E. histolytica* are 10-15μm in diameter and contain one to four nuclei



Iodine stained *Entamoeba coli* cyst

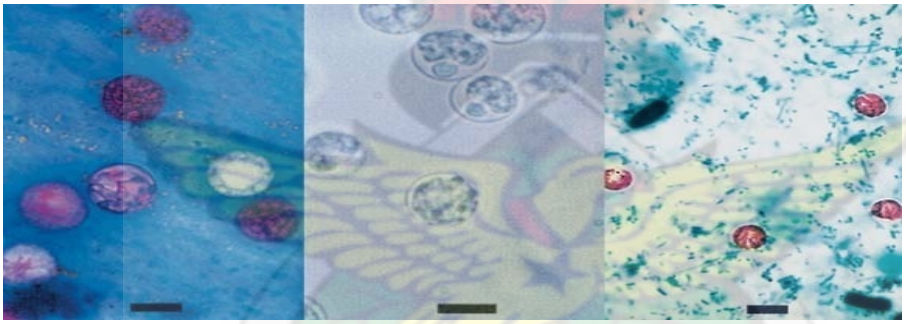
Source: Colour Atlas and Textbook of Diagnostic Microbiology

Cysts of *E. coli* are 15-30μm in diameter and contain one to eight nuclei with irregular peripheral chromatin: karyosome not central.

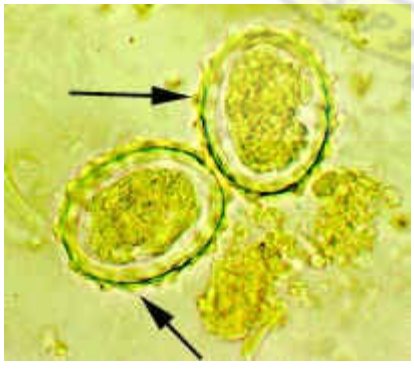


Source: Colour Atlas and Textbook of Diagnostic Microbiology

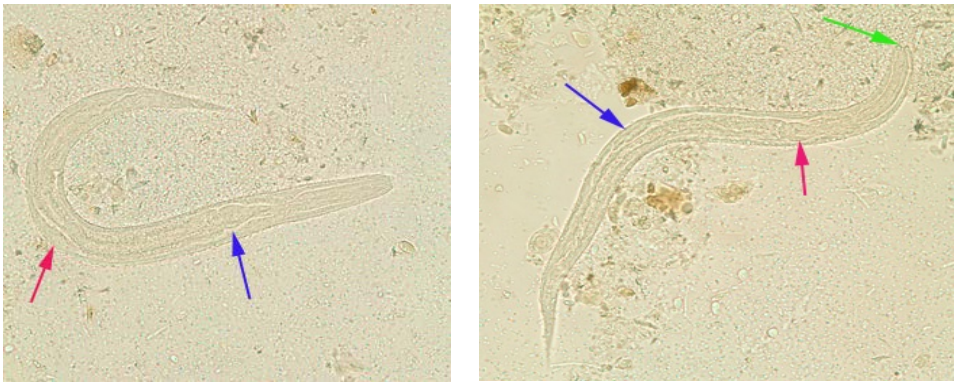
Iodine stained *Iodamoeba butschlii:* 9-15µm in diameter and have one nucleus in mature cysts usually eccentrically placed. Glycogen is present as a compact well defined mass staining dark brown with iodine.



Modified kinyoun acid-fast stain (modified Ziehl-Neelsen staining) Oocysts from *Cryptosporidium parvum* in stool smear.
Bench Aids for the Diagnosis of Intestinal Parasites. Geneva: WHO; 1998.



Typical fertilized egg with thick outer mammillated coat (60 x 45µm) of *Ascaris lumbricoides.*
Source: (Furjanic *et al*, 1999)



Source: CDC.2010: DPDx

Diagram A **Diagram B**

A: Rhabditoid larva of *S. stercoralis* in an unstained wet mount of stool. Notice the rhabditoid oesophagus (blue arrow) and prominent genital primordium (red arrow). **B:** Rhabditoid larva of *S. stercoralis* in an unstained wet mount of stool. Notice the prominent genital primordium (blue arrow), rhabditoid oesophagus (red arrow) and short buccal canal (green arrow).

Appendix 6.0. The Questionnaire

KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY AND KOMFO ANOKYE TEACHING HOSPOITAL, SCHOOL OF MEDICAL SCIENCES, DEPARTMENT OF CLINICAL MICROBIOLOGY

QUESTIONNAIRE ON A COMPARATIVE STUDY OF INTESTINAL PARSITIC INFECTIONS AND ASSOCIATED RISK FACTORS AMONG PRIMARY SCHOOL CHILDREN IN FOUR NEIGHBOURING COMMUNITIES WITHIN KUMASI: AYIGYA, KENTINKRONO, ABOABO, MANHYIA, GYINYASE AND KYIRATRE

ID. Number…….. Class…….

1. Gender [ ] Male [ ] Female
2. Which age group do you belong to?[ ] 5-6 [ ] 7-8 yrs [ ] 9-10yrs [ ] 11-12yrs
3. Place of Residence. [ ] Ayigya [ ] Kentinkrono [ ] Aboabo [ ] Manhyia
4. What is the main source of drinking water in your place of residence? [ ] Pipe-borne Water [ ] Borehole/ Well [ ] River/stream [ ] other (Specify…….
5. What type of sanitary facilities does your family usually use? [ ] No facility (e.g Bush/field [ ] W.C [ ] Pit latrine [ ] KVIP
6. Do you wash your hands after defecation? [ ] Yes [ ] No [ ] Sometime
7. If yes, with what? [ ] Soap and water [ ] Water only
8. Do you wash your hands before and after eating? [ ]Yes [ ] No [ ] Sometimes
9. If yes, with what? [ ]Soap and water [ ] Water only
10. Where do you buy your food? [ ] bring food from the house [ ] around the school compound [ ] do not buy food
11. Do you know the reason why you wash your hands? [ ] Known [ ] Unknown
12. Have you been dewormed recently? [ ] Yes [ ] No
13. What is the main source of drinking water in your school? [ ] Pipe-borne Water [ ] Borehole [ ] Well [ ] River/stream [ ] Other (Specify)……
14. Does the school you attend have toilet facilities? [ ] Yes [ ] No
15. If Yes, What Type? [ ] Pit latrine [ ] W.C [ ] Public Toilet
16. Has any deworming exercise being organized in the school/ community recently? [ ] Yes [ ] No
17. What type of dewormer was administered? …….
18. Foot wear behaviour. [ ] usually [ ] sometimes [ ] Never
19. Religion; [ ] Christian [ ] Islam
20. Ethnicity; [ ] Akan [ ] Northern [ ]

21. Parent’s occupation; [ ] farmer [ ] civil servants [ ] Trader [ ] Carpenter [ ] businessman.