Bovine fasciolosis is an economically important parasitic disease of cattle caused by Fasciolidaetrematodes of the genus Fasciola

Marie Teshager

teshagermarie@gmail.com

ABSTRACT: Bovine fasciolosis is an economically important parasitic disease of cattle caused by Fasciolidaetrematodes of the genus Fasciola. The two most important species of this genus, *Faciola hepatica* and *Faciolagigantica*, are commonly known as liver flukes. The distribution of fasciolosis is worldwide, however, the distribution of *Faciola hepatica*, is limited to temperate areas and highlands of tropical and sub-tropical regions while *Fasciolagigantica*, which predominates in tropical area. The definitive hosts for Faciola hepatica are most mammals among which sheep and cattle are the most important once. The geographic distribution of trematode species is dependent on the distribution of suitable species of snails. Ethiopia has a high livestock population but productivity is low as a result of diseases, malnutrition and other management problems. Both *Faciola hepatica* and *Faciolagigantica* species of genus Faciola are found in Ethiopia and are transmitted by Lymnaea truncatula and Lymnae natalensis, respectively and various reports indicated that it is a serious problem of livestock production in Ethiopia causing considerable economic losses. Diagnosis based on clinical signs, seasonal occurrence and a previous history of fasciolosis. Fasciolosis causes a substantial economic loss which includes death, loss in carcass weight, reduction in milk yield, condemnation of affected liver, decline production and productive performances, exposure of animals to other diseases due to secondary complications.

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Introduction

Among many parasitic problems of farm animals, fasciolosis is a major disease, which imposes direct and indirect economic impact on livestock production, particularly of sheep and cattle (Menkir *et al.,* 2007). Fasciola hepatica and Fasciolagigantica are the two liver flukes commonly reported to cause fasciolosis in ruminants. The life cycle of these trematodes involves snail as an intermediate host (Walker *et al.,* 2008).

Bovine fasciolosis is an economically important parasitic disease of cattle caused by Fasciolidaetrematodes of the genus Fasciola. The two most important species of this genus, F.hepatica and F.gigantica, are commonly known as liver flukes. The distribution of fasciolosis is worldwide, however, the distribution of F. hepatica, is limited to temperate areas and highlands of tropical and sub-tropical regions (Soulsby, 1986) and Fasciolais commonly recognized as liver flukes and are responsible for wide spread morbidity and mortality in cattle characterized by weight loss, anemia and hypoproteinemia. The two most important species are Fasciola hepaticafound in temperate area and in cooler areas of high altitude in the tropics and subtropics and Fasciolagigantica, which predominates in tropical area (Troncy, 1989).

The definitive hosts for F.hepatica are most mammals among which sheep and cattle are the most important once. The geographic distribution of trematode species is dependent on the distribution of suitable species of snails. The genus Lymnaea in general and L.trancatula in particular is the most common intermediate hosts for F. hepatica. This species of snail was reported to have a worldwide distribution (Urquhart *el al.*, 1996).

Ethiopia has a high livestock population but productivity is low as a result of diseases, malnutrition and other management problems. Both F. hepatica and F. gigantica are found in Ethiopia and are transmitted by *Lymnaeatruncatula* and *L. natalensis,* respectively and various reports indicated that it is a serious problem of livestock production in Ethiopia causing considerable economic losses. Recently, small-scale traditional irrigation scheme is expanding in many parts of the country. Hence, it is anticipated that implementation of irrigated agriculture is creating favorable habitat for intermediate snail vector thereby influence the occurrence of fasciolosis (Michael *et al*., 2004).

Fasciolosis is a wide spread ruminant health problem and causes significant economic losses to the livestock industry in Ethiopia (Regassa *et al*.,2012). In Ethiopia, *F. hepatica* and *F.gigantica* infections occur in areas above 1800 m and below 1200 m 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 conductive for both snail hosts, and mixed infections prevail (Yilma and Malones, 1998). Fasciolosis caused by F. hepaticaand F. gigantica, is one of the most prevalent helminthes infections of ruminants in different parts of the world including Ethiopia (WHO, 1995; Okewole *et al*.,2000). Fasciolosis causes a substantial economic loss which includes death, loss in carcass weight, reduction in milk yield, condemnation of affected liver, decline production and productive performances, exposure of animals to other diseases due to secondary complications and cost of treatment expenses. 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 (Anne and Gary, 2006).

In Ethiopia, bovine faciolosis is caused by the species *F.hepatica* and *F.gigantica* has been reported. They also a significant economic loss due to liver condemnation which affected by faciola and moreover, the studies also showed that fasciolosis has higher economic significance on animal production and productivity. The economic losses due to fasciolosis throughout the world are enormous and these losses are associated with mortality, morbidity, reduced growth rate, condemnation of fluky, liver, increased susceptibility to secondary infections and expense due to control measures (Malone *et al* 1998). Therefore; the objective of this paper is collect a recent data and update information to review bovine faciolosis in Ethiopia, and its current status and economic significance.

2. BOVINE FACIOLOSIS IN ETHIOPIA

2.1 Etiology

Bovine faciolosis is caused by the two most important species of *F. hepatica* and *F. gigantca* which are commonly known as liver flukes. They are categorized in the phylum platyhelminthes, class trematoda, family faciolidae and genus fasciola. *F. hepatica* mostly found predominantly in temperate areas & highlands that are in the cooler areas of high altitude in tropics & subtropics while *F. gigantica* predominates in tropical areas(soulsby,1982).

2.1.1 Hosts, intermediate hosts and sites

Hosts for *F. hepatica* are most mammals and sheep & cattle are the most important. The intermediate hosts are snails of genus L ymnae. The most common *L. truncatula* is amphibious snail with a worldwide distribution and highlands of Africa. Site of adult liver flukes are bile ducts / gall bladder and immature flukes are Liver parenchyma. Aberrant flukes in unusual hosts like man, horse, (lungs, etc). Hosts for *F. gigantica* are sheep & cattle and IH are Lymnea, *L. natalensis* in Africa which is a primarily aquatic snail and are found in as treams irrigation cannels and marshy swamps. Site for adults are bile duct/gall bladder and immature flukes are liver parenchyma (Urquhart *et al*., 1996).

2.1.2 Morphology

Adult *F. hepatica* is a leaf-shaped & grey-brown in color About 2.5 - 3.5 cm in length & 1 cm in width. Anterior end is conical and markedly distinct & have broad shoulders. Edges converge caudally; hence has pointed posterior end and tegument covered with backwardly projecting spines and have obvious oral & ventral suckers present. Eggs are Oval, operculate, golden-yellow, and large, with zygote when passed in faeces; about twice the size of strongyle eggs. (Soulsby,1982). Morphology of larger liver fluke *F. gigntica* is 2.5 - 7.5 cm long and more leaf-like than *F. hepatica*. Apical cone is smaller than *F. hepatica* and has no shoulder; edges run collaterally so wider posterior end than *F. hepatica* (Soulsby, 1982).

2.3 Life cycle

Adult flukes are always oviparous and lay eggs with an operculum or lid at one polein the bile ducts and shed eggs into the bile, which enter the intestine. Eggs passed in the faeces of the mammalian host develop and hatch, releasing motile ciliated miracidia. This takes 9–10 days at optimal temperatures of 22–26°C and little development occurs below 10°C. The liberated miracidium has a short life-span and must locate a suitable snail within about 3 hours if successful penetration of the latter is to occur (Merck, 2005).

Under the stimulus of light, the miracidium releases an enzyme, which attacks the proteinaceous cement holding the operculum in place. The latter springs open like a hinged lid and the miracidium emerges within a few minutes. The miracidium, propelled through the water by its cilia, does not feed and must, for its further development, find a suitable snail within a few hours. It is believed to use chemotactic responses to ‘home’ in on the snail and, on contact, it adheres by suction to the snail and penetrates its soft tissues aided by a cytolytic enzyme. The entire process of penetration takes about30 minutes, after which the cilia are lost and the miracidiumdevelops into an elongated sac, the sporocyst, containing a number of germinal cells. These cells develop into rediae, which migrate to the hepato-pancreas of the snail; rediae are also larval forms possessing an oral sucker, some flame cells and a simple gut (Taylor *et al*.,2007)

From the germinal cells of the rediae arise the final stages, the cercariae, although if environmental conditions for the snail are unsuitable, a second or daughter generation of rediae is often produced instead. The cercariae, in essence young flukes with long tails, emerge actively from the snail, usually in considerable numbers. The actual stimulus for emergence depends on the species, but is most commonly a change in temperature or light intensity. Once a snail is infected, cercariae continue to be produced indefinitely, although the majority of infected snails die prematurely from gross destruction of the hepato-pancreas. Typically the cercariae swim for some time, utilizing even a film of water, and within an hour or so attach themselves to vegetation, shed their tails and encyst. This stage is called a metacercaria. Encysted metacercariae have great potential for survival extending to months (Urquhart *et al*.,1996).

In infected snails, development proceeds through the sporocyst and redial stages to the final stage in the intermediate host, the cercaria; these are shed from the snail as motile forms, which attach themselves to firm surfaces, such as grass blades, and encyst there to form the infective metacercariae. It takes a minimum of 6–7 weeks for completion of development from miracidium tometacercaria, although under unfavorable circumstances a period of several months is required. Infection of a snail with one miracidium can produce over 600 metacercariae. This is the essential point of the life cycle is that whereas one nematode egg can develop into only one adult, one trematode egg may eventually develop into hundreds of adults. This is due to the phenomenon of asexual multiplication, parthenogony, in the molluscan intermediate host, i.e. the production of new individuals by single larval forms (Taylor *et al*., 2007).

Metacercariae may remain viable many months unless they become desiccated. Where they migrate to the liver the time required for this transit can vary and results in delayed development rates, which affects the efficacy of some treatments because many are effective against flukes only later in their development. The young flukes penetrate the liver capsule and tunnel through the parenchyma for 6–8 week, growing and destroying tissue. They then enter small bile ducts and migrate to the larger ducts and, occasionally, the gall bladder, where they mature and begin to produce eggs Snails may extend the developmental period by hibernating during the winter (Merck, 2005).

Metacercariae ingested by the final host excyst in the small intestine. Once ingested, the outer cyst wall is removed mechanically during mastication. Rupture of the inner cyst occurs in the intestine and depends on a hatching mechanism, enzymatic in origin, triggered by a suitable oxidation-reduction potential and a carbon dioxide system provided by the intestinal environment. The emergent juvenile fluke then penetrates the intestine and migrates to the predilection site where it becomes adult after several weeks. Then migrate through the gut wall, cross the peritoneum and penetrate the liver capsule. The young flukes tunnel through the liver parenchyma for 6–8 weeks, and then enter the small bile ducts where they migrate to the larger ducts and occasionally the gallbladder and reach sexual maturity. The prepatent period is 10–12 weeks. The minimal period for the completion of one entire life cycle is ~17 wk. Adult flukes may live in the bile ducts of sheep for years; most are shed from cattle within 5–6 months that is the longevity of *F.hepatica* in untreated cattle usually less than one year. The life cycle of *F.gigantica* is similar to F.hepatica except the difference is in the time scale of cycle and hasa prepatent period 13-16 weeks(Soulsby,1982).

2.4 Epidemiology

2.4.1 occurrence, distribution and mode of transmission

In general the distribution of Fasciolosis is worldwide. However, the distribution of *F. hepatica* is limited to temperate areas and high lands of tropical and subtropical regions (Soulsby, 1986). The distribution of *F. hepatica*is mostly encountered in temperate areas, and in cooler areas of high altitude in the tropics and subtropics, while *F. gigantica* predominates in tropical areas. Snails are their intermediate hosts and amphibious snails of the genus Lymnaeaspecies are widely distributed throughout the world and *L. trunculata* is the most common of them all (soulsby, 1982 and Taylor *et al*.,2007). The presence of snail intermediate host and therefore the distribution of the parasite are limited to geographic areas where appropriate snail species is present and is dependent on season (Mihreteab *et al*., 2010).

Large numbers of metacercaria will usually be produced when there is optimal availability of suitable snail habitats, optimum temperatures and optimum moisture is present. For e.g.in Ethiopia the occurrence is closely associated to presence of suitable environmental conditions for the development of snails. 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 veils (permanent natural habitats); or temporary man-made depressions filled with water (tractor tracks etc.) (Biniam *et al*., 2012). 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. Transmission occurs through ingestionmetacercaeiae during grazing of contaminated marshy areas (Urquhrt *et al*., 1996)

2.4.2 Risk factors

According to Urquhart *et al* (1996)' and 'Taylor *et al* (2007)'there are three main factors affecting influencing the production of large number of metacercariae necessary for the out break of faciolosis:

1. Availability of suitable snail habitats:

L. truncatula prefers wetmud to free water, and permanent habitats include the banks of ditches or streams and the edges of small ponds. Following heavy rainfall or flooding, temporary habitats may be provided by hoof marks, wheel ruts or rain ponds. Fields with clumps of rushes are often suspect sites. Though a slightly acid pH environment is optimal for *L. truncatula*, excessively acid pH levels are detrimental, such as occur in peat bogs, and areas of sphagnum moss.

2. Temperature:

A mean day/night temperature of 10°C or above is necessary both for snails to breed and for the development of *F.hepatica* within the snail, and all activity ceases at 5°C. This is also the minimum range for the development and hatching of *F.hepatica* eggs. However, it is only when temperatures rise to 15°C and are maintained above that level, that a significant multiplication of snails and fluke larval stages ensues.

3. Moisture:

The ideal moisture conditions for snail breeding and the development of *F. hepatica* within snails are provided when rainfall exceeds transpiration, and field saturation is attained. Such conditions are also essential for the development of fluke eggs, for miracidia searching for snails and for the dispersal of cercariaebeing shed from the snails.

2.5 Pathogenesis

Pathogenesis is varies according to the number of metacercariae ingested, the phase of parasitic development in the liver and the species of host involved. Acute hepatic fasciolosis is caused by the passage of young F. hepatica through the liver parenchyma. Clinical signs occur 5-6 weeks after the ingestion of large numbers of metacercariae. By this time, the migrating flukes are large enough to do substantial mechanical damage to the liver. Acute hepatic insufficiency and hemorrhage result quiescent spores of Clostridium novyi may become activated by the anaerobic necrotic conditions created in the liver parenchyma by migrating F. hepatica, causing infectious necrotic hepatitis ('black disease') in sheep and cattle. This migration has also been thought to stimulate the development of occasional cases of bacillary hemoglobinuria in cattle (Radostits *et al*., 2007).

Chronic hepatic fasciolosis develops only after the adult flukes establish in the bile ducts. Here results in calcification of the bile ducts and enlargement of the gallbladder. The calcified bile ducts often protrude from the liver surface cause cholangitis, biliary obstruction, fibrosis, and a leakage of plasma protein across the epithelium. Although this protein can be re-absorbed in the intestine, there is poor utilization and retention of nitrogen leading to hypoalbuminemia. There is also a loss of whole blood due to the feeding activities of the flukes. This exacerbates the hypoalbuminemia and eventually gives rise to anemia. Chronic infection may limit growth rate and feed conversion in growing heifers and growth rate in beef cattle. *F.hepatica* infection has been reported to increase the susceptibility of cattle to Salmonella Dublin and predispose to prolonged infection and fecal excretion. Aberrant migration of the flukes is more common in cattle and encapsulated parasites are often seen in the lungs(Taylor *et al*., 2007).

2.6 Clinical signs

Acute fasciolosis not mostly common cattle however if the disease is observed clinically; it is manifested by; dullness, weakness, lack of appetite, pallor and edema of mucosa and conjunctivae, pain when pressure is exerted over the area of the liver. In heavy infections, where anemia and hypoalbuminaemia are severe, submandibular edema frequently occurs. With smaller fluke burdens, the clinical effect is minimal and the loss of productivity is difficult to differentiate from inadequate nutrition. It must be emphasized that diarrhoea is not a feature of bovine fasciolosis unless it is complicated by the presence of *Ostertagia* species (Radostits *et al*., 2007).The chronic form is most important in cattle and seen in late winter that is early spring; and occurs as a result of ingesting moderate numbers (200–500) of metacercariae over longer periods of time; signs include anemia, unthriftiness, submandibular edema, and reduced milk production, but even heavily infected cattle may show no clinical signs butclinically, these are difficult to detect since the fluke burdens are usually low and anemia is not apparent. The main effects are a reduction in milk yield and quality, particularly of the solids-non-fat component (Merck, 2005).

2.7 Diagnosis

This is based primarily on clinical signs, seasonal occurrence, prevailing weather patterns, and a previous history of fasciolosis on the farm or the identification of snail habitat. It can also diagnosed by examination of faeces for fluke eggs (note: eggs of *Fasciola* are browny yellow and eggs of Paramphistomidae are colorless) (Urquhart *et al.*, 1996).

2.8Treatment

Triclabendazole is a compound specifically for use against F. hepatica in sheep (10 mg/kg) and cattle (12 mg/kg). Higher doses are required for the control of F. gigantica in buffalo.26 It is highly effective against all stages of fluke from 2 days old in sheep and 2 weeks in cattle27 and is the drug of choice in outbreaks of acute fluke disease. Albendazole is a broad-spectrum compound also active against nematodes and cestodes. It is effective against adult F. hepatica at a dose-rate of 7.5 mg/kg in sheep and 10 mg/kg in cattle. It is ovicidal and will kill any *F. hepatica* eggs present in bile ducts or the alimentary tract at the time of treatment. Netobimin (20 mg/kg) is metabolized to albendazole in the body and has similar activity against F. hepatica (Radostitis *et al*., 2007).

At present there is only one drug, triclabendazole, which will remove the early immature (around 2 weeks of age in cattle) parenchyma stages. Apart from triclabendazole, the two drugs most commonly used for sub acute or chronic fasciolosis are nitroxynil and oxyclozanide also used to treat facilosis. Albendazole, ricobendazole and netobimin are also effective against adult fluke at an increased dosage rate. In lactating cows, where the milk is used for human consumption, the above drugs are either banned or have extended withdrawal periods. An exception isoxyclozanide, which is licensed for use in lactating animals in many countries and has a milk-withholding time of up to 3 days. Resistance to flukicides is not a problem in cattle (Taylor *et al*., 2007).

2.9 Control

Preventive measures are required in endemic areas as fasciolosis can cause death without warning or significant production losses. An integrated strategic approach is more cost beneficial than reliance on routine dosing and is less likely to induce anthelmintics resistance, but requires detailed knowledge of the local epidemiological cycle. Reduction of pasture contamination with metacercariae will reduce future risk. This can be done by preventing the snails from becoming infected with F. hepatica or by diminishing the size of the snail population. To achieve the first objective, adult flukes should be eliminated from the bile ducts of all grazing stock in spring and early summer. This prevents egg excretion and minimizes the numbers of snail-seeking miracidia at this crucial stage in the epidemiological cycle. There may however be wildlife sources of F. hepatica eggs which cannot be controlled in this way. Snail numbers can be reduced by restricting the size of their habitat. This can be done, where feasible, by draining boggy areas and by making sure that ditches, land drains; water troughs etc. are well maintained (Radiostis *et al*.,2007).

3. STATUS OF BOVINE FACIOLOSIS IN ETHIOPIA AND ITS ECONOMIC SIGNIFICANCE

3.1 Status of bovine faciolosis in Ethiopia

In Ethiopia, both *F. hepatica* and *F. gigantica* have been reported to exist in many parts of the country. The prevalence of bovine faciolosis based on copropsy result varies from 11.5% in buno province to 87% in Debre Berhan. Abattor studies have also reported up to 88.5% prevalence in Debre Berhan. Faciolosis in sheep and cattle in animal's resultsthat shows low productivity (low weight gain, low milk production etc.). Also in many countries including Ethiopia, livers from animals infected with *F .hepatica* and *F. gigantica* are condemned as unsuitable for human consumption (Zerihun,2006). The prevalence of bovine faciolosis in Ethiopia has been reported by different researchers at different areas. Since those areas are suitable to the snail intermediate host (Yosef *et al.*, 2014). The presence of fasciolosis due to *F. hepatica* and *F. gigantica* at abattoir surveys in some parts of the country has long been known and its prevalence and economic significance have been reported by several workers (Rahmatho *et al.*, 2010 ; Biniam *et al.*, 2012;Yosef *et al.*, 2014;Regassa*et al.*, 2012; Mihreteab *et al*, 2010; Alula *et al*., 2013; Ibrahim *et al*., 2009).

Table 1 the prevalence of bovine faciolosis in different areas of Ethiopia

Study area	Prevalence (%)	F. hepatica (%)	F. gigantica (%)	Mixed infection (%)	References
Hawassa Municipal abattoir, southern Ethiopia	28.6	58.9	10.6	14.7	(Rahmatho *et al., 2010)*
in and around Woreta, Northwestern Ethiopia	41.41	NR	NR	NR	(Biniam *et al*., 2012)
in and Around Bedelle District, Ethiopia	20.8	NR	NR	NR	(Yosef *et al*., 2014)
Bishooftu Municipal Abattoir, Central Ethiopia	21.6	18.3	6.4	3.1	(Regassa *et al*., 2012)
at Adwa Municipal Abattoir, North Ethiopia	32.3	13.9	7.7	6	(Mihreteab *et al*, 2010)
in Nekemte Municipal abattoir	21.9	14.1	5.2	2.6	(Alula *et al*., 2013)
At Kombolcha Industrial Abattoir, Ethiopia	39.6	24.3	7.14	5	(Ibrahim *et al*., 2009)

NR: not recorded and the percentage of F. hepatica F.gigantica are among positive animals

3.2 Economic significance

Bovine facilosis is an important economical disease in Ethiopia. In line to the economic importance of bovine fasciolosis in Ethiopia is associated with loss of productivity (indirect loss) and due to condemnation of infected liver (direct loss). This reported by different researchers in different places and at different time (Tolosa and Tigre, 2006; Shiferaw *et al*., 2011; Zeleke *et al*., 2014; Abunna *et.al.,2010; Yohannes et al.,2012*).

Table 2 direct and indirect economic loss of faciolosis

Study area	Direct loss (due to liver condemination)/annum	Indirect loss (due to loss of productivity)/ETB/ annum	Total/ETB	Reference
Jimma municipal abattoir	54,063.34 ETB	NR	NR	(Tolosa and Tigre, 2006)
In and Around Assela	37, 767.6 ETB	660, 933 ETB	698,700.6 ETB	(Shiferaw *et al*., 2011)
Mettu Municipal Abattoir	47,570.00 ETB	466,150.00 ETB	513,720ETB	(Zeleke *et al*., 2014)
Soddo municipal abattoir	4000 USD	NR	NR	(Abunna *et al., 2010)*
Mekelle municipal abattoir	2245 USD	NR	NR	*(Yohannes et al., 2012)*

NR, not recorded; USD, United States dollar; ETB, Ethiopian birr

4. CONCLUSION AND RECOMMENDATIONS

In general it could be concluded that Fasciolosis was one of major problem for livestock development in Ethiopia and closely linked to the presence of biotypes suitable to the development of snail intermediate host. Both *L. truncatula* and *L. natalensis* which are the intermediate hosts of *F.hepatica* and *F. gigantica* respectively which causes bovine faciolosis are found in Ethiopia. The species of *F. hepatica* is predominantly occurring than *F. gigantica* and sometimes mixed infection also occur. There is highly significant economic loss that accounts in thousands birr due to direct and indirect effect of faciolosis in Ethiopia. Therefore; based on the above conclusions the following recommendations are provided:

- ❖ Strategic anthelmintic treatment with appropriate fluckicide drug should be practiced
- ❖ A combination of control measures included drainage, fencing and snails had to be used to ensure a satisfactory and degree of control in the long run.
- ❖ Seasonal control applied.

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