

## CHAPTER 1.0 INTRODUCTION

### 1.1 Background

Schistosomiasis or bilharzia is a disease affecting many people in the developing countries and is one of the major parasitic diseases of huge public health importance today. It is considered the second most important human parasitic diseases after malaria in terms of morbidity and mortality (Chitsulo *et al*, 2000). In many areas within sub-Saharan Africa, it continues to drain the socio-economic development of already impoverished rural communities. Bilharzia or bilharziosis is the eponym for schistosomiasis after Theodor Bilharz, who first described the cause of urinary schistosomiasis in 1851 (Jordan and Webbe, 1982).

There are five species of flatworms that cause schistosomiasis. Each species causes a different clinical presentation of the disease. Schistosomiasis may localize in different parts of the body and its localization determines its particular clinical profile (Jordan *et al*, 1993). *Schistosoma mansoni* and *Schistosoma intercalatum* cause intestinal schistosomiasis. *Schistosoma haematobium* causes urinary schistosomiasis. *Schistosoma japonicum* and *Schistosoma mekongi* cause Asian intestinal schistosomiasis (Caatinga *et al*, 2000).

Schistosomes have a typical trematode vertebrate-invertebrate life cycle, with humans being the definitive host. The life cycles of all five human schistosomes are broadly similar. The most common way of getting schistosomiasis is by wading or swimming in lakes, ponds and other bodies of water which are infested with the snails (usually of the *Biomphalaria*, *Bulinus*, or *Oncomelania* genus) that are the natural reservoirs of the schistosomiasis pathogen (Caatinga *et al*, 2000).

Parasite eggs are released into the environment from infected individuals, hatching on contact with fresh water to release the free-swimming miracidium. Miracidia infect fresh-water snails by penetrating them. After infection, close to the site of penetration, the miracidium transforms into a primary (mother) sporocyst. Germ cells within the primary sporocyst then begin dividing to produce secondary (daughter) sporocysts,

which migrate to the snail's hepatopancreas. Once at the hepatopancreas, germ cells within the secondary sporocyst begin to divide again, this time producing thousands of new parasites, known as cercariae, which are the larvae capable of infecting mammals. Cercariae emerge daily from the snail host in a circadian rhythm, dependent on ambient temperature and light. Young cercariae are highly motile, alternating between vigorous upward movements and sinking to maintain their position in the water. Cercarial activity is particularly stimulated by water turbulence, shadows and human skin chemicals (Andrade and Bina, 1983).

Penetration of the human skin occurs after the cercaria have attached to the exposed skin. The parasite secretes enzymes that break down the skin to enable penetration of the cercarial head. As the cercaria penetrates the skin it transforms into a migrating schistosomulum stage. The newly transformed schistosomulum may remain in the skin for 1-2 days before locating a post-capillary venule where the schistosomulum travels to the lungs and it undergoes further developmental changes necessary for subsequent migration to the liver. Eight to ten days after penetration of the skin, the parasite migrates to the liver sinusoids. *S. japonicum* migrates more quickly than *S. mansoni*, and usually reaches the liver within 6-8 days of penetration. Juvenile *S. mansoni* and *S. japonicum* worms develop an oral sucker after arriving in the liver, and it is during this period that the parasite begins to feed on red blood cells. The nearly-mature worms pair, with the longer female worm residing in the gynaecophoric channel of the male. Adult worms are about 10 mm long. Worm pairs of *S. mansoni* and *S. japonicum* relocate to the mesenteric or rectal veins. *S. haematobium* schistosomula ultimately migrate from the liver to the perivesical venous plexus of the bladder, ureters and kidneys through the haemorrhoidal plexus. Parasites reach maturity in 6-8 weeks, at which time they begin to produce eggs. Adult *S. mansoni* pairs residing in the mesenteric vessels may produce up to 300 eggs per day during their reproductive lives. *S. japonicum* may produce up to 3000 eggs per day (Bina and Prata, 1990).

Many of the eggs pass through the walls of the blood vessels, and through the intestinal wall, to be passed out of the body in faeces. *S. haematobium* eggs pass through the

ureteral or bladder wall and into the urine. Only mature eggs are capable of crossing into the digestive tract, possibly through the release of proteolytic enzymes, but also as a function of host immune response, which fosters local tissue ulceration. Up to half the eggs released by the worm pairs become trapped in the mesenteric veins, or will be washed back into the liver, where they will become lodged. Worm pairs can live in the body for an average of four to five years, but may persist up to 20 years. Trapped eggs mature normally, secreting antigens that elicit a vigorous immune response. The eggs themselves do not damage the body. Rather, it is the cellular infiltration resultant from the immune response that causes the pathology classically associated with schistosomiasis (Bina and Prata, 1990).

The objective of this community based cross-sectional study was to determine the prevalence of liver disease due to *Schistosoma mansoni* infection amongst the population of Game Village in the Siavonga District. The study was carried out using clinical, laboratory and ultrasound examinations, including the Knowledge, Attitude and Practices (KAP) questionnaire to collect data. The study focused mainly on the prevalence of liver disease due to *Schistosoma mansoni* infection on the study population living in a schistosomiasis endemic area of the Game Village in the Siavonga District of Zambia.

## **1.2 Statement of the Problem**

In Zambia about 2 million people are affected with schistosomiasis (MoH, 2006). The investigations carried out in the Siavonga District by Mubila and Robinson (2002) and Chimbari *et al*, (2003) found the prevalence of *Schistosoma mansoni* infection to be 32% respectively. Another study undertaken by the Schistosomiasis Control Initiative (2007) in the Siavonga District found the prevalence of *Schistosoma mansoni* infection to be 77%.

The general signs and symptoms of gastro-intestinal tract (GIT) infections like abdominal pain, diarrhoea, abdominal enlargement and passing blood in stool are common in many cases. Despite the abdominal syndrome being common, the clinical

importance of *Schistosoma mansoni* infection has not been determined. No study has provided data on the prevalence of liver disease in school-children and adults infected with *Schistosoma mansoni* in the Siavonga District.

### **1.3 Justification of the Study**

This study is new in this part of Zambia and the data that was collected and analysed was intended to be used to understand the clinical picture of the disease in the *Schistosoma mansoni* infected individuals. The parasitological examination of specimens and the assessment of knowledge, attitude and practices (KAP) questionnaire was important in that it provided an opportunity for designing intervention strategies in the management of the disease and putting in place long-term strategies for sustainable morbidity control.

### **1.4 Literature Review**

Schistosomiasis is found in tropical countries in Africa, the Caribbean, Eastern South America, East Asia and in the Middle East. *Schistosoma mansoni* is found in parts of South America and the Caribbean, Africa, and the Middle East. *S. haematobium* is found in Africa and the Middle East and *S. japonicum* in the Far East. *S. mekongi* and *S. intercalatum* are found focally in Southeast Asia and Central West Africa, respectively. Although it has a low mortality rate, schistosomiasis can be very debilitating. About 600 million people are thought to be at risk of schistosomiasis infection and 200 million are estimated to be infected in 74 countries (Chan *et al*, 1996). One hundred and twenty million people have symptoms of passing eggs in stool and urine. Twenty million have symptoms of stunted growth, anaemia, and chronic ill health. The current estimated total number of individuals with morbidity and mortality due to schistosomiasis infection in sub-Saharan Africa may be as high as 200 000 deaths per year due to non-functioning kidney and bladder cancer in respect of *Schistosoma haematobium* infection and haematemesis in case of *Schistosoma mansoni* infection.

Schistosomiasis is a chronic disease. The chronic forms of the disease are either intestinal or hepatosplenic. The intestinal form may have no symptoms, but blood in the

stools is the most common complaint in the absence of hepatosplenic involvement. Pathology of *S. mansoni* and *S. japonicum* schistosomiasis includes Katayama fever, hepatic perisinusoidal egg granulomas, symmers' pipe stem periportal fibrosis, portal hypertension, and occasional embolic egg granulomas in brain or spinal cord. Pathology of *S. haematobium* schistosomiasis includes haematuria, scarring, calcification, squamous cell carcinoma and occasional embolic egg granulomas in brain or spinal cord. Bladder cancer diagnosis and mortality are generally elevated in affected areas (Caatinga *et al*, 2000).

Many infections are subclinically symptomatic, with mild anaemia and malnutrition being common in endemic areas. Acute schistosomiasis (Katayama's fever) may occur weeks after the initial infection, especially by *S. mansoni* and *S. japonicum*. Manifestations include abdominal pain, cough, diarrhoea, eosinophilia, extremely high white blood cell count, fever, fatigue and hepatosplenomegaly. Occasionally central nervous system lesions occur. Cerebral granulomatous disease may be caused by ectopic *S. japonicum* eggs in the brain, and granulomatous lesions around ectopic eggs in the spinal cord from *S. mansoni* and *S. haematobium* infections may result in a transverse myelitis with flaccid paraplegia. Continuing infection may cause granulomatous reactions and fibrosis in the affected organs which may result in manifestations that include colonic polyposis with bloody diarrhoea (*Schistosoma mansoni* mostly), portal hypertension with haematemesis and splenomegaly (*S. mansoni*, *S. japonicum*); cystitis and ureteritis (*S. haematobium*) with haematuria which can progress to bladder cancer, pulmonary hypertension (*S. mansoni*, *S. japonicum*, more rarely *S. haematobium*), glomerulonephritis and central nervous system lesions (Caatinga *et al*, 2000).

Microscopic identification of eggs in stool or urine is the most practical method for schistosomiasis diagnosis. For the measurement of eggs in the faeces of presenting patients, the scientific unit used was eggs per gram of stool. Stool examination should be performed when infection with *S. mansoni* or *S. japonicum* is suspected and urine examination should be performed if *S. haematobium* is suspected. Eggs can be present in

the stool in infections with all *Schistosoma* species (Katz and Zicker, 1975). The examination can be performed on a stool smear (1 to 2 mg of faecal matter). Since eggs may be passed intermittently or in small amounts, their detection can be enhanced by repeated examinations and/or concentration procedures (such as the formalin-ethyl acetate technique). In addition, for field surveys and investigational purposes, the egg output can be quantified by using the Kato-Katz technique (20 to 50 mg of faecal matter) or the Ritchie technique. Eggs can be found in the urine in infections with *S. haematobium* at the recommended time of collection of between noon and 3 pm). Detection is enhanced by centrifugation and examination of the sediment. Quantification is possible by using filtration through a Nucleopore membrane of a standard 10 ml of urine followed by egg counts on the membrane. Investigation of *S. haematobium* should also include a pelvic x-ray as bladder wall calcification is highly characteristic of chronic infection. Tissue biopsy (rectal biopsy for all species and biopsy of the bladder for *S. haematobium*) may demonstrate eggs when stool or urine examinations are negative. The eggs of *S. haematobium* are ellipsoidal with a terminal spine, *S. mansoni* eggs are also ellipsoidal but with a lateral spine, *S. japonicum* eggs are spheroidal with a small knob (Katz and Zicker, 1975).

Schistosomiasis is treated using a single oral dose of the drug praziquantel. While praziquantel is universally used and it is safe and highly effective in curing an infected patient, it does not prevent re-infection by cercariae and is thus not an optimum treatment for people living in endemic areas. There is a second drug available for treating *Schistosoma mansoni* called oxamniquine. As with other major parasitic diseases, there is ongoing and extensive research into developing a vaccine that will prevent the parasite from completing its life cycle in humans (WHO, 1985).

The current means of controlling the disease is through chemotherapy, vector elimination, improved sanitation and health education. However, these measures are temporary and expensive and have not reduced the disease burden. In addition widespread use of chemotherapy requires continued repeated treatments and indefinite

surveillance. There is also the possibility of drug resistance (Joseph *et al*, 2004). The use of molluscicides is very expensive and may be detrimental to the environment hence the need to develop schistosomiasis vaccine to complement drug therapy. The encouragement towards the development of schistosomiasis vaccine is derived from the scientific findings that re-infection mediates partial immunity in human populations found in endemic areas therefore schistosomiasis vaccine should be developed which can reduce or delay morbidity after intermitted rounds of chemotherapy (Butterworth, 1994).

Prevention is by eliminating the water-borne snails which are natural reservoirs for the disease. This is usually done by identifying bodies of water, such as lakes and ponds which are infested by adding niclosamide, acrolein, copper sulfate or endod (*Phytolacca dodecandra*) to the water in order to kill the snails. In some cases, urbanization, pollution, and/or consequent destruction of snail habitat has reduced exposure, with a subsequent decrease in new infections (Jordan *et al*, 1993).

Hepatosplenic schistosomiasis is the most important clinical manifestation of *Schistosoma mansoni* infection (Bina, 1997). The hepatic lesion is caused by a granulomatous response to eggs of *S. mansoni* with subsequent periportal fibrosis, portal hypertension, splenomegaly, oesophageal varices, and recurrent haematemesis (Kloetzel, 1962; Arap *et al*, 1976; Sleight *et al*, 1985; Gryseels, 1991a; Abdel and Strickland, 1993). Development of hepatic granulomas is due to host immune cells monocytes, lymphocytes, neutrophils and eosinophils (MLNE) response to deposited schistosome eggs and is followed by fibrosis of periportal veins (Smithers and Doenhoff, 1982; Andrade *et al*, 1989; Boros, 1989).

Ultrasonography has become a valuable tool in the assessment of morbidity due to *S. mansoni* infection (Doehring *et al.*, 1992). It is safe, painless, non-invasive and superior to physical examination and is inexpensive, rapid, portable and simple to operate. It causes no biological hazards to the patient or operator. It has high sensitivity and

specificity of between 80 and 100 percent and it either complements or replaces many invasive techniques such as endoscopy, liver biopsy, pyelography, cystoscopy, angiography and other invasive techniques (Abdel-Wahab and Strickland, 1993). The classification adopted by the World Health Organization (Abdel-Wahab *et al.*, 1992) includes ultrasound image pattern degrees of hepatic fibrosis based on measurement of the periportal tract thickness.

Although there has been important success in the control of schistosomiasis in some countries like China and Brazil and some countries within Sub-saharan Africa like Tanzania, schistosomiasis has almost doubled in the last decade with the growing population (WHO, 1993), partly due to irrigation projects and migration of rural populations into urban areas. These and many other factors have led to schistosomiasis being recognized as an important public health problem in large cities of endemic countries (WHO, 1985). It is known that ecology, occupation and economic factors play important role in human schistosomiasis (Warren, 1973; Jordan and Webbe, 1982). Schistosomiasis being a behaviour-related disease it portrays complex interactions between human behaviour, social, economic, cultural beliefs and causes of the disease (Huang and Manderson, 1992).

Most of the available data on clinical pathology due to *Schistosoma mansoni* infection and its occurrence come from Brazil, where the disease has been extensively investigated (WHO, 1998).

The acute phase of schistosomiasis due to *Schistosoma mansoni* infection is associated with the onset of the female parasite laying eggs about five weeks after infection and granuloma formation around eggs trapped in the liver and intestinal wall with hepatosplenomegaly, leucocytosis and eosinophilia. This phase of infection is often asymptomatic. However, the clinical syndrome include fever, nausea, headache, chills, irritating cough and in extreme cases diarrhoea with blood and mucus. This occurs mostly in non-immune individuals frequently from urban areas exposed for the first time in endemic areas and symptoms last from a few weeks to several months. The chronic



phase of the disease which manifest a few years after infection are either intestinal or hepatosplenic. The intestinal form of schistosomiasis manifests itself a number of years after infection with blood in the stools as the most common complaint (Cheever and Duvall, 1982).

There is a cellular reaction with granuloma formation around eggs trapped in the tissues with subsequent fibrosis (Cheever and Duvall, 1982). All areas of both the small and large intestines may be involved but most severe lesions are seen in the large intestines and rarely in the small intestines even though large numbers of eggs may be deposited here. Colonic polyps are also sometimes seen. The hepatosplenic form of schistosomiasis again is seen a number of years after infection. The pathology is similar to that seen in the intestines. Most individuals with hepatosplenic schistosomiasis also have splenomegaly. However, hepatosplenic schistosomiasis can occur without enlargement of the spleen (WHO, 1998).

In *Schistosoma mansoni* infection the current estimated total number of individuals with morbidity and mortality in sub-Saharan Africa may be as high as 393 million people at risk of infection, 54 million people are infected, 8.5 million have hepatomegaly, 6.3 million have splenomegaly, 290 thousand have ascites, 93 thousand have haematemesis and 130 thousand die of haematemesis per year (Nokes *et al*, 1999). The current global disability-adjusted life years (DALYs) lost as a result of schistosomiasis infection stands at 4.5 million (WHO 2000). Tropical Africa is the most severely affected with 85% of the cases (WHO 1993).

In children, schistosomiasis causes growth retardation, anaemia, poor school performance, cognitive impairment and memory deficits (McGarvey *et al*, 1993; 1996). The highest intensities of infection are found in children between 5 to 16 years old (Bedwani *et al*, 1998). In adults the infection affects economic productivity (WHO 1993 and Nokes *et al*, 1999). Severity of schistosomiasis infection is attributed to host exposure to infection in various water bodies because of lack of access to safe water and sanitation and host immunity to challenging the infection (Butterworth, 1994). As a

behavior related disease, the risk of infection with schistosomiasis is associated with age, sex, and occupation of individuals (Gryseels, 1991b). Conditions responsible for disease severe morbidity such as liver disease are not completely understood although parasite burden seems to be a major determinant (Sleigh *et al*, 1986).

Determination of the costs arising from schistosomiasis infections, disability and death is a subject of continued research. Despite of the limited data collected on health statistics, conclusions can be drawn about the economic impact of schistosomiasis.

In the Philippines, the work days lost as a result of *Schistosoma japonicum* infection have been estimated up to 40 per infected person per year. In Ghana *Schistosoma haematobium* infection contributes to the loss of 4.4 work days per infected person per year. In Kenya and Zimbabwe, there is around 10% reduction in exercise performance in children with *Schistosoma mansoni* infection (WHO 1993).

There is a delay of 5 to 15 years from the time of *Schistosoma mansoni* infection to the development of severe disease. Hepatosplenomegaly develops in about 10% of the infected people. Liver disease, with oesophageal varices and bleeding is seen in varying degrees but affects up to 7% of the infected individuals in endemic areas, mostly those harbouring heavy worm loads (WHO, 1993).

*Schistosoma mansoni* infection has also been associated with *Salmonella* species and *Staphylococcus aureus* (WHO, 1998). These organisms are found in the tegument or the intestinal tract of adult schistosomes suggesting that schistosomiasis is a source or reservoir of other infections.

Studies have shown that adults have lower intensities of schistosomiasis infection than children, suggesting that resistance develop with age, manifesting around the age of puberty (Joseph *et al*, 2004).

A study was conducted in Brazil on 164 subjects to determine morbidity due to

*Schistosoma mansoni* infection. Parasitological examination detected 40% prevalence of *Schistosoma mansoni* infection. Geometric mean egg count (GMEC) ranged between 24 - 1784 eggs per gram of stool. Ultrasound examination detected 5% of subjects with normal liver, 64% with early periportal fibrosis (EPPF), 25% with moderate periportal fibrosis (MPPF) and 6% with advanced periportal fibrosis (APPF). Periportal fibrosis was validated by direct correlation between portal thickness (PT) and portal vein diameter, PT and spleen vein diameter, PT and spleen size (Amelia *et al*, 2000).

A study carried out in Egypt investigated 1480 subjects to determine the prevalence of morbidity associated with *Schistosoma mansoni* infection using physical, parasitological and ultrasound examinations. Parasitological examination detected 28% prevalence of *Schistosoma mansoni* infection. Geometric mean egg count (GMEC) was 81.3 eggs per gram of stool. Ultrasound examination detected 1.4% subjects with advanced periportal fibrosis. Splenomegaly and periportal fibrosis (PPF) correlated well with *Schistosoma mansoni* infection (Abdel-Wahab *et al*, 2000).

A survey on 792 subjects in Sudan was conducted to determine morbidity due to *Schistosoma mansoni* infection. Parasitological examination detected 70% prevalence of *Schistosoma mansoni* infection. Geometric egg count GMEC was less than 100 and averaged 34 and 38 eggs per gram of faeces. Ultrasound method detected 58% cases of early periportal fibrosis (EPPF), 9% cases of moderate MPPF and 3% advanced cases of APPF in individuals. Early PPF was detected in 50 -70% children and adolescents. Moderate PPF was detected in 45 -58% men between 21-30 years old. Advanced PPF with splenomegaly was detected in 6% of individuals, mostly adult men. These observations led to the suggestion that intensity of infection duration and gender are important in hepatic disease progression (Qurashi-Muhamed *et al*, 1999).

In Uganda, 460 subjects were examined for morbidity due to schistosomiasis. Parasitological examination detected 84% prevalence of *Schistosoma mansoni* infection. Geometric egg count (GMEC) was 81 eggs per gram of stool. Ultrasound detected 10% subjects with advanced periportal fibrosis APPF (Frenzel *et al.*, 1999).

In Zambia studies carried out in the Siavonga District by Mubila and Robinson (2002) and Chimbari *et al*, (2003) showed *Schistosoma mansoni* prevalence of 32% respectively. Four years later another study undertaken in the Game Village in the Siavonga District by the Schistosomiasis Control Initiative (2007) found the prevalence of *Schistosoma mansoni* infection at 77%.

## **1.5 Objectives**

### **1.5.1 General Objective**

To determine the prevalence of liver disease in *Schistosoma mansoni* infected school children and adults of the Game village in Siavonga District.

### **1.5.2 Specific Objectives**

1.5.2.1 To determine the prevalence and intensity of schistosomiasis infection in school children and adults of the Game village in Siavonga District.

1.5.2.2 To determine the prevalence and intensity of other possible hepato-splenomegaly confounding infections.

1.5.2.3 To determine the physical symptoms of schistosomiasis in school children and adults of the Game village in Siavonga District.

1.5.2.4 To determine liver disease in *Schistosoma mansoni* infection as observed by ultrasound examination in school children and adults of the Game village in Siavonga District.

1.5.2.5 To determine the epidemiological knowledge, attitudes and practices (KAP) of schistosomiasis in school children and adults of the Game village in the Siavonga District.

## **CHAPTER 2.0 MATERIALS AND METHODS**

### **2.1 Study Design**

This was a community-based cross-sectional study carried out from June to October 2007.

### **2.2 Study Site**

The study was conducted at a community school in the Game Village situated about 5 km away from the Siavonga District in the eastern direction on the shores of Lake Kariba. The village had no access to safe water and sanitation facilities. The source of water for various activities, including domestic and farming, was the Lake Kariba and the surrounding ponds where most of the women and children go to swim and do their gardening. This scenario exposed them to high risk of contracting schistosomiasis and other soil and water-borne diseases.

### **2.3 Study Population**

The study was carried out at the Game Village in the Siavonga District. The Siavonga District is situated 200 km away from Lusaka on the shores of Lake Kariba in the Southern Province of Zambia and had a population of about 73,000 people. The Siavonga District had about 22 government, community and private schools with a total enrolment figure of 3,630 school-children. The Siavonga District had 16 health centres, two hospitals, one situated at the Chirundu border-post and the other in Siavonga Town. Most of the activities of the majority of the people were fishing and subsistence farming as the soils are good and much of the land is bordered by the man-made Lake Kariba. Other people work in lodges and guest houses found on the shores of Lake Kariba.

### **2.4 Inclusion Criteria**

School-children between the ages of 5 and 15 years from the Game Community School were recruited onto the study. Also recruited were adults between 16 and 85 years old from the Game Village who consented to participate in the study. A total of 269 participants were recruited.

## **2.5 Exclusion Criteria**

Those excluded from participating in the study were children aged below 5 years, children not attending school, persons not willing to participate in the study, seriously sick patients including those with viral hepatitis, carcinoma and individuals who had received treatment for schistosomiasis and soil transmitted helminths in the last 6 months.

## **2.6 Ethical Approval and Informed Consent**

Ethical approval for this study was obtained from the University of Zambia Research Ethics Committee. Before recruitment of study participants in the Siavonga District, informed consent was obtained from parents and guardians after holding a meeting with them to explain why such a study was being carried out in their area. Written consent was also obtained from the Head Teacher of the School.

## **2.7 Determination of the Prevalence of *Schistosoma mansoni* Infection**

To determine the prevalence of *Schistosoma mansoni* infection in the study population, two stool containers were given to all the 269 study participants on the first day and were requested to submit one stool sample between 10 am and 2 pm to coincide with peak egg excretion in stool. The second stool sample was submitted the following day at the same time. Samples were processed the same day of collection using the Kato-Katz method (Katz *et al*, 1972). Stool samples were sieved through nylon screen 80 mesh and smeared over a plastic template. The template had a hole in the middle measuring 6mm in diameter and 1.5 mm in thickness. The plastic template was placed on a microscope slide and the hole in the middle measuring 6mm in diameter and 1.5 mm in thickness was filled with stool samples previously sieved through nylon screen 80 mesh to hold 41.7 mg of faeces. The plastic template filled with stool sample was gently removed from the microscope slide by lifting it leaving 41.7 mg of faeces on the slide. The faeces on the slide were covered with cellophane previously soaked in malachite green. The microscope slide was then over-turned onto a small piece of old news print paper. Pressure was applied on the opposite side of the microscope slide to spread faeces the microscope slide to make a thick stool smear. Two thick smears from each stool sample

were prepared for examination under a binocular microscope at X10 magnification. The prevalence of *Schistosoma mansoni* infection was determined by observation of specific *Schistosoma mansoni* eggs with a characteristic lateral spine on a thick smear of stool of each study participant.

### **2.8 Determination of the Intensity of *Schistosoma mansoni* Infection**

The intensity of *Schistosoma mansoni* infection was determined by counting the observed specific *Schistosoma mansoni* eggs on a Kato-Katz thick smear of stool of each participant. The number of eggs counted on each slide was multiplied by 24 in order to calculate the number of *Schistosoma mansoni* eggs per gram of faeces and were classified according to different intensity categories (WHO 2000) as light infection with egg counts between 1 - 99 eggs per gram of stool, moderate infection with egg counts between 100 – 399 eggs per gram of stool and heavy infection with egg counts greater or equal to 400 eggs per gram of stool as in Table 3. The geometric mean egg counts (GMEC) per gram of faeces for the total number of positives was calculated by adding up all individual *Schistosoma mansoni* egg intensities counted per gram of faeces of each slide and the totals were computed in Microsoft Excel Software using the formula:  $n\text{-th } \sqrt[n]{(x)(y)\dots(z)}$  where n-th represented the root of the total number of slides counted for intensities whereas (x)(y)...(z) represented the product of the total number of slides counted for intensities.

### **2.9 Determination of the Prevalence of *Schistosoma haematobium***

To determine the prevalence of *Schistosoma haematobium* infection in the study population of the Siavonga District, one 60ml urine sample container was given to all the 269 study participants on the first day and were asked to submit urine samples between 10 am and 2 pm to coincide with peak egg excretion for examination. Samples were processed within one hour of collection on the same day. Each urine sample collected was physically examined for visible haematuria. This was followed by dipping one strip of rapid Hemastix reagent (Bayer Diagnostics Europe Limited, Dublin, Ireland) to detect the presence of micro-haematuria. The urine samples were thoroughly mixed after which 10ml of urine was withdrawn from the container using a clean 10ml capacity

syringe. Urine in the syringe was filtered by passing it through a filter holder containing an isopore membrane filter (Vestergaard Frandsen Kolding, Denmark) which was fixed onto the syringe. The filter holder was later detached from the syringe, placed onto an adsorbent tissue and opened to remove the membrane filter from inside the holder. The membrane filter was then placed on a clean slide and examined on the microscope at X10 magnification. Specific *Schistosoma haematobium* eggs with a characteristic terminal spine of each study participant were observed in urine.

#### **2.10 Determination of the Intensity of *Schistosoma haematobium***

The intensity of *Schistosoma haematobium* infection was determined by counting the observed specific eggs with terminal spine in 10 ml of urine and was expressed as eggs per 10 ml of urine (WHO, 2000).

#### **2.11 Determination of the Prevalence and Intensity of Other Possible Hepato-splenomegaly Confounding Infections**

The prevalence and intensities of infections in Hookworms, *Ascaris lumbricoides*, and *Trichuris trichuria* were detected in stool by Kato-Katz method as in *S. mansoni* infection.

#### **2.12 Determination of the Physical Symptoms of Schistosomiasis.**

The physical symptoms of schistosomiasis in the study participants were determined by palpation of the liver and spleen. Two nurses with no previous knowledge of the parasitological results of the study participants conducted the physical examination. Fingers were used to palpate the liver and the spleen. One finger represented 2 cm of enlarged liver or spleen (WHO, 1991 and 2000).

#### **2.13 Determination of the Liver Disease caused by *Schistosoma mansoni* Infection**

To determine the features in the liver suggestive of liver disease caused by *Schistosoma mansoni* infection, a portable ultrasound machine ALOKA SSD-500 fitted with a 3.5 MHz convex probe transducer (Imai, Tokyo, Japan) and powered from a portable generator was used by two independent qualified and experienced ultrasonographers to



examine all the study participants using the method previously described (Dittrich *et al*, 1983). Individuals were required to lie on the examination couch with the abdomen area exposed for scanning. Ultrasound gel was applied directly onto the abdomen area and the scanning probe moved across the abdomen for 5 - 10 minutes per individual (WHO, 2000).

#### **2.14 Determination of the Epidemiological Knowledge, Attitudes and Practices of Schistosomiasis**

*Schistosoma mansoni* is clinically known to induce blood in stool, abdominal pains, and diarrhoea and hepatosplenic involvement usually in chronic cases. Epidemiological knowledge, attitudes and practices of schistosomiasis infection among school children and adults in Siavonga District were determined by using a standard knowledge, attitudes and practices (KAP) questionnaires which were administered to the community by teachers and local social health workers to help with data collection. Individual participants were asked questions (Appendix I). The KAP questionnaires were translated in the local language which participants understood best. The questionnaires were administered to all the study participants in order to evaluate their understanding of the different aspects of the disease. At the end of the exercise, members of the research team collected all KAP questionnaire forms from individual participants to evaluate the answers.

#### **2.15 Data Analysis**

Analysis of data for this study was conducted using Microsoft Excel Software, SPSS version 10.0, Epi Info version 3.3.2. Confidence Intervals (CI) at 95% for prevalence and intensity were computed in Excel using the formula:  $P \pm 1.96 \sqrt{(PQ/n)}$  where P was prevalence,  $Q = 100 - P$  and n = sample size. The Confidence Intervals (CI) for geometric mean egg counts for infection intensities were computed in Excel using the formula:  $\bar{A} \pm 1.96 (STDEV / \sqrt{n})$  where  $\bar{A}$  was the geometric mean egg count intensity, STDEV was the standard deviation of the sample variables and  $\sqrt{n}$  was the square root of the sample size (Douglas, 1991).

Index of the liver size (ILS) was computed using the formula  $ILS = (PSL^2 + MCL^2 + MAL^2) \times 0.2618$  where PSL was the measurement of parasternal length, MCL was the measurement of mid clavical line, and MAL was the measurement of mid axillary line (SCI, 2007).

## **CHAPTER 3.0 RESULTS**

### **3.1 Study Population**

Game Village had a population of about 3,600 people with one community school and one health centre. The school had classes ranging from grades 1 to 6 and had an enrolment figure of 247 school children comprising (117 or 47.4%) boys and (130 or 52.6%) girls. Pupils in grade 1 were (50 or 20.2%), (36 or 14.6%) pupils were in grade 2, (58 or 32.5%) pupils were in grade 3, (35 or 14.2%) pupils were in grade 4, (26 or 10.5%) pupils were in grade 5 and (42 or 17%) pupils were in grade 6.

There were 269 individuals from the Game Village recruited to participate in the study. Females comprised (149 or 55.4%) and males comprised (120 or 44.6%). There were (148 or 55%) school children between the ages of 7 and 15 years who were recruited to participate in the study, and these comprised (76 or 51.3%) females and (72 or 48.7%) males. There were (121 or 45%) adults between the ages of 16 and 85 years. Female adults comprised (73 or 60.3%) whereas male adults comprised 48 or 39.7%. More children (148 or 55%) participated in the survey than adults (121 or 45%) because adults were probably engaged in occupation, such as in government offices and fishing on Lake Kariba. Children were easy to recruit onto the study because they were found at school most of the time (Table 1).

Table 1: Distribution of the Study Population according to Age groups and Sex.

	<b>School-children</b>		
<b>Age groups (yrs)</b>	<b>Females</b>	<b>Males</b>	<b>Total</b>
7 – 9	27	25	52
10 – 12	29	27	56
13 – 15	20	20	40
<b>Sub Total</b>	<b>76 (51.3%)</b>	<b>72 (48.7%)</b>	<b>148 (55%)</b>
	<b>Adults</b>		
16 – 25	29	21	50
26 – 35	21	8	29
36 – 45	12	8	20
46 – 55	4	5	9
56 – 65	4	3	7
65 - 85	3	3	6
<b>Sub Total</b>	<b>73 (60.3%)</b>	<b>48 (39.7%)</b>	<b>121 (45%)</b>
<b>Grand Total</b>	<b>149 (55.4%)</b>	<b>120 (44.6%)</b>	<b>269</b>

### 3.2 Prevalence and Intensity of *Schistosoma mansoni* Infection

#### 3.2.1 Prevalence of *Schistosoma mansoni* Infection

The prevalence of *S. mansoni* infection in the study population of Game Village as determined by the Kato-Katz method (Katz *et al*, 1972) was found to be (175 or 65%) with 95% CI of (59.3 – 70.7). Infected females and males were (93 or 34.5%) and (82 or 30.5%), respectively. The infection rate was double (119 or 44.2%) in school children than in the adults (56 or 20.8%). The infection rates among female and male school children were comparable, (61 or 22.6%) and (58 or 21.6%), respectively. More adult females than adult males were infected, (32 or 11.9%), and (24 or 8.9%), respectively (Table 2).

Table 2: Prevalence of *Schistosoma mansoni* infection by sex in school children and adults of Game Village, Siavonga

Sex	Study population N = 269		School children n = 148		Adults n = 121	
	Pos	Neg	Pos	Neg	Pos	Neg
Females	93 (34.5%)	56 (20.8%)	61 (22.6%)	15 (5.6%)	32 (11.9%)	41 (15.2%)
Males	82 (30.5%)	38 (14.1%)	58 (21.6%)	14 (5.2%)	24 (8.9%)	24 (8.9%)
Total	175 (65%)	94 (34.94%)	119 (44.2%)	29 (10.8%)	56 (20.8%)	65 (24.16%)

The highest prevalence rate of *S. mansoni* infection was noted in school children aged 10 to 12 years. Older children from 13 to 15 years and young adults from 16 to 25 years of age showed identical prevalence rates of *S. mansoni* infection among the participants studied. Thereafter the trend in infection rates declined with increasing age (Figure 1).

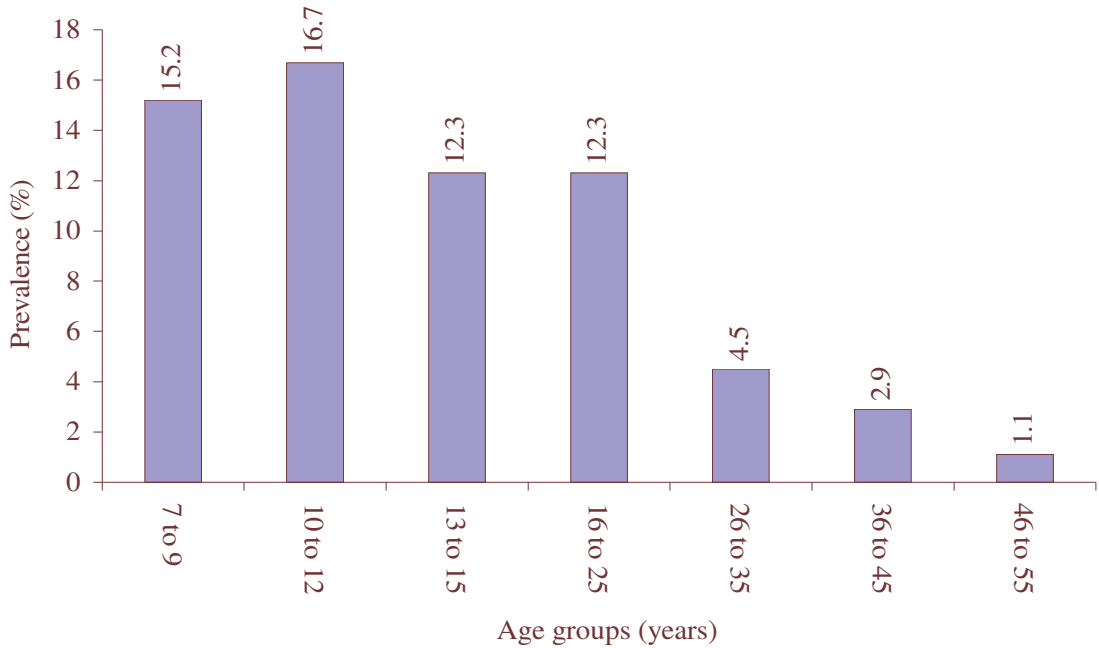


Fig 1: Prevalence of *S. mansoni* infection in the study population of Game Village, Siavonga by age groups.

The highest prevalence rate of *S. mansoni* infection was observed in females aged between 10 to 12 years. Older children from 13 to 15 years and young adults from 16 to 25 years of age showed the same prevalence rates of *S. mansoni* infection in both female and male. Among the adults, the prevalence rate of *S. mansoni* infection was lowest in older adults between 46 to 55 years of both sexes. It was also consistently observed that more females than males in all the age groups were infected except in the ages of 13 to 25 years (Figure 2).

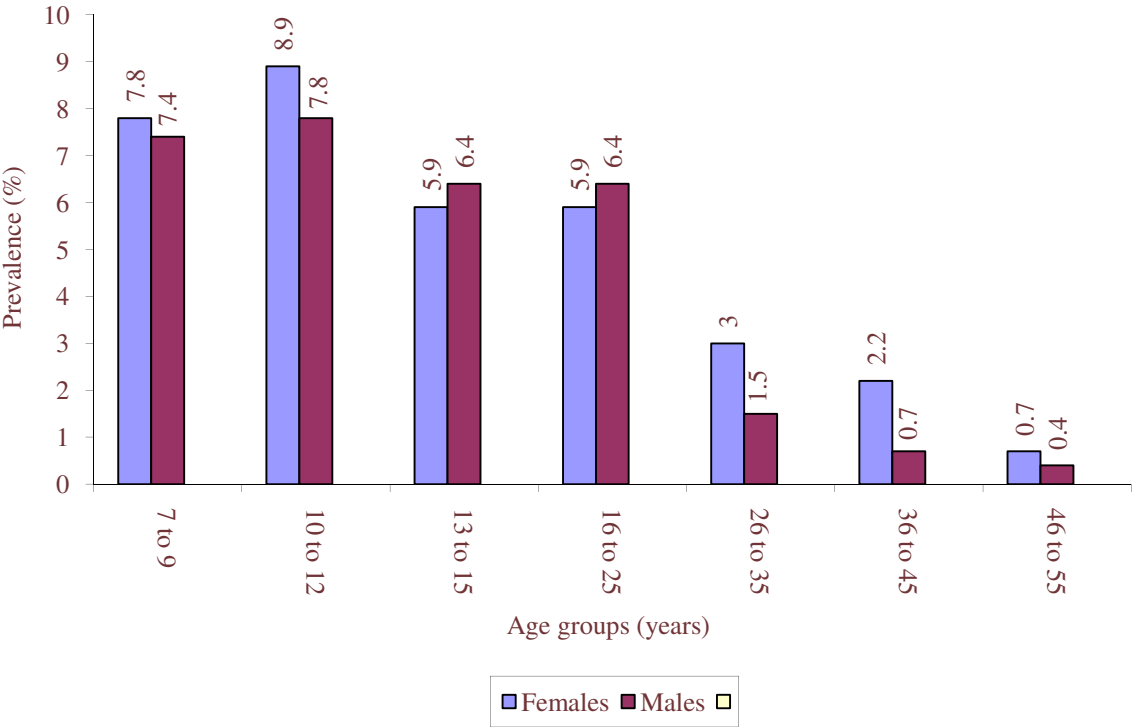


Fig 2: Prevalence of *S. mansoni* infection in the study population of Game Village by age group and sex.

### 3.2.2 Intensity of *Schistosoma mansoni* Infection

The 175 individuals found infected with *S. mansoni* were grouped into different categories of intensities of infection according to WHO, (2000) classification. The Kato-Katz method was employed to determine the intensity of infection in the 175 positive cases of *Schistosoma mansoni* infection found in Game Village of Siavonga district. Light, moderate, and heavy infection intensities were recorded in 61.2%, 33.7%, and 5.1% of the cases, respectively. It was also found that, among the 119 infected school children, 40.6% had light infection intensities, 23.4% and 4% showed moderate and heavy intensities of infections, respectively. Of the 56 infected adults 20.6%, 10.3%, and 1.1% had light, moderate and heavy intensities of infections, respectively. The findings show that only 9 or 5.1% cases had heavy intensities of *Schistosoma mansoni* infection (Table 3).

Table 3: Intensity of *Schistosoma mansoni* infection in the study population of Game Village, Siavonga.

WHO Category of Intensity	Positives in study population N == 175		Positives in school children n = 119		Positives in adults n = 56	
	Positives	% pos	Positives	% pos	Positives	% pos
Light Intensity (1 – 99 eggs per gram of stool)	107	61.2%	71	40.6%	36	20.6%
Moderate Intensity (100 – 399 eggs per gram of stool)	59	33.7%	41	23.4%	18	10.3%
Heavy Intensity (> or = 400 eggs per gram of stool)	9	5.1%	7	4%	2	1.1%



The mean egg counts per gram faeces were highest in males between 13 and 25 years of age. The lowest mean egg counts were observed in adults aged 26 to 45 years, particularly in the males who also had the lowest infection intensities. The egg output in the age groups between 46 and 55 years, was not different from that of the young study participants below the ages of 26 years in both females and males. The geometric mean egg counts (GMEC) was found to be 63.8 per gram of faeces for the 175 *S. mansoni* infected study participants (Figure 3).

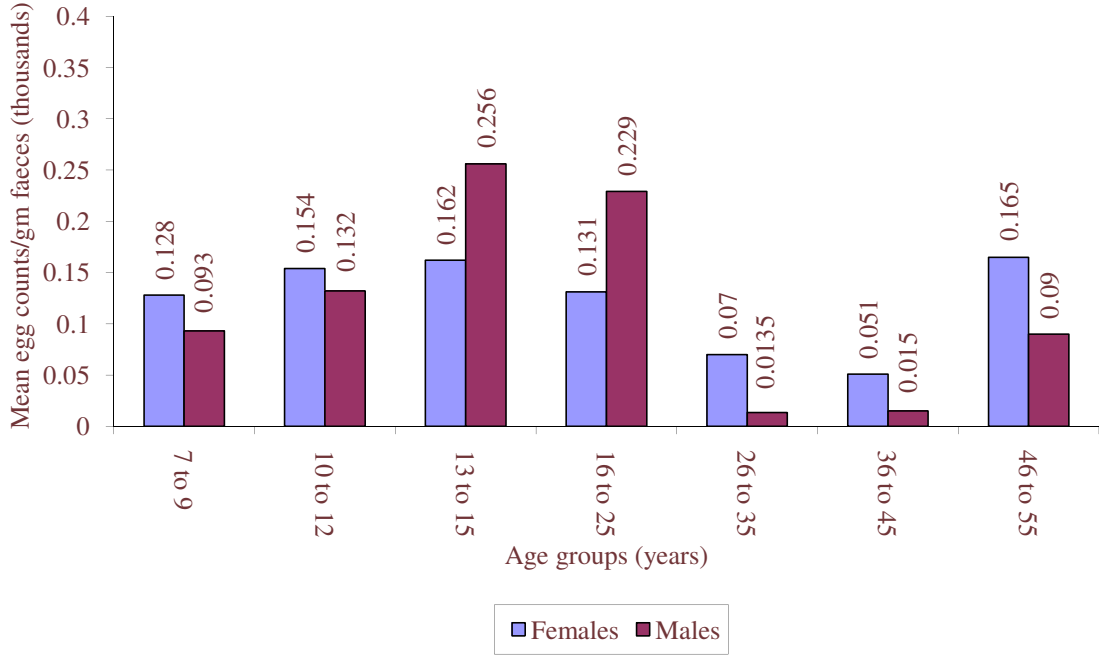


Fig 3: Intensity of *S. mansoni* infection by age group and sex in the study population of Game village, Siavonga.

More male school children than female school children were moderately infected. In general, the infection intensities in the females were more than those of the males in light infection categories in both school children and adults (Figure 4).

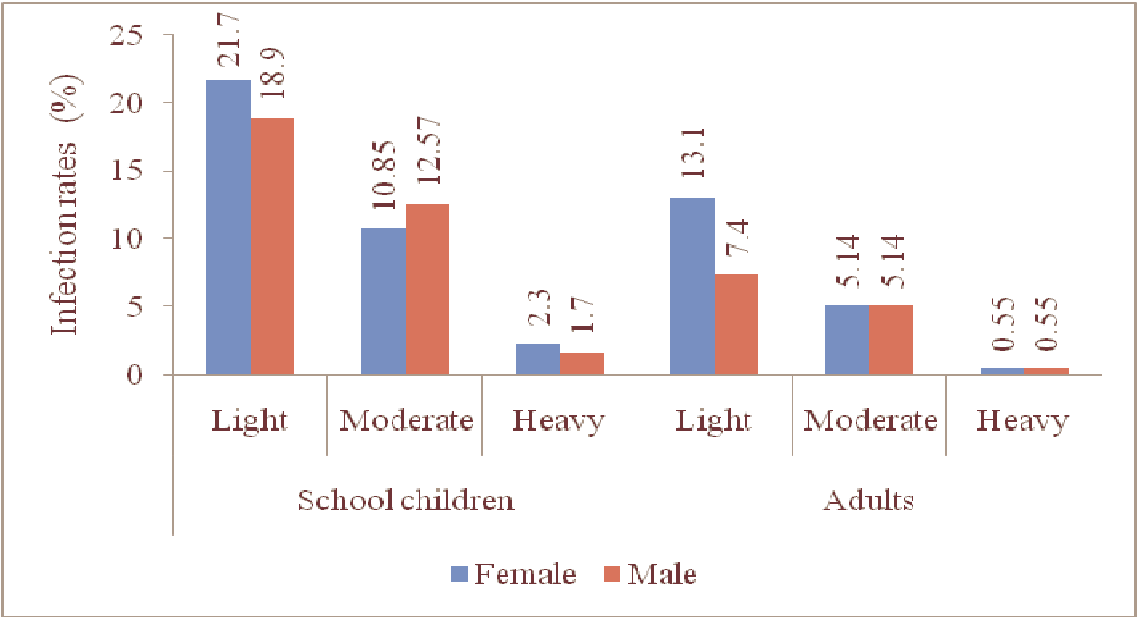


Fig 4: Infection rates of different categories of intensities in school children and adults of Game Village, Siavonga by sex.

### 3.3 Prevalence and Intensity of *S. haematobium* Infection

#### 3.3.1 Prevalence of *S. haematobium* Infection

The overall prevalence of *S. haematobium* infection in the study population as determined by microscopic examination of 10 ml of urine was found to be 21/269 or 7.8% with 95% CI of (4.4 – 10.6). The prevalence of *S. haematobium* infection in the study population as determined by rapid Hemastix reagent strips for haematuria was found to be 61/269 or 22.7%, higher than that found by microscopy, although no single urine sample showed visible haematuria.

#### 3.3.2 Intensity of *S. haematobium* Infection

The study showed that all the 21 cases of *S. haematobium* infection were of the light category with egg counts of less than 50 eggs per 10 ml of urine according to the WHO

classification of infection intensities in urinary schistosomiasis (WHO, 2000). The geometric mean egg count (GMEC) was found to be 4.98 eggs per 10 ml of urine.

### **3.4 Prevalence and Intensity of Other Possible Hepato-splenomegaly Confounding Infections.**

Confounding infections that are possible causes of Hepato-splenomegaly, such as hookworms (*Necator americanus* and *Ancylostoma duodenale*) and round worms like *Ascaris lumbricoides* and *Trichuris trichiura* were determined by the Kato-Katz method. Malaria parasites of *Plasmodium falciparum* infection from the finger prick blood of study participants were determined by examination of the Giemsa stained blood slides.

#### **3.4.1 Prevalence and Intensity of Hookworm Infections.**

The prevalence of hookworm infection was determined in all the study participants but was noticed in the age group between 7 and 15 years and was found to be (4.1%) or 11/269 with 95% CI of (1.7 – 6.5). Of the hookworm infected participants, 4 were single hookworm infections, 4 were co-infections with *S. mansoni*, and 1 was co-infected with *S. haematobium*. Multiple infections with hookworm, *S. mansoni* and *S. haematobium* were recorded in 2 individuals both males aged 11 and 13 years.

The intensity of hookworm infection was found to be 7/11 or 64% classified as light infection with egg counts between 1 – 1,999 eggs per gram of stool and 4/11 or 36% classified as moderate infection with egg counts between 2,000 – 3,999 eggs per gram of stool (WHO, 1985). The geometric mean egg count (GMEC) of hookworm infection was found to be 44 eggs per gram of faeces.

#### **3.4.2 Prevalence and Intensity of *Ascaris lumbricoides* Infection.**

The prevalence of *Ascaris lumbricoides* infection in the study population was found to be (17/269 or 6.3%) with 95% CI of (3.4 – 9.2). Single infections of *A. lumbricoides* were found in 3 cases. Co-infection with *S. haematobium* was found in 1 case. Co-infections with *S. mansoni* were found in 10 cases and multiple infections with, *S. mansoni* and *S. haematobium* were found in 3 individuals. There were no co-infections

of *A. lumbricoides* and *T. trichiura* recorded, and neither were there co-infections recorded between hookworm and the *Ascaris lumbricoides* or *Trichuris trichiuria*. Co infections of *S. mansoni*, *S. haematobium* were not recorded.

The intensity of *A. lumbricoides* infection was found to be (13/175 or 7.43%) light infection with egg counts between 1 – 4,999 eggs per gram of stool, and (4/175 or 2.3%) were moderate infections with egg counts between 5,000 – 49,999 eggs per gram of stool. No heavy infections were recorded. The geometric mean egg count (GMEC) of *A. lumbricoides* infection was found to be 542 eggs per gram of faeces.

### **3.4.3 Prevalence and intensity of malaria infection caused by *Plasmodium faciparum***

Malaria infection caused by *Plasmodium faciparum* parasites were also examined by Giemsa method in blood of study participants by preparing thick blood smears on slides. Blood slides were fixed by air drying, and then stained by dipping in Giemsa stain for 30 minutes. After staining, they were briefly rinsed in plain water and air dried again before examining under the microscope using standard operating procedures without the microscopists knowing the status of the study participants. No single participant was found with malaria infection.

Haemoglobin concentration of study participants were measured with a portable HemoCue kit. (HemoCue, Angelholm, Sweden). Blood samples were collected from finger pricks by drawing drops of blood using microcuvettes which were inserted into the HemoCue haemoglobin machine for haemoglobin readings. Guidelines on anaemia levels of human subjects in general were obtained from World Health Organization recommended values (WHO, 1996). For children less than 11 years, anaemia was defined as haemoglobin less than 115 grams per litre of blood. For children aged between 12 and 14 years, anaemia was defined as haemoglobin less than 120 grams per litre of blood.

### **3.5 Physical Symptoms of Schistosomiasis**

Physical symptoms of schistosomiasis in the study participants were determined by physical medical examination, and liver and spleen palpation and did not reveal any overt morbid features of *S. mansoni* infection except in one individual where splenomegaly was observed.

One individual (Case 1) was found with the spleen mid clavical line (MCL) size of 10 cm outside the normal (MCL) spleen size of 0 cm and the spleen mid axillary line (MAL) size of 12 cm outside the normal (MAL) spleen size of 0 cm respectively.

Another individual (Case 6) was found with the spleen mid clavical line (MCL) and mid axillary line (MAL) sizes of 16 cm and 18 cm, respectively. Both were outside the normal (MCL) and (MAL) spleen sizes of 0 cm (Table 4).

### **3.6 Liver disease in *Schistosoma mansoni* infection as observed by Ultrasound examination**

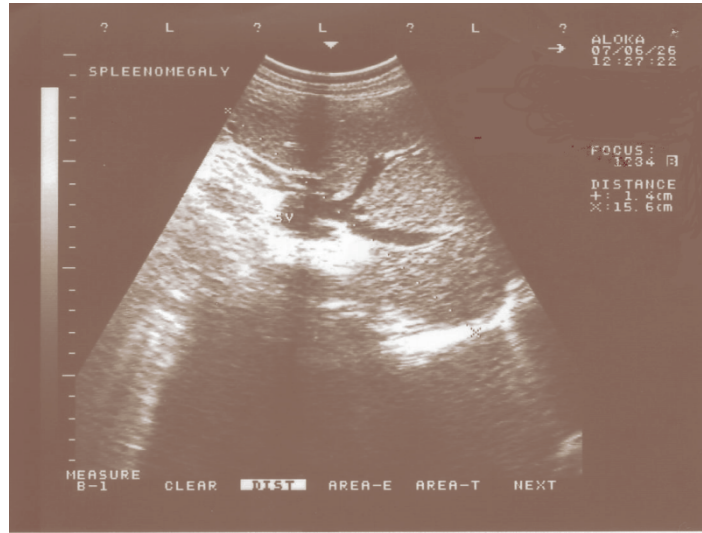
All study participants were examined for liver damage by use of ultrasound machine. The study showed that there were 6 individuals or 3.4% of the infected study participants who had liver damage. Ninety eight percent or 263 study participants showed normal liver condition as observed by ultrasound examination. The 6 individuals found with abnormal liver also had *Schistosoma mansoni* infection and were aged between 12 and 24 years of age, comprising 4 females and 2 males. Four of the 6 study participants with liver disease were school children between 12 and 15 years of age, and 2 were young adults as summarised (Table 4).

The intensity of the infection did not appear to influence the development of liver disease as the cases found were recorded in all categories of intensity of *Schistosoma mansoni* infection such as 2 cases with light infection, 3 cases with moderate infection, and 1 case with heavy infection. Co infections were absent in all but one (Case 6), suggesting that co-infections were not a confounding factor in this study.

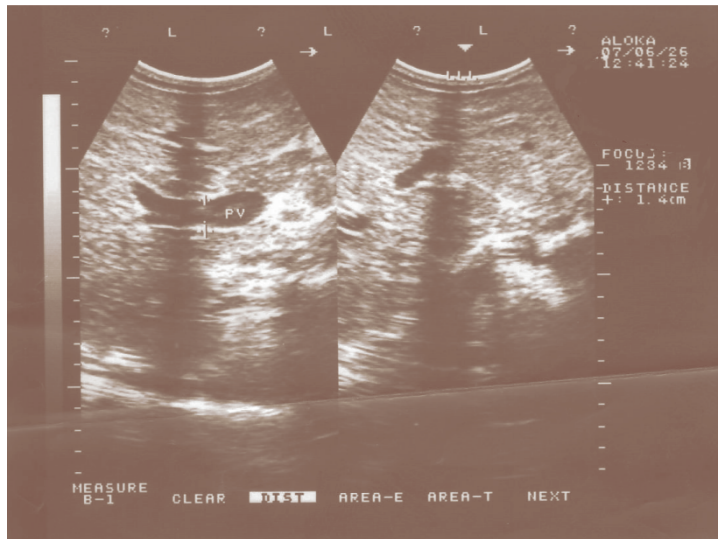
Table 4: Summary of *S. mansoni* cases with features of liver disease

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Age	18	15	15	24	12	14
Sex	F	M	F	M	F	F
<b>Microscopy</b>						
Intensity	Moderate 384 epg	Moderate 198 epg	Light 72 epg	Moderate 198 epg	Light 30 epg	Very heavy 1404 epg
<i>S.haematobium</i> co-infection	No	No	No	No	No	Yes
Hookworm	No	No	No	No	No	No
Ascaris	No	No	No	No	No	No
Trichuris	No	No	No	No	No	No
Malaria	No	No	No	No	No	No
Hb – level g/l	68 g/l	41 g/l	89 g/l	144 g/l	126 g/l	109 g/l
<b>Physical Examination</b>						
Splenomegaly	Yes	No	No	No	No	No
<b>Ultrasound Examination</b>						
Liver parasternal line	9.1 cm	12 cm	8.1 cm	8.4 cm	8.5 cm	11.6 cm
Portal vein diameter	14 mm	14 mm	11 mm	13 mm	8 mm	9 mm
Portal systemic collaterals	Yes	No	No	No	No	No
Periportal fibrosis	No	No	No	Yes	Yes	No
Pipe stems	No	No	No	Yes	Yes	Yes
Spider thickening	Yes	Yes	Yes	No	No	Yes
Prominent peripheral rings	Yes	No	No	Yes	Yes	Yes
Ruff	No	Yes	Yes	Yes	Yes	Yes
Patches	Yes	No	No	No	No	No

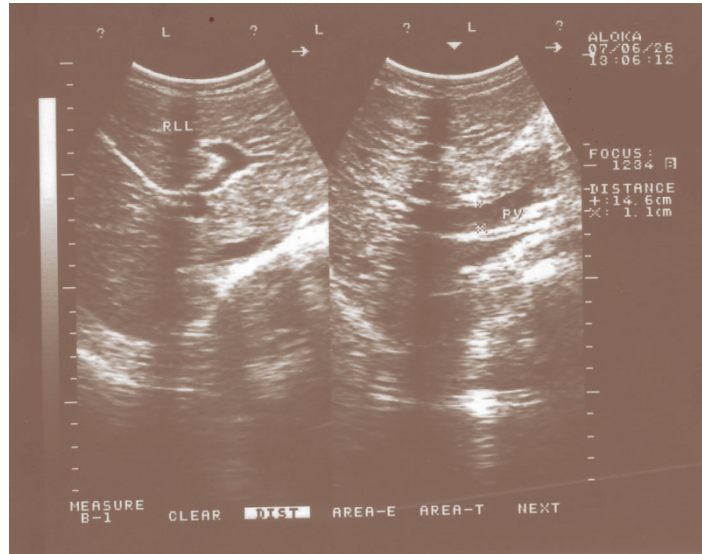
Case 1



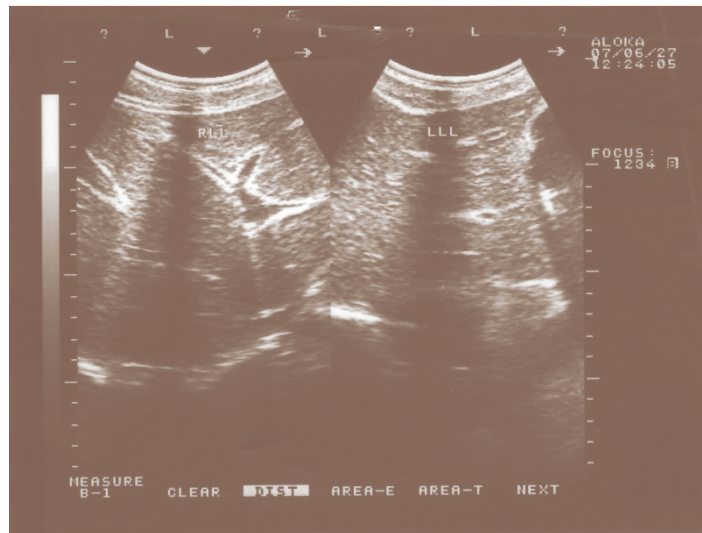
Case 2



Case 3

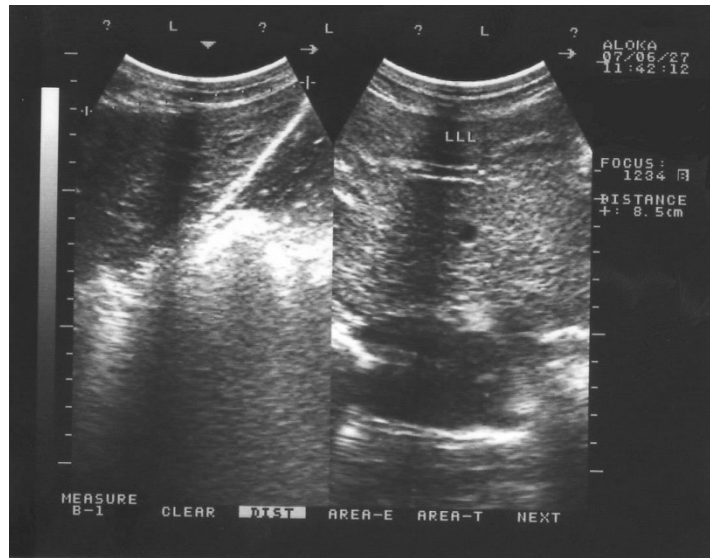


Case 4

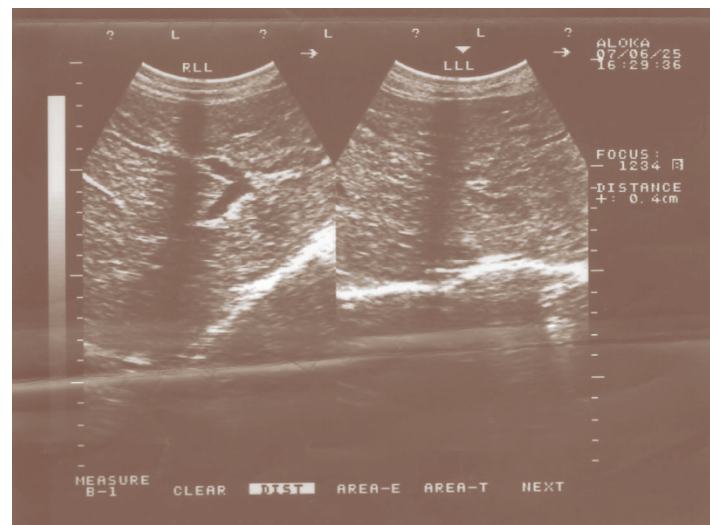




Case 5



Case 6



### 3.7 Anaemia

Haemoglobin levels of study participants were measured with a portable HemoCue kit (HemoCue, Angelholm, Sweden) to determine anaemia. Haemoglobin levels of 115 grams to 180 grams per litre of blood were considered not anaemic according to the guidelines on anaemia (WHO, 1996).

Anaemia was defined as haemoglobin levels less than 115 grams per litre of blood. Based on the (WHO, 1996) guidelines, anaemia was found in 70 individuals or 26% of the study population. There were more cases of anaemia in school children with *S. mansoni* infection (52/269 or 19.3%) in both sexes than in *S. mansoni* infected adults (18/269 or 6.7%) suggesting that more school children had low haemoglobin levels in blood than adults. More cases of anaemia were observed in male adults without *S. mansoni* infection (4/269 or 1.48%) than in male school children 3/269 or 1.1% (Table 5). Among the 6 individuals with *S. mansoni* infection and features of liver disease, 4 had anaemia with haemoglobin levels of less than 115 g/l of blood and comprised 3 females aged 14 years, 15 years, 18 years and 1 male aged 15 years, suggesting that more females were anaemic than males.

Table 5: Summary of anaemia cases with Hb less than 115 g/l of blood.

<i>S. mansoni</i> Infection	School children		Adults		Totals
	Females	Males	Females	Males	
Positives	23	19	6	1	49
Negatives	7	3	7	4	21
Totals	30	22	13	5	70

### 3.8 Knowledge, Attitude and Practices (KAP) of Schistosomiasis

All study participants were administered with a structured questionnaire on knowledge, attitudes and practices (KAP) to determine the epidemiological knowledge of schistosomiasis in the study population. Among the respondents 157 or 58.4% indicated having known what bilharzia was, 135 or 50.2% reported having known what causes

bilharzia and had been treated for schistosomiasis at least once but did not know when they were treated, 61 or 26.6% said that they knew the symptoms of bilharzia. Regular abdominal pains (144 or 53.5%) were the most overt symptom reported among the participants. Some participants (53 or 19.7%) indicated not being sure of how bilharzia is prevented, 20 or 7.4% reported to perceive that bilharzia was a sexually transmitted disease, 144 or 53% reported having toilets both at school and at home. About 160 or 59.4% said that they used wells as source of drinking water but used stream water for bathing. It was further observed that majority of the study participants (27 or 10%) had lived in Game Village for 10 years. Others (18 or 6.7%) age 7 and 15 years had lived in the Village since birth (Appendix I).

## CHAPTER 4.0. DISCUSSION

More children participated in the survey than adults because adults were occupied with other work like working in government offices and fishing in Lake Kariba. Children were a captive audience because they were found at school at specific times, and that schools participate in the School Health and Nutrition Programmes where the children could be examined.

Results showed that more than double the number of school children were infected with *Schistosoma mansoni* than adults probably because children between the ages of 5 to 16 years old have more water contact behaviour than adults since this disease is waterborne (Bedwani *et al*, 1998). It is also observed that more females than males were infected with *Schistosoma mansoni* because females spent more time in infested water performing domestic chores as was indicated during verbal discussions, unlike their male counterparts who spent more time at work in government offices and fishing in the waters of Lake Kariba.

In the present study, carried out over a period of four months in a population of 3,600 of Game village of Siavonga, 269 study participants were recruited. The 65% prevalence of *S. mansoni* infection found in Siavonga's Game village was lower than the 77% that was previously determined in the unplanned settlement, an area very close to the Game village (SCI, 2007). This reduction in the prevalence could be attributed to the Schistosomiasis Control Initiative programme activities that were introduced in 2005 to 2007 in the area.

It was observed that children between the ages of 7 to 15 years and the young adults between 16 to 25 years were more infected than adults aged between 26 to 55 years in both sexes. There was no difference in the prevalence of *S. mansoni* infection between female and male school children probably because children of both sexes were exposed to the infected water at equal frequencies. The highest prevalence rate of *S. mansoni* infection was in school children aged 7 to 12 years old.

It was also observed that more females were infected than males in all the age groups except the age 13 to 25 years old where more males than females were infected. The prevalence rate of *S. mansoni* infection sharply declined with age after the age of 26 years. Most infected participants (61%) were classified with light intensity infections, but only 5.1% carried heavy infections which could be attributed to the anti schistosomiasis control activities in the area, including deworming projects. Among the adults, the prevalence rate of *S. mansoni* infection was lowest in older adults between 46 to 55 years of both sexes. It was also consistently observed that more females than males in all the age groups were infected except in the ages of 13 to 25 years.

The contribution of immunity to infection rates in schistosomiasis has not been well explained. Existence of immunity against schistosomiasis is likely to be partial and probably does not play an important role in controlling the prevalence and intensity of infection (Smithers, 1976). This would explain why prevalence does not completely start to diminish until after the age of 26 years as seen in this study. Survival of populations in endemic areas largely depends on innate immunity where only a small proportion of an infecting inoculum of cercariae develop into adult worms, also occurrence of non-specific immunity and occurrence of immunoregulatory factors that suppress immunopathology and the disease (Warren, 1973), which partly explains the low morbidities experienced in this study

The mean egg counts per gram faeces were highest in males between 13 and 25 years of age and the lowest in adults aged 26 to 45 years. The most likely explanation would be that younger participants spend more time in infected water than the older age group which make up the main national workforce in offices and activities not related to water.

The egg output in the oldest group 46 to 55 did not differ from the younger generation, but substantially higher than young adults aged 26 to 36 years. This result can be best understood in the context that people retire early from employment in this country, and the aging population tends to turn to the old fashioned way of making a livelihood, such as fishing. Most elderly people are not in formal employment and would thus spend

more time in water related activities as it is known that worm burdens are dependent upon the rate of exposure to contaminated water. This could explain why 2 of the 9 heavily infected participants, were in the age group of 46-55 years. Another explanation would be loss of partial immunity with increased age and loss of passive immunity a finding that can be attributed to increase in age by children (Warren *et al*, 1972).

The physical symptoms of schistosomiasis in the study participants showed that 2 or 0.74% individuals had splenomegaly. This was consistent with the Uganda study findings were ultrasound detected up to 10% subjects with advanced periportal fibrosis APPF (Frenzel *et al.*, 1999). Hookworm and *Ascaris lumbricoides* infections found showed that these infections can occur in association with schistosomiasis and could influence the development and progression of liver disease due to schistosomiasis.

There were more cases of anaemia in school children with *S. mansoni* infection (19.3%) in both sexes than in *S. mansoni* infected adults (6.7%) indicating that more school children were anaemic than adults in this study.

Of the 175 *S. mansoni* infected participants, 6 cases or 3.4% were found with features of liver disease as observed by ultrasound examination. Four cases were females and 2 cases were male adults. Among the 2 adult cases, one case was male without anaemia while the other case was female with anaemia of haemoglobin level less than 115 g/l of blood. Among the 4 cases of school children, 1 case was male and anaemic, 3 cases were female of which 2 cases were anaemic and 1 case was without anaemia. The high number of anaemia cases found among females of the 6 cases could probably be attributed to the fact that females were in their early years of teenage life after puberty and could have been menstrating hence the low haemoglobin levels as no single person was found with malaria parasites in their blood by Giemsa method.

Epidemiological knowledge of schistosomiasis in the study population was assessed by knowledge, attitudes and practices (KAP) structured questionnaire and yielded important results which showed that study participants had a fair knowledge of

schistosomiasis.

Age, as observed in most schistosomiasis surveys is a major determinant of schistosomiasis infection. Children between 10 to 15 years were thought to be at the highest risk of infection (Sama and Ratard, 1994). This fact has equally been highlighted in this study where it is also observed that the prevalence of *Schistosoma mansoni* infection was high in females in both school children and adults. This observation could probably be attributed to the increased water contact activities as a result of domestic chores females perform.

The present study showed that of the 175 *S. mansoni* infected participants (6 or 3.4%) of the study participants in the Game village had suspected liver disease as determined by clinical, parasitological and ultrasound examinations. This shows less liver disease than that reported (WHO, 1993) where it was shown that hepatosplenomegaly developed in about 10% of *Schistosoma mansoni* infected people. Liver disease, with oesophageal varices and bleeding develops in up to 7% of the *Schistosoma mansoni* infected individuals in endemic areas, mostly those harbouring heavy worm loads (WHO, 1993). However, this study noticed 15 cases of individuals vomiting blood although no oesophageal varices were recorded. The revelations of this study result has a positive implication to the community of Game village in the sense that people of this area now will be more careful with their way of living. On the contrary, the result has a negative implication on the part of health care providers. It clearly shows that not much is being done to educate the community on health care matters related to usage of untreated water from unprotected wells and streams for domestic purposes and encouraging people to seek health care from hospitals or clinics once strange symptoms are noticed.

## **CHAPTER 5.0 CONCLUSION**

This study has concluded that *S. mansoni* was the most prevalent infection found in Game Village, Siavonga. It was also concluded that children have a higher chance of contracting schistosomiasis than adults. It was further concluded that females have a higher chance of contracting schistosomiasis because of the nature of activities they perform than males. The 3.4% liver disease found due to *S. mansoni* infection was observed in the young between the ages 12 to 24 years old and was regardless of the intensity of infection.



## **CHAPTER 6.0 RECOMMENDATIONS**

Intensive health education and provision of quality health care facilities, good housing, and clean water to the community is recommended. Efforts must continue to persuade stakeholders, non-governmental organizations and donors to fund operations towards school health and nutrition programmes.

## CHAPTER 7.0 REFERENCES

Abdel-Wahab, M.F., G. Esmat., A. Farrag., Y.A. el-Boraey. and G.T. Strickland. 1992. "Grading of hepatic schistosomiasis by the use of ultrasonography". American Journal of Tropical Medicine and Hygiene., 46, 403–408.

Abdel-Wahab, M.F., G. Esmat., E. Medhat., S. Narooz., I. Ramzy., Y. EI-Boraey. and G.T. Strickland. 2000. "The epidemiology of schistosomiasis in Egypt". Menofia Governorate. American Journal of Tropical Medicine and Hygiene., 62, 28 – 34.

Abdel-Wahab, M.F. and G.T. Strickland. 1993. "Abdominal ultrasonography for assessing morbidity from schistosomiasis". Transaction of the Royal Society of Tropical Medicine and Hygiene., 87 , 135-7.

Amelia, Ribeiro. De Jesus., Delfin, Gonzalez.Miranda., Roberval, Gonzalez.Miranda., Ilma, Arau´ Jo., Andre´A, MagalhaˆEs., Marcus, Bacellar and Edgar, M.Carvalho. 2000. "Mobidity associated with *S. mansoni* infection determined by ultrasound in an endemic area of Brazil, Caatingi Do Maura." American Journal of Tropical Medicine and Hygiene., 63, 1– 4.

Andrade, A.N., E.A.E. Van Mark. and Bastos, C.I. 1989. "Esquistossomose mansonic cerebral." Arg. Neuropsiquiat., 47, 100 – 104.

Andrade, Z.A. and J.C. Bina. 1983. "The pathology of the hepatosplenic form of *Schistosoma mansoni* infection." Memorial Institute. Oswaldo Cruz., 78, 285 – 305.

Arap Siongok, T.K., A.A. Mahmoud. and J.H. Ouma. 1976. "Morbidity in *Schistosoma mansoni* in relation to intensity of infection; study of a community in Machakos, Kenya." American Journal of Tropical Medicine and Hygiene., 25, 273–84.

Arap Siongok, T.K., A.A. Mahmoud. and J.H. Ouma. 1976. "Morbidity in *Schistosoma mansoni* in relation to intensity of infection; study of a community in Machakos,

Kenya." *American Journal of Tropical Medicine and Hygiene.*, 25, 273–84.

Bedwani, R., E. Renganathan. and K.F. El. 1998. "Schistosomiasis and the risk of bladder cancer in Alexandria, Egypt." *British Journal on Cancer.*, 77, 1186 – 1189.

Bina, J.C. and A. Prata. 1990. "A Evoluçãõ Natural da Esquistossomose mansoni em uma Area Endêmica. Bahia." *Cedre.*

Bina, J.C. 1997. "Estudo de Variações que Podem Influenciar na Evoluçãõ da Esquistossomose mansônica: Efeito da Terapêutica Específica e da Interrupçãõ da Transmissãõ." *Reveal., Patol Tropical* 26, 69–128.

Boros, D.L. 1989., "Immunopathology of *Schistosoma mansoni* infection." – *Clinical Microbial Reveal.*, 2, 250 – 269.

Butterworth, A.E., 1994. "Human immunity to schistosomes: some questions." *Parasitology Today.*, 10, 378 – 380.

Caatinga Do Moura Amélia Ribeiro De Jesus, Delfin Gonzalez Miranda, Roberval Gonzalez Miranda, Ilma Araújo, Andréa Magalhães, Marcus Bacellar, and Edgar M. Carvalho, 2000. "Morbidity associated with *schistosoma mansoni* infection determined by ultrasound in an endemic area of Brazil" *The American Society of Tropical Medicine and Hygiene.*, 1 - 4

Chan, M.S., H.L. Guyatt., D.A.P. Bundy. and G.F. Medley. 1996. "Dynamic models of schistosomiasis morbidity." *American Journal of Tropical Medicine and Hygiene.*, 55, 52 – 62.

Cheever, A.W. and R.H. Duvall. 1982. "Migration of worm pairs within the mesenteric veins of mice." *Transaction of the Royal Society of Tropical Medicine and Hygiene.*, 76,

Chimbari, M.J., E. Dhloho., E. Mwadiwa. and Mubila, L. 2003. "Transmission of schistosomiasis in Kariba, Zimbabwe, and a cross-sectional comparison of schistosomiasis prevalences and intensities in the town with those in Siavonga in Zambia." *Annual of Tropical Medicine.*, 97, 605-16.

Chitsulo, L., D. Angels., A. Montessor. and L. Savioli. 2000. "The global status of Schistosomiasis and its control." *Acta Tropica.*, 77, 41-51.

Dittrich, M., S. Milde., E. Dinkel., W. Baumanni. and D. Weitzel. 1983. "Sonographic biometry of liver and spleen size in childhood." *Pediatric Radiol.*,13, 205–211.

Doehring-Schwerdtfeger, E., I.M. Abdel-Rahim., R. Kardorff., C. Kaiser., D. Franke., J. Schlake., J. Richter., M. Elsheikh., Q. Mohamed-Ali. and J.H. Ehrich. 1992. "Ultrasonographic investigation of periportal fibrosis in children with *Schistosoma mansoni* infection, reversibility of morbidity twenty-three months after treatment with praziquantel." *American Journal of Tropical Medicine and Hygiene.*, 46, 409–415.

Douglas, G. A., 1991. "Practical statistics for Medical Research." 165 – 171.

Frenzel, L. G., E. Odongo-Aginya., C.M. Ndugwa., T. Lroni-Lakwo., U. Schweigmann., U. Vester., N. Spannbrucker. and E. Doehring. 1999. "Evidence for a long-term effect of a single dose of praziquantel on *Schistosoma mansoni*-induced hepatosplenic lesions in northern Uganda." *American Journal of Tropical Medicine and Hygiene.*, 60, 927-931.

Gryseels, A., 1991a. "Morbidity due to *Schistosoma mansoni* and its control in Sub-Saharan Africa." *Parasitology Today.*, 7, 244–8.

Gryseels, A., 1991b. "The epidemiology of schistosomiasis in Burundi and its

consequences for control." Transaction of the Royal Society of Tropical Medicine and Hygiene., 85, 626 – 653.

Huang, Y. and L. Manderson. 1992. "Schistosomiasis and the social patterning of infection." Acta Tropica., 51, 175 – 194.

Jordan, P. and G. Webbe. 1982. Epidemiology, Treatment and Control of Schistosomiasis. International Edition, Great Britain.

Jordan, P., G. Webbe. and R. Sturrock. 1993. Human schistosomiasis. Wallingford, England, CAB.

Joseph, S., F.M. Jones., K. Walter. and A.J. Fulford. 2004. "Increase in Human T helper 2 Cytokine responses in *S. mansoni* worm and worm tument antigens are induced by treatment with praziquantel." Journal of infectious Diseases., 190, 835 – 42.

Katz, N., A. Chaves. and J. Pellegrino. 1972. "A simple device for quantitative stool thick smear technique in schistosomiasis mansoni." Reveal Institute of Medicine in the Tropics Saõ Paulo., 14, 8–17.

Katz, N. and F. Zicker. 1975. "Correlation between symptomatology and intensity of *Schistosoma mansoni* infection in inhabitants from endemic areas in Minas Gerais State—Brazil." Brasilia Medica., 11(1,2), 55–59.

Kloetzel, K., 1962. "Splenomegaly in *Schistosoma mansoni*." American Journal of Tropical Medicine and Hygiene., 25, 273–84.

Ministry of Health report. 2006. "Bilharzia in Zambia".

McGarvey, S.T., G. Aligui., K.K. Graham., P. Peters., G.R. Olds and R. Olveda. 1996. "Schistosoma japonica and childhood nutritional status in northeastern Leyte, the

Philippines, a randomized trial of praziquantel versus placebo." *American Journal of Tropical Medicine and Hygiene.*, 54, 498 – 502.

McGarvey, S.T., G. Wu., and S. Zhang. 1993. "Child growth, nutritional status and *Schistosoma japonica* in Jiangxi, People's Republic of China." *American Journal of Tropical Medicine and Hygiene.*, 48, 547 – 553.

Mubila, L and D. Rollinson. 2002. "Snail parasite compatibility and prevalence of *S. haematobium* on the shores of Lake Kariba, Zambia." *Annals of Tropical Medicine and Parasitology.*, 96(2), 165 – 173(9).

Nokes, C., S.T. Mc Garvey and L. Shine. 1999. "Evidence for an improvement in cognitive function following treatment of *Schistosoma mansoni* infection in Chinese Primary School children." *American Journal of Tropical medicine and Hygiene.*, 60, 556 – 565.

Qurashi Mohamed, A., M.A.E. Nasr-Eldin., A.A. Abdelhameed., A. Mergani., S. Rahoud., E.K. Elagib., K.O. Saeed., A. Laurent., M.A.M. Magzou and A.J. Dessein 1999. "Susceptibility to periportal (symmers) fibrosis in human *Schistosoma mansoni* infections, evidence that intensity and duration of infection, gender, and inherited factors are critical in disease progression." *Journal of Infectious Diseases.*, 180, 1298-1306.

Sama, M.T. and R.C. Ratard. 1994. "Water contact and schistosomiasis infection in Kumba, south-western Cameroon." *Tropical Medical Parasitology.*, 88, 629 – 634.

Schistosomiasis Control Initiative Zambian baseline analysis for children and adults report, 2007.

Sleigh, A.C., K.E. Mott. and R. Hoff. 1985. "Three-year prospective study of the evolution of Manson's schistosomiasis in northeast Brazil." *Lancet.*, 2, 63–6.

Sleigh, A.C., K.E. Mott., R. Hoff., J.H. Maguire and J.T.P. Silva. 1986. "Manson's schistosomiasis in Brazil: 11 – year evaluation of successful disease control with oxamniquine." *Lancet.*, 1, 635 – 637.

Smithers, S.R. 1976. *Adv. Parasitology* 14., 399.

Smithers, S.R. and M.J. Doenhoff. 1982. Schistosomiasis, in: *Immunology of parasitic diseases*, S. Cohen, and K.S. Warren, editors, 527 – 607, Oxford, England: Blackwell scientific.

Warren, K.S., Cook, J.A. and Jordan, P. 1972. *Transaction of the Royal Society of Tropical Medicine and Hygiene.*, 66, 65.

Warren, K.S. 1973. "Regulation of the prevalence and intensity of schistosomiasis in man: immunology or ecology?" *Journal of Infectious Diseases* 127., 595 – 609.

WHO 1985. "The control of schistosomiasis." Report of a WHO Expert Committee. Geneva, World Health Organization Technical Report Series 728.

WHO 1991. "Proposal for a practical guide to the standardized use of ultrasound in the assessment of pathological changes." TDR/SCH/Ultrasound/91.3.

WHO 1993. "The Control of Schistosomiasis." Second report of a WHO Expert Committee. Geneva, World Health Organization Technical Report Series 830.

WHO 1996. Report of an Informal Consultation on helminth infection and anaemia in girls and women. Geneva, World Health Organization. Document WHO/CTD/SIP/96.1.

WHO 1998. Report of the Informal Consultation on schistosomiasis control. Geneva, World Health Organization. Document WHO/CDS/CPC/SIP/99.2.

WHO 2000. "A Practical Guide To Standardized Use of Ultrasonography For the Assessment of Schistosomiasis related Morbidity." Niamey, Niger, World Health Organization. Document TDR/STR/SCH/00.1.



## CHAPTER 8.0 APPENDIX I

**Knowledge, Attitude and Practices (KAP) questionnaire administered to 269 study participants of Game Village.**

No	Question	% response			
		Yes	%	No	%
1	Do you know what bilharzia is?				
2	Do you know how bilharzia is caused?				
3	Do you know the symptoms of bilharzia?				
4	Do you know how to prevent bilharzia?				
5	Do you know the treatment for bilharzia?				
6	Does bilharzias cause abdominal pain				
7	Do you have a toilet/latrine at school or home?				
8	Have you lived in Game village for 10 years?				
9	Have you lived in Game village since birth				
10	Where do you get drinking water when at school or home? Is it a well?				

