Regional Veterinary Laboratories Report

October 2022

Regional Veterinary Laboratories (RVLs) carried out necropsy examinations on 625 carcases and 74 foetuses during October 2022. Additionally, 2,192 diagnostic samples were tested to assist private veterinary practitioners with the diagnosis and control of disease in food-producing animals. This report describes a selection of cases investigated by the Department of Agriculture, Food and the Marine's (DAFM) veterinary laboratories in October 2022.
The objective of this report is to provide feedback to veterinary practitioners on the pattern of disease syndromes at this time of the year by describing common and highlighting unusual cases. Moreover, we aim to assist with future diagnoses, encourage thorough investigations of clinical cases, highlight available laboratory diagnostic tools and provide a better context for practitioners when interpreting laboratory reports.
In addition, this month's report contains advice on using laboratory diagnostics to investigate bovine neonatal enteritis.

CATTLE

Pneumonia and enteritis were the most common causes of death in cattle submitted to the RVLs in October 2022.

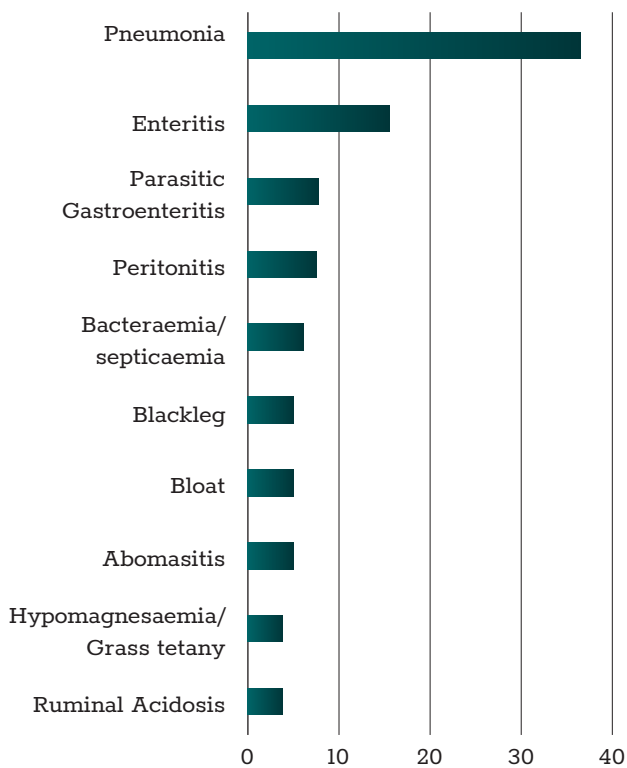

Table 1: The most common causes of death diagnosed in cattle submitted to DAFM RVLs in October 2022.

GASTROINTESTINAL TRACT

Abomasitis

A 16-day-old calf was submitted to Kilkenny RVL. The calf was severely dehydrated. There was a marked abomasitis; there was marked hyperaemia of the mucosa, the abomasal folds were oedematous and there was multifocal emphysema. *Escherichia coli* was cultured from multiple organs suggesting a bacteraemia. Histopathology revealed an abomasitis with *Sarcina* sp. visible. *Sarcina* are fastidious Gram-positive anaerobic bacteria. Associated gross lesions include emphysema and oedema of the abomasal wall, mucosal hyperaemia and haemorrhage, and rupture of the abomasum. Until more is known of the specific risk factors for the involvement of *Sarcina* spp., the practical advice includes maintaining good hygiene when preparing and handling milk feeds for young ruminants.

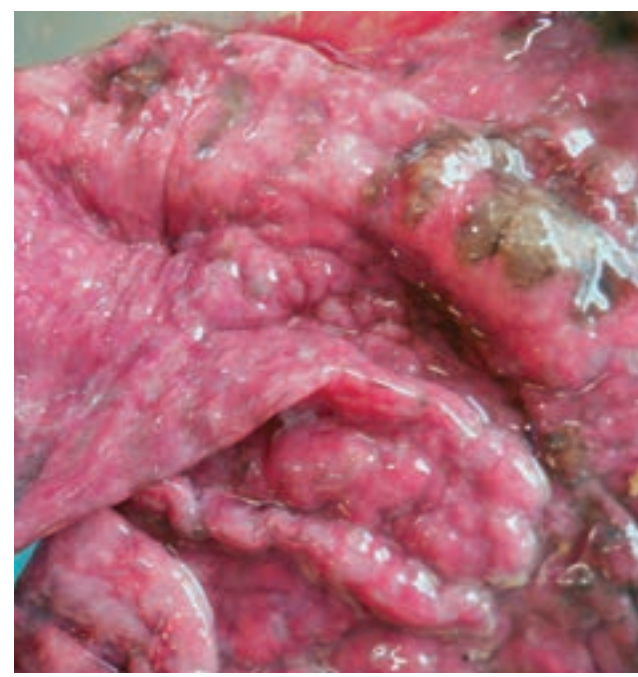

Figure 1: Marked abomasitis with emphysema from which *Sarcina* sp. were identified. Photo: Aideen Kennedy.

Perforated abomasal ulcer

An eight-day-old calf was submitted to Kilkenny RVL. The calf was severely dehydrated. There was diffuse peritonitis. There were multifocal abomasal ulcers and one perforated abomasal ulcer. The calf also had an umbilical infection and was a ruminal drinker. Concurrent disease or stress can be risk factors for abomasal ulcers and therefore these conditions may have contributed to ulcer development in the current case.

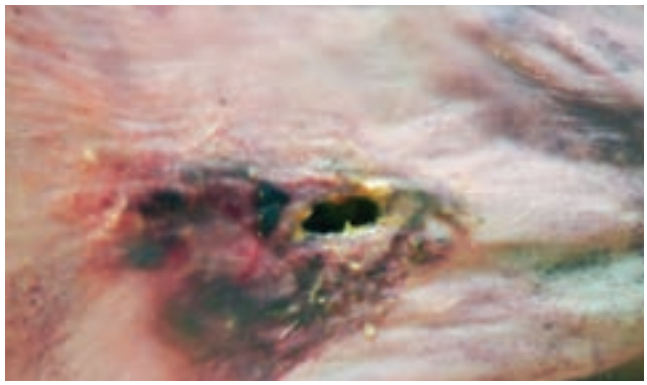

Figure 2: A perforated abomasal ulcer. Photo: Aideen Kennedy.

Congenital hepatic fibrosis/Cerebrocortical necrosis

Athlone RVL examined an eight-month-old dairy heifer with a history of having had 'summer scour' problems in the group earlier in the summer. The weanling was found with a slight stagger, treated by a vet for neurological signs and subsequently euthanised. On gross post-mortem examination, the liver was enlarged with white tracts of fibrosis on the surface, resulting in an uneven surface, and the parenchyma was similarly fibrosed and difficult to cut. The small and large intestinal contents were loose, but the faeces were firm, and there was no ulceration in the mouth, pharynx or oesophagus. Ulceration of the proximal digestive tract is associated with summer scour syndrome. The cerebral cortex fluoresced under ultraviolet light suggesting cerebrocortical necrosis (CCN). Histopathology of the brain confirmed that the main changes seen were consistent with CCN. Liver histopathology showed replacement of 20-50 per cent of the hepatic parenchyma by robust, portal-to-portal bridging, mature, collagenous stroma; and within this stroma were markedly increased numbers of small calibre bile duct profiles that often had irregular to absent lumina, were tortuous with occasional branching, and frequently occurred at the limiting plate directly adjacent to periportal hepatocytes. The changes in the liver were consistent with congenital hepatic fibrosis.

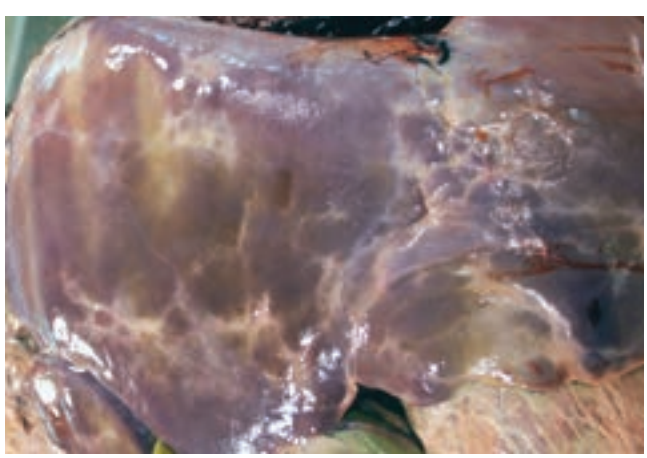

Figure 3: White tracts of connective tissue on the surface of a liver, likely due to congenital hepatic fibrosis. Photo: Denise Murphy.

Parasitic gastroenteritis

An eight-month-old weanling with a history of scour, which had been unresponsive to anthelmintic and antibiotic treatment, was submitted to Kilkenny RVL. On gross examination, there was a thickened abomasum with a 'Morocco leather' appearance to the mucosa. McMaster results showed a strongyle count of less than 50 eggs per gram (EPG), but this may be explained be recent anthelmintic treatment. On histopathology, the morphological changes were consistent with parasitism, with nematodes visible in the abomasal mucosa.

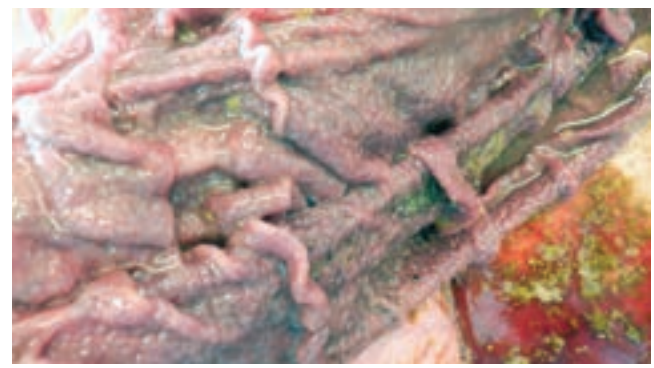

Figure 4: The 'Morocco leather ' appearance of thickened abomasal mucosa in a case of parasitic gastroenteritis. Photo: Aideen Kennedy.

RESPIRATORY TRACT

Parasitic bronchitis

Athlone RVL examined a three-year-old heifer with a history of having been coughing for the previous week. She was treated by the vet but died. On gross post-mortem examination, there was diffuse subpleural and interlobular emphysema and bullae in the caudodorsal lung lobes bilaterally, and some collapsed lobules. Lungworm (*Dictyocaulus viviparus*) were visible in the trachea and bronchial tree. There were haemorrhages on the trachea and bronchial mucosa and lots of stable foam/froth. Viral and bacterial polymerase chain reaction (PCR) test results were negative. Histopathology was consistent with a diagnosis of parasitic bronchitis or 'hoose pneumonia'.

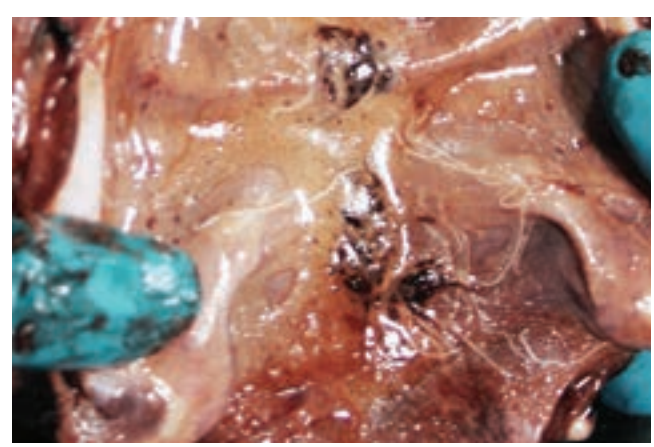

Figure 5: Adult lungworm (*Dictyocaulus viviparus*) in the proximal trachea, near the larynx. Photo: Denise Murphy.

Sligo RVL diagnosed a case of severe lungworm infection in a three-month-old calf.

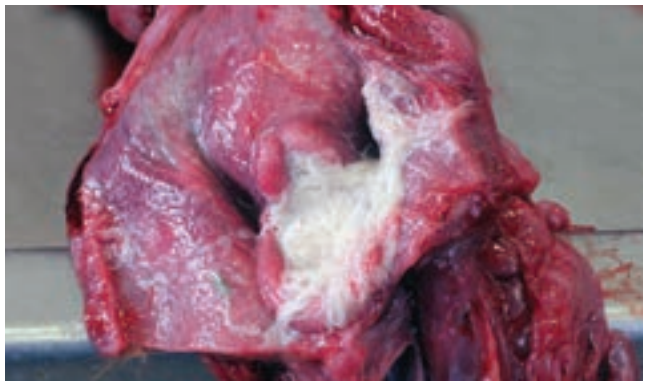

Figure 6: Adult lungworm (*Dictyocaulus viviparus*) spilling out from the trachea through the larynx. Photo: Colm O'Muíreagaín.

CARDIOVASCULAR SYSTEM

Atrial septal defect

Athlone RVL examined an 18-month-old bull with a history of ongoing respiratory problems; the animal received treatment for pneumonia, relapsed and was euthanised. On gross post-mortem examination, the liver was enlarged and had an uneven surface and there was a 'nutmeg' pattern on cut surfaces. The pericardial sac was distended with clear fluid, the heart was enlarged and there was an atrial septal defect. Bilaterally, the apical portions of the cranial lung lobes were fibrosed and consolidated due to a chronic bronchopneumonia. A diagnosis of a congenital atrial septal defect and secondary chronic bronchopneumonia was made.

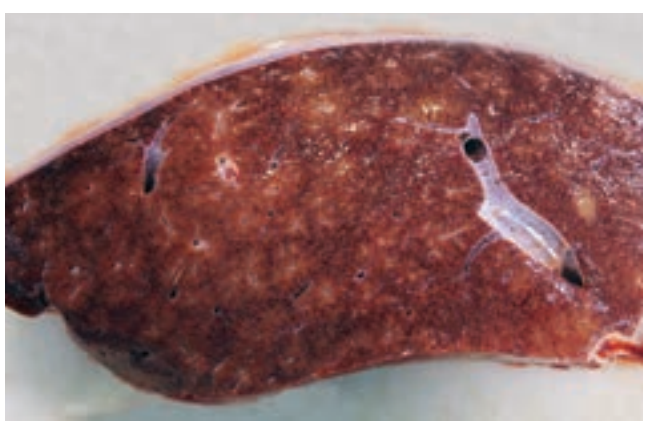

Figure 7: Congested hepatic parenchyma giving the classic 'nutmeg' effect in a case of atrial septal defect. Photo: Denise Murphy.

MUSCULOSKELETAL

Blackleg

Limerick RVL examined a seven-month-old Aubrac cross weanling that was found collapsed with blood coming from its nostrils and died shortly afterwards. Blackleg lesions with a rancid, spoilt butter smell were discovered in the hindquarter and intercostal muscles. There was a fibrinous pericarditis, and the lung was covered in blood. *Clostridium chauvoei* was confirmed by fluorescent antibody technique (FAT), and it was established that the animal had not been vaccinated.

POISONINGS

Yew poisoning

Limerick RVL examined a dry suckler cow and a yearling that had died suddenly, after moving to pasture previously grazed by cows and calves. There had been a history of unexplained deaths in this field over the years. Yew (*Taxus baccata*) plant material was identified in the rumen of both animals. Yew trees contain taxine alkaloids which are cardiotoxins, causing sudden death in the case of acute toxicity or ataxia, bradycardia, dyspnoea, tremors, collapse and death in the case of subacute toxicity. The highest level of taxines occur in the yew plant at wintertime.

Figure 8: Leaves of the Yew tree (*Taxus baccata*) found in the rumen contents of a cow that had died suddenly. Photo: Brian Toland.

Rumen fluke

Rumen flukes are parasites of ruminants and occur worldwide. They have a snail intermediate host. The adult parasites live in the rumen (stomach) and the immature larval forms live in the small intestine. Clinical disease is due to intestinal damage caused by massive numbers of larvae in the small intestine (larval paramphistomosis). The adult worms in the rumen are not usually considered to cause disease. The current preliminary scientific research suggests that the main/only rumen fluke in cattle in Ireland is *Calicophoron daubneyi* which uses the mud snail *Galba (Lymnaea) truncatula* as its intermediate host; this is the same snail that acts as the intermediate host for the liver fluke *Fasciola hepatica*.

Clinical disease occurs occasionally with clinical signs including dullness, dehydration, rapid weight loss, severe watery scour which may contain traces of blood, and swelling under the jaw (bottle-jaw). Severely affected animals may die due to dehydration. As the clinical signs described here are not exclusive to rumen fluke infection, diagnosis by clinical signs alone is unreliable. However, a severe scour and weight loss and abnormally low levels

of albumin in blood samples combined with a history of grazing wet 'flukey' ground, especially in the late summer or autumn would raise suspicions. Demonstration of rumen fluke in a faecal sample (eggs/larvae) would help confirm the diagnosis, however testing for the larvae rather than fluke eggs is not routinely available in laboratories and, generally, it is only in severe cases that larvae are detected in faeces. Therefore, a negative result does not rule out larval paramphistomosis.

Control of rumen fluke should focus on reducing the possibility of exposure to rumen fluke larvae on pasture. Restricting access to fields, or parts of fields, which are or have been wet or waterlogged will reduce exposure to contaminated herbage. Most of the drugs that control liver fluke DO NOT kill rumen fluke. Although not specifically licensed in Ireland for the treatment of rumen fluke, it has been reported in the scientific literature that oxyclozanide can kill both mature and immature stages of this parasite. Cattle may occasionally show transient scouring and inappetence, and dairy animals may have decreased milk yield following treatment.

SHEEP

Bacteraemia/septicaemia and pneumonia were the most common causes of death in ovine carcases submitted to the RVLs in October 2022.

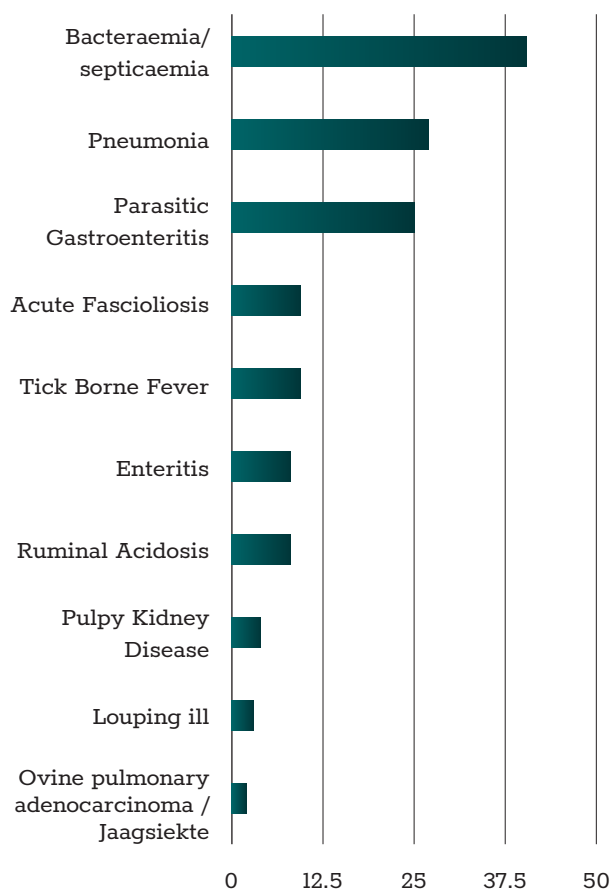

Table 2: The most common causes of death diagnosed in ovine carcases submitted to DAFM RVLs in October 2022.

GASTROINTESTINAL TRACT

Acute fasciolosis

Limerick RVL examined a six-month-old Suffolk cross lamb that was purchased a week earlier and was dosed with albendazole. Necropsy revealed a severely damaged liver with extensive haemorrhage and numerous fluke present, there was a high strongyle count of 5,850 EPG and the lamb also tested positive for tick-borne fever, *Bibersteinia trehalosi* and *Mannheimia haemolytica*. For clinical outbreaks of acute and sub-acute fluke, the only effective treatment is triclabendazole. Drenched sheep should be moved to clean pasture afterwards. Fluke egg counts will not detect acute and sub-acute cases as fluke eggs are not being produced yet.

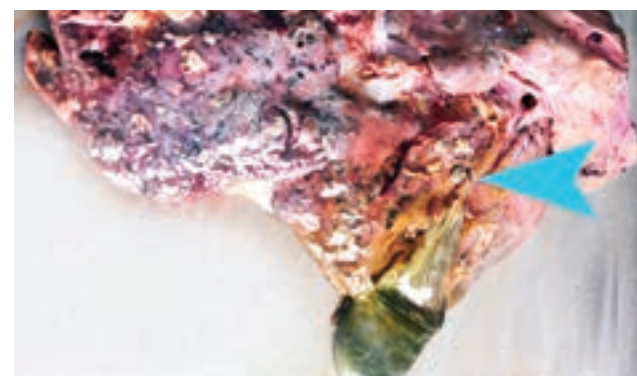

Figure 9: Ovine liver with extensive haemorrhage due to acute fascioliosis, with a mature fluke visible (blue arrow). Photo: Brian Toland.

RESPIRATORY TRACT

Laryngeal chondritis

Limerick RVL examined a three-year-old pedigree Texel ram with a history of sudden death at grass. The ram was running with a group of breeding ewes. On gross examination, there was subcutaneous oedema of the head and neck, pulmonary oedema and focally extensive abscessation of the larynx probably a sequel to laryngeal chondritis '(Texel throat'). Culture of the laryngeal lesion yielded a mixed bacterial growth. Culture of the lung yielded *Listeria ivanovii*. A large number of strongyle eggs (8,500 eggs per gram) and coccidial oocysts were detected in the faeces.

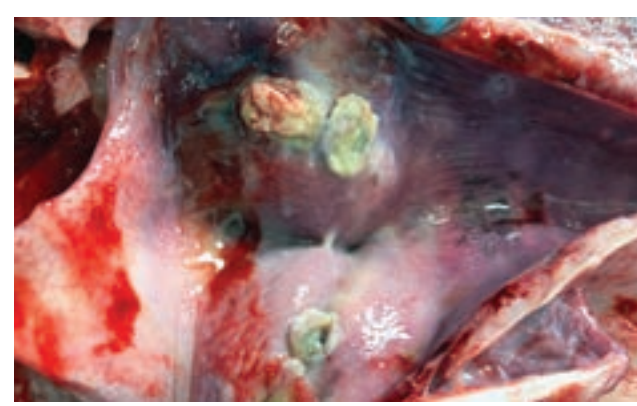

Figure 10: Multifocal abscessation of the larynx, probably a sequel to laryngeal chondritis. Photo: Alan Johnson.

Bacteraemia/septicaemia

Multiple cases of infection due to *B. trehalosi* were submitted to Kilkenny RVL in October. One case involved the submission of four lambs from one flock; six had died in the flock overnight having been noticed slightly off-form the previous evening. Two of the lambs had areas of consolidation in their lungs, while two had diffusely congested lungs. Oesophageal ulcers were noted in two cases. *B. trehalosi* was cultured from multiple organs from each of the lambs, suggesting a bacteraemia or septicaemia. High strongyle egg counts were reported in three of the lambs, so a review of parasite control was recommended along with a review of vaccination protocols. *B. trehalosi* can be associated with ulcerative lesions covered by yellow plaques of fibrin on the tongue, larynx, and oesophagus.

Figure 11: Oesophageal ulcers with exudate near the larynx (left). Photo: Aideen Kennedy.

POISONINGS

Copper toxicity

A nine-month-old ram was found dead and submitted to Kilkenny RVL. On opening the carcase, the tissues appeared jaundiced. The kidneys were dark in colour. The urine was port-wine-coloured, and the faeces were soft. Liver and kidney copper levels were very elevated supporting a diagnosis of copper toxicity. In addition, there was a very high strongyle count of over 4,000 EPG. Stress can precipitate copper release from the liver and the parasite burden may have contributed to this.

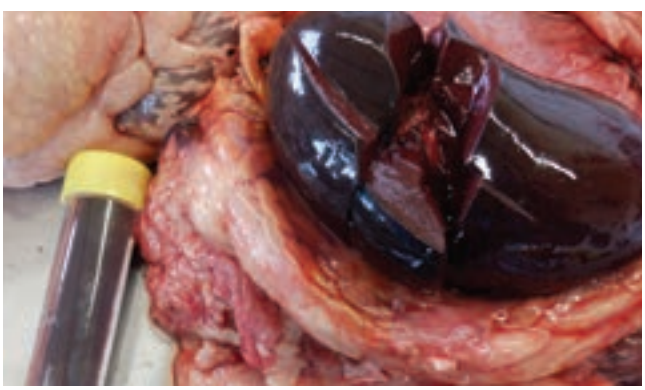

Figure 12: Dark kidney and port-wine-coloured urine (left) from a ram in which copper toxicity was diagnosed. Photo: Aideen Kennedy.

USING LABORATORY DIAGNOSTICS TO INVESTIGATE NEONATAL ENTERITIS

When performing an investigation of neonatal enteritis on farm it is advisable to submit samples from a number of affected animals. Animals that have been sick or that have received treatment for a prolonged period of time are not suitable candidates for sampling. Ideally, three to five diarrhoea samples should be collected from early, affected, and untreated calves. Faecal samples should only be submitted in screw-top plastic containers (Figure A). In addition, blood samples (serum/red top) should be submitted to check if there has been adequate passive transfer of maternal antibodies from colostrum to the calf. Five to ten blood samples should be taken from calves aged less than ten days old, and these samples should not be collected from sick animals.

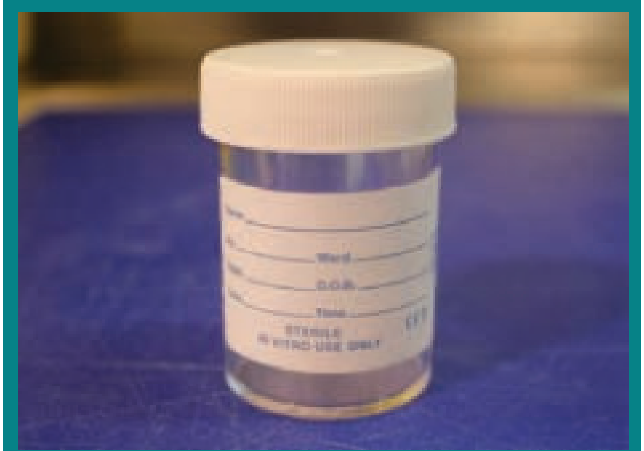

Figure A: Suitable screw-top plastic container for neonatal faecal samples.

If in doubt about sample selection, contact the laboratory directly for advice.