Liver fluke may infect most mammals, including man. In South Africa the more common fluke is *Fasciola gigantica* with *Fasciola hepatica* occurring in a few localities only. The intermediate hosts are the semi-aquatic snails *Lymnaea natalensis* for *F. gigantica*, *L. truncatula* for *F. hepatica* and *L. columella* for both. (Figure 1) *Lymnaea natalensis* prefers warmer climates than the other two snails, and fasciolosis is not seen in semi-arid or arid parts of the country. The parasites occur in the bile ducts and the life cycle is indirect. The minimum period of development from miracidium to cercaria is 17 days and cercaria can be shed after 36 days (25 days for *F. hepatica*). The entire developmental period spans 13 - 16 weeks (8 – 12 weeks for *F. hepatica*).
Snail shells. Left, *Lymnaea columella*, middle, *Lymnaea truncatula* and right, *Lymnaea columella*. Note the opening of the body is on the right-hand side, when the tip of the shell points away.
The golden eggs of *Fasciola* get their colour from bile pigments (top left). A miracidium (top right) will enter a snail and from large redia (bottom left) that will form much smaller cercaria that have single tails (bottom left). The cercaria escape from the snail and encyst on solid objects to form the metacercaria (bottom right).

The pathogenesis in cattle and sheep is the same, but sheep react severely to the parasites. The young fluke grows fast and increases 8-fold in size in 15 days as it bores through the parenchyma of the liver. In massive infections the host usually dies of internal haemorrhage, while milder infections cause hepatic fibrosis and hyperplastic cholangitis.

During the initial 6-7 weeks of their migration, the flukes cause a mild anaemia due to the blood that seeps through into the migration tracts. The loss of plasma protein caused by the flukes is less important than the anaemia. When the worms arrive in the bile ducts, anaemia develops rapidly, as the worms are avid blood suckers.

The three factors that are basically responsible for the anaemia are an increased rate of destruction, a compensatory increase in haemopoiesis and continued draining of iron reserves, very similar to what happens in haemonchosis.

With peracute infections, animals die without showing any clinical signs. This is the result of massive internal haemorrhage that occurs with these infections.
Acute infections result in the death of animals while the flukes are still migrating in the parenchyma (after 6-7 weeks). If the animal can be examined, there is evidence of severe macrocytic anaemia and high AST levels. Fluke eggs are not present in the faeces.

Internal haemorrhage as a result of peracute fasciolosis is shown top left, and the effect on the liver in the top right illustration. Note the tiny flukes on the black rectangle (top right), which are responsible for the lesions. Fibrosis of the liver (bottom left) and thickened bile ducts (bottom right) are characteristic of chronic fasciolosis.

Subacute cases show a rapid loss of body mass, severe anaemia and high fluke egg counts. Death occurs from 12-20 weeks after infection.

Gradual wasting, severe anaemia with ascites, oedema, bottle jaw and high fluke egg counts are seen with chronic infections. Death may occur more than 20 weeks after infection.
Severe anaemia as indicated by the metaplasia of yellow bone marrow to red (left) and distinct submandibular oedema (right)

Usually there is no obvious reaction to the passage of the young flukes through the intestinal wall and on the peritoneum, except for small haemorrhagic foci where the flukes have temporarily attached. Parasites may sometimes be found in ascitic fluid. In heavy and repeated infections, peritonitis, either exudative (acute) or proliferative (chronic), occurs.

In acute cases of penetration of the liver, there are fibrino-haemorrhagic deposits on the serous surfaces and in chronic cases there are fibrous tags and adhesions (Figure). In the liver parenchyma itself there are numerous haemorrhagic migration tracts and in heavy infections the liver has a streaked appearance. Tracts from which the debris has been cleared appear yellowish as a result of eosinophil infiltration (Figure). Some flukes do not reach the bile ducts and form cysts in the parenchyma. These cysts consist of a connective tissue capsule and a dirty brown content of blood, debris and excrement from the fluke. Aberrant migration is uncommon, but flukes have been found in the lungs.

Mature flukes are present in the bile ducts where they obstruct the flow of bile, cause mechanical irritation, and produce toxins. Changes in the bile ducts are more severe on the left side. The bile ducts stand out as firm, whitish, branching cords. When these ducts are incised, flukes, detritus and clumps of eggs in a brown fluid may pour out.
Fibrous tags on the liver surface (top left), migration tracts filled with eosinophils (top right), aberrant migration to the lung, where the parasite caused localized haemorrhage (bottom left) and flukes, and detritus expelled from a bile duct (bottom right)

The lesions are initially limited to the larger ducts, but with time and repeated infections the smaller portal units are also affected. This is due to direct irritation by the flukes, superimposed infections (bacterial) and biliary stasis. The lesions heal by fibrosis and large areas of the parenchyma may become completely fibrotic. With repeated infections, the left lobe of the liver may become atrophied, indurated and have an irregular surface. With light infections the lesions are milder and are sometimes not detectable.

Cattle are generally resistant and adult worms live only for 9-12 months before being expelled. Few worms survive for 3-5 years. The extensive fibrosis and pronounced tissue reaction apparently are responsible for the resistance. Treatment with anthelmintic that will kill both young and adult flukes prevents clinical signs and such cattle develop a strong resistance. If they graze on contaminated pastures, their resistance becomes even stronger.

Adult sheep appear to be more resistant to the infection but not to the effects of the flukes, while young animals are fully susceptible.
Selected helminthoses in domestic ruminants: Infections of the liver

Clinical signs, seasonal occurrence, prevailing weather patterns and history of fasciolosis on the farm form part of establishing a diagnosis. The autopsy is pathognomonic. Faecal egg counts are significant as the size, shape and coloration of the eggs are diagnostic. The number of eggs per gram of faeces can give an indication of the size of the infection, i.e. the intensity.

Liver function tests, particularly the GLDH (glutamate dehydrogenase) and GT (gamma glutamyl transpeptidase) can give an indication of liver damage, which may be due to fasciolosis. Antibodies of the flukes may be detected with the ELISA (enzyme-linked immunosorbent assay) performed on milk or serum, and the passive haemagglutination tests.

**Strobilate (adult) cestodes**

*Stilesia hepatica*

As the name implies, the worms occur in the bile ducts of a variety of ruminants. Two round paruterine organs form 2 distinct white lines that run parallel down the length of the gravid proglottides.

The life cycle is unknown. The intermediate hosts are reputedly soil mites of the family Oribatidae, which need rich, moist soil, and in which the infective stage, the cysticercoid, occurs. They cause neither clinical signs nor significant liver pathology, although massive infections may cause mild cholangitis. The worms are widespread throughout the subcontinent and are usually only encountered at autopsy or at the abattoir. Large numbers of livers are condemned because of the presence of this worm and represents considerable economic loss. Control by disturbing the habitat of the soil mites by ploughing, especially near kraals or places where dung accumulates.
The larval cestodes or metacestodes

*Taenia hydatigena*

The final host is a canid, domestic dogs and jackal included. The immatures (metacestodes) are occasionally encountered in cattle. A large infection causes a condition known as *hepatitis cysticercosa*, which is characterized by migration tracts through the parenchyma of the liver. When fully grown, the metacestodes exit the liver in the region of the gall bladder and each forms the characteristic *Cysticercus tenuicollis* on the serosa. A differential diagnosis for *hepatitis cysticercosa* is fasciolosis and little can be done to treat the condition in the bovine. However, regularly deworming of all domestic dogs, properly cooking all meat (especially offal) before it is fed to dogs and preventing cattle to consume the tapeworm eggs in the faeces, do a lot to control the occurrence.
Selected helminthoses in domestic ruminants: Infections of the liver

Echinococcus granulosus

Cystic echinococcosis or hydatidosis is fairly common in sheep and goats, and occasionally encountered in cattle. Once again, domestic dogs and jackal are the final hosts and ruminants are infected by eating or drinking food or water contaminated by faeces of these carnivores. The target organ for the metacestodes is the liver and the lungs, is commonly known as a hydatid cyst. Surgery is impractical, and as a rule animals are not treated. It is a zoonosis that receives world-wide attention.

Sheep’s liver with numerous hydatid cysts (left) and a cyst in the heart of a bovine, opened to show the hydatid ‘sand’ or protoscoleces (right)