SELECTED HIGHLIGHTS FROM APHA PIG DISEASE SURVEILLANCE REPORTS
MARCH 2015

- Severe reproductive disease due to porcine circovirus 2
- Coccidiosis with *Salmonella* causing diarrhoea and death in replacement gilts
- Gastric ulceration occurring during Gläser’s outbreak
- Respiratory disease outbreaks due to swine influenza and PRRS virus

Reproductive disease

Severe reproductive disease due to porcine circovirus 2

A diagnosis of porcine circovirus 2-associated reproductive disease was made following submission of several litters of mummified and stillborn piglets delivered at term to Thirsk. This is only the second confirmed GB case of PCV2-associated foetopathy, but has been reported elsewhere in the field in Europe and North America and following experimental infection of pregnant sows. Disease manifested on two linked units as severe SMEDI, with mummified pigs and stillbirths (Figure 1), not abortions and sows were well. PCV2 antigen was detected by immunohistochemistry associated with severe myocarditis lesions in foetal hearts. No other infectious agent was identified. The units are now revaccinating replacement gilts for PCV2 prior to service.

Figure 1: Mummified and stillborn piglets delivered at term due to PCV2-associated foetopathy

PRRS virus sequencing following an outbreak of reproductive disease

An outbreak of porcine reproductive and respiratory syndrome (PRRS) was investigated further by sequencing the virus detected. There had been a few abortions, lethargy and inappetance in a group of 20 sows in one dry sow paddock on the 700-sow outdoor herd. Five PRRSv PCR-positive sow sera were sequenced and the virus in all five showed similarity to each other between 99.7 and 100%, indicating that just one strain was present. The strain in the sows was 91.6% and 89.4% similar to the two vaccine strains and no closely related PRRS virus strain (>98.5% homology) was identified in the GB database.
Enteric Disease

Type C clostridial enterotoxaemia causing sudden deaths of neonatal pigs
Severe enteritis, with variable degrees of jejunal necrosis and haemorrhage was found in three dead neonatal piglets submitted to Bury St Edmunds from an indoor breeder-finisher to investigate a problem of sudden deaths in piglets aged between three and ten-days-old. The problem had been ongoing in the herd for two months and in the current batch of 25 litters, most were affected with between one and six piglets showing signs in each litter. The lesions in the small intestines were suggestive of clostridial enterotoxaemia and this was confirmed by detection of alpha and beta toxins pointing to involvement of Clostridium perfringens type C. One piglet had necrotic gingival lesions associated with clipped teeth and this piglet also had hypogammaglobulinaemia indicating low colostral intake.

Enteritis due to Escherichia coli
Enteric colibacillosis was confirmed by isolation of Escherichia coli Abbotstown strain from the small intestine of a four-week-old piglet which was found dead in a group of 25 on a small 80-sow indoor herd. On-farm post-mortem examination revealed reddened small intestines with red-brown watery contents. No other enteropathogens were identified.

Monophasic variant of Salmonella causing mortality and wasting in growers
Salmonellosis due to a monophasic Typhimurium-like 4, 12:i:- strain phage type 193 was confirmed as the cause of necrotic typhlocolitis in two nine-week-old pigs. They were euthanased to investigate the cause of wasting, blue ears and about 10% mortality in a group of 1000 pigs on an indoor nursery-finisher, 200 of which were showing signs. The pigs were vaccinated at weaning for Mycoplasma hyopneumoniae, PCV2 and PRRSV and no evidence of disease due to these pathogens was found. Monophasic Salmonella variants like this one have been diagnosed in an increasing proportion of salmonellosis incidents in pigs in recent months.

Gastric ulceration occurring during a Glässer’s outbreak
Fatal haemorrhage from ulceration of the pars oesophagea was found to be the cause of death of an eight-week-old pig from a batch of 850 recently treated for Glässer’s disease. Since then occasional deaths totalling 26 had occurred over a ten-day period of which the pig submitted to Bury St Edmunds was an example. The carcase was markedly anaemic and cranioventral pulmonary consolidation was present affecting about 15% of the lungs, no viral or bacterial pathogens were identified although the recent antimicrobial treatment may have affected culture results. One can speculate that earlier Haemophilus parasuis infection caused a period of inappetance and predisposed to the gastric ulceration in the pig and a submission with a similar history was reported last month. Submission of further deaths would be worthwhile and these cases will be reviewed to see if there are any common features.

Coccidiosis with Salmonella causing diarrhoea and death in replacement gilts
A problem of diarrhoea and some deaths was reported in batches of nine-month-old replacement gilts after arrival on a commercial outdoor breeding unit. Six gilts had died in the last six batches delivered, deaths occurring around nine days after arrival. Two gilts were submitted to Starcross and both had a severe extensive necrotic enteritis (Figure 2) particularly affecting the distal small intestine and proximal large intestine.

Figure 2: Small intestinal necrosis due to coccidiosis in replacement breeding gilt
Coccidial counts of 6200 and 2300 oocysts/gram were detected in caecal contents and Salmonella Reading was isolated from the intestinal contents of one. Intestinal histopathology confirmed coccidia as the main cause of the necrotic enteritis and there was no evidence of involvement of Brachyspira species. Coccidiosis at this age is unusual in pigs and involves certain pathogenic Eimeria species. When it does occur, it is typically seen in a similar scenario to that present in this case; in replacement breeding pigs soon after moving onto commercial pig units. This is likely to be due to their exposure to a contaminated environment soon after arrival and has been described previously (Gaudie and others, 2005. Veterinary Record 157:517-8). Outdoor training paddocks into which replacement breeding gilts are introduced on arrival can be the source of infection if used long-term.

Respiratory Disease

Two outbreaks of swine influenza in growers
Swine influenza and Glässer’s disease were diagnosed at Bury St Edmunds when three dead pigs were submitted with severe fibrinous polyserositis. About 80% of 4,500 eight to ten-week-old pigs on the outdoor rearing site were showing signs of respiratory disease with some meningitis and about 10% mortality. It is likely that the swine influenza accounted for the high morbidity of respiratory signs and concurrent Glässer’s disease for the cases of meningitis and death.

Two pigs were submitted to Thirsk to investigate sudden death of eight pigs from a group of 1000 eight-week-old post-weaned pigs over the previous four days, with a few others showing meningitis-like signs. Post-mortem examination revealed lesions suggestive of acute bronchopneumonia in both pigs and an extensive peritonitis in one. Active swine influenza was confirmed in the pig with peritonitis by PCR and supportive histopathological lesions in the lungs, while in the other pig, PRRS virus was detected. No bacterial pathogens were isolated.

Swine influenza suspected in outdoor breeding gilts
An outbreak of swine influenza was also suspected but not confirmed in a group of 210 in-pig gilts in an outdoor herd. Fifty of the group were coughing, off food and lethargic over the period of a week with new cases developing each day, they showed a poor response to medication but none died. No swine influenza virus was detected by PCR in nasal swabs but significant titres were detected to the pandemic H1N1 2009 swine influenza strain in five gilts, providing supportive evidence of infection – detection by PCR or seroconversion being required to confirm a diagnosis.

Respiratory disease and sudden deaths due to porcine reproductive and respiratory syndrome
Respiratory disease due to PRRS virus was diagnosed at Bury St Edmunds when viscera including fresh pluck were submitted from a nine-week-old pig which had been postmortemed on-farm. There was a moderate cranioventral pneumonia from which no bacteria were isolated but PRRSv was detected by PCR in lung and histopathology revealed a subacute interstitial pneumonia consistent with PRRS. Ten percent of 800 pigs on the indoor rearing unit were affected with respiratory disease with seven sudden deaths over the two weeks prior to submission. There is similarity in clinical signs in this outbreak and those due to swine influenza described above and this emphasises the value of diagnostic investigation to establish a cause when outbreaks occur.

Another PRRS outbreak was diagnosed at Thirsk when three, six-week-old piglets were submitted to investigate respiratory signs in first stage weaners. Affected piglets were described as showing heavy breathing, low-level coughing, not thriving and having reduced feed and water intake. At the time the pigs did receive in-feed Penicillin and the piglets were vaccinated against PCV2 associated disease and Mycoplasma. Post-mortem examination revealed fibrinous pleuritis and pericarditis and pneumonia with grey/pink areas of consolidation in the cranioventral parts of all lung lobes. Histopathology and immunohistochemistry confirmed PRRS virus involvement in the pneumonia and PRRS virus was detected by PCR in pooled serum samples. No bacteria were isolated, but cultures may have been affected by the penicillin with which the pigs were being treated.
Systemic Disease

PCV2-associated myocarditis in preweaned pigs following disease in sows
Swabs for culture and fixed hearts for histopathology were submitted from two pigs as part of an investigation into sudden deaths prior to weaning in three-week-old piglets. On-farm post-mortem examinations revealed lesions suggestive of septicaemia and *Streptococcus suis* type 1 and 14 was cultured from one pig. In the second pig, histopathology revealed severe chronic active nonsuppurative myocarditis and significant labelling of PCV2 antigen by immunohistochemistry, consistent PCV2-associated myocarditis. The pig was the progeny of sows affected by PCV2-associated foetopathy described above and had been from a batch affected at birth (25% mummies and stillbirths), the chronic nature of the heart lesions suggests that the disease in this pig was residual from in-utero infection although it was not possible to determine the timing of the myocarditis lesions.

Outbreaks of disease due to *Streptococcus suis* type 2
Three outbreaks of streptococcal septicaemia due to *Streptococcus suis* type 2 were diagnosed. One was in two five-week-old pigs submitted from a 2000-pig nursery-finisher. Five deaths were reported with another four pigs showing respiratory signs. No viral disease was detected in the submitted pigs in which the organism was isolated from meninges and liver. The second was in 12-week-old pigs in which eleven had died over a three-day period, with three others with nervous signs, from the batch of 1100 on the indoor finisher unit. The third was in eight-and-a-half-week-old pigs, two of which died suddenly overnight and submitted to Thirsk. Post-mortem investigation revealed polyarthritis, pneumonias and suspected meningitis and *S. suis* type 2 was isolated from lung, joints and meninges. *Pasteurella multocida* was also isolated from the lung of one of the pigs and no underlying swine influenza or PRRS virus infections were detected. *S. suis* type 2 remains the most commonly isolated *S. suis* in APHA submissions.

Urinary Disease

Smallholder sow with pyelonephritis
A sow was submitted from a smallholding with nine sows and one boar. She was weaned three weeks earlier and had died following three days lethargy and anorexia despite antibiotic and anti-inflammatory treatment. Severe chronic bilateral pyelonephritis was found from which *Escherichia coli* was isolated mixed with a *Streptococcus* species from some sites but in heavy pure growth from one – the *E. coli* was considered likely to be the significant clinical isolate and is one of the main pathogens involved in urinary tract infections in pigs. The chronic nature of the lesions indicated disease had been ongoing for several weeks and it is important that sows, especially farrowing/lactating sows are encouraged to get up to drink and urinate at regular intervals. Ensuring good hygiene and access to clean water at all times was also advised. Pyelonephritis is much less common now on commercial units than in the past.