"THE PREVALENCE, INTENSITY AND MORBIDITY OF SCHISTOSOMIASIS AMONG THE SCHOOL CHILDREN FROM RUSINGA ISLAND, LAKE VICTORIA IN KENYA."

BY

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A THESIS SUBMITTED IN PART FULFILMENT FOR THE DEGREE OF MASTER OF PUBLIC HEALTH (M.P.H.) AT THE UNIVERSITY OF NAIROBI.

- DECLARATION -

This thesis is my original work and has not been presented for a degree in any other University;-

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1. SUMMARY

A cross-sectional descriptive study of schistosomiasi was carried out on the school children residing on Rusinga Island of Lake Victoria in Kenya.

Between February and December, 1986 a total of 2620 pupils (79.5%) out of 3294 registered in the Island's schools were examined. 247 pupils (9.4%) were found to be infected with schistosome eggs; (88.3% with schistosoma mansoni, 11.7% with S. haematobium and 0.2% with both).

Of those infected with S. haematobium 24(82.8%) had haematuria (confirmed by reagent strips) and out of these 24, 21(91.3%) had more than 50 eggs per 10ml of their urine specimens.

Of those with S. mansoni infection, 76(73,1%) had bloody stools (confirmed by occult blood test) out of whom 70(97.2%) had more than 100 eggs per gram of their faecal specimens. Only three pupils out of the 218 with S. mansoni had hepatomegaly but all three had more than 200 eggs per gram of their faecal specimens. There was no relationship between splenomegaly and S. mansoni infection as only two (1.9%) of those infected had splenomegaly whereas 49(3.7%) had splenomegaly but no S. mansoni infection.

Intensity of schistosomiasis was found to have no apparent effect on the academic performance of the infecte pupils nor did it influence their weight for age.

Potential intermediate snail hosts for S. mansoni identified from the Island included Biomphalaria pfeifferi B. choanomphala and B. sudanica while those for S. haematobium were Bulinus (Physopsis) africanus, B (Ph) nasatus, and B. (Ph) globosus.

Apart from schistosomiasis 172 pupils (6.6%) had Ascaris lumbricoides, 136 (5.2%) had Trichuris trichiura, 67(2.6%) had hookworm, 44(1.7%) had Entamoeba histolytica and 21 pupils (0.8%) had Giardia lamblia.

2. INTRODUCTION

2.1. THE DISEASE:

Schistosomiasis (bilharziasis) is a visceral parasitic disease caused by trematodes (blood flukes) of the genus schistosoma. The adult male and female trematodes live in either mesenteric venules or/and vesical venules of human definitive host over a life span of several years. There are five different species of schistosomes infecting possibly 200 million people in the world with 500 - 600 million people exposed to the threat of infection according to the World Health Organization (WHO/SCHISTO/83.71). S. haematobium (found all over Africa, the Middle East and Cyprus), S. mansoni (found in Egypt and all over Africa South of the Sahara, parts of South America and Caribbean Island), S. intercalatum (found in parts of West and Central Africa), S. japonicum (found in Japan, China, Thailand and Laos) and S. mekongi (found in the Mekong River area between Laos and Cambodia) are the five species which normally infect man.

The African species of the genus schistosoma may be divided into two species complexes on the basis of major morphological and biological differences:

The S. haematobium species complex:

The ovary of the female worms is situated in the posterior part of the body and the uterus contains 10 - 100 eggs with (normally) well developed terminal spines. The number of testes in the male worm is

3 - 6 and the intermediate snail host species all belong to the genus Bulinus. The schistosoma species belonging to this species complex comprise S. haematobium, S. intercalatum, S. bovis, S. mattheei, S. margrebrowiei and S. leiperi.

The S. mansoni species complex:

The ovary of the female worm is situated in the anterior part of the body and the uterus contains 1 - 2 eggs with (normally) well developed lateral spines. The number of testes in the male worm is 6 - 9 and the intermediate snail host species all belong to the genus Biomphalaria. The schistosoma species belonging to this species complex comprise S. mansoni, S. rodhaini and S. edwardiense.

S. mansoni, S. haematobium and S. intercalatum are considered being the human schistosoma species (only man serves as primary definitive host) in Africa. However, infection of man with some of the non-human schistosoma species have been shown to occur, for example, S. mattheei (Pitchford, 1959). S. margrebrowiei (Walkiers, 1928), S. rodhaini (Haenens and Santele, 1955), and human infection with S. bovis have been found throughout Africa[1], but man does not function as a primary definitive host for these species. Also, naturally acquired infections with the human schistosoma species have been recorded in large number of African mammalian species but they play no significant role in their

transmission. Natural hybridization between schistosomes occurs in man, namely between male S. haematobium and female S. mattheei in parts of Southern Africa (giving rise to S. mattheei like-egg found in human urine), between S. haematobium and female S. intercalatum in Cameroun and between the biologically markedly different strains of S. haematobium in parts of Ghana.

S. mansoni and S. haematobium are the only human species found in Kenya and both have similar life cycles (see figure 1) developing over a succession of stages, namely, the egg, miracidium (see figure 2), first and second stage sporocysts, cercaria (see figure 3), schistosomulum and adult (see figure 4). When viable eggs produced by female trematodes pass out with urine (S. haematobium) and faeces (S. mansoni) of infected individuals, they normally contain mature miracidia. The eggs hatch in water and the liberated larvae (miracidia) have to enter suitable fresh-water snail hosts within a few hours else they die. In the snail each miracidium develops into a first stage sporocyst within a few days and by asexual reproduction lasting about four weeks the first stage sporocysts develop into second stage sporocysts which in turn grow and multiply into cercariae. From a single miracidium as many as 100,000 cercariae may be produced - all of the same sex, taking approximately 4 - 5 weeks (S.mansoni) and 5 - 6 weeks (S. haematobium) depending on temperature, age of the snail and the number of miracidia infecting it.

Cercariae are relatively short-lived (up to 48 hours), the period during which they have to find and penetrate the

skin of a defenitive host. The process of penetrating takes a few minutes but the length of time of penetration till eggs excretion commences is 5 - 6 weeks (S. mansoni) and 10 - 12 weeks (S. haematobium). Soon after penetration the cercaria changes its appearance to become a tail-less schistosomulum (tail-less and worm-like). It stays in the skin for 3 - 5 days before entering the peripheral blood vascular system or the lymphatic system to be carried passively to the lungs via the right heart. The schistosomulum undergoes modifications over a period of 2 - 3 days in the lungs before passively carried by the blood flow to the left heart then to the systemic circulation and by chance to the hepatic portal system where final growth and maturation takes place. Mature female and male worms pair and mate before migrating to either mesenteric venules (S. mansoni) or the vesical plexus (S. haematobium).

Infection with S. haematobium

Clinical features of S. haematobium infection include the earliest symptom at the site of cercarial penetration in the form of severe itching (cercarial dermatitis). This lasts 2 - 3 days and may be accompanied by small red papules (Barlow and Meleney, 1949). About eight weeks later an allergic reaction (pyrexia, headache, generalised pains, anorexia, nausea, vomiting, hepatalgia and splenomegaly) may appear with accompanying blood eosinophilia (as high as 80%) and even eggs may be found in urine.

The stage of established infection occurs 10 - 12 weeks after cercarial penetration and is usually manifested by transitory haematuria and egg extrusion while symptoms of irritability of the bladder manifest as dysuria and frequency. The main histopathological features of the disease appear at the stage of late infection. Calcified eggs scattered in the dense collageneous bladder tissue cause fibrosis which may lead to bladder neck obstruction and later carcinoma of the bladder. In countries endemic for S. haematobium infection, the high frequency of squamous cell bladder cancer has been reported (Cheever, 1978).

S. haematobium infection is endemic in 52 African and Eastern Mediterranean countries. It is estimated that at least 180 million persons are at risk of infection and about 90 million persons are infected (WHO, 1980; Iarotski and Davis, 1981). The highest prevalence and the major proportion of heavy infections are observed in school age children. The excretion of blood and protein in the urine of infected children, as well as adults is directly correlated with the number of S. haematobium eggs in the urine (Mott et al, 1983), and about 98-100% of all persons who have more than 50 eggs of S. haematobium per 10ml of urine have haematuria.

Contamination of snail habitats with S. haematobium eggs is due essentially to infected persons urinating directly into water. Bulinus species involved in natural transmission of S. haematobium in Kenya are Bulinus (Physopsis) africanus, B(Ph) nasatus and (B(Ph) globosus.

Parasitological diagnosis based on recovering S. haematobium eggs from the patient's urine is the method now used to provide a definitive diagnosis of an active infection. Once diagnosed treatment is carried out by use of the current safe oral antischistosomal drugs, praziquantel (Biltricide^{R}) at a dose of 40mg/kg body weight stat and metrifonate (Bilarci^{R}) at a dose of 7.5-10mg/kg body weight given in three doses at two week intervals. These drugs can effectively stop the progress of and reverse the morbidity due to S. haematobium infection (WHO, 1980; Iarotski & Davis 1981; Mott et al; 1983)

Infection with S. mansoni

Following cercarial penetration, S. mansoni infection may present as an irritating macular, papular skin rash for several days. This may be accompanied with fever, allergic manifestation with eosinophilia, hepatosplenomegaly and lymphadenopathy.

In chronic S. mansoni infection the majority of patients are symptom - free although bloody stools are frequently associated with S. mansoni infection. Other patients may present with intermittent diarrhoea with periods of normal bowel movement or constipation. However, the severity of pathological manifestations (hepatomegaly, splenomegaly or hepatosplenomegaly) is normally correlated with intensity of infection, and intensity of transmission is in general corresponding with the prevalence rates that are normally found in highly endemic areas. Hepatosplemegaly is uncommon before the age of 10 years but common between 10 - 20 years of age. Some individuals with only light infections may experience severe disease while some having heavy infections

may experience only limited disease. Oesophageal varices due to portal hypertension may rapture causing haematemesis or melaena. Portal hypertension is mainly due to liver fibrosis caused by deposit of S. mansoni eggs, granulomas and cellular infiltrations. The process may also cause ascites.

Contamination of snail habitats with S. mansoni eggs is due to infected persons defaecating either directly in water or the faeces deposited on the banks of water bodies being washed into the fresh water by rain or fluctuating water levels. Biomphalaria species involved in natural transmission of S. mansoni in Kenya are B. pfeifferi, B. sudanica and B. choanomphala.

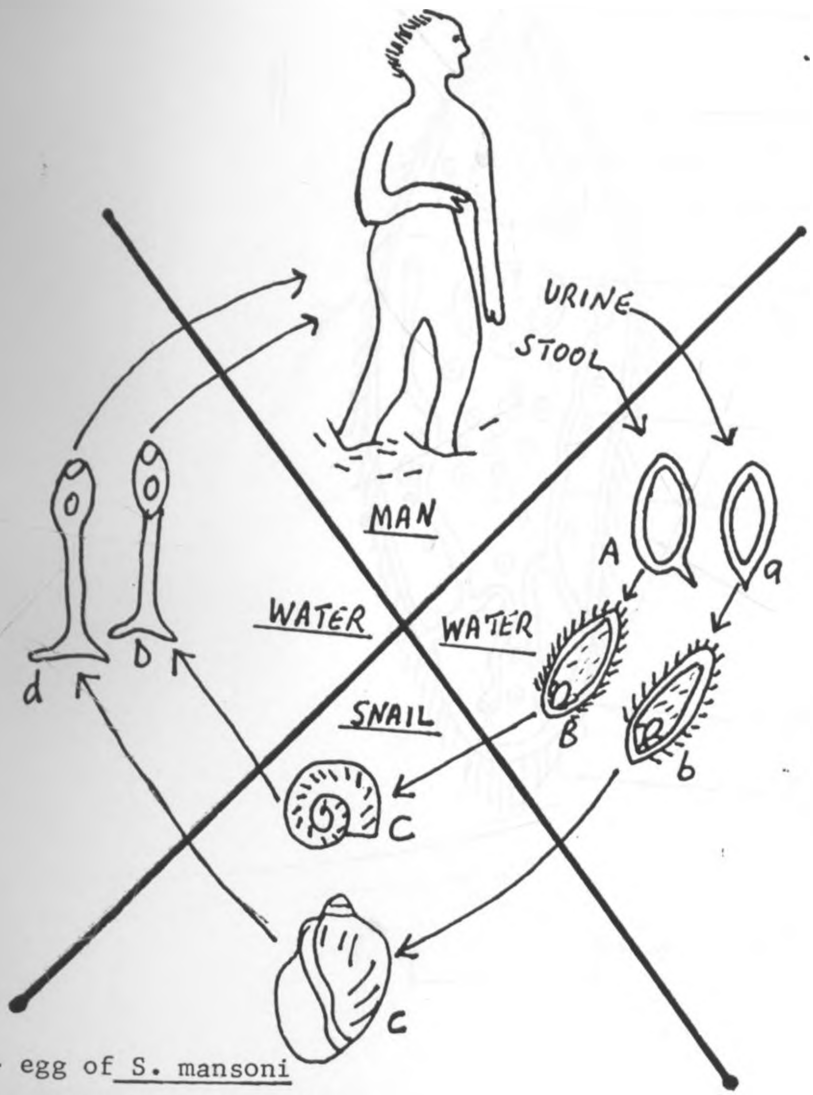
Recovering S. mansoni eggs from the patient's stool provides a definitive diagnosis of active infection. A diagnosed infection is currently treated with Praziquantel at an oral dose of 40mg/kg body weight once or Oxamniquine (Vansil^{R}) at an oral dose of 30mg/kg body weight given over two days.

The epidemiology of human schistosomiasis involves three major components; the schistosome parasite, the human definitive host, and the molluscan intermediate snail host. Each of these components is subject to considerable variations mediated directly or indirectly by the numerous factors which influence the common environment. The dynamics of transmission of human schistosomes, and thereby the epidemiology of human schistosomiasis, therefore exhibit great variability and complexity as governed by local geological, geographical and climatic conditions.

The primary objective of schistosomiasis control has always been to reduce or eliminate morbidity due to the disease. Morbidity due to schistosomiasis is directly correlated with the number of schistosome eggs being eliminated in urine (S. haematobium) or in faeces (S. mansoni) and the intensity of schistosomiasis is in general proportional to the prevalence rate. For effective reduction of morbidity in schistosomiasis, one has to reduce the female adult worms which produce the eggs in man. This can easily be done by using the current safe oral antischistosomal drugs (chemotherapy).

Chemotherapy deals with only one of the three major components of human schistosomiasis - the schistosome parasite. The human definitive host needs also to be educated on the safe ways of disposing of his waste into the environment and on what types of water are safe for him to use for his domestic, recreational, occupational and religious purposes. In other words, health education alone can reduce the transmission of schistosome eggs from man to the intermediate snail hosts. Until the last decade, schistosomiasis control was synonymous with snail control (Farooq et al, 1966). More efforts were made to eliminate the third component responsible for the epidemiology of human schistosomiasis (the molluscan intermediate snail host) than to eliminate the other two components. Snail control can be effected through three different methods. Environmental control (through modification of the snail habitat), biological control (the use of biological competitors, predetors and parasites of snails) and chemical control (the use of molluscicides).

FIGURE 1. Life cycle of Schistosoma haematobium and Schistosoma mansoni.



A- egg of S. mansoni

ā- egg of S. haematobium

B- miracidium of S. mansoni

b- miracidium of S. haematobium

C- intermediate snail host of genera Biomphalaria.

c- intermediate snail host of genera Bulinus.

D- cercaria of S. mansoni.

d- cercaria of S. haematobium

FIGURE 2. Morphology of the schistosome miracidium

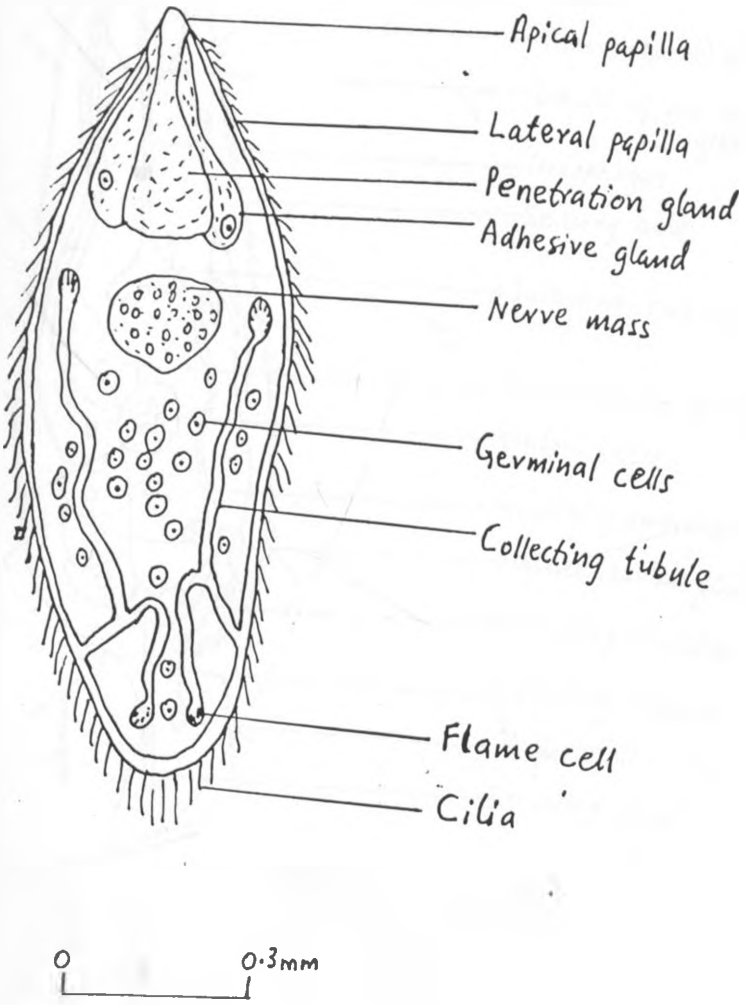


FIGURE 3. Morphology of the schistosome cercaria

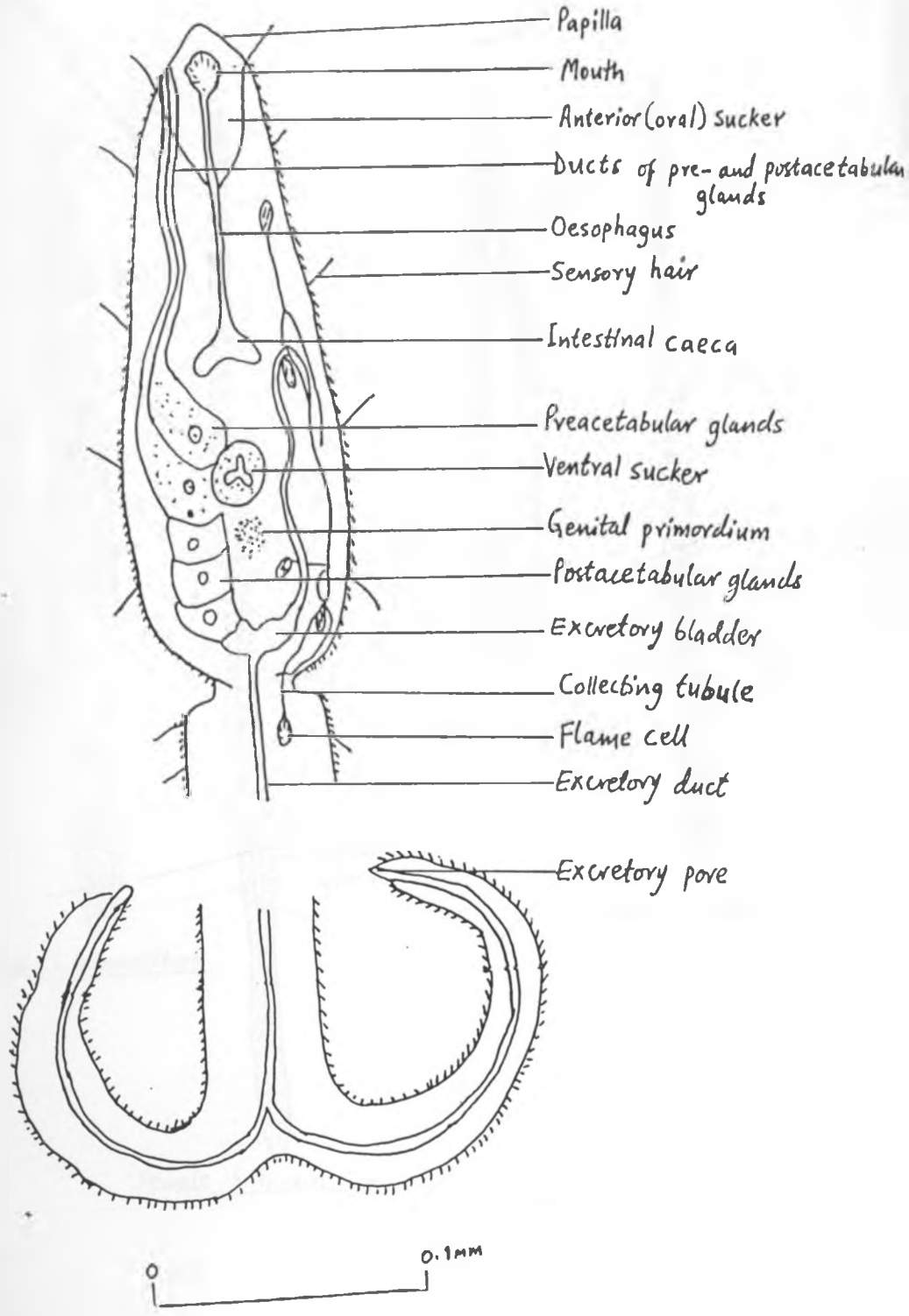
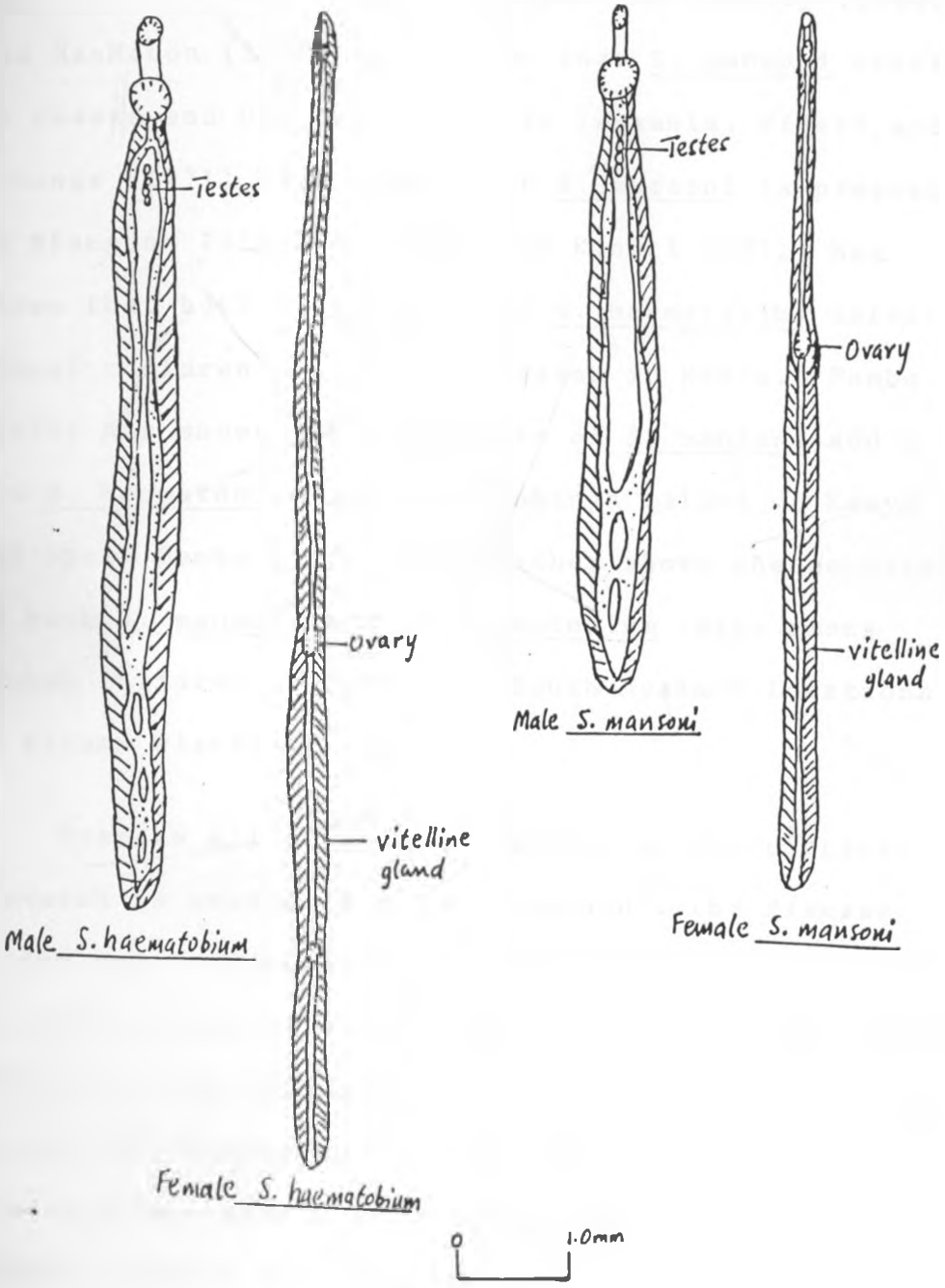


FIGURE 4. Major morphological characteristics of S. haematobium and S. mansoni species.



2.2. THE STUDY

Both *S. mansoni* and *S. haematobium* have long been known to be endemic in the Lake Victoria basin, East Africa. For example Forsyth and Bradley (1964) and MacMahon (1967) have shown that *S. mansoni* occurs in Mwanza and Ukerewe Island in Tanzania, Wijers and Munanga (1971) have shown that *S. mansoni* is present in Mfangano Island of Kenya and Kinoti (1971) has shown that both *S. mansoni* and *S. haematobium* infect school children of the Kano Plains in Kenya. Pamba (1974) has shown the occurrence of *S. mansoni* and a few *S. haematobium* cases in Rusinga Island of Kenya and again Pamba (1977) has further shown the occurrence of both *S. mansoni* and *S. haematobium* cases among school children of North and South Nyakach Locations in Kisumu District, Kenya.

Despite all this data, further epidemiological research is needed in order to measure the disease burden and to identify specific risk factors relating to schistosomiasis with a view to improving the disease control at the community level in conformity with the concept of Primary Health Care programme. When resources are made available, thorough descriptive studies (rather than complex computer simulations) should lead to rapid and effective control measures.

This type of baseline epidemiological study can provide a foundation on which a national control programme can be implemented and designed using a Community-based

disease control strategy. Furthermore, a disease like schistosomiasis may show a high prevalence in the community but only a minority of those infected have serious clinical disease. On the account of the foregoing observations, this study was undertaken to determine not only the prevalence of schistosomiasis among the school children of Rusinga Island, but also the morbidity relating to intensity of the infection and its other possible effects.

3. OBJECTIVES AND RATIONALE

3.1. General Objective :

To determine the prevalence, intensity and morbidity of schistosomiasis, relating the intensity of infection to the academic performance and weight for age among the school children from Rusinga Island in Kenya.

3.2. Specific Objectives:

3.2.1. To determine age-specific and overall prevalences and intensities of *S. mansoni* and *S. haematobium* infections among the school children from Rusinga Island.

3.2.2. To determine the prevalence of associated morbidity (bloody stools, hepatomegaly and splenomegaly for *S. mansoni* infection and haematuria for *S. haematobium* infection) among schistosome positive and non-schistosome positive school children.

3.2.3. To relate morbidity (bloody stools, hepatomegaly, splenomegaly and haematuria), academic performance and weight for age to intensity of schistosomal infection among the school children.

3.2.4. To evaluate the associated morbidity (bloody stools, hepatomegaly, splenomegaly and haematuria) as a screening method for diagnosing schistosomiasis.

3.4. Rationale:

The Integrated Rural Health programme was designed by the Ministry of Health in Kenya to take care of all health problems in the rural areas. Due to budgetary constrains the control of communicable diseases like schistosomiasis has suffered in favour of curative services. Reduction in prevalence and intensity of schistosomiasis, for example, should be within the scope of the Ministry of Health to undertake at any level of the health care delivery system.

Pamba (1974) showed that S. mansoni occurred in all parts of Rusinga Island with a prevalence of 30% among the school children and that S. haematobium was rare. Routine laboratory records at the only hospital on the Island showed that during the year 1983, 14.9% of all stools examined in the out-patient had S. mansoni eggs and 3.4% of all urine examinations had S. haematobium eggs. Figures recorded for the next two years were 27.3% with S. mansoni eggs, none with S.haematobium eggs (1984), and 22.1% with S. mansoni eggs and 4.6% with S. haematobium eggs (1985).

On the account of the above observations, and while still the Medical Officer of Health for South Nyanza District I undertook to survey the schistosomiasis situation on Rusinga Island so that with the result at hand enough antischistosomal drugs would be despatched from Nairobi. Furthermore, since S. mansoni infections have been shown to be asymptomatic in many patients it is only through such an epidemiological survey that asymptomatic cases could be detected. I chose to survey the schools only because previous studies have shown that the highest prevalence and the major proportion of heavy infections are observed in school-age (5 - 20 years) children. In addition it would be easier and quicker to reach school children in schools than to get enough villagers to provide stool and urine samples within such a short time.

At the time funds were made available for the project, I was already at the University of Nairobi undertaking post-graduate studies in Public Health. However, the Division of Diseases Control and Research (DDC&R) based in Homa Bay went on with the survey as had been planned by myself. But funds ran out after only four of the eleven schools had been surveyed from February to May, 1986. Fortunately, three months later it was made possible for me to continue with the project as part fulfilment for my Master of Public Health (M.P.H.) degree at the University of Nairobi, and by the end of the year most of the required data had been collected.

4. METHODOLOGY

4.1. The Study Area:

Rusinga Island is one of the Lake Victoria Islands situated in the Republic of Kenya (see figures 1, 5, and 6 plus Appendix II). Administratively, the Island is a location sub-divided into three sub-locations (Kamasengere, Waware and Kaswanga) within Mbita division of South Nyanza district. Situated between 0.51^{o}S and 0.62^{o}S latitudes and between 34.24^{o}E and 34.37^{o}E longitudes, the Island has an area of 50 square kilometres and a population of about 12,000 people (projected from the 1979 Kenya's national population census), giving a population density of about 240 people per square kilometre.

As at 31st July, 1985, the Island had 10 primary schools (Kamasengere, Nyamuga, Kamayoge, Wanyama, Waregi, Kaswanga, Kakrigu, Uya and Utajo) with 3052 pupils and one secondary school (Tom Mboya) with 242 pupils. Apart from the pupils from the secondary school, the majority of primary school pupils have been living on the Island since birth. There is only one hospital situated to the North West of the Island and one village Polytechnic situated near the Chief's Office to the South-East of the Island.

The Island is formed from sedimentary rocks which rise from the Lake shores situated at 1125 metres above the sea level to the central parts where the altitude reaches upto 1500 metres above the sea-level. It is

separated from the main-land by a 200 metre man-made causeway (completed in 1983) and there is a dry weather road around the Island joining the mainland net-work of roads through the causeway.

Rusinga Island is a relatively dry area. The mean annual rainfall is 760mm with only one period of heavy rains from March to May when Subsistence crops (maize, sorghum, cassava and sweet potatoes) are planted. The lake is the major source of water though during the rainy months the Islanders use rain-water collected from roofs, seasonal streams, and ponds. Nyamita stream provides some water even during the dry season. Fishing is the major occupation on the Island. Dholuo and Abasuba are the only local dialects but English and Swahili are extensively taught in the schools.

4.2. The Study Design:

This was a descriptive cross-sectional study based on school children and lasting about one year. It involved stool and urine screening for schistosome eggs, a physical examination and an oral medical questionnaire.

4.3. The Subjects:

The total population of study was 3294 school children from the eleven established schools on the Island: Kamasengere primary (414 pupils), Nyamuga primary (361 pupils), Kamayoge primary (252 pupils),

Wanyama primary (433 pupils), Waregi primary (333 pupils), Agiro primary (212 pupils), Kaswanga primary (284 pupils), Kakrigu primary (292 pupils) Uya primary (185 pupils), Utajo primary (286 pupils) and Tom Mboya secondary school (242 pupils). A total of 2620 pupils (79.5% of the total) were examined.

The 20.5% absenteeism was due mainly to the apparent inflation of pupil's lists by some headmasters such that the figures collected from the District Education Office did not correspond with the actual number of pupils found registered in some schools visited. A few pupils, however, though present, refused to produce stool and urine due to some cultural beliefs while others did not attend school on the particular day due to different domestic problems. Any pupils admitted to hospital due to schistosomiasis would not have had their stool and urine examined. This would result in an underestimate of the actual cases of schistosomiasis.

FIGURE 5: MAP OF KENYA SHOWING THE LOCATION OF RUSINGA ISLAND.

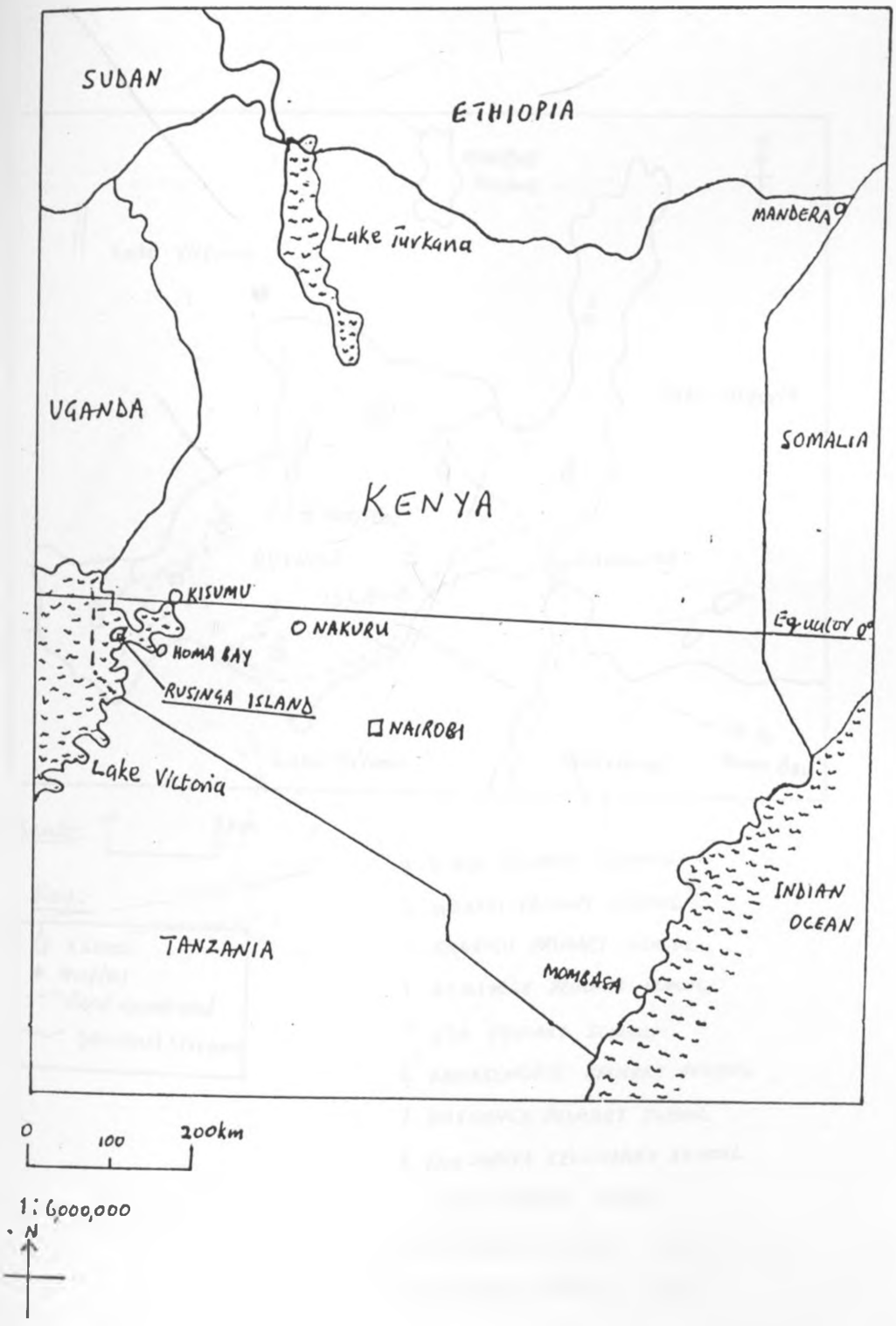
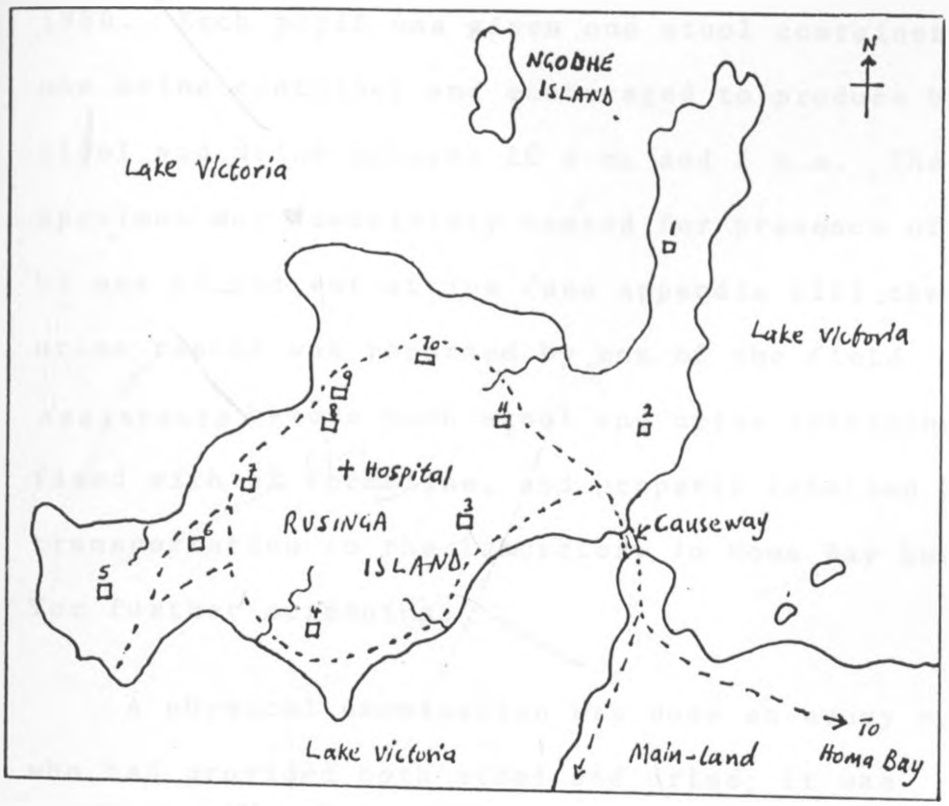


FIGURE 6 : A SKETCH MAP OF RUSINGA ISLAND SHOWING SCHOOLS WHERE THE EPIDEMIOLOGICAL SURVEY OF SCHISTOSOMIASIS WAS CARRIED OUT:



Scale: 0 3km

Key:

- School
- + Hospital
- --- Rural access road
- ~ Seasonal streams

1. UTAJO PRIMARY SCHOOL
2. WAREGI PRIMARY SCHOOL
3. KAKRIGU PRIMARY SCHOOL
4. KAMAYOGE PRIMARY SCHOOL
5. UYA PRIMARY SCHOOL
6. KAMASENGERE PRIMARY SCHOOL
7. KASWANGA PRIMARY SCHOOL
8. TOM MBOYA SECONDARY SCHOOL
9. AGIRO PRIMARY SCHOOL
10. WANYAMA PRIMARY SCHOOL
11. NYAMUGA PRIMARY SCHOOL

4.4. Materials and Methods:

Stools and urine samples were collected in all eleven schools from 2nd February to 28th October, 1986. Each pupil was given one stool container and one urine container and encouraged to produce both stool and urine between 10 a.m. and 2 p.m. The urine specimen was immediately tested for presence of blood by use of reagent strips (see appendix III) then this urine result was recorded by one of the field assistants before both stool and urine specimens were fixed with 5% formaline, and properly labelled before transportation to the laboratory in Homa Bay hospital for further screening.

A physical examination was done on every pupil who had provided both stool and urine; it was performed blind (without knowledge of stool and urine results) and by the principal investigator, throughout the study. Before the physical examination was performed, each pupil had to answer standardised medical questionnaires about history of bloody stools and haematuria. In addition, the name, sex, age, weight, academic position in class and the pupil's activity in relation to the lake were recorded (see appendix I). Every pupil was then examined in a supine position and if the liver was palpable, the maximum distance from the right costal margin in the mid-clavicular line (or from the xiphoid in the mid-sternal line) to the edge was measured using a centimeter

ruler. The spleen edge palpated below the left costal margin in the anterior axillary line was measured according to Hackett scale (see appendix IX).

Back at the laboratory, each stool specimen was examined for occult blood (see appendix VI) then by the Formol-ether concentration technique (see appendix VII) for S. mansoni eggs, plus eggs, larvae and cycts of other intestinal parasites. Due to the limited supply of materials for quantitative parasitology, only stools positive for S. mansoni eggs were further processed for Kato thick smear technique (see appendix VIII), One smear was made from each positive sample and the egg count read in duplicate by two laboratory technicians independent of each other: in addition further 10% random sample of all slides examined each day was re-examined by the principal investigator assisted by one of the Senior Laboratory Technologists, as quality control measures.

Each urine specimen positive for haematuria was directly examined by syringe filtration technique using the limited number of Nuclepore^{R} membrane filters available (see appendix V). The rest of the urine specimens (1402) were examined for S. haematobium eggs by the sedimentation technique (see appendix IV), and only positive urines (5) further quantitatively examined by syringe filtration technique.

Further analysis on the data collected from the pupils was done back at the Department of Community Health in Nairobi. But the Island's shores and inland parts were surveyed for possible transmission sites harbouring potential intermediate snail hosts. The analysis included the age-specific and overall prevalences and intensities of schistosomiasis according to sex. Prevalences of other intestinal parasites according to age and sex were also analysed. The morbidity due to schistosomiasis was analysed according to intensity of infection.

Data from academic positions for every pupil in class for the past two terms were used to classify the pupils into three academic levels; A, B and C. Those in group 'A' were pupils who attained upper third academic positions in rank (averaged from the past two previous end-of-term examinations) in their respective classes while group 'B' were the next middle third and group 'C' were the last third academic positions. An attempt was then made to relate the attainment of these grades to the intensity of schistosomiasis.

Likewise, data from pupils' weight were used to classify pupils into three different groups of weight for age according to standard paediatric anthropometric tables. The first group were those pupils who had $\geq$ 90% of the standard weight, the second group had 75 - 89% of the standard weight and the last group were those pupils who had < 75% of the standard weight for age. These groups were also used to relate the intensity of schistosomiasis to weight for age.

Comparisons were also made between data from the 1974 schistomiasis survey and that of 1986 and the results of the survey at Kamayoge Primary School before and after treatment with praziquantel, a drug considered to be safe and effective against both S. mansoni and S. haematobium.

4.5. Ethical Considerations:

- The objectives of the study were explained to the pupils in the presence of their headmaster by the principal investigator before oral consent was obtained. There was total co-operation from all the schools visited.

- No special incentives were provided to the pupils in order to participate but all positive cases for schistosomiasis and other intestinal parasites were treated with appropriate drugs to prevent morbidity or complications due to the parasites.

- Participants with other severe illnesses were either treated, or referred to the nearest hospital,

- The only inconveniences to the school children were interruptions to their lessons and demands for stool and urine specimens but these did not outweigh the benefits.

5. RESULTS

5.1. Composition of pupils examined:

Table 1 summarises the composition of the pupils by sex and age. There were a total of 2620 pupils examined out of 3294 (79.5%) pupils enrolled in the schools. These included 1054 (40.2%) female pupils with a mean age of 10.8 years (5-20 years range), a mean weight of 34.6kg, (10-73kg range) as illustrated in Table 2. The male pupils were 1566(59.8%) with a mean age of 12.6 years (5-22 years range) and a mean weight 39.4kg (14-7kg range).

5.2. The prevalence of Schistosomiasis:

Table 4 summarises prevalences of schistosomiasis and other intestinal parasites according to school. It also illustrates the number of enrolled (3294) and the actual number of pupils examined (2620).

A total of 247 pupils (9.4%) were positive with schistosome eggs with the highest prevalence (32.7%) at Kamayoge Primary School and the lowest (1%) at Kaswanga Primary School. 434 pupils (16.6%) had other intestinal parasites.

The age specific and overall prevalence of *S. mansoni* and *S. haematobium* infections is summarised in table 5(a) while table 5(b) summarises the age-specific and overall prevalences of the

infection by other intestinal parasites. Figure 8 also illustrates in a histogram form the prevalence of schistosomiasis according to age and sex. The age specific prevalence for schistosomiasis was highest (12.6%) in the males aged 15-19 years and lowest (7.5%) in the females aged 5-9 years. The overall prevalence for schistosomiasis was 9.4% (including 7.7% for females and 10.6% for males; 8.3% for S. mansoni, 1.1.% for S. haematobium and 0.2% for mixed infections). Among other intestinal parasites A. lumbricoides had the highest prevalence (6.6%) which went up to 12.5% among the males aged 5-9 years, T. trichiura had an overall prevalence of 5.2%, Hookworm (2.6%); E. histolytica (1.7%) and G. lamblia (0.8%). Figure 7 illustrates eggs and cysts detected from stool and urine specimens collected from the pupils.

5.3. The Instensity of Schistosomiasis:

The intensity of S. mansoni infection according to age is summarised in tables 6(a) and figures 9 and 10(a). The intensity (geometric mean of eggs per gram faeces) of infection was highest (90.7) among the 10-14 years age group and lowest (51.3) among the 5-9 years age group. According to eggs count category 51-100 eggs per gram faeces showed the highest frequency (47,1%) while >200 eggs per gram faeces showed the lowest (4.8%).

The intensity of S. haematobium infection (geometric mean of eggs per 10ml urine) is summarised in Tables 7(a), and 7(b) and figures 9 and 10(b). The intensity of infection was highest (85.4) among the ≥ 20 years of age and lowest (44.1) among the 5 - 9 years age group. According to eggs count category 76 - 100 eggs per 10ml of urine showed the highest frequency (31%) while 1 - 25 eggs/10ml of urine showed the lowest (10.3%).

TABLE I

COMPOSITION OF THE SCHOOL CHILDREN STUDIED BY SEX AND AGE (RUSINGA ISLAND):

Sex	Number examined	Per cent (%)	Age in years	
			Range	Mean
Female	1054	40.2	5-20	10.8
Male	1566	59.8	5-22	12.6
Total	2620	100.0	5-22	11.9

TABLE 2

COMPOSITION OF THE SCHOOL CHILDREN STUDIED BY SEX AND WEIGHT

Sex	Number examined	Per cent (%)	Weight in kilogrammes	
			Range	Mean
Female	1054	40.2	10-73	34.6
Male	1566	59.8	14-79	39.4
Total	2620	100.0	10-79	37.4

TABLE 3 SCHOOLS FROM RUSINGA ISLAND SURVEYED FOR SCHISTOSOMIASIS (February to October, 1986)

SCHOOL	Number examined			No. +ve			5–9 year-age group						10–14 year-age group						15–19 years-age group						≥20 years-age group					
							No. examined			No. +ve			No. examined			No. +ve			No. examined			No. +ve			No. examined			No. +ve		
	Girls (F)	Boys (M)	Total (T)	F	M	T	F	M	T	F	M	T	F	M	T	F	M	T	F	M	T	F	M	T	F	M	T	F	M	T
KAMASENGERE	137	141	278	10	17	27	50	48	98	3	5	8	61	63	124	6	8	14	25	29	54	1	4	5	0	2	2	0	0	0
NYAMUGA	156	178	334	5	5	10	56	57	113	2	1	3	68	71	139	1	3	4	32	47	79	2	1	3	0	3	3	0	0	0
KAMAYUGE	85	120	205	16	51	67	22	23	45	3	11	14	42	68	110	8	29	37	21	26	47	5	11	16	0	3	3	0	0	0
WANYAMA	151	228	379	8	12	20	52	76	128	3	6	9	84	131	215	5	5	10	15	19	34	0	1	1	0	2	2	0	0	0
WIREGI	125	145	270	23	24	47	41	48	89	8	8	16	78	112	190	13	15	28	6	23	29	2	1	3	0	0	0	0	0	0
AGIRO	77	81	158	1	1	2	28	34	62	1	0	1	37	30	67	0	0	0	11	14	25	0	1	1	1	3	4	0	0	0
KASWANGA	79	127	206	0	2	2	23	25	48	0	1	1	45	72	117	0	0	0	11	26	37	0	0	0	0	4	4	0	1	1
KAKRIGU	95	124	219	5	10	15	33	25	58	2	2	4	53	59	112	2	7	9	8	37	45	1	0	1	0	4	4	0	0	0
UYA	50	81	131	3	8	11	26	38	64	3	4	7	22	41	63	1	1	2	1	0	1	0	0	0	1	2	3	0	0	0
UTAJO	92	166	258	9	7	16	31	26	57	2	0	2	58	117	175	7	6	13	5	20	25	0	0	0	0	0	0	0	0	0
TOM MBOYA	7	175	182	1	29	30	0	0	0	0	0	0	0	2	2	0	0	0	5	139	144	1	25	26	2	34	36	0	4	4
TOTAL	1054	1566	2620	81	166	247	362	400	762	27	38	65	548	766	1314	43	74	117	140	380	520	12	43	55	4	57	61	0	5	5

PREVALENCE OF SCHISTOSOMIASIS PLUS OTHER INTESTINAL PARASITES AMONG SCHOOL CHILDREN OF RUSINGA ISLAND.

TABLE 4

SCHOOL	Number of Pupils	Number examined	NO. +ve for schisto.	Prevalence (%)	Other intestinal parasites	
					NO. +ve	Prevalence (%)
KAMASENGERE PRIMARY	414	278	27	9.7	21	7.5
NYAMUGA PRIMARY	361	334	10	3.0	35	10.5
KAMAYOGE PRIMARY	252	205	67	32.7	25	12.2
WANYAMA PRIMARY	433	379	20	5.3	83	21.9
WAREGI PRIMARY	333	270	47	17.4	44	16.3
AGIRO PRIMARY	212	158	2	1.3	22	13.9
KASWANGA PRIMARY	284	206	2	1.0	59	28.6
KAKRIGU PRIMARY	292	219	15	6.8	36	16.4
UYA PRIMARY	185	131	11	8.4	25	19.1
UTAJO PRIMARY	286	258	16	6.2	56	21.7
TOM MBOYA SECONDARY	242	182	30	16.5	28	15.4
TOTAL	3,294	2,620	247	9.4	434	16.6

TABLE 5: PREVALENCE OF SCHISTOSOMIASIS PLUS OTHER INTESTINAL PARASITES ACCORDING TO AGE AND SEX:

5(a)

SEX	Age in years	NO. exam.	NO. +ve	o/o +ve	POSITIVES S. haem. NO.	S. haem. o/o	S. mansoni NO.	S. mansoni o/o	Mixed NO.	Mixed o/o
FEMALES	5-9	362	27	7.5	1	0.3	26	7.2	0	0
	10-14	548	43	7.8	5	0.9	38	6.9	0	0
	15-19	140	11	7.9	2	1.4	9	6.4	1	0.7
	≥ 20	4	0	0	0	0	0	0	0	0
	TOTAL	1054	81	7.7	8	0.8	73	6.9	1	0.1
MALES	5-9	383	39	10.2	2	0.5	37	9.7	0	0
	10-14	746	74	9.9	10	1.3	64	8.6	4	0.5
	15-19	380	48	12.6	5	1.3	43	11.3	0	0
	≥ 20	57	5	8.8	2	3.5	3	5.3	0	0
	TOTAL	1566	166	10.6	19	1.2	147	9.4	4	0.2
TOTAL	5-9	745	66	8.9	3	0.4	63	8.5	0	0
	10-14	1294	117	9.0	17	1.3	100	7.7	4	0.3
	15-19	520	59	11.3	7	1.4	52	10.0	1	0.2
	≥ 20	61	5	8.2	2	3.3	3	4.9	0	0
	TOTAL	2620	247	9.4	29	1.1	218	8.3	5	0.2

5(b)

Other intestinal parasites										
A. lumbricoides		T. trichiura		Hookworm		G. lamblia		E. histolytica	
NO.	o/o	NO.	o/o	NO.	o/o	NO.	o/o	NO.	o/o
44	12.1	30	8.3	6	1.7	4	1.1	2	0.5
34	6.2	28	5.1	10	1.8	1	0.2	16	2.9
3	2.1	5	3.6	8	5.7	0	0	5	3.6
0	0	0	0	0	0	1	25	1	25
81	7.7	63	6.0	24	2.3	6	0.6	24	2.3
48	12.5	32	8.3	7	1.8	6	1.6	4	1.0
29	3.9	31	4.1	13	1.7	6	0.8	11	1.5
14	3.7	10	2.6	20	5.3	1	0.3	5	1.3
0	0	0	0	3	5.3	2	3.5	0	0
91	5.8	73	4.7	43	2.7	15	1.0	20	1.3
92	12.3	62	8.3	13	1.7	10	1.3	6	0.8
63	4.9	59	4.6	23	1.8	7	0.5	27	2.1
17	3.3	15	2.9	28	5.4	1	0.2	10	1.9
0	0	0	0	3	4.9	3	4.9	1	1.6
172	6.6	136	5.2	67	2.6	21	0.8	44	1.7

FIGURE 7 : EGGS AND CYSTS DETECTED FROM STOOL AND URINE COLLECTED FROM SCHOOL CHILDREN OF RUSINGA ISLAND.

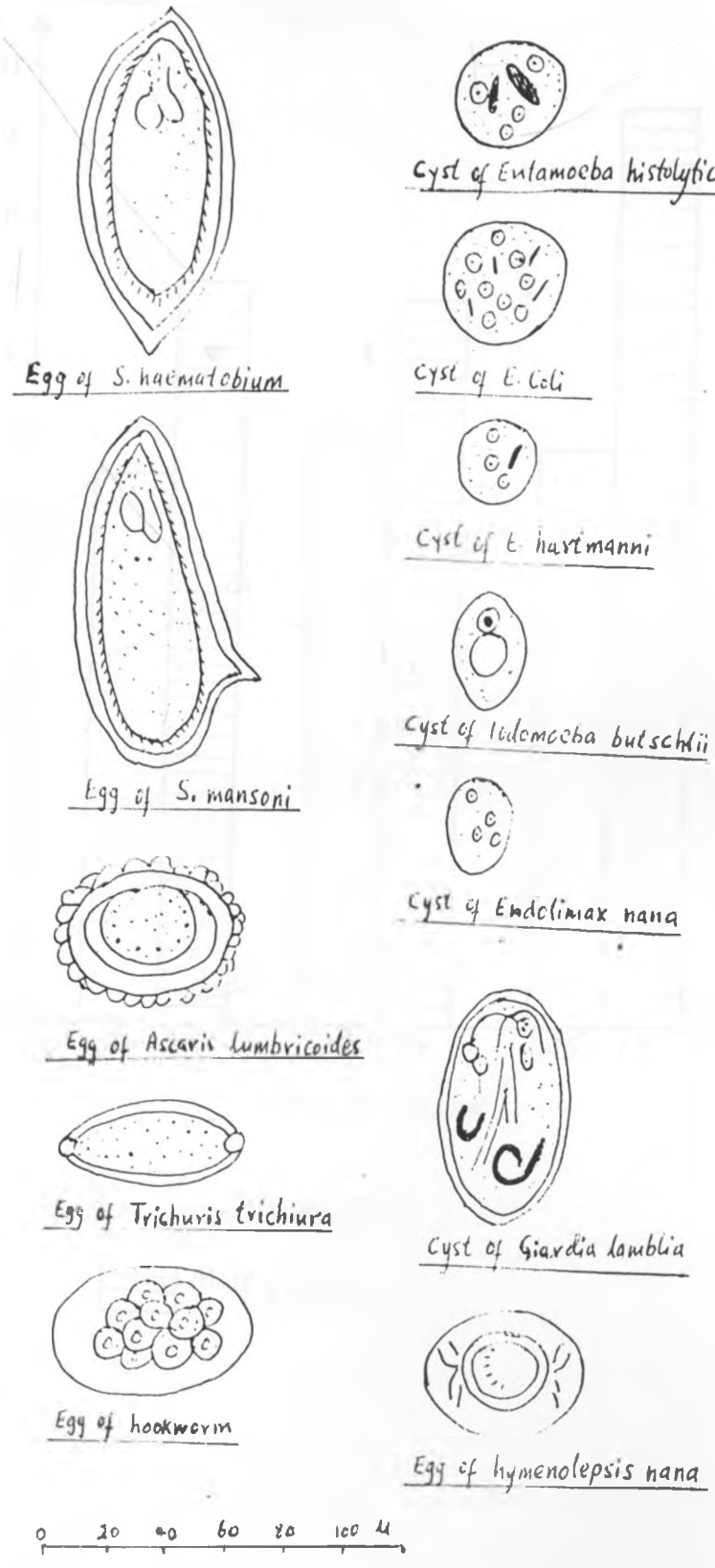
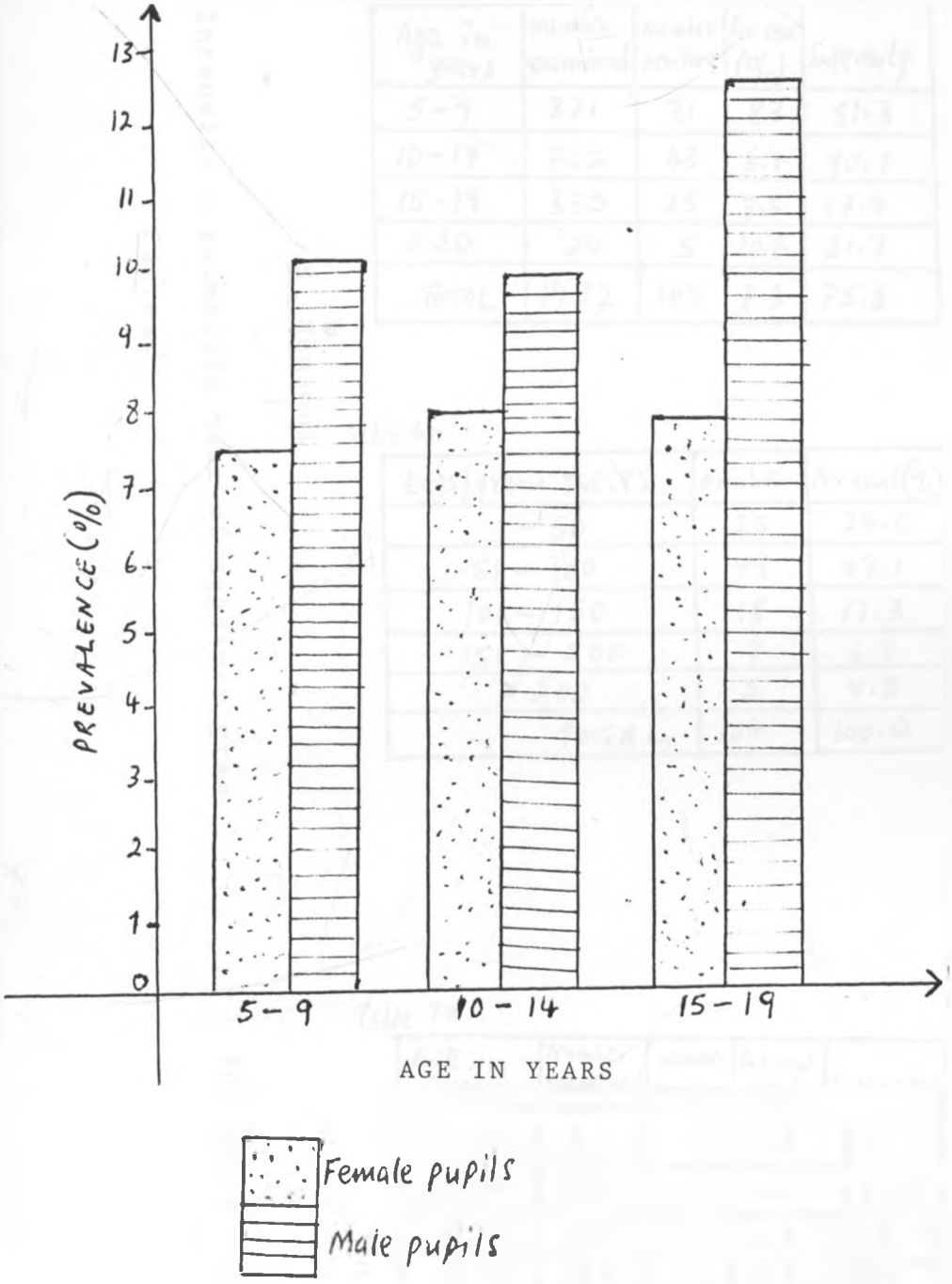


FIGURE 8 PREVALENCE OF SCHISTOSOMIASIS ACCORDING TO AGE AND SEX AMONG SCHOOL CHILDREN OF RUSINGA ISLAND.



INTENSITY OF SCHISTOSOMIASIS AMONG THE POSITIVE SCHOOL CHILDREN

Table 6a

Age in years	Number examined	Number positive	Per cent (%)	Intensity
5-9	371	31	8.3	51.3
10-14	702	43	6.1	90.7
15-19	335	25	7.5	77.4
≥20	24	5	20.8	81.7
TOTAL	1432	104	7.3	75.3

S. Mansoni

Intensity = geometric mean of eggs per gram faeces.

Table 6b

Eggs/gram faeces	Number	Per cent (%)
1 - 50	25	24.0
51 - 100	49	47.1
101 - 150	18	17.3
151 - 200	7	6.7
> 200	5	4.8
TOTAL	104	100.0

Table 7a

Age in years	Number examined	Number positive	Per cent (%)	Intensity
5-9	371	3	0.8	44.1
10-14	702	17	2.4	63.5
15-19	335	7	2.1	77.4
≥ 20	24	2	8.3	85.4
TOTAL	1432	29	2.0	67.6

Table 7b

Eggs/10ml urine	Number	Per cent %
1-25	3	10.3
26-50	5	17.2
51-75	8	27.6
76-100	9	31.0
>100	4	13.8
TOTAL	29	100.0

S. haematobium

Intensity = geometric mean of eggs per 10ml of urine.

FIGURE 9: INTENSITY OF SCHISTOSOMIASIS ACCORDING TO AGE AMONG INFECTED SCHOOL CHILDREN OF RUSINGA ISLAND

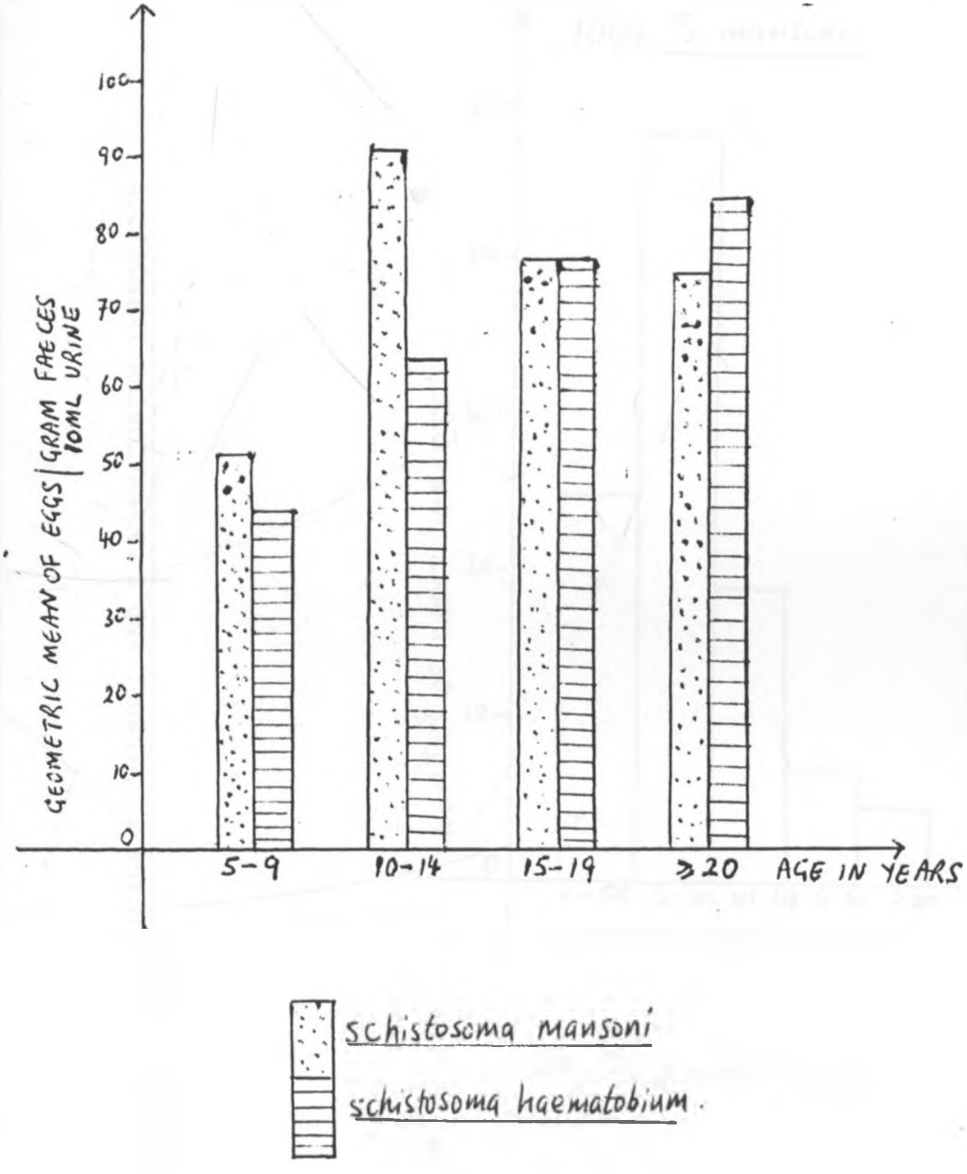
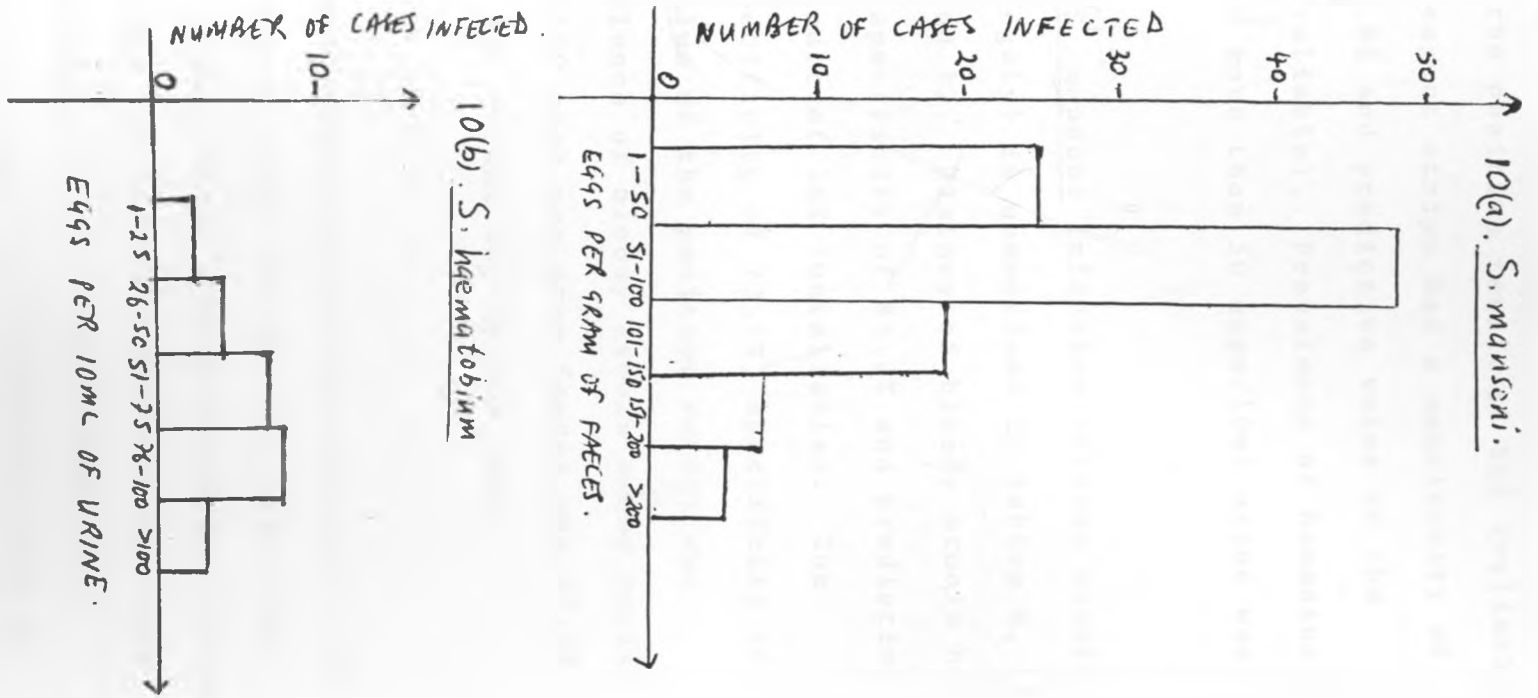


FIGURE 10 EGG COUNTS FOR POSITIVE S. MANSONI AND S. HAEMATOBIUM INFECTIONS



5.4. The morbidity due to schistosomiasis:

Morbidity due to S. haematobium infection (haematuria) is summarised in Tables 8(a), 8(b) and 8(c). History of haematuria had a sensitivity of 58.6%, specificity of 99.7% and predictive value of the positive result of 81% (reliable). Haematuria detected by reagent strips had a sensitivity of 82.8%, specificity of 99.6% and predictive value of the positive result of 80% (reliable). Prevalence of haematuria among those infected with more than 50 eggs/10ml urine was 95.5%.

Morbidity due to S. mansoni infection (bloody stools, hepatomegaly and splenomegaly) is summarised in Tables 9, 10 and 11 plus figures 11 and 12. History of bloody stools had a sensitivity of 36.5%, specificity of 85.2% and predictive value of the positive result of 16% (unreliable). The occult blood test had specificity of 73.1%, specificity of 84.4% and predicitive value of the positive result was 25% (unreliable). Prevalence of bloody stools among pupils infected with more than 100 eggs per gram faeces was 97.2% giving a statistically significant chi-square test ($X_1^2 = 68.72$; $P < 0.005$).

Prevalence of hepatomegaly among those infected with S. mansoni was 2.9% (3 pupils only) but all the three had more than 200 eggs per gram of their faecal specimens giving a statistically significant one tailed Fisher's exact test ($P = 5.5 \times 10^{-6}$). Hepatomegaly had a sensitivity of 2.8%, specificity of 99.5% giving the predictive value of the positive result only 30% (unreliable). Prevalence of hepatomegaly among pupils not infected with S. mansoni

was 0.5%. 70% of pupils with hepatomegaly were in the 10-14 year age group, 20% in the 5-9 year age group and 10% in 15-19 year old group giving a statistically non-significant one tailed Fisher's exact test (P = 0.47).

Splenomegaly had a general prevalence of 3.5% (1.9% among the S. mansoni infectives). Specificity of splenomegaly was 96.3% giving unreliable predictive value of the positive result at only 4%. 66.7% of those with splenomegaly were in the 5-9 year old group; 33.3% in the 10-14 year old group and none above 14 years of age. One of the pupils with S. haematobium infection had splenomegaly.

Morbidity due to mixed (double) infections is summarised in Table 12. Four of the pupils with mixed infections were males in the 10-14 year age group, the other was a female pupil aged 16 years.

Table 15 and figure 16 summarise cases of schistosomiasis from Kamayoge Primary School before and after treatment with praziquantel. Of the 67 positive cases treated in May, 58(86.6%) were re-examined in October and 53(91.4%) were found negative of schistosome eggs.

5.5. Possible effects of schistosomiasis on academic performance and weight for age:

There seemed to be no direct effect on academic performance and weight for age among the pupils examined in relation to the intensity of schistosoma infection. These findings are summarised in Tables 13, 14, and figures 13, 14 and 15.

The distribution of the infectives into the academic grades was not significantly related to the intensity of infection. For statistical analysis the table was reduced to 3X4 table by combining the last 2 eggs count categories: this gave a non-significant chi-square. This also applied to their distribution among the three different grade categories of weight for age (Chi-square =0.2302, df=2; $P > 0.10$). It is to be noted that the worm-loads among the infectives were too low to cause severe morbidity. Also, the gradings were done relative to the other pupils and it would be wrong to assume that those who were graded under grade A on academic performance were of very high intelligence.

5.6. Relative Contribution to Transmission of Schistosomiasis on the Island:

Table 17 and figure 16 illustrate water contact sites where potential snail intermediate hosts were collected. Nearly all the sites visited were being used by the Islanders for bathing, swimming, washing domestic articles like clothes and utensils, watering cattle, sheep and goats, and fishing. The majority were also surrounded by homes some of which had no pit latrines (for faecal disposal) indicating potential site contamination and active transmission of schistosomiasis.

There were potential intermediate snail hosts for both *S. mansoni* transmission (*Biomphalaria pfeifferi*, *B. choanomphala* and *B. sudanica*), and *S. haematobium* transmission *B(Ph) africanus*, *B. (Ph) nasatus* and

B (Ph) globosus). The snails collected were examined for the presence of schistosome infections (see appendix X) and 13 out of 26 specimen of Biomphalaria collected were found to be infected with cercariae which could not further be identified due to lack of facilities at the laboratory. None of the 78 specimens of Bulinus collected were found infected with cercariae. Nyamita stream provided 8 of the infected snail specimens and this correlated with the fact that the nearby school (Kamayoge) provided the highest number of infectives (67) giving a point prevalence of 32.7%.

Table 16 and figure 17 summarise the prevalences of S. mansoni during the 1974 epidemiological survey and the 1986 survey (this study) from the same schools. Generally there has been a considerable reduction of schistosomiasis prevalence (from 41.1% in 1974 to 5.9% in 1986) in the schools which were surveyed. Each of the six schools surveyed showed a downward trend.

TABLE 8: MORBIDITY DUE TO S. HAEMATOBIUM INFECTION (HAEMATURIA):

8(a) History of Haematuria

		Infection with S. haematobium		
		Present	Absent	TOTAL
History of Haematuria	Present	17	4	21
	Absent	12	1399	1411
	TOTAL	29	1403	1432

Sensitivity of the test = 58.6%

Specificity of the test = 99.7%

Predictive value of the positive result = 0.81 (reliable).

8(b) Haematuria by reagent strips.

		Infection with S. haematobium		
		Present	Absent	TOTAL.
Haematuria by Reagent Strips	Present	24	6	30
	Absent	5	1397	1402
	TOTAL	29	1403	1432

Sensitivity of the test = $\frac{24 \times 100}{29}$ = 82.9%

Specificity of the test = $\frac{1397 \times 100}{1403}$ = 99.6%

Predictive value of the positive result = 0.80 (reliable).

8(c) Intensity versus haematuria.

		Infection with S. haematobium		
		< 50 eggs/10ml urine	≥ 50 eggs/10ml urine	TOTAL
Haematuria by Reagent strips	Present	2	21	23
	Absent	5	1	6
	TOTAL	7	22	29

Prevalence of haematuria among those infected with more than 50 eggs/10ml urine = 95.5%

Chi-square (X_1^2) test = 13.45:

P< 0.005 (statistically significant).

TABLE 9:

MORBIDITY DUE TO S. MANSONI INFECTION

(BLOODY STOOLS):

9(a) History of Bloody stools.

		Infection with S. mansoni		
		Present	Absent	TOTAL
History of Bloody stools	Present	38	197	235
	Absent	66	1131	1197
	TOTAL	104	1328	1432

Sensitivity of this test = 36.5%.

Specificity of the test = 85.2%.

Predictive value of the positive result = 0.16 (unreliable).

9(b) Occult blood test.

		Infection with S. mansoni		
		Present	Absent	TOTAL
Bloody stools by occult blood test	Present	76	233	309
	Absent	28	1095	1123
	TOTAL	104	1328	1432

Sensitivity of occult blood test = 73.1%.

Specificity of occult blood test = 82.4%.

Predictive value of the positive result = 0.25 (unreliable).

9(c) Intensity versus Bloody stools.

		Infection with S. mansoni		
		<100 eggs/g stool	≥100 eggs/g stool	TOTAL
Bloody stools by occult blood test	Present	6 (7.9%)	70 (92.1%)	76
	Absent	26 (92.9%)	2 (7.1%)	28
	TOTAL	32	72	104

Prevalence of bloody stools among those infected with ≥ 100 eggs/g faeces = 97.2%.

Chi-square (X_1^2) test = 68.72: $P < 0.005$ (statistically significant).

TABLE 10:

MORBIDITY DUE TO S. MANSONI INFECTION (HEPATOMEGALY)

10(a) Hepatomegaly versus infection

	Infected	Not infected	TOTAL
Hepatomegaly	3	7	10
No hepatomegaly	101	1321	1422
TOTAL	104	1328	1432

Sensitivity of the test = 2.8%

Specificity of the test = 99.5%

Predictive value of the positive result = 0.3 (unreliable).

10(b) Infection versus age.

Age in years	Infected	Not infected	TOTAL
≤ 9	0	2	2
≥ 10	3	5	8
TOTAL	3	7	10

P = 0.47 (by one tailed Fisher's exact test).

There is no relationship.

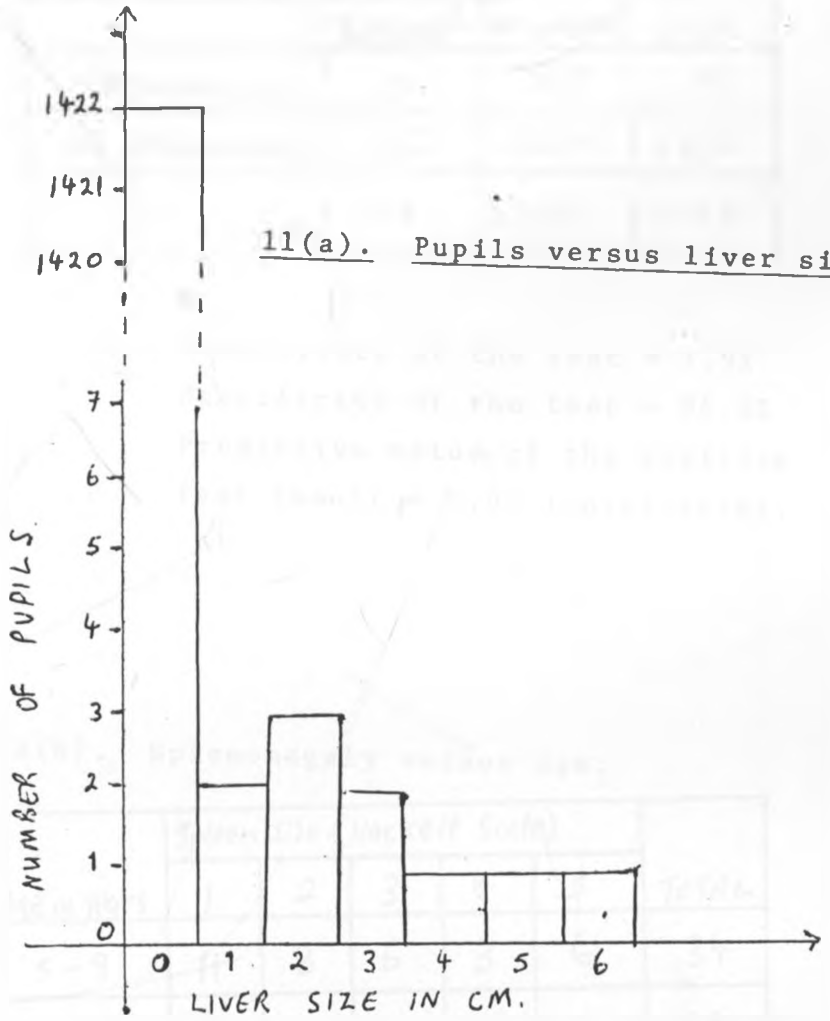
10(c) Hepatomegaly versus intensity of infection.

	Eggs per gram stool		
	≤ 200	≥ 201	TOTAL
Hepatomegaly	0	3	3
No hepatomegaly	101	0	101
TOTAL	101	3	104

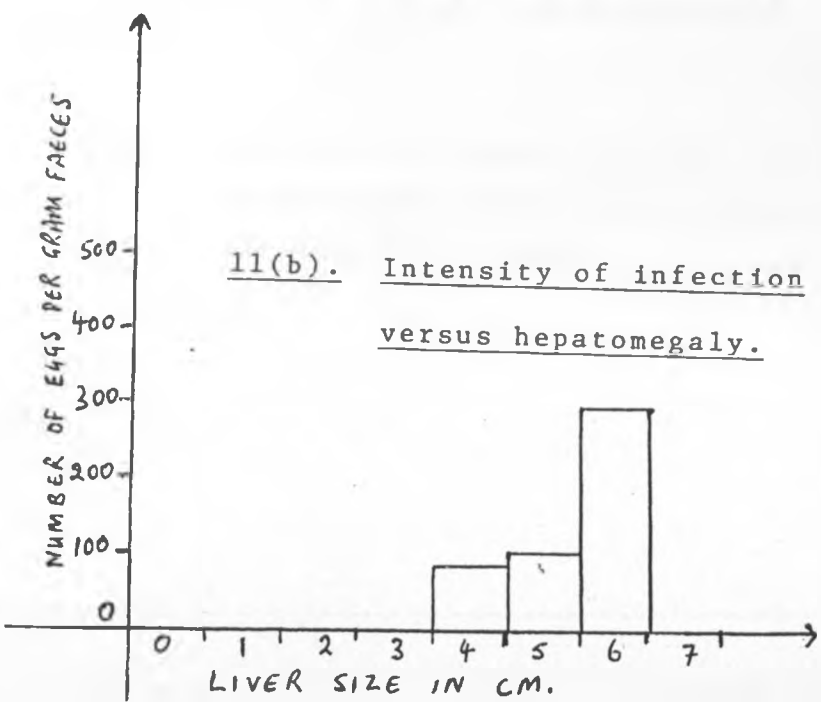
P = 5.5×10^{-6} (by Fisher's exact test - one tailed).

There is relationship.

FIGURE 11: LIVER SIZES OF PUPILS EXAMINED



11(a). Pupils versus liver sizes



11(b). Intensity of infection versus hepatomegaly.

TABLE 11:

MORBIDITY DUE TO S. MANSONI INFECTION (SPLENOMEGALY):

11(a) Splenomegaly versus infection.

	Infected	Not infected	TOTAL
Splenomegaly	2	49	51
No splenomegaly	102	1279	1381
TOTAL	104	1328	1432

Sensitivity of the test = 1.9%
Specificity of the test = 96.3%
Predictive value of the positive test result = 0.04 (unreliable).

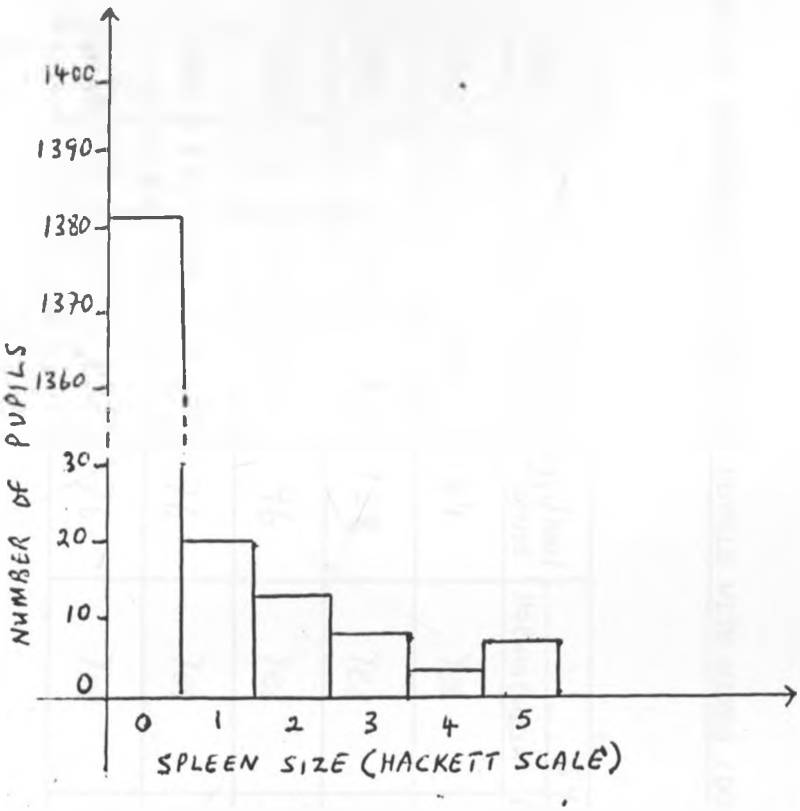
11(b). Splenomegaly versus age.

	Spleen size (Hackett Scale)					
Age in years	1	2	3	4	5	TOTAL
5-9	11	8	6	3	6	34
10-14	9	5	2	0	1	17
TOTAL	20	13	8	3	7	51

N.B: There was no single case of splenomegaly among pupils above the age of 14 years.

FIGURE 12: SPLEEN SIZES AMONG PUPILS EXAMINED

12(a) Pupils versus spleen sizes.



12(b) Splenomegaly versus age.

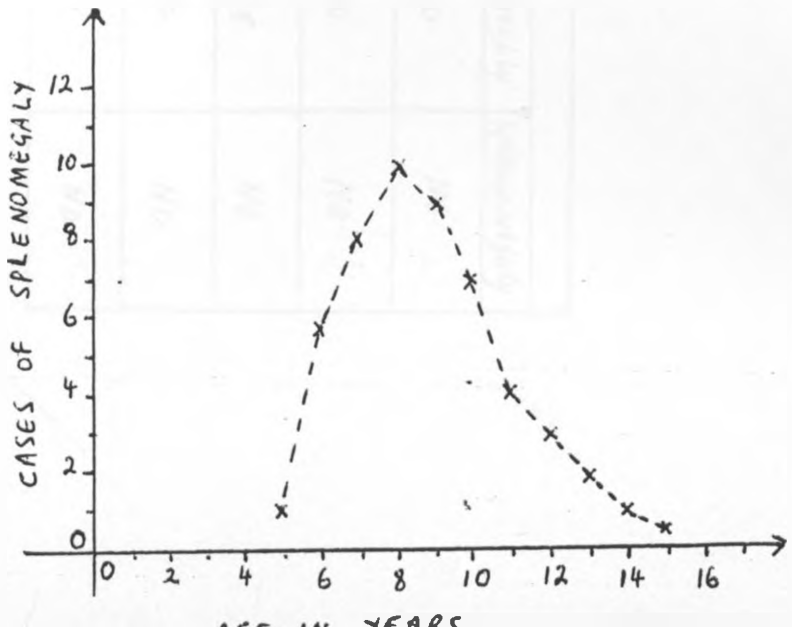


TABLE 12: MORBIDITY IN THE FIVE PUPILS WITH MIXED (DOUBLE) INFECTIONS:

Sex	Age in years	Wt in Kg	Eggs/g stool	Eggs/10ml urine	SYMPTOMS			
					Haematuria	Bloody stools	Hepatomegaly	Splenomegaly
Male	11	26	228	44	Yes	Yes	NO	NO
Male	11	30	116	128	Yes	Yes	NO	NO
Male	12	26	224	96	Yes	Yes	Yes	NO
Male	14	56	164	76	Yes	Yes	NO	NO
Female	16	70	132	76	Yes	Yes	NO	NO

TABLE 13:

CASES OF SCHISTOSOMIASIS IN RELATION TO ACADEMIC POSITIONS IN CLASS:

13(a) Intensity of infection versus academic grades.

CASES OF SCHISTOSOMIASIS	ACADEMIC GRADES			TOTAL
	A	B	C	
1-50eggs/g faeces 1-25eggs/10ml urine	10	8	6	24
51-100eggs/g faeces 26-50eggs/10ml urine	19	18	20	57
101-150 eggs/g faeces 51-75 eggs/10ml urine	12	11	10	33
151-200 eggs/g faeces 76-100 eggs/10ml urine	4	3	4	11
≥ 201 eggs/g faeces ≥ 101 eggs/10ml urine	3	2	3	8
TOTAL	48	42	43	133

No apparent relationship.

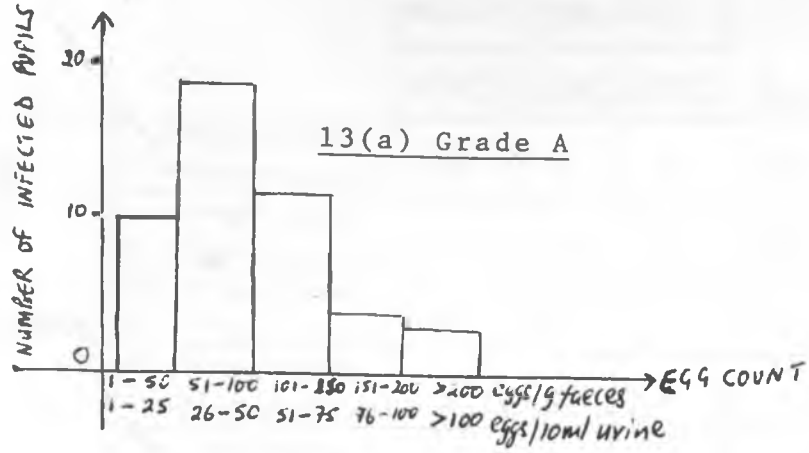
13(b) Infection versus academic grades.

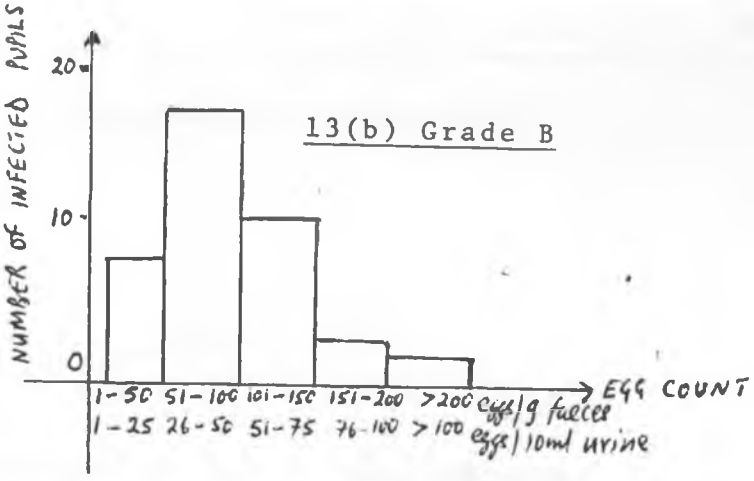
		ACADEMIC GRADES			TOTAL
		A	B	C	
Infection with Schistosomes	Present	48	42	43	133
	Absent	416	433	450	1299
	TOTAL	464	478	493	1432

Chi-square (X_1^2) test = 1.376 $P > 0.10$

(No relationship).

FIGURE 13: SCHISTOSOMIASIS IN RELATION TO ACADEMIC POSITIONS IN CLASS





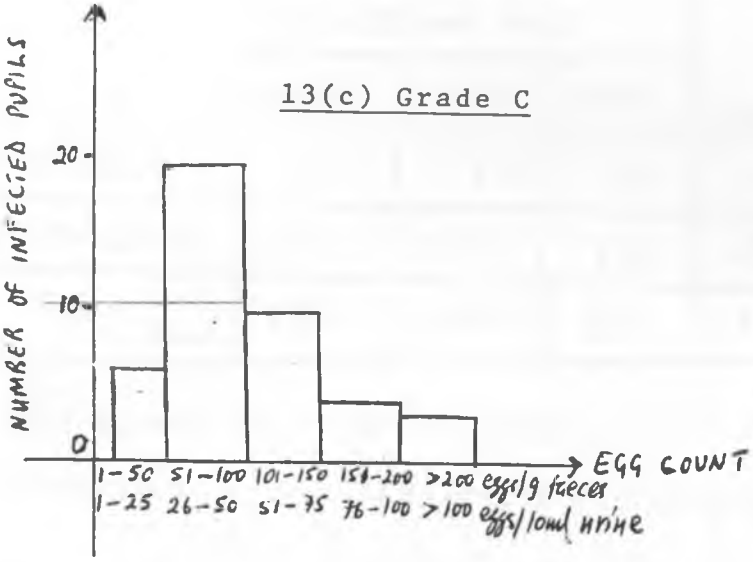


TABLE 14:

SCHISTOSOMIASIS IN RELATION TO WEIGHT FOR AGE

14(a) Intensity of schistosomiasis (infection) versus weight for age.

		WEIGHT FOR AGE			
		≥ 90%	75-89%	< 75%	TOTAL
CASES OF SCHISTOSOMIASIS	1-50 eggs/g faeces 1-25 eggs/10ml urine	16	11	1	28
	51-100 eggs/g faeces 26-50 eggs/10ml urine	27	24	3	54
	101-150 eggs/g faeces 51-75 eggs/10ml urine	12	16	1	29
	151-200 eggs/g faeces 76-100 eggs/10ml urine	6	5	1	12
	>200 eggs/g faeces >100 eggs/10ml urine	3	6	1	10
NON-CASES OF SCHISTOSOMIASIS		621	596	82	1299
	TOTAL	685	658	89	1432

No apparent relationship.

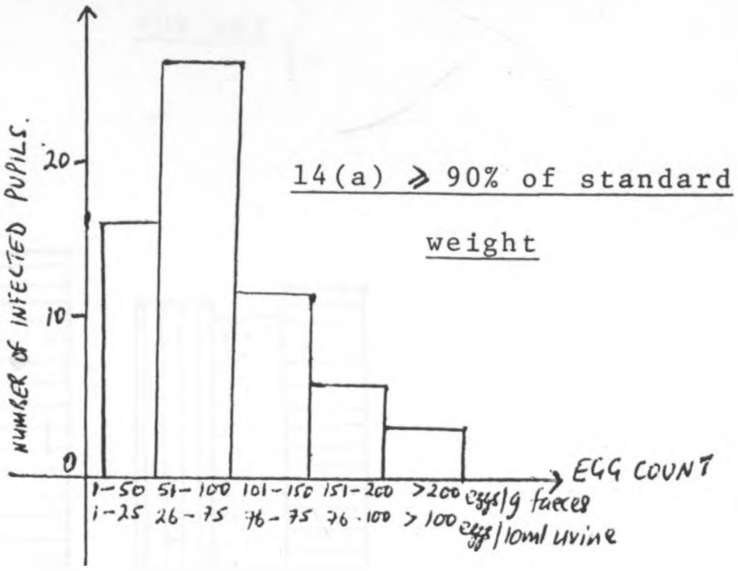
14(b) Weight for age versus infection

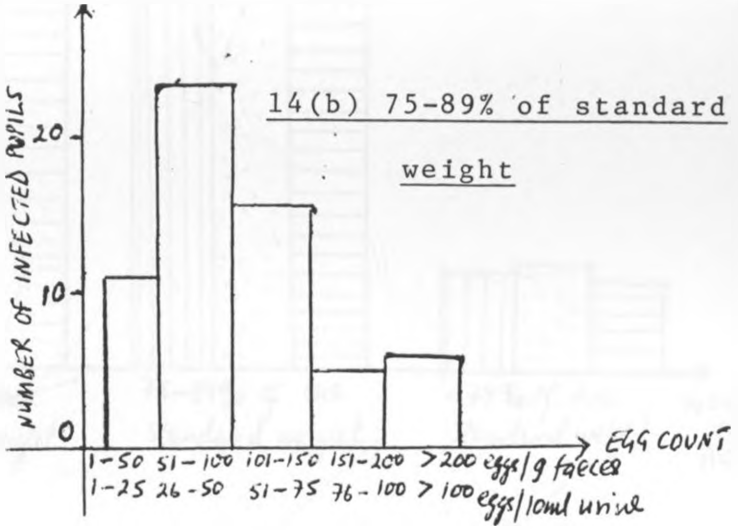
	WEIGHT FOR AGE			
	≥ 90%	75-89%	< 75%	TOTAL
Infected Cases	64	62	7	133
Non-infected cases	621	596	82	1299
TOTAL	685	658	89	1432

Chi-square (X^2_2) test = 0.2302 $P > 0.10$.

Statistically no significant difference.

FIGURE 14: SCHISTOSOMIASIS IN RELATION TO WEIGHT FOR AGE





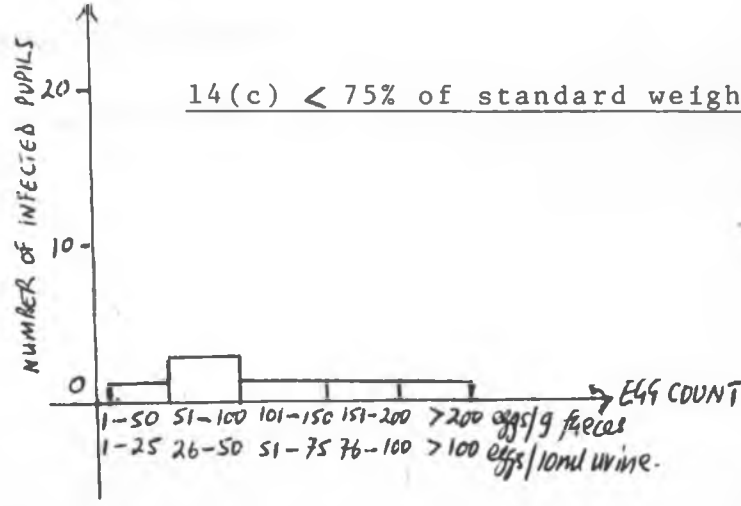


FIGURE 15: SCHISTOSOMIASIS IN RELATION TO WEIGHT FOR AGE

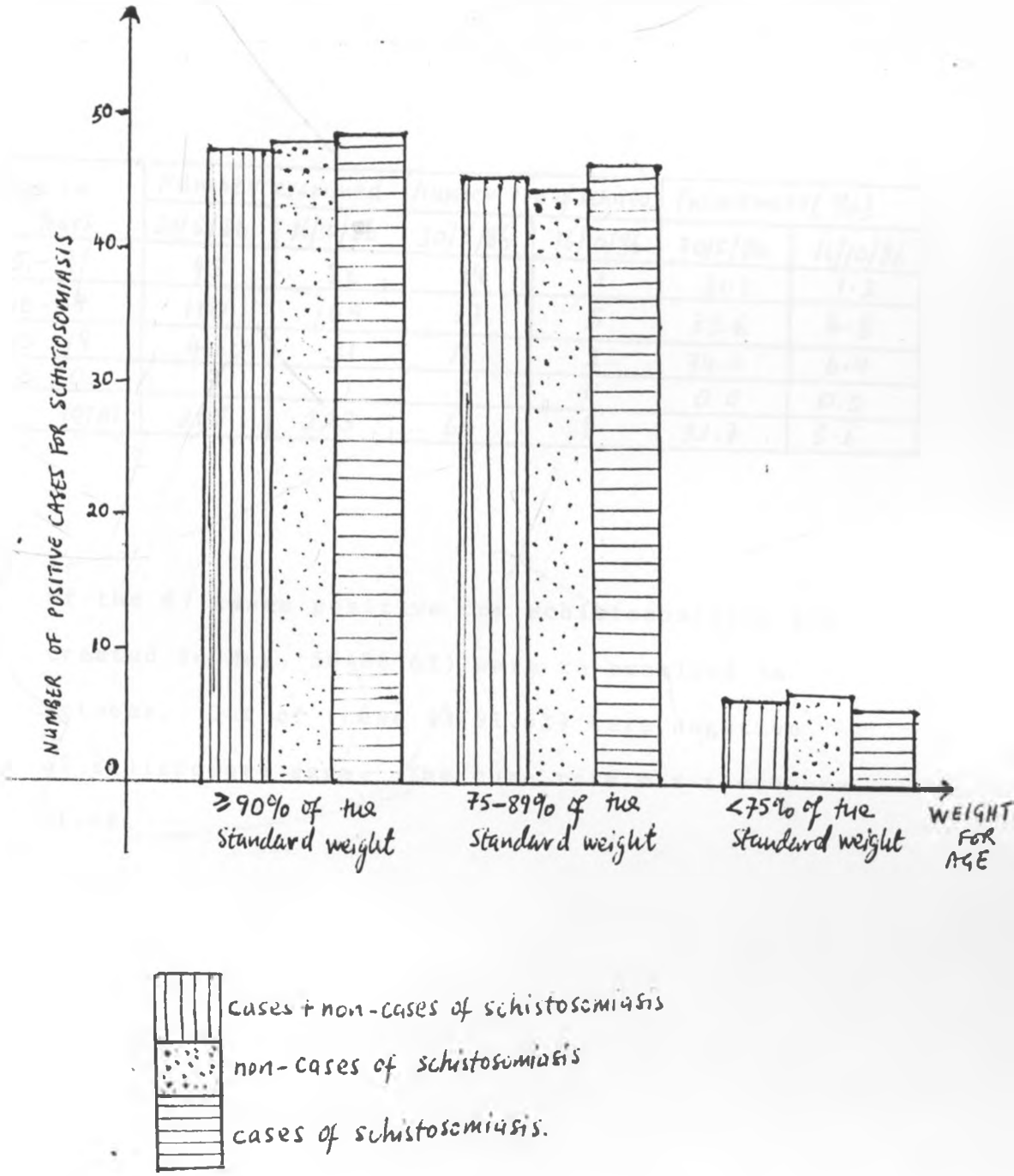


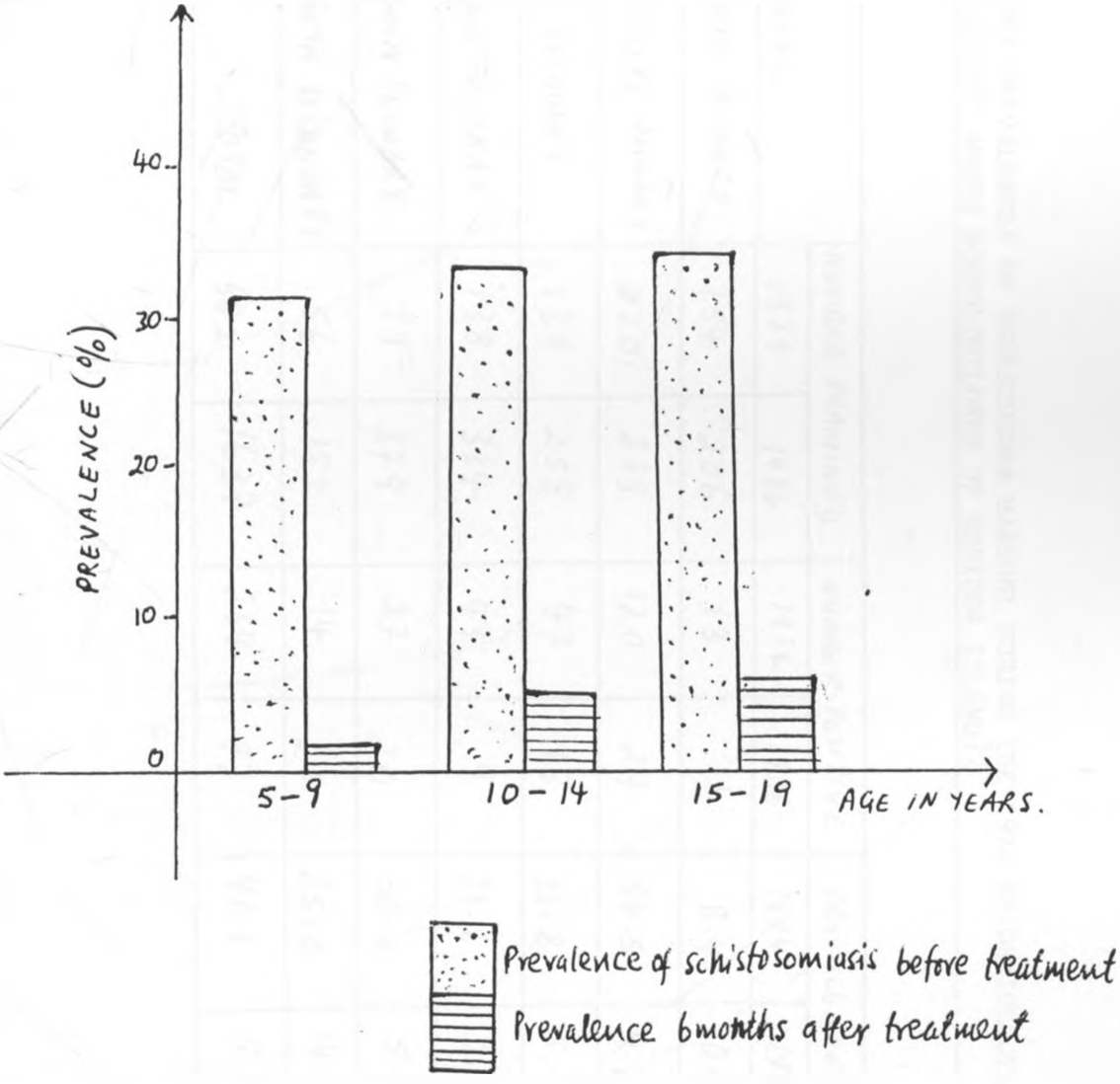
TABLE 15:

PREVALENCES IN KAMAYOGE PRIMARY SCHOOL BEFORE AND AFTER TREATMENT

Age in Years	Number examined		Number +ve of schisto.		Prevalences (%)	
	30/5/86	16/10/86	30/5/86	16/10/86	30/5/86	16/10/86
5 - 9	45	86	14	1	31.1	1.2
10 - 14	110	105	37	5	33.6	4.8
15 - 19	47	31	16	2	34.0	6.4
≥ 20	3	1	0	0	0.0	0.0
TOTAL	205	223	67	8	32.7	3.6

Of the 67 cases positive for schistosomiasis and treated in May, 58(86.6%) were re-examined in October. Out of these 53(91.4%) were negative of schistosome eggs. The cure rate was therefore, 91.4%.

FIGURE 16: PREVALENCE OF SCHISTOSOMIASIS AMONG SCHOOL CHILDREN OF KAMAYOGE PRIMARY SCHOOL, RUSINGA ISLAND, BEFORE AND AFTER TREATMENT WITH PRAZIQUANTEL

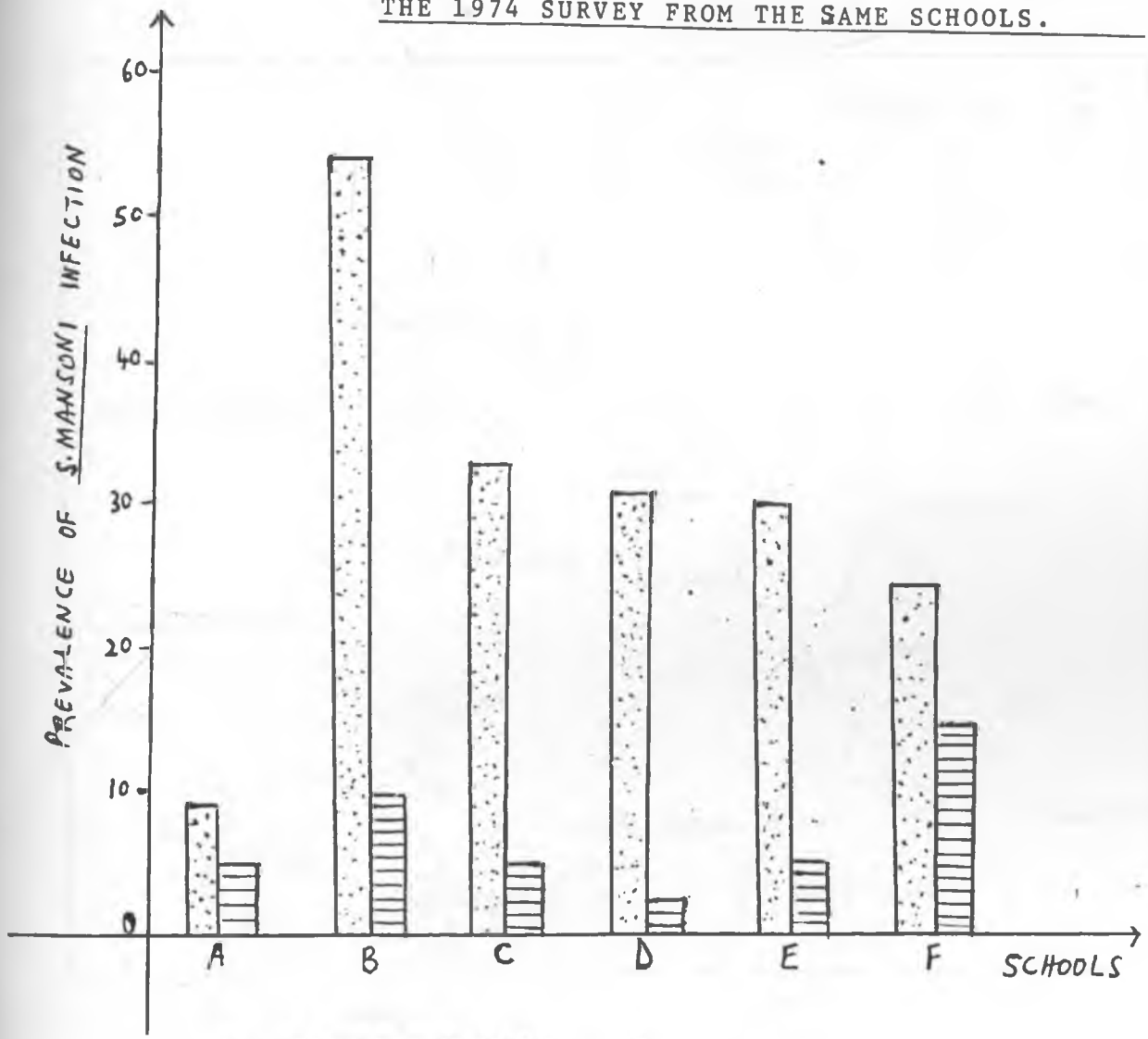


COMPARISON BETWEEN THE PREVALENCES OF SCHISTOSOMA MANSONI DURING THE 1974 EPIDEMIOLOGICAL SURVEY AND THE 1986 SURVEY AMONG SCHOOLCHILDREN OF RUSINGA ISLAND:

TABLE 16:

SCHOOL	NUMBER EXAMINED		NUMBER POSITIVE		PREVALENCE (%)	
	1974	1986	1974	1986	1974	1986
KASWANGA PRIMARY	154	206	13	1	8.4	0.5
KAMASENGERE PRIMARY	220	278	120	27	54.5	9.7
UTAJO PRIMARY	139	258	47	13	33.8	5.0
NYAMUGA PRIMARY	138	334	43	9	31.2	2.7
WANYAMA PRIMARY	89	379	27	20	30.3	5.3
TOM MBOYA SECONDARY	56	182	14	27	25.0	14.8
TOTAL.	642	1637	264	97	41.1	5.9

FIGURE 17: PREVALENCE OF S. MANSONI INFECTIONS DURING THE 1986 EPIDEMIOLOGICAL SURVEY FROM SIX SCHOOLS OF RUSINGA ISLAND AS COMPARED WITH THE 1974 SURVEY FROM THE SAME SCHOOLS.



A – KASWANGA PRIMARY

B - KAMASENGERE PRIMARY

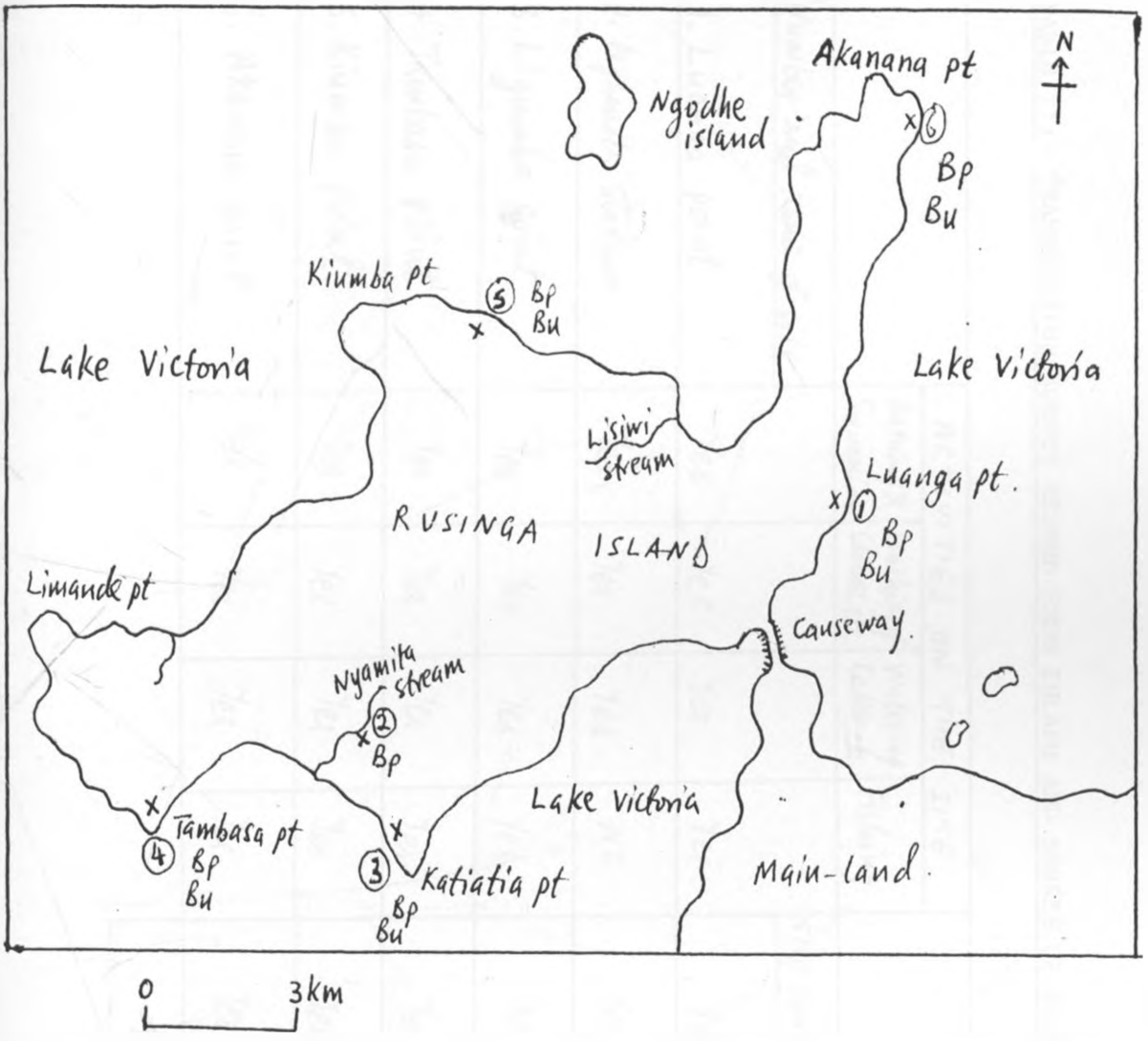
C - UTAJO PRIMARY

D - NYAMUGA PRIMARY

E - WANYAMA PRIMARY

F - TOM MBOYA SECONDARY

FIGURE 16: A SKETCH MAP OF RUSINGA ISLAND SHOWING HUMAN WATER CONTACT SITES WHERE SNAIL INTERMEDIATE HOSTS FOR SCHISTOSOMIASIS WERE COLLECTED.



Bp = Biomphalaria species – B. pfeifferi, B. sudanica and B. choanomphala

Bu = Bulinus species – B(Ph) africanus, B(Ph) nasatus and B(Ph) globosus

TABLE 17: TRANSMISSION SURVEY RECORD FROM INLAND AND SHORES OF RUSINGA ISLAND.

	ACTIVITIES ON THE SITE					INTERMEDIATE HOSTS (SNAILS)			
	Bathing & Swimming	washing of Clothes, etc	watering Cattle, etc	Fishing		Number collected		Number infected	
Number and Name of site					SITE CONTAMINATION	Bp spp	Bu spp.	Bp spp.	Bu spp
1. Luanga point	Yes	Yes	Yes	Yes	Yes	3	16	1	0
2. Nyamita stream	Yes	Yes	Yes	NO	Yes	12	0	8	0
3. Ligumba point	Yes	Yes	Yes	Yes	Yes	4	17.	2	0
4. Tambasa point	Yes	Yes	Yes	Yes	Yes	3	14	1	0
5. Kiumba point	Yes	Yes	Yes	Yes	Yes	0	13	0	0
6. Akanana point	Yes	Yes	Yes	Yes	Yes	5	18	1	0
					TOTAL	26	78	13	0

6. DISCUSSIONS AND CONCLUSIONS

6.1. The Prevalence of Schistosomiasis:

With the prevalence at 9.4% among the school children, Rusinga Island is a hypoendemic schistosomiasis area. This is because school children usually represent the age groups with the highest prevalence and intensity of schistosoma infection. Where transmission has been more or less constant for a number of years as is the case here, school children should provide good information on the overall prevalence and intensity of infection in the whole population.

The prevalence was highest among the male school children aged 15 - 19 years (12.6%) and lowest among the female aged 5 - 9 years. (7.5%). In schistosomiasis prevalence and intensity of infection are generally directly related and show usually similar patterns of variation with age. Generally both increase up to the 10-20 year-age group, followed by a decline. Factors responsible for the declining rates of prevalence and especially intensity, with increasing age, include decline in water contact with increasing age plus most probably slow development of resistance to infection. The sex related difference in prevalence may be explained by the differences in the frequency of water contact plus the duration and amount of body surface exposed to the water. In fact, during this study it was observed that more male children went to the lake to swim than the female ones. This relates well with Farooq et al. (1966)

who noted that infection rates of swimmers were more than double those of non-swimmers.

6.2. The Intensity of Schistosomiasis:

The intensity of schistosomiasis was lowest among 5 - 9 year age group (51.3 for S. mansoni and 44.1 for S. haematobium) and highest between 10 and 20 years (up to 90.7). This is comparable with the finding of Christensen et al. (1985) who noted that in areas where the endemic situation has remained essentially unchanged for many years, the intensity of infection usually rises to a maximum between the ages of 10 and 25 years.

6.3. Morbidity due to Schistosomiasis:

Morbidity due to schistosomiasis was marked in those with more than 50 eggs per 10ml of their urine specimens and more than 100 eggs per gram of the stool specimens. 95.5% of pupils with haematuria had more than 50 eggs/10ml of their urine specimens whereas prevalence of bloody stools among those infected with more than 100 eggs/g of stool was 97.2%. These compare well with Christensen et al. (1985) who observed that apart from haematuria, areas with low rates of prevalences and intensity of infection, urinary schistosomal disease is of no major medical importance. Cook et al. (1974) observed that S. mansoni is frequently symptom - free although blood in stools is normally associated with intensity of infection.

Only three pupils out of 218(2.9%) with S. mansoni had hepatomegaly. Kloetzel, (1963), Cook et al., (1974) and

Cheever, (1968) noted that while hepatomegaly is associated with intensity of S. mansoni infection, some individuals with light infection are found with severe disease and some habour heavy infections causing apparently little harm.

Splenomegaly in those infected with S. mansoni was only 1.9% and 3.7% among those uninfected. Prata and Shroeder, (1967),[13] Bina et al., (1978)[13], observed that splenomegaly is more common in caucasians than Negroes in spite of similar prevalence and intensity in the groups. Rusinga Island being a malaria endemic area should have persons with splenomegaly related to malaria. The majority of these were in the 5 - 9 year age group followed by 10 - 14 years age group and none above 14 years. This age distribution is most probably due to development of resistance to malaria among the older pupils. The extensive use of antimalarials might have contributed to the surprisingly low spleen rates.

Epidemiological information on the history of haematuria had a predictive value of 81% while screening the urine specimens from the same pupils for haematuria by reagent strips had a predictive value of 80%. Both methods would therefore be reliably used to assess the prevalence of S. haematobium infection when direct quantitative microscopic techniques are not feasible.

Morbidity due to S. mansoni infection was on the other hand rather unreliable in assessing the prevalence of infection. History of bloody stools had a predictive value of 16% while that of occult blood test was 25%. Hepatomegaly had a predictive value of 30% and that of splenomegaly was only 4%. These methods are therefore not valid to be used in

assessing the prevalence of S. mansoni infection in Rusinga Island where other intestinal parasites give rise to bloody stools and malaria gives rise to splenomegaly.

6.4. Academic Performance and Weight for Age:

The apparent absence of a direct effect on academic performance and weight for age among the pupils examined, in relation to the intensity of schistosomiasis is difficult to explain. What one has to realise is that the worm-loads among the pupils infected were generally too low to cause any severe morbidity. Heavily infected children experience disability and reduce scholastic abilities, but such effects are not pronounced in lightly infected children. In highly endemic areas, the physical working capacity of heavily infected adults are generally reduced. Such effects are much less pronounced in areas of low or moderate endemicity.[6]

Differences in susceptibility among races of man and/or differences in the pathobiological potential of different strains of S. mansoni may be responsible for the fact that severe S. mansoni - induced morbidity is often uncommon in Africa South of Sahara whereas it is a major health problem in Egypt and Sudan.

6.5. Transmission of Schistosomiasis on the Island:

Generally, temperature conditions are favourable throughout the year on the Island and this can sustain the snail host populations along the lake shores. But any transmission taking place inland is determined by rainfall pattern of the area which is between March and May each year.

With the low prevalence and intensity of schistosomiasis on the Island, availability of chemotherapy at the hospital and increased efforts in health education, it can generally be concluded that there is relatively little of schistosomal transmission taking place on Rusinga Island at present.

Under less intensive transmission conditions, loss of infection may exceed establishment of new infections resulting in declining prevalence rates. There has been also a reduction in the frequency of possible water contact activities due to the introduction of free primary school education. Over 75% of the school-going age are enrolled in the Island's schools and it is interesting to note that nearly every pupil examined during this tudy had scabies, a water-wash type of skin infection.

Most of the pupils who had S. haematobium infection were from the secondary school and temporary residents of the Island. With presence of potential intermediate snail hosts (Bulinus) transmission of S. haematobium might have been introduced into the previously free area.

7. RECOMMENDATIONS

Though this was a simple baseline descriptive study it has provided a foundation upon which effective future national control programmes for schistosomiasis could be planned. It is being recommended:-

1) - That another epidemiological survey is carried out one month after the 1987 long rains to determine the incidence of the infection.

2) - That since improved feasible methods for disease control can only be developed through applied research in the field, with the availability of manpower now in the districts, epidemiological surveillance of common local communicable diseases should be integrated in the rural health services.

3) - That there is a need for long-term repeated interventions if a final eradication of schistosomiasis from Rusinga Island is to be achieved.

4) - That since the study was carried out during the dry season when some intermediate snail hosts are known to aestivate (Bulinus nasatus and Bulinus globosus), there is a need for thorough malacology during the long rains in order to determine whether transmission of S. haematobium takes place on the Island or not.

From my findings, schistosomiasis is seen not to be a health problem for the Islanders at present but it should be monitored carefully so that any change from its present trend for the better is arrested promptly.

REFERENCES

1. Abraham, S.B., (1980).
Control of Communicable Diseases in Man.
The American Public Health Association, 13: 305-308

2. Appleton, C.C. (1986).
Annals of Tropical Medicine and Parasitology, 80 (3): 367-368.

3. Barlow, C.H. and H. E. Meleney, (1949).
American Journal of Tropical Medicine and Hygiene, 29, 79.

4. Cheever, A.W. (1968).
A quantitative post-mortem study of schistosoma mansoni in Man.
American Journal of Tropical Medicine and Hygiene, 17, 38-64.

5. Choudhry, A.W. (1974).
Bilharzia, Snails and Control in East Africa.
Danish Bilharzia Laboratory, Copenhagen, Denmark.

6. Christensen, N. and G. Gøtsche (1983).
Techniques for diagnosing urinary and intestinal schistosomiasis.
Danish Bilharzia Laboraory, Copenhagen, Denmark

7. Cook, J.A., (1974).
A controlled study of morbidity of Schistosoma mansoni in St. Lucian children based on quantitative egg excretion. American Journal of Tropical Medicine and Hygiene, 23, 625.

8. Farooq, M. (1967).
Progress in Bilharzia Control.
World Health Organization chronicle, 21, 175-184

9. Forsyth, D.M. and D.J. Bradley, (1974).
Irreversible damage of schistosoma haematobium in school children.
Lancet, 2: 169.

10. Haenens, G.D. and A. Santele, (1955).
Ann. Soc. Belg. Med. Trop., 35, 497.

11. Husting, E.L. (1983).
Human water contact activities related to transmission of bilharziasis (schistosomiasis).
American Journal of Tropical Medicine and Hygiene, 86: 23-35.

12. Iarotski, L.S. and A. Davis, (1981).
The schistosomiasis problem in the world: results of a WHO questionnaire survey.
Bulletin of the World Health Organization, 59: 115-127

13. Jordan, P. and G. Webbe, (1982).
Schistosomiasis: epidemiology, treatment and control.
William Heinemann Medical Book Ltd., London

14. Kinoti, G.K. (1971 a, 1971 b.)
Epidemiology of schistosoma mansoni and schistosoma haematobium on the Kano plains of Kenya.
Transactions of the Royal Society of Tropical Medicine and Hygiene, 65: 637, 646.

15. Kloetzel, K. (1963).
Some quantitative aspects of diagnosis and epidemiology in schistosoma mansoni.
American Journal of Tropical Medicine and Hygiene, 12, 334.

16. Kristensen, T.K., (1985).
WHO collaborating centre for applied malacology.
Danish Bilharzia Laboratory, Copenhagen, Denmark.

17. McMahon, J.E. (1967).
A study of some clinico-pathological manifestations in schistosoma mansoni infections in Tanzania.
Annals of Tropical Medicine and Parasitology, 61 (3): 302-309.

18. Mott, K.E., Dixon, H., Osei-Tutu, E. and E.C. England (1983).
Relationship between intensity of schistosoma haematobium infection and clinical haematuria and proteinuria. Lancet, 1: 1005-1008.

19. Pamba, H.O. (1974, 1977).
Schistosomiasis in Nyanza Province
East African Medical Journal, 51 (8), 4 (2-3).

20. Peters, P.A. El Elamy, M., Warren, K.S. and A.A.F. Mahmoud, (1980). Ouick Kato smear for field quantification of schistosoma mansoni eggs.
American Journal of Tropical Medicine and Hygiene, 29 217-219.

21. Pitchford, R.J. (1959).
Natural schistosome infections in South African rodents.
Transactions of the Royal Society of Tropical Medicine and Hygiene, 53: (2) 213

22. Pitchford, R.J., (1973).
Observations on the ecology of schistosoma mattheei.
Journal of South African Veterinary Association, 44: 405-420.

23. National Population Census (1979).
Central Bureau of Statistics
Government Printer, Nairobi, Kenya.

24. Ritchie, L.S. (1948).
An ether sedimentation technique for routine stool examination.
Bulletin of U.S. Army Medical Department, 8: 326-328.

25. Walkiers, J. (1928).
Ann. Soc. Belg. Méd. Trop., 8, 21

26. Wijers, D.J.B. and P.N. Munanga (1971).
East African Medical Journal, 48: 135.

27. World Health Organization (1980).
Epidemiology and Control of schistosomiasis
Technical Report Series No. 643, Geneva.

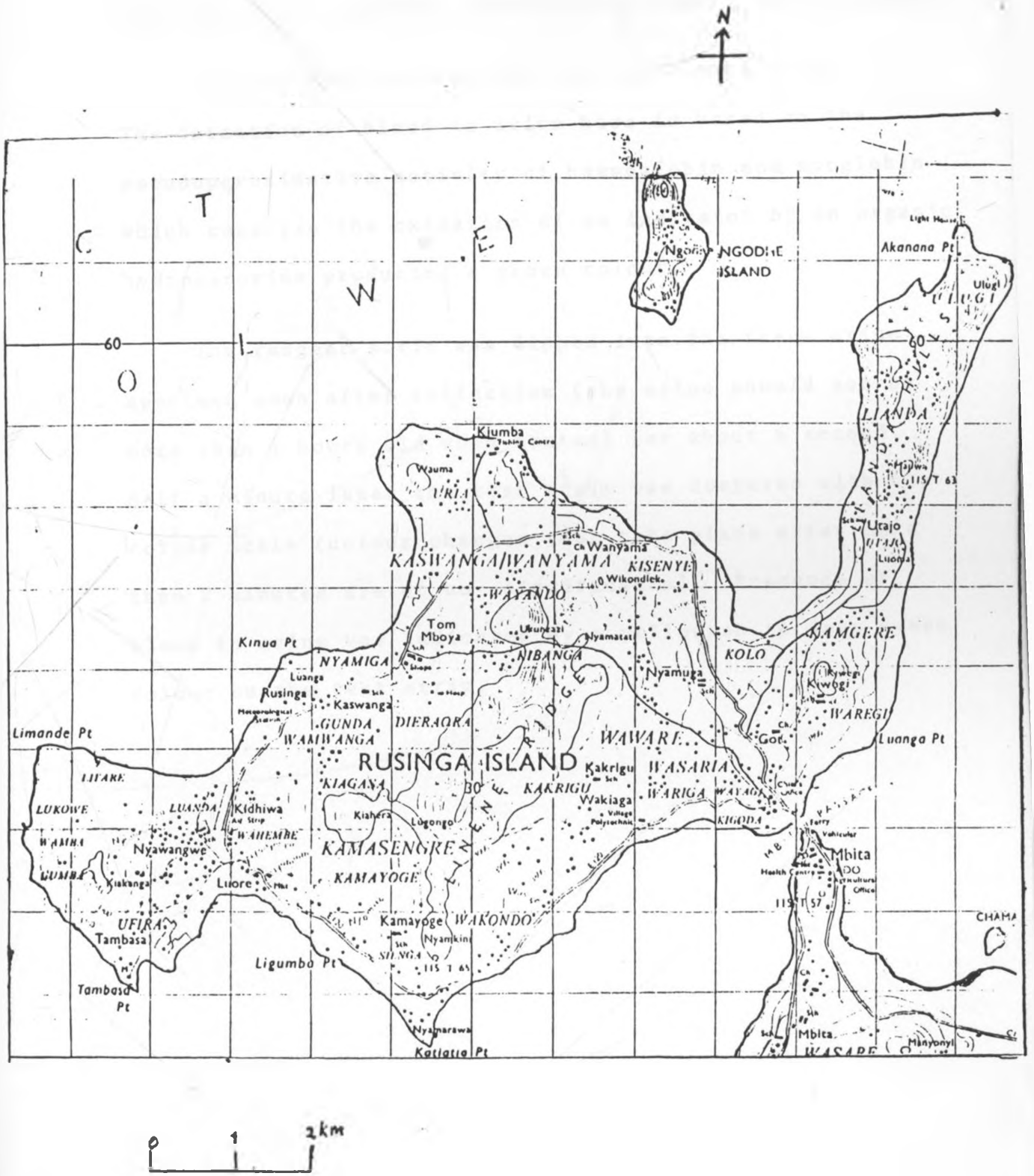
APPENDIX 1

DISTRICT: South Nyanza LOCATION: Rusinga Island SCHOOL: ..... CLASS:..... .. DATE: .....

WATER SOURCE:

SPECIMEN: NO:	Name	Sex	Age (Years)	Weight (kg)	Academic Position in class	Water Contact Bathing(B) Swimming(S) Washing(W) Fishing (F)	History of Haematuria	History of Bloody stoo	Liver Size (cm)	Spleen Size in Hackett Scale	Urine egg Count per 10mls.	Stool e Count p Gram

APPENDIX II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KISENYE
Wikondiek
WAYANDO
Tom Mboya
Kinua Pt
NYAMIGA
NIBANGA
Nyamuga
KOLO
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Kaswanga
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DIERAORA
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LUMBE
Kiakanga
Liore
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APPENDIX III

TEST FOR BLOOD IN URINE (HAEMATURIA) USING REAGENT STRIPS

The reagent strips used were the Combi 9 type. The detection of blood in urine here is based on the pseudoperoxidative activity of haemoglobin and myoglobin which catalyze the oxidation of an indicator by an organic hydroperoxide producing a green colour.

The reagent strip was dipped into the fresh urine specimen soon after collection (the urine should not be more than 4 hours old when tested) for about a second. Half a minute later the test strip was compared with the colour scale (colour changes that take place after more than 2 minutes are of no significance). Presence of blood in urine was indicated by a yellowish to deep green colour on the test strip.

APPENDIX IV

SEDIMENTATION TECHNIQUE (For diagnosis urinary schistosomiasis).

The procedure used was without centrifugation. A large urine sample was poured into a sedimentation glass (300 - 500 ml), and left for 30 minutes to settle. At the end of this time a deposit should be seen at the bottom of the glass (S. haematobium eggs should be in the deposit if the sample is positive) and without disturbing the deposit the supernatant urine was poured off leaving the deposit in a little urine.

Using the pasteur pipette, all the deposit was transferred onto the slides and covered carefully with coverslips. The preparation was then examined under a microscope with 50 - 100 X magnification for S. haematobium eggs.

APPENDIX V

SYRINGE FILTRATION TECHNIQUE (For diagnosing urinary schistosomiasis).

The filter membrane (only Nuclepore^{R} polycarbonate filters were available in limited numbers) was placed inside the filter holder using forceps then the filter holder re-assembled. The urine specimen was then mixed thoroughly (by drawing in and out of the syringe 10 times and 10ml withdrawn into a 10ml syringe. The filled syringe was connected to the wide end of the prepared filter holder with Nuclepore^{R} filter and the 10ml urine sample was slowly forced through the filter into a container with water and disinfectant. Removing the syringe from the filter holder, it was filled with air which was again injected slowly through the filter before the filter-holder was opened and the filter membrane removed using forceps.

The Nuclepore^{R} filter membrane was then placed on a microscope slide with the deposit (eggs?) facing downwards. With a pasteur pipette one drop of saline was placed onto the filter membrane and the specimen was then examined for S. haematobium eggs under a microscope with X 100 magnification. Before the next urine sample filtration, both the syringe and the filter holder were washed with detergent while the used Nuclepore^{R} filter was disposed of.

APPENDIX VI

OCCULT BLOOD TEST:

The reagent powder (Gregerson, Weber, Therenson-Rolland and Adler) reacts by detection of blood in faecal matter based on the pseudoperoxidative activity of haemoglobin and myoglobin which catalyze the oxidation of an indicator (benzedine) in the presence of oxygen (barium peroxide).

A small portion of reagent powder was dissolved in 3 - 4 ml of acetic acid (50%) then a portion of faecal specimen (size of wheat grain) placed on a microscope slide resting on a white piece of paper. A few drops of the dissolved re-agent was added onto the small faecal specimen placed on the slide. An appearance of blue colour indicated presence of blood in the faeces.

This is a very sensitive test that can detect 0.5 ml of blood added in 100 gm of faecal matter. As such pupils were adviced neither to eat meat nor to brush their teeth using hard tooth-brush, 48 hours before stool specimens were collected.

APPENDIX VII

FORMOL - ETHER CONCENTRATION TECHNIQUE (For diagnosing intestinal schistosomiasis).

Approximately 0.2 gram of faeces was placed in a mortar using an applicator stick. 7 ml of a 10% solution of formaline in saline was added and the faeces suspended in the formaline by using a pestle. Having placed one layer of surgical gauze in a small glass funnel, the suspension of faecal material was poured through the glass funnel into a 10ml glass centrifuge tube. The 3ml of ether was then added to the centrifuge tube which was stopped and shaken vigorously for 30 seconds before centrifuging the suspension at 5000 r.m.p. for 2 minutes.

Following centrifugation, four layers were distinguished in the tube: ether at the top separated from formaline by a fatty deposit and a pellet of deposit at the bottom. By loosening the fatty deposit at the interface of the liquids by use of an applicator stick, the three layers of supernatant were poured off without disturbing the deposit. A drop of Lugol's iodine was added to the deposit before transferring it to a microscope slide using a pasteur pipette. Having placed a coverslip, the preparation was examined under 50 - 100 X magnification microscope for S. mansoni eggs, plus eggs of other intestinal parasites as well as protozoal cysts.

APPENDIX VIII

KATO THICK SMEAR TECHNIQUE (For diagnosing intestinal schistosomiasis).

Coverslips made from cellophane strips (wettable) were soaked in a 50% gycerine - malachite green solution (100ml of gylcerine + 100ml distilled water + 1ml of a 3% aqueous malachite green solution) for at least 24 hours before use. To ensure that only smooth stool sample was used, any stool specimen containing fibrous material was placed on a piece of disposable paper then a screen mesh size 250μm was placed over it forcing the specimen through the screen.

A template (perforated plate which can contain exactly 50 mg faecal material in the hole) was then placed on a glass microscope slide and the template hole completely filled with smooth faecal sample using a small wooden spatula, levelling the faeces to the surface of the template. The template was carefully removed to leave all the faecal material on the slide and none sticking to the template.

The 50mg faecal specimen was covered on the slide with one of the soaked cellophane strips and any excess glycerine solution on the upper surface of the cellophane wiped off with a small piece of absorbant paper. The slide was inserted against a smooth surface and the faecal material spread evenly under the cellophane by pressing it against the surface. The specimen was left for clearing for at least 24 hours before examining for S. mansoni eggs on a

microscope with 50 - 100 X magnification. The number of schistosome eggs counted in the specimen was multiplied by 20, to obtain the number of eggs per gram of the patient's faeces.

APPENDIX IX

THE HACKETT SCALE:

The spleen size was measured by palpating the spleen edge below the left anterior axillary line according to the Hackett Scale:-

0 = Normal spleen, not palpable on deep inspiration.

1 = Spleen palpable only on deep or at least more than normal inspiration.

2 = Spleen palpable on normal breathing but not projected below a horizontal line half-way below the costal margin and the umbilicus measured along a line dropped vertically from the left nipple.

3 = Spleen with the lowest palpable point projected more than half-way to the umbilicus but not below a line drawn horizontally through it.

4 = Spleen with the lowest palpable point the umbilical level but not projected more than half-way towards a horizontal line through the symphysis pubis.

5 = Spleen with lowest palpable point below the lower limit of class 4.

APPENDIX X

EXAMINATION OF SNAILS FOR PRESENCE OF SCHISTOSOME INFECTION:

The field collected snails were examined for schistosome infections using the simplest technique applicable to a rural laboratory without enough rodents.

Each snail was placed into a beaker containing 10 ml of fresh water and the beaker placed in natural sunlight from 10 a.m. to 2 p.m. (4 hours). Using a pasteur pipette with a rubber bulb a drop of water from the beacher was placed onto a microscope slide and examined for presence of cercariae using a microscope with 10 - 40 X magnification.