Liver Fluke – the parasite and disease.

Liver fluke disease (fasciolosis) is caused by the trematode, or flatworm, parasite, *Fasciola hepatica*. Disease can result from the migration of large numbers of immature flukes through the liver, from the presence of adult flukes in the bile ducts, or both. Liver fluke can infect all grazing animals (and can infect humans) but they mainly affect sheep and cattle, 'and are typically more pathogenic (i.e. damaging) in sheep.

Figure 17. Liver fluke
(a) Adult liver fluke parasite, *Fasciola hepatica* (scale bar = 0.5cm) © Sinclair Stammers; (b) Mud snail intermediate host, *Galba truncatula* (circled, scale bar = 0.5cm) © Grace Cuthill, Moredun Research Institute

Life-cycle

Compared to other helminths, the life-cycle is complex, involving an intermediate mud snail host, *Galba* (formerly, *Lymnaea) truncatula*, and several free-living stages. The role of the snail, which prefers muddy conditions, particularly areas associated with poor drainage, means that the incidence of liver fluke is greater in wetter areas of the country and in years when there is high summer rainfall. With the capacity of the snail to multiply rapidly (100,000 offspring in 3–4 months), coupled with the high fecundity of the liver fluke and its ability to multiply within the snail, there is potential for very high levels of contamination of pastures and heavy fluke burdens in animals.

Adult fluke in the host animal lay eggs that are passed out onto pasture in the faeces. When the temperature exceeds 10ºC, a ciliated larval stage known as a miracidium develops within the egg, hatches and uses thin films of moisture to migrate, actively seeking the mud snail intermediate host. Miracidia can only survive for a few hours outside the snail. Within the snail they undergo two further developmental stages, including multiplication (x1000s), eventually becoming infective cercariae, which emerge from the snail when the temperature and moisture levels are suitable.

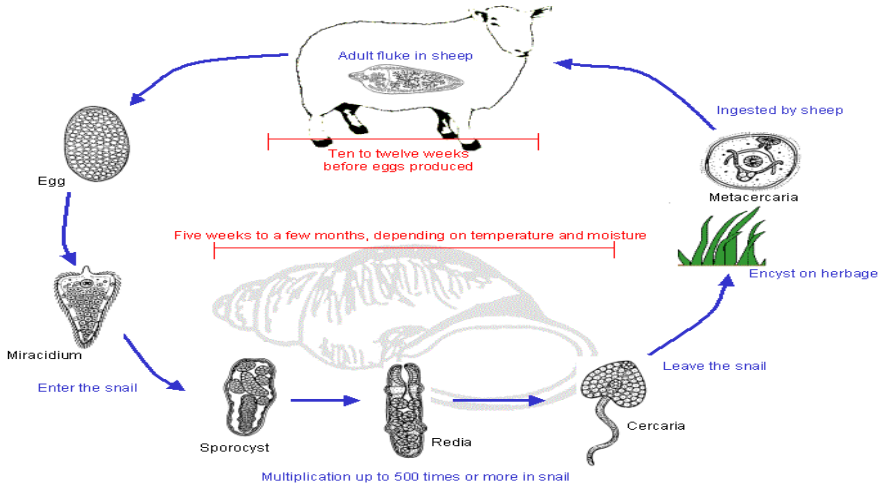


Figure 18. Life-cycle of the liver fluke, Fasciola hepatica
(Drawings courtesy of Drs Oldham, Jacobs and Fox)

The cercariae migrate onto wet herbage, encysting as metacercariae (cysts), the highly resilient infective stage of the liver fluke, where they wait to be eaten. Once ingested, the immature flukes migrate through the intestinal wall to the liver, causing considerable tissue damage as they tunnel. The infection is patent (i.e. egg-laying) 10–12 weeks after the metacercariae are ingested. The whole cycle typically takes 18-20 weeks.

Epidemiology

The hatching of fluke eggs and the multiplication of snails depend on adequate moisture and temperatures greater than 10ºC. These conditions usually occur from May–October in the UK, although patterns have been changing in recent years. The incidence of liver fluke disease is highest in years when rainfall is above average during May–July. The epidemiology of liver fluke is often viewed as the result of two distinct cycles of snail infection and pasture contamination.

❖ *Summer infection of snails*

In wet summers, snail populations multiply rapidly and snails are infected by hatching miracidia from May–July. If wet weather continues, the snails shed massive numbers of cercariae onto pasture from July–October. Conversely, if the climate in May–July is dry or cold, fewer snails appear, fewer fluke eggs hatch and levels of contamination in the autumn are much lower. Clinical fasciolosis, resulting from summer infection of snails, arises usually from ingestion of large numbers of metacercariae over a short period of time in July–October. Summer infection of snails is still the predominant epidemiological pattern of fluke infection seen in the UK.

❖ *Winter infection of snails*

Less commonly, snails can become infected in late summer or early autumn and development is delayed as the snails become dormant and hibernate. The cercariae are then not shed onto the pasture until the following spring. This can produce an unexpected but significant infection in herds or flocks in the spring. There is a liver fluke forecast for the UK, based on the prevailing climatic conditions in different parts of the country, and how these are likely to impact on the snail host and subsequent fluke infection risk on pasture (see NADIS for up-to-date information). Information on local and regional weather patterns, which underpin the NADIS Fluke Forecast. Further information about current GB fluke prevalence is also available at the Animal and Plant Health Agency Disease Surveillance Dashboard.

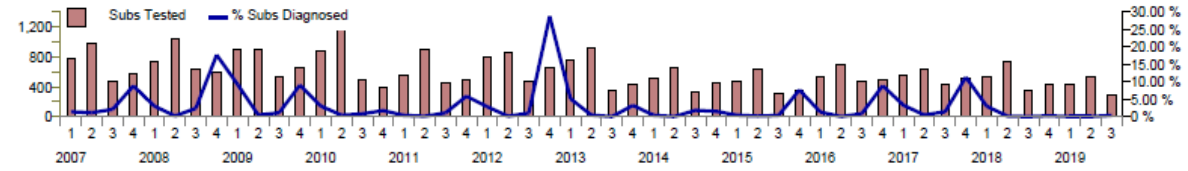
Fasciolosis

Liver fluke disease in sheep occurs in three main clinical forms – acute, subacute and chronic fasciolosis (figure 19). The form that occurs depends on the numbers of infective metacercariae ingested and the period of time over which they are ingested. Recently, milder winters and wetter summers have seen changing patterns in parasite epidemiology and acute disease has been reported earlier in the season (figure 20). Table 21 outlines the clinical signs and treatment options for each form of the disease.

Table 21. Diagnosis and treatment of fasciolosis in sheep

Disease type	Typical Peak incidence	Clinical signs	Fluke numbers	FEC (epg)	Recommended Treatment Choice
Acute	July to December	**Sudden death** or dullness, anaemia, dyspnoea, ascites and abdominal pain.	1000+ mainly immature	0	Triclabendazole. Treat all sheep and move to a lower risk (drier) pasture if possible OR re-treat after 3 weeks. Further deaths may occur post-treatment from liver damage incurred.
Subacute	October to January	**Rapid weight loss, anaemia,** submandibular oedema and ascites in some cases.	500-1000 adults and immatures .	<100	Treat with a fasciolicide active against mature and immature fluke. If sheep cannot be moved to lower risk pasture, re-treat after 5-8 weeks.
Chronic	January to April	**Progressive weight loss, anaemia,** submandibular oedema, diarrhoea and ascites.	200+ adults	100+	All fasciolicides are active against the mature fluke involved in chronic disease. Treat and move to lower risk pasture.

(a)



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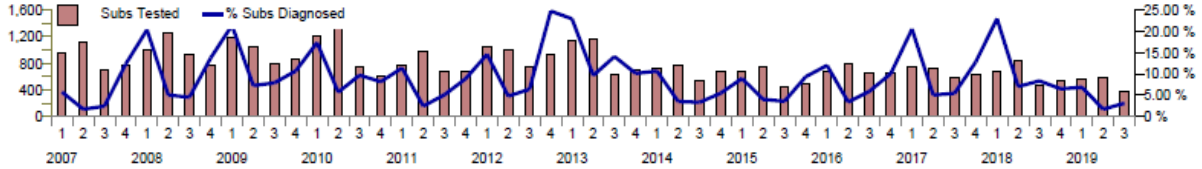

Figure 20. Seasonal patterns of liver fluke disease since 2007
Showing spikes in (a) acute and (b) chronic fasciolosis in sheep, following the wet summer of 2012 and mild winter of 2013. Bars represent the number of samples tested (post-mortem & FEC), lines represent the % of submissions diagnosed as liver fluke. Information kindly supplied by Sian Mitchell, Animal & Plant Health Agency.

(a)

(b)

(c)

Figure 19. Clinical signs of liver fluke disease in sheep. (a) acute fluke; (b) chronic fluke; (c) submandibular oedema or 'bottlejaw'. Images © APHA & Philip Skuce, Moredun Research Institute

Production losses

Liver fluke disease, whether the acute or subacute form of disease, can have a serious financial impact on a sheep farm, with immediate production losses of up to 10 per cent caused by weight loss or reduced weight gain (NADIS). Chronic disease can halve profits by reducing the lamb crop, through reduced ewe fertility and increased ewe mortality. Losses are estimated at £3 to £5 per infected sheep (AHDB). Fluke is also the second highest cause of liver condemnation at the abattoir, with current annual condemnation rates running at approximately 10%.