AN ABATTOIR SURVEY ON THE OCCURRENCE AND PREVALENCE OF SWINE PARASITIC HELMINTHS OF PUBLIC HEALTH AND ECONOMIC IMPORTANCE IN KENYA

BY

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Declaration

This project report is my original work and has not been presented for a degree in any other University.

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Dedication

This project report is dedicated to my parents Andrew and Sarah, my wife Lydiah, son Enock and daughter Ruth.

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Abstract

Although there have been systematic and well coordinated efforts in documenting the prevalences of parasitic diseases of the pig in several countries of the world, very little has been done in Kenya. Parasitic helminths, specifically, have negative impacts on the health of the pigs, health of the consumers of pork and other products and the economics of the swine industry. The present study focused mainly on those parasites encountered during routine post-mortem meat inspection in Kenya with emphasis on those of public health and economic importance.

Using records kept by the Ministry of Agriculture and Rural Development, a retrospective abattoir survey was carried out to determine the occurence and prevalences of the various pig parasitic helminths of public health and economic importance encountered during the routine abattoir meat inspection over a ten year period (1988-1997) in Kenya. In addition, a cross-sectional survey was carried out in Nairobi's Farmers' Choice and Ndumboini abattoirs where routine physical meat inspection was done. Faecal and diaphragm muscle samples were collected for laboratory faecal egg analyses and trichinoscopy respectively. Faecal samples were quantitatively analysed for faecal helminth egg counts (eggs per gramme of sample) using the McMaster Technique, while the diaphragm muscle samples were analysed by trichniscopy for *Trichinella spiralis* larvae.

A total of 642,295 pigs were slaughtered during the 1988-1997 period. Out of these, 31,365 (4.9%) had *Ascaris suum* (indicated by the presence of milk spots in the liver), 4,733 (0.7%) had hydatid cysts in the liver, lungs and spleen, 215

(0.03%) had lung worms, 165 (0.03%) had liver flukes and 38 (0.01%) had nodular worms belonging to *Oesophagostomum dentatum* and 23 (0.004%) had *Cyticercus cellulosae*.

The cross-sectional post-mortem meat inspection performed revealed that *Ascaris suum* had a prevalence of 27.1%, hydatid cysts had 5.7% while *Oesophagostomum dentatum* had a prevalence of 5.7%. On quantitative fecal egg counts of strongyles, *Ascaris suum*, *Trichuris suis* and coccidia showed prevalences of 50.9%, 10.7%, 3.6% and 15.2% respectively. *Trichinella spirallis* was never detected.

These results therefore show that *Ascaris suum* had the highest prevalence. Generally, the prevalences of the other pig parasitic helminths were low.

The presence of these parasites which are also of public health (zoonotic) importance in this country therefore calls for interventionary efforts in order to avert potential health hazards to consumers of pork and its products.

CHAPTER ONE

1.0. Introduction

Helminths constitute the major parasites of the pig with economic and public health importance. These Parasites have negative impacts on the health of the pigs, economic returns of the owners and health of the consumers. These parasites have been reported worldwide and have been known to adversely affect the swine industry. The life cycles of a number of these parasites and their economic impacts on pig production have been widely studied and documented (Paiaro, 1993).

Specifically, major internal parasites of swine include the helminths (nematodes, trematodes, cestodes and acanthocephala) and protozoa. Parasites of public health importance include *Taenia solium, Trichinella spiralis, Toxoplasma gondii* and cystic *Echinococcus granulosus* (Gamble, 1997).

Several surveys have been conducted to determine the occurences and prevalences of these internal parasites in various countries. High prevalences have been recorded among the production systems which are unhygienic and poorly managed (Ayaji and Arabs, 1988). Recent studies have shown that superior housing and hygiene in combination with routine antihelminthic usage have led to decreasing prevalences of these parasites (Pattison, *et al.*, 1980b; Morris *et al.*, 1984; Roepstorff and Jorsal, 1990; Dangolla *et al.*, 1997).

The most common intestinal parasites of swine have been shown to be unevenly distributed among different age groups (Pattison *et al.*, 1980a; Morris *et al.*, 1984: Roepstorff and Jorsal, 1989). *Isospora suis* and *Strongyloides*

ransomi are common in piglets, *Ascaris suum* and *Trichuris suis* in growers, and *Oesophagostomum* spp., *Hyostrongylus rubidus* and *Eimeria* spp. in adults. These age-dependent distributions are probably caused by different host-parasite relationships, especially the immunogenicity of the parasite (Pattison *et al.*, 1980a; Murrell, 1986; Roepstorff and Nansen, 1994).

Several investigators have studied different aspects of swine parasites in different countries of the world. Ferreira and Borges-Ferreira (1984), reviewed the prevalence, pathogenicity and economic importance of helminths of swine in Portugal. Andersen (1976), showed that the negative influence of *Ascaris suum* upon growth is caused by both the larvae during migration through the lungs and liver and by the presence of adult worms in the intestines. Wagner and Polley (1997), conducted an abattoir survey of market pigs in Saskatchewan to determine the prevalence and intensity of *Ascaris suum*. Further the prevalence of parasites associated with pork and pork products and their public health risks were studied by Gamble (1997) and recorded that *Trichinella spiralis* and *Taenia solium* are the most prevalent. Boretti *et al.* (1987), carried out a survey of parasites and diseases of pigs in the Mata de Ponte Nova in Brazil and reported that helminths are the most prevalent. Salivu, *et al.* (1990), carried out a survey of gastrointestinal parasites in pigs in Nigeria where high rates of infection were attributed to the poor management practices and poor sanitary conditions. The prevalences of intestinal parasites in pigs and cost benefit analysis of different treatments were studied by Bavia *et al.* (1987). The workers found that all antihelminthic treatments gave a favourable cost benefit analysis.

An abattoir survey of gastrointestinal parasites of pigs was conducted in Malaysia by Lee *et al.* (1987). In this survey, the overall prevalence of parasites was 85%. Prevalences of parasites of pigs transmissible to humans in New Zealand were studied by Fairley (1996) while Du-Yupan *et al.* (1995), investigated the parasites of pigs in Guizhou province of China. In Nordic countries (Denmark, Finland, Iceland, Norway and Sweden), *Ascaris suum, Oesophagostomum* spp., *Isospora suis* and *Eimeria* spp. are common intestinal parasites of swine whose prevalences and geographical distributions were studied by Roepstorff *et al.*(1998). Large fatteners and gilts are most frequently infected with *Ascaris suum* with maximum prevalences of 25-35% in Denmark, Norway and Sweden, 13% in Iceland and 5% in Finland (Roepstorff *et al.*, 1998). Studies carried out in Tanzania showed that pig parasites occurs with *Oesophagostomum* species having a prevalence of 40%, *Ascaris suum* (12%), *Strongyloides ransomi* (9%) and *Trichuris suis* (5%) (Esrony *et al.*, 1997).

There have been systematic and well coordinated efforts directed at documenting the prevalences of parasitic diseases of pigs in several countries, but very little has been done in Kenya. Ng'ang'a (1974) studied the situation of pig hydatidosis covering the 1959-1970 period while Gathura *et al.* (1988), carried out a survey on the prevalence of hydatidosis for an 11 year period, 1977 to 1987. However no study has been carried out to determine various aspects of the various helminths infecting pigs in Kenya.

1.1. Objectives

The objectives of this study were to carry out a retrospective abattoir survey on:

(i) the occurrence,

(ii) prevalence of helminthic infections in pigs slaughtered in Kenya over a ten-year period (1988 to 1997) with emphasis on those of public health and economic importance and

(iii) to show the temporal trends of these infections over this period.

CHAPTER TWO

2.0. Review of literature

2.1. Parasitic helminths of swine

An overview of the life cycles and pathogenesis of the common helminths of the pigs has been done by Murrell (1986). The parasites have attracted allot of interest worldwide in the recent past due to economic and public health implications in the swine industry.

2.1.1. Nematodes of the swine

These constitute the most important group of the parasites of the swine.

2.1.1.1. *Ascaris suum* (the large roundworm of the pig)

Globally *Ascaris suum* is one of the most prevalent parasites in swine. It commonly occurs in highly intensive production systems (Roepstorff and Nansen, 1994). *Ascaris suum* are large , cream-coloured roundworms found in the small intestines. They can also be found in the bile ducts, stomach and large intestines. Normally, the adult worms in the small intestine are limited to only five to ten worms. However, they usually live for long periods and produce large numbers of eggs (Olson *et al.*,1958). The adult females may measure up to 12 inches long and a thickness of about one-quarter inch. The adult males are comparatively shorter and thinner. The larval stages can be found in several tissues throughout the body, but mainly in the liver and lungs (Murrell, 1986).

Morphologically, *Ascaris suum* is identical to the human parasite, the

Ascaris lumbricoides. However they differ in their physiological requirements (Abdulrachman and Joe, 1954). Antigenic analysis has shown that *A. suum* shares 11 antigens with *A. lumbricoides* (Imperato *et al.*, 1968). Some biochemical differences have also been reported (Kurimoto, 1974). Observattions from experimental and accidental laboratory infections have shown that the 'pig' form can infect man (Lysek, 1967) and the 'human' form can develop in pig (Galvin, 1968). Examination of the isoenzymes in the two species with gel electrophoresis showed 14 loci that were monomorphic in *Ascaris suum* and were identical in electrophoretic mobilities in *A. lumbricoides* (Nadler, 1987). *Ascaris suum* can also affect apes, cattle, sheep and squirrels (Soulsby, 1982).

Estimates of daily *Ascaris suum* female egg production generally are in the range of 200,000 eggs (Brown and Cort, 1927; Sinniah, 1982). The eggs have a thick shell which is considerably resistant to environmental factors (Roepstorff and Murrell, 1997). Embryonation and larval development are dependent on temperature. In temperate regions the development occurs mainly in summer season (Connan, 1977; Stevenson, 1979).

The pigs become infected by ingesting the embryonated infective eggs along with food and water or in case of suckling piglets, infection comes from contaminated skin of the sow. The eggs hatch in the small intestine (largely in the duodenum) under the influence of intestinal conditions, especially partial pressure of carbon dioxide (pCo_2) (Clarke and Perry, 1988). The second stage larvae(L_2) penetrate the intestinal wall and are carried to the liver through the

blood stream and then to the lungs. It has been reported that the L_2 larvae exclusively invade the walls of the caecum and colon, and not the small intestine as is generally believed (Murrell *et al.*, 1997). The larvae are arrested in the capillaries and some may pass through and be carried by the circulating blood to other organs. The larvae in the lung capillaries may escape into the alveoli and pass up the trachea to the pharynx. They are then swallowed and pass down the oesophagus into the stomach. In the stomach, they grow into adult worms and move to the small intestines (Olson *et al.*, 1958).

In heavy infestations, there may be vomiting, impaction of the bowel due to obstruction of the intestinal lumen by worms, jaundice, anaemia, emaciation and pendulous abdomen (Bernardo *et al.*, 1990b). The presence of larvae in the lungs may cause pneumonia which would be characterised by coughing with some exudation. Growth is stunted and the animal is unthrifty. The young pigs may show neurological signs (Soulsby, 1982). The negative impact of *Ascaris suum* upon growth is caused both by larvae during migration through the lungs and liver and by the presence of adult worms in the intestine (Andersen, 1976).

During post-mortem examination, apart from presence of adult worms in the intestines, pathological lesions are found in the liver and lungs. Roneus (1966), produced the first extensive study of the liver "white spots". He indicated that there are two types, namely the "White spots" caused by the granulating tissue due to the migration of larvae and the lymphonodular type which consists of lymphocytes, fibroblasts and histiocytes.

Ascariosis is mainly diagnosed during post-mortem meat inspection by

observing the presence of adult worms in the small intestines, the characteristic lesions in the liver and by the characteristic ascarid eggs in faecal samples (Pattison *et al.*, 1980b; Morris *et al.*, 1984; Roepstorff *et al.*, 1988).

Infections with *Ascaris suum* may stimulate the development of strong protective immunuty (Eriksen, 1981; Eriksen *et al.*,1992a,b), which depends on the level and length of exposure period. In single infections, it has been shown that the number of established adult worms may be negatively correlated to the size of the inoculated dose (Andersen *et al.*, 1973; Jorgensen *et al.*, 1975). Pigs heavily exposed over some months, incoming larvae may be killed by acquired immunity even before they reach the liver (Urban *et al.*, 1988; ; Eriksen *et al.*, 1992a). There is limited resistance, as the larvae easily migrate and establish in parasite naive baconers and sows (Eriksen *et al.*, 1992a).

Piperazine has been widely used as the drug of choice in the treatment of ascariosis in pigs. Pyrantel tartate at 22mg /kg body weight, Levamisole at 8mg/kg body weight and Hygromycin B have also been used. Ivermectin has been shown to be 100% effective against ascariosis (Ottesen, 1990).

2.1.1.2. *Oesophagostomum* (Nodular worm of the pig)

These nodular worms include, *Oesophagostomum dentatum, O. quadrispinulatum, O. brevicaudum* and *O. georgianum* (Roepstorff, 1986; Talvik *et al.*, 1997). *O. dentatum* is the most common species. They all parasitise different parts of the large intestine (Jacobs, 1967). Infections with nodular worms are widespread among pigs throughout the world (Urquhart *et al.*, 1996).

The major characteristic of the parasites belonging to genus *Oesophagostomum* is the cervical groove, which is a transverse cuticular depression extending laterally for varying lengths. The eggs are typical thin-shelled strongyle eggs which are segmented when laid (Soulsby, 1982). The nodular worms have a direct life cycle. Experimentally, the worm burden in the caecum and colon of infected pigs have been reported to range from 5,000-15,000 (Pattison *et al.*, 1979, 1980a). The number of larvae present in the large intestinal mucosa are higher (Rose and Small, 1980a). Infections with nodular worms stimulate limited immunity which moderates the intestinal worm burden (Pattison *et al.*, 1979; Roepstorff *et al.*, 1987a).

The females are highly fecund, and in heavily infected sows may have epgs of 3,000-14,000 without showing clinical signs (Roepstorff, 1991). Once passed out in feces these eggs hatch and develop into the infective third stage larvae (L_3). The latter can be ingested by the pigs with feed or water. Both the eggs and the free-living, pre-infective larvae are sensitive to desiccation, but the infective third stage larvae (L_3) are resistant and may survive in the environment for an approximately 1 year (Rose and Small, 1980b). The ingested larvae migrate to the large intestine where they encyst in the mucosa and submucosa to form small nodules (Soulsby, 1982). The fourth stage larvae emerge from the nodules to occupy the lumen where they mature.

Unthriftness, weakness, constipation and mucoid bloody scouring are the notable clinical signs. Apart from loss of condition, these may lead to secondary infections which may end up in death, hence considerable economic loss

(Corwin *et al.*, 1986). In severe infections, pseudomembranes may be shed and passed out in feces carrying with them large numbers of worms (Soulsby, 1982).

Diagnosis is done during post-mortem meat inspection by observing the nodules with worms in the walls of the large intestine. Hypertrophy of the regional lymph nodes, thickening of the intestinal wall and diphtheritic membrane, as well as oedema of the mesocolon may also occur. Diagnosis may also be done by identification of the cultured larvae (Honer, 1967).

Treatment has been done using phenothiazine at 50mg/kg body weight, piperazine at 110mg/kg body weight, Dichlorvos at 15.1mg/kg body weight. Thiabendazole, hygromycin, levamisole and pyrantel tartate have also been used. The efficacy of ivermectin against *Oesophagostomum* and other gastrointestinal nematodes of the pig has been well documented (Baarth *et al.*, 1980; Stewart *et al.*, 1981a; Brokken *et al.*, 1984; Marchiondo *et al.*, 1987; Benz *et al.*, 1996).

2.1.1.3. *Trichuris suis* (Whipworm of the swine)

These are small nematodes with anterior aspects thinner than the posterior parts. The male has a single spicule and the testis is convoluted throughout its length. The female has a single ovary, uterus and the vulva opens at the junction of the anterior and posterior parts. The life cycle is direct with a 6-8 days pre-patent period. The larvae develop within the resistant eggs in which they may remain infective for years (Hill, 1957). The infection induces a strong immunity (Powers *et al.*, 1959).

The eggs of *T. suis* are passed in the feces. They are barrel-shaped, thick-shelled and dark-brown in colour and have a clear, transparent plug at either pole. Under favourable conditions, the eggs become embryonated or develop into the infective larval stage. The pigs become infected through ingestion of the embryonated eggs in feed, water, or when rooting in contaminated soil. The larvae grow to maturity in the caecum and the large intestine. Heavy infections may lead to inflammation of the bowel wall leading to unthriftness, weakness and emaciation.

At post-mortem, very little damage is usually observed. However, in heavy infections necrosis, oedema and haemorrhage of the mucosa may be seen. Ulcer-like lesions may be present in the caecum and colon. There may be nodule formation which are granuloma-like and contain the anterior portion of the worms, eggs and phagocytes. There may be an inflammatory reaction with lymphocytic infiltration and excessive mucus production. The mucosa of the large intestine may be replaced by a necrotic diphtheritic membrane (Beer and Lean, 1973).

Diagnosis is usually done by detecting the characteristic eggs in faeces and also the post-mortem lesions.

Control is normally done by practising standard swine sanitation system. *Trichuris suis* eggs are more resistant to environmental factors than free_living infective larvae and will be more difficult to control in outdoor systems (Roepstorff and Murrel; 1997).

Treatment is rarely done. But if carried out ivermectin, fenbedazole,

pyrantel, levamisole, thiabendazole and hygromycin B have been used (Radostists *et al.*, 1994).

2.1.1.4. *Metastrongylus* (Lungworms of the swine)

The important species are *Metastrongylus pudendotectus, M. apri and M. salmi*. Though the primary host is the pig, Chandler (1955) reported *Metastrongylus* in man while Swhartz and Alicata (1935) reported infections in guinea pigs and dogs. *M. apri* is the vector of swine influenza. Earthworm acts as the intermediate host.

The parasites are fairly long and slender white worms.The adults live in the bronchi and bronchioles of the lungs (Dunn *et al.*, 1955). The adult worms apparently orient themselves with their heads down or facing the terminal branches of the trachea where, according to Soliman (1951), they ingest inflammatory exudate as it is coughed up.

The thick-shelled embryonated eggs are either coughed, ciliated up or are swallowed, and finally passed in the faeces. They are then ingested by earthworms, the intermediate hosts, where the infective L_3 larvae develop. Eggs in the soil and the infective larvae within the earthworms may remain viable for several years (Spindler, 1938). The pigs become infected by ingesting earthworms containing the infective larvae. The larvae are then released and penetrate the intestinal wall. They then enter the blood stream via the lymphatic system and finally into the lungs where they enter into the air passages.

Clinically, metastrongylosis is characterised by coughing, difficult

breathing, loss of appetite and retarded growth. Heavy infections cause bronchitis and pneumonia (Stockdale, 1976).

At post-mortem inspection there may be wedge-shaped areas of vesicular emphysema accompanied by small irregular pale red areas of consolidation along the ventral border of the diaphragmatic lobe of the lung. In the lungs observed changes are inflammation, haemorrhages, hyperplasia, hardening of the lungs and loss of respiratory function (Nguyen, 1996). The bronchi are thickened and dilated. Dunn (1956) indicated occasional "milk spots" on the liver due to accidental migration of the larvae through the liver. In the young pigs, parasitic pneumonia may be present as well as giant cell formations associated with the presence of large numbers of lung worm eggs in the alveoli (Stockdale, 1976).

Diagnosis can be made during post-mortem inspection where the presence of the lung worms have been demonstrated by chipping and firmly pressing the diaphragmatic edge of the lung lobe (Corwin *et al.*, 1986). In addition, fecal examination of embryonated eggs is a good aid to diagnosis.

Treatments of lung worms have been done by use of cyanacethydrazide at 15mg/kg b.wt. and diethylcarbamazine citrate at 50mg/kg b.wt. Thiabendazole and levamisole have also been used.

2.1.1.5. *Hyostrongylus rubidus* (The red stomach worm of the pig)

These are small slender red worms which occurs worldwide (Radostists, *et al.*, 1994). They have some common epidemiological characteristics with *Oesophagostomum* spp. Pigs are infected by ingesting the free-living third stage

larvae (L_3). *Oesophagostomum* and *Hyostrongylus* spp. have short prepatent periods and comparable bionomics (Rose and Small, 1982).

Hyostrongylus rubidus lay typical strongyle eggs which are passed out in feces. The larvae become infective in pasture and soil in about 7 days. Pigs become infected by swallowing infective larvae in feed or water. Further development takes place in the stomach mucosa, where worms reach maturity (Masaba and Herbert, 1978).

Heavy infections cause inflammation and thickening of the stomach wall leading to gastric upsets. Pigs become unthrifty and young ones may die. Diagnosis is confirmed at post-mortem by observing the hyperaemic and ulcerative lesions on the stomach wall. Diagnosis can also be done by culturing the faecal eggs and examining the resultant larvae.

Treatment using dichlorvos, levamisole , cambendazole and thiabendazole have been used and found to be effective against *Hyostrongylus rubidus*.

2.1.1.6. *Ascarops* (Thick stomach worms)

The main thick stomach worms belong to the species *Ascarops strongylina*. These are small red colored spirurod nematodes with narrow cuticular wings on the left side (Soulsby, 1982). They live in the stomachs of a wide range of mammals and are of worldwide distribution (Dunn, 1978). They have an indirect life cycle with coprophagous beetles as intermediate hosts (Soulsby, 1982). Eggs are oval with thick shells and embryos are well developed

before oviposition. The males have unequal spicules. In the females, the vulva is anterior to the middle of the body (Soulsby, 1982).

Eggs are passed out in feces where they are consumed by various species of coprophagus beetles. The larvae hatch and develop within a cyst to the third infective larval stage in the body cavity of the beetles (Soulsby, 1982). Pigs become infected by eating the beetles and larvae develop to maturity in the stomach.

Infected pigs do not exhibit any noticeable symptoms. However, at necropsy pseudomembrane may be formed at the pyloric end of the stomach where these worms are found attached to the wall. Red patches may be found around the pin prick openings made by the worms and there may be gastritis and small ulcerations.

Presence of characteristic eggs in the feces confirm the diagnosis.

Carbon disulphide at a rate of 0.1ml/kg b.wt. has been used in the treatment of this worm.

2.1.1.7. *Physocephalus sexalatus*

These are small red-colored spiruroid nematodes with trilobed lips. The head is marked off from the body by an inflated cuticular ending in a circular demarking margin just anterior to the posterior end of the pharynx (Soulsby, 1982). The entire male tail is twisted about three turns and the vulva in the female is posterior to the middle of the body. The eggs are oval and slightly flattened at the poles. Embryos are well developed in the shell prior to

oviposition. These nematodes have an indirect life cycle where coprophagous beetles act as intermediate hosts (Soulsby, 1982).

These worms causes unthriftiness and loss of condition , hence an economic loss to the farmer. Treatment of this worm is by use of Sodium fluoride.

2.1.1.8. *Gongylonema pulchrum* (Gullet worm of the swine)

These worms have cuticular bosses on the anterior ends and cervical alae which are symmetrical and relatively broad. The worms are found between the mucosa and muscle tissue of the tongue. The egg is elliptical and smooth-shelled and contains an embryo when passed in the feces. A number of dung beetles act as intermediate hosts and the pig becomes infected by ingesting them with infective larvae (Soulsby, 1982).

These worms are of public health concern during meat inspection due to aesthetic reasons. This leads to an economic loss due to consumer rejection. Otherwise they are not pathogenic to human beings. Carbon disulphide is effective against this worm.

2.1.1.9. *Strongyloides ransomi* (The intestinal thread worm of swine)

This worm has a direct life cycle that includes free-living adult males and females and parasitic parthenogenetic females in the small intestines. The parasitic females are tiny and transverse vulva with protruding lips which lies posterior to the middle of the body. The eggs are ellipsoidal, thin-shelled and

contain embryos when laid.

The small embryonated eggs are passed in feces where they hatch and the resultant rhabditiform larvae may develop into filariform or infective larvae. The infective larvae are capable of penetrating the skin. They proceed into the lungs via the bloodstream. From the alveoli of the lungs they enter into the bronchi, oesophagus, stomach and then into the small intestines, where they become adults. Prenatal infection has been reported to occur (Stone, 1964). Young piglets may acquire postnatal infection from the colostrum of the sow (Moncol and Batte, 1966). They found out that the infective larvae were concentrated in the adipose tissue rather than in the mammary glands. *Strongyloides ransomi* is capable of developing a free-living generation of adult males and females.

Infected pigs may be restless and irritable, have anorexia, reduced growth rate, diarrhoea, vomiting and intestinal haemorrhage. In severe cases death occurs.

Percutaneous infection may lead to skin eruptions. During post-mortem infection, petechial haemorrhages may be evident in the lungs, heart and intestinal mucosa.

Post-mortem examination is the main aid to diagnosis. Examination of feces for the presence of characteristic eggs can also confirm the diagnosis.

Treatment has been carried out using thiabendazole at 50mg/kg b.wt. administered with feed and levamizole at 5mg/kg b.wt. subcutaneously for three days or 15mg/kg b.wt. orally.

2.1.1.10. *Stephanurus dentatus* (The kidney worms)

These are thick, large worms which are found in the liver, fat surrounding the kidneys and the ureters. These worms have a direct life cycle with a long prepatent period of 9-16 months (Batte *et al.*,1960). The larvae have been found in the brain, spinal cord, musculature, mesenteries and pancreas of infected animals.

The eggs are quite large and strogyle-like. They are thin-shelled eggs are passed out in urine in the early stages of segmentation. Under favourable conditions the eggs hatch and the larvae develop into the infective stage (L_3). The developing stages are very sensitive to direct sunlight and desiccation (Spindler and Andrews, 1955). Pigs become infected either by oral route or by skin penetration. The larvae move to the stomach, intestinal wall or in the tissues beneath the skin, the peritoneal cavity and mesenteric lymph nodes where they moult to L_4 before entering the liver through portal vessels (Lichtenfels and Tromba, 1972). They wander about and grow to about 3/4 of an inch before the final moult after which they migrate to the kidneys (Batte *et al.*,1960). On reaching the tissues and fat around the kidneys, they form cyst-like structures which open into the pelvis of the kidney or directly into the ureters, through which the eggs are passed to the bladder and then to the exterior (Anderson *et al.*,1973). Within the capsules around the kidneys, the worms reach full maturity and lay eggs. During larval migration from the liver to the kidneys, some go astray and find their way into the spleen, loin muscles, spinal cord, lungs and

other organs where they become encapsulated (Batte *et al.*, 1960).

Heavy infection of the liver by these immature worms causes weakness and emaciation, sometimes a dropsical abdomen and possibly death. Weakness and even paralysis of the hindquarters may occur as a result of damage to the kidneys.

Condemnations of livers, kidneys and trimming of loins and other valuable parts of carcasses result in important economic losses (Stewart *et al.*, 1964; Batte *et al.*, 1975). Condemnation of the whole carcase may occur if large portions are affected. The liver condemnation index (lb. liver condemned / numbers of pigs killed) in U.S.A. gradually increased from the 1970's and was recorded at 0.48 lb in 1980. The latest published figure available is 0.51 lb for 1990 (USDA Food Safety and Inspection Service, 1990). Infection of pigs with kidney worms can also cause reduction in growth rate and efficiency of feed utilization (Hale and Marti, 1983).

At post-mortem, it has been observed that damaged liver cells are replaced by hard connective tissue which appears greyish-white and may be superficial or extend deep into the substance of the liver (Anderson *et al.*, 1973). There may be verminous necrotic abscesses in the liver parenchyma and the surrounding lymph nodes, which in heavy infections may also be seen in the pancreas, spleen and lungs (Batte *et al.*, 1960). Thrombi may be seen in the portal vein or blood vessels deep in the liver. Affliction of the lymph tissue is a prominent feature of the disease. Fibropurulent cysts and tracts around the ureters and kidneys may occur. Abscesses may also be seen in the kidney

parenchyma, accompanied by eosinophilia (Batte *et al.*, 1960). Extensive damage may be caused to the kidneys by the presence of larvae and mature worms in the surrounding fat tissues. Pleuritis and peritonitis may occur.

Post-mortem examination or the presence of characteristic eggs in the urine combined with the finding of adult worms in cysts around kidneys and liver lesions offer confirmatory diagnosis.

Fenbendazole and ivermectin are effective against larval and adult stages of *Stephanurus dentatus* (Stewart *et al.*, 1981a, b). Levamisole is efficacious against adult stages (Stewart *et al.*, 1977).

Prevention and control is done by exposing the susceptible eggs and larvae to sunlight and / or dry weather conditions (Spindler and Andrews, 1955). Watering and feed troughs should be placed on high ground and young pigs should be separated from adult animals because eggs are mainly shed by the older animals (Batte *et al.*, 1960). All these combined with improved methods of hygiene in the piggery can control the spread of the worm.

2.1.1.11. *Trichinella spiralis* (The trichina worm)

This is a zoonotic nematode parasite of serious public health implications (Kagan, 1959; Schantz *et al.*, 1975; Gajadhar *et al.*, 1997). It has a worldwide distribution. Primarily this parasite infects man and pig, but has been reported in ox, sheep, horse, dog, cat, rabbit and rat (Roepstorff and Nansen, 1994). The recent taxonomic revision of this worm included 8 taxa (Pozio *et al.*, 1992). But *Trichinella spiralis* remains the most thoroughly studied species. The adult

worms are found in small intestine, but the larvae affect the skeletal muscles (neck, tongue, diaphragm and jaw), although lesions may be found in the myocardium, lungs, brain and meninges (Kotula *et al.*, 1984; Kapel *et al.*, 1998). Although the distribution is cosmopolitan, the prevalence tends to vary with the types of feeding programms (Zimmerman *et al.*, 1962). Jeffries *et al.*(1966), estimated that the number of infected garbage-fed pigs was much lower than infected grain-fed pigs.

Epidemiology of human trichinellosis has been mostly studied in the United States of America where previously about 200-300 clinically recognised cases were reported to the Public Health Service (Kagan, 1959). But currently, its morbidity is on the decline although it is still recognised as an important health problem (Schantz, 1991). Kagan (1959), estimated that there may be 25-50 million Americans with *Trichinella* larvae in their muscles. The 1993 outbreak of human trichinellosis in Canada showed that the disease was linked to the consumption of wild boar meat originating from 2 farms in the province of Ontario (Gajadhar et al., 1997). The same workers reported absence of human cases of *Trichinella* from the consumption of pork from domestic Canadian pigs. Trichinellosis was unknown in Africa south of Sahara until 1961, when exceptionally heavy infections were seen in 11 young men on the lower slopes of Mount Kenya who had eaten the flesh of a bush pig, *Potamochoerus porcus* (Forrester *et al.*, 1961). When high infection rates were found in hyenas and other carnivores in the Rift-Valley area, then it was accepted that the infection was indigenous in Kenya (Nelson *et al.*, 1963).

Infection in animals and man is acquired by eating raw or undercooked flesh (e.g. pork) containing encapsulated larvae (Pozio *et al.*, 1993). Domestically, rats are probably the most highly infected 'natural' hosts and pigs become infected by eating infected pork scraps or occasionally rats which enter their stalls (Kagan, 1959; Gajadhar, 1997). Improperly cooked infected sausages are the major sources of the parasite to humans (Pozio *et al.*, 1993). Upon ingestion of infected materials, larvae are released in the duodenum and may be found there as early as one hour after the initial infection (Wright, 1979; Dunn and Wright, 1985). Deeply embedded in the mucosa, larvae feed, grow and rapidly become sexually mature. After copulation, which takes place in the small intestines, the female burrows into the mucous membranes by way of the glands of Lieberkuhn and makes its way to the lymph. Large numbers of embryos are deposited, which enter the lymphatic system and blood stream and are carried to the skeletal muscles (Wang and Bell, 1986a, b), especially those of the diaphragm, jaws, tongue, larynx and eye (Kapel *et al.*, 1998). On reaching the desirable muscles, the larvae penetrate the sarcolema of the muscle fibres. Here they become enclosed in cysts, usually one to a cyst, although as many as seven have been recorded in a single cyst. Viable cysts may remain intact for at least 30 years (Gullota and Froscher 1983), although calcification occurs gradually to destroy the larvae and capsule.

Pigs are quite tolerant to the parasite and symptoms are seldomly seen in natural infections. Experimentally, there may be loss of appetite, colic pains, paralysis of the hind-quarter, incontinence of urine and feces, diarrhoea,

decreased weight gains stiffness of the muscles and itching (Scholtens *et al.*, 1966). Trichinellosis has serious consequences in man, where a massive infection of muscles by the larvae produces severe and painful symptoms that may end in death (Schantz *et al.*, 1977).

Diagnosis is usually made after slaughter where the larvae are easily demonstrated by trichinoscopy or by digestion of muscular tissue in acidified pepsin solution and examination under microscope. It has been established that direct parasitological methods for the diagnosis of trichinellosis cannot detect low level infections (Van Knapen *et al.*, 1981). Enzyme-linked immunosorbent assay (ELISA) has yielded more promising results because of its high sensitivity and takes shorter time to perform (Ruitenberg *et al.*, 1975, 1976; Van Knapen *et al.*,1981; Haralabidis *et al.*, 1989, 1992, 1993). Boireau *et al.* (1997), has used a modified ELISA.

No satisfactory treatment against trichinellosis has been found. However, thiabenzole has been tried. Thorough cooking of all garbage, including cooked pork and sausage before feeding pigs and preventing rats from entering piggeries may aid in controlling trichinellosis (Pozio *et al.*, 1989). Pork and its products should be properly cooked before consumption by human. Deep freezing is also an effective method of control (Pozio *et al.*, 1989)

2.1.2. Acanthocephalids

2.1.2.1. *Macracanthorhynchus hirudinaceus* (The thorny headed worm)

These are large nematode-like worms superficially resembling ascarids (Soulsby, 1982). Their large sizes have made them a valuable models for working on their nervous system and other fundamental studies (Crompton and Nickol, 1985). They are of worldwide distribution where pigs are raised. They are mainly found in the small intestines of pigs, wild boars and occasionally dogs and monkeys (Soulsby, 1982). They occasionally infect human where they may cause intestinal perforations (Radomyos *et al.*,1989).

The eggs contain larvae when laid and are passed out with feces. They are then eaten by beetle grubs in which infective larval stage develops (Soulsby, 1982). Pigs acquire the infection by ingesting these grubs containing the infective larvae. The latter develop and mature in the small intestines (Soulsby, 1982). The adult worms are normally found attached to the intestinal wall.

The infected animals show no specific symptoms, but in heavy infections there may be unthriftness. At post-mortem, inflammation of the mucosa at the site of attachment and wounds with caseous material may be seen. Perforation of the intestinal wall may occur and peritonitis caused by leakage of intestinal contents may be fatal (Soulsby, 1982).

Examination of the characteristic eggs in the feces and observation of lesions and worms in the small intestine are good aids to diagnosis. Levamisole and ivermectin are used in the treatment of this worm.

2.1.3. Cestodes

2.1.3.1. *Cysticercus cellulosae* (The Pork "measles")

These are the larval stages of the cestode, *Taenia solium* (Flisser, 1988). *Cysticercus cellulosae* are small, lemon-shaped bladders about half an inch long and a quarter inch wide with a protuberance on one side, which is the scolex. These larval stages, are found primarily in the muscles of the pig and wild boar. Occasionally they occur in the muscles, spinal cord and brain of human with serious and often fatal results (Macias and Ordonez, 1970). The adult worm, *T. solium* is exclusively a parasite of human where the strobila develops in the intestine. Thus, human can act both as a definitive and an intermediate host (Flisser, 1988). The muscles commonly involved include the muscles of the heart, tongue, diaphragm, masseter and triceps brachialis.

Pigs become infected by ingesting *T. solium* eggs (Gonzalez *et al.*, 1993). The eggs hatch in the gastrointestinal tract, penetrate the intestinal wall and find their way into circulation where they are spread throughout the body (Panthak *et al.*, 1995). In the skeletal muscles, they develop into cysts.

Pig cysticercosis is an important parasitic disease not only because it leads to a decrease in production, but also it maintains human taeniasis and cysticercosis (Aluja *et al.*, 1987). The resultant cysticercotic or measly pork is of greatly reduced value, thus economic loss due to condemnations at abattoirs (Gonzalez *et al.*, 1993).

Human beings become infected by eating infected pig flesh or contaminated food, drink or by autoinfection where they develop into adult

worms in the intestine.

Taeniasis / Cysticercosis complex is of great public health importance, because of its fatal outcome in human infection as a result of self-infestation and the development of the cystic stage in the brain. In the brain metacestodes remain viable for longer periods than in muscles (Aluja *et al.*, 1996). Human neurocysticercosis is considered to be the commonest parasitic disease of the central nervous system (Del Brutto and Sotelo, 1988; Schantz, 1989).

Taeniasis / Cysticercosis complex is common in Central and South America (Schenone *et al.*, 1982) and other regions where ecological and social conditions permit its dissemination (Mahajan, 1982). In Latin America and Asia neurocysticercosis is endemic and recognised as a major public health problem (Acha and Aguilar, 1964; Coker Van *et al.*, 1981; Botero, 1984; Mignard *et al.*, 1986; Dumar *et al.*, 1989; Tsang and Wilson, 1995). It is the most prevalent disease of the central nervous system in the USA. Most of the patients have been reported to originate from Mexico or other states of Latin America (Earnest *et al.*, 1987). In Mexico, a clinical incidence of swine cysticercosis of 7% has been reported (Flisser, 1988). In Africa, the infection is probably more widespread than is apparent, as it is poorly documented. In West Cameroon, post-mortem examination of pigs showed a prevalence of 24% with 2% of the human population showing *Taenia solium* antibodies (Zoli *et al.*, 1987). In 1974, an outbreak of the disease occured in the Ekari people of West New Guinea where a prevalence of 18% was recorded (Gajdusek, 1978).

The method most commonly employed for the diagnosis of pig cysticercosis is the post-mortem inspection of carcasses. Immunodiagnosis of this cysticercosis represents an important tool in providing quantitative epidemiological data, since it can be easily applied to a large number of living animals (Gonzalez *et al.*,1990).

Improvements in public health and animal husbandry have led to the virtual eradication of the human and porcine cysticercosis in developed countries, but such measures are too expensive for immediate implimentation in less developed countries (Gemmell *et al.*, 1988).

No effective treatment is available in the pig, though flubendazole (Tellez-Giron *et al.*, 1981) has been used. However, in human, praziquantel and metriphonate and are possible alternatives to surgery.

2.1.3.2. *Cysticercus tenuicollis* (The bladder worm)

The adult worm, *Taenia hydatigena* lives in the intestines of dogs and other carnivores. The larval stage is mostly found in sheep and cattle, but sometimes in pigs (Soulsby, 1982). Domestic animals acquire the infection by ingesting droppings of infected dogs. The eggs hatch in the intestines and embryos find their way into the liver via the blood-stream. In the liver, they burrow out and may be found on the surface of the liver or more commonly, attached to the omentum or organs in the peritoneal cavity (Soulsby, 1982). The larvae are usually found in a cyst filled with fluid and may be very large measuring several inches in diameter when mature. Dogs become infected by

ingesting the bladder worm.

Diagnosis is usually confirmed at post-mortem meat inspection. Control measures involve treating dogs for the adult worm (using drugs such as praziquantel, mebendazole, fenbendazole and bunamidine) and preventing the ingestion of the infective larval stage by dogs through proper meat inspection (Soulsby, 1982).

2.1.3.3. Hydatid cysts

Hydatidosis is a zoonosis with worldwide distribution. It is caused by the larval stages of *Echinococcus granulosus* (Matossian *et al.*, 1977; Schwabe, 1986; Rausch, 1995) and represents a problem of medical, veterinary and economic relevance in endemic areas (Perdomo *et al.*, 1988;). Animal hydatidosis is an economically very important disease because of the losses caused by the condemnation of organs from the infected animals slaughtered at the abattoir and also due to indirect losses from their reduced production (Hovorka, 1963; Simko and Filo, 1988). In Kenya, the main parasite is *E. granulosus* which occurs in different subspecies (Nelson and Rausch, 1963). Domestic dogs are the main definitive hosts (Nelson and Rausch, 1963; Eugster, 1978).

E. granulosus is a small taeniid worm which varies in length from 2-7 mm and posses 3-4 proglotids. The penultimate proglotid is mature and the terminal proglotid is gravid and is usually about half the length of the worm. The cestode is hermaphrodite (Dunn, 1978).

The adult worms are found in the intestines of domestic dogs and a wide variety of wild carnivores (Eugster, 1978). The eggs are typical taenid eggs and measure 23-36 by 25-30um. The mature proglotids containing the eggs are excreted in the feces of the infected carnivores. The eggs released from the proglotids are infective.They are ingested by intermediate hosts, which include the herbivores, man and pigs (Eugster, 1978). In the small intestines, the eggs hatch and the liberated oncospheres penetrate the walls of the intestines and make their way to the portal vein from where they get to the liver (Soulsby, 1982). In the liver, most of them become lodged in the sinusoids and commence a slow development of the hydatid cysts. Others escape through the vascular by-pass into systemic circulation where they become trapped in the capillaries of the lungs and develop hydatid cysts while a few may escape and are conveyed via the arteries to other parts of the body such as the bones, brain, kidneys etc (Eugster, 1978; Macpherson, 1985). The life cycle is completed when the definitive host ingests hydatid cysts containing protoscolices which evaginate and penetrate deeply between the villi into the crypts of Lieberkuhn and mature.

Animals with hydatid cysts show no clinical signs, but reports from Russia (Ramazanov, 1978) indicate that the infection has an effect on production of the animal. Several workers have carried out studies in various countries of Africa to determine the prevalence of hydatid disease in domestic animals as shown below:

Table 1: Prevalence of Hydatid disease in various domestic animals in various countries of Africa.

Country	cattle	sheep	goats	camels	pigs	horses	ref.
Kenya	15.8	7.6	3.1	80	0.9	-	(Gathura *et al.*, 1988)
Libya	5.6	8.3	1.6	36.4	-	-	(Gusbi *et al.*, 1990)
Egypt	0	1.3	-	31	4.6	-	(Rahman *et al.*, 1992)
Tunisia	8	2.8	-	17	19	-	(Brahmi, 1973)
Algeria	5.4	14.3	21.2	42.1	0.7	1.8	(Larbaoni *et al.*, 1980)
Mauritania	-	-	-	53.7	-	-	(Pangui and Ould, 1991)
Niger	2.2	2.8	2	-	-	-	(Pangui *et al.*, 1992)
Nigeria	1.5	7.1	18.4	70.9	5	-	(Dada *et al.*, 1979)

Human hydatidosis is a serious and fatal condition (Schantz, 1991). It is one of the most important and widespread helminthic zoonoses (Schantz, 1991). In Kenya, Turkana District has been reported to have the highest prevalence of the disease in the world, with an infection rate of 96 cases per every 100,000 of the population per year (O'Leary, 1976).

Diagnostic aids such as radiography, radioisotope scanning, ultrasonic echotopography and computerised axial topography have been used for diagnosis of hydatidosis. However, they only define space occupying lesions and cannot specifically identify a hydatid cyst. Various serological tests have been applied in the confirmation of presumptive clinical diagnosis of hydatidosis. These include: intradermal test, indirect haemagglutination test, latex agglutination test, immunoelectrophoresis, indirect fluorescent antibody test, enzyme-linked immunosorbent assay, double diffusion test, complement fixation test, counter-immunoelectrophoresis and radioimmunoassay (Schantz and Gottstein, 1986; Di Felice and Siracusano, 1987).

There is no satisfactory chemotherapy for human and animal hydatidosis. Mebendazole has been tried but its results have been unpredictable and adverse reactions such as exfoliative dermatitis and renal damage have been reported (Kern, 1979; African Medical Research Foundation, 1979). Albendazole has been used with promising results (Okello, 1986). Surgical treatment of human hydatidosis remains the only practical treatment although the operations are risky, expensive and often cysts recur (O'Leary, 1976).

In Kenya, many control measures have been put in place including dog

registration, dog treatment, centralisation of slaughter facilities and public education (Macpherson *et al.*, 1984). But, due to cultural habits and the nomadic life of the communities mainly affected, control of the disease is still a hard task to fulfil.

2.1.4. Trematodes of the pig

2.1.4.1. *Paragonimus kellicotti*

This is a fleshy, spinous worm of fairly large size living as an adult in the lungs of many different hosts. Pigs are probably accidental hosts. Pigs acquire infection by ingesting the metacercarial stages found in the intermediate hosts which include crayfish and snail (*Pomatiopsis lapidaria*) (Soulsby, 1982). The ingested metacercariae penetrate the intestinal wall and wander to the pleural cavity, enter the lungs and encyst. Large brown operculate eggs are coughed up and may be demonstrated in the sputum or feces of definitive host (Soulsby, 1982).

At post-mortem, there are adhesions, local inflammatory reaction, and even fibrosis in the lungs. There is no known available effective treatment against this worm.

2.1.4.2. *Paragonimus westermani* (The lung fluke)

This is a fluke having a fleshy body, concave ventrally and convex dorsally with a spiny cuticle. The parasites are found in the lungs, and sometimes in the brain, spinal cord and other organs of the pig, other animals

and man in America, China, Japan, Malaysia and Africa (Soulsby, 1982). There is no effective treatment against this worm.

2.1.4.3. *Fasciola hepatica* (The liver fluke)

This is a flat, leaf-like worm which infects the bile ducts and, in its immature form, the liver parenchyma of sheep and cattle. It is also found in pigs (Dalchow *et al.*, 1971). The latter are infected if they are grazed in on low swampy country. The intermediate hosts are the snails where the metacercariae leave and encyst on grass or other plants and pigs pick up the infection when they swallow these metacercariae encysted on grass (Nansen *et al.*, 1972). This disease can be controlled by keeping pigs off swampy land. Treatment can be effected by subcutaneous injection of carbon tetrachloride.

2.1.4.4. *Fasciolopsis buski*

These are trematodes which live in the small intestines of human and pigs and are significant not only in terms of their economic importance, but also as a public health problem owing to their potential public health risks (Chandra, 1984; Sharma and Gogoi, 1986). Measuring up to 80 mm in length, they are some of the largest trematodes found in humans (Chandra, 1984). These parasites are found in many countries in the Orient. Pigs serve as reservoir hosts.

The life cycle of this parasite is similar to that of *Fasciola hepatica.* The worms produce eggs (up to 25,000 eggs per worm per day) that are passed in the host's feces (Sharma and Gogoi, 1986). The intermediate host is a snail, and the

cercariae that emerge from the snail encyst on vegetation. Humans are infected when they eat vegetables contaminated with metacercariae.

Chronic infections with this parasite lead to inflammation, ulceration, haemorrhage and abscesses of the small intestine (Soulsby, 1982). These can ultimately lead to death of the host. Diagnosis of the disease is based on observing eggs in the feces.

2.1.4.5. *Dricocoelium dentriticum*

This is a small fluke with a rather elongated body, being narrow anteriorly and broader posteriorly. It is found in Europe and various other parts of the world (Soulsby, 1982). Snails act as intermediate hosts. Diagnosis is based on fecal examination for eggs and necropsy findings. Treatment is done using benzimidazole, albendazole and praziquantel.

2.1.4.6. *Schistosoma japonicum*

This is an elongated trematode. The female is usually carried by the male in the ventral groove. It is found in the portal and mesenteric blood vessels of the pig, other animals and man in Africa and the Far East (Soulsby, 1982).

The females lay eggs in the capillaries of the mucosa or submucosa of the intestine. The eggs are then passed out in the feces where they are taken up by snails of the *Oncomelania* genus, which are the intermediate hosts. The pigs are infected by coming in contact with water containing the cercariae which penetrate the skin and gain access to the blood circulation.

Praziquantel and oxamniquine have been used for the treatment of infections with this parasite (Ghandour *et al.*, 1989).

CHAPTER THREE

3.0. Materials and Methods

3.1. Records of post-mortem meat inspection of pigs

Records covering the period 1988-1997 were obtained from the registry of the Ministry of Agriculture, Livestock Development and Marketing. The monthly and annual records from the slaughterhouses in the various districts indicating the number of carcasses and organ condemnations during the routine post-mortem meat inspection as a result of presence of parasites were obtained. The data were tabulated by month, year and District.

3.2. Post-mortem examination of pig carcasses in local slaughter houses (Farmers' Choice and Ndumboini)

The pigs were tagged as P1-P140 on the ears and ante-mortem inspection perfomed before slaughter. The plucks, stomachs and intestines for every pig were likewise tagged immediately after evisceration. Post-mortem examinations were then carried out on every carcass and its organs (lungs, heart, liver, stomachs and intestines) putting emphasis on helminthic parasitic conditions.

3.3. Faecal sampling

Faecal sampling on pigs slaughtered at Ndumboini (in Kiambu) and Farmers Choice (in Nairobi) pig slaughter houses was carried out randomly.

The faecal samples were collected from the rectums of the freshly slaughtered pigs immediately after evisceration. A total of 112 faecal samples

were collected. All samples were labelled and taken to the laboratory the same day where they were kept in refrigerater at 4^0C for a maximum of two days before processing. They were quantitatively analyzed using modified McMaster egg-counting technique (Whitlock, 1948; Roepstorff and Nansen, 1998) for purposes of determining the common helminths.

Briefly, this was done by dissolving 2g of a faecal sample in 28ml of concentrated salt solution (920g of Magnesium sulphate boiled in 1,000ml of water and specific gravity adjusted to 1.28) with thorough stirring. The mixture was then filtered using a strainer and solids discarded (Henriksen and Aagaard, 1976). Using a teat pipette, the McMaster chamber was filled with the solution and eggs examined and counted (in cm^2 of the slide) under low power(100X) of the microscope. The type of eggs were determined by their characteristic appearance, size and shape. The count obtained was multiplied by 100 to get the number of eggs per gram of faeces (e.p.g.). An animal was considered infected with a parasite if at least one egg was detected in the McMaster chamber. The type of helminth eggs and the number of eggs per gramme (e.p.g.) were recorded.

3.4. Muscle sampling

The Muscle samples were obtained from the thick part of the diaphragm for purposes of determining the presence of *Trichinella spiralis* using trichinoscopy. A total of 132 muscle samples were randomly collected and examined for *Trichinella spiralis* larvae. Most of the samples were examined on

the same day of collection while those not examined were kept at 4^0C for a maximum of 96 hours before examination. The examination for the larvae of *Trichinella spiralis* was done by longitudinally cutting each muscle sample into 12 small pieces along the muscle fibres as near as possible to the tendons using scissors. These were then placed in a compressory and examined for the *Trichenella spiralis* larvae using a trichinoscope.

3.5. Data management and analyses

The annual prevalence was defined as the percentage of the number of animals infected to the total number of animals slaughtered over that year. The overall prevalence was calculated as a percentage of the total number of animals infected by all the parasites of concern to the total number of animals slaughtered over the period of study. The data were also analyzed statistically using repeated measures of Analysis Of Variance (ANOVA) (Huitema, 1980) to test for significant differences in the annual prevalences of the various pig parasitic helminths of public health and economic importance over the ten year period, 1988-1997.

CHAPTER FOUR

4.0. Results

4.1. Prevalences of pig helminths for the 1988-1997 period based on national meat inspection records

A total of 642,295 pigs were slaughtered over the 10-year period. Among these, 36,539 pigs had parasitic helminths, giving an overall mean prevalence of 5.7%. Specifically, 31,365 (4.9%) had milk spots in livers caused by *Ascaris suum*, 4,733 (0.7%) had hydatid cysts in the lungs, livers and spleens. Lung worms were found in 215 (0.03%) pigs and 165 (0.03%) had liver flukes. Thirty eight (0.01%) had nodular worms due to *Oesophagostomum dentatum* in the large intestines. Twenty three (0.004%) had *Cysticercus cellulosae* in the heart. Table 1 shows the mean annual prevalences of the important pig parasitic helminths of public health and economicimportance recorded in that period.

Results showing the annual trends of the abattoir pig helminth infections for the period 1988 to 1997 as compared to total number of pigs slaughtered are depicted in Figure 1. There was a slow increase in the number of pigs slaughtered over the years as compared to the increase in helminth infected pigs which though they maintained a higher level of infection, fluctuated up and down throughout the 10-year period. 1996 had the highest number of pigs slaughtered while 1989 had the lowest number of pigs slaughtered. 1989 had the lowest number of pigs infected while 1993 showed the highest number of pigs infected. However, the overall mean annual prevalence was highest in 1988 and lowest in 1989.

TABLE 2: THE ANNUAL ABATTOIR PREVALENCE OF PIG PARASITIC HELMINTHS IN KENYA FOR THE 1988-1997 PERIOD

YEAR	A.S. (%)	H.C. (%)	L.W (%)	L.F. (%)	N.W. (%)	C.C. (%)	O.P. (%)
1988	10.12	0.59	0.13	0.00	0.00	0.009	10.84
1989	0.51	0.05	0.05	0.00	0.00	0.00	0.61
1990	0.55	2.37	0.06	0.00	0.01	0.00	3.00
1991	8.58	1.17	0.00	0.21	0.00	0.00	9.96
1992	5.60	0.70	0.003	0.003	0.00	0.01	6.36
1993	7.99	0.84	0.06	0.02	0.00	0.003	8.91
1994	5.56	0.67	0.02	0.00	0.008	0.01	6.27
1995	2.92	0.25	0.003	0.03	0.006	0.00	3.22
1996	3.56	0.78	0.02	0.003	0.01	0.00	4.37
1997	4.75	0.18	0.007	0.00	0.01	0.003	4.95
M.P	4.88	0.74	0.03	. 0.03	0.006	0.004	5.70

KEY:

A.S.= *ASCARIS SUUM* H.C.= HYDATID CYSTS

L.F.= LIVER FLUKES N.W.= NODULAR WORMS

L.W.= LUNG WORMS C.C.= *CYSTICERCUS CELLULOSAE*

O.P.= OVERALL PREVALENCE M.P.= MEAN PREVALENCE

In Figure 1, the number of pigs slaughtered showed a general increasing trend over the years while the number of infected animals showed slight fluctuations. However, the annual infection rates fluctuated throughout with increases and decreases as shown in Figure 2. Increases were recorded in 1990, 1993, 1994, 1995 and 1996 while decreases were noted in 1989, 1991, 1992 and 1997. However, the number of pigs infected with helminths increased steadily

from 1992 to 1996. The overall annual prevalences decreased between 1993 and 1995, then increased between 1995 and 1998. Statistically, *Ascaris suum* showed a significant difference (p= 0.0001) in the mean annual prevalences to all the other helminth parasites. The same was noted for hydatid cysts. However there was no significant difference among lungworms, liver flukes, nodular worms and *cysticercus cellulosae.*

The abattoir trends in prevalences of the specific pig helminthosis are summarized in Figures 3 to 8. The occurence of *Ascris suum* was recorded throughout the period of study. Comparatively, Figure 3 shows that *Ascaris suum* maintatained a significantly high prevalence over the years as compared to the other parasites. In 1988 the infection rate of *A. suum* was highest (prevalence of 10.12%), but declined to the lowest level between 1989 and 1990 (prevalence of 0.51% and 0.55% respectively). Figure 3 shows fluctuations in the infection rates over the 10-year period. Increases were reported in 1991, 1993, 1996 and 1997 while decreases were recorded in 1989, 1992, 1994 and 1995.

Hydatid cysts were recorded throughout the 10-year period as shown in Figure 4. The annual mean prevalences of hydatid cysts decreased over the years though it peaked (2.37%) in 1990. Figure 4 shows that hydatid cysts annual mean prevalences were significantly much lower than that of *Ascaris suum.*

Prevalences of lung worms fluctuated over the years (Figure 5), with a marked decline between 1988 and 1991 followed by a steady increase between 1992 and 1993. No lung worms were recorded in 1991. The occurence of Liver

from 1992 to 1996. The overall annual prevalences decreased between 1993 and 1995, then increased between 1995 and 1998. Statistically, *Ascaris suum* showed a significant difference (p= 0.0001) in the mean annual prevalences to all the other helminth parasites. The same was noted for hydatid cysts. However there was no significant difference among lungworms, liver flukes, nodular worms and *cysticercus cellulosae.*

The abattoir trends in prevalences of the specific pig helminthosis are summarized in Figures 3 to 8. The occurence of *Ascris suum* was recorded throughout the period of study. Comparatively, Figure 3 shows that *Ascaris suum* maintatained a significantly high prevalence over the years as compared to the other parasites. In 1988 the infection rate of *A. suum* was highest (prevalence of 10.12%), but declined to the lowest level between 1989 and 1990 (prevalence of 0.51% and 0.55% respectively). Figure 3 shows fluctuations in the infection rates over the 10-year period. Increases were reported in 1991, 1993, 1996 and 1997 while decreases were recorded in 1989, 1992, 1994 and 1995.

Hydatid cysts were recorded throughout the 10-year period as shown in Figure 4. The annual mean prevalences of hydatid cysts decreased over the years though it peaked (2.37%) in 1990. Figure 4 shows that hydatid cysts annual mean prevalences were significantly much lower than that of *Ascaris suum.*

Prevalences of lung worms fluctuated over the years (Figure 5), with a marked decline between 1988 and 1991 followed by a steady increase between 1992 and 1993. No lung worms were recorded in 1991. The occurence of Liver

flukes was not reported between 1988 and 1990, in 1994 and in 1997 as shown in Figure 6. The prevalences of liver flukes fluctuated downwards over the years though there was an increase in 1991. The prevalences of nodular worms remained significantly low throughout the study period (Figure 7).

Carcasses with *Cysticercus cellulosae* were reported in Nairobi, 6 in 1992, 8 in 1994 and 2 in 1997; Kiambu, 4 in 1988 and 1 in 1997 and in Nakuru, 2 in 1993. Although the prevalences of *Cysticercus cellulosae* remained the lowest throughout the study period (Figure 8), they were relatively high between 1988 and 1995. Records were available from 27 districts which were studied (Appendix 8). There were no records from other districts either because the Veterinary Department had not taken over the meat inspectorate services from the Ministry of Health or due to religious reasons especially in the Coast and North-eastern Provinces where consumption of pork is significantly low. The Farmers Choice slaughter house in Nairobi led in the pig slaughtering activity with a total of 536,954 pigs slaughtered between 1988-1997, followed by Kirinyaga (24,891), Kiambu (18,037), Kakamega (13,732), Busia (9,610), Murang'a (9,144), Nakuru (8,905), Embu (5,845), Nyandarua (2,351), Nyeri (1,943), Kilifi (1,937), Vihiga (1,759), Bungoma (1,537), Kwale (1,190), Kisumu (1,053), Mombasa (893), Taita Taveta (859), Teso (456), Kitale (435), Thika (274), Uasin Gishu (106), Kericho (105), Laikipia (84), Machakos (64), Lugari (62), Kisii (41) and Kajiado (28).

The annual occurences of the various pig helminth infections over the 10-year period (1988-1997) are as shown in appendices 1 to 8.

Appendix 8 shows the occurences and distribution of the various pig helminths in the studied districts over the 10-year period. Appendices 9 to 14 shows the national overall monthly and annual occurences of the pig helminths over the same period. Appendix 15 shows monthly occurences and distributions of the various pig helminths reported in abattoirs for each year of the 10-year period. Appendix 16 shows the annual mean prevalences of the various parasites over the 10-year period. Appendix 17 shows the type and the ranges of eggs per gram of faecal samples collected and analysed in the laboratory.

4.2. Prevalences of pig helminths based on post-mortem meat inspection between May and July, 1998 at Farmer's Choice (Nairobi) and Ndumboini (Kiambu) abattoirs

The results of the post-mortem meat inspection performed between May and July in 1998 are sown in Table 3. *Ascaris suum* (milk spots in the livers and adult worms in the small intestines), hydatid cysts (in the livers, lungs and spleen), and *Oesophagostomum* species nodules in the mucosa of the large intestines) were observed. Of the 140 carcarsses inspected, 38 (27.1%) had *Ascaris suum* with 22 (15.7%) having milk spots in the livers caused by the larvae, while 16 (11.4%) carcasses had large round worms, *Ascaris suum* in the small intestines. Five (3.6%) carcasses and 3 (2.1%) carcasses had hydatid cysts in the livers and lungs respectively with an overall prevalence of 5.1%. 8 (5.7%) of the carcasses had nodules in the large intestines due to *Oesophagostomum dentatum*.

4.3. Prevalences of pig helminths based on laboratory analysis of the muscle and faecal samples obtained from Farmer's Choice and Ndumboini abattoirs

Out of 132 diaphragm muscle samples collected, none was positive for *Trichinella spiralis* on trichinoscopy.

There were 112 fecal samples collected. The results are shown in Table 4. Of these, 12 (10.7%) were positive for ascarid eggs with a range of 100-5,000 e.p.g., 57 (50.9%) were positive for strongyle eggs with a range of 100-4,600 e.p.g., 4 (3.6%) were positive for trichuris eggs with a range of 100-4,400 e.p.g. and 17 (15.2%) were positive for coccidial oocysts.

TABLE 3: The mean monthly pig abattoir prevalences due to helminth infections, May to July 1998 at Ndumboini and Farmers' Choice abattoirs

Type of Parasitic condition	Organ affected	Prevalence(%)
Milk spots	Liver	15.7
Ascariasis	Small intestines	11.4
Hydatid cysts	Liver	3.6
	Lungs	2.1
Pimply guts	Large intestines	5.7

TABLE 4: The prevalence of pig helminths from the results of fecal egg counts

Type of Helminth eggs	Prevalence (%)
Ascarid eggs	12.7
Trichurid eggs	4.3
Strongyle eggs	50.9

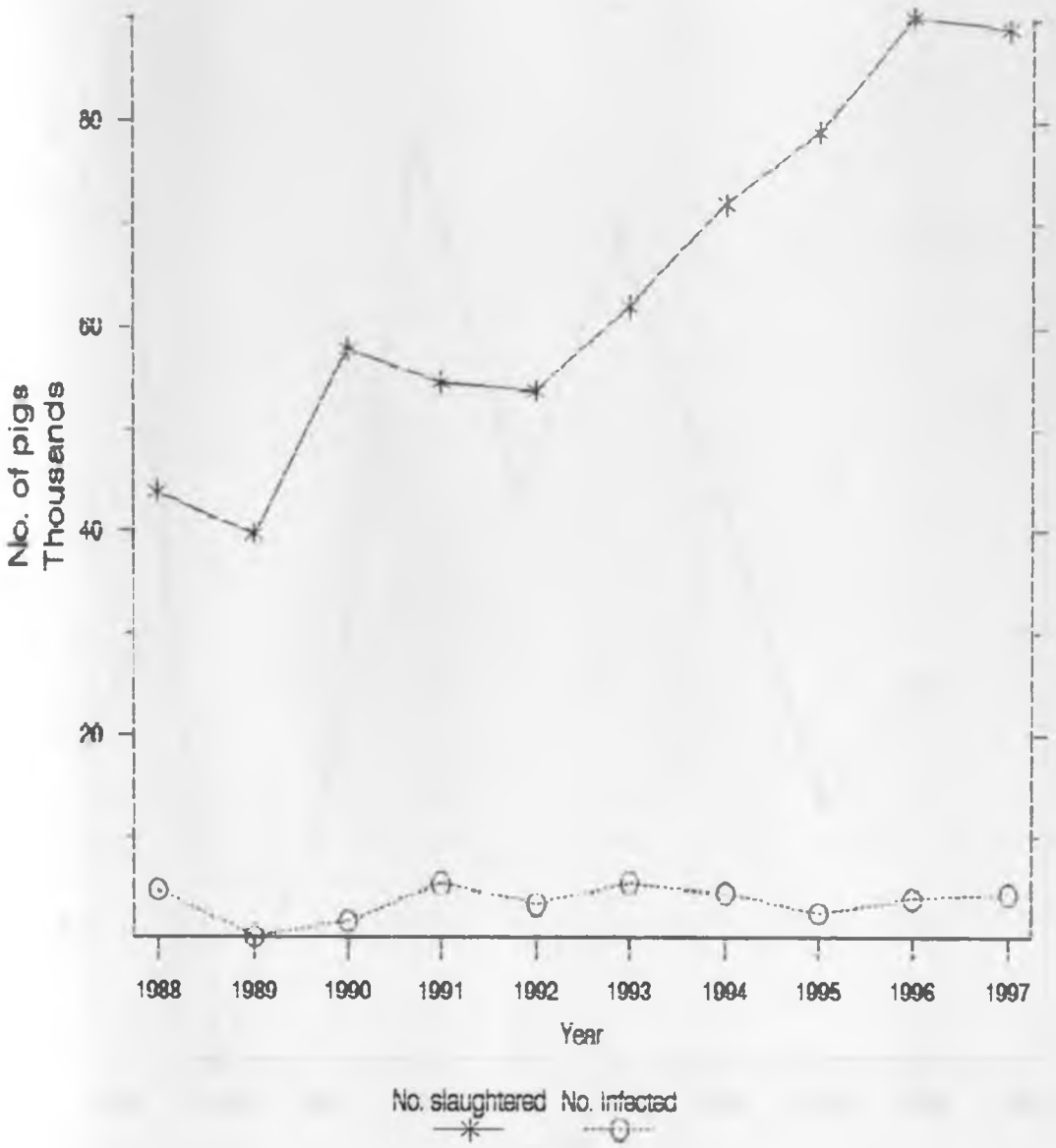

Fig. 1. Trends of the overall helminth infestation of pigs in Kenya for the 1988-1997 period

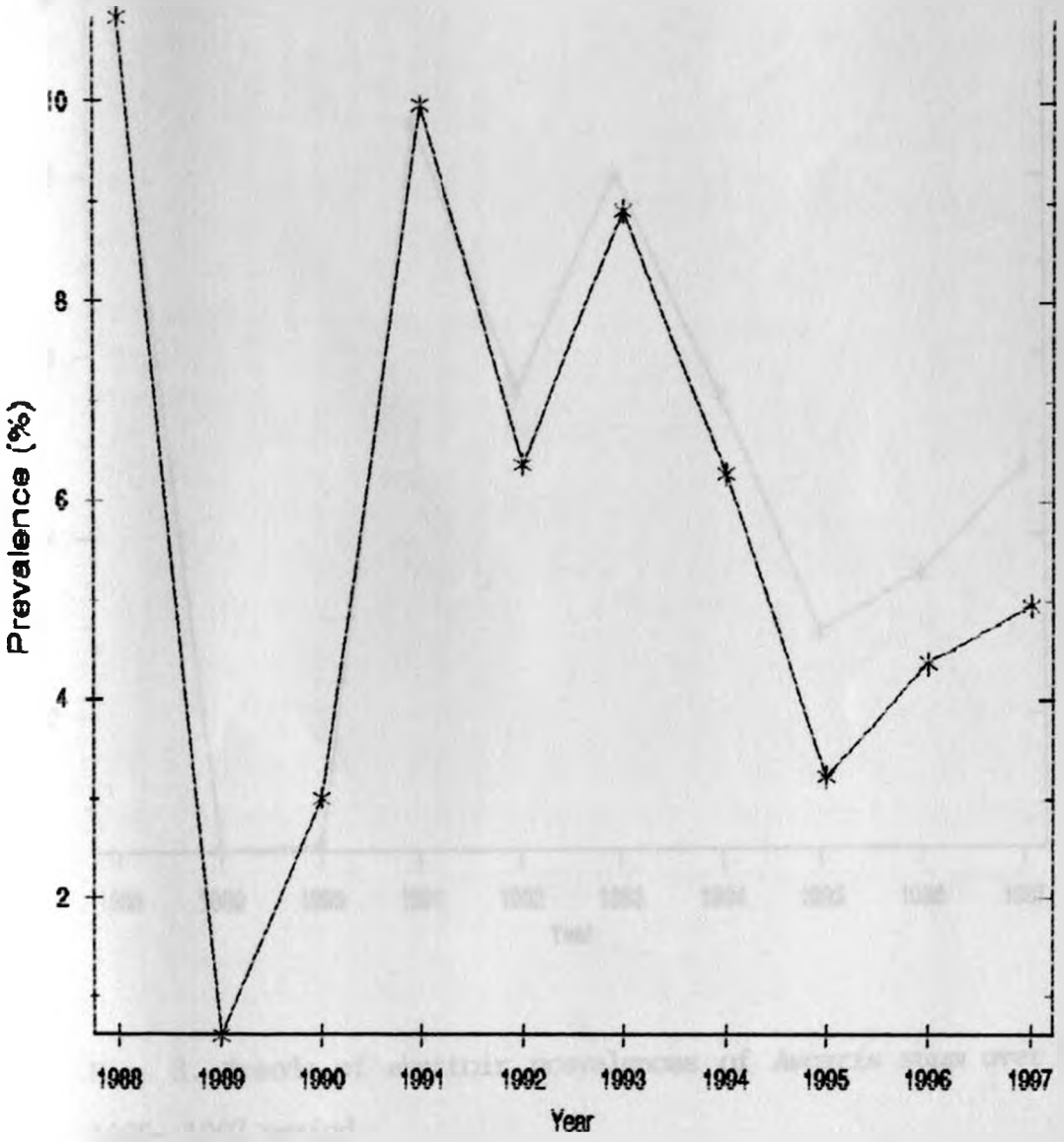

Fig. 2. Trends of the overall abattoir prevalences of helminth infestation of pigs in Kenya for the 1988-1997 period

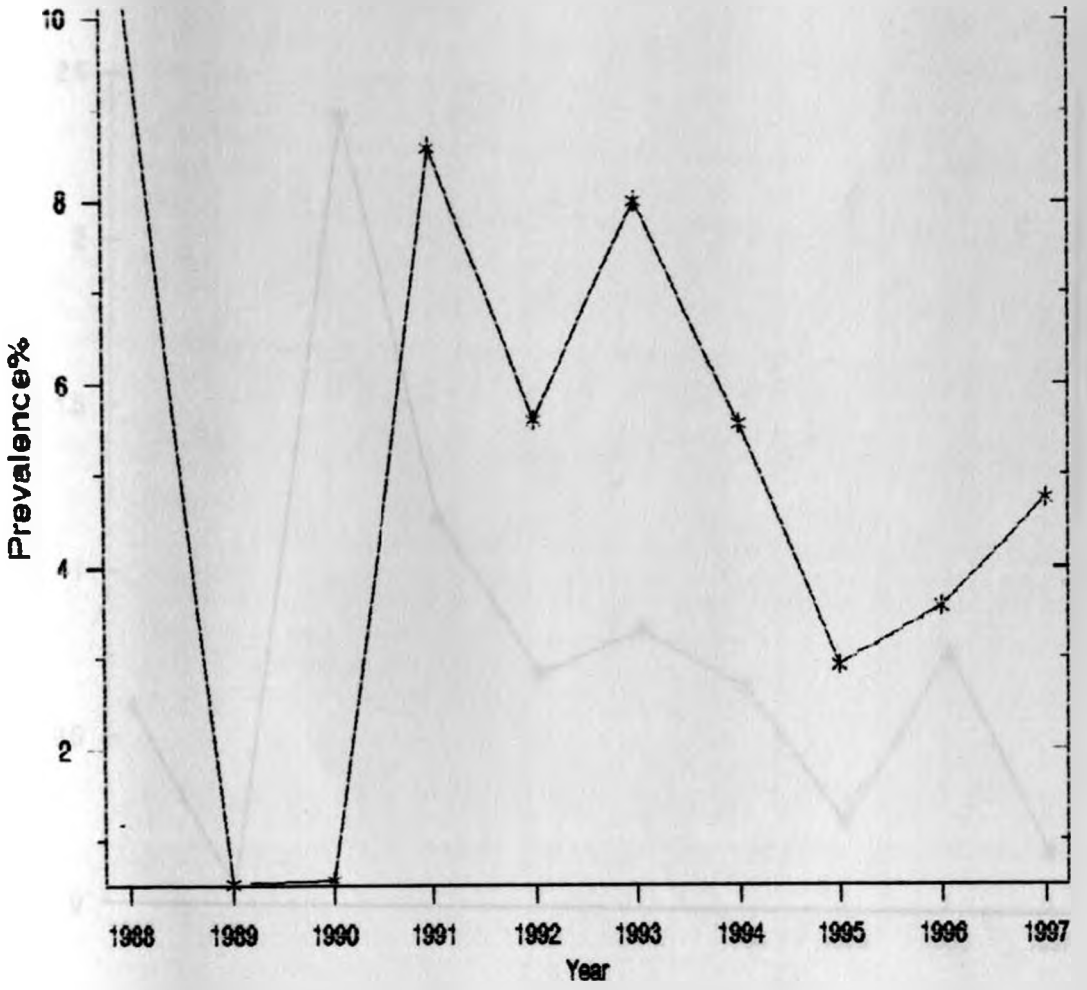

Fig. 3. Trends of abattoir prevalences of *Ascaris suum* over the 1988– 1997 period

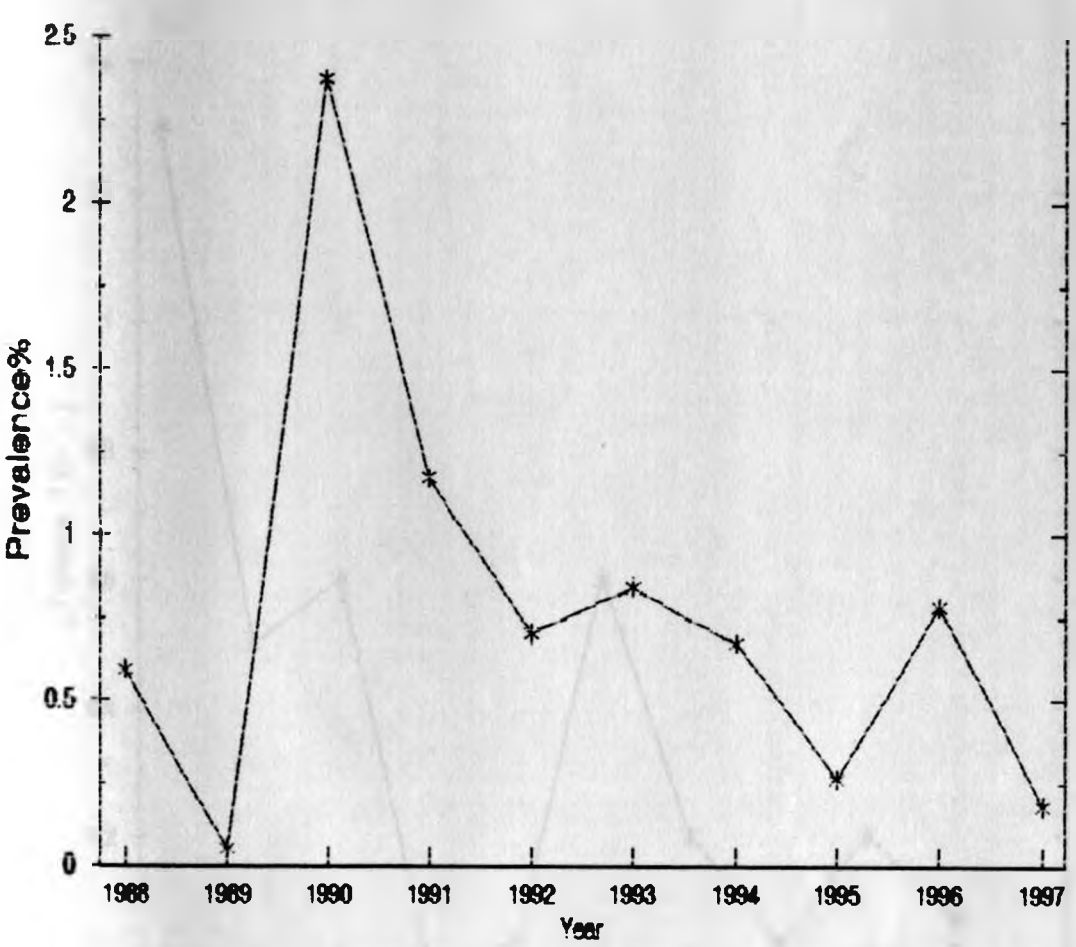

Fig. 4. Trends of abattoir prevalences of pig hydatid disease over the 1988–1997 period

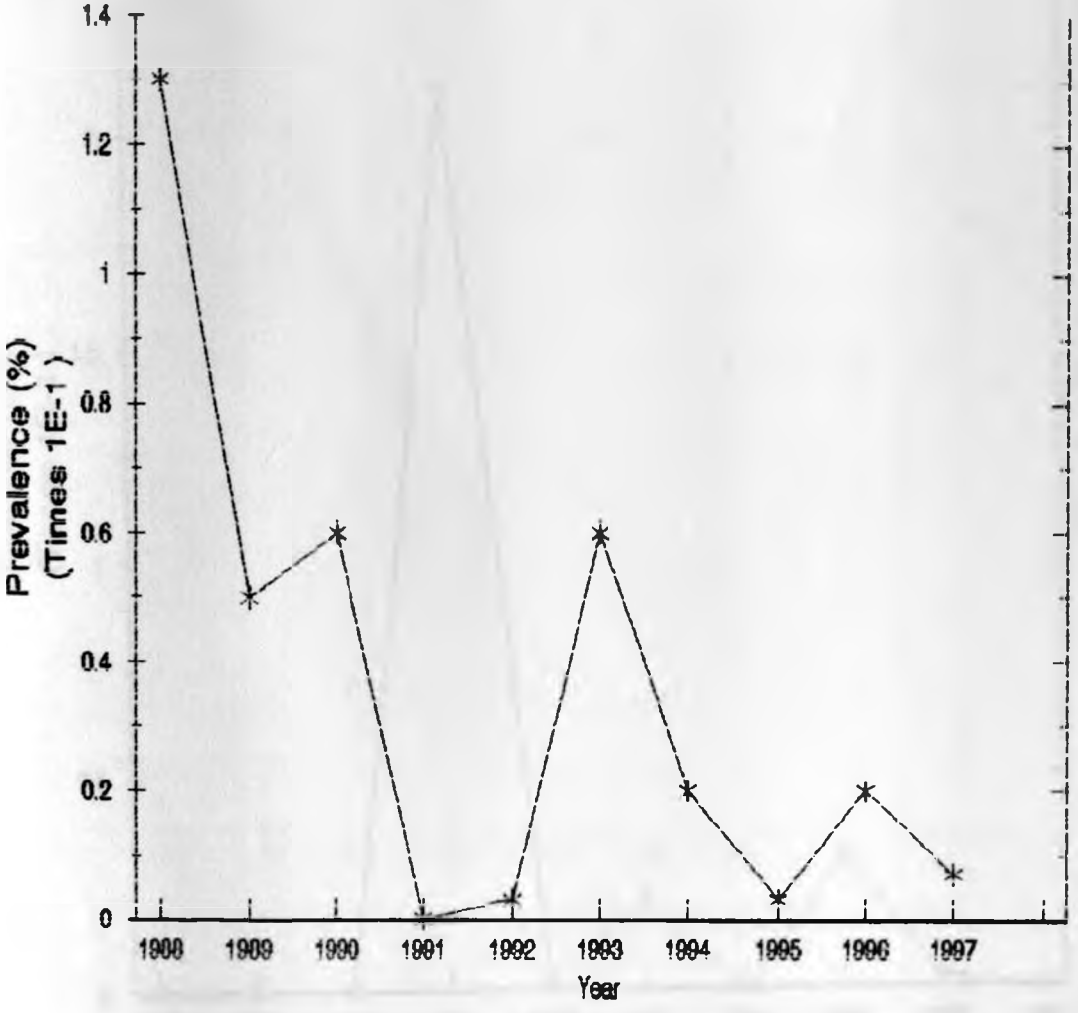

Fig. 5. Trends of abattoir prevalences of pig lung worms over the 1988-1997 period

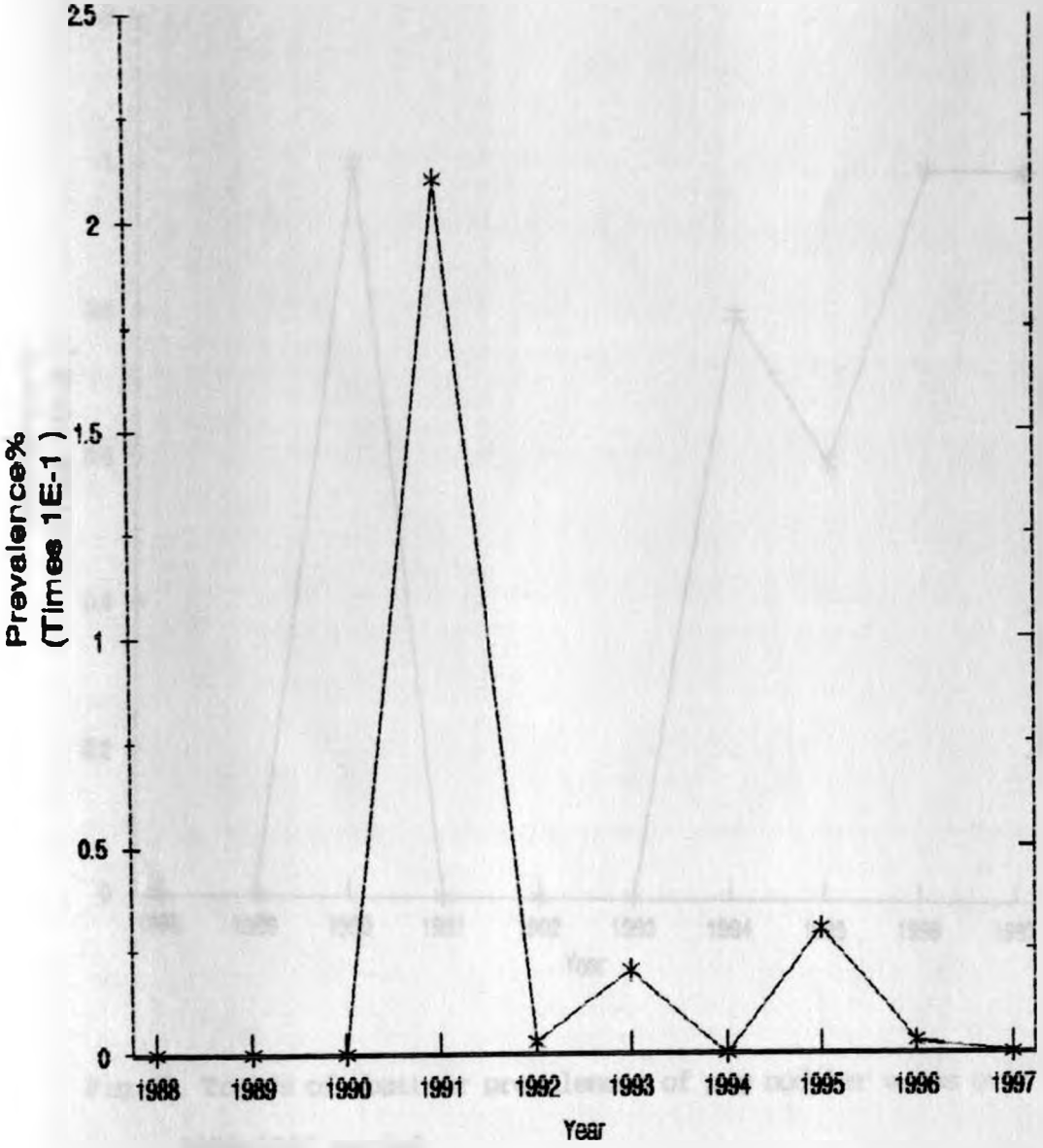

Fig. 6. Trends of abattoir prevalences of pig liver flukes over the 1988–1997 period

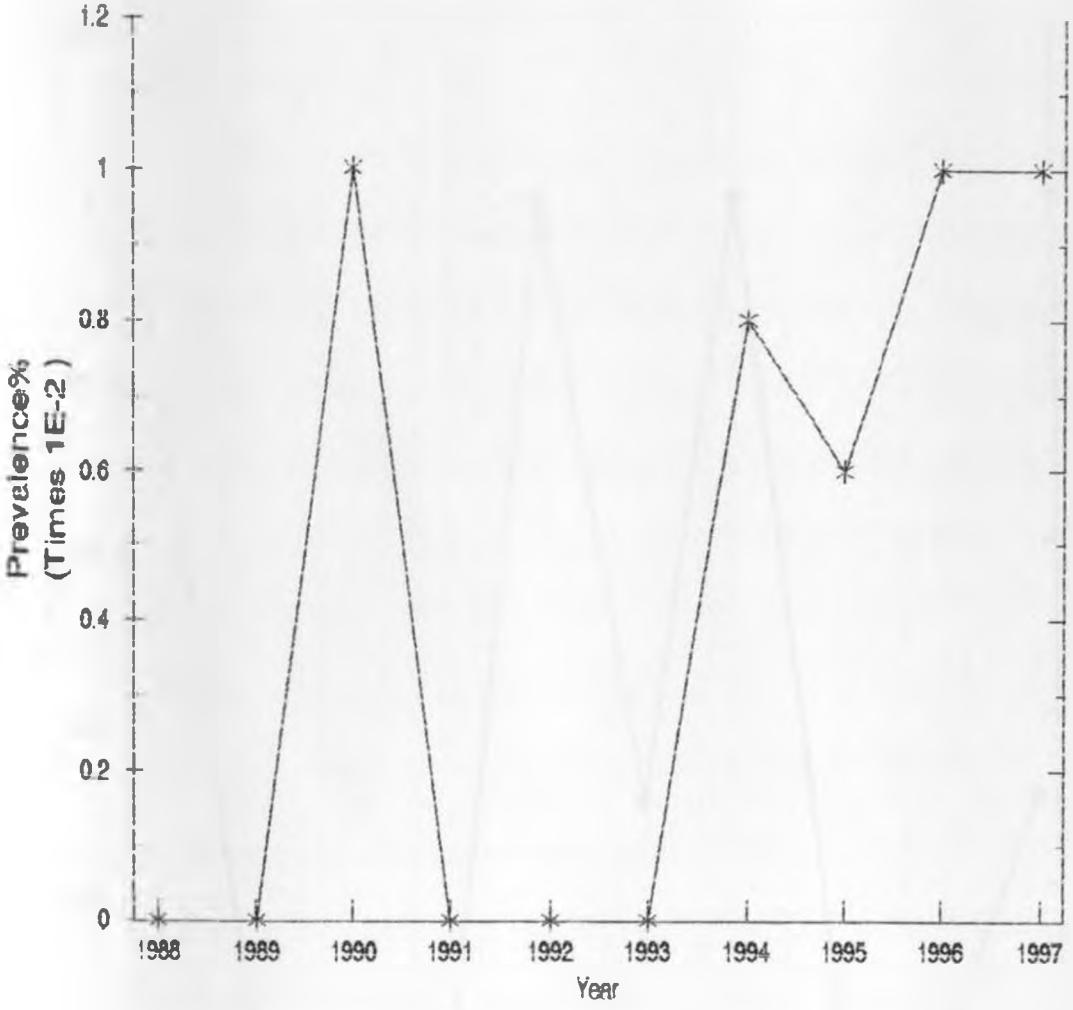

Fig. 7. Trends of abattoir prevalences of pig nodular worms over the 1988-1997 period

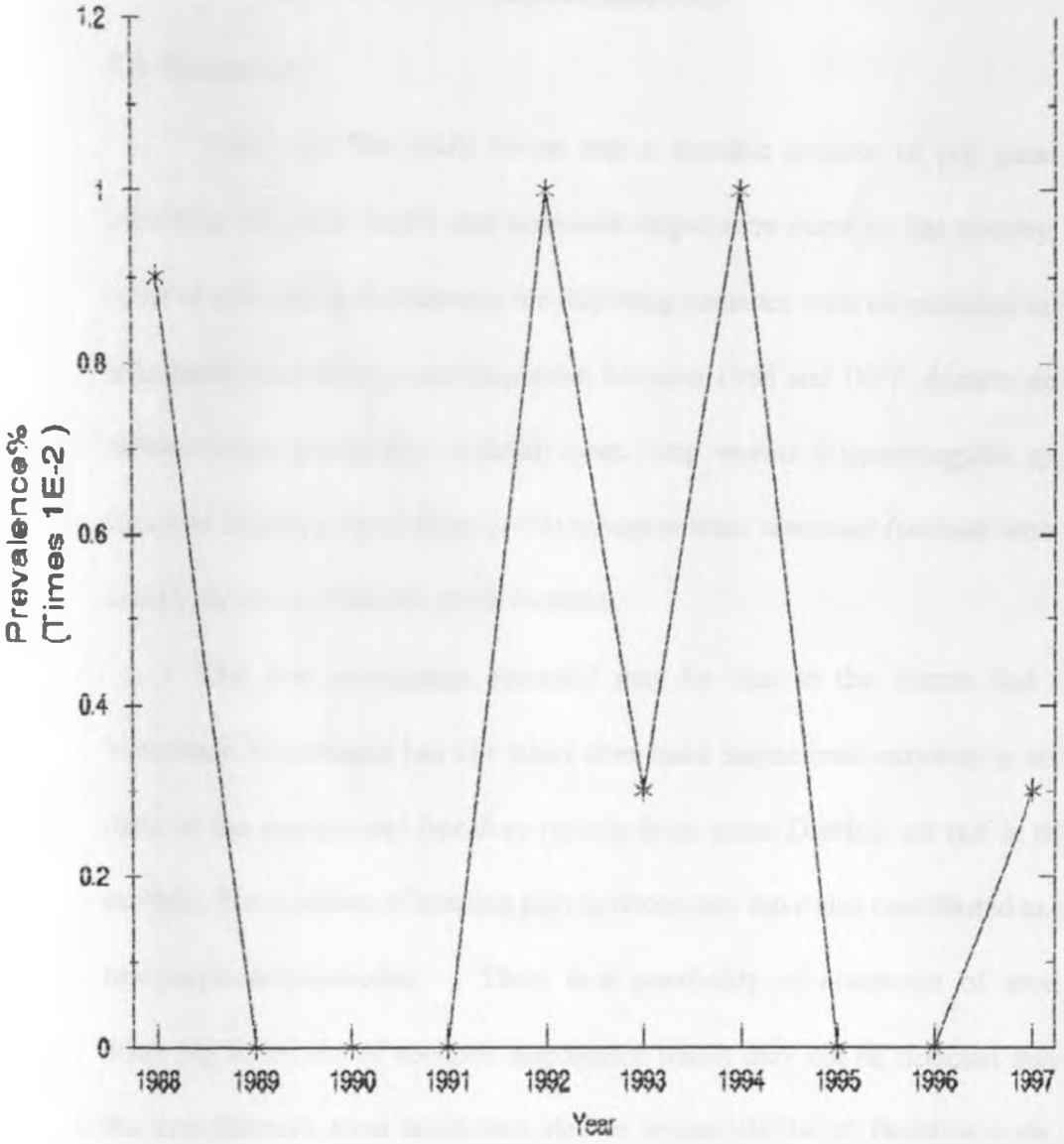

Fig. 8. Trends of abattoir prevalences of *Cysticercus cellulosae* over the 1988–1997 period

CHAPTER FIVE

5.0. Discussion

Results of this study reveal that a sizeable number of pig parasitic helminths of public health and economic importance occur in this country. In order of descending prevalences the following parasites were encountered in the slaughterhouses during meat inspection between 1988 and 1997: *Ascaris suum*, *Echinococcus granulosus* (hydatid) cysts, lung worms *(Metastrongylus* spp.), *Fasciola hepatica* (liver flukes), *Oesophagostomum dentatum* (nodular worms) and *Cysticercus cellulosae* (pork measles).

The low prevalences recorded may be due to the reason that the Veterinary Department has not taken over meat inspectorate services in some parts of the country and therefore records from some Districts are not in their custody. The tradition of keeping pigs in-doors may have also contributed to the low prevalences recorded. There is a possibility of occurence of several other pig helminths of zoonotic importance which may not be detected during the post-mortem meat inspection due to unavailability of facilities such as equiped laboratories within the slaughterhouses. Parasites may also be present in some stages that are not visible during routine meat inspection but may be observed if better diagnostic procedures were employed. The prevalence of *Ascaris suum* could have been higher since the absence of milk spots in the liver does not mean the absence of the parasite in the small intestines. This is evidenced by the results obtained from the various samples prospectively collected and analysed in the laboratory between May and July, 1998. Faecal

samples when quantitatively analysed for helminth eggs showed the occurence of *Ascaris suum* (10.7%), *Trichuris suis* (50.9%), Strongyles (3.6%) and cysts of coccidia (15.2%). At the same time, fecal egg counts may not be very reliable in that they may give false-negative results due to infections with immature worms of one sex only (Jungersen *et al.*,1997) or false positive results due to coprophagia (Boes *et al.*,1997).

Results obtained from the post-mortem examinations carried out at the Ndumboini and Farmers' Choice slaughterhouses showed high prevalences of liver ascariasis (15.7%) and 11.4% of the small intestinal ascariasis. These were followed by hepatic hydatid cysts (3.6%) and pulmonary hydatidosis (2.1%) and *Oesophagostomum* spp. (5.7%) as pimply guts in the large intestines. These prevalences were relatively higher than those computed from the records kept by the Ministry of Agriculture and Rural Development. Probably this is due to under reporting, improper recording and improper records keeping. In addition this may be attributed to incomplete inspection as evidenced by the absence of records on gastro-intestinal findings especially on ascariasis, missing of records from some districts for certain months or years.

Ascaris suum had the highest prevalence as compared to other parasites reported in Kenya as seen in the current study. This agrees with studies carried out in other countries of the world where *Ascaris suum* has been found to be more prevalent in in-door pig rearing system than out-door production system (Roepstorff and Jorsal, 1990). In the past, it was mandatory for pigs to be permanently kept indoors in Kenya. This tradition is still practised in most pig

rearing areas of the country. However, in some areas especially the western part of the country, pigs are reared in both the out-door and in-door production systems. Overall, the prevalence of *A. suum* in Kenya obtained in the current study is far below that recorded in other countries of the world. For example, Roepstorff and Jorsal (1989), reported a prevalence of 88% in Denmark.

During the post-mortem inspection carried out in this study, some pigs were found to have *Ascaris suum* in the small intestines, but there were no milk spots in the livers. Further, some pigs did not simultaneously have milk spots in their livers and adult worms in the small intestines. But on analysing faecal samples, ascarid eggs were present. This is probably due to the fact that milk spots have been reported to disappear within 3-6 weeks.Hence their presence only provides information on how recently the infection had occured (Eriksen *et al.*, 1980). Practical experience has shown that very often there is a poor correlation between uptake of infective eggs, establishment of intestinal worm burdens, presence of specific antibodies and numbers of milk spots per condemned liver, both in natural and eperimental infections (Eriksen *et al.*, 1980; Bernardo *et al.*, 1990a; Eriksen *et al.*, 1992a; Bogh *et al.*, 1994).

The prevalence (0.7%) of hydatidosis obtained f

rom the abattoir survey in this study was not only far below that recorded in other countries of Africa for the same animal species, but also lower than the prevalence of 0.9% earlier recorded by Gathura *et al.* in 1988 in this country. This prevalence was equal to that recorded in Algeria (Larbaoui and Alloula, 1980). Dada *et al.* (1979) recorded a prevalence of hydatid cysts of 5% in

Nigeria, while Rickard (1979) reported a prevalence of 28% in Tunisia, while Rahman *et al*., (1992) reported a prevalence of 4.6% in Egypt. Earlier studies carried out in Kenya, other domestic animals showed higher prevalences of hydatidosis than the ones recorded in this study. Gathura and Kamiya (1990), reported that 30% of cattle, 15% of goats and 13% of sheep harboured the infection. The low prevalences recorded for pigs in this study may be because most pigs in Kenya are mainly reared in-doors, therefore minimising exposure of pigs to dog feces which are the main sources of infection.

The low prevalence (0.03%) of lungworms (*Metastrongylus* spp.) reported in this study may be explained by the fact that this helminth requires earthworms as intermediate hosts and thus it is restricted to pig herds reared in the out-door production systems which are uncommon in Kenya. This compares well with the results of earlier studies carried out in Denmark (Roepstorff and Jorsal, 1989).

According to the current study, Liver flukes in the pigs are reported to occur in this country, though at a low prevalence. Although at present, there are no data available on human fascioliasis in Africa its public health risks cannot be ignored. The biology of the larval stages of the flukes, and also of the snail hosts, is influenced to a great extend by ecological factors such as temperature, water quality and speed of water current (Bundy et al., 1983, Fagbemi, 1984).

The prevalence of *Cysticercus cellulosae* over the ten year period was very low (0.004%). But, there is a feeling that there is under reporting mainly due to inadequate inspection since the only methods employed are visualisation,

palpation and incision with no laboratory examination. In recent years, knowledge concerning taeniasis-cysticercosis (*Taenia solium*) in the human patients has made considerable progress in the field of immunology, diagnostic procedures, treatment and molecular biology (Schantz *et al.*, 1992). In spite of this wealth of knowledge, the disease is prevalent in developing countries (Cruz *et al.*, 1989; Schantz *et al.*, 1992) where pork is consumed. Human neurocysticercosis (due to consumption of pork infected with *C. cellulosae)*, is a condition which has persisted over the years notwithstanding the available information. In the Hospital of Neurology in Mexico City, 13% of all neurosurgeries performed during 1995 were cases of neurocysticercosis (Sotelo *et al.*, 1996). In the developed world, the transmission of taeniasis/ cysticercosis has been virtually eradicated through the practice of strict meat inspection, the confinement of pigs and the obligatory installation of toilets in rural dwellings. These measures have not been enforced in most developing countries (Pawlowski, 1990; Nascimento *et al.*, 1995). The low prevalence of *Cysticercus cellulosae* in Kenya as noted in the current study may be due to the fact that most pig farmers rear their pigs in confinement. This may also be attributed to successful campaign by the government and non-governmental organisations in educating the public on general hygiene which include installation of at least one pit latrine in every homestead, though this has not been well implemented in the pastoralist areas because of nomadism. Fortunately, there is no pig rearing in these areas.

The absence of *Trichinella spiralis* in the sampled diaphragm muscles

may not be enough to rule out the presence of the parasite over the study period (1988-1997) because the sample size may have been small and the study was not done long enough. Also, diagnosis based on trichinoscopy or any other advanced method has not been widely used in the slaughterhouses in this country.

Reports on the pig gastrointestinal parasites based on the monthly and annual meat inspection records as seen in the present study was scanty in Kenya. This might be due to the little attention given to stomachs and intestines during inspection because their utilisation for human consumption is substantially low in Kenya. The examination carried out in the slaughterhouses and the laboratory results of the faecal samples collected during this study, revealed the presence of large round worms, *Ascaris suum* in the small intestines, *Oesophagostomum* spp., *Trichuris suis* and coccidial oocysts in the large intestines.

Faecal sample analysis revealed that there was a very high number of pigs shedding strongyle eggs. These eggs could either be from *Oesophagostomum* species or *Hyostrongylus rubidus*. Larval cultures (Alicata, 1935) were not performed to differentiate the two.

The main pig abattoir in Kenya, Farmers Choice received pigs for slaughter from almost all pig-rearing areas in Kenya and therefore records of parasites observed in this slaughterhouse would have reflected the distribution of the parasites in the pig rearing areas of the country.

In general, the overall mean annual prevalences of the parasites observed are far below those reported from other countries of the world for the same animal species. An abattoir survey of gastrointestinal parasites in pigs in

Malaysia (Lee *et al.*, 1987) and Nigeria (Salivu *et al.*, 1990) showed high rates of parasitic infections with an overall prevalence of over 85%. This was attributed to poor management practices and poor sanitary conditions. The low prevalences could be attributed to the fact that pigs were strictly reared in-doors in Kenya as opposed to other countries where free-range system is practised.

Though the prevalences of the various pig parasites in Kenya are lower than those recorded in other countries, there is a need for well co-ordinated abattoir surveys to determine the prevalences of these diseases within each of the pig rearing areas. There is also need to study the type of pig husbandry practised and climatical changes in these areas so as to determine their influence in the transmission of these diseases. This will help in planning of pig helminth control and / or eradication programmes.

Helminth infections in the pigs cannot easily be eradicated, but they can be controlled to prevent serious loss in terms of economy, animal and public health. Several effective anthelminthics are available for controlling helminth infections in pigs in Kenya. However, in view of the cost of drugs, chemotherapeutic means may not be financially viable in this country. Therefore, to formulate suitable recommendations concerning appropriate control measures, the seasonal transmission pattern of these parasitic infections in the enzootic areas should be determined.

5.1. conlusions

1. Pig parasitic helminths of public health and economic importance occurs in Kenya. They include: *Ascaris suum*, *Echinococcus* (hydatid) cysts, Liver flukes

(*Fasciola* spp.). Lungworms (*Metastrongylus* spp.), Nodular worms (*Oesophagostomum* spp.), pork measles (*Cysticercus cellulosae*). ***Trichinella spiralis*** was not reported in this country during the 1988-1997 period. It was not also detected in the sampled diaphragm muscles.

2. *Ascaris suum* had a significantly higher prevalence of 4.9% than other helminths according to the records kept by the Ministry of Agriculture and Rural Development However this was higher according to the actual inspection carried out in the slaughter houses and sampled faecal samples where prevalences of 27.1% and 10.7% respectively were recorded.

3. Abattoir data are inaccurate indicators of prevalences and distributions of pig helminthiasis. However they only suggest the existence of the infections in the field.

CHAPTER SIX

6.0. REFERENCES

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CHAPTER SEVEN

7.0. APPENDICES

Appendix 1: the distribution of pig parasites recorded in slaughter houses in Kenya over a ten year period (1988-1997)

YEAR	A.S	H.C.	L.W	L.F	N.W.	C.C	T.N.I.	T.N.S.
1988	4,442	258	58	---	---	4	4,762	43,915
1989	202	19	21	---	---	---	242	39,694
1990	320	1,370	37	---	6	---	1,733	57,853
1991	4,684	641	---	113	---	---	5,438	54,577
1992	3,012	376	14	14	---	6	3,422	53,776
1993	4,951	520	40	10	---	2	5,523	62,003
1994	4,004	479	16	---	6	8	4,513	71,993
1995	2,308	204	2	25	5	---	2,544	79,057
1996	3,210	702	21	3	12	---	3,948	90,249
1997	4,232	164	6	---	9	3	4,414	89,178
TOTAL	31,365	4,733	215	165	38	23	36,539	642,295

KEY

T.N.S.== TOTAL NO. OF PIGS SLAUGHTERED

T.N.I.== TOTAL NO. OF PIGS INFECTED

A.S. == *ASCARIS SUUM*

H.C. == HYDATID CYSTS

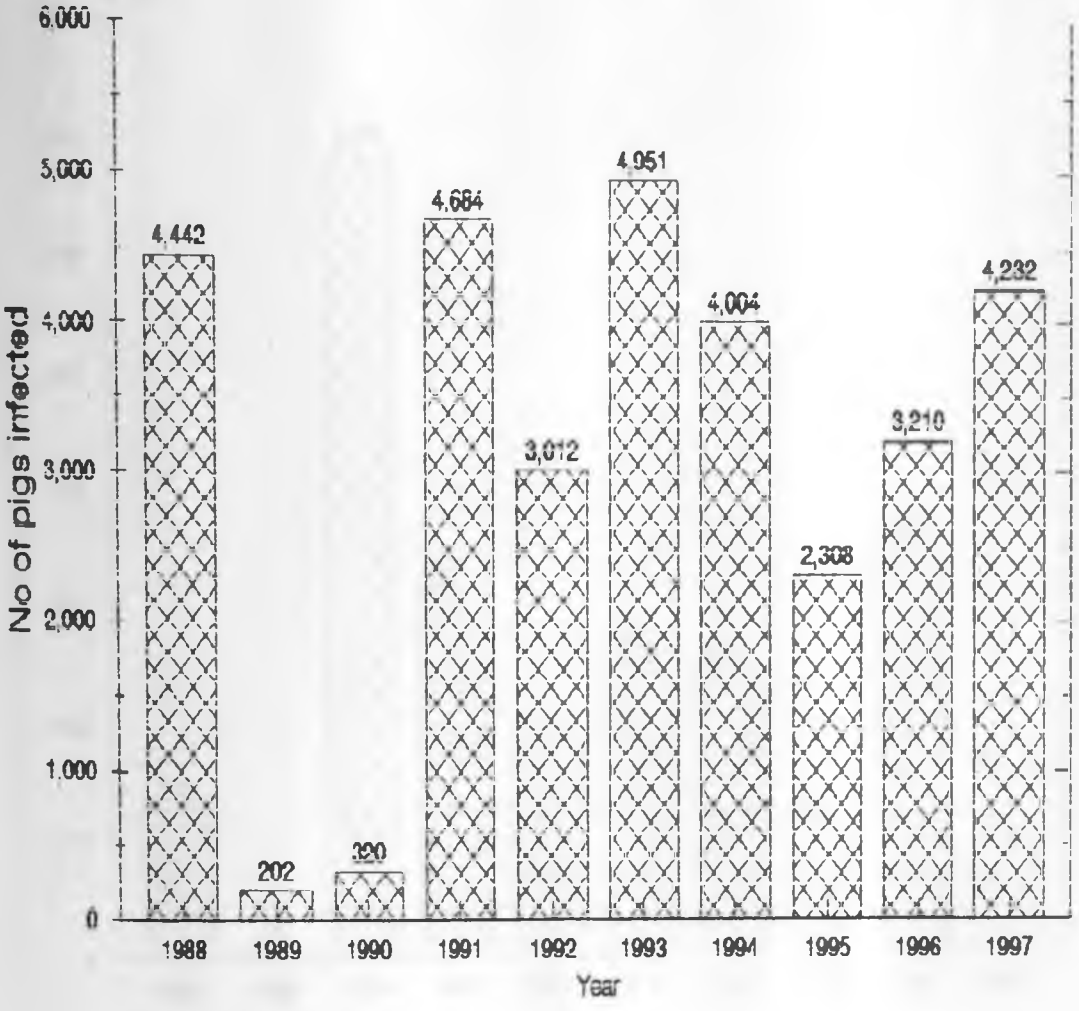
C.C.==*CYSTICERCUS CELLULOSAE*

L.W. == LUNG WORMS

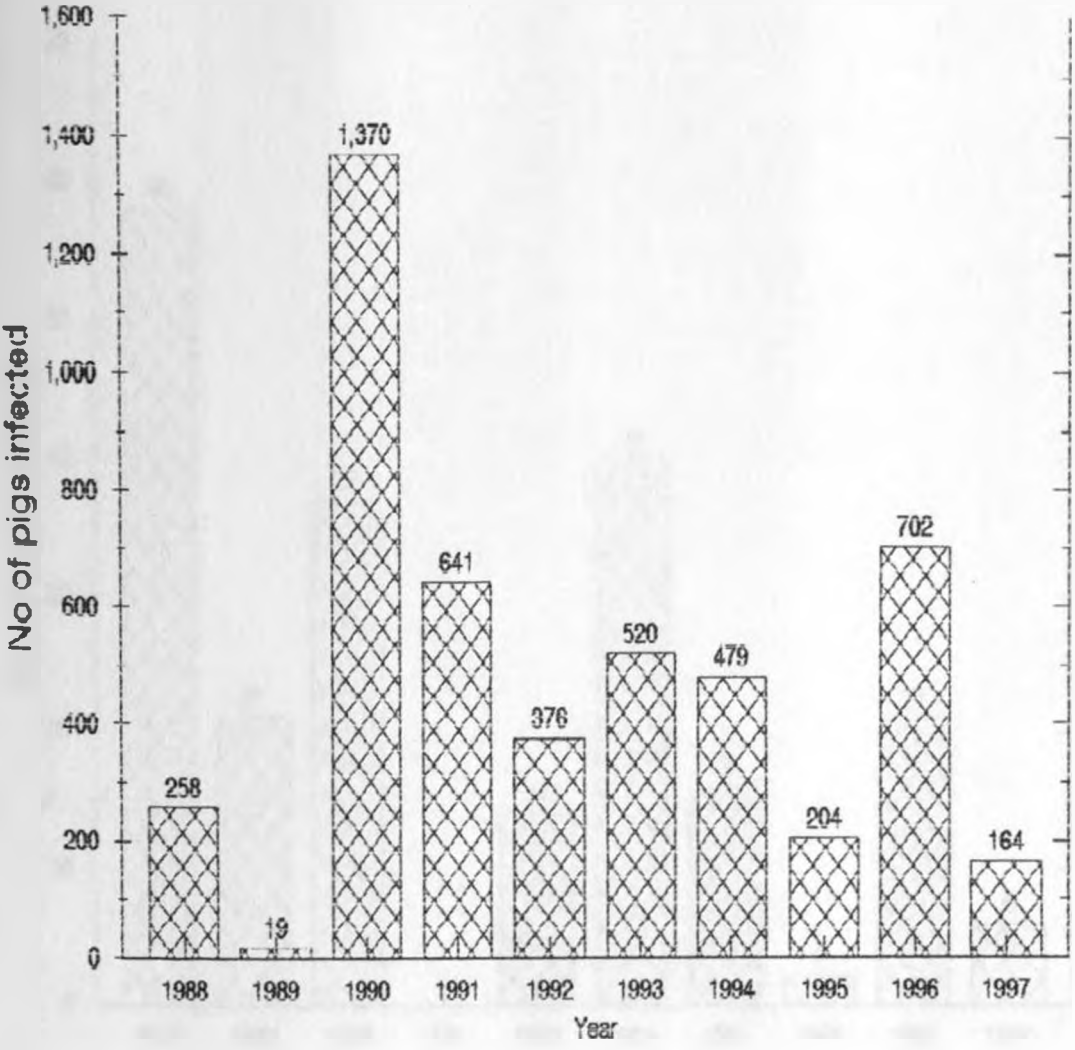
L.F. == LIVER FLUKES

N.W. == NODULAR WORMS

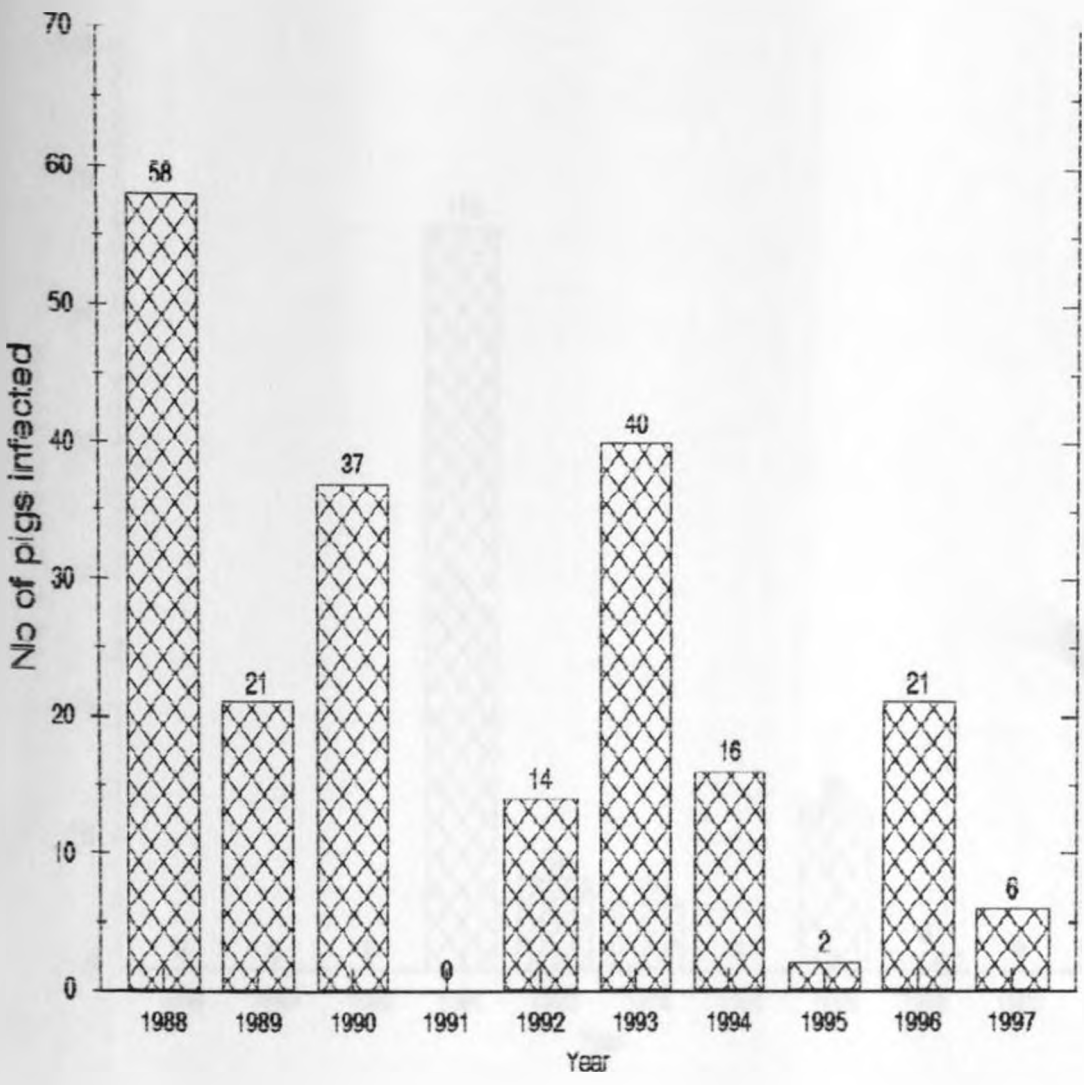
Appendix 2. Annual occurences of *Ascaris suum* for the 1988–1997 period



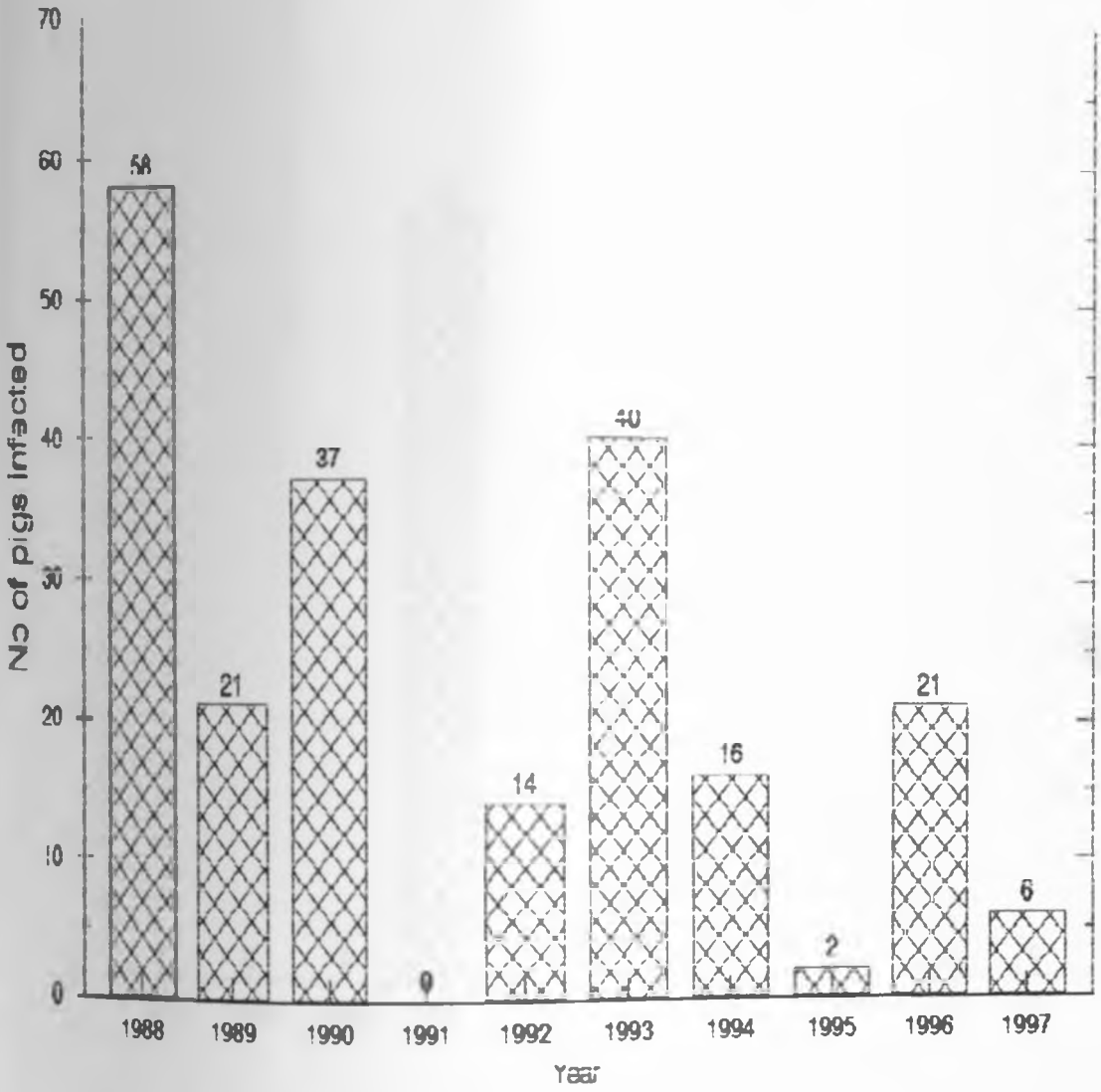
Appendix 3. Annual occurences of swine hydatid disease for the 1988–1997 period



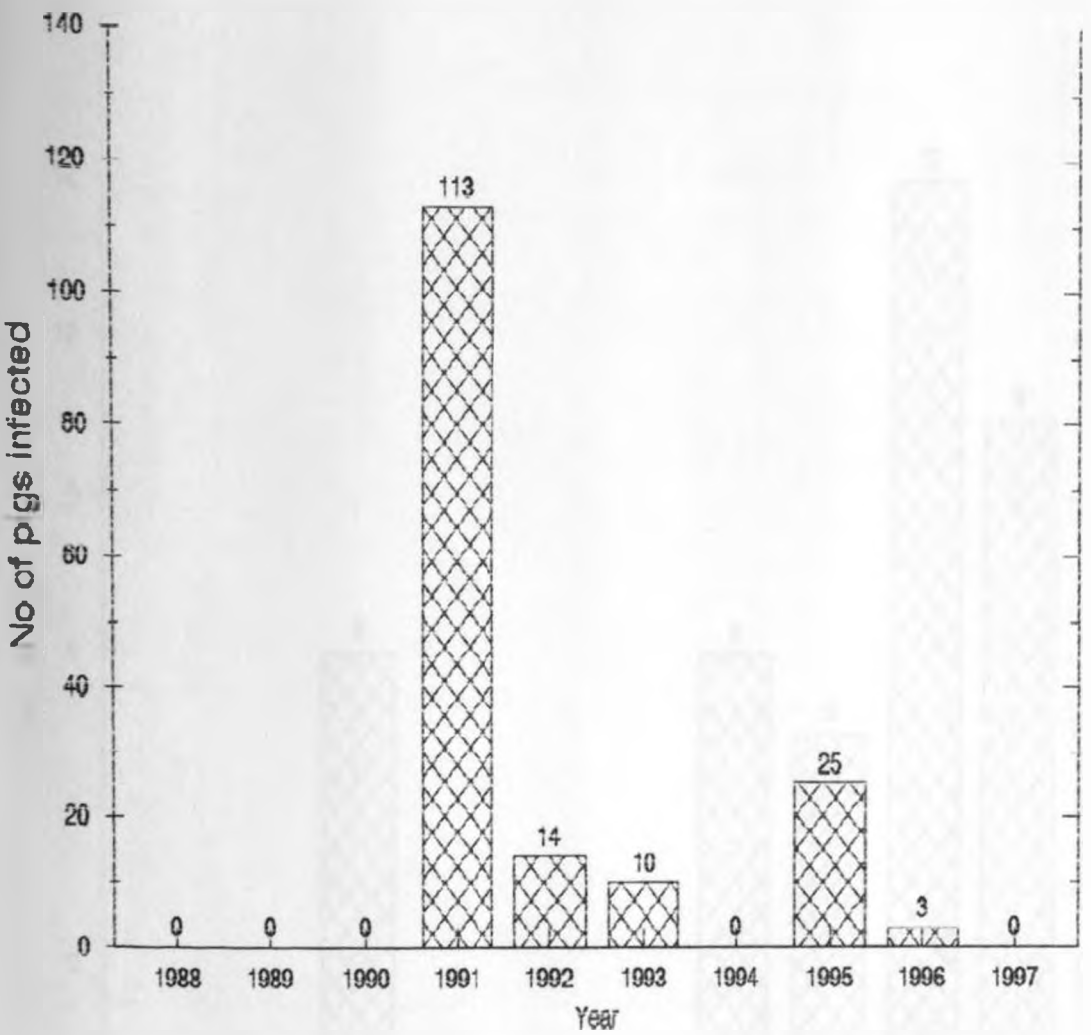
Appendix 4. Annual occurences of swine lung worms for the 1988-1997 period



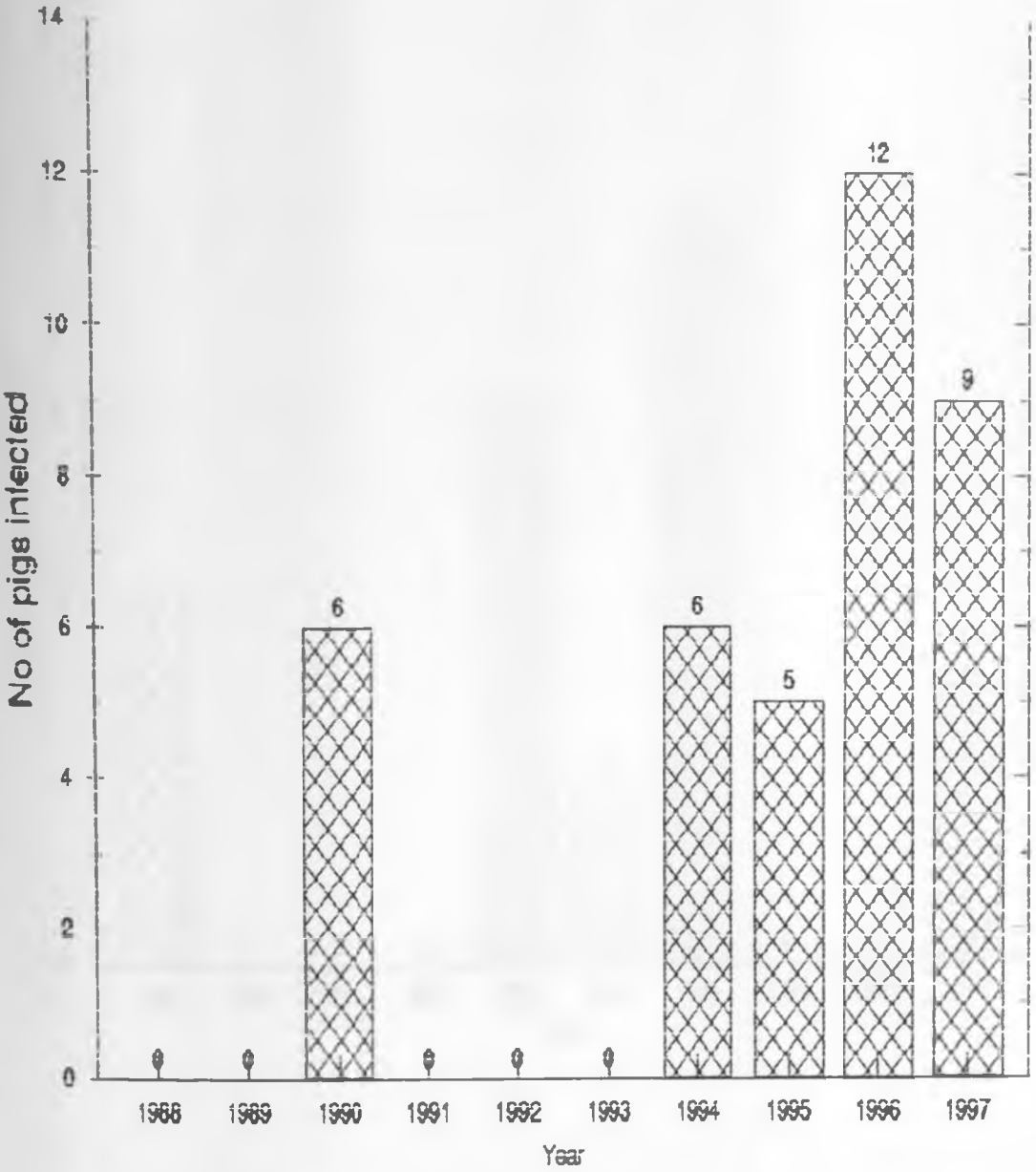
Appendix 4. Annual occurences of swine lung worms for the 1988-1997 period



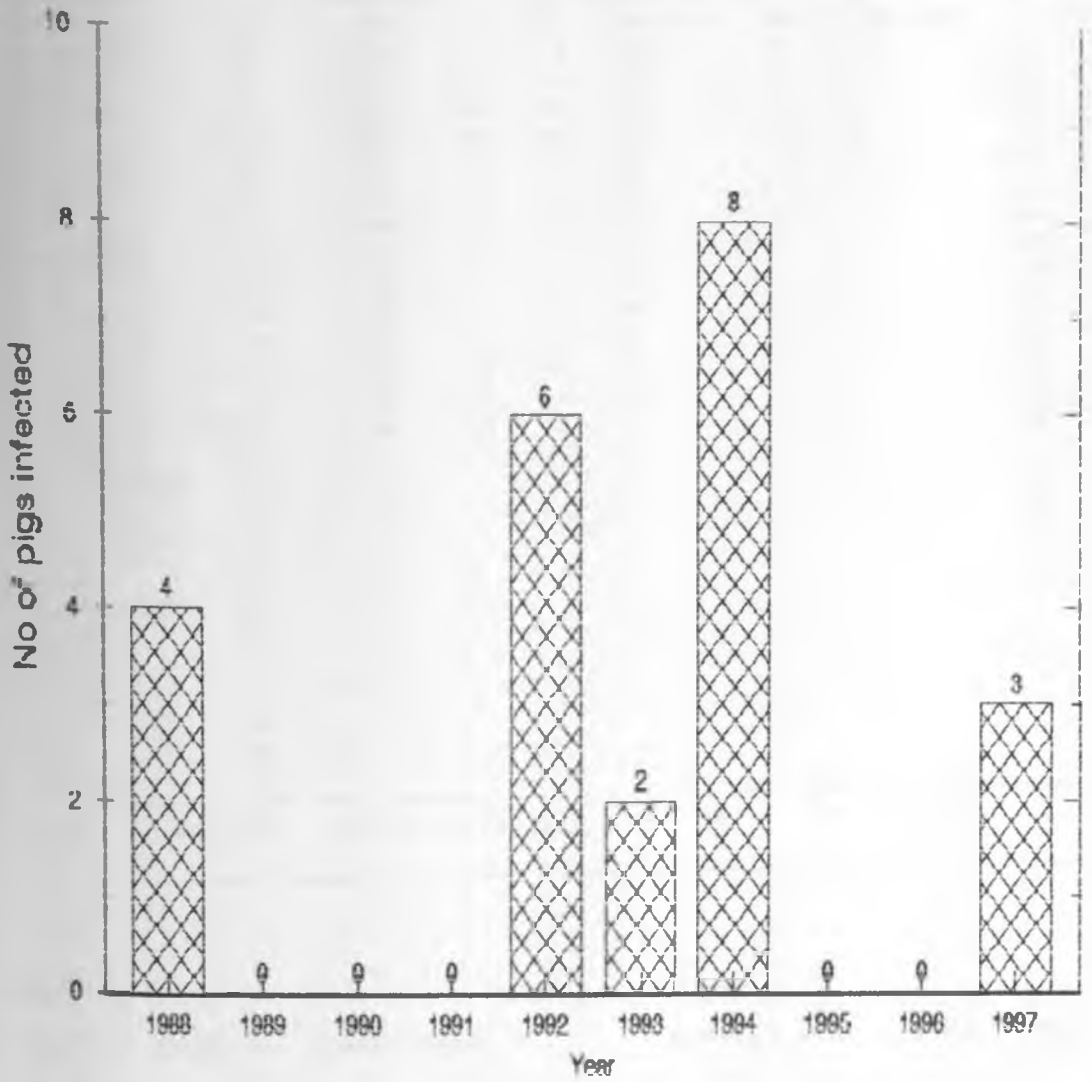
Appendix 5. Annual occurences of swine liver flukes for the 1988-1997 period



Appendix 6. Annual occurences of swine nodular worms for the 1988-1997 period



Appendix 7. Annual occurences of *Cysticercus cellulosae* for the 1988-1997 period



Appendix 8: The number of cases of the various pig parasites as reported in meat inspection records from various districts in Kenya (1988-1997)

DISTRICT	T.N.S.	A.S.	H.C.	L.W.	L.F.	N.W.	C.C.	T.N.I.
NAIROBI	536,954	23,387	3,719	65	159	---	16	27,346
KIRINYAGA	24,891	1,959	150	6	---	6	---	2,121
KIAMBU	18,037	1,851	271	---	---	---	5	2,127
KAKAMEGA	13,732	1,642	260	92	2	5	---	2,001
BUSIA	9,610	645	46	33	---	---	---	724
MURANG'A	9,144	878	55	---	2	17	---	952
NAKURU	8,905	244	26	---	---	---	2	272
EMBU	5,845	378	25	3	---	---	---	406
NYANDARUA	2,351	93	28	3	1	---	---	125
NYERI	1,943	9	---	---	---	---	---	9
KILIFI	1,937	49	---	---	---	---	---	49
VIHIGA	1,759	21	---	---	1	---	---	22
BUNGOMA	1,537	82	80	---	---	---	---	162
KWALE	1,190	20	18	---	---	6	---	44
KISUMU	1,053	48	50	---	---	---	---	98
MOMBASA	893	---	---	---	---	---	---	---
TAITA TAVETA	859	---	---	---	---	---	---	---
TESO	456	15	---	13	---	---	---	28
TRANS NZOIA	435	---	---	---	---	---	---	---
THIKA	274	33	2	---	---	---	---	35
UASIN GISHU	106	---	---	---	---	---	---	---
KERICHO	105	---	---	---	---	---	---	---
LAIKIPIA	84	---	---	---	---	---	---	---
MACHAKOS	64	2	3	---	---	---	---	5
LUGARI	62	9	---	---	---	4	---	13
KISII	41	---	---	---	---	---	---	---
KAJIADO	28	---	---	---	---	---	---	---
TOTAL	642,295	31,365	4,733	215	165	38	23	36,539

KEY

T.N.S.: TOTAL NO. SLAUGHTERED

INFECTED

A.S.---- *ASCARIS SUUM*

L.W.----LUNG WORMS

N.W.----NODULAR WORMS

CELLULOSAE

T.N.I. : TOTAL NO.

H.C.----HYDATID CYSTS

L.F.-----LIVER FLUKES

C.C.----*CYSTICERCUS*

Appendix 9: Number of liver condemnations reported in Kenya due to presence of *Ascaris suum*

YEAR	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
1988	736	664	83	250	250	237	248	230	248	256	257	283
1989	39	23	18	11	24	15	15	4	12	13	10	18
1990	25	34	38	20	22	18	27	30	23	24	15	44
1991	387	350	374	377	398	402	402	402	396	380	395	421
1992	17	20	15	394	328	430	343	245	7	488	336	389
1993	323	446	418	559	509	511	339	454	300	319	231	542
1994	436	44	149	26	423	190	224	155	515	431	682	330
1995	49	74	9	98	114	191	182	254	72	325	429	423
1996	379	201	214	261	75	296	274	277	215	273	323	422
1997	253	198	25	225	408	228	201	195	362	671	595	639
T.I.	2644	2453	1663	2221	2551	2518	2255	2246	2150	3180	3273	3511
M.I.	264	245	166	222	255	252	226	225	215	318	327	351

KEY

T.I.= TOTAL NUMBER OF LIVERS INFECTED WITH *ASCARIS SUUM*

M.I.= MEAN NUMBER OF LIVERS INFECTED WITH *ASCARIS SUUM*

ix 10: Number of pig carcass condemnations reported in Kenya due to presence of *Cysticercus cellulosae*

EB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
0	2	0	0	0	0	0	0	0	1	0
0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	5	0	1
0	0	0	0	0	0	0	0	0	2	0
0	1	0	3	0	1	2	0	0	0	1
0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0
0	0	0	2	0	0	0	0	0	1	0
0	3	0	5	0	1	2	0	5	4	2
0	0.3	0	0.5	0	0.1	0.2	0	0.5	0.4	0.2

al number of carcasses infected with *C. cellulosae*

n number of carcasses infected with *C. cellulosae*

Appendix 11: Number of liver and lung condemnations reported in Kenya due to presence of hydatid cysts

YEAR	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
1988	59	63	57	15	11	10	0	8	6	12	2	15
1989	1	0	3	0	2	0	3	2	1	4	0	3
1990	78	96	119	115	129	109	114	119	107	118	126	140
1991	44	52	49	50	64	53	58	51	51	51	47	71
1992	1	4	9	42	51	60	24	38	91	16	15	25
1993	76	38	33	62	25	34	88	39	26	37	22	40
1994	92	34	77	65	55	24	17	11	29	57	16	2
1995	0	0	17	12	48	20	40	21	13	35	8	10
1996	32	33	40	53	88	102	97	143	66	20	12	16
1997	25	3	4	2	10	11	7	54	18	11	12	17
T.I.	408	323	408	416	483	423	448	486	408	361	260	339
M.I.	40.8	32.3	40.8	41.6	48.3	42.3	44.8	48.6	40.8	36.1	26.0	33.9

KEY

T. I. = TOTAL NUMBER OF ORGANS INFECTED WITH HYDATID CYSTS

M. I. = MEAN NUMBER OF ORGANS INFECTED WITH HYDATID CYSTS

Appendix 12: Number of pigs reported to be infected with *Oesophagostomum dentatum* in Kenya

EAR	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
1988	0	0	0	0	0	0	0	0	0	0	0	0
1989	0	0	0	0	0	0	0	0	0	0	0	0
1990	1	0	2	0	0	0	0	0	0	0	1	2
1991	0	0	0	0	0	0	0	0	0	0	0	0
1992	0	0	0	0	0	0	0	0	0	0	0	0
1993	0	0	0	0	0	0	0	0	0	0	0	0
1994	0	0	0	0	2	0	0	1	0	0	0	3
1995	0	0	0	1	0	1	0	0	0	0	3	0
1996	2	8	1	0	0	0	0	1	0	0	0	0
1997	0	0	0	3	0	0	0	0	1	2	1	2
T.I.	3	8	3	4	2	1	0	2	1	2	5	7
M.I.	0.3	0.8	0.3	0.4	0.2	0.1	0.0	0.2	0.1	0.2	0.5	0.7

KEY

T. I. = Total number of pigs infected with *O. dentatum*

M. I. = Mean number of pigs infected with *O. dentatum*

Appendix 13: Number of lungs condemned due to presence of lung worms in Kenya

YEAR	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
1988	9	2	9	5	3	4	11	1	3	5	4	2
1989	2	1	2	0	0	1	4	3	4	1	0	5
1990	6	1	3	0	2	9	7	3	0	4	0	2
1991	0	0	0	0	0	0	0	0	0	0	0	0
1992	2	0	1	0	3	3	1	2	0	0	0	2
1993	0	0	0	0	0	0	0	0	0	0	0	0
1994	2	0	1	1	3	0	3	2	0	1	0	3
1995	0	0	0	0	0	0	0	2	0	0	0	0
1996	2	0	2	0	2	2	0	6	0	0	1	6
1997	1	1	3	0	0	0	0	0	0	0	0	1
T.I.	24	5	21	6	13	19	26	19	7	11	5	21
M.I.	2.4	0.5	2.1	0.6	1.3	1.9	2.6	1.9	0.7	1.1	0.5	2.1

KEY

T. I. = Total number of lungs infected with lungworms

M. I. = Mean number of lungs infected with lungworms

Appendix 14 Number of livers condemned due to presence of liver flukes in Kenya

YEAR	JAN	FEB	MAR	APR	MAY	JUN	JUL	AUG	SEP	OCT	NOV	DEC
1988	0	0	0	0	0	0	0	0	0	0	0	0
1989	0	0	0	0	0	0	0	0	0	0	0	0
1990	0	0	0	0	0	0	0	0	0	0	0	0
1991	10	6	15	13	10	9	8	12	13	7	3	7
1992	1	1	0	0	2	3	1	3	2	1	0	0
1993	0	0	0	0	0	0	0	0	0	0	10	0
1994	0	0	0	0	0	0	0	0	0	0	0	0
1995	1	0	0	0	0	2	8	0	0	0	14	0
1996	0	0	1	0	1	0	1	0	0	0	0	0
1997	0	0	1	0	1	0	1	0	0	0	0	0
T.I.	12	7	17	13	14	14	19	15	15	8	27	7
M.I.	1.2	0.7	1.7	1.3	1.4	1.4	1.9	1.5	1.5	0.8	2.7	0.7

KEY

T. I. = Total number of livers infected with liver flukes

M. I. = Mean number of livers infected with liver flukes

Appendix 15: The number of cases of parasitic helminth infections reported in pig abattoirs monthly for a period of ten years (1988-1997) in Kenya

1988

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	736	664	83	250	250	237	248	230	248	256	257	283	4442
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	---	---	---	---	---	---	---	---	---	---	---	---	---
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	1	---	2	---	---	---	---	---	---	---	1	---	4
Hydatid cysts	59	63	57	15	11	10	---	8	6	12	2	15	258
Liver flukes	---	---	---	---	---	---	---	---	---	---	---	---	---
Metastrongylus	9	2	9	5	3	4	11	1	3	5	4	2	58
T. slaughtered	6515	5320	6039	2567	2839	2577	2717	2918	2684	3137	3192	3400	43,915

Appendix 15: Cont.

1989

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY.	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	39	23	18	11	24	15	15	4	12	13	10	18	202
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	---	---	---	---	---	---	---	---	---	---	---	---	---
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	---	---	---	---	---	---	---	---	---	---	---	---	---
Hydatid cysts	1	---	3	---	2	---	3	2	1	4	---	3	19
Liver flukes	---	---	---	---	---	---	---	---	---	---	---	---	---
Metastrongylus	2	1	2	---	---	1	4	3	4	1	---	5	21
T. slaughtered	3207	3119	3350	3186	3392	3063	3327	3229	3243	3521	3422	3635	39,694

Appendix 15: Cont.

1990

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	25	34	38	20	22	18	27	30	23	24	15	44	320
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	1	---	2	---	---	---	---	---	---	---	1	2	6
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	---	---	---	---	---	---	---	---	---	---	---	---	---
Hydatid cysts	78	96	119	115	129	109	114	119	107	118	126	140	1370
Liver flukes	---	---	---	---	---	---	---	---	---	---	---	---	---
Metastrongylus	6	1	3	---	2	9	7	3	---	4	---	2	37
T. slaughtered	5136	4556	5042	4426	4755	3957	4479	4818	4501	5323	5365	5492	57,853

Appendix 15: Cont.

1991

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY.	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	387	350	374	377	398	402	402	402	396	380	395	421	4684
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	---	---	---	---	---	---	---	---	---	---	---	---	---
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	---	---	---	---	---	---	---	---	---	---	---	---	---
Hydatid cysts	44	52	49	50	64	53	58	51	51	51	47	71	641
Liver flukes	10	6	15	13	10	9	8	12	13	7	3	7	113
Metastrongylus	---	---	---	---	---	---	---	---	---	---	---	---	---
T. slaughtered	4976	4353	4072	4396	4234	3997	4643	4852	4662	4827	4422	5143	54,577

Appendix 15: Cont.

1992

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	17	20	15	394	328	430	343	245	7	488	336	389	3012
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	---	---	---	---	---	---	---	---	---	---	---	---	---
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	---	---	---	---	---	---	---	---	---	5	---	1	6
Hydatid cysts	1	4	9	42	51	60	24	38	91	16	15	25	376
Liver flukes	1	1	---	---	2	3	1	3	2	1	---	---	14
Metastrongylus	2	---	1	---	3	3	1	2	---	---	---	2	14
T. slaughtered	4307	4148	4590	4323	3984	4426	4322	4671	4626	4390	4747	5232	53,776

Appendix 15: Cont.

1993

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	323	446	418	559	509	511	339	454	300	319	231	542	4,951
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	---	---	---	---	---	---	---	---	---	---	---	---	---
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	---	---	---	---	---	---	---	---	---	---	2	---	2
Hydatid cysts	76	38	33	62	25	34	88	39	26	37	22	40	520
Liver flukes	---	---	---	---	---	---	---	---	---	---	10	---	10
Metastrongylus	---	---	---	---	---	---	---	---	---	---	---	---	---
T. slaughtered	3986	4232	5017	5635	4524	4516	4798	5446	5155	4866	6827	6999	62,003

Appendix 15: Cont.

1994

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY.	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	436	443	149	26	423	190	224	155	515	431	682	330	4004
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	---	---	---	---	2	---	---	1	---	---	---	3	6
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	---	---	1	---	3	---	1	2	---	---	---	1	8
Hydatid cysts	92	34	77	65	55	24	17	11	29	57	16	2	479
Liver flukes	---	---	---	---	---	---	---	---	---	---	---	---	---
Metastrongylus	2	---	1	1	3	---	3	2	---	1	---	3	16
T. slaughtered	5132	5398	6502	5087	5516	4854	5603	1175	6378	6461	6592	7525	71,993

Appendix 15: Cont.

1995

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	49	74	97	98	114	191	182	254	72	325	429	423	2308
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	---	---	---	1	---	1	---	---	---	---	3	---	5
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	---	---	---	---	---	---	---	---	---	---	---	---	---
Hydatid cysts	---	---	17	12	48	20	40	21	13	35	8	10	204
Liver flukes	1	---	---	---	---	2	8	---	---	---	14	---	25
Metastrongylus	---	---	---	---	---	---	---	2	---	---	---	---	2
T. slaughtered	5984	5519	6858	5357	6384	5974	6450	6736	6758	7152	7742	8143	79,057

Appendix 15: Cont.

1996

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	379	201	214	261	75	296	274	277	215	273	323	422	3210
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	2	8	1	---	---	---	---	1	---	---	---	---	12
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	---	---	---	---	---	---	---	---	---	---	---	---	---
Hydatid cysts	32	33	40	53	88	102	97	143	66	20	12	16	702
Liver flukes	---	---	1	---	1	---	1	---	---	---	---	---	3
Metastrongylus	2	---	2	---	2	2	---	6	---	---	1	6	21
T. slaughtered	6957	6723	6676	7062	7144	6550	7632	7928	7435	8488	8324	9330	90,249

Appendix 15: Cont.

1997

MONTHS OF THE YEAR

PARASITE	JAN.	FEB.	MAR.	APR.	MAY	JUN.	JUL.	AUG.	SEP.	OCT.	NOV.	DEC.	TOT.
Ascaris suum	253	198	257	225	408	228	201	195	362	671	595	639	4232
T. spiralis	---	---	---	---	---	---	---	---	---	---	---	---	---
Trichuris suis	---	---	---	---	---	---	---	---	---	---	---	---	---
Oes. dentatum	---	---	---	3	---	---	---	---	1	2	1	2	9
H. rubidus	---	---	---	---	---	---	---	---	---	---	---	---	---
S. dentatus	---	---	---	---	---	---	---	---	---	---	---	---	---
M. hirudinaceus	---	---	---	---	---	---	---	---	---	---	---	---	---
C. cellulosae	---	---	---	---	2	---	---	---	---	---	1	---	3
Hydatid cyst	25	3	4	2	10	11	7	54	18	11	12	17	164
Liver flukes	---	---	1	---	1	---	1	---	---	---	---	---	3
Metastrongylus	1	1	3	---	---	---	---	---	---	---	---	1	6
T. slaughtered	8042	7382	7366	7073	7346	7179	7250	7240	7298	7898	8069	8469	89,178

Appendix 16: Abattoir prevalences of pig helminth parasites in Kenya for a ten-year period (1988-1997)

YEAR	A.S.(%)	H.C.(%)	L.W.(%)	L.F.(%)	N.W.(%)	C.C.(%)	O.P.(%)
1988	10.1	0.59	0.13	0.00	0.00	0.009	10.84
1989	0.51	0.05	0.05	0.00	0.00	0.00	0.61
1990	0.55	2.37	0.06	0.00	0.01	0.00	3.00
1991	8.58	1.17	0.00	0.21	0.00	0.00	9.96
1992	5.6	0.70	0.003	0.003	0.00	0.01	6.36
1993	7.99	0.84	0.06	0.02	0.00	0.003	8.91
1994	5.56	0.67	0.02	0.00	0.008	0.01	6.27
1995	2.92	0.26	0.003	0.03	0.006	0.00	3.22
1996	3.56	0.78	0.02	0.003	0.01	0.00	4.37
1997	4.75	0.18	0.007	0.00	0.01	0.003	4.95
M.P.	4.88	0.74	0.03	0.03	0.006	0.004	5.70

KEY:

A.S.== *ASCARIS SUUM* H.C.== HYDATID CYSTS

L.W.== LUNG WORMS L.F.== LIVER FLUKES

N.W.== NODULAR WORMS C.C.== *CYSTICERCUS CELLULOSAE*

M.P.== MEAN PREVALENCE

O.P.== OVERALL ANNUAL PREVALENCE OF ALL HELMINTHIC PARASITES

Appendix 17: Quantitative fecal egg counts (e.p.g. == eggs per gram) reported from fecal samples collected from pigs at the time of slaughter in Kenya

PIG	STRONGYLE	ASCARID	TRICHURID
1	0	0	0
2	200	0	0
3	300	0	0
4	0	0	0
6	1000	0	0
7	0	0	0
8	200	0	0
10	800	0	0
11	100	0	0
12	300	0	0
13	0	0	4,400
14	0	0	0
15	200	0	0
16	0	0	0
17	0	0	0
18	0	0	0
19	0	0	0
20	0	0	0
21	0	0	0
22	100	0	0
25	0	0	0
26	0	0	0
27	0	0	0
28	0	0	0
29	0	0	0
30	0	0	0
31	0	0	0
32	100	0	0
33	0	0	0
34	1,800	0	100
35	200	0	0
36	400	0	0
37	700	0	3,000
38	1,400	2,800	1,200

PIG	STRONGYLE	ASCARID	TRICHURID
39	0	0	0
40	300	0	0
41	700	0	0
42	500	3,500	0
43	100	5,000	0
44	0	300	0
45	0	0	0
46	500	900	0
47	0	1,500	0
48	300	500	0
49	100	0	0
50	100	0	0
51	0	0	0
52	100	0	0
53	0	0	0
54	100	0	0
55	100	0	0
66	0	0	0
67	0	0	0
68	100	0	0
69	2,400	0	0
70	300	0	0
71	200	0	0
72	100	0	0
73	1,100	0	0
75	500	100	0
76	0	0	0
78	4,600	0	0
79	100	100	0
80	100	0	0
81	0	0	0
82	200	0	0
83	0	0	0
84	200	0	0
85	900	0	0
86	200	700	0
87	300	100	0
88	0	0	0

PIG	STRONGYLE	ASCARID	TRICHURID
90	0	0	0
93	0	0	0
94	200	0	0
95	500	0	0
96	0	0	0
97	0	0	0
98	0	0	0
99	0	0	0
100	100	0	0
101	0	0	0
102	100	0	0
103	100	0	0
105	300	0	0
106	0	0	0
107	100	0	0
108	100	0	0
109	0	0	0
110	0	0	0
111	0	0	0
112	100	0	0
113	200	0	0
114	0	0	0
115	0	0	0
116	300	0	0
117	0	0	0
118	0	0	0
119	0	0	0
120	0	0	0
121	200	0	0
122	0	0	0
123	200	0	0
124	100	0	0
125	0	0	0
126	100	0	0
127	600	0	0
128	0	0	0