AHVLA Disease Surveillance Report
March 2014

- Necrotising pleuropneumonia due to *Mycoplasma bovis* in adult cows
- Copper toxicosis in sheep
- Porcine sapelovirus causing nervous disease
- Avian tuberculosis in a backyard flock

**CATTLE**

*Musculoskeletal disease*

**Salmonella Dublin cervical osteomyelitis**

*Salmonella* Dublin infection was diagnosed at Carmarthen as the cause of osteomyelitis of the distal cervical vertebrae of a 10-week-old Holstein Friesian heifer calf. The infection had resulted in purulent infection of the intervertebral space between the sixth and seventh cervical vertebrae. The affected calf was initially reported to be tender on its feet, becoming slow to rise. It subsequently became recumbent and was euthanased. Postmortem examination confirmed the presence of necrotic roughening of the apposing vertebral surfaces and a purulent exudate, and *S* Dublin was isolated in culture (figure 1). Spinal osteomyelitis is an unusual presentation of infection by *S* Dublin in calves and is only seen sporadically. Fourteen cases were reviewed in Ireland by Healy and others (1997) who reported an age range of 2 to 9 weeks, no breed or sex predisposition and a range in time of clinical presentation from 5 days to 8 weeks with a mean of 20 days. The calves exhibited difficulty in rising with a tendency to knuckle or kneel on the forelimbs, and in more than half of the cases pain was elicited on manipulation of the neck. Unusually, four animals were successfully treated with the remaining ten calves euthanased.

**Respiratory disease**

**Necrotising pleuropneumonia caused by *Mycoplasma bovis***

A severe necrotising pleuropneumonia was identified postmortem in an adult cow at Preston, which prompted reporting the finding as a possible case of contagious bovine pleuropneumonia (CBPP). The animal had been
euthanased after a two week history of pyrexia, weight loss and progressive respiratory signs. Eight of the dairy herd of 350 were reported to have been affected, with two deaths. There was approximately 10 litres of yellow to red fluid containing large fibrin clots within the thorax. The cranioventral areas of the lungs and the cranial parietal pleura were covered in thick yellow fibrin with haemorrhages. The lung lobes were adhesed to each other and to the adjacent pleura and the pericardial sac. The cranial and middle lung lobes were dark red and firm and there was a large amount of yellow exudate in the interlobular septa exhibiting a ’marbling’ pattern (figure 2)<>, with subpleural and interlobular emphysema within the caudal lobes. There was also fibrinous thickening of the pericardium and epicardium. As the severe pleuropneumonia and pericarditis resembled that of CBPP suspicion of possible notifiable disease was reported to Animal Health colleagues and a disease investigation was carried out. Laboratory testing confirmed Mycoplasma bovis and Trueperella pyogenes infection, and histopathology revealed a severe necrotising pleuropneumonia which demonstrated extensive labelling for Mycoplasma bovis using immunohistochemistry. There was no evidence of Mycoplasma mycoides subspecies mycoides, the cause of CBPP.

**Interstitial pneumonia in housed fattening bulls**

Interstitial pneumonia was diagnosed postmortem at Shrewsbury in a group of 60 fattening bulls. An acute onset of signs of pneumonia was described in 10 of the animals with two of the bulls dying. The outbreak occurred in the oldest group of animals on the premises, with younger calves, which were fed the same diet, being unaffected. A postmortem examination was carried out by the practitioner who described ‘diffuse pneumonic pathology’ and submitted samples for diagnostic testing. No viruses were detected by fluorescent antibody testing and histopathology confirmed a severe subacute interstitial pneumonia. The pathology was suggestive of a haematogenous rather than an aerogenous insult. This indicated the potential for pneumotoxic injury caused by the generation of 3-methyl indole, ingestion of a known intoxicant such as mouldy sweet potatoes, or an idiopathic cause. Outbreaks have been reported in feedlot cattle in North America but sporadic cases are also diagnosed in housed young stock in this country. Contributing factors appear to be a dusty environment, dietary components (such as mouldy sweet potatoes) and the feeding of growth promoters (although those recognised in the USA are not licensed in the UK). A ‘rumen modifier’ was being added to the diet of the animals in this case, although whether it contributed to the outbreak, is uncertain.

**Reproductive disease**

**Hyposelenaemia in heifers**

Hyposelenaemia was diagnosed in a group of 32 primiparous heifers. Five of the seven heifers which had calved in the last month were reported to have had calves which were alive but weak and with flaccid muscles, and died within an hour of birth. A high proportion of the heifers also retained placentae. The five heifers were blood sampled and had profoundly low
selenium status with glutathione-peroxidase activities of between 6.1 and 9.6 U/ml RBCs (AHVLA consider adequate values >30 U/ml RBCs). Low blood copper concentrations were also found in four of the five animals (plasma copper concentrations 2.4 to 6.7 μmol/l (reference interval 9-19 μmol/l)).

SMALL RUMINANTS

Enteric diseases

Triclabendazole resistance
Chronic liver fluke and potential resistance to triclabendazole continues to be of concern. The RVC and Preston both reported instances where chronic fasciolosis was observed at postmortem examination where sheep had received recent or repeated treatment with triclabendazole over the preceding weeks. Further investigation of inefficacy of triclabendazole would include a Faecal Egg Count Reduction Test and review of the grazing practices, stock management and flukicide use on the farm.

AHVLA contributed to the SCOPS parasite forecasting when early cases of Nematodirosis were reported by Regional Laboratories. Parasite alerts and forecasts are available from www.scops.org.uk and www.Nadis.org.uk

Respiratory diseases

Maedi-visna
Starcross assisted in the investigation of thin coughing ewes in a flock of 500. The flock did not breed its own replacements; ewe lambs were bought in late summer. Over recent years between 5 and ten thin and constantly coughing ewes were observed at housing each year in November. The coughing apparently responded to a treatment of anthelmintic but there was no weight gain. This year a postmortem examination was performed by the farm's veterinary surgeon on a sacrificed ewe. Lung and associated lymph nodes and a blood sample were submitted to AHVLA for examination. Histological examination of the lung tissue revealed a diffuse interstitial pneumonia and a marked reactive lymphadenosis, consistent with a diagnosis of Maedi-visna. The blood sample gave a weak positive result in the M-V agar gel immunodiffusion test (AGIDT). This is an infrequent diagnosis in the southwest.
Reproductive diseases

Abortion
The most common abortion diagnoses made by AHVLA from January to March 2014 are shown in figure 3 below. Other diagnoses include *Trueperella pyogenes*, dystocia, Schmallenberg and Yersinia.

![Abortion diagnoses in sheep 2013 and 2014](image)

It is unclear why there has been an increase in the diagnoses of Enzootic abortion of ewes (EAE) in 2014; nevertheless, it does stress the importance of flock health planning in assessing biosecurity and preventing the introduction of infectious disease onto farms.

Skin
Starcross diagnosed caseous lymphadenitis (CLA) after *Corynebacterium pseudotuberculosis* was isolated in pure culture from aspirated pus from swollen head lymph nodes of a nanny goat. A number of nannies due to begin kidding had developed swollen parotid and submandibular glands. Mixed flora, including *T pyogenes*, was cultured from an aspirate from a second nanny. Blood samples were tested using the CLA ELISA. The CLA culture positive goat was also ELISA positive, whilst the blood sample from the goat with mixed culture was ELISA negative. The CLA ELISA test has not yet been fully validated for use in goats. In sheep the specificity can be calculated as 98% and the sensitivity at 87%. In CLA affected flocks/herds clinical signs and bacteriology should take precedence over serology in the diagnosis of infection. It is intended that in this particular herd the blood test will be used for screening the goats to facilitate changes to management practices.
Other diseases
Copper toxicosis was diagnosed on three occasions during April. At Carmarthen a farmer reported a number of stillbirths and late abortions in his flock and some ewes had died following stillbirth. Ewes were fed silage and concentrate and were fully vaccinated. At postmortem examination of a two-year-old Suffolk cross ewe (one of three to die in a flock of 60 ewes), the carcase was jaundiced and the liver bright orange in colour. The liver copper level was 15,391 µmol/kg DM (reference interval 300-8000 µmol/kg dry matter (DM)). The owner was advised to evaluate the complete diet for copper levels. At Penrith, a liver copper level of 15507 µmol/kg DM in an adult Blue-faced Leicester ewe was also recorded. The carcase appeared jaundiced when examined by the farm’s veterinary surgeon. The sheep had recently been dosed with a copper-containing bolus.

The physiological state of late pregnancy can be a risk factor for copper toxicosis as stress and/or reduced food intake can result in liver pathology and the release of copper into the blood stream during fat mobilization.

At Sutton Bonington chronic copper intoxication with an acute presentation was diagnosed in a Texel ram lamb which had had vague malaise for two days prior to death. Blood analysis for copper concentration was 141.0 µmol/l (reference interval 9-19 µmol/l); concentrations >50-70 µmol/l are sufficient evidence to diagnose copper poisoning.

Postmortem examination by the PVS confirmed the typical gross pathology consistent with a haemolytic crisis. Six Texel ram lambs had died over a week with similar signs. The ram lambs were being fed corn ad libitum with a copper concentration of 15-19ppm and some pea straw. The week prior to the disease onset the pea straw had been changed to hay. Interestingly, the rams had received one week’s worth of calf concentrate in November 2013. This was considered critical in the aetiology by copper loading the liver. The high plane of continuing nutrition would have maintained high liver copper concentrations and the forage change to hay had possibly had an impact on the liver, perhaps due to a short period of stress and inappetance leading to fat mobilisation thereby precipitating the haemolytic crisis. Advice was given regarding minimising stress and discussing with a nutritionist the short term introduction of in-feed molybdenum to act as a copper antagonist.

PIGS
Respiratory Diseases
Finisher deaths due to septicaemic pasteurellosis
Pasteurellosis was diagnosed by Bury St Edmunds when two fresh plucks were submitted to investigate widespread respiratory disease in 18-week-old pigs on an indoor finishing unit. Clinical signs were reported to have been ongoing for more than two weeks and the pigs had been treated with in-water antimicrobial. There was cranioventral pulmonary consolidation in both plucks and a fibrinous pericarditis. Pasteurella multocida was isolated from multiple sites. Histopathology was consistent with a bacterial pulmonary infection and there was no convincing histological evidence of either viral or mycoplasmal involvement; PCRs for swine influenza and porcine reproductive and
respiratory syndrome (PRRSV) did not detect these viruses. There was also no evidence of porcine circovirus (PCV)-2-associated disease. As respiratory disease had been ongoing for several weeks prior to submission, earlier involvement of viruses, in particular swine influenza, could not be ruled out. Bloods were submitted from the cohort of pigs at the same time as the plucks and swine influenza serology pointed to previous exposure to pandemic H1N1 2009. The fibrinous pericarditis associated with *P. multocida* infection, and certain features of the histopathology, suggested that the pasteurellosis was septicaemic and could account for the deaths of the pigs.

### Nervous Disease

**Unusual outbreak of oedema disease causing ataxia**

Oedema disease was diagnosed at Langford as the cause of nervous signs mainly consisting of ataxia, weakness and recumbency as well as malaise and anorexia. The small herd comprised three groups of pigs; 45 recently weaned, 29 weighing around 17kg, and 14 older pigs weighing between 70 and 130kg. The pigs were bought in from different units at the same time. At least 14 pigs across all three groups died over about three weeks. Postmortem findings were non-specific with no typical oedema of the eyelids, gastric mucosa or mesocolon visible. Pulmonary oedema and increased peritoneal fluid with scant fibrin stranding was found in some of the six pigs submitted. Initially *Streptococcus suis* meningitis was suspected but no pathogens were isolated from the meninges. Histopathology supported the possibility of oedema disease when a fibrinoid vasculopathy was detected in brain and lymph node and *E. coli* serotype O139:K82 (strain E4) was isolated in pure culture from the small intestines of subsequent submissions confirming the cause of disease. Amongst the submissions was one pig with diarrhoea due to a different manifestation of the E4 *E. coli* infection. This pig had a haemorrhagic enteropathy and small intestinal thickening and corrugation not unlike porcine intestinal adenomatosis. E4 *E. coli* was isolated in pure growth from the intestines.

This outbreak was unusual in that oedema was not evident before, or at, postmortem examination, possibly because some pigs were euthanased and lesions had not fully developed. Oedema disease most commonly affects pigs one to four weeks after weaning. Here much older animals were also affected, which may have been due to later challenge following their transport and introduction into an environment contaminated with the oedema disease-producing strain of *E. coli*. Control can be difficult and centres on hygiene, controlling feed levels, high quality nutrition and sometimes a reduction in stocking rate.

**Sapelovirus outbreak causing nervous disease**

Unusual nervous signs and deaths in post-weaned pigs prompted the submission of initially a fixed brain and then affected pigs to Thirsk from a 400-sow farrow-to-finish unit. This unit also had problems with *Streptococcus suis* meningitis which usually responded to antimicrobial treatment. However, prior to submission, one to two pigs from the same pen in each batch of 200 pigs weaned fortnightly presented with different nervous signs that did not
respond to treatment. Pigs were affected approximately two weeks after weaning and were reported to be going off their front legs and walking backwards. They progressively worsened showing nystagmus, then lateral recumbency, but no paddling. Gross postmortem examination was unremarkable but brain histopathology revealed a severe nonsuppurative and necrotising polioencephalitis and panencephalitis. These findings were indicative of a neurotropic viral infection and were very similar to those in a recent outbreak of porcine sapelovirus in the Bury St Edmunds region. Immunohistochemistry and PCR for porcine sapelovirus confirmed this diagnosis and ruled out teschovirus involvement. Affected pigs did not respond to nursing and it was advised that cases presenting with signs typical of the sapelovirus be culled promptly if they showed no response to antimicrobial treatment. Interestingly, this is the second outbreak of sapelovirus in 2014. The most recent case diagnosed by AHVL was in 2008.

*Musculo-skeletal Disease*

**Hindlimb weakness due to severe polyarthritis in finishers**
Following the diagnosis of porcine sapelovirus earlier in rear on a nursery-finisher unit, several pigs around 14-weeks-old were reported to be showing hind limb weakness and reluctance to rise. Some of these had died and there had been a poor response to antimicrobial treatment. There was concern that this was a further manifestation of porcine sapelovirus. One typical case was euthanased and submitted to Bury St Edmunds. A severe fibrinosuppurative polyarthritis (figure 4) was present affecting all joints including the atlanto-occipital joint. No bacterial pathogens were isolated and Mycoplasma hyosynoviae was not detected by PCR. The pig had been treated prior to submission and this may have affected culture results; the detection of polyarthritis explained the clinical signs and ruled out the involvement of porcine sapelovirus.

*HOIDS*

**Commercial Layers**

*Coccidiosis*
Acute caecal coccidiosis was diagnosed in 20-week-old layers submitted to investigate a sudden rise in mortality. At postmortem examination, the carcases were found to be markedly pale, and the caecal branches were distended and filled with blood. Histopathology of the caeca revealed blood-filled luminal contents and large coccidial schizonts within sloughed pieces of autolysed mucosa consistent with those of *Eimeria tenella*. This was an unusual diagnosis as the birds were housed in an enriched colony system, but the problem was localised within a small area of the house where there was potential access to droppings from a conveyor belt.
**Spirochaetosis**

Avian intestinal spirochaetosis was seen in a submission of 34-week-old free-range layers which were experiencing a slight drop in egg production just after attaining peak production. Most of the examined carcases were out of lay. One in particular showed ovarian regression and abnormal yellow-mustard discolouration of the caecal contents from which *Brachyspira intermedia* was isolated. Spirochaetosis in layers is often a problem of young flocks in the early part of lay, where it can be associated with drops in egg production, failure to reach peak of production and failure to gain bodyweight.

**Broilers**

**Colisepticaemia and aspergillosis**

*E. coli* septicemia and fungal pneumonia were diagnosed in two houses of four-day-old chicks originating from a young parent flock and submitted to investigate a cumulative mortality of approximately 2%. Postmortem examination revealed swollen livers and spleens, occasional cases of pericarditis, inflamed navels and congested lungs with discrete pin-point creamy coloured nodules. *Aspergillus fumigatus* was isolated from the nodules and moderate growths of *E. coli* were also isolated.

**Tenosynovitis**

A diagnosis of tenosynovitis associated with avian reovirus was made in a submission of 20-day-old broilers with a history of slight increase in culling due to lameness. Postmortem examination revealed bilateral angulation/rotation of legs at the level of the hock joints, with mild swelling of the achilles and digital flexor tendons and occasional focal thickening of the pericardium. Histological examination of a range of tissues revealed a chronic lymphocytic pericarditis and a chronic lymphocytic tenosynovitis of the achilles tendons with lymphocytes, plasma cells, early germinal centre formation and localised granulocytes. A reovirus was isolated in tissue cultures from the affected tendon and heart tissues. Reoviruses are common inhabitants of the intestinal tract in birds showing no clinical signs; whilst the majority of strains are considered non-pathogenic, infections with pathogenic reovirus in broilers are on the increase, and the identification of new types has been reported (Troxler and others 2013). The pathogenic strains are mainly associated with poor, uneven growth and lameness with splayed legs and partial to complete tendon ruptures. Age-related resistance in broilers depends on the level of maternal antibodies and the virulence of field strains. This case followed a previous diagnosis made on the same premises.
**Backyard and hobby flocks**

**Mycoplasmosis**

Infectious sinusitis associated with *Mycoplasma* infection was diagnosed in two batches of birds from a hobby flock ranging between 15 to 25 weeks of age and a 4-year-old bird, with a history of respiratory signs, periorbital swelling and a drop in egg production in the older birds. The main findings at postmortem examination included conjunctival congestion and excessive mucus in the nasal passages. In the younger birds there was also evidence of poor bone mineralisation with weak bones and widening of the metaphysis of the tibiotarsal bones. Histological examination undertaken on the tracheas, conjunctivas and nasal passages revealed a lymphoplasmacytic tracheitis, conjunctivitis and rhinitis with germinal centre formation suggestive of possible *Mycoplasma* involvement. Examination by Denaturing Gradient Gel Electrophoresis (DGGE) detected several species of *Mycoplasma* including *M synoviae*, in the trachea of the younger birds, although *M gallisepticum* was not detected. In addition, a hypophosphataemic pattern of rickets was confirmed histologically in the younger birds.

**Avian tuberculosis**

Seven of a group of 18-month-old backyard layers purchased as a group of 20 pullets had died over the previous four months after becoming inappetent, losing weight and showing general malaise. One bird was submitted for postmortem examination. There were miliary white or cream-coloured foci throughout the liver and numerous larger nodules in the spleen, which was enlarged to 3 cm diameter (figure 5). Acid/alcohol-fast bacilli were detected within these lesions on histological examination and the findings were consistent with mycobacterial infection (avian TB). The cause of death of the previous six hens was unknown as none were submitted for examination.

**References**


This summary is produced by the AHVLA and is drawn from reports provided by the AHVLA laboratories at Aberystwyth, Bury St Edmunds, Carmarthen, Langford, Lasswade, Leahurst, Luddington, Newcastle, Penrith, Preston, Royal Veterinary College, Shrewsbury, Starcross, Sutton Bonington, Thirsk, Truro,
Weybridge and Winchester. AHVLA monthly reports are available online at http://www.defra.gov.uk/ahvla-en/category/publications/disease-surv/surv-reports/