Parasitic gastroenteritis in cattle
Unusual presentation of *Actinobacillus suis* causing spinal abscesses
First APHA diagnosis of oedema disease in East Anglia for many years
Infectious coryza confirmed in a hobby breeding flock

**CATTLE**

*Alimentary disease*

**Coccidiosis:** Outbreaks of coccidiosis were reported by several VI Centres. There were good examples of the variation in presentation and challenge to diagnosis that can arise with this disease. Starcross investigated poor thrift in a group of 20 heifer replacement calves aged around six months. Diarrhoea was reported to have been a feature and they had been treated for coccidiosis. Postmortem examination revealed dehydration and pale mucous membranes. The content of the large intestine was green fluid with only 100 oocysts per gram present; however, histopathological examination confirmed a severe chronic coccidial typhlocolitis. In comparison, the sudden onset of haemorrhagic diarrhoea and death of an unweaned calf on a rearing unit was investigated. Postmortem examination was done by the practitioner who reported severely haemorrhagic colonic mucosa and submitted intestinal content in which there was only a very low count of 1100 oocysts per gram. The peracute presentation in this case is suggestive of prepatent coccidiosis and microscopical examination of the large intestinal mucosa could have been diagnostic.

**Parasitic gastro-enteritis** was also reported on several farms, in some cases associated with significant morbidity and mortality. Carmarthen investigated poor thrift in a group of yearling steers which had been housed for three weeks. The group last received anthelmintic in August. Around 10 of the group of 30 were in poor condition and three animals had died. Postmortem examination of the emaciated animal which weighed only 170 kg revealed a few thickened bile ducts in the liver, which may have been caused by past
liver fluke infection, as no liver fluke were present. There were many raised nodules on the mucosal surface of the abomasum (figure 1). Worm counts confirmed 28,400 mature *Ostertagia* spp. and 9,700 immature worms in the abomasum, with 44,900 *Cooperia* spp., 2,600 *Nematodirus* spp. and 80,700 immature worms in the small intestine. This was associated with a faecal worm egg count of 4,600 trichostrongyle-type eggs per gram. This outbreak of endoparasitism could have been prevented if the animals had received a suitable anthelmintic when they were housed.

Fig. 1 Raised nodules typical of ostertagiosis on the mucosal surface of the abomasum.

**Attaching effacing E. coli** (AEEC) infection was diagnosed as the cause of enteritis in two week old dairy calves. A live two week-old-dairy bull calf was submitted to Shrewsbury from a herd where, over the past four months, diarrhoea and malaise were reported in seven calves, six of which had died. The calves responded poorly to antibiotic and fluid therapy. Postmortem examination revealed watery fluid in the stomachs and intestines with slight reddening of the large intestinal mucosa. No pathogens were found using standard tests on the intestinal contents but histopathology identified small intestinal villous atrophy and a typhlocolitis associated with AEEC. The villous atrophy suggested past infectious insult and would have contributed to the diarrhoea and weakness. AEEC infection has been identified in several farmed species and can only be diagnosed by histopathology, hence examination of freshly dead animals is necessary. The exact aetiology of the disease is uncertain; bacterial colonisation of the small or large intestine can occur and co-infection may occur with other enteropathogens. The presence of the large populations of *E. coli* in the large intestine undoubtedly contributes to endotoxaemia in the affected calves. Control of outbreaks principally relies on hygienic calf rearing. A review of the disease was made by Wales and others (2005).

**Nervous disease**

*Cerebrocortical necrosis* (CCN) had been previously diagnosed in a group of 60 bull calves aged between four and six months. The protein balancer in the diet was replaced; however, two further cases of neurological disease were reported. Postmortem examination was undertaken by the practitioner and the brain was submitted for histopathology which identified a subacute to chronic encephalitis with bacteraemic localisation the likely cause. The origin
of the bacteraemia was not identifiable but as the animals are on a high concentrate diet, subacute acidosis and rumenitis were suggested.

**Urinary disease**

**Urolithiasis** was diagnosed at Thirsk in a group of seven and eight month old fattening steers. The cattle had been weaned and housed about a week previously and were being fed silage and a creep feed similar to that which they were receiving before being weaned. One of the 46 steers was found dead without prior signs of illness and was examined postmortem. The urethra was obstructed within the penis (Fig 2) and the kidneys had numerous pale foci throughout the cortices which were adhesed to the capsules, and there was pus filling the pelvises (Fig 3). The ureters were dilated with purulent contents and the bladder, which was ruptured, was irregularly thickened with numerous uroliths in the lumen. Pure growths of *Proteus mirabilis* were isolated from the kidney and peritoneum. Analysis of the uroliths confirmed magnesium, phosphate and oxalate content which indicated that the urolith formation was secondary to urinary tract infection. This suggested that the affected animal was likely to be a ‘one-off’; nevertheless a review of the diet was recommended.

![Fig 2: Uroliths obstructing the penile lumen](image)

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Fig 3: Kidneys

**SMALL RUMINANTS**

*Musculoskeletal Disease*

**Erysipelas:** Two severely lame lambs were submitted for post-mortem examination to RVC to investigate 14 lambs that had started to “walk funny” and had stiff hind legs. They were out of 40 that had not been ready for slaughter in early September. No foot lesions were present and an on-farm postmortem examination revealed no obvious changes; selenium and manganese blood levels were unremarkable; two or three calves appeared to be similarly affected; all affected animals had grazed previously flooded ground; severe chronic polyarthritis with marked thickening of joint capsules was found (Fig 5) in the two lambs submitted.

No bacteria were isolated from joint cultures. Erysipelas titres in both lambs were 1/20480, consistent with polyarthritis due to erysipelas. In chronic cases the bacteria may be absent or in very low numbers; culture using nutrient broth may be helpful to detect these bacteria. Serology on serum or joint fluid is a good alternative test.
Enteric disease

Clostridial enterotoxaemia: Two lambs were submitted to Carmarthen to investigate 10 out of 400 recently purchased store lambs that died shortly after housing. They were fed ad lib starter pellet and hay with access to mains water. They were treated at housing with ivermectin, a flukicide, a long acting tetracycline injection and vitamins but the vaccination history was unknown. Signs in affected lambs included bloating and scouring. Postmortem examinations revealed well grown lambs with liquid faeces on the tail and hindquarters. One lamb had a bloated rumen, a pale and friable liver that had ruptured with an adherent blood clot and bloody fluid in the abdomen. Both lambs had a bloat line of the oesophagus suggesting that they were bloated when they died. Both had a large amount of concentrate in the rumen and liquid large intestinal contents and also an increased amount of clear pericardial fluid containing a large gelatinous clot that was considered suggestive of a clostridial enterotoxaemia. Small intestinal content was positive for Clostridium perfringens epsilon toxin in both lambs. Histopathology showed brain lesions indicating increased vascular permeability and of white matter vacuolation that could be consistent with an acute enterotoxaemia due to C. perfringens type D. The rapid change of diet to a primarily concentrate diet led to the bloating, scouring and the clostridial enterotoxaemia.

Concentrate feeds should be introduced gradually into the diets of ruminants. Control of C. perfringens enterotoxaemia in lambs is by vaccination.

Thirsk also investigated sporadic mortalities (10/165) which were detected over a few weeks in a group of March/April born lambs weaned in July/September. The group were on grass with additional oats after weaning and had recently been changed to stubble turnips with an oat based creep feed. The lambs were vaccinated once with Ovivac P in October but did not get their second vaccination. Pulpy kidney was suspected; however post mortem examination on two lambs submitted separately revealed the rumens to be well filled with some plant fibre and large amounts of grains (Fig 6). Large amounts of grains were also present in the abomasum. The pH of the rumens was 4, confirming a diagnosis of ruminal acidosis.
Parasitic Gastroenteritis in Goats

High trichostrongyle egg counts of 3,850, 6,000 and 2,800 eggs per gram were associated with scouring and weight loss in goats in submissions to Shrewsbury. In one case there was concurrent *Salmonella anatum* infection. This salmonella can originate from environmental sources including water fowl, e.g. wild ducks.

Parasitic gastro-enteritis (PGE) was identified in a six- to seven-year-old goat which was submitted to Penrith after being found dead on a small holding. The goat was acquired at approximately two to three years of age and had never been in the best of condition suffering from intermittent diarrhoea. The *Trichostrongyle* type egg count was high at 2350 worm eggs per gram of faeces. It had mastitis and coagulase negative *Staphylococcus* sp. (CNS) was isolated on culture. The CNS may have caused an opportunistic infection secondary to the PGE associated debility. Histopathological examination found evidence of chronic PGE and worming advice was given.

PGE is a potential problem in goats of all ages as they are unable to develop protective immunity with age even after repetitive exposure to pasture challenge. This highlights the importance of Flock Health Plans, which should be developed in partnership with veterinary surgeons and using SCOPS principles.

Reproductive

As the lambing season gets underway abortion and stillbirth basic sample requirements for investigation, taken from the APHA Guidance on sample and test selection, are presented below.

Whole fetus and placenta is the submission of choice with maternal serum, or fetal fluids and fetal stomach contents with placenta.

<table>
<thead>
<tr>
<th>Condition / cause</th>
<th>Sample type</th>
<th>Recommended tests</th>
<th>Further information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enzootic abortion (Chlamyphilia abortus)</td>
<td>Placenta</td>
<td>Gross examination, stained smears</td>
<td>A positive result on a stained smear confirms the diagnosis. Positive maternal serology indicates exposure</td>
</tr>
<tr>
<td>Maternal Blood – single</td>
<td></td>
<td>Ab ELISA</td>
<td></td>
</tr>
<tr>
<td>Toxoplasmosis</td>
<td>Placenta</td>
<td>Gross examination, IFAT on placenta</td>
<td></td>
</tr>
<tr>
<td>Thoracic fluid</td>
<td></td>
<td>IFAT on fetal fluid</td>
<td>IFAT on fetal fluid is the test of choice. If fluid is not available carry out IFAT on placenta</td>
</tr>
<tr>
<td>Maternal blood – single</td>
<td></td>
<td>LAT on maternal blood</td>
<td>Positive maternal serology indicates exposure</td>
</tr>
<tr>
<td>Campyloobacter and other bacterial infections</td>
<td>Fetal stomach contents and liver</td>
<td>Culture and stained smears.</td>
<td>Send whole fetus and placenta if samples do not resolve a diagnosis</td>
</tr>
</tbody>
</table>

**PIGS**

*Alimentary Disease*

**Rotaviral enteritis in neonatal piglets in mainly gilt litters:** Live affected piglets were submitted from an outdoor unit to investigate diarrhoea mainly occurring in gilt litters and starting when piglets were one to two-days-old. The piglets had good milk clots in the stomach but gammaglobulin concentrations in two of the piglets suggested low to negligible colostral antibody transfer, and rotavirus was detected in small intestinal contents. Being from gilt litters where immunity to resident pathogens may not have fully established, and the poor colostral antibody transfer in some of the pigs, are both factors which may have predisposed to disease.
Colitis due to *Brachyspira pilosicoli*: Three incidents of colitis due to *Brachyspira pilosicoli* were diagnosed. In one, diarrhoea was described affecting 20% of the group of housed 16-week-old finishers. In the second, it was the cause of loose faeces in housed 15-week-old finishers. In this case less than 5% were affected and there was no associated mortality. In the third, no morbidity figures were available but disease was affecting nine-week-old pigs in a straw-based system.

**Respiratory disease**

**Porcine reproductive and respiratory syndrome diagnosed on a continuous unit:** An increase in mortality to 5%, respiratory disease and pasty-grey diarrhoea in the 2-3 weeks after weaning prompted submission of three dead pigs to Bury St Edmunds from a continuous outdoor unit. PRRS-associated pneumonias were confirmed in two by immunohistochemistry and PCR – the pigs had been vaccinated for PRRS, the immunohistochemical detection of PRRSv in the lungs indicates that in spite of vaccination, the virus was implicated in the respiratory disease. *Streptococcus suis* types 2 and 8 were isolated from the lungs as likely secondary infections and one of the pigs also had salmonellosis due to *S. Typhimurium* Copenhagen phage type 193. The continuous nature of the unit was likely to be facilitating persistence of PRRS on the unit with susceptible pigs being introduced at regular intervals.

**Systemic disease**

**Erysipelas outbreaks in unvaccinated pigs:** *Erysipelothrix rhusiopathiae* was isolated from heart valves with lesions of vegetative endocarditis submitted to Shrewsbury from two, five-month-old pigs, to investigate the cause of malaise and sudden deaths. Sixteen pigs were unwell in a group of 400 and 13 had died on a farm with 1900 pigs. The continuous nature of the unit and strawed system without regular cleaning and disinfection may have allowed accumulation of high levels of contamination with *E. rhusiopathiae*. Once clinical cases occur, this adds to the infection challenge to pen-mates and an outbreak can ensue. Improving hygiene and pig flow is an important part of control and occasionally vaccination of growing pigs is merited where successive batches of pigs are affected.

**Nervous disease**

**First APHA diagnosis of oedema disease in East Anglia for many years**

Oedema disease was diagnosed as the cause of malaise, nervous signs, swollen eyes and death of 30 pigs from a group of 600 eight-week-old indoor growers over a period of a week. Three dead pigs were submitted, two had swollen eyelids as shown in Figure 7 and there were other non-specific lesions including reddening of the skin of the ventral abdomen, fibrin stranding and excess fluid in the pleural cavities and prominent meningeal blood vessels. The clinical signs had raised the suspicion of oedema disease and this was confirmed by isolation of haemolytic *Escherichia coli* strain E4 (serotype O139:K82) from the small intestine, a strain known to be associated with oedema disease. Brain histopathology revealed multifocal asymmetric acute necrotising panencephalopathy and other histological features characteristic of oedema disease and reflecting the effect of the shiga-like
toxin elaborated by the *E. coli*. This is the first diagnosis of oedema disease in the East Anglian region for many years; interestingly, increased diagnoses of oedema disease elsewhere in the country have been noted in 2014 although the reasons for this are unclear. Oedema disease tends to affect rapidly growing pigs and feed factors including changes in diet or nutrient quality may be involved.

![Swollen eyelids in a pig with oedema disease](image)

**Fig 7: Swollen eyelids in a pig with oedema disease**

**Musculoskeletal Disease**

**Unusual presentation of *Actinobacillus suis* causing spinal abscesses**

A breeder-finisher unit reported sudden onset lameness in pigs from one to seven-weeks-old with a poor response to antimicrobial treatment. Between 5-10% of each batch of pigs was affected with pigs becoming unwilling to stand or move. Three such cases aged from one to four-weeks-old were submitted to Thirsk for investigation, one of which was showing hindlimb paresis and another with obviously swollen hind-limb joints. There was no evidence of infection at the sites of tail docking or teeth clipping, nor at the navels. One pig was found to have a spinal abscess affecting cervical vertebra 7, another to have a spinal abscess at thoracic vertebrae 2 and 3 and the third had a suppurative polyarthritis, multiple abscessation in the lungs and a fibrinopurulent pleuritis. *Actinobacillus suis* was isolated from the spinal abscesses which were in two one-week-old pigs while *Trueperella pyogenes* was isolated from the joints and lung of a four-week-old pig and probably represents secondary infection. *A. suis* colonises the respiratory tract of pigs and is most commonly encountered causing septicaemia in young piglets. It can also be involved in respiratory disease and septicaemia in older pigs and in localised infections following thromboembolic spread, as is likely to have occurred in this outbreak. The trigger for this outbreak remains unclear.
MISCELLANEOUS AND EXOTIC FARmed SPECIES

Hepatic lipidosis and coccidiosis in an alpaca: Hepatic lipidosis and coccidiosis caused by *Eimeria macusaniensis* was diagnosed at Starcross as the cause of death in an adult female alpaca that had been found dead, being one of a group of 13. The main gross post mortem findings included a friable pale liver, oedematous lungs, sub pleural and cardiac haemorrhages and soft, blood and mucus covered faeces. Parasitology identified an *Eimeria macusaniensis* count of 7450 oocysts per gram. Histological examination of the liver revealed severe hepatic lipidosis: possible causes of this included a nutritional imbalance, an underlying endocrine or other condition. *E. macusaniensis* can result in severe disease three weeks after exposure and two weeks before patency. Younger animals may present with diarrhoea whereas older animals show weight loss, poor weight gain, ill thrift, lethargy and loss of appetite, often with no diarrhoea. Colic may also occur. Hepatic lipidosis (in association with hepatic encephalopathy) was discussed in the Miscellaneous and Exotic Farmed Species emerging threats report April to June 2013 which can be found at http://webarchive.nationalarchives.gov.uk/20140707141401/http://www.defra.gov.uk/ahvla-en/publication/mis-exotic-survreports/

BIRDS

*Broilers*

**Endocarditis:** Valvular endocarditis and septicaemia due to *Enterococcus cecorum* infection were seen in a submission of 20-day-old broilers with a history of increased mortality. Postmortem examination revealed marked enlargement of the liver and spleen, vegetative endocarditis and fibrinous pericarditis. Cultures of spleen and heart yielded pure growths of a gram positive coccus which were further identified in the spleen as *E. cecorum*. This organism is part of the normal gut flora of broilers but can also be pathogenic in birds from approximately 14 days onwards, possibly associated with particular pathogenic clones of the organism.

*Commercial layers*

**Fowl cholera:** A small, 150-bird free range layer flock was affected with egg drop and respiratory disease including sneezing and nasal discharge, at eight months of age. All the birds were affected and three birds had died. Postmortem examination of five birds showed mucopurulent exudate in the infraorbital sinuses of all birds, pneumonia in one bird and airsaccultis, pleurisy, peritonitis and pericarditis in another bird. Pathogenic mycoplasmas were not detected. *Pasteurella multocida* was isolated from the respiratory tract of four of the birds, confirming a diagnosis of fowl cholera. The tendency
to affect upper rather than lower respiratory tract in this case may have accounted for the low mortality rate which might often be expected to be higher in cases of fowl cholera. The mortality rate did not increase over the two weeks following diagnosis; however response to treatment was poor and the owner decided to depopulate the flock.

Gizzard erosion: A few dead birds were being found daily in a flock of 4,000 free range layers aged 24 weeks. There was no effect on egg production in the flock. The combs of the birds were pale, and postmortem examination confirmed pallor in the carcases of the affected birds. This was associated with haemorrhage into the gastro-intestinal tract and also dark gizzard contents with patchy thickening and red-brown discoloration of the gizzard lining. Histopathological examination of the gizzards revealed focal ventriculitis with loss and regeneration of the glandular epithelium and occasional deep basophilic intranuclear inclusion bodies, features similar to those seen in broilers affected by adenovirus ventriculitis (gizzard erosion). Adenoviruses are considered ubiquitous and can cause a range of conditions in poultry including gizzard erosion in broilers, but this condition is much less recognised in layer chickens.

Turkeys
Erysipelas: Erysipelas was diagnosed in turkey flocks. In one case, 160 birds from a flock of 8000, twenty-week-old turkeys died over four days. The birds were in two pole-barns and no clinical signs were reported other than found dead. The gross postmortem findings were similar in all birds and were consistent with an acute systemic infection; in particular all the birds had enlarged spleens. *Erysipelothrix rhusiopathiae* was isolated from multiple systemic sites. In another case the disease was diagnosed as the cause of death in a three month old turkey. The small unit of 275 birds were being reared for the Christmas market and there was a history of 20 deaths over a three week period. Marked hyperaemia of the carcase with a fibrinous peritonitis, pericarditis, splenitis and airsacculitis were observed at postmortem examination and *E. rhusiopathiae*, was recovered in septicaemic distribution. In another flock the organism was isolated from the liver and a swab submitted from a 16-week-old turkey from a flock where 25 from a group of 400 had died. There was a history of Erysipelas on the farm.

Backyard flocks
Infectious coryza: An outbreak of respiratory disease was investigated in 21-week old birds in a hobby breeding flock, which comprised predominantly imported but also some homebred stock. The birds had reared well, but a few days after being moved from rearing to breeding accommodation, initially two or three birds were affected with nasal and ocular discharge and periorbital swelling, and two days later another eight birds were affected. Postmortem examination of affected birds confirmed mostly unilateral swelling of the periorbital region, with clear to mucoid exudate in the sinuses, accompanied by caseation in two birds. Bacterial culture undertaken on a sinus swab
yielded suspect *Aivibacterium paragallinarum*, the causative agent of infectious coryza, subsequently confirmed by 16S rRNA sequencing. No pathogenic Mycoplasma organisms were detected in pooled tracheal and conjunctival swabs. There was a good clinical response to antibiotic medication in the water. Infectious coryza is a contagious respiratory disease first confirmed in Britain in 2010 (Welchman and others 2010) and can be transmitted by asymptomatic carrier birds.

**Game birds**

**Gapeworm in black grouse:** Chronic gapeworm (*Syngamus trachea* infection) was diagnosed in a 30-week-old captive black grouse which had died suddenly. Postmortem examination revealed that the bird had been in poor condition, and there was marked consolidation of the left lung, thinning of the tracheal cartilage and gape worms were present along the length of the tracheal lumen. Gapeworm infection can be a problem in birds kept in outdoor enclosures as the parasite’s larvae may remain infective and viable in earthworms and other transport hosts for several years.

**References**


This summary is produced by the APHA and is drawn from reports provided at the time of reporting by APHA laboratories at Bury St Edmunds, Carmarthen, Lasswade, Leahurst, 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)