

A Review on the Public Health and Economic Significance of Fasciolosis

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Abstract: This work was undertaken to review and compile available literature information on the characteristics of the parasite; about its epidemiology, public health significance and economic impact of bovine Fasciolosis. *Fasciola*, which belongs to phylum platyhelminthes, is caused by parasitic flat worms of liver fluke which infest the liver of various animals. Bovine fasciolosis is a parasitic disease of cattle caused by trematodes usually *Fasciola gigantica* and *Fasciola hepatica* in the tropics. The life cycle of these trematodes involves snail as an intermediate host. The disease is found in vast water lodged and marshy grazing field condition anticipated to be ideal for the propagation and maintenance of high prevalence of fasciolosis. This disease is widely distributed in areas where cattle are raised and there is a niche for Lymnaeid snail. It is an important limiting factor for bovine production. It causes several economic losses. The losses may be direct or indirect. The flukes cause severe liver damage and results in total condemnation of liver. Diagnosis of bovine fasciolosis is based on clinical sign, grazing history, seasonal occurrence, examination of faeces by laboratory tests and post-mortem examination. In cattle, chronic form of the disease is more common and drugs like rafoxanide, nitroxylin and Triclabendazole are more effective. The prevention and control of the disease should be targeted on regular application of anthelmintics, reducing the population of snail intermediate hosts through drainage of their habitats and restriction of the movement of animals from snail intermediate host habitats or fencing these dangerous zones.

Key words: Fasciolosis • Public Health Significance • Economic Significance

INTRODUCTION

Despite its huge population size, Ethiopian livestock productivity remains marginal due to various diseases, malnutrition and management constraints [1]. Parasitism is the major problem that affects the productivity of livestock worldwide [2]. Among many parasitic problems of domestic animals, fasciolosis is the most important parasitic disease in domestic ruminants throughout the world. The disease is caused by digenean of the genus *Fasciola* commonly referred to as liver flukes. The two species most commonly implicated as the etiological agents of fasciolosis are *F. hepatica* and *F. gigantica* [3].

Fasciola can infect a wide variety of domesticated animals, wildlife and people [4] and are the most prevalent helminth infection of ruminants in different parts of the world including Ethiopia [5, 6]. *Fasciola hepatica* has a cosmopolitan distribution [2]. Infection with *F. gigantica*

is regarded as one of the most common single helminth infection of ruminants in Asia and Africa. Its economic importance is mostly obvious when the disease causes mortality, but even subclinical infections have been shown to cause high losses from reduced feed efficiency, weight gains, milk production, reproductive performance, carcass quality and work output in draught animals and from condemnation of livers at slaughter [7, 8].

Active infections of *F. gigantica* in cattle are common in lower altitude settings but appear to diminish with increasing elevation [4]. In Ethiopia, *F. hepatica* and *F. gigantica* infections occur in areas above 1800 meters and below 1200 meters above sea level, respectively, which has been attributed to variations in the climatic and ecological conditions such as rainfall, altitude and temperature and livestock management system. In between these altitude limits, both species coexists where ecology is conducive for both snail hosts and mixed infections prevail [9].

Both *F. hepatica* (high land) and *F. gigantica* (low land) type of liver flukes cause severe losses in Ethiopia where suitable ecological conditions for the growth and multiplication of intermediate host snails are available [10]. The disease is found in vast water lodged and marshy grazing field condition anticipated to be ideal for the propagation and maintenance of high prevalence of fasciolosis. In Ethiopia, the highlands contain pockets of water logged marshy areas. These provide suitable habitats year round for the snail intermediate hosts [11]. Its prevalence has shown to range from 11.5% to 87% [1].

Fasciola hepatica was shown to be the most important fluke species in Ethiopian livestock with distribution over three quarter of the nation except in the arid northeast and east of the country. The distribution of *F. gigantica* was mainly localized in the western humid zone of the country that encompasses approximately one fourth of the nations [12]. The incidence of the disease in bovines has increased worldwide in recent years as a possible consequence of global climate changes [13].

Fasciolosis can occur as subclinical, acute, or chronic based on the number of metacercariae ingested. The acute forms of the disease are primarily due to mechanical damage caused by simultaneous migration of immature flukes in the hepatic parenchyma [14, 15]. Chronic fasciolosis develops when the adult parasites migrate to the bile ducts and cause cholangitis, biliary obstruction and fibrosis [2]. Fasciolosis is not lethal in cattle and bovines rarely acquire the acute form of the parasitosis [14].

The life cycle of these trematodes involves snail as an intermediate host [3]. The complex nature of the life cycle and epidemiology of snail-borne disease presents challenges for predictive mapping at the herd-level, as well as disease management and animal husbandry at the individual-level [4]. Diagnosis of fasciolosis is may be established based on prior knowledge of the epidemiology of the disease in a given environment; observation of clinical signs, information on grazing history, seasonal occurrence and standard examination of feces in the laboratory [16]. More rational prophylactic programs based on local epidemiological information are needed for sound fasciolosis control strategies in Ethiopia [9]. Therefore; this review is undertaken to review and compile available literature information on the characteristics of the parasite about its occurrence in man and animals, its method of transmission, diagnosis and control of the disease which will increase our understanding about the disease.

MATERIALS AND METHODS

Etiology: Fasciolosis is caused by parasitic flat worms of liver fluke which infest the liver of various animals specially sheep and cattle [17]. Infection of domestic ruminants with *F. hepatica* (temperate liver fluke) and *F. gigantica* (tropical liver fluke) are most common causes of fasciolosis [3, 18].

Taxonomic Classification: Taxonomic classification of *Fasciola* presented in the phylum platy helminthes, class trematoda, sub class digenea, super family fasciolidea, genus *Fasciola* and species *F. hepatica* and *F. gigantica* [19].

Morphology: *Fasciola* parasites are large hermaphrodites worm with leaf shaped body and spiny tegument [20]. Grossly, the young fluke at the time of entry in to the liver is 1.2 mm in length, undifferentiated between species and lancet like in appearance [17]. When it becomes fully mature in the bile ducts, it is leaf shape, grey brown in color and round. It is around 3.5 cm in length and one cm in width. The anterior end is conical and marked off by distinct shoulder from the body. Adult *F. hepatica* has flat leaf like body typical of flukes and measures 20-30 mm in length and 8-15 cm in diameter wide [21].

It has anterior elongation (a cephalic cone) on which oral and ventral suckers are approximately of equal size are located. The intestine of adult parasites highly branched with numerous diverticulae extending from anterior to posterior end of body. A pair of testes also highly branched and located in the posterior half of the body. The relative compact, ovary located just above the testes and linked to short convoluted uterus opening to genital pore above the ventral sucker. The vitellaria are highly diffuse and branched in the lateral and posterior region of the body. *F. gigantica* is a parasite very similar to *F. hepatica* and its length may vary from 25-75 mm long and 15 mm wide. In addition cephalic cone is proportionally shorter than that of *F. hepatica* and its body even more leaf like in shape [17, 19].

Microscopically the tegument has backwardly projecting spines. An oral and ventral sucker may be readily seen. The egg is oval, operculated, yellow and large (150 x 90 µm) and about twice the size of trichostrogyle eggs [21].

Epidemiology: Flukes found all over the world being totally depends on water as the medium for infection of both the intermediate and definitive hosts [22].

They are very discriminating in their choice of snail hosts [23]. *F. hepatica* is a temperate species above 1800 meters mean above sea level and found in all continents including highlands of Ethiopia and Kenya except Antarctica. *F. hepatica* is the major cause of liver fluke diseases in Ethiopia and its tropical counterpart, infects various animal species, but mostly herbivores. It affects ruminants much more than man [9].

This is one of the most economically important parasitic diseases of livestock, causing disease in cattle and other domestic animals in Latin America, Africa, Europe and China. It is most common in Bolivia, Ecuador, Egypt and Peru, but is also found in European countries, including France, UK, Spain and Portugal. The incidence has apparently increased over the last 20 years [24]. *F. gigantica* on the other hand is widely distributed in tropical countries of Africa and Asia. In Ethiopia, it is found at altitude below 1200-2560 m.a.s.l. Mixed infection by two species can be encountered at 1200-1800 m.a.s.l [9].

The disease is distributed in countries with high cattle and sheep production, human fasciolosis occurs, excepting Western Europe, in developing countries. It occurs only in areas where suitable conditions for intermediate hosts exist [25]. Liver fluke transmission is dependent on the presence of its snail intermediate host; therefore, the distribution of the parasite is limited to geographic areas where the appropriate snail species is present. The intermediate host is a fresh water snail of the genus *lymnaea*. *L. natalensis* (truly aquatic) and *L. truncatula* (amphibious) [17] is the most important intermediate host for *F. hepatica* in different parts of the world [22] and in Ethiopia [26].

It is an amphibious or mid dwelling snail which prefers moist temperature conditions (15-22°C) though it appears that variants found in the tropics have adaptation to higher temperature mostly in the lowland areas and can breed and survive at 26°C with sufficient moisture. The intermediate host of *F. gigantica* is *L. natalensis* and *L. acuminata*. *L. natalensis* is recognized intermediate hosts for *F. gigantica*. *L. acuminata* is a strictly aquatic snail often found in Africa [9].

Large numbers of metacercaria will usually be produced when there is optimal availability of suitable snail habitats, optimum temperatures and optimum moisture is present. This frequently results in seasonal patterns of emerging disease in certain parts of the world for example, in Britain metacercaria may appear on pastures from August to October and also in May to June. Suitable snail habitats will include all areas where

snails may survive in clear water or mud such as the edges of streams, ponds, rivers and vleis (permanent natural habitats); or temporary man-made depressions filled with water (tractor tracks etc.) [27].

A slightly acid environment may be more optimal. Temperature requirements are mean day/night temperatures of 10°C at which both the snails and the flukes will propagate. Below 5°C all activity will stop and above 15°C significant increase in both snails and fluke larval stages may be seen, with the optimum being 22 - 26°C. Moisture levels are described as optimal when rainfall exceeds transpiration and when field saturation is achieved [27].

Most cases of acute fascioliasis result from ingestion of metacercariae in late summer from eggs hatching in the same year. These eggs were shed during the winter and spring by adult worms inhabiting the bile ducts of chronically-infected untreated sheep and cattle that survived infection in the previous year. However, some metacercariae do survive on pasture over winter, particularly if the conditions are mild. In addition, immature fluke infections in snails cease development when the intermediate host enters hibernation deep in the mud at the onset of winter [28].

Life Cycle: Completion of the life cycle of the liver fluke *Fasciola hepatica* is closely dependent on climatic conditions. Factors such as the survival and development rate of the fluke eggs, availability and distribution of the snail intermediate host *Lymnaea truncatula*, rate of development of infection within the snails and survival on pasture of the metacercaria larvae are closely linked to the temperature and humidity of the environment. The timing and intensity of the metacercarial peak on pasture in late summer and autumn in any year are also closely related to the climatic conditions [28].

The life cycle of fasciolosis begins with release of unembryonated eggs into the biliary ducts which are then passed in the stool of herbivores (definitive hosts) or humans (incidental hosts). Eggs of *Fasciola* are passed in the feces or immature eggs are discharged in the biliary ducts and in the feces [17].

The eggs develop and hatched then finally, release motile ciliated miracidia; this takes 9-10 days at optimum temperatures, which invade a suitable snail intermediate host. Librated miracidium has short life span and must locate suitable snail within about three hours. If success penetration is occurred in infected snail, develop proceed through the sporocyst and radial stage to the final stage in the intermediate host [17, 29].

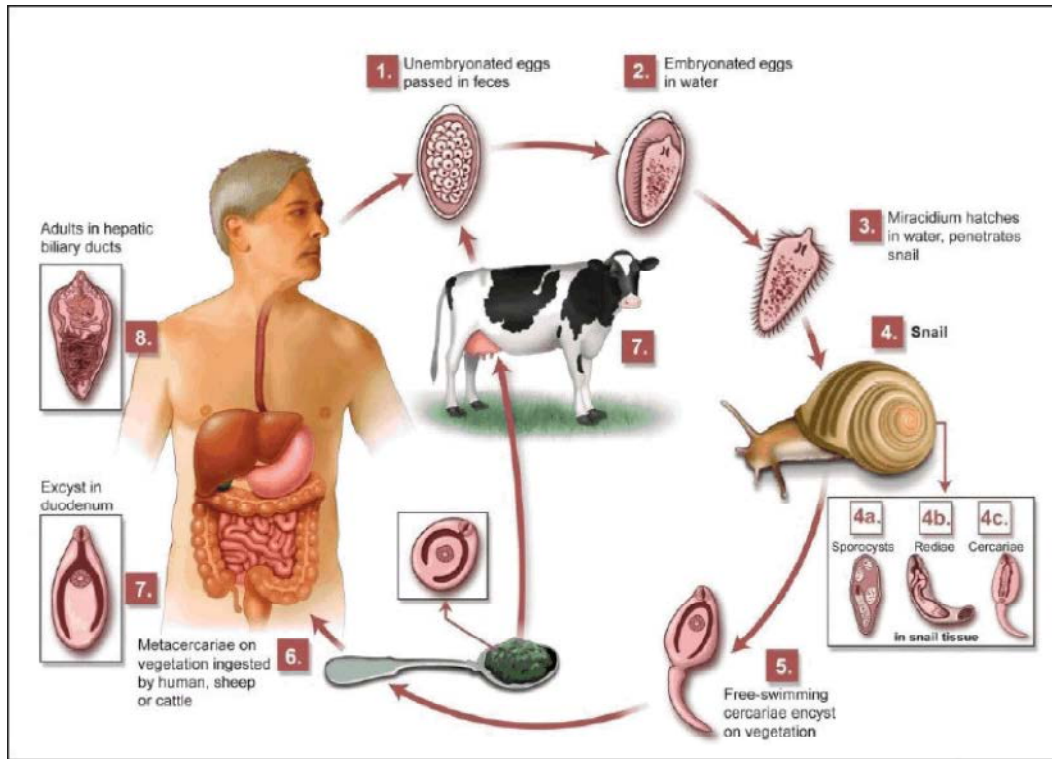


Fig. 1: Life cycle of *Fasciola* species

Source: CDC [31] Adapted and modified from: [http://www.dpd.cdc.gov/dpdx/fasciola_hepatica - cycle -jpg](http://www.dpd.cdc.gov/dpdx/fasciola_hepatica_cycle.jpg).

In the snail the parasites develop into cercariae, which are released from the snail and encyst as metacercariae on aquatic vegetation or other surfaces, which are infective stage of fluke. Mammals become infected by eating contaminated vegetation. After ingestion, the metacercariae excyst in the duodenum and migrate through the intestinal wall, the peritoneal cavity and the liver parenchyma into the biliary ducts, where they develop into adults. The adult flukes live in the large biliary ducts of the mammalian host [30].

When ambient temperature rise above 5-6°C (45-46°F), miracidium must find and invade snail within 24-30 hours. Young *Fasciola hepatica* migrates through hepatic parenchyma for about 4-5 weeks growing from 0.1-10 mm. The essential point of life cycle is that, trematode eggs can develop eventually in to hundreds of adults [21]). The life cycle of *F. gigantica* is similar to that of *F. hepatica*. The main difference is being in the time scale of cycle. Most parasitic phases are longer and pre-patent period is 13-16 weeks [19].

Pathogenesis: The development of infection in definitive host is divided into two phases: the parenchymal (migratory) phase and the biliary phase [29, 32]. The

parenchymal phase begins when encysted juvenile flukes penetrate the intestinal wall. After the penetration of the intestine, flukes migrate within the abdominal cavity and penetrate the liver or other organs.

F. hepatica has a strong predilection for the tissues of the liver [33]. Occasionally, ectopic locations of flukes such as the lungs, diaphragm, intestinal wall, kidneys and subcutaneous tissue can occur. During the migration of flukes, tissues are mechanically destroyed and inflammation appears around migratory tracts of flukes [34]. The second phase (the biliary phase) begins when parasites enter the biliary ducts of the liver. In biliary ducts, flukes mature, feed on blood and produce eggs. Hypertrophy of biliary ducts associated with obstruction of the lumen occurs as a result of tissue damage [35].

The fibrotic response of liver to fluke induced damage varies with host. The severe reaction in cattle, which induce calcification of the bile ducts, appear to hinder the establishments and feeding of challenge infections thereby re-enforcing immune response [2].

Clinical Signs: Clinical signs of fasciolosis are always closely associated with infectious dose (amount of ingested metacercariae). In sheep and

cattle are, as the most common definitive host, clinical presentation is divided into 2 types [32, 35].

Acute Fasciolosis: is associated with mostly immature flukes and usually seen in autumn and early winter, 2-6 weeks after ingestion of metacercariae in large numbers. Infectious dose is between 800-5000 ingested metacercariae. Immature flukes migrate through the liver parenchyma and create migratory tracts, which results from direct trauma, coagulative necrosis and release of toxic excretions from the flukes (e.g. Catepsins) [35].

Lesions may vary from mild (low infestations) to severe in heavy or repeat infestations. The liver may be enlarged and hemorrhagic with fibrous to fibrous exudates on the capsular surface (usually the ventral lobes). The migratory tracts may be visible as dark acute hemorrhagic streaks to, more yellowish white streaks typical of post necrotic scarring and granulation [29]. Sheep suddenly die without any previous clinical signs. Ascites, abdominal hemorrhage, icterus, pallor of membranes, weakness, lethargic, anemia may be observed in sheep [17, 21].

Chronic Fasciolosis: is associated with mature flukes and seen mainly in late winter/early spring. It is usually 4-5 months after ingestion of moderate numbers of metacercariae. Mature flukes, which are present in the bile ducts, cause necrosis and ulceration of the epithelium giving rise to peribiliary inflammation and severe hyperplasia of the epithelial layer [35].

Mechanical irritation by their spines and suckers, biliary retention and the production of toxic or irritant products by the flukes may contribute to lesions. Infectious dose is 200-800 ingested metacercariae. It is the most clinical syndrome in sheep and cattle. It occurs when the parasite reaches hepatic bile duct asymptomatic or gradual development of bottle jaw and ascites (ventral edema), emaciation, weight loss due to persistent diarrhea [17, 36].

In blood, anemia, hypo albuminemia and eosinophilia may be observed in all types of fasciolosis [35]. Elevation of liver enzyme activities, such as glutamate dehydrogenase (GLDH), gamma-glutamyltransferase (GGT) and lactate dehydrogenase (LDH), is detected in chronic fasciolosis from 12-15 week after ingestion of metacercariae [29]. Acquired resistance to *F. hepatica* infection is well known in adult cattle. Calves are susceptible to disease but in excess of 1000 metacercariae are usually required to cause clinical fasciolosis.

Importance of cattle fasciolosis consist in economic losses caused by condemnation of livers at slaughter and production losses especially due to reduced weight gain [38].

Diagnosis: A tentative diagnosis of fasciolosis may be established based on prior knowledge of the epidemiology of the disease in a given environment, observation of clinical signs, information on grazing history and seasonal occurrence. Confirmatory diagnosis, however, is based on demonstration of *Fasciola* eggs through standard examination of feces in the laboratory, postmortem examination of infected animals and demonstration of immature and mature flukes in the liver [16, 19, 21].

In animals, intra vital diagnosis is based predominantly on feces examinations and immunological methods. However, clinical signs, biochemical and hematological profile, season, climate conditions, epidemiology situation and examinations of snails must be considered [25]. Moreover, the fluke eggs are detectable in feces 8–12 weeks post-infection. In spite of that fact, fecal examination is still the only used diagnostic tool in some countries (like Ethiopia). While coprological diagnosis of fasciolosis is possible from 8-12 week post-infection (WPI), *F. hepatica* specific-antibodies is recognized using ELISA or Western blot since 2-4 week post-infection. Therefore, these methods provide early detection of the infection [39].

The simple sedimentation technique using tape water (cup sedimentation) has shown to be superior. Since eggs are not shed continuously, repeated examination are necessary and examination may fail despite the presence of flukes.

Treatment: Chemotherapy with drug remains the most cost effective way of treating parasitic disease and it is usually at the heart of any controlling campaign. Less frequent strategic treatment with a possible yearly rotation of an anthelmintic or anthelmintic combination that are effective against both immature and adult fluke have been reported to provide the best method of successful control of fasciolosis [41]. Drugs differ in their efficacy, mode of action, price and viability [13].

Triclabendazole (Fasinex) is the most widely used drug for the control of fasciolosis in ruminants. It is highly effective against immature (juvenile) and adult stages of *Fasciola* species and frequent treatments within the

pre-patent period can reduce the fluke infection to a negligible level [42, 43]. Triclabendazole (TCBZ) is used in control of fasciolosis of livestock in many countries. In animals, TCBZ resistance was first described in Australia, latter in Ireland and Scotland and more recently in the Netherlands [36]. Considering this fact, scientists have started to work on the development of new drug. Recently, a new fasciolicide was successfully tested in naturally and experimentally infected cattle in Mexico. This new drug is called 'Compound Alpha' and is chemically very similar to TCBZ [42, 44].

Albendazole is broad-spectrum drug especially for use against cestodes and flukes. Closentel will kill the majority of flukes greater than 4 weeks old. Clorsulon is applied in combination with ivermectin for combined flukes and round worm control both immature and adult [13, 16]. Nitroxylin and oxclosanide are less effective against immature flukes and should be used in the treatment of chronic fasciolosis (adult flukes). Treated cattle should be moved to clean pastures wherever possible [43].

Prevention and Control: Generally control of fasciolosis can be achieved by reducing the IH (snail), by chemical (Niclosamide and Copper sulphate) or biological, strategic application of Anthelmintics and reduction of the number of snail by drainage, fencing and other management practice and reduction in the risk of infection by planned grazing management [45].

Novel approaches for control include, vaccination [46], breeding *Fasciola* resistant animals or using competition in snail intermediate hosts between *Fasciola* and other trematode species [47]. Over time, cattle may develop a partially protective immune response to *F. hepatica*. The interaction of such factors as age of the host, innate resistance of the host, previous exposure of the host and present level of parasite exposure determine the degree of parasite establishment and the pathologic impact of the infection [48].

Cattle, unlike sheep, develop a degree of natural resistance to second and subsequent infections by incoming juvenile flukes. Worm survival is poorer in cattle as compared to sheep and the rate of maturation is slower. As a result of these differences, cattle rarely suffer from acute fasciolosis. However, chronic infections are common in over-wintered stock, necessitating one or more treatments during the housing period with flukicide active against the adult worms, in order to reduce the risk of pasture contamination after turn-out [28]. Treatment of animals to reduce the reservoir and reduce stock losses

has been used. In some areas of the world where fasciolosis is found (endemic), special control programs are in place or are planned [31].

In prophylactic uses of fluke Anthelmintics is aimed at; reducing pasture contamination by fluke egg at a time most suitable for their development; removing fluke population at a time of heavy burdens or at a period of nutritional and pregnancy stress to the animal [21].

Molluscides are usually impractical because of the large body of water involved and their possible effect on fish which may form an important part of local food supply [2]. The efficacy of molluscide is doubtful as snails have considerable capacity for multiplication and spread [16]. The amount of snail habitat present on individual farms related to pasture witness condition, such as premises with low-laying, heavy clay soils with a high water table, is also an important condition in evaluating the need for fluke control [28].

Public Health Significance of Bovine Fasciolosis:

Fasciolosis is an important disease caused by *F. hepatica* and *F. gigantica*. Recently fasciolosis is recognized as emerging human disease. WHO has reported that 2.4 million people were infected with *Fasciola* and further 180 million people are at risk of infection [49]. The records of natural infection in humans are mostly correlated with regions that are endemic for animal fasciolosis [50].

Most of the areas with a high endemicity of human fasciolosis involve *F. hepatica*. The disease, largely caused by *F. hepatica*, is reported to occur in France, England, Netherlands and Australia. Disease caused by *F. gigantica* occurs less commonly and has been described in the USSR, Indochina, West Africa and Hawaii. However, in Asia the distribution of *F. hepatica* and *F. gigantica* overlaps and this makes it difficult to identify the particular species involved, which is often referred to simply as *Fasciola* species. Water has been cited as the source of human infection, whether directly by drinking or indirectly by contaminating vegetables or kitchen utensils [49].

Man is usually infected accidentally by eating raw aquatic plants such as, watercress, with encysted metacercariae. These metacercariae can survive for long periods because the plants are usually submerged.

Many infected persons are asymptomatic during the migration of the larvae, though some experience fever and pain in the right upper quadrant of the abdomen with an associated eosinophilia and general malaise of varying degree, including myalgia and urticari [52]. The parasite causes inflammation of the mucosa of the caecum and

ascending colon with attendant symptoms of diarrhea. It causes ill health in a large number of individuals and deaths among untreated patients, especially children, have been attributed to this infection. Direct parasitological techniques, indirect immunological tests and other non-invasive diagnostic techniques are presently used for human fasciolosis diagnosis [49].

Many drugs have been used to treat fasciolosis in human patients. Dehydroemetine was considered the therapy of choice a few decades ago, but its toxicity allowed bithionol to become the drug of choice for years, despite its long treatment course [53]. The prevention of human fasciolosis may be achieved by strict control of watercress and other metacercariae-carrying aquatic plants for human consumption, especially in endemic zones. Among vegetables incriminated, freshwater plant species differ according to geographic zones and human dietary habits [49].

Economic Significance of Bovine Fasciolosis: When cattle become clinically ill as a result of a parasitic infection, the economic ramifications are clear. However, clinical disease is uncommon and cattle tend to harbor parasitic infections that are clinically unapparent. Although infected cattle may not appear ill, subclinical parasitic infections are recognized as causing economically important reductions in animal productivity. Economic losses from liver flukes may result directly from increased liver condemnations at slaughter and indirectly from decreased livestock productivity. Although direct losses are easier to measure, indirect losses are considered to be far more economically important [54].

Fasciolosis considered as an important factors for bovine and ovine production. In general infection of domestic ruminant with *F. hepatica* and *F. gigantica* causes significant economic loses estimated as over USD 200 million per annum to the agricultural sector worldwide, with over 600 million animal infected [55]. Beef producers are affected by increased culling of cows, reduced sale weights of culled cows, lowered reproductive performance in the brood cow herd, reduced calf weaning weights and reduced rates of growth in stockers. Economic losses in feedlots result from reduced feed-conversion ratios and lowered average daily gains and fluke-infected dairy cows produce less milk [1, 56].

Precise economic benefits of liver fluke control are difficult to quantify because of interactions among physiologic, nutritional, associated disease and climatic/geographic factors [57]. Interaction among these

factors causes tremendous variation in pasture infectivity and the physiologic consequences of infection from year to year and ranch to ranch [48].

Economic losses in Africa and other parts of the world is due to the condemnation of liver as unsuitable for human consumption and associated with poor carcass conformation and predispose the animal to other infectious principally clostridium disease, weight loss, loss in productivity and loss due to death [29]. More recently, Tolosa *et al.* [58] and Fufa *et al.* [59] have reported financial losses of 6300 USD and 4000 USD per annum, respectively due to liver condemnations at slaughter houses.

CONCLUSION

Fasciolosis is one of the major problems of livestock development, especially in cattle, by inflicting direct and indirect economic losses and its occurrence is closely linked to the presence of suitable habitat for the development of snail intermediate host. Hence, this disease deserves serious attention by various stakeholders in order to promote the livestock production. Strategic application of Anthelmintics to reduce pasture contamination with fluke eggs should be adapted. In addition, control of intermediate host (snails) through drainage of their habitats should be practiced. Moreover, keeping the animals off from snail intermediate host habitats or fencing these dangerous zones should also be included in the prevention and control strategy of the disease.

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