

Treatment and Control of Liver Fluke (*Fasciola hepatica*) in Sheep and Cattle.

Key Recommendations

1. Investigate any unexplained losses or disease that could be due to liver fluke including:
 - Sudden deaths.
 - Ill thrift.
 - Increased barren rates.
 - Liver condemnations.
 - Unexplained metabolic disease in dairy cows.
2. Include liver fluke treatment and control as part of the flock/herd health plan.
3. Assess the impact of liver fluke and the effectiveness of treatment and control measures using:
 - Faecal samples.
 - Post-mortem examinations.
 - Blood samples.
 - Fluke forecasts.
 - Abattoir feedback.



The number of disease outbreaks due to the liver fluke *Fasciola hepatica* has increased in recent years with unprecedented numbers of sheep affected in 2012/13 (Figure 2) The disease poses a threat to animal welfare and may also cause economic loss through mortality, reduced production, liver condemnation, predisposition to other diseases and the cost of treatment and control.

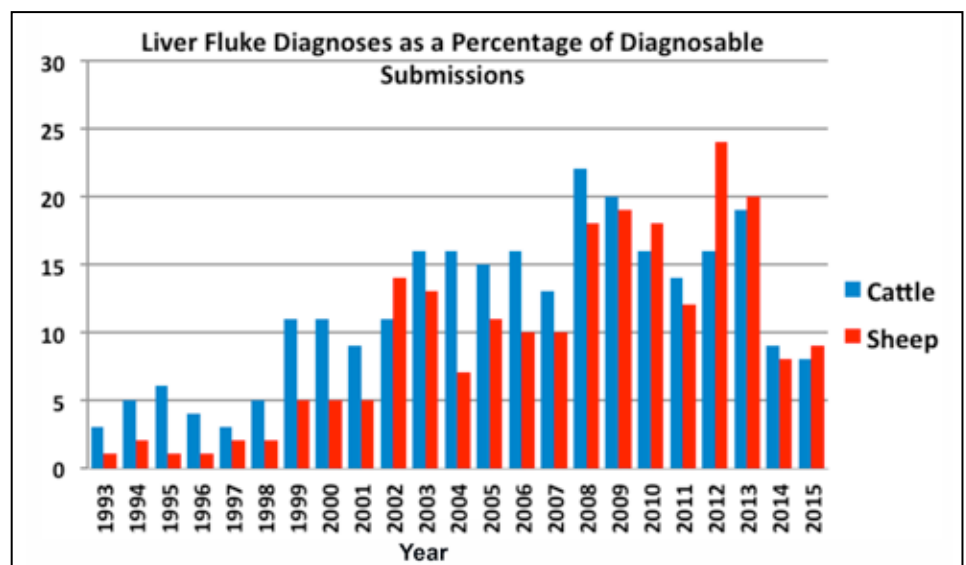


Figure 2: Outbreaks of fasciolosis as a percentage of diagnosable submissions

Life Cycle

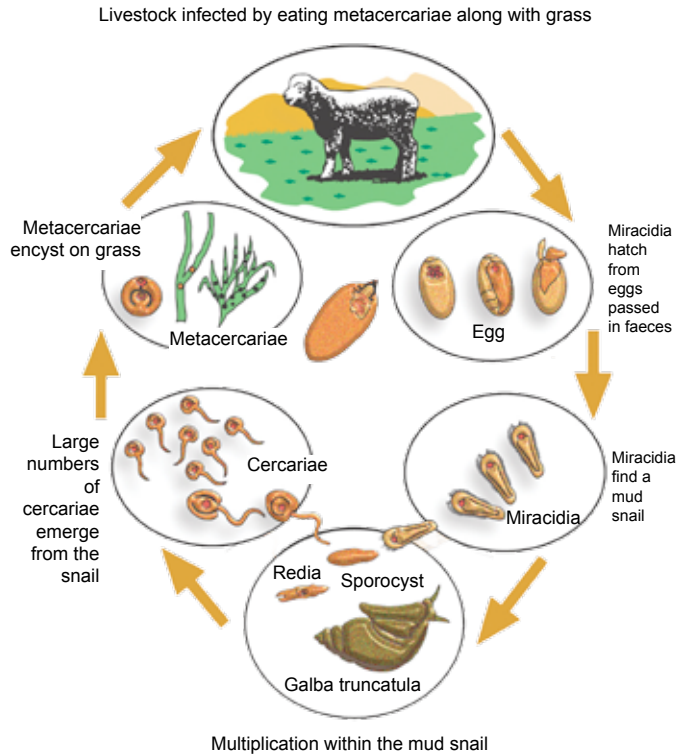


Figure 3: Life cycle of *Fasciola hepatica*.

Liver fluke eggs passed in the faeces of a mammalian host (e.g. cattle, sheep, deer, rabbits) develop and hatch into motile ciliated miracidia. This takes nine days at the optimal temperature of 22 to 26°C. Development at lower temperatures takes longer and does not occur below 10°C. The liberated miracidia have a short lifespan and must come into contact with their intermediate host, the mud snail *Galba truncatula*, within three hours if successful penetration is to occur. Multiplication occurs within infected snails and development proceeds through sporocyst and redial stages to cercariae which are the final snail stage. These are shed from the snail as motile forms and attach themselves to firm surfaces such as grass blades where they encyst to form the infective metacercariae. It takes a minimum of six to seven weeks to complete development from miracidia to metacercariae and under unfavourable conditions a period of several months is required. Cattle and sheep, (or other mammalian final hosts), ingest metacercariae which excyst in the small intestine, migrate through the gut wall, cross the peritoneum and penetrate the liver capsule. The young flukes tunnel through the liver parenchyma for six to eight weeks and then enter the small bile ducts where they mature to adults in around four weeks. During this period they migrate to the larger bile ducts and the gall bladder. The average time from ingestion of metacercariae to the presence of fluke eggs in the faeces is 10 to 12 weeks making the minimum period for completion of one entire life cycle of *Fasciola hepatica* 17 to 19 weeks.

Epidemiology

The risk of disease due to liver fluke is closely linked to summer rainfall which provides optimal conditions for *Galba truncatula*. The extent of the habitat of *G. truncatula* relates to climatic conditions and soil hydrology. Ideal conditions for survival and multiplication of snails include a slightly acidic environment and slow moving water to carry away waste products. Permanent habitats therefore include the banks of ditches or streams and the edges of small ponds. Following heavy rainfall hoof marks,

wheel ruts (Figure 4) or flooded areas may provide temporary habitats. Fields with clumps of rushes are common snail sites as these have a slightly acidic pH (Figure 5). The wet conditions required for snail breeding are achieved when rainfall exceeds transpiration. Such conditions also facilitate the development and hatching of *F. hepatica* eggs, the search for snails by miracidia and the dispersal of cercariae after shedding from snails. A mean day/night temperature of 10°C or above is necessary for snail breeding, the development of *F. hepatica* within the snail and the development and hatching of fluke eggs. As the mean temperature increases during late spring and early summer the development time for the stages of liver fluke outwith the final host (the suprapopulation) becomes shorter reaching a minimum of five weeks in mid-summer. The minimum temperature requirements for the development of a suprapopulation of *F. hepatica* in the UK have historically prevailed from April to October with relatively minor variations. As a result the main factor influencing the magnitude of snail populations is summer rainfall. These climatic factors dictate that the majority of snails become infected in the summer by miracidia developed from eggs deposited in the spring and early summer. This results in increased pasture levels of metacercariae from mid August



Figure 4: *Galba truncatula* snail in a wheel rut.



Figure 5: Field likely to contain suitable snail habitats

onwards and clinical disease in sheep from September onwards. The higher prevalence of fasciolosis in recent years has been largely associated with milder weather and summers with above

average rainfall. The development of flukicide resistance in some populations of *F. hepatica* will also play a role. Increased rainfall raises the water table thereby permitting *G. truncatula* to extend its habitat while milder temperatures prolong the length of the development period for the suprapopulation. In Scotland this has been reflected in the spread of the disease from poorly drained pastures in the west to traditionally drier farms in the east which had previously been free of liver fluke (Figure 6).

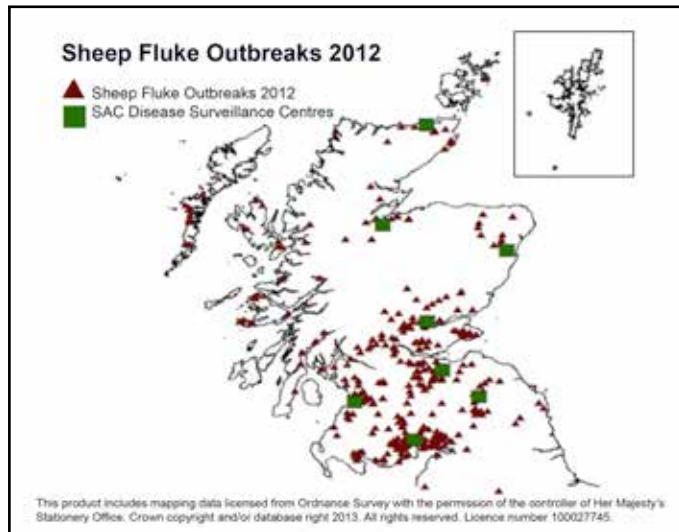


Figure 6: Locations of ovine fasciolosis outbreaks in 2012

Diagnosis

Clinical Signs

Liver fluke disease (fasciolosis) is usually classified as acute, subacute or chronic depending on the number and ages of flukes present in the liver. This classification is arbitrary and there is considerable overlap between these categories. It should always be remembered that fasciolosis is a group problem even though only a few individuals may be showing typical clinical signs.

Acute fasciolosis: Outbreaks are usually seen in late autumn/early winter and are associated with large numbers of immature flukes in the liver parenchyma. Following a wet summer the risk period may extend into the late winter and early spring. Clinical signs are most common in sheep and are due to the ingestion of large numbers of metacercariae from heavily infected pasture over a short period of time. The immature flukes tunnel through the liver parenchyma causing acute haemorrhagic anaemia and hypoalbuminaemia. Sudden deaths, often associated with liver haemorrhage (Figure 7), occur in animals where sufficient numbers of immature flukes are present. Other animals in the group may be weak, pale and in some cases exhibit abdominal pain with a palpably enlarged liver.

Subacute fasciolosis: This is usually seen from late autumn to spring and also presents as an acute haemorrhagic anaemia with eosinophilia and hypoalbuminaemia. This form of the disease occurs when large numbers of metacercariae have been ingested over a longer period of time, or the number ingested at any one time has not been sufficient to cause acute fasciolosis. It is not so rapidly fatal and animals may show clinical signs for one or two weeks prior to death. Affected sheep lose condition rapidly, become markedly anaemic with obvious pallor (Figure 8) and may have an enlarged liver and resent abdominal palpation.

Submandibular oedema (bottle jaw) may also develop in some cases (Figure 9). Large numbers of immature flukes will be present in the liver parenchyma but are not as numerous as in cases of acute fasciolosis. In addition a proportion of the fluke



Figure 7: Fatal liver haemorrhage in a case of acute fasciolosis.



Figure 8: White conjunctiva due to fluke induced anaemia.

population will have developed further and be found as adults in the bile ducts.

Chronic fasciolosis: This is due to the blood feeding activities of adult flukes in the bile ducts (Figure 1) and is most frequently seen in the winter and spring. It is characterised by ill thrift, anaemia, eosinophilia and hypoalbuminaemia. Submandibular oedema develops in severe cases and emaciation and death will follow if the animal remains untreated.



Figure 9: Submandibular oedema secondary to hypoalbuminaemia.

Subclinical fasciolosis: Smaller burdens of liver fluke may be responsible for subclinical disease resulting in lowered productivity reflected in inadequate food conversion rates, reduced lambing percentages and poor milk yields. It may precipitate metabolic disease in peri-parturient dairy cows.

Post-mortem Examination

Post-mortem examination of a fresh carcase is the quickest and most reliable method of diagnosis if fasciolosis is suspected to be the cause of death. The size of recovered flukes can give an indication of when infection occurred. Necropsy can also identify other important diseases such as pneumonia or parasitic gastroenteritis. If fasciolosis is not the cause of death inspection of the liver will give an indication of the level of fluke challenge. Outbreaks of fasciolosis in sheep and cattle may be exacerbated by sudden deaths due to Black disease. This is caused by multiplication of the bacterium *Clostridium novyi*, previously dormant in the liver, in response to the anaerobic conditions produced by migrating flukes. Rapid death is due to toxæmia, and may occur despite limited fluke damage in the liver.

Laboratory Testing

In the live animal, laboratory diagnosis of fasciolosis is based on a variety of tests carried out on faecal, blood or milk samples (Table 1).

Fluke Egg Count: This is a long established method of detecting the presence of adult flukes but is not suitable for the diagnosis of acute disease caused by immature flukes. Six to ten faecal samples should be submitted to the testing laboratory and can be pooled to reduce cost.

Coproantigen ELISA: This test detects the excretory/secretory (ES) antigens of *F. hepatica* in faeces and a positive result indicates active infection with late immature or adult liver flukes. It can diagnose infection two to three weeks before fluke eggs can be detected in the faeces. Where fluke burdens are low there may be no temporal advantage over a fluke egg count.

Antibody ELISA: This test detects antibodies to the ES antigens of *F. hepatica* in serum or milk and can give a positive result from two to four weeks post infection. A positive result does **not** confirm active infection as antibodies can be detected for up to ten months after fluke death e.g. following treatment. The test is useful in animals in their first grazing season as they can be used as sentinels for infection. It can also be used to screen bulk tank milk and gives an estimate of the percentage of seropositive cows in the milking herd.

Despite the production of antibodies there is no evidence that sheep or cattle ever become immune to *F. hepatica* and untreated sheep can remain persistently infected for life. Bovine livers contain more fibrous tissue which limits the damage caused by immature flukes. In addition fibrosis of the bile ducts, in response to infection, makes it more difficult for flukes to feed. Both factors can limit the size of fluke burdens in cattle and reduce the lifespan of individual flukes.

Liver Enzymes: Elevation of glutamate dehydrogenase (GLDH) levels can be useful for the diagnosis of acute fasciolosis as early as two to three weeks post infection. Raised gamma glutamyl transferase (GGT) levels can be detected once flukes enter the bile ducts six to eight weeks after infection.

Serum Proteins: Infection with significant numbers of liver flukes will cause hypoalbuminaemia possibly with concurrent hyperglobulinaemia.

Haematology: EDTA blood samples are rarely collected but can be used to demonstrate anaemia via the measurement of packed cell volume (PCV). An eosinophilia may also be detectable.

Note that changes in biochemical and haematological parameters are not specific to infection with liver flukes. Clinical signs together with the results of other tests e.g. detection of liver fluke eggs, should always be taken into account when establishing a diagnosis.

Faecal PCRs capable of diagnosing active infection at the two week stage have been developed but are not commercially available. There is ongoing research into the development and feasibility of pen side tests.

Table 1: Summary of laboratory tests used to assist in the diagnosis of fasciolosis.

Test	Type of sample	Reference range	Disease stage detected
Fluke egg count (FEC)	Faeces	N/A	Adult
Coproantigen ELISA	Faeces	N/A	Adult and late immature
Aspartate aminotransferase* (AST)	Clotted blood	<150 iu/litre	From 2-3 weeks post infection
Glutamate dehydrogenase (GLDH)	Clotted blood	<25 iu/litre	From 2-3 weeks post infection
Gamma glutamyl transferase (GGT)	Clotted blood	<50 iu/litre	From 6-8 weeks post infection
Albumen	Clotted blood	30-40g/litre	Immature and adult
Differential WBC (eosinophil) count	EDTA blood	<1 x10 ⁹ /l	Immature and adult
PCV	EDTA blood	25-45 l/l	Immature and adult
Antibody ELISA	Clotted blood or bulk tank milk	N/A	From 2 to 4 weeks post infection but can remain positive for up to 10 months after fluke death.

*AST is not liver specific

Treatment and Control

Control measures should be part of a flock/herd health plan and have three main objectives:

- Appropriate treatment of animals to prevent disease or production loss and reduce infection in the snail population.
- To risk assess and manage grazing areas to avoid ingestion of high numbers of metacercariae.
- Quarantine treatment of added animals.

Elimination of liver flukes by treatment limits the availability of *F.hepatica* eggs and therefore miracidia to snail populations. Before any scheme of snail control is undertaken an assessment of potential snail habitats should be made as these may be localised or extensive. The best long term method for permanent eradication of extensive snail habitats is drainage (Figure 10) but this may be impractical or prohibitively expensive. Techniques such as subsoiling to reduce compaction are also worthwhile. Where snail habitats are localised (Figure 11) fencing off wet areas is useful, particularly in a dry year. If possible graze susceptible stock (especially sheep) on lower risk areas from mid-August onwards as metacercariae will be absent, or fewer in number, in these areas. Lower risk areas could include fields with no snail habitats (Figure 12), fields not grazed by sheep earlier in the year, stubble fields, re-seeds or alternative forages such as brassicas. With the latter be alert to any risk of infection from grass run backs. During autumn it may also be appropriate



Figure 11: Heifer standing in a permanently wet area of localised snail habitat.



Figure 10: Improving drainage can reduce the area of snail habitat.

to allocate lower risk areas to certain classes of stock such as lactating dairy cows or animals approaching slaughter weight. High risk fields will have large numbers of metacercariae on the pasture. They will contain areas of permanent snail habitat and may have been heavily grazed by sheep earlier in the year.

Metacercariae have a lifespan measured in months, with the potential to survive over winter in spite of frosts or snow. This means that high risk areas which remain un-grazed in autumn can still pose a danger to livestock introduced to them later in the winter. Metacercarial numbers peak in October/November and then start to fall due to ingestion, trampling and natural mortality. This explains why acute fasciolosis is most commonly diagnosed in autumn. Once temperatures rise in spring new grass growth will be preferentially eaten, with any surviving metacercariae found in the senescent layer of vegetation.



Figure 12: Low risk field with no snail habitat.

On farms where lower risk areas are limited or unavailable, try to reduce the stocking density in autumn. Finish or sell lambs as soon as possible or consider housing to finish. Monitoring live-weight gains could give an early warning of a problem requiring investigation - infection with *F. hepatica* is only one possible cause of poor growth rates.

Flukicides

All flukicides are effective against adult fluke but activity against immature stages is variable (Table 2). It is extremely important to check product datasheets for information about expected efficacy before treating animals. This is particularly the case in autumn when immature flukes may predominate. Decisions on the timing and frequency of flukicide usage should take into account the annual fluke risk forecast, previous farm

history, abattoir feedback and results of monitoring tests. Triclabendazole resistant liver flukes have been detected in the UK, Ireland and many other countries worldwide. Deaths and ill thrift should be investigated promptly both before and after treatment and instances of suspected lack of efficacy reported to the pharmacovigilance unit of the Veterinary Medicines Directorate (www.gov.uk/government/organisations/veterinary-medicines-directorate).

Monitoring for resistance: Resistant liver flukes survive treatment with a dose of flukicide that would normally be expected to kill them. This advantage is passed genetically to their offspring so that the next generation of *F. hepatica* is also resistant. Monitoring for resistance is complicated by the long prepatent period and the variable efficacy of flukicides against immature stages. Any investigation into suspected treatment failure must first rule out other explanations such as under-dosing, inappropriate product use, reinfection and/or maturation of immature flukes before the possibility of resistance is considered. It is important that any testing carried out is timed to avoid any adverse effects on animal welfare. Animals must be weighed, drenching guns checked for accuracy and treatments administered using good dosing practice.

Fluke Egg Count Reduction Test (FECRT): This test can only be carried out when adult flukes are present in the liver, so may not be appropriate in autumn. Individually identified faecal samples are collected from the same ten animals pre-treatment and **three** weeks later, with fluke egg counts carried out on both occasions. Following successful treatment with triclabendazole the number of fluke eggs should fall by at least 90 to 95%. The FECRT can also be used to monitor the efficacy of closantel which due to its stunting effect on surviving immatures is expected to suppress egg output for more than three weeks post treatment. The FECRT is only suitable for screening other flukicides at times when ALL flukes in the liver are expected to be adult. Also note that the expected efficacy of actives other than triclabendazole may be less than 95% (Table 2). Measurement of liver enzymes can provide supplementary information which may assist with interpretation of results.

Coproantigen Reduction Test (CRT): Faecal samples are collected as above both pre-treatment and **two** weeks later. Samples are tested with the coproantigen ELISA and are expected to be negative post treatment. The reduction in mean percentage positivity can also be calculated and should be at least 90%. This test has been shown to be useful following treatment with triclabendazole when late immature or adult flukes are present. It can be used to screen other actives at times when all flukes in the liver are expected to be adult.

Active Ingredient	Age of fluke in weeks													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Albendazole									50-70%			80-99%		
Oxyclozanide														
Closantel							50-90%			91-99%				
Nitroxylnil														
Triclabendazole			90-99%		99-100%									

Table 2: Efficacy of flukicides against different stages of *Fasciola hepatica*. Adapted from Fasciolosis, p229, Ed. JP Dalton

Histopathology: This involves the recovery of flukes from the bile ducts of sheep three to four days after flukicide treatment. The flukes are formalin fixed and their tegument and digestive/reproductive tracts are examined histologically for evidence of damage.

Egg Hatch Assay: *F. hepatica* eggs are triggered to hatch following exposure to triclabendazole sulphoxide. Significant differences are seen in the percentage of eggs hatching from triclabendazole resistant flukes compared to eggs from triclabendazole sensitive flukes.

The latter two tests are not commercially available at the time of writing.

Quarantine Treatments: There are three main reasons to quarantine treat added animals:

- To kill *F. hepatica* in the liver and prevent disease and production losses.
- To prevent the introduction of *F. hepatica* onto fluke free farms where snail habitats exist.
- To prevent the introduction of *F. hepatica* which are resistant to any of the available flukicides, particularly triclabendazole.

The time of year and source of the added animals should be taken into account when planning quarantine treatments. Two possible strategies for sheep are:

1. Treat with triclabendazole on arrival and closantel or nitroxylnil six weeks later.
OR
2. Treat with closantel on arrival and nitroxylnil six weeks later.

Animals should be held off pastures with snail habitats for as long as possible but ideally for at least three weeks. Faecal samples can be collected to check that treatments have been effective. Straying stock, farm to farm movement of fluke infected wildlife or waterborne dissemination of snails and metacercariae could also introduce flukicide resistant *F. hepatica* to a holding. Snails may also be transported on the feet of birds.

Control

Liver fluke risk varies both annually and from farm to farm making it impossible to produce a universal blueprint for treatment. The following section contains examples of treatment programmes which could be adapted to suit individual farm circumstances. Local knowledge and experience must always be taken into account when planning treatment programmes.

Sheep: On fluke infected farms treat with a flukicide active against immature *F. hepatica* in October and again in January. Additional treatments, given four to six weeks after these routine doses, may be required if a high disease risk is forecast. The same flukicide should ideally not be used year after year for autumn/winter treatments however in years where triclabendazole is not used extra treatments may be required. On lower risk farms, and/or in lower risk years, consider using closantel or nitroxylnil instead of triclabendazole in October and January. A faecal egg count or coproantigen ELISA test could be carried out to determine if treatment in January is required.

On holdings where triclabendazole resistance has been confirmed then closantel or nitroxylinil should be used in the autumn and winter. Depending on the fluke risk a second treatment may be required three to four weeks later. Some researchers have suggested that in the face of an outbreak of acute fasciolosis triclabendazole should continue to be used alongside a second flukicide. Their hypothesis is that a proportion of the fluke population may remain susceptible to triclabendazole.

A flukicide targeting adult flukes should be used in late spring/early summer. This dose is important to prevent infection of snails and thereby reduce fluke challenge in the autumn. It is not recommended to use triclabendazole at this time and there is no need to treat young lambs. Collect faecal samples for testing if you are unsure if treatment is required.

Beef: On beef enterprises the timing of treatment should be related to the date of housing and will depend on the chosen product. A common strategy is to inject nitroxylinil six to eight weeks after housing. On high risk farms, and/or in high risk years, an additional treatment at housing should be considered in order to reduce the impact of liver flukes on performance. The risk of re-infection following treatment means that out wintered cattle may require more than one dose. This is particularly important in advance of spring calving to avoid the risk of excessive condition loss and poor yields of colostrum and milk. Collect faecal samples for testing if you are unsure whether treatment is required.

Dairy: There are restrictions on the administration of many flukicides to both milking cows and pregnant heifers, so close attention should be paid to the datasheet prior to use. Products containing triclabendazole, with milk withdrawal periods of six to seven weeks, are suitable for use at drying off in the autumn and early winter. Once the milking herd has been housed for 10 to 12 weeks all flukes in the liver will be adult so products containing albendazole or oxcylozanide can be used at drying off or during the dry period.

The use of combination products in any class of stock should be considered carefully and avoided if inappropriate. Their use can result in mistimed fluke treatments, administration of an inappropriate flukicide for the time of year or unnecessary use of wormer thus increasing selection pressure for the development of anthelmintic resistance.

Take advice if you are unsure when to treat and which product to use. It is good practice to monitor treatment efficacy whenever possible. As wildlife such as deer and rabbits can be infected with *F. hepatica* it is not possible to eradicate liver fluke on farms with snail habitats.

Forecasting Disease Risk

The risk of severe outbreaks of fasciolosis increases following wet summers. Forecasting systems developed in the 1950s and 60s such as the wet day and the Mt methods are still in use today and are calculated using rainfall, temperature and evapotranspiration data. More sophisticated methods based on computer models incorporating climatic, geographic, farm, necropsy, abattoir and soil hydrology data are under development with the aim of producing forecasts at postcode or even farm level. Forecasts

can be found at www.nadis.org.uk and may be published in the farming press.

Researchers have modelled the impact of climate change on the prevalence of fasciolosis over the next sixty years and predict unprecedented levels of risk and serious disease epidemics particularly in western Scotland, Wales and south west England. On a more positive note, teams of scientists are continuing to carry out research into *F. hepatica*, including the development and trial of prospective vaccines.

Further useful information about liver fluke can be found at www.scops.org.uk and www.cattleparasites.org.uk.

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