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Review Article

Review on *Fasciolosis*, its Effect on Meat Quality/Hazards and Economical Importance

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ABSTRACT

Fasciolosis is a parasitic disease caused by commonly known species of liver fluke that are *Fasciola hepatica* and *Fasciola gigantica* that affects most population of ruminants and exists worldwide. The disease is characterized by both acute and chronic forms of liver lesion. Acute *Fasciolosis* is associated with migrating of immature flukes through the liver parenchyma and chronic *Fasciolosis* is accompanied by weight loss, decreased meat and milk production. The disease aggravation depends on distribution of intermediate hosts *Lymnaeidae* snail species in areas where cattle and sheep produced. Abattoirs are important in terms of assurance of the quality of meat and meat products for human con-sumption. More importantly, abattoirs aim at controlling animal and zoonotic diseases. The investigation of *Fasciolosis* at abattoirs has provided useful information regarding the prevalence of diseases and the economic losses caused due to liver condemnation worldwide. Presence of liver fluke appears associated with lower weight, carcass conformation and fatness score deteriorate when liver fluke is present. Carcasses of animals infected by *Fasciola* have poorer conformations, lower fat scores, and less weight gains than carcasses of non-infected cattle. According to international researchers report, *Fasciola* infect more than 300 million cattle and 250 million sheep worldwide and causes significant economic losses to global agriculture estimated at more than US $3 billion annually through production loss, such as a reduction of milk and meat yields. Productivity and economical losses due to *Fasciolosis* are incurred through a combination of factors, either through death of stock or reductions in meat and milk quality, growth rate and reproductive capacity, finally carcass quality and liver condemnations. This shows the significant impact of *Fasciolosis* on production and affects the economy. Therefore, it is desirable to strengthen control measures for the disease in order to minimize these losses.

Keywords: *Fasciolosis*; Meat quality; Edema; Economy

INTRODUCTION

Fasciolosis is caused by the hepatic trematode (*F. gigantica and F. hepatica*), a systemic parasitic disease of domestic ruminants, which is economically significant parasitic disease of most mammalian species. It is causing heavy loss in production and productivity, morbidity and mortality, growth retardation, sterility; poor feed utilization, poor quality of meat and milk, condemnation of affected livers and expense due to control measures [1]. *F. hepatica* infect more than 300 million cattle and 250 million sheep worldwide and, together with *F. gigantica* causes significant economic losses to global agriculture estimated at more than US$3 billion annually through production loss, such as a reduction of milk and meat yields [2,3]. The disease is characterized by both acute and chronic forms of infection. Acute *Fasciolosis* is related with juvenile flukes migrating through the liver parenchyma. In this, grossly the liver is enlarged and hemorrhagic with fibrinous to fibrous exudates on the capsular surface. Numerous hemorrhagic spots and focal necrosis are found on the cut surface of liver parenchyma. The migratory tracts from direct trauma of this parasite are grossly seen as dark acute hemorrhagic streaks or typical post necrotic scarring and granulation [4]. Histopathologically necrotic migratory tracts occurred by immature flukes migrating through

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the liver parenchyma is seen and tissue cytology, in correlation with histopathology is also used in diagnosis of liver pathology [5]. Chronic *Fasciolosis* is known by weight loss, weakening, pallor of mucous membranes, anemia, ventral edema, appetite changes, soft stool consistency or diarrhea, stomach hypotonia and decreased milk production [6].

Apart from its great veterinary importance throughout the world, *Fasciola* is emerging and secondary most spread zoonotic infection affecting numerous human populations in which they can being infected from accidental ingestion of parasite larvae passed into the environment with faeces from definitive hosts. The increasing importance of *Fasciolosis* is also related to its pathogenicity and immunity, immunological impact on children in long-term infection particularly in endemic areas and in every continent of the world. This disease is pronouncedly complicated, including difficulties in diagnosis, its great morbidity. The diagnosis of acute *Fasciolosis* is implemented by the determination of serum hepatic enzyme activities which are released from the damaged hepatic cells [7]. Clinical diagnosis and positive fecal egg count along with increasing level of Gamma-Glutamyl Transferase (GGT) confirm for the presence of chronic *Fasciolosis*. Confirmatory diagnosis for *Fasciolosis* is based on demonstration of characteristics *Fasciola* eggs through standard examination of feces in the laboratory and presence of immature and mature flukes in the liver through postmortem examination [8]. On post mortem *Fasciola* infected liver is an irregular outline, and pale and firm. Gross pathology of chronic *Fasciolosis* is characterized by reduced in size of the organ and thickened bile ducts [9]. Liver examination at slaughter house is considered to be the most direct, reliable and cost-effective technique for the diagnosis of liver fluke infection [10]. The lesions in the liver are caused due to the toxic and mechanical effects of liver flukes, the excretory products of the parasites, and the decomposed products of *Fasciola* spps in the bile and liver tissues, all of which affect the complex vascular and biliary system in the liver [11].

Several control methods against ruminant *Fasciolosis* are available and can either be used independently and or as a combination of two or more of them. These methods involve elimination of the intermediate host, control of the parasite itself, adoption of good grazing practices (avoiding marshy pastures), regular and rational use of anthelmintic or a combination of all these strategies. However, anthelmintic are the most preferred due to the private nature of such treatments [12]. Abattoirs are important in terms of assurance of the quality of meat and meat products for human consumption. The carcass of animal parasitized by liver fluke has lower weight and lower fat level. Condemnation of the liver of infected animals caused a significant financial loss as well as the loss protein food source. This loss, if not eliminated through effective control strategies, would have a major impact on the sustainability of the livestock industry. The aim of this review is to discuss the effects of *Fasciolosis* on meat quality/hazards and to highlight the economic impacts of *Fasciolosis*. Therefore, to assess the impact of *Fasciola* on the weights of quality carcasses, it is important to test the interaction between *Fasciola* with other associated factors to manage the economic loss due to liver condemnation and reduction of productivity.

LITERATURE REVIEW

Etiology and morphology of *fasciola*

Fasciolosis, which is also referred to as distomatosis or liver fluke disease, is a parasitic disease caused by the trematode of the genus *Fasciola*. The most important species are *F. hepatica* and *F. gigantica*, found in the temperate and tropical regions of the world respectively. Larger fluke, F. gigantica is restricted to warmer regions including parts of Africa and Asia. Both species overlap in many areas of Africa and Asia. Adults of each species can concurrently occur in the same animal host, either because of local overlap or because of livestock movement [13]. The two *Fasciola* species may hence interbreed resulting in hybrids species. They are hermaphrodites and are found in the bile ducts of a large number of herbivorous ruminants, equine, pigs, rabbits. The adult parasite has a flat leaf-like body, typical of flukes, and measures 20 to 30 mm long by 8 to 15 mm wide. They are dorsoventrally flattened, the tegument is covered with scaly spines, and they have two suckers (distome arrangement with the oral sucker and acetabulum close together). They have a bifurcate blind gut and each worm is hermaphroditic, possessing both male and female reproductive organs. The pair of testes, also highly branched, is located in the posterior half of the body. The relative compact ovary is located just above the testes and is linked to a short-convoluted uterus opening to a genital pore above the ventral sucker. The vitellaria are highly diffuse and branched in the lateral and posterior region of the body. It has an anterior elongation (a cephalic cone) on which the oral and ventral suckers, which are approximately of equal size. The intestine of the adult parasite is highly branched, with numerous diverticulae extending from the anterior to the posterior of the body.

Fasciola gigantica is the largest of the human liver and lung flukes. It is a parasite very similar to F. hepatica, with length that may vary 25 to 75 mm long by 15 mm wide. It has a shorter cephalic cone, a larger ventral sucker and a more anterior position of the testes in Figures 1-3. The egg of F. hepatica measures 150µm by 90µm in size and very similar in shape to that of F. gigantica. The egg of the latter is larger in size (200 µm x 100 µm). *Fasciola* eggs should be distinguished from the eggs of other flukes, especially from the large eggs of Paramphistome. *Fasciola* eggs has a yellowish-brown shell with an indistinct operculum and embryonic cells whereas Paramphistome egg has transparent shell, distinct operculum with embryonic clear cells, and possess a small knob at their posterior end .

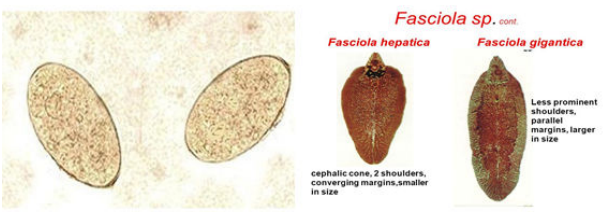

Figure 1: *Fasciola* Eggs (100X) and Morphology of *Fasciola* spp.

Intermediate hosts of *fasciola*

The intermediate hosts for *Fasciola* spp. are true water snails belonging to the phylum Mollusca, class Gastropoda and subclass Pulmonata. They belong to the family Lymnaedae and super species Lymnaea (Radix) auricularia sensu lato. At least 20 snail species have been identified as intermediate hosts for one or more *Fasciola* spp. Snails of the genus Lymnaea, the most common L. truncatula, are an amphibious snail with a wide distribution throughout the world and in Ethiopia [14]. Other important Lymnaea vectors of *Fasciola* outside Europe are L. tomentosa in Australia and New Zealand, L. cumella in North America, Australia and New Zealand, L. bulimoidis in Southern USA and the Caribbean, L. humlis in North America, L. viator in South America and L. diaphena in South America. On the other hand, snails of the genus Lymnaea, L. auricularia, other snails L. natalensis, L. rufescens and L. rubiginas are IH of F. gigantica. All are primarily aquatic and found in streams, irrigation channels and marshy swamps [15].

Life cycle of *fasciola*

Infestation with *Fasciolosis* is usually associated with grazing wet land and drinking from the snail infesting watering places [16]. The life cycle of *Fasciola* spp. is a typical of digenetic treamatodes. Adult flukes in the bile duct shed eggs into the bile, which enter to the intestine. Eggs reach the outside by passing down the common bile duct and passed in the feces of mammalian host. Hatching occurs in moist conditions only after the first larval stage, miracidium, has formed and when ambient temperature rises above 5-6 °C (41-43 °F). Miracidia must find and invade the tissue of suitable host snails within 24-30 hours. leave the snails as cercaria. These attach to herbage and transform in to metacercaria by secreting a tough protective cyst wall. After ingestion by the final hosts, each metacercaria releases an immature fluke which crosses the intestinal wall and migrate across the peritoneal cavity to the liver. The migration is sometimes misdirected and ectopic flukes can be found in lungs, particularly in cattle. The minimal period for the completion of an entire longevity of F. hepatica in untreated cattle is usually less than one year. Life cycle of F. hepatica is therefore 17-18 weeks. The prepatent period is 10-12weeks [17].

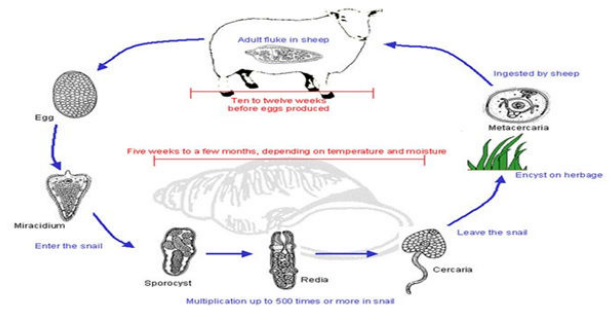

Figure 2: Life-cycle of the liver fluke, *Fasciola* hepatica source,Tagesu (2017).

Epidemiology of *fasciola*

The geographical distribution of F. hepatica and F. gigantic is determined mainly by the distribution patterns of the snails that have a role as intermediate hosts and depends on the grazing habitat preference of the animal. The most important intermediate hosts of *Fasciola* are Lymnaea truncatula and Lymnaea natalensis. In developed countries, the incidence of F. hepatica ranges up to 77% [18]. Temperature and rainfall affect both the spatial and temporal abundance of snail hosts and the rate of development of fluke eggs and larvae, the three most important factors that influence the occurrence of *Fasciolosis* are availability of suitable snail habitat, temperature and moisture. Metacercaria can survive up to 3 months after harvesting in hay from endemic highland areas that are consumed by the ruminants in arid and lowland areas, particularly during the dry season when suitable grazing pastures are scarce; local crowding of animals along the banks of streams and ponds during the dry season. When nutritional conditions are generally compromised also provides an important dynamic for infection transmission [19].

Pathogenesis of *fasciola*

Pathogenesis of *Fasciolosis* varies according to the parasitic development phases; parenchymal and biliary phases. The parenchymal phase occurs during migration of flukes through the liver Parenchyma and is associated with liver damage and hemorrhage. The biliary phase coincides with parasite residence in the bile ducts and results from the haematophagic activity of the adult flukes and from the damage to the bile duct mucosa by their circular spines. In the bile ducts of some permissive hosts, such as the sheep, rabbit, rat and mouse, the biliary stage of the disease is common. In others, such as cattle and humans, few flukes survive beyond the migratory phase and biliary disease is relatively rare. Hepatic pathology, even when only limited areas of the liver are damaged, results in significant disturbances in mitochondrial bioenergetics metabolism of carbohydrates, proteins, lipids and steroids, as well as bile flow and bile composition [20].

The pathology associated with diseases are caused by the inflammation of the bile ducts which causes thickening of the lining and eventually leads to fibrosis that results in reduced flow of the bile and back pressure builds leading to atrophy of the liver parenchyma and cirrhosis. The complexity arises from several sources. Maturation of flukes involves development and growth for over 12-16 weeks during which time the fluke travels between and within organs. Because an individual fluke may pass the same part of the liver twice (or more) during these peregrinations, fresh and resolving lesions caused by the sequential insults may be found in the same section of tissue; as the migratory fluke grows the size of its track through the liver increases as does the damage and the inflammatory response. Resistance develops with age so that adult cattle are quiet resistant to infection. Hypoalbuminemia and hyperglobulinemia commonly occur in liver fluke infections in all host species. During the parenchymal stage of the infection, liver damage caused by the migrating flukes compromise liver function, which in sheep and calves is reflected in a decline in plasma albumin

concentrations, attributed partly to reduced rate of synthesis and partly to an expansion of the plasma volume.

Figure 3: Chronic liver fluke infection in cattle, with bile duct distension (Gross) and bottle jaw.

Clinical signs of *fasciola*

In domestic ruminants, an adverse effect of acute or chronic fasciolosis includes decreased weight gain and milk production, decreased female fertility, work power and mortality. Acute fasciolosis due to the migration of juvenile flukes occurs as disease outbreak following a massive, but relatively short-term, intake of metacercariae. The high fluke intake is often the result of certain seasonal and climatic conditions combined with a lack of appropriate fluke control measures. It typically occurs when stocks are forced to graze in heavily contaminated wet areas because of overstocking and/or drought [21]. Animals suffering from acute fasciollosis especially sheep and goat, may display no clinical signs prior to death; while some may display abdominal pain and discomfort and may develop jaundice. In some cases, the liver capsule may rupture and fluid may lick into the peritoneal cavity causing death due to peritonitis. More commonly, on ingestion of fewer metacercaria, fever and eosinophilia is seen. Death usually results from blood loss due to hemorrhage and tissue destruction caused by the migratory juvenile flukes in the liver resulting in traumatic hepatitis. This is more commonly seen in sheep than in other hosts and in post mortem examination revealed inflammatory lesion and fibrosis due to fasciolosis [22]. Sub-acute *Fasciolosis* is caused by ingestion of a moderate number of metacercaria and is characterized by anemia, jaundice and ill thrift. The migrating fluke causes extensive tissue damage, hemorrhage and in particular liver damage resulting in severe anemia liver failure and death in 8-10 weeks [23].

The chronic form of the disease is the most common in cattle and it occurs when small numbers of flukes finally enter the bile duct and infection becomes patent. This results in a chronic wasting disease from slow acquisition of liver flukes for months or even years. Clinical signs of chronic *Fasciolosis* are variable, it occurs when the parasite reaches the hepatic bile duct, but often includes: weight loss, anaemia, bottle jaw, diarrhoea, constipation. Fluke infection may predispose to other conditions due to impaired liver function and can reduce milk yield and fertility. Affected animals are reluctant to travel.

Diagnosis of *fasciola*

The most common method of diagnosis is by faecal egg counts and pathological lesions in the liver during abattoir examination. It may consist of tentative and confirmatory procedures. A tentative diagnosis of *Fasciolosis* may be established based on prior knowledge of the epidemiology of the disease in a given environment; observations 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. The latter is helpful in deciding the intensity of infection. There are other laboratory tests (enzymatic and/or serological procedures used to qualify the infection mainly for research purposes. Serological diagnostic method often used to detect antibodies, such as indirect ELISA. Serological assays are detecting infections due to immature forms where fecal egg output is often nil. Such tests allow the detection of substance like cathepsin L. (cysteine proteases), excretory secretory products, detection of Ag in milk, and ELISA detection of antibodies against the fluke's plasma concentration of Gamma-Glutamyltransferase (GGT), which are increased with in the bile duct damage [24].

Treatment of *Fasciolosis*

Livestock are often treated with flukicides, chemicals toxic to flukes, including bromofenofos, triclabendazole and bithionol . For the treatment of acute *fasciolosis*, it is essential to choose a product which is highly effective against the juveniles that damage the liver parenchyma. For chronic disease a compound active against adult fluke is required. Triclabendazole (12 mg/kg) is considered as the most common drug due to its high efficacy against adult as well juvenile flukes. Clorsulon is supplied in combination with ivermectin for combined fluke and around warm control in cattle. Nitroxynil is given sub cutaneously at 10 mg/kg and has good efficacy against the adult fluke but the dose has to be increase by up to 50% to obtain adequate control of acute disease. Ivermectin, which is widely used for many helminthic parasites, has low effectivity against F. hepatica, as does praziquantel [25].

Control and prevention of *fasciola*

Several control methods against ruminant *Fasciolosis* are available and can either be used independently and as a combination of two or more of them. The control of F. hepatica in cattle in temperate regions where the disease is prevalent is often based on strategically timed flukicide treatment, which is determined by studying the seasonal transmission dynamics in numerous locations throughout the world. The tropical trematode infection caused by F. gigantica may be less amenable to this approach because the intermediate hosts L. (Radix) natalensis are true water snails and cattle can come into contact with infected snails while grazing around water bodies all year round. Reducing the number of intermediate snail hosts by chemical or biological means and drainage, strategic application of anthelmintics, fencing and other management practices and reduction in the risk of infection by planned grazing management are used to control fasciolosis. In most fasciolosis endemic areas, the control of the intermediate snail host population offers a good opportunity for the reduction of

transmission and is generally effective when combined with one or more other methods such as chemotherapy or environmental sanitation. Although eradication of the snail hosts is the most effective method of total fluke controls this, however, is often very difficult in low-lying, wet areas with a mild climate [26]. Strategic anthelmintic treatment based on epidemiological and meteorological data is important for the control of flukes [27]. Molluscicides use for the control of snail intermediate hosts is a potential tool for the control of fluke infections. Before considering chemical control of snails, the topography of habitant should be noted because; it is often very difficult to apply them effectively. They are also not species-specific, may destroy edible snails highly used as food in some communities and expensive. Effective control of most trematode infections is based on strategically applied chemotherapy. Combination of environmental manipulation, intermediate host control, sanitation and chemotherapy are believed to be efficient but very expensive. The action of drug against both immature and mature flukes, side effect and cost are considered to choose a flukicidal drugs. Chemotherapy with drugs remains the most cost-effective way of treating parasitic diseases, and is usually at the heart of anymajor control campaign [28].

Effects of *Fasciolosis* on meat quality/hazards

Abattoirs are important in terms of assurance of the quality of meat and meat products for human consumption. The pathogenic effects of the liver flukes on the host organism start with the ingestion of infective stages (encysted metacercaria) with vegetation or freshwater. After that migration of juvenile forms through the hepatic parenchyma, flukes reside and graze on the mucosa of the bile ducts, which result in the massive tissue damage [29]. In the *Fasciolosis* infected animals, the damage to the host species is either due to the mechanical or chemical effects by *Fasciola* spp. or by the hosts inflammatory and immune responses. The mechanical injury usually occurs when the infective metacercariae migrate through the liver capsule and hepatic tissue. This migration in turn is associated with trauma, hemorrhages and necrosis of the liver tissue, followed by subsequent granulation which ultimately leads to liver cirrhosis. The lesions in the liver are only partially a result of mechanical action of liver fluke, because the injury of the liver can be induced by parasites excretory products, decomposed products of parasites, bile and hepatic tissue. Pathological changes, caused by mechanical and toxic effects of F. hepatica, affect the complex vascular and biliary system in the liver [30]. The chemical effects involve a strong Th2 response, which in turn is associated with the production of cytokines thereby contributing to the overall pathophysiological condition. Bottle jaw syndrome could be as a result of massive *Fasciola* infection causing liver destruction leading to cessation of protein synthesis.

Hepatic pathology, even when only limited areas of the liver are damaged, results in significant disturbances in mitochondrial bioenergetics metabolism of carbohydrates, proteins, lipids and steroids, as well as bile flow and bile composition. The higher prevalence of *Fasciolosis* is found in older cattle, probably due to a longer exposure time [31], and it would be expected that F. hepatica infected animals taken later to the slaughter house, in which it is possible to under estimate the effects of *Fasciolosis* in older animal populations with a higher prevalence and an "ideal slaughter weight" [32]. The interaction of age and liver fluke infection in relation to the final weight of the meat is better evidenced in young animals (~23-37 months), possibly because they are more susceptible to the effects of parasitic infections than the older animals. The farmer and the market determine the weight at which an animal is arrive to the abattoir and farmers will delay sending animals infected with F. hepatica until they reach the desired weight. The lower weights of the carcasses of cattle with F. hepatica could be due to the alterations generated by this parasite in the hepatic parenchyma, lowering its capacity for synthesis and metabolism [33].

In addition to the differences observed in the estimated weights across the age range, there is also an association of F. hepatica with poor carcass conformations and lower fat scores. Meat is an important source of protein and valuable commodity in resource poor communities [34]. *Fasciolosis* produces biochemical changes in enzymatic activities and lower levels of total proteins, albumin, globulins, glucose, creatinine, urea, cholesterol, triglycerides, and lipoproteins, which may be responsible for the poor carcass conformations, low fat scores, and low body weights of infected animals [35]. *Fasciola* spps infection can causes the release of reactive oxygen species, which cause cell wall damage and hepatic tissue necrosis. These are influencing the biochemical parameters in the serum, and determination of specific liver enzymes functions as a useful tool for diagnosis of hepato-biliary diseases. Increased serum AST could be related to the degenerative changes and hepatocellular necrosis produced by migration of juvenile flukes through the liver parenchyma. Moreover, the cellular changes from parasitism increase the permeability of the hepatic cells and in turn result in the release of the enzymes into the serum. Elevation in serum GGT levels is an indicator of chronic changes, cholestasis and epithelial damage in bile ducts caused by presence of adult flukes in biliary tract. These are resulting GGT increase provide sensitive indications of liver injury and is the best marker, while AST is less sensitive. The degenerative changes found in hepatocyte and biliary cirrhosis in the liver histopathology of cows and cattle infected with *Fasciola*. This is producing hypoalbuminaemia, hyperglobulinaemia and Hypoproteinaemia, this also present in the fallow deer and sheep.

The hypoalbuminaemia is occurred due to decreasing albumin synthesis caused by liver damage. This produces cholangitis, anaemia, biliary obstruction, destruction and fibrosis of hepatic tissue. The hypoproteinaemia is occurred due to severe infection of the liver produced by destruction of liver parenchyma resulted in drastic alteration in protein values. The hyperglobulinaemia is happened due to increased production of α and ß globulin and as a result of immune response to infection. The consequences of liver damage resulting from the migrating flukes' compromises liver function which is reflected in changes of plasma proteins (albumin, globulins) concentration. The detected hypoglycemia at *Fasciola* infection is attributed to the disturbance of glucogeneogenesis results from hepatic disorders; increasing of the ketone bodies during gastroenteritis resulting reduced in voluntary feed intake and blood glucose depression

with depression of the hepatic glucogenic pathways in the infected animals. Liver parenchymal damages inflicted by huge worm load could possibly be the cause of hypoglycemia. Increasing of creatinine and serum urea levels are occurred due to *Fasciola* infection. Glomerulopathy is related with *Fasciolosis* and buffaloes are suitable as naturally existing experimental model of renal injury by circulating immune complexes [36].

Fasciola spp., which is considered as one of the extrinsic stressors, may affect the normal physiological equilibrium and thus the stress response. These factors are all significant, affecting the conversion of muscle into meat. Furthermore, the reasons for the variance in both the biochemical parameters and the eating quality traits being attributable to intra-animal variability under stress conditions are not entirely clear. Depending on the source of the stress, animals may experience fear, dehydration, and hunger; increased fatigue; and physical injury, which further contribute to potential changes in their energy homeostasis, intracellular ion dynamics, protease system, and proteins in skeletal muscle and also stress has effects on the behavioral and physiological status and ultimate meat quality [37, 38]. Thereafter, the animal's physiological and metabolic functions, which dominate the postmortem biochemical changes, are affected. As a result, the intensity of the stressor and the susceptibility of the animal to the stressors are of major significance in animal welfare and meat quality development. The carcass of animal parasitized by liver fluke has lower weight and lower fat level. Nitric oxide level is significantly elevated in the cattle infected with *Fasciola* spp. As compared with that in the uninfected animals. The high nitric oxide levels in the liver of rats experimentally infected with F. hepatica, which causes cytotoxicity to cells due to the ability of NO to generate peroxynitrite, with subsequent initiation of various oxidative reactions, including modification of nucleic acids, lipids, and proteins, which causes tissue injuries [39]. The oxidative changes of muscle protein cause considerable physical and chemical changes in the conformation, bioavailability, solubility, aggregation, and capability to undergo proteolysis. These relationships can affect the basic quality traits, nutritional values, and functionalities of carcass. The imbalance between pro-oxidants and endogenous antioxidant prompt the elevated production of free radicals, such as reactive nitrogen species. Over flow of free radicals elicits oxidative stress, resulting in dangerous effects on cellular biomacromolecules such as DNA, protein, and lipids. Free radicals accumulated in response to oxidative stress impede the mitochondrial integrity and cell membrane through lipid peroxidation, which substantially increase the risk of oxidative reactions during the postmortem aging of meat product. Moreover, the meat becomes susceptible to oxidative process due to the high levels of unsaturated fatty acids and various indicators such as transition metals, pigments, and certain oxidoreductase enzymes. Thus, oxidation has been described as a major cause affecting muscle protein function and the nutritional, sensory, and shelf-life quality of animal products. Lipid peroxidation is a free radical-mediated Chain of reaction. ROS,such as hydroxyl radical (-OH), hydrogen peroxide (H2O), superoxide anion (O2-), and hydroperoxyl radical (H-), are the most prominent initiators of these chain reactions. Protein oxidation involves direct reaction with an ROS or an oblique reaction with secondary products of lipid peroxidation, leading to the covalent modification of a protein [40].

Economic loss due to *Fasciolosis*

Fasciolosis causes high economical loss in sheep, goat, buffaloes and cattle, but are of particular importance in sheep and cattle to livestock producers (FAO, 1994). Worldwide productivity losses due to *Fasciolosis* were, F. Hepatica infects more than 300 million cattle and 250 million sheep worldwide and, together with F. gigantica, causes significant economic losses to global agriculture estimated at more than US$3 billion annually through production loss, such as a reduction of milk and meat yields. Economic losses from *Fasciolosis* may result directly from increased liver condemnation or indirectly from decreased livestock productivity. Although direct losses are easier to measure, indirect losses are considered to be far more economically important. Other than the herd-level losses, *Fasciolosis* also results in losses associated with liver condemnation in slaughtered animals and in addition to liver condemnation, the disease causes economic losses due to reduced weight gains, poor feed utilization, poor quality carcass, unthriftiness, reduction in working power, carcase conformation and fatness score deterioration, condemnation in large number of infected livers, increased susceptibility to secondary infection and expense due to control measure, reduction in growth rate and reduced productivity[41-45]. The lower weights of the carcasses of cattle with F. hepatica could be due to the alterations generated by this parasite in the hepatic parenchyma, reducing its capacity for synthesis and metabolism.

The damage due to the migration of the immature flukes and presence of the adults in the bile ducts affects cattle metabolism, leading to depressed haematocrit, hypoalbuminaemia and eventually emaciation [46]. We could speculate that the catabolic processes associated with the parasitism could interfere with adequate muscle and fat development, hindering carcass conformation and the deposition of fat. Carcass conformation and fatness grades influence the price paid by the abattoir to the beef producers as reported by [47], who investigated the fluke serology levels on meat juice and by using abattoir records [48]. The progressive weight loss (the amount of weight loss may also, however, be dependent on age, level of nutrition and intensity of infection) and lead to serious economic losses and pale mucous membranes in infected cattle may be attributed to anemia caused by *Fasciolosis*. Direct economic loss is result from condemnation of liver affected by *Fasciolosis*. The annual losses from liver condemnation are assessed by considering the overall annually slaughtered animals in the abattoir. The annual losses are calculated by using the following formula according to [49]

Direct losses due to liver condemnation, ALc=ASw X LC X P,

Where: ALc: Annual Loss from liver condemnation. ASw: mean annual Animal slaughtered at abattoir. LC: mean cost of one liver in abattoir, P: prevalence rate of the disease at the abattoir.

Indirect losses due to carcass weight loss ACW: CSR x CL x BC x P;

Where ACW=annual loss from carcass weight,

CSR=average number of cattle slaughtered per year in the study area

CL=carcass weight loss in individual cattle due to *Fasciolosis*

BC=average market price of beef in the study areas

P=prevalence rate of *Fasciolosis* in the study area

The total economic loss due to *Fasciolosis* in the study area is estimated from the summation of ALC+ACW

In Ethiopia; *Fasciolosis* is considered to be of a great economic importance by both species of *Fasciola* specially in cattle. Major economic losses due to *Fasciolosis* have been described when animals grazing in irrigated system. Areas with seasonally flooded pastures, grazing areas of lake shores, slowly flowing water ways and banks of rivers are favourable environment for breeding of vectors of *Fasciola*. The economic significant of *Fasciolosis* in the highlands of Ethiopia has been reported by several workers. An estimate of the economic loss due to ovine *Fasciolosis* in the Ethiopian high lands was made based on available data on mortality weight loss, reduced reproductive efficiency and liver condemnation at slaughter. The economic effects of *Fasciolosis* were identified and models for estimating the financial loss presented. For example, the annual loss due to endoparasitism including *Fasciolosis* in Ethiopia was estimated at 700 million. Decreased productivity alone due to ovine *Fasciolosis* was estimated at 200 million birrs by the year 2010. Ovine *Fasciolosis* in Ethiopia is very frequent and causes a significant economic loss either in loss or decrease productivity and loss of body condition [50]. Ovine *Fasciolosis* losses were estimated at 48.8million Ethiopian Birr per year of which 46.5%, 48.8% and 4.7% were due to mortality, productivity (weight loss and reproductive wastage) and liver condemnation, respectively in Table 1 [51-64].

Annual loss due to fasciolosis	Study area	References
4,674.2 USD	(Adwa)	44
6300 USD	(Jimma)	53
4000 USD	(Wolyta soddo)	54
(52,981ETB)	-	55
59,387ETB (2969USD)	-	3
63072 ETB ($ 1,182,600	Nekemte	56
1,505, 856 ETB ($ 43, 024.458)	Wolaita sodo	63
154,188 ETB ($ 4,405.37),	Wolaita sodo	57
215,000 ETB ($ 6,142.85),	Dire dawa	34
154,490ETB ($ 4414)	Jimma	58
4000 USD	Soddo	50
6300 USD	Jimma	59
(1833 USD).	Kombolcha	60
1, 692.00 ETB	Menz lalo midir,	-
27, 572.64 USD	Adwa	64
(4,674.2 USD	-	61
1,026,951.12USD	Mekelle	-
(8312.5 USD)	Hawassa	-
268,536.21 USD	Tigray	62

Table 1: Some information on annual economic loss due to *Fasciolosis* in Ethiopia.

DISCUSSION AND CONCLUSIONS

Fasciolosis is a common trematode parasitic disease of domestic ruminants which is caused by *Fasciola* spp. with important implications for animal health and welfare and farming economics. It is causing a great reduction of revenue through lowering in productivity of animal in terms of less growth rate, meat and milk production, fertility, feed efficiency and draught power. The disease aggravation depends on distribution of intermediate hosts Lymnaeidae snail species in areas where cattle and sheep produced. *Fasciolosis* is the main cause of organ and liver condemnation during post mortem inspection at slaughter house. As meat is the main source of protein for human it should be clean and free from diseases of particular importance to the public like *Fasciolosis*. Although data are somehow limited, F. hepatica is one of the extrinsic oxidative stress factors that affect slaughtered animals, causing biochemical and metabolic changes in the early postmortem period. These changes have serious effects on meat quality aspects. Carcasses of bovines infected by *Fasciola* have poor quality like; less conformations and bioavailability poorer fat scores, and less weight gains than meat of non-infected cattle. Condemnation of the liver of infected cattle caused a significant financial loss as well as the loss protein food source this shows the significant impact of *Fasciolosis* on production and affects the economy. Based on these conclusive remarks the following recommendations are forwarded:

- Drainage of swampy area and regular deworming of animals before and after the rainy season are important recommendations in the reduction of the intermediate host.
- To assess the impact of *Fasciola* on the weights of quality carcasses, it is important to test the interaction between *Fasciola* with all factors to manage the economic loss due to liver condemnation and reduction of productivity.

- Animal should be regularly treated with the appropriate anthelmintics and awareness should be created on the prevention and control methods of *Fasciolosis*.
- Government bodies should create effective policies in collaboration with veterinarians to control andprevent the disease
- Future research should be needed to unravel the intricate mechanisms involved in the effects of F*asciolosis* on the changes that occur upon slaughter.

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CONFLICT OF INTEREST

The author declares there is no conflict of interest

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