SRUC Veterinary Services Monthly Report for September 2023

OVERVIEW

- **Death due to *Dictyocaulus viviparus* reinfection syndrome in a fattening bull.**
- **Fog fever in a Limousin heifer following a move to silage aftermath**
- **Hepatogenous photosensitisation secondary to chronic copper toxicity in lambs**

GENERAL INTRODUCTION

Despite a few dry days at the beginning of the month September proved to be warm and wet. When compared with the 1991 to 2020 period total rainfall was 136 per cent and sunshine 117 per cent of average. The mean temperature was 1.5 degrees higher making it the third warmest September in a 140-year series.

DISEASE ALERTS

The following conditions were reported by SRUC VS disease surveillance centres in December 2022. Given similar climatic and production conditions, they could also be important this year.

- **Ill thrift and death of dairy calves associated with low environmental temperatures**
  Depending on air speed, the lower critical temperature for calves from birth to three weeks-of-age lies between 10 and 15˚C. Below this the calf needs to use energy to maintain its body temperature. Milk replacer concentration should be increased to 150 g/l fed at a rate of at least 3 litres twice a day. Sufficient dry bedding should be available and there should be no draughts at calf level. The use of calf jackets and feeding three times a day are other options to consider.

- **Rumen acidosis in store lambs**
  Store lambs are at risk of rumen acidosis following the introduction of concentrate feeds. Lambs fed ad lib can also be affected when hoppers are re-filled after the feed has inadvertently been allowed to run out. Concentrate feeds should be introduced gradually and lambs should ideally be fed for a few weeks before hoppers are introduced. Barley as part of a home mixed ration is best fed whole. Wintery conditions can heighten the risk by increasing lamb reliance on concentrate feed.

CATTLE

Toxic conditions

A two-year-old Limousin heifer was found dead in a field where a large limb had recently fallen from an oak tree. Postmortem examination found reduced rumen fill with numerous brown and a small number of green oak leaves within the contents. No whole or part acorns were identified. Dry, firm faeces within the large intestine suggested constipation which is reported in cases of oak (*Quercus* spp) toxicity. There can be several days delay between ingestion of oak and the development of clinical signs therefore inspection of rumen contents may be unrewarding. Both acorns and oak leaves contain phenols and tannins such as gallic acid and pyrogallol that are toxic when ingested in sufficient quantities. These compounds bind and precipitate proteins leading to renal failure. Unfortunately, carcase autolysis made assessment of the viscera challenging and it was not possible to carry out histopathology to look for evidence of renal tubular necrosis. Oak poisoning was proposed as the cause of death based on the evidence available.

Parasitic diseases

A 14 -month-old Aberdeen Angus fattening bull was purchased in March and injected with a long acting moxidectin product prior to being turned out with 29 others in May. It was found dead in September and submitted for postmortem examination. This identified a relatively small area of wedge-shaped consolidation suggestive of parasitic bronchitis in the left caudal lung lobe. No lungworms were detected in the airways however, histopathology revealed acute eosinophilic bronchopneumonia in the caudo-dorsal lung lobes and sections of nematodes were visible within bronchioles and alveoli. The visceral pleura in these areas had appeared greenish and in hindsight this was thought to be a result of eosinophilic infiltration rather than autolysis. The histopathology was typical of lungworm reinfection syndrome and considered severe enough to be the cause of death. It was postulated that haemorrhages found within the thymus, trachea and neck muscles could have been a result of dyspnoea. The findings indicated that the bull had been exposed to *Dictyocaulus viviparus* in the past, but that phase one immunity had waned allowing larvae to reach the lungs and trigger a phase 2 hypersensitivity reaction. The pre-purchase grazing and anthelmintic history was unknown, but the farmer confirmed that losses due to lungworm had previously occurred in the area the bulls had been grazing. Re-infection syndrome is often associated with a move to an area of high larval challenge.

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A more typical lungworm outbreak involved a group of March-born dairy calves that had been turned out in June. Respiratory signs were noticed in mid-August, and as a result they were treated with oral levamisole. They initially improved but were housed three weeks later and treated with antibiotics after failing to fully recover. Five calves died over the next two weeks and a recumbent, dyspnoeic heifer was euthanased for postmortem examination. The lungs were hyperinflated with small patches of peri-bronchial consolidation. Small numbers of *D viviparus* were observed in the trachea, with very large numbers present within the bronchi (Fig 1). It was concluded that the lungworm burden was due to reinfection during the three-week period between anthelmintic treatment and housing. Detailed history taking is important when investigating possible treatment failure in these types of cases.

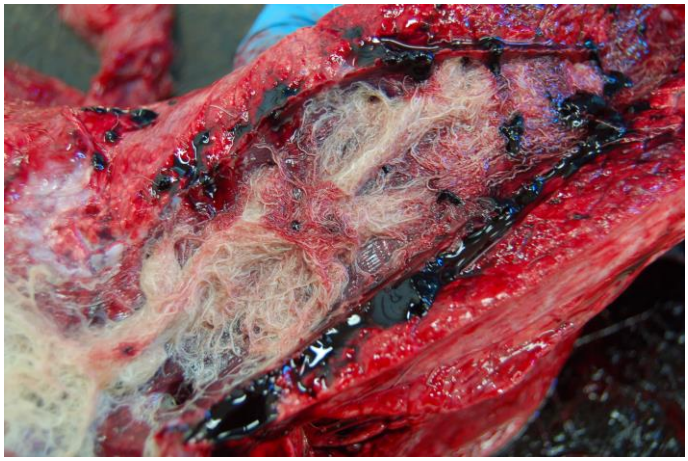

Figure 1 – *Dictyocaulus viviparus* worms in the airways in a previous case of fatal lungworm infection

Alimentary tract disorders

A 19-day-old Holstein heifer was euthanased to investigate an ongoing issue with calf deaths. Three calves with ocular lesions had been reported and *Escherichia coli* K99 enteritis had recently been diagnosed. The carcase weighed only 25kg and there was perineal hair loss secondary to diarrhoea. Bilateral corneal oedema and hypopyon were noted. Internally there was no body fat and serous body cavity effusions suggested hypoproteinaemia. A small number of 0.5 cm diameter circular lesions were present on the oesophageal mucosa with a further dozen similarly sized target lesions found on the rumen mucosa (Fig 2). There was evidence of moderately extensive lung consolidation with interlobular oedema. The intestines and mesentery were slightly oedematous and there was extensive oedema in the connective tissues adjacent to the colon (Fig 3). Single 1 to 1.5 cm long ulcerative/necrotic lesions were found in both the caecum and colon. Culture and PCR testing established that the pneumonia was due to *Pasteurella multocida* and histopathology confirmed that bovine papular stomatitis (BPS) was the cause of the mucosal lesions in the upper digestive tract. Large intestinal lesions were consistent with the coronavirus detected in faeces but there were additional areas of mucosal ulceration, abundant bacterial colonies and fibrin that were suggestive of salmonellosis. Thrombosis of blood vessels is a feature of *Salmonella* spp infection and was proposed as the explanation for the oedema around the colon. *Salmonella* spp were not isolated which may have reflected recent antibiotic treatment. Increased mortality rates in artificially reared calves are often shown to be multifactorial with management factors equally if not more important than infection disease. Infection with BPS is usually a feature of ill thriven calves and the intestinal pathology was considered to be the most significant finding in this case. Investigation of ongoing losses was advised to try and build a clearer picture of the primary issue.

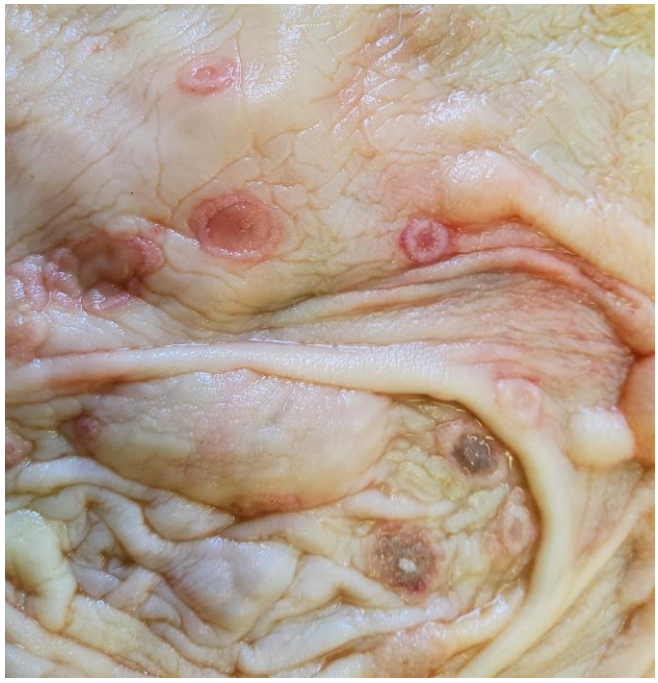

Figure 2 – Bovine papular stomatitis lesions on the rumen mucosa of a pre-weaned dairy calf

Figure 3 – Mesocolon oedema in a pre-weaned dairy calf

Respiratory tract diseases

A 30-month-old Limousin heifer at grass became lethargic and dyspnoeic and was found dead a few days later. It was one of ten heifers that had been moved to a silage aftermath three weeks previously. The lungs were heavy, and the parenchyma appeared wet with copious blood-stained fluid within the bronchi. Localised areas of emphysema were also noted. Histopathology described an acute interstitial pneumonia with hyaline membrane formation and type II pneumocyte hyperplasia but no evidence of viral infection. Taken together with the history these findings were consistent with a diagnosis of fog fever. Fog fever occurs when adult animals are moved from poor to better quality grazing. Tryptophan from the grass is metabolised to 3-methylindole within the rumen and then absorbed leading to acute destruction of lung cells. Fog fever is an important differential diagnosis for respiratory disease in adult cattle during autumn and it is important to distinguish it from bacterial or parasitic pneumonia.

SMALL RUMINANTS

Toxic conditions

A lowland flock reported a 6 per cent incidence of photosensitisation with cases only seen in Texel cross lambs. Biochemistry revealed variable increases in GGT suggesting a hepatogenous aetiology, and hypoalbuminaemia was evident in chronic cases. Inspection of the field failed to detect any potentially significant plants and two affected animals were euthanased for postmortem examination. Liver histopathology revealed a severe chronic hepatopathy with hepatocellular pleomorphism, portal fibrosis and bile duct proliferation consistent with chronic copper toxicity. High liver (37,800 µmol/kg and 27,500 µmol/kg DM; reference range 314 – 7850 umol/kg DM) and kidney copper levels (1850 µmol/kg and 1520 µmol/kg DM; reference range <787 umol/kg DM) were confirmed in both lambs. The source of copper was 2g copper boluses intended for sheep over 25 kg in weight. These had been administered in early June when the youngest lambs were only four weeks-of-age. Five deaths that occurred shortly afterwards were not investigated and may have been due to copper toxicity. Pre-weaned animals are efficient at absorbing copper and longer-term consequences in this case included poor growth rates and photosensitisation associated with excessively high liver copper levels.

Parasitic diseases

A hill flock established via the purchase of six-year-old draft Swaledale ewes over an eight-year period reported an annual ewe mortality rate of 20 per cent. Only a quarter of the ewes were reported to be in good enough body condition to allow them to return to the hill after weaning. Despite this, lamb mortality and growth rates were not a cause for concern. Four, seven to nine-year-old ewes with a history of ill thrift were euthanased for investigation of the problem. Postmortem examination detected *Dictyocaulus filaria* worms in the bronchi in all four which is often an indication that there is a concurrent gastrointestinal worm burden. This proved to be the case with strongyle egg counts ranging from 500 to 3250 eggs per gram (epg). Histopathology confirmed severe gastrointestinal parasitism in all cases. One ewe tested positive for Johne's disease, but no other evidence of iceberg diseases was detected. Parasitic gastroenteritis should be included in the list of differential diagnoses when investigating ill thrift in hill ewes. Other causes of ill thrift must be excluded as high worm burdens can be present contemporaneously with other disease issues.

Alimentary tract disorders

A five-month-old Texel cross lamb from a group of 60 ewes and lambs was found dead and submitted for postmortem examination. The group had been wormed with a benzimidazole drench nine days earlier and the shepherd was concerned about possible liver fluke infection. The conjunctivae appeared white, and the carcase was pale. A 1 cm diameter circular hole was found in the wall of the right pharynx with haemorrhage and necrosis of the underlying tissues. A large amount of clotted blood was present in the rumen giving a final diagnosis of fatal haemorrhage secondary to a dosing gun injury. No evidence of liver fluke was found but acute fasciolosis would be a differential diagnosis for anaemia at this time of year. This age group of lambs can be used as sentinels for infection with *Fasciola*

hepatica in autumn and winter. During September and October 57 submissions comprising 361 individual samples were tested in the *F hepatica* serum antibody ELISA. Only seven positive results (1.9 per cent) spread over five submissions were found indicating low liver fluke challenge on the majority of farms that carried out screening.

Nervous system disorders

Pyrexia, trembling and deaths started in a group of cross ewes and lambs four days following a move to new grazing. One carcase was submitted for postmortem examination and several ticks were noted. Additional findings included pericardial and pleural effusions and splenomegaly. Bacterial cultures were unrewarding, and *Clostridial perfringens epsilon* toxin was not detected. Neuropathology described meningoencephalitis consistent with louping ill and characterized by neuronecrosis and neurophagia. Disease usually results after an incubation period of between 8 and 13 days.[1] This indicates that sheep became infected on the original pasture prior to the move.

BIRDS

Pigeons

A three-month-old racing pigeon was submitted to investigate ill health in a group of 50 young birds where five individuals had become lethargic with loose faeces. They had all had been treated with an injectable antibiotic plus an oral anti-coccidial, and this was the only bird to have died. The group had received a paramyxovirus vaccine and were reported to be in good condition. Postmortem examination confirmed that the bird had been eating prior to death but the cause of death was not clear. Histopathological examination of tissue from the bursa of Fabricius revealed an acute to subacute bursitis with lymphocyte depletion, follicular cysts, necrosis and intranuclear/intracytoplasmic inclusion bodies consistent with circovirus infection. There was additional acute focal pneumonia with mixed bacterial infection. Pigeon circovirus infection causes immunosuppression increasing susceptibility to a range of infections. The virus is shed in faeces and saliva and birds up to four months-of-age are most susceptible. Control relies on good hygiene and biosecurity plus treatment of secondary infections.

References:

1 **Jeffries CL, Mansfield KL, Phipps LP, *et al.* Louping ill virus: an endemic tick-borne disease of Great Britain. J Gen Virol 2014;95(5):1005-1014**