OVERVIEW

- Losses due to type 1 ostertagiosis in dairy youngstock at grass.
- Outbreaks of autumn nematodirosis in lambs.
- Deaths due to Yersinia pseudotuberculosis septicaemia in turkey poults.

GENERAL INTRODUCTION

The mean temperature for September was 0.4 °C below the 1981 to 2010 average. It was a wet month in the northwest with many places having over 150 per cent of average rainfall, but much drier in Aberdeenshire. Overall, rainfall was 128 per cent of average and sunshine was 99 per cent of average.

DISEASE ALERTS

The following conditions were reported by SRUC VS disease surveillance centres in January 2018. Given similar climatic and production conditions, they could also be important this year.

- **Rhododendron toxicity in sheep**

  This is a risk when sheep inadvertently gain entry to woodlands and gardens; or following snowfall when access to grass is reduced. Affected sheep display abdominal pain, vomiting, bruxism, and ataxia, before progressing to recumbency and death. Lesions of secondary pasteurella pneumonia may be found on postmortem examination so it is important to check the rumen content for the typical dark green glossy leaves which confirm the diagnosis. Ingestion of the shrub Pieris japonica produces similar symptoms as it also contains the polycyclic diterpenes that are responsible for toxicity.

- **Cerebrocortical necrosis in store lambs**

  See review article in body of report.

CATTLE

**Nutritional and metabolic disorders**

A group of 40 cows with calves at foot was gathered in order for the cows to be scanned. All the animals appeared healthy when returned to the field but a well grown five-month-old simmental cross calf was found dead the next morning. The carcase was submitted to Dumfries for postmortem examination where the most significant finding was sticky grey rumen content with the consistency of wet cement. The rumen pH of 5.2 was considered significant as 48 hours had elapsed between the animal being found dead and the postmortem being carried out. Ad-lib creep feed was available and it was suspected that this calf had eaten an excessive amount on being returned to the field.

**Parasitic diseases**

Type 1 ostertagiosis was diagnosed as the cause of death of dairy youngstock on three farms. The first case involved a group of 23-month-old holstein heifers that had failed to thrive since returning from wintering in spring. Coughing, but no diarrhoea, was reported and the second animal to die was submitted for investigation. Dictyocaulus viviparus worms up to 4.3 cm in length were found in the airways, and lesions consistent with ostertagiosis were detected in the abomasum. The strongyle egg count was 400 epg despite treatment with a pour on product containing moxidectin ten days earlier. The second case involved a group of eight-month-old calves experiencing diarrhoea and weight loss. The carcase of the first animal to die was examined and severe ostertagiosis was diagnosed. The strongyle egg count was 350 epg and histopathology confirmed severe mucosal lesions including nematodes with morphology consistent with Ostertagia within the abomasal glands. Small numbers of dilated glands contained purulent exudate consistent with secondary bacterial infection. This group had been treated with a pour on product containing moxidectin five weeks earlier. The third herd reported the loss of eight, five-month-old calves from a group of 250, and a further 30 were ill. Following the initial deaths the group had been moved to a new field and the worst affected calves treated for suspected coccidiosis. Four carcases were examined and ostertagiosis with superimposed purulent abomasitis consistent with secondary bacterial infection was confirmed in all cases.

**Figure 1a – Parasitic abomasitis due to ostertagiosis complicated by secondary bacterial infection**
Part-funded by the Scottish Government as part of its Public Good Veterinary Advice Services

Figure 1b - Abomasum (fundus), 7 month old Holstein heifer, ostertagiosis and secondary purulent abomasitis. Extensive elongation and mucous metaplasia of glands associated with nematode profiles in mucosa (black arrows) and in the abomasal lumen (black arrowhead). The mucosal nematodes are located within a degenerating gland containing purulent exudate (blue arrows). The exudate extends into the subjacent oedematous submucosa (blue arrowhead).

This group had been treated with a pour on product containing doramectin five weeks earlier and strongyle egg counts ranged from 800 to 2350 epg. The findings confirmed treatment failure following the use of pour on products in all three herds and further investigation was recommended. Ideally faecal samples should be collected two weeks post treatment to check that pour on treatments have been effective. It was postulated that the dry weather in the first half of the grazing season had reduced worm challenge slowing the development of immunity.

Generalised and systemic conditions

The third, three-month-old calf to die from a group of 29 over a ten day period was submitted to Inverness for postmortem examination. The carcass was very pale, the left lung was haemorrhagic, and unclotted blood was present within the pericardium. Haemorrhages were noted on the liver, kidneys, jejunal mucosa, and within the calvarium and sinuses. Bracken poisoning was suspected but bone marrow histopathology did not support this theory. Following further investigation a diagnosis of sudden death due to clostridial enterotoxaemia type D was reached based on brain swelling including tentorial notching of the occipital cortices associated with perivascular protein-rich fluid exudation on neuropathology, and the detection of epsilon toxin in small intestinal content.

The most significant finding on postmortem examination of a high yielding, four-year-old Holstein cow was a large blood clot ventral to the cerebellum.

Figure 2 – Brain stem haemorrhage in a case of clostridial enterotoxaemia type D in a dairy cow.

Further examination of the brain revealed multiple foci of malacia and associated haemorrhage within the thalamus and extending into the rostral mid brain. Histopathology confirmed a necrotising encephalopathy associated with perivascular protein-rich oedema fluid accumulation that strongly supported enterotoxaemia due to Clostridium perfringens type D as the cause of death. Prominent haemorrhages, such as surrounding the brainstem in adult cattle, and rarely more generalised in younger cattle and sheep, are features of a small proportion of Clostridium perfringens type D enterotoxaemia cases. It is not clear if this is a consequence of epsilon toxin induced vascular injury or a secondary complication.

Reproductive tract conditions

A dairy herd of 210 cows calving all year round reported a single abortion at six months gestation. The herd were grazed during the day and housed at night when they had access to a total mixed ration consisting of silage, draff, barley, maize, dark grains and soya. The foetus was submitted to Ayr along with the placenta which was grossly thickened with areas of purulent debris. Rhizopus arrhizus was recovered from the placenta only with cultures of foetal stomach contents remaining sterile. Histopathology confirmed a severe, multifocal, suppurrative, necrotising placentitis and identified fungal hyphae in association with the lesions. This demonstrated that the isolate was significant as the cause of the placentitis and not a contaminant from the environment.
Nervous system disorders

Ayr diagnosed louping ill in a one-month-old suckled calf that was reported to be pyrexic, ataxic and trembling. Louping ill serology was positive with a predominance of IgM confirming recent infection. The same diagnosis was reached in an eight-year-old suckler cow exhibiting ataxia and hyperaesthesia on a second holding. Additionally serum magnesium was below reference range in this case at 0.3mmol/l (reference range 0.8 to 2 mmol/l), and it was suggested that this was a consequence of anorexia related to the louping ill.

SMALL RUMINANTS

Nutritional and metabolic disorders

A three-year-old North Ronaldsay ewe died suddenly and a postmortem examination was carried out on farm. Diffuse petechiation, particularly within the thoracic cavity, was reported as the main finding. Histological examination of the liver revealed a severe necrotising hepatopathy and chronic portal lesions with pigment accumulation consistent with hypercuprosis that was confirmed on liver analysis (27,200 µmol copper/kg dry matter (DM); reference range 314 to 7,800 µmol/kg DM). A food safety investigation was not required on the basis of this result. North Ronaldsay sheep traditionally subsist by grazing seaweed on the foreshore of their native island and have developed an enhanced ability to absorb copper. This adaptation to a low copper environment makes them highly susceptible to chronic copper toxicity when the breed is managed on grass.1

Parasitic diseases

The carcase of a four-month-old Texel lamb was submitted for investigation following the death of twelve lambs from a group of 150 over a two week period. The group had recently returned from summer grazing and had been treated with a benzimidazole drench two weeks earlier. Affected animals were reported to be dull, diarrhoeic, and losing condition. Parasitic gastroenteritis was confirmed as the cause of death following the recovery of 4,000 Teladorsagia circumcincta worms from the abomasum and 10,300 Nematodirus battus from the small intestine. This confirmed treatment failure and further investigation was advised. 37 outbreaks of nematodiosis were recorded in June 2018, which is when the disease traditionally peaks in Scotland; with a further seven confirmed in September. This is in contrast to the situation in Northern Ireland where diagnoses of nematodiosis are reported to peak in autumn.2

Alimentary tract disorders

A group of 257 spring born lambs moved to winter grazing following treatment with a benzimidazole drench. An EID bolus was also administered to the 157 ewe lambs intended to be retained for breeding. Two weeks later 10 of the ewe lambs were found dead within a 48 hour period and a further six presented with ataxia and circling progressing to recumbency. Postmortem examination of three lambs was carried out at Aberdeen with similar findings in each case. Mucosal defects were found in the soft palate that communicated with large abscesses within the soft tissues dorsally. In two cases purulent exudate extended into the atlanto-occipital joint and in one lamb necrotic debris was adherent to the spinal cord in this area. Fusobacterium necrophorum and Trueperella pyogenes were isolated from abscess and brain swabs. The history, clinical presentation and gross findings were all consistent with dosing gun injuries with subsequent damage to the cranial cervical spine and bacterial meningitis. Inspection of the dosing equipment used was advised.

Reproductive tract conditions

An organism of the Corynebacterium renale group was isolated from a swab collected from a chronic discharging preputial lesion on a three-year-old Suffolk tup. These organisms produce urease that breaks down the urea in urine to ammonia. The latter is irritant to epithelia and induces ulcerative penile and preputial lesions. High protein diets may predispose to the condition therefore a review of nutrition can be of value in addition to antimicrobial treatment.

Musculo-Skeletal conditions

Perth diagnosed bacterial myositis and peritonitis in a four-month-old male crossbred lamb that was one of sixteen to die over a two day period. Approximately 2 litres of red cloudy fluid was found in the abdominal cavity and there was a pale raised area on the peritoneal surface corresponding to a region of haemorrhage and fibrin between the overlying muscle layers of the left flank. Histopathology findings were consistent with severe bacterial myositis and secondary peritonitis, however the changes were not consistent with myositis due to Clostridium chauveoi or Histophilus somni. A large mixed bacterial population was evident and considered to be consistent with environmental contamination by opportunistic bacteria secondary to penetration of the abdominal wall. On farm postmortem examination of a second animal also revealed peritonitis. A clostridial vaccine had been administered four days previously and suboptimal injection technique was suspected to be the cause of the losses.

Renal diseases
A Lleyn cross gimmer was submitted to Dumfries for investigation of ill thrift affecting this age group of sheep. Widespread petechiation was detected subcutaneously and on the omentum and mesentry. The kidneys were dark brown but there was no other evidence to suggest a haemolytic crisis. Histopathology revealed generalised chronic glomerulitis, severe protein-losing nephropathy and tubular haemosiderosis raising the possibility of an immune mediated glomerular lesions and secondary tubular changes, possibly accompanied by chronic intravascular haemolysis or coagulopathy. This suggested that this was a one off case and not representative of the group. The only finding potentially relevant to the rest of the group was footrot and blowfly strike affecting three of the four feet.

PIGS

Generalised systemic diseases

A nine-year-old male pig that had been treated with meloxicam for five years became anorexic and dyspnoea and died despite antibiotic treatment. Postmortem examination findings included congested, oedematous lungs and a serous pericardial effusion. Periarticular fibrosis and osteophyte production affected both elbows which, together with erosion of the medial cartilage, explained the need for long term therapy with a non steroidal anti-inflammatory drug. Bacteriology and neuropathology were unremarkable but histopathology revealed lesions consistent with disseminated intravascular coagulation (DIC). Long-term NSAID use is recognised as a cause of DIC in humans and it was postulated that pigs could be similarly affected. Three, 14-week-old Norfolk black turkeys died over a ten day period and around 10 per cent of the flock were reported to be lame. Dramatic pulmonary congestion and oedema was found on post mortem examination along with hepatosplenomegaly, congestion of the subcutaneous tissues, and haemorrhagic ascitic fluid. Yersinia pseudotuberculosis was isolated in pure growth from the lung, liver and spleen, confirming septicaemia as the cause of death. Y. pseudotuberculosis has previously been reported as a cause of septicaemia and osteomyelitis in turkeys. Hepatic miliary foci which are commonly found in cases of yersiniosis were not detected in this case. Good hygiene practices and preventing contact with the faeces of wild birds can assist in preventing outbreaks.

Alimentary tract disorders

Three recently weaned piglets were found dead one morning on a breeding to finishing indoor unit. The heating in the building had been reduced from 28 to 24 degrees and the piglets had been seen huddling for warmth. Postmortem examination found evidence of enteritis and a haemolytic crisis. Histopathology revealed generalised chronic glomerulitis, severe protein-losing nephropathy and tubular haemosiderosis raising the possibility of an immune mediated glomerular lesions and secondary tubular changes, possibly accompanied by chronic intravascular haemolysis or coagulopathy. This suggested that this was a one off case and not representative of the group. The only finding potentially relevant to the rest of the group was footrot and blowfly strike affecting three of the four feet.

BIRDS

Poultry

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Poultry

Two hens and a turkey exhibiting weakness and respiratory signs were submitted to Inverness for postmortem examination. One hen had a grossly enlarged abdomen due to an accumulation of yellow, translucent ascitic fluid. The ovary was enlarged and the oviduct obstructed by a four centimetre diameter soft tissue mass. Multiple grey foci were noted in the mesentery and serosa of the intestinal tract. Histopathology confirmed the mass to be an adenocarcinoma of the oviduct. The other two birds were both very thin with multiple pale caseous masses throughout the viscera. Acid-alcohol fast bacilli were detected on Ziehl-Neelsen stained smears from the masses, consistent with a diagnosis of avian tuberculosis.

MISCELLANEOUS

A 13-month-old deer with a three week history of weight loss and scour was euthanased for investigation of the problem. It was the only animal affected from a group of 70 purchased in May. Tissues were submitted to St. Boswells for examination and the small intestine was found to be thickened and corrugated with dark mucosa. The mesenteric lymph nodes were greatly enlarged with firm white centres but no evidence of necrosis or calcification. Smears from both the intestines and lymph nodes were positive for acid fast bacilli typical of Mycobacterium avium paratuberculosis and histopathology confirmed mycobacterial enteritis and lymphadenitis with features typical of Johne's disease.

Exotic animals

A two-day-old bison calf from a wildlife park was found dead despite being appearing well and on its feet the previous day. Postmortem examination revealed a
fractured rib and evidence of an ascending umbilical infection from which *Escherichia coli* was cultured. It was suggested that, if the fracture occurred during parturition, pain may have increased the time taken to stand predisposing to navel infection and preventing the timely intake of colostrum.

References:
1 Haywood S, Simpson DM, Ross G, Beynon RJ. The greater susceptibility of North Ronaldsay sheep compared with Cambridge sheep to copper induced oxidative stress, mitochondrial damage and hepatic stellate cell activation. *J Com Path* 2005; 133:114-27
4 Wallner-Pendleton E, Cooper G. Several outbreaks of *Yersinia pseudotuberculosis* in Californian turkey flocks. *Av Dis* 1983; 27(2):524-6
Cerebrocortical Necrosis in Ruminants

Cerebrocortical necrosis (CCN) refers to a clinical condition in ruminants associated with thiamine deficiency or, relatively uncommonly in the UK, excessive sulphur intake. While all ages can be affected, the condition is most commonly found in sheep under one year, and cattle from six to 18 months of age. The disease is often sporadic but outbreaks can occur. Onset of disease may follow a change to pasture or feed in the preceding weeks, and an association with high-carbohydrate low-forage diets has been made.

Signs are consistent, with hyperaesthesia, central blindness and a star-gazing posture being the first evidence of a problem, progressing to ataxia and recumbency within 12 to 48 hours. Facial twitching, cerebellar tremors, nystagmus and opisthotonus are often seen, and affected animals may develop seizures when handled.

Postmortem examination findings may be subtle, and in the early stages consist of cerebrocortical swelling with flattening of affected gyri and sometimes cerebellar coning. The brain may have a yellowish appearance and auto-fluoresce on exposure to UV light. More chronic cases may have visible separation of the friable necrotic grey matter from the underlying white matter cores. The presence of auto-fluorescence, the histological features, and their distribution often distinguish this condition from other causes of cerebrocortical necrosis such as lead intoxication or water deprivation associated sodium toxicity.

The aetiology of CCN in ruminants is not yet fully understood. Thiamine is a critical cofactor in the carbohydrate metabolism which sustains the brain, and energy depletion in the cerebral tissue is responsible for the necrosis. Several mechanisms have been postulated including overgrowth of thiaminase producing rumen flora, ingestion of feed rich in thiaminases, a failure of thiamine absorption, and excessive thiamine excretion in faeces. Excessive dietary sulphur intake (e.g. cruciferous plants) has also been implicated. Sulphur cleaves thiamine, but it may also exert an effect by direct reduction of thiamine production in the rumen, and by reducing availability of thiamine phosphate esters which are critical for cerebral metabolism.

Treatment consists of parenteral administration of vitamin B1, repeated over three days. In severely or chronically affected individuals treatment may be unrewarding and euthanasia is preferable, but if treated promptly many animals will make a rapid and full recovery.

In sheep, while this condition is diagnosed year-round, SRUC data shows a seasonal distribution, with a greater number of cases occurring during the summer and autumn months. This is consistent with the data on age at diagnosis, which demonstrates peaks of disease around four months, and again at seven months of age. This may reflect factors such as parasitism, nutritional pressures or altered rumen flora, for example, at weaning.

This places CCN as an important differential diagnosis in cases with central nervous system signs in sheep of these ages, since other common conditions of similar presentation (particularly louping ill encephalitis) may have a poorer prognosis.
CCN diagnoses by month
SRUC 2008-2018 (sheep)

Diagnosis of CCN by age
SRUC 2008-2018
Cerebrocortical necrosis in a store lamb. The cut surfaces of the fixed brain reveal well defined areas of faint yellowish discoloration of the cerebral grey matter (A) that autofluoresce when viewed under ultraviolet light (365nm) (B).