

Rumen fluke in cattle in the UK: a review

This article tracks the emergence of rumen fluke in cattle in the United Kingdom. As well as a parasitological summary, clinical signs, diagnosis, treatment and control options are discussed.

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The first diagnosis of rumen fluke made in the author's practice was in April 2012. This was made by the Animal Health and Veterinary Laboratories Agency (AHVLA) Langford on a mixed faecal sample from six organic dairy cows, where ten newly acquired animals were showing ill thrift and milk drop. It was unknown at that time how significant the diagnosis of rumen fluke was, and treatment for both liver fluke and rumen fluke was initiated for all ten animals.

In April 2008, Foster and others reported the identification of *Paramphistomum* species (rumen fluke) on five cattle farms in England and Wales since June 2007. In June 2008, Murphy and others reported paramphistome eggs increasingly being found in bovine faecal samples submitted to Irish Veterinary Laboratories for routine parasite diagnosis. Heavy burdens of rumen fluke had also been observed during post-mortem examination of a number of store cattle and suckler cows in poor condition. In all cases the diagnosis of *Paramphistomum* species was complicated by concomitant parasitic or nutritional disease or poor husbandry.

In July 2008 (VLA Surveillance Report) the first occurrence of *Paramphistomum* in East Anglia (as larvae in the reticulum of a recently slaughtered healthy bullock) was reported. In September of the same year SAC Veterinary Services reported rumen fluke (SAC Veterinary Services, 2008). This was found on two occasions in cattle: first as larvae in the rumen and reticulum during a post-mortem examination, and second as fluke eggs in a submitted faecal sample.

In December 2011 an outbreak of clinical disease and mortality in adult sheep associated with immature paramphistome infection was identified in Scotland (Mason et al, 2012), followed by the first diagnosis of clinical disease in young cattle associated with immature paramphistome infection in England in November 2012 (Millar et al, 2012).

Figure 1 shows the count diagnoses for rumen fluke in cattle, made by AHVLA in England and Wales up to 2nd August 2013. All these diagnoses are eggs in faeces only, apart from the

two clinical cases diagnosed by Millar and others (2012). They therefore only represent infection rather than disease. It appears rumen fluke were not recorded by AHVLA prior to 2010. However, they have been described in UK livestock as far back as the 1950s in an abattoir study in Glasgow (Skuce et al, 2013).

Since April 2012 adult rumen fluke have been incidental findings in the rumen of four dairy cattle post-mortem examinations performed by the author's practice. As reported incidence in the UK increases, should we be concerned and what does the large animal practitioner need to know?

Description

Grossly the adults are small, conical (pear-shaped) maggot-like flukes about 1.0 cm long and light red in colour when fresh (Figure 2). Larval stages are less than 5.0 mm, fresh specimens having a pink colour (Taylor et al, 2007). Microscopically the egg resembles that of *Fasciola hepatica* being operculate, but it is slightly larger (about 130–180 µm), clear rather than yellow and more coarsely granular (Taylor et al, 2007) (Figures 3 and 4).

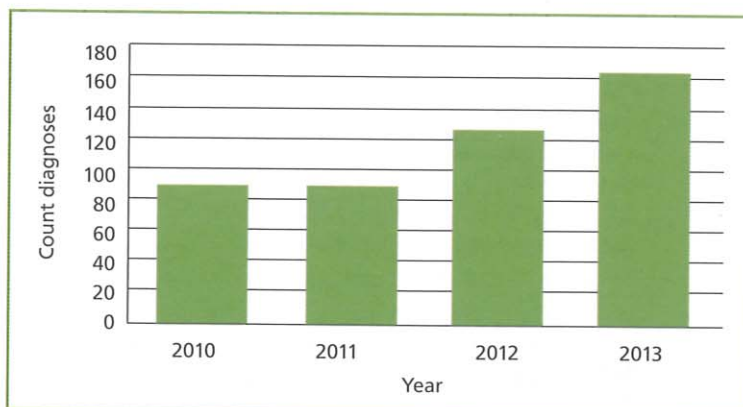


Figure 1. Count diagnoses for rumen fluke made by AHVLA in England and Wales up to 2nd August 2013. Up to date figures courtesy of M. Millar, AHVLA Langford.



Figure 2. Adult rumen fluke in the rumen of an adult dairy cow.

Hosts

Rumen fluke occur worldwide. Definitive hosts include cattle, sheep, goat, deer, buffalo and antelope (Figure 5). Intermediate hosts are water snails principally *Planorbis* and *Bulinus* (Taylor et al, 2007).

One of the most significant recent developments in our understanding of rumen fluke has come from a paper by Gordon et al (2013). They have demonstrated that rumen fluke from imported and home-bred cattle and sheep in Scotland belong to the species *Calicophoron daubneyi* (formerly *P. daubneyi*) rather than *Paramphistomum cervi*, the species presumed to be most common in Great Britain. Gordon and others conclude that *C. daubneyi* is the most common rumen fluke of domestic ruminants in Scotland.

What's in a name? The greatest significance of this finding is that unlike *P. cervi*, *C. daubneyi* can use the same intermediate host snail species as *Fasciola hepatica* or liver fluke known as *Galba truncatula* (Figure 6). Liver fluke has a widespread distribution in the UK which is only possible due to the distribution of its hosts. If rumen fluke can spread via these same hosts then the potential distribution throughout the UK is significant.

C. daubneyi is the most common rumen fluke in continental Europe. Gordon and others (2013) also confirmed that the flukes taken from one of the cases described by Miller and others (2012) were *C. daubneyi* giving greater power to the argument that *C. daubneyi* is UK wide.

Life cycle

The life cycle of rumen fluke is similar to liver fluke in that they require two hosts; a mammalian definitive host and a snail intermediate host (De Waal, 2010).

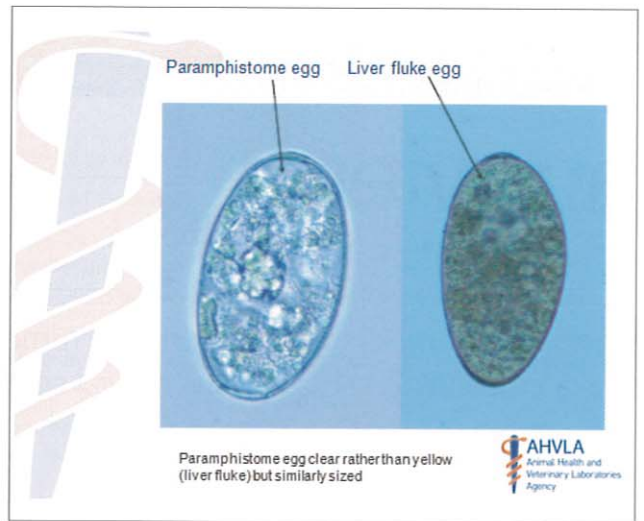


Figure 3. A rumen fluke egg is clear rather than yellow like a liver fluke egg. Photo courtesy of M. Millar, AHVLA Langford.

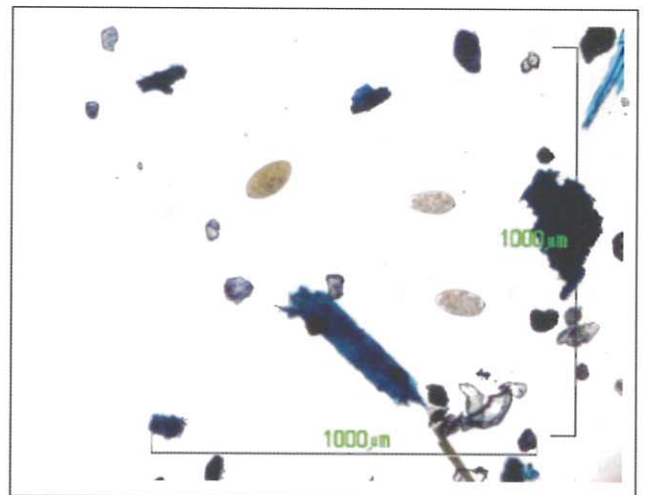


Figure 4. Rumen fluke and liver fluke eggs are both usually between 130–180 μm. Photo courtesy of M. Millar, AHVLA Langford.

Infected animals excrete eggs in the faeces. The eggs develop and hatch under suitable conditions (temperature and moisture) when they have been freed from the faecal mass. Development of the egg takes a minimum of 4 weeks (at 17°C) before miracidia hatch. The newly hatched miracidia then find a suitable intermediate host aquatic snail (De Waal, 2010).

Development of infection in the snail takes a minimum of 5 weeks and infected snails can live and shed cercariae for up to 1 year. After emerging from the snail, cercariae encyst forming metacercariae on the ventral surface of sub-aquatic plants where they can remain viable for up to 6 months. The final host ingests the encysted metacercariae on the herbage (De Waal, 2010).

After ingestion, metacercariae excyst and larvae attach and feed for up to 6 weeks on the mucosa of the duodenum. They may also be found in the ileum, jejunum and abomasum, before moving to the forestomachs (Foster et al, 2008). In the rumen the fluke attach to their predilection sites on the dorsal surface of the anterior ruminal pillar, and the dorsal and ventral aspects of



Figure 5. African buffalo — a definitive host of rumen fluke.



Figure 6. *Galba truncatula*.

the posterior ruminal pillar. Here they continue to grow to reach their maximum size 5 to 9 months after infection. The minimum prepatent period is 3 to 4 months (De Waal, 2010).

Epidemiology

Rumen fluke often depend for their continuous endemicity on permanent water masses, such as lakes and ponds, from which snails are dispersed into previously dry areas by flooding during heavy rains (Figure 7). Rumen fluke eggs deposited by animals grazing these areas hatch and infect snails. Subsequent production of cercariae often coincides with receding water levels making them accessible to grazing ruminants (Taylor et al, 2007).

Foster et al (2008) highlight the fact that the VLA carries out several thousand faecal examinations per year on bovine faecal samples and yet it is only recently that rumen fluke eggs have been noted. They suggest it may be that conditions conducive to the survival of *fasciola* have led to increased exposure of cattle to rumen fluke. Changing farm practices — for example, more organic farming, reduced use of herbicides and increasing incidences of flooding resulting in animals accessing water courses — might also account for increased exposure and infection levels.

Previous infection and the age of the host animal afford some protection against re-infection. Acute disease is usually seen in young cattle less than 2 years of age. Older (adult) animals often continue to harbour low burdens of adult parasites and are therefore important reservoirs of infection for snails (De Waal, 2010).

Pathology

The adult parasites in the forestomachs are well tolerated, even when many thousands are present and feeding on the wall of the rumen or reticulum (Taylor et al, 2007). Immature flukes embed in the mucosa of the upper ileum and duodenum and are plug feeders, which can result in severe erosion of the duodenal mucosa (Taylor et al, 2007) (Figures 8 and 9).

Clinical signs

Clinical disease is rare and only associated with heavy burdens of immature fluke at the intestinal phase of infection. A small number of severe cases have been reported in Ireland with the following clinical signs:

- Dullness
- Dehydration
- Rapid weight loss
- Severe watery scour which may contain traces of blood
- Anaemia
- Hypoproteinaemia
- Sub-mandibular oedema (Animal Health Ireland, 2011).

Left untreated, severely affected animals may die due to dehydration (Animal Health Ireland, 2011). Mortality in acute outbreaks can be as high as 90% (Taylor et al, 2007).

The first case of clinical disease reported due to immature rumen fluke in England was diagnosed in two cattle post-mortem examination submissions to the AHVLA (Millar et al, 2012). One submission was from a calf from a group of 20 preweaned suckler calves aged 6 to 8 months, and the other was two animals from a group of 31 weaned cattle aged 7 to 14 months. Both groups had been grazing waterlogged pasture, and non-responsive watery diarrhoea and severe condition loss over 3 weeks were reported, with at least four deaths from each group. All the young cattle from both groups presented with diarrhoea and some were severely depressed, dehydrated and anorexic, although the dams from the first group were unaffected (Millar et al, 2012).

Diagnosis

Laboratory testing will readily detect eggs in faeces of an animal carrying adult rumen fluke, but it is much more difficult to confirm disease caused by the immature flukes, as there are no conclusive findings in faeces or blood samples (Animal Health Ireland, 2011).

A history of grazing pasture containing snail habitat coupled with the clinical signs, should first alert the clinician to the differential diagnosis of clinical disease due to immature rumen fluke. Faecal examination is of limited value since the acute disease occurs during the prepatent period. However, large numbers of paramphistome eggs can sometimes be present in faeces during acute disease as the intestinal phase may also be accompanied by large numbers of adult flukes in the forestomach (Taylor et al,

2007). In very severe cases observed by the Irish Regional Veterinary Laboratories, large numbers of immature rumen flukes have also been found in faeces (Animal Health Ireland, 2011).

Definitive diagnosis during the acute disease phase is reached only by detection of larval fluke at post-mortem examination (Foster et al, 2008).



Figure 7. Permanent water masses such as flooded ditches can allow dispersal of snails during flooding.

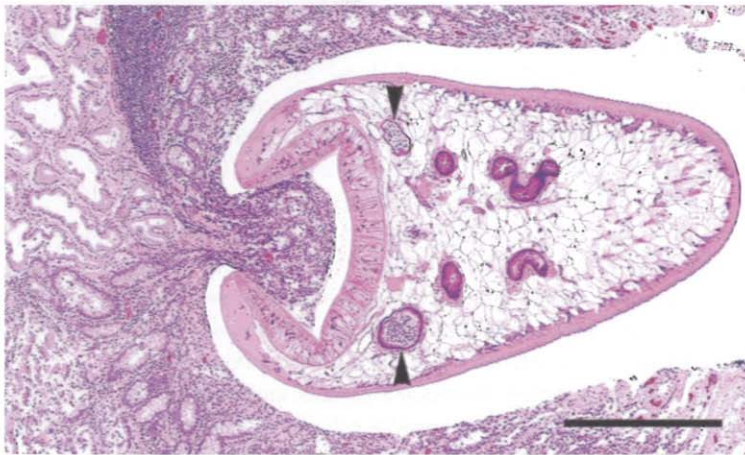


Figure 8. Immature rumen fluke with plug of duodenum. Photo courtesy of M. Millar, AHVLA Langford.

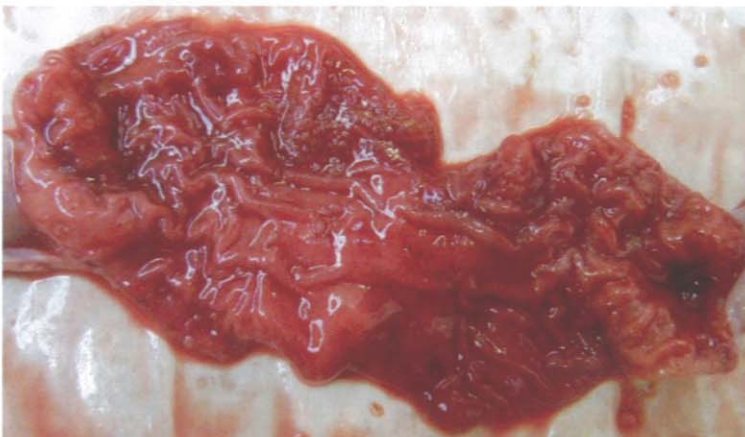


Figure 9. Duodenum showing marked reddening with immature flukes (resembling bubbles) on the mucosa. Photo courtesy of M. Millar, AHVLA Langford.

Treatment

Rumen fluke are not killed by the common flukicides used in cattle and sheep (AHVLA, 2013). The efficacy of four anthelmintics against *C. daubneyi* in naturally infected dairy cattle was determined by Arias and others (2013). The best results were achieved with closantel and oxclozanide with faecal egg count reduction (FECR) values of 97–99% and cattle positive by coprology reduction (CPCR) values of 85–93%. The observation of a similar efficacy with closantel and oxclozanide led the authors to recommend the administration of closantel in those countries where oxclozanide is not available. Closantel is not licensed for use in dairy cattle in the UK.

The AHVLA (2013) recommends products containing oxclozanide as the only proven treatment for rumen fluke but emphasise effectiveness is variable, particularly against larval stages. AHVLA (2013) also emphasise oxclozanide only has activity against adult liver fluke, so if used in the autumn/winter there is the risk of acute liver fluke disease particularly in sheep. Conversely the improvement in production reported in animals treated with oxclozanide may be due to effective treatment of adult liver fluke and not a perceived treatment of rumen fluke.

Ultimately advice must be to not treat rumen fluke unless clinical signs are present. Animal Health Ireland (2011) stresses that the detection of rumen fluke eggs in faecal samples or the detection of adults in small numbers in the rumen is not in itself a reason to institute specific control measures, as light infections appear to have no effect on animal health or productivity. The routine implementation of a preventative dosing regimen for rumen flukes is rarely justified, except on farms where severe disease and losses have been confirmed in the past (Animal Health Ireland, 2011). Despite this, sales of flukicides containing oxclozanide in Ireland increased by 600% in 2012 (Skuce et al, 2013).

Animal Health Ireland (2011) further highlight that apart from the economic costs that arise from unnecessary use of any anti-parasitic drug, it is especially important to treat rumen flukes sensibly and sparingly, given that there is only one effective compound (oxclozanide). The development of oxclozanide-resistant strains of rumen fluke must be avoided at all costs. The development of such strains is a distinct possibility if a single compound like oxclozanide is used indiscriminately over several years (Animal Health Ireland, 2011).

Control

Control advice to farmers may seem unnecessary with the current situation of rumen fluke in the UK. In Ireland where the situation appears more advanced, advice centres on biosecurity, reduction of exposure, and treatment. It is worth being aware of this advice in case the situation in the UK changes.

Biosecurity advice focuses on preventing introduction of rumen flukes onto the farm. Bought in animals of unknown origin or animals that are from known rumen fluke-infested farms should be tested by faecal examination and dosed with an effective product prior to allowing access to pasture, if they have rumen fluke eggs in their faeces (Animal Health Ireland, 2011).

Reducing the possibility of exposure to rumen fluke larvae on pasture is the same as reducing exposure to liver fluke. Restrict-

ing access to fields, or parts of fields, that are prone to flooding or water-logging will reduce exposure to contaminated herbage. The fencing off of drains, ponds and watercourses should also reduce exposure (Animal Health Ireland, 2011).

Conclusions and the future?

Should the clinician, therefore, be concerned about the emergence of rumen fluke in the UK?

It appears rumen fluke are on the increase, both from reports in the literature and records from AHVLA (Figure 1). There has also been the first confirmed clinical case of disease due to immature rumen fluke in the UK (Millar et al, 2012). Gordon and others (2013) highlight that the morphological similarity of rumen fluke eggs may cause a false positive result in the faecal egg count for liver fluke. If rumen fluke eggs are mistaken for liver fluke eggs, this could result in a false diagnosis of liver fluke treatment failure. Could it also be that until recently there has been a failure to differentiate rumen fluke eggs from liver fluke eggs in faecal samples? This combined with improving climatic conditions (wet summers) could account for the significant increases in rumen fluke egg counts? Liver fluke remains a far more important disease than rumen fluke at present, but differentiating between the two is essential.

Why has there only been one clinical case due to immature rumen fluke reported back in November 2012 and none since? Is this a one-off or is it more likely that clinicians are not recognising the disease?

With any new emerging disease there are always more questions than answers. Millar and others (2012) who diagnosed the first clinical case in the UK offered the following advice: 'we recommend that colleagues in practice remain alert to the possibility of this disease, and consider it as a potential differential diagnosis in outbreaks of diarrhoea in susceptible ruminants, particularly where there is a history of grazing pasture being prone to flooding'. Armed with this advice, appropriate diagnostics can be initiated and if necessary suitable treatment and control programmes put in place. **LS**

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Continuing Professional Development

In order to test your understanding of this article, answer these multiple choice questions, or if you are a subscriber, go online at www.ukvet.co.uk, and find many more multiple choice questions to test your understanding.

Adjunctive tests

- How many hosts does the rumen fluke require to complete its life cycle?
 - 1
 - 2
 - 3
 - 4
- How long can a snail infected with the cercariae of rumen fluke shed them for?
 - 1 year
 - 2 years
 - 3 years
 - 4 years
- Where could you not find immature rumen fluke in the cow?
 - Abomasum
 - Duodenum
 - Jejunum
 - Liver

For answers please see page 254

KEY POINTS

- The number of diagnoses of rumen fluke in cattle in the United Kingdom has increased considerably over the last 3 years.
- It may be that conditions conducive to the survival of liver fluke have led to increased exposure of cattle to rumen fluke.
- Clinical disease is rare and only associated with heavy burdens of immature fluke at the intestinal phase of infection.
- Laboratory testing will readily detect eggs in faeces of an animal carrying rumen fluke, but it is much more difficult to confirm disease caused by the immature flukes, as there are no conclusive findings in faeces or blood samples, a post-mortem examination is usually required.
- Control advice centres on biosecurity, reduction of exposure, and treatment.
- Products containing oxyclozanide are the only proven treatment for rumen fluke but effectiveness is variable, particularly against larval stages.