PRRS in pigs
APHA Disease Surveillance Report
December 2014

- Hemlock poisoning and Tick Borne Fever in sheep
- Necrotic enteritis in laying hens

CATTLE

Systemic disease

**Lymphoma:** A lymphoma was identified by histopathological examination of the sternal bone marrow of a 16 day old calf. The animal had exhibited bleeding from injection sites, and at postmortem examination widespread haemorrhages on internal organs were described by the practitioner. On the basis of the clinical and pathological findings bovine neonatal pancytopenia (BNP) was suspected, especially as previous cases had been confirmed in the herd. However, histopathological findings of almost complete replacement of the intertrabecular architecture by sheets of cells with varying nuclear morphology and loss of haematopoietic cells and adipocytes, were consistent with neoplasia, most likely lymphoma. The possibility of enzootic bovine leucosis, a notifiable disease caused by bovine leukaemia virus, should be considered for all lymphomas in cattle; however, it is recognised as being a disease of adult cattle. In contrast, sporadic lymphomas are diagnosed in all ages of cattle and include, as in this case, a congenital form in calves.

**Black disease:** Infectious necrotic hepatitis (‘Black disease’) was the cause of death of a seven month old Aberdeen Angus calf, which was the second to die in a group of 12 in a suckler herd. Both calves had been lethargic for between 24 and 48 hours before death. The group had been housed six weeks previously but had received no anthelmintic or flukicide treatment. They had been vaccinated against clostridial myositis (‘blackleg’). Postmortem examination revealed extensive ecchymotic haemorrhages in the subcutaneous tissue of the neck and over the thorax. An irregular area of pale necrosis with a haemorrhagic border, typical of Black disease, was present in the liver (figure 1). This disease is often triggered by migrating liver fluke, and treatment with a product effective against immature *Fasciola hepatica* was recommended for the remaining animals in the group.
Vaccination against *Clostridium novyi* can also be employed on farms where the disease has been identified.

Fig 1. Necrotic area with a haemorrhagic border in the liver of a seven-month-old suckler calf caused by *Clostridium novyi* infection (Black disease).

**Nervous disease**

**Copper toxicity** was diagnosed in a three month old dairy cross calf. It had developed acute onset fitting, which was unresponsive to attempted sedation, and died. No specific pathology was identified on postmortem examination other than haemorrhages over the heart and patchy lung consolidation. Attempted bacterial culture of the brain proved sterile and there was no evidence of hypomagnesaemia. Histopathological examination of the brain revealed a vacuolar encephalopathy which was consistent with that seen in animals with hepatic disease. The liver showed biliary replication and portal fibrosis associated with much increased copper accumulation. Biochemical analysis of the liver confirmed a copper concentration of 15,914 µmol/kg (APHA reference interval 300-8000 µmol/kg). The hepatic pathology was considered to be very similar to a previously reported case in a calf (Wada and others 1995). This case is comparable with Wilson's disease of humans which is associated with an inherent abnormality leading to the accumulation of copper.

**Respiratory disease**

**IBR:** An outbreak of infectious bovine rhinotracheitis (IBR) was confirmed by postmortem examination of an adult Holstein-Friesian. It was the third cow of a ‘flying’ herd of 220 which died over a 14 day period, despite treatment, with signs of respiratory disease. Nine other cows were also affected, each of the cows exhibiting reduced milk yield, loss of condition, heavy breathing and coughing. The animals were housed two months previously, and each week, an average of six cows, which were purchased from local markets, were added to the herd. No vaccination was employed. Postmortem examination
revealed an extensive pneumonia with a necrotising tracheitis (Figure 2). Additionally there was chronic abscessation within the abdomen associated with a tyre wire which presumably had penetrated the reticular wall, with a second wire found within the lumen of the reticulum.

![Necrotising tracheitis in a cow with IBR](image)

**Fig 2: Necrotising tracheitis in a cow with IBR**

**SMALL RUMINANTS**

*Systemic disease*

**Parasitic gastroenteritis (PGE)** was the most frequently diagnosed disease in sheep & goats across all the regions. Parasitological examination of three faecal samples by Bury St Edmunds revealed significant *Trichostrongyle*-type egg counts (up to 11400 epg) and *Nematodirus* spp (up to 700 epg) in spite of treatment with a macrocyclic lactone product three weeks previously. Penrith diagnosed PGE in hill sheep wintering on a lowland farm. Following dipping and dosing with a combined wormer and flukicide they had been put onto "clean" grazing but diarrhoea was noticed and three lambs had died. At post-mortem examination a worm egg count of 1,150 epg and a total worm count in the thousands confirmed the diagnosis. Anthelmintic resistance to levamisole was suspected.

Very high worm counts were recorded at post mortem examination by Carmarthen in untreated 20 month old ewe lambs. A gastrointestinal tract worm count revealed 2700 *Teladorsagia spp*. plus 900 immature worms in the abomasum and 28000 *Trichostrongylus spp.* and 1100 immature worms in the small intestine. A worm egg count of faeces showed 2700 trichostrongyle-type egg per gram.

There is increasing evidence of anthelmintic resistance and lack of an informed worming strategy on some farms. Implementation of a worm control strategy is essential and should include monitoring efficacy of treatments with simple post-drench worm egg counts on pooled samples with further laboratory investigation if resistance is suspected.
PGE was also identified in goats. In a Shrewsbury case, abortions and malaise were reported in a group of 22 goats housed at the start of November but not wormed since July. Three had died. PGE was confirmed at postmortem examination with an estimated 60,400 immature worms in the abomasum and 29,000 *Trichostrongylus* sp. in the small intestine.

**Systemic disease**

**Multicentric lymphoma** was diagnosed by the University of Bristol in an 11-year-old Texel cross pet wether which had lost weight and had intermittent diarrhoea and colic. At postmortem examination widespread lymphadenopathy was found (Figure 3). The abomasal mucosa was markedly thickened and corrugated (Figure 4) and there were adhesions between the caecum and colon with reddening of the serosa. Histopathological examination revealed widespread lymphoma involving the skin, abomasum, intestinal tract and lymphoid tissue. The reported colic and intermittent diarrhoea were due to the large intestine lymphoma and development of adhesions. Lymphoma in small ruminants is rare and (with the exception of Bovine Leucosis virus which can cause disease in both cattle and sheep) the cause is unknown and is usually sporadic.

Fig 3 Markedly enlarged retropharyngeal lymph nodes in a sheep with lymphoma

![Markedly enlarged retropharyngeal lymph nodes in a sheep with lymphoma](image1)

Fig 4 Thickened and corrugated abomasum

![Thickened and corrugated abomasum](image2)
Hemlock poisoning and Tick-Borne Fever:
Shrewsbury investigated mortalities in a group of 420 recently weaned ewes. The ewes had been treated with a cypermethrin pour-on and moved onto open hillside. The group was later moved into a closed area with bracken. Ten days later 12 ewes were found dead and 13 others were ill with several recumbent and paddling. On closer inspection dilated pupils, mild hyperaesthesia, and a fine tremor were noted. At post mortem examination several live ticks were found on the carcase. Inclusion bodies were detected in blood smears and PCR for *Anaplasma phagocytophilum* confirmed Tick Borne Fever (TBF). Histological examination of organs provided evidence of multisystemic inflammation perhaps associated with low level septicaemia. TBF causes initial pyrexia and subsequent immunosuppression. However histology of the heart described lesions that could be compatible with intoxication. The group of sheep were immediately moved off the field but mortalities continued with two to three sheep dying every day. A VIO visited the farm and while some improvement had occurred after treatment with oxytetracycline, deaths continued. The field was examined and several Umbelliferae plants were present which had the majority of their stems and leaves missing. Plants were collected and sent to the SAC for examination. A ewe that had displayed neurological signs was submitted for post-mortem examination. Extensive histological examination showed damage to the nerves of the spinal cord potentially related to a neurotoxic agent.

Inflammation was evident in a wide variety of organs including the gastrointestinal tract, particularly in the fore stomachs. This was likely to be due to secondary infection following gastrointestinal atony resulting from impaired transmission of nervous stimulus after ingestion of toxins. The presence of hemlock (Fig 5, *Conium maculatum*) was confirmed by SAC CVS. The overall presentation of the case suggested that hemlock poisoning had occurred in addition to the Tick Borne Fever.
PIGS

Upward trend in porcine reproductive and respiratory syndrome diagnoses

Analysis of the seasonality of porcine reproductive and respiratory syndrome (PRRS) diagnoses in GB shows that the last quarter (October to December) of 2014 had the highest quarterly diagnostic rate for PRRS since 2004 when the PCR test was introduced, improving diagnosis. Six percent of the APHA PRRS diagnoses were reproductive disease outbreaks. The remainder were all in post-weaned pigs. Disease due to PRRS in post-weaned pigs is often associated with other pathogens or disease conditions, in part reflecting the immunosuppressive nature of the virus. The three most common diagnoses made in submissions with PRRS at APHA in 2014 were *Streptococcus suis*, salmonellosis and pasteurellosis. This helps explain why clinical signs in PRRS outbreaks can be variable. Figure 6 shows the clinical signs reported to APHA for outbreaks during 2014; respiratory signs, pigs found dead and wasting being the three most commonly described. The fact that PRRS is often identified together with other pathogens emphasises the importance of full diagnostic investigation in disease outbreaks. There is guidance on accessing diagnostic support and on sampling and testing available on this link: [http://ahvla.defra.gov.uk/vet-gateway/surveillance/diagnostic-support.htm](http://ahvla.defra.gov.uk/vet-gateway/surveillance/diagnostic-support.htm)

Figure 6: Clinical signs for APHA PRRS diagnoses in 2014 (excluding reproductive outbreaks)

Respiratory Disease

Several incidents of respiratory disease involving porcine and reproductive syndrome: Porcine and reproductive syndrome (PRRS) virus was detected by PCR in pooled sera from 17-week-old indoor finishers which had been coughing for several weeks in the Bury St Edmunds region. Fifty percent of the pigs were affected with low mortality.
PRRS was also diagnosed at Bury St Edmunds in ten to 12-week-old pigs from an indoor breeder-finisher unit together with bacterial lung infections. A group of 50 pigs were showing skin reddening and three had died in the four days prior to submission. The pigs were vaccinated for PCV2 and *Mycoplasma hyopneumoniae* but not for PRRSv. One pig had a severe fibrinous polyserositis, and all three submitted pigs had variable degrees of cranioventral consolidation with 45% of the lung showing lesions in the worst-affected pig. *Pasteurella multocida* and *Streptococcus suis* type 2 were isolated from the lung of one pig and PRRSv was detected in the lung of the pig with polyserositis revealing field challenge with the virus.

A pig was submitted to Thirsk to investigate ongoing respiratory disease on a finishing unit. The pigs were vaccinated for *Mycoplasma hyopneumoniae* and PCV2. Pigs were bought in at 35-40kg for fattening and coughing began after the pigs had been on the unit for about six weeks. There was an initial clinical response to antibiotic treatment but then a number of pigs developed ill-thrift and this problem escalated, with 20 of a group of 200 pigs dying in the week prior to submission. The pig submitted was very pale with a rectal prolapse which had been scavenged. The pig had pneumonia with a bacterial component involving *Pasteurella multocida* and active PRRSv infection was detected by PCR and was likely to be playing a significant role. It is likely that excessive coughing led to rectal prolapse due to increased abdominal pressure and that this was cannibalised by other pigs leading to fatal haemorrhage in the submitted pig. Pigs with rectal prolapses need to be detected earlier and removed from the pen for treatment/management as advised by the attending veterinary surgeon.

**Endocarditis due to erysipelas with gastric ulceration:** Erysipelas was diagnosed at the University of Bristol in two Gloucester Old Spot gilts, from a group of 13 pigs on a small holding. Both pigs had a short history of malaise and apparent abdominal pain. At postmortem examination, one gilt had a large haemorrhaging gastric ulcer at the pars oesophagea and the stomach was full of blood (Figure 7). The other gilt had a smaller non bleeding ulcer, but both had severe chronic vegetative lesions on the left atrio-ventricular valves (Figure 8) from which *Erysipelothrix rhusiopathiae* was isolated. Stomach ulceration is not a specific manifestation of erysipelas and in this case it is possible that illness due to endocarditis affected the gilts’ appetites and contributed to ulcer formation. Maintaining vaccination of pigs against erysipelas is an important routine preventative measure; the gilts in this small herd had not been vaccinated though the older sows had been.
Iron deficiency anaemia in wasting piglets on a smallholding: Iron deficiency was diagnosed as the cause of ill thrift and lethargy in two piglets submitted to Sutton Bonington from a small open farm with five breeding sows. The holding reported that around a third of piglets in the last two litters had shown poor growth. One piglet submitted live had very pale mucous membranes and haematology confirmed profound anaemia with a red blood cell count of 1.82 x10^12/l (reference interval 6-9). The carcase showed profound pallor and watery blood. Biochemistry revealed iron deficiency. Additional laboratory testing did not identify any other concurrent aetiologies for the anaemia and advice was given on provision of iron supplementation.

Nervous Diseases

Severe outbreaks of bowel oedema causing sudden death and nervous signs: Two diagnoses of bowel oedema were made by Sutton Bonington, both quite severe and both associated with E. coli strain E4 (serotype O139:K82) which is recognised as being associated with bowel oedema. In the first incident, the disease caused 50 deaths from a group of 300 pigs over a three-day period with a clinical presentation of collapse and nervous signs within 24 hours. Disease affected 14-week-old pigs on a 1,000-head finishing unit. At post-mortem examination the eyelids of the submitted pigs were noted to be swollen. A red fibrin deposit was noted over the small and large intestinal serosa and the distal half of the small intestine contained dark, haemorrhagic contents indicating enteric pathology in addition to nervous disease. Other organ systems were grossly unremarkable and intestinal bacteriology recovered a heavy growth of haemolytic E.coli strain E4.

In the second incident, bowel oedema was diagnosed following the submission of two dead pigs from a smallholding. The pigs were six-months-old and had been clinically normal the previous day. On the morning of submission, the owner found three pigs were dead and four others were showing varying degrees of ataxia and nervous signs, including seizures and
high pitching squealing. Both submitted pigs had markedly swollen eyelids and subcutaneous tissues at the extremities were also oedematous. The intestinal mesentery was oedematous and the content of the distal small intestine was liquid with a hint of haemorrhagic streaking. A heavy growth of haemolytic *E. coli* serotype E4 was recovered from the intestines of both pigs.

**BIRDS**

*Commercial Layers*

**Necrotic enteritis:** An unusual case of necrotic/ulcerative enteritis was seen in a submission of 35-day-old commercial layer pullets with a history of increased mortality. Postmortem examination revealed moderately dehydrated carcases, with enlarged livers and spleens, mucoid and haemorrhagic intestinal contents and single or multiple variably sized foci of mucosal necrosis resembling “Turkish towel” in the upper and mid small intestine. Bacterial cultures of liver and spleen were largely unrewarding. Histological examination revealed widespread acute necrosis of the intestinal mucosa with numerous rod shaped bacteria, typical of Necrotic Enteritis associated with *Clostridium perfringens* infection. There was no evidence of coccidial infection which is often a predisposing factor in the development of intestinal necrotic lesions. Histological examination of bursas and spleens revealed subacute bursitis and splenic lymphocyte depletion which were highly suggestive of a possible viral challenge such as Infectious Bursal Disease Virus (IBDV). Immunosuppression and enhanced predisposition to secondary bacterial infection caused by a possible IBDV challenge is perhaps the most likely explanation of this outbreak which was unusual in layer pullets.

*Broilers & Broiler Breeders*

**Blackhead and colibacillosis:** Blackhead and *E. coli* septicaemia were seen in 25-week-old broiler breeders submitted with a history of six deaths in a house of approximately 3,600 birds. Postmortem examination revealed distension of the caeca with a cast of necrotic and caseous material, pericarditis, swollen livers and spleens and tags of fibrinopurulent exudate in the abdominal cavity and reproductive tract. Bacterial culture the livers yielded heavy growths of *E. coli*. Histopathological examination of caecum revealed granulomatous lesions associated with histomonad-like organisms consistent with caecal blackhead. The findings were therefore suggestive of concurrent colibacillosis and histomonosis in the birds in this house.
**Backyard flocks**

**ILT:** A group of 60 point-of-lay chickens began showing ocular and respiratory signs 48 hours after eight of the group returned unsold from market. Treatment with antibiotic was started after about a week. After ten days a few birds had recovered, five had died but most were still showing clinical signs. Two birds which were “gaping” and had a nasal discharge were submitted for examination. Post mortem examination revealed that the conjunctvae of both birds were thickened and mucus was present in the right conjunctival sac of one of the birds. The same bird had foci of mucoid exudate in the lungs. Similar but milder pathology was seen in the other bird. The tracheas also contained excess mucus. No significant bacteria or *Mycoplasma* species were detected in the respiratory tracts. However a herpes virus was identified from tissue culture of trachea confirming a diagnosis of Infectious Laryngotracheitis (ILT). Exposure to subclinically or mildly affected birds is a major cause of spread of ILT and indirect spread via contaminated handling crates, equipment, clothing etc. can also occur.

**Myelocytoma:** The death of a five year old free range female chicken was due to a myelocytoma. This was a single bird affected of a flock of 12 chickens and for the two weeks before it died, the chicken had been unable to walk. The left leg was stretched out behind the bird with increased muscular tone reported. At necropsy there was a pale mass dorsal to the kidneys, mainly on the left side. The mass was identified on histopathology as a myelocytoma and a nerve was identified trapped within the tumour. The signs seen in this chicken are likely to be associated with the tumour pressing on the sciatic nerve. Infection with avian leucosis virus is the most common cause of myelocytoma in chickens.

**WILDLIFE**

**Mass mortality of Black Headed gulls** (*Chroicocephalus ridibundus*): A mass die-off of several hundred gulls occurred on the roof of commercial buildings on an industrial estate. Seven buildings with extensive low pitched roofs were joined together, each with different varieties of exhaust outlets. The area was known to be a night roost for black headed gulls. The gull deaths were only identified when maggots were seen inside one of the buildings, coming through the air conditioning system. Poor weather prevented immediate safe collection of carcases but a report was made to the Defra wild bird helpline and bodies were delivered to Shrewsbury VI Centre. There was marked autolysis in all carcases and a lack of food in the intestinal tracts with only small amounts of fibrous material in the gizzards. A visit was made to the premises and although a large number had been cleared (Figure 10), it was still possible to see dead birds on other industrial units particularly those which had hot air exhaust outlets in use 24 hours a day. The carcases were not visible at street level and no sick or dying birds had been observed by the workforce. The unusual sequence of events leading to delayed recognition of the incident, with marked decomposition of the carcases, limited examinations. No evidence was found of avian influenza.
viruses, West Nile virus, botulinum toxins or common bacterial pathogens. Further toxicological examinations are being considered and no further carcases have been seen. The precise cause of the deaths has not been determined.

Figure 9. Black headed gull carcases on roof of industrial buildings. Carcases from the building in the foreground have been removed.

A mass mortality of seabirds – cause unknown: A report of dead and dying seabirds in early winter on a shoreline/estuary area in south Wales was investigated by the submission of seven carcases, 3 oystercatchers (Haematopus ostralegus), 2 black headed (Chroicocephalus ridibundus) and 2 herring gulls (Larus argentatus) to APHA Carmarthen Veterinary Investigation Centre under the Diseases of Wildlife and the Avian Influenza Virus (AIV) in Wild Birds Schemes. Postmortem examination did not detect any obvious oil contamination. The birds were in varying body condition with some showing evidence of damage by scavengers around or after death. Tests for AIV and West Nile virus were negative. No significant bacteria were isolated and tests for botulinum toxins (C and D) on intestinal contents were also negative. Histopathology on the freshest carcases did not detect any significant lesions that could be responsible for death. Analysis for likely chemical poisons (carbamates, metaldehyde, neonicotinoids and rodenticides) was also negative. It was concluded that trauma related to the recent stormy weather was likely responsible for the deaths.

Suspected electrocution in a Peregrine (Falco peregrinus): A peregrine was found dead on the ground at a power station. The peregrine had been in good bodily condition with evidence of recent feeding as it had feathers, soft tissue and bone in the crop and gizzard. There were no signs of traumatic damage to the musculoskeletal system but there was marked blackening of
the tissues lining the oropharynx with the tongue and roof of the oropharynx having a blackened charred appearance (Figure 11). The findings suggested that the bird had died rapidly with damage to the oropharynx compatible with electrocution.

Figure 10: Peregrine oropharynx with charred appearance to the soft tissues

![Image of oropharynx]

**Salmonella Dublin in wildlife:** S. Dublin was cultured from the intestine of a siskin (*Carduelis spinus*) which had thick caseous material present in the crop. The bird was found dead in a rural garden surrounded by livestock farms. Twelve birds had died in the garden where food was put out for garden passerines. The bird was severely autolysed. It is unclear if the oesophageal lesion was caused by salmonellosis, or by trichomonosis with the S Dublin being an incidental finding. S. Dublin has been isolated very occasionally from wild mammals and birds by the Diseases of Wildlife Scheme over the years. Species affected since 2004 have included magpie (*Pica pica*), fallow deer (*Dama dama*) on two occasions, otter (*Lutra lutra*) and a fox (*Vulpes vulpes*). It is assumed that these infections occur as a result of spill-over of infection from cattle or cattle farm environments to wild species. S Dublin is host adapted to cattle and one of the most frequent salmonella isolates from cattle herds in England. It was unlikely that S Dublin infection was responsible for all the losses nevertheless the garden owners were advised that the infection was potentially zoonotic.

**References**


This summary is produced by the APHA and is drawn from reports provided at the time of reporting by the APHA laboratories at Bury St Edmunds, Carmarthen, Lasswade, Penrith, Shrewsbury, Starcross, Sutton Bonington, Thirsk and Weybridge and third party external postmortem providers to APHA (University of Bristol School of Veterinary Sciences, Royal Veterinary College, SAC Consulting Veterinary Services St Boswells). APHA monthly reports are available online at [https://www.gov.uk/government/publications/disease-surveillance-reports-2014](https://www.gov.uk/government/publications/disease-surveillance-reports-2014)