Bovine Astrovirus: an emerging pathogen in the UK
APHA Disease Surveillance Report
April 2015

- Botulism in lambs
- Summary of small ruminant submissions, first quarter 2015
- Clostridial enteritis in neonatal piglets

CATTLE

Musculoskeletal disease
Blackleg: Clostridial myositis or ‘blackleg’ was diagnosed at Carmarthen in a 10-month-old Charolais heifer, which was one of two to die in a group of 14 that had been at grass for three weeks. Areas of black emphysematous skeletal muscle were present on the brisket and in both forelimbs. A fluorescent antibody test (FAT) on a smear of affected muscle tissue was positive for Clostridium chauvoei. The animals had been given only one injection of a blackleg vaccine before turnout, rather than the two doses recommended to provide significant immunity. As follicular activity was present on the animal’s ovaries it was postulated that bruising caused by the heifers riding each other could have predisposed to the myositis. APHA comments that clostridial vaccines are cheap, highly effective and that their appropriate use should always be considered as part of active farm health planning.

Nervous disease
Bovine astrovirus infection was identified at Carmarthen associated with sudden onset progressive nervous signs, including focal seizures, in a three-year-old Limousin bull. The animal was euthanased and the brain obtained for examination. Histopathology revealed a non-suppurative encephalitis indicating the likelihood of a neurotropic viral infection. Immunohistochemistry for louping ill virus, which can cause such lesions, was negative, but astrovirus RNA was detected by PCR in the fixed tissue. Bovine astrovirus is a relatively newly recognised cause of nervous signs in cattle and the disease is poorly understood. Studies in both the USA and Switzerland have previously identified the virus in cattle with non-suppurative encephalitis, and there have been several other cases confirmed in UK cattle.
A cerebral vasculopathy typical of *Clostridium perfringens* epsilon intoxication was diagnosed postmortem in a suckler cow. The animal was one of a group of four cows in a small-holder's herd. The animal had become recumbent and exhibited apparent blindness and uncontrolled kicking. There was no menace response and some frothing at the nares. Death occurred within a few hours despite symptomatic treatment with minerals, a multivitamin and a non-steroidal anti-inflammatory drug. There was no gross pathology in any organs; however, histopathological examination of the brain confirmed the diagnosis. Epsilon intoxication is more commonly diagnosed in sheep in which it has a reasonably consistent presentation (‘pulpy kidney disease’). In comparison, the disease is more variable in cattle with cases sporadically diagnosed in neonatal calves (Watson and Scholes 2009), growing animals and adult cows. The ‘trigger’ for the disease is unknown. Vaccination could be considered, as in sheep, to prevent disease.

**Alimentary disease**

**Abomasitis** was diagnosed on postmortem examination of a three-week-old Belgian Blue cross suckler calf. It was the second calf to die in a group of 15 calves which had been turned out to pasture with their dams one week previously. The abomasal mucosa was dark red, swollen and oedematous and had a few small erosions. Gram positive rod-shaped bacteria with typical morphology of *Clostridium sordellii* were identified on direct microscopy and the causative bacteria were isolated in anaerobic culture. *C. sordellii* infection was first identified by the then Veterinary Investigation Service in associated with abomasitis in lambs (Lewis and Naylor 1998), and similar disease has since been diagnosed in dairy calves, but it is rarely found in suckler calves. The underlying aetiology, in particular what triggers the onset of disease, is poorly understood.

**Respiratory disease**

**Necrotising pleuropneumonia**: *Trueperella pyogenes* and *Fusobacterium necrophorum* subspecies *necrophorum* infections were demonstrated as the cause of a severe necrotising pleuropneumonia in a seven-day-old dairy heifer. It was the tenth loss from 30 calves in a herd of 300 cows. Postmortem examination confirmed diffuse unilateral lesions affecting all of the right side lung lobes, remarkably sparing the left side lobes. This was thought to have occurred due to the animal being recumbent for three days before it died. Low colostral status and cryptosporidiosis were also identified.
**SMALL RUMINANTS**

**Systemic disease**

**Septic polyarthritis:** Shrewsbury identified joint ill affecting 100/600 lambs in a lowland flock. Clinically there was slight joint swelling, commencing at a week old, which progressed to very swollen joints with bursitis and muscle wastage. Response to treatment was poor and many lambs became recumbent and died. The ewes had lambed inside. The lambs' navels were treated with iodine soon after birth and long acting penicillin was given to them at turnout. Two lambs were presented for postmortem examination. One had marked swelling of the stifles and the other had swelling of the limb joints and muscles. All joints contained large volumes of floccular purulent fluid which extended into the adjacent muscles and tendons. There was pus present at the atlas joint in both lambs and also distension of the costrochondral junctions with purulent material. *Streptococcus dysgalactiae* was cultured from the joints of both lambs. Concern regarding the welfare of the remainder of the lambs was raised with the practitioner and a farm visit was offered. Euthanasia of those unlikely to respond to treatment and chronic cases was advised.

In a second case *Streptococcus dysgalactiae* was isolated from joint swabs submitted from lambs from 2 additional holdings. There were 15/100 affected, with 10 deaths, on the first holding and again response to antibiotic treatment was poor. In the second case 7/80 lambs aged two to three weeks were affected.


This report notes that regarding *Streptococcus dysgalactiae*, 77 isolates were identified over the three year period, multiple resistance was not detected, and in 2011 all of the isolates tested were resistant to tetracycline; this figure declined to 85% in 2013. Resistance was also seen to tylosin in 4% (one isolate) each year. The isolates were susceptible to all of the other antibiotics tested.

**Enteric disease**

**Insufficient milk, coccidiosis** and **Nematodirus** infestation were the cause of diarrhoea and poor condition in a group of lambs from a city farm. At least 15/80 were dull and had watery bloody green diarrhoea. Clinical signs started
a week after turning onto non-rested pasture. Both submitted lambs were in poor body condition and showed evidence of insufficient feed intake, with dry forage present in the rumen. There was only grass in the abomasum of one and soil and water with no milk in the abomasum of the second. The mesenteric lymph nodes were enlarged. The mucosa of the caecum and colon was thickened with a “pimpled” texture. The kidneys were pale and enlarged in one lamb. Faecal examination identified a moderate trichostrongyle burden, including *Nematodirus battus* eggs, and very high coccidial oocyst counts (363,000 and 839,500 oocysts per gram). Advice on treatment and supplementary milk provision was given.

*Salmonella Diarizonae* and *Listeria monocytogenes* were cultured from the faeces of a ewe scouring after lambing. Postmortem examination by the submitting vet identified enteritis with ulcers in the large intestine. In total 10/500 ewes died and an additional 6 aborted.

**Nervous disease**

**Border disease** was confirmed on two holdings where young lambs were reported to be “shivering”. Pyrexia was also reported in one case and signs consistent with “hairy shakers”, including long curly fleece and muscle tremors, in the other. The second holding also reported an abortion rate of 5-6% and increased numbers of stillbirths. Border disease virus was detected on the first holding with Bovine Viral Diarrhoea virus (BVDv) on the second. BVD virus has been previously occasionally identified as the cause of ‘border disease’ in sheep. Two surveys have reported the pestivirus types associated with border disease outbreaks. The first in 1997 of isolates from Britain, New Zealand and Sweden over an 18 year period found 70% of the isolates were Border Disease virus with 30% BVDv type 1 (Vilcek and others 1997). A second survey at the Moredun Institute in 2006, of mostly Scottish isolates, confirmed 80% border disease virus and 20% BVDv type 1 (Willoughby and others 2006). BVD in sheep is an important consideration for cattle eradication programmes.

**Botulism:** Dramatic losses occurred in a group of yearling lambs associated with poultry litter. 1000 lambs had been split into two adjacent fields, 600 in one field and 400 in the other. The field with 600 contained a large amount of poultry manure ready for spreading two trailer-loads of which were not fenced off. Three weeks after the lambs had entered the field 70 were found dead on one day and 30 others were showing various degrees of recumbency, weakness and flaccid paralysis. A postmortem examination by the practitioner revealed a rumen full of poultry litter. The lambs were immediately removed from the field but new cases continued to occur in the group for approximately 14 days with the majority being euthanased. In total 230 of the group of 600 lambs died.
Box report contribution
During the first four months of 2015 small ruminant diagnostic submissions to APHA and to our Partner Post mortem providers (RVC, University of Bristol, University of Surrey, SAC CVS and Iechyd Da) in England and Wales were submitted to investigate disease from animals with the presenting signs described in table 1. The presenting sign is included as a selection on the submission form and is important in our understanding of syndromic trends in disease.

Table 1. Diagnostic submissions by presenting sign January – April 2015

<table>
<thead>
<tr>
<th>Presenting Sign</th>
<th>Submissions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abortion</td>
<td>1025</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>263</td>
</tr>
<tr>
<td>Wasting</td>
<td>240</td>
</tr>
<tr>
<td>Found dead</td>
<td>226</td>
</tr>
<tr>
<td>Unknown</td>
<td>134</td>
</tr>
<tr>
<td>Malaise</td>
<td>107</td>
</tr>
<tr>
<td>Nervous</td>
<td>62</td>
</tr>
<tr>
<td>Repro</td>
<td>59</td>
</tr>
<tr>
<td>Recumbent</td>
<td>55</td>
</tr>
<tr>
<td>Skin</td>
<td>47</td>
</tr>
<tr>
<td>Other</td>
<td>45</td>
</tr>
<tr>
<td>Respir</td>
<td>34</td>
</tr>
<tr>
<td>Musc/Skel</td>
<td>27</td>
</tr>
<tr>
<td>Lame</td>
<td>26</td>
</tr>
<tr>
<td>Eye</td>
<td>13</td>
</tr>
<tr>
<td>Gt</td>
<td>12</td>
</tr>
<tr>
<td>Healthy</td>
<td>10</td>
</tr>
<tr>
<td>Masticlin</td>
<td>9</td>
</tr>
<tr>
<td>Milkdrop</td>
<td>4</td>
</tr>
<tr>
<td>Urinary</td>
<td>1</td>
</tr>
</tbody>
</table>

Predictably, submissions to investigate abortion were highest and the most commonly diagnosed causes of abortion included: *Chlamydophila abortus* (36.02%), toxoplasmosis (28.99%), *Campylobacter* (15.44%)

For presenting sign diarrhoea the most commonly diagnosed diseases were PGE (22.22%), cryptosporidiosis (20.47%); rotavirus disease (13.45%), coccidiosis (5.85%) and chronic fasciolosis (4.68%)
PIGS

Enteric Disease

Rotavirus causing diarrhoea prior to weaning in small herd and in neonates in commercial herd: Rotavirus was identified as the cause of the diarrhoea in faecal samples submitted to Starcross to investigate diarrhoea in six-week-old preweaned piglets in a Saddleback herd. Porcine epidemic diarrhoea virus (PEDv) was not detected in routine testing for surveillance. PED outbreaks have now been reported in several EU countries but not the UK; the outbreaks in the EU have involved a reportedly milder PEDv strain (INDEL, OH851-like) which differs from those known as virulent from North America and Asia. APHA are testing samples submitted from pigs with diarrhoea for PEDv using PCR and BPEX are funding this testing for units in England. High mortality in sucking piglets with diarrhoea would be of particular concern and should be promptly tested for PEDv. While testing takes place, strict biosecurity measures should be implemented to help limit spread of the virus and there is advice available on the BPEX website http://development.bpex.org.uk/R-and-D/Pig-Health/pedv.aspx.

Neonatal diarrhoea affecting about 50% of recent litters was investigated in a herd which had diagnosed rotaviral enteritis a year previously. The rotavirus infection had been managed by controlled exposure in late pregnancy which had worked fairly well until recently. The diarrhoea was described as watery, starting from two-days-old and affected sow and gilt litters. Three acutely affected live piglets were submitted to Thirsk for investigation. The findings were consistent with neonatal enteritis with watery yellow floccular contents in the large intestine. Rotavirus was detected using PAGE testing and histopathological findings in the intestines supported the diagnosis.

Clostridial enteritis outbreaks continue in neonatal piglets: Two incidents of clostridial enterotoxaemia were diagnosed at Bury St Edmunds. In the first, live three-day-old piglets with diarrhoea were submitted from an outdoor breeding unit on which 10 of a batch of 150 litters were affected with malodourous yellow watery diarrhoea. Affected litters were from any parity of sow and about half of the piglets in these litters were affected. All three piglets had marked necrosis affecting mainly the jejunum; histopathology confirmed acute necrotising enteritis. Clostridium perfringens alpha toxin but not beta toxin was detected in small intestinal contents suggesting type A enterotoxaemia; however, beta toxin is labile and, given the necrosis, it was thought more likely that this was type C disease. No other enteropathogen was detected, including no porcine epidemic diarrhoea virus, and there was no evidence of hypogammaglobulinaemia in the piglets.

In the second incident, three dead five- to seven-day-old piglets were examined to investigate diarrhoea and death from four to five-days-old in gilt litters on an outdoor breeding unit. About 20 of the batch of 200 gilt litters were affected and about 60 piglets had died. Severe acute necrotic enteritis was present in all three piglets and clostridial disease was suspected. This was supported by the detection of Clostridium perfringens alpha toxin in small intestinal contents from two of the piglets. When clostridial disease occurs in
slightly older piglets, as in this case, concurrent enteropathogens may be playing a role; none were detected and no coccidial oocysts were detected by histopathology or faecal oocyst counts. Diagnosis of coccidiosis due to *Isospora suis* in young piglets can sometimes be problematic as oocyst output may be low; submission of live affected piglets, when possible, allows the intestines to be fixed within minutes of death and the coccidial forms can be detected by histopathology.

**Respiratory Disease**

**Inclusion body rhinitis with systemic involvement:** Tissues were submitted to Thirsk from three and five-week-old pigs to investigate the cause of sneezing from three weeks of age in piglets on a unit with 80 sows. Some were reported to respond to antibiotic treatment while others declined with weight loss and ultimately needed to be euthanased. Histopathology revealed severe lymphocytic inclusion body rhinitis including lymphohistiocytic portal hepatitis and extensive lymphohistiocytic interstitial/tubulointerstitial nephritis with occasional megalocytic cells with intranuclear inclusion bodies. These findings were consistent with severe cytomegalovirus rhinitis and systemic infection. There was also evidence of secondary bacterial infection and of bacterial colitis in one of the piglets. Previous cases of systemic cytomegalovirus have been diagnosed at Thirsk, with evidence of preceding swine influenza virus infection although that was not identified in this case.

**Inclusion body rhinitis as part of a disease complex after weaning:** Problems since before Christmas of respiratory disease and wasting affecting around 20% of pigs from two weeks after weaning in a 700-sow herd were investigated by submission of three seven-week-old piglets to Thirsk. Piglets appeared to be in good health and good condition at weaning and were vaccinated against PCV2-associated disease at three-weeks-old. Post-mortem investigation revealed a degree of enteritis in all three pigs, pneumonias of variable severity and upper respiratory tract lesions with the mucosa of the turbinates appearing swollen, reddened and covered with mucus (Fig 2). Further testing, including histopathology, revealed the presence of inclusion body rhinitis and bronchopneumonia involving *Actinobacillus pleuropneumoniae* and *Bordetella bronchiseptica* infections. No PRRS or swine influenza viruses were detected. The enteric lesions were found to be due to a combination of rotaviral and *E. coli* 0157:K,V17(V17) infections. The *E. coli* strain is a recognised porcine pathogen associated with enteric disease. Viral diseases did not appear to be underlying the mixed diseases present and factors affecting disease challenge in the postweaning period including pen hygiene mixing, ventilation, age segregation, and other stresses may be relevant.
Fig 2: Rhinitis evident on nasal turbinates in a typical case of inclusion body rhinitis (porcine cytomegalovirus infection).

**Systemic Disease**

**Multiple diagnoses of disease due to *Streptococcus suis* type 2:** Multiple diagnoses were made, although with differing clinical and pathological findings; several are described here and under nervous disease. In one, six sudden deaths from a group of 700 eight-week-old pigs prompted submission of a dead pig. The pig had a moderate cranioventral pneumonia and *Streptococcus suis* type 2 was isolated. Streptococcal disease was the cause of death and no viral involvement was detected but lung histopathology suggested earlier swine influenza infection. When pandemic H1N1 2009 swine influenza emerged in 2009, it was not uncommon for streptococcal disease to occur concurrently with swine influenza.

Another diagnosis of disease due to *S. suis* type 2 was made when a fresh pluck was submitted to investigate respiratory signs and sudden deaths with poor response to antibiotic treatment in 14-week-old housed pigs. Ten percent of 1200 pigs were reported to be showing respiratory disease, with 15 deaths. There was a vegetative endocarditis affecting the left atrioventricular valve from which *S. suis* type 2 was isolated. Whilst this pig may have been representative of those dying, it may not have been typical of the respiratory disease and no other respiratory pathogen was identified.

**Nervous Disease**

**Streptococcal meningitis remains a prominent diagnosis in growers:** *Streptococcus suis* type 2 was diagnosed as the cause of cases of meningitis in one pen of 13-week-old rearing gilts, despite *S. suis* vaccination. Twenty-five out of 40 in the pen were affected, with seven deaths. Those treated promptly with penicillin responded well. One gilt was submitted in good body condition and with excess cloudy fluid in both stifle joints: *S. suis* type 2 was isolated from liver, meninges and joints. On two further farms, *S. suis* type 2 was also isolated from meningeal swabs collected during on-farm post-mortem examinations performed to investigate sudden deaths and signs of meningitis in six-week-old and nine-week-old pigs.

Two six-week-old pigs were submitted to Thirsk to investigate the cause of lameness and shaking, with some pigs showing slight swelling above the eyes, but not of the eyelids as in oedema disease. Joints were not obviously
swollen and pigs were responding to antimicrobial treatment. Postmortem investigation revealed evidence of severe polyarthritis, pneumonia, possible septicaemia and meningitis. There was slight subcutaneous oedema along the ventral body in one pig. *S. suis* type 2 was isolated from both pigs, but to confuse matters a pure growth of *E. coli* 0139:K82 (E4) was also isolated from the small intestine of one of the pigs suggesting the possibility of oedema disease. Although it was suspected that streptococcal infection was the main cause of the problem, as a precaution, brain histopathology was undertaken and confirmed severe purulent meningitis consistent with streptococcal meningitis with no histological evidence of CNS lesions of oedema disease.

In contrast to the above cases involving *S. suis* type 2, when four-week-old pigs were submitted to Bury St Edmunds to investigate an increase in mortality to between 10 and 15% in the previous three batches of weaned pigs, it was *S. suis* type 1 which was found to be responsible. The pigs were showing tremors, incoordination and recumbency prior to death. Submitted piglets were in good body condition and had excess turbid yellow fluid in all joints including the atlanto-occipital joint in one pig. *S. suis* type 1 was isolated from the meninges of these pigs in pure growth and is likely to have been causing polyarthritis as well as meningitis although it was not isolated from the joints.

Musculoskeletal Disease

**Osteoporosis leading to pathological fractures in weaned first-litter sows:** A number of cases of suspected pathological fractures in first-litter sows occurred shortly after being weaned. A similar problem had occurred a few years ago after which the diet was changed (additional calcium included) and the problem resolved and it transpired that additional calcium was no longer being provided to gilts prior to being served. A typical affected sow which had delivered 16 live piglets and had weaned 10 good piglets was euthanased and submitted to Thirsk having been found off her legs four days after weaning. Postmortem examination revealed multiple fractures of both humeri and both femurs including the femoral necks and the cortices of the long bones were thinner than expected. It was suspected that the fractures were mainly pathological with the possibility of some degree of trauma contributing after weaning when sows were mixed, particularly as all four limbs were involved. Bone analysis showed normal Ca:P ratios and bone ash which is consistent with lactational osteoporosis as bone is normal in structure but reduced in mass and thus more prone to fracture. The heavy demands on gilts for calcium for both growth and lactation, especially when they have good litters means that osteoporosis is most commonly seen in late lactation or in weaned first-litter sows as on this unit. A shortage of dietary calcium and poor bone reserves predispose to its occurrence and these findings prompted an urgent review of the diet and management of gilts.
BIRDS

Commercial Layers

Necrotic enteritis: Dehydration and occasional necrotic enteritis associated with coccidiosis and immune-depression were seen in two submissions of 32- and 35-day-old commercial layer pullets with a history of increased mortality. Postmortem examination revealed moderately dehydrated and uneven carcases, some of which showed multiple, small foci of mucosal necrosis and pseudomembrane formation in the lower small intestine associated with coccidial infection. Histological examination of the intestinal mucosa confirmed focal areas of acute necrosis with numerous rod shaped bacteria and abundant coccidial forms. Examination of bursa and spleen tissue also revealed a subacute bursitis and splenic lymphocyte depletion suggestive of a primary infectious bursal disease virus (IBDV) challenge most likely predisposing to secondary infections. Necrotic enteritis is relatively uncommon in young layer flocks but was likely to have been consequential to the immunosuppressive effects of IBDV.

Mycoplasma gallisepticum infection: Three point-of-lay pullets were submitted to investigate respiratory disease and high mortality in a group of 410 recently purchased birds, of which approximately a third had died. Suspicion of notifiable disease had been reported prior to the submission of the birds but had been negated. At postmortem examination all three birds showed evidence of upper respiratory tract disease, including sinusitis and one bird showed evidence of pneumonic changes. Both Mycoplasma gallisepticum and M. pullorum were detected in lung tissue, the former being a significant cause of chronic respiratory disease in chickens and likely to have accounted for the signs reported. No other specific respiratory pathogens were detected.

Backyard Flocks

Pullorum disease: Salmonella Pullorum was identified from cultures that were submitted for identification. The cultures were isolated from the livers from young chickens (10- to 12-day-old) with a history of sudden deaths and/or wasting. This Salmonella causes a disease that was previously known as bacillary white diarrhoea (BWD) although it is now referred to as Pullorum disease. It is mainly seen in chicks less than three weeks of age and can initially be seen as excessive numbers of dead-in-shell chicks or death shortly after hatching. Affected birds show a variety of non-specific signs such as being dull with a tendency to huddle, respiratory distress, lack of appetite and white runny droppings that adhere to the feathers around the vent. Mortality varies considerably and can be up to 100% in extreme cases. In growing birds, lameness can be seen with swollen hocks causing poor growth rates. The most important route by which Pullorum disease is spread is vertically from an infected parent bird via the ovary to the egg, but the disease can also spread by horizontal contact, for example between infected chicks or pullets. Serological testing can be used to eliminate any affected or carrier birds from...
the adult population and replacement birds should preferably be purchased from flocks known to be free of the disease but this can be difficult in the small hobby chicken circuit or backyard flocks.

**Avian Encephalomyelitis:** Sudden onset weakness, paresis and tremor affecting two groups of chicks in a backyard flock reared for shows led to the submission of chicks to the University of Bristol aged ten days and three weeks. Paresis was first seen in the older group (at three weeks) but was then noted in the younger chicks too. The diet was changed when the signs were first noted but more birds continued to be affected. Gross postmortem examination was unremarkable but histopathological examination of brain and spinal cord tissue showed non-suppurative encephalomyelitis typical of avian encephalomyelitis virus (AEV). AEV typically causes a range of neurological signs in in chicks up to three weeks of age. The virus can be transmitted both vertically through the egg but horizontal infection can also occur after hatching, and the virus can survive for long periods in the environment. The clinical signs of AEV are rarely seen, at least in the commercial sector, because of widespread vaccination of parent birds and the resultant protection afforded by maternal immunity. Outbreaks of AEV rarely recur in backyard birds unless new, susceptible birds are introduced.

**References**

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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 and Thirsk, and partner external postmortem providers to APHA (University of Bristol School of Veterinary Sciences, Royal Veterinary College, SAC Consulting Veterinary Services St Boswells, University of Surrey at Buxton, Derbyshire). APHA laboratory services at Weybridge provide diagnostic testing for surveillance. These providers contribute to the VIDA diagnoses recorded on the APHA FarmFile database and comply with standardised
diagnostic criteria and laboratory testing requirements. APHA monthly reports are available online at https://www.gov.uk/government/publications/disease-surveillance-reports-2015