SELECTED HIGHLIGHTS FROM APHA PIG DISEASE SURVEILLANCE REPORTS
MAY 2015

- Swine influenza detected on farm experiencing an abortion outbreak
- Outbreaks of porcine reproductive and respiratory syndrome prominent
- Meningitis, endocarditis, arthritis and septicaemia due to *Streptococcus suis*
- Osteomyelitis in limb bones causing unresponsive lameness

OUTBREAKS OF PIGLET SEPTICAEMIA DUE TO *KLEBSIELLA PNEUMONIAE*:
ALERT FOR FURTHER OUTBREAKS THIS SUMMER

Each summer since 2011 APHA (formerly AHVLA) has diagnosed outbreaks of septicaemia in piglets due *Klebsiella pneumoniae* subsp. *pneumoniae* (Kpp). All but one of these cases has occurred in East Anglian herds, one outbreak in 2014 was diagnosed in the South West of England. The seasonal nature of disease occurring between May and September is illustrated in the figure below.

The most common features of the outbreaks are:

- Sudden or very rapid deaths
- Preweaned pigs affected from 10-days-old to point of weaning
- Non-specific lesions (septicaemic) at post-mortem examination
- Outdoor pig units
- Mortality variable 1-6 % of pigs born (one herd 16%)
- Appears self-limiting but has recurred in two herds in subsequent years

One septicaemia outbreak in East Anglia in 2014 involved concurrent severe mastitis in lactating sows. Post-mortem examination and bacteriology are required to confirm a diagnosis. The same emerging strain of Kpp (sequence type 25) has been involved in all outbreaks to date. The website link below includes details of the clinical presentation and pathology. Veterinary surgeons wishing to submit or discuss diagnosis of possible cases which may arise during the coming months, should contact an APHA Veterinary Investigation Officer.

Reproductive disease

Active swine influenza detected on farm experiencing an abortion outbreak
In the last monthly report, an abortion outbreak investigated by the University of Bristol was described in which no infectious cause was established but serology on aborting sows showed high antibody titres to swine influenza. Previous outbreaks of acute swine influenza in pregnant sows have occasionally caused small outbreaks of late stage abortion and stillbirths due to the maternal effect of the viral infection and two sows were seen to be dyspnoeic in the dry sow yards at the time of the abortions. It was agreed that if respiratory disease continued, that further samples would be submitted. Nasal swabs were subsequently submitted for Defra-funded swine influenza testing from pigs showing acute respiratory disease in the first two weeks after weaning. Swabs from two pigs were PCR-positive for both for influenza M gene and the pandemic H1N1 2009 strain of the virus confirming active swine influenza infection on the unit.

Reproductive failure and weak live-born piglets due to PRRS
Porcine reproductive and respiratory syndrome virus (PRRSv) was detected in all three foetuses tested from one of two litters sampled to investigate infertility, abortions mummification, stillbirths and poor viability piglets occurring in a weaner-producer unit in the Bury St Edmunds region. Sows were vaccinated against PRRSv and those of parity 1 to 3 were particularly affected. The virus showed very close similarity to that detected on a nursery-finisher unit receiving piglets from this breeding unit on which there was increased mortality as described below. An earlier submission of foetuses from this unit did not identify PRRSv but, on that occasion, only two foetuses were submitted, one of which was severely autolysed. This case demonstrates the value of submitting multiple freshly aborted litters to increase the chances of detecting an infectious cause, if present.

Alimentary Disease

Post-weaning mortality associated with PRRS challenge and enteric disease
Live pigs were submitted to Bury St Edmunds as part of a series from a continuous nursery-finisher site with a 24-month history of elevated mortality (varying from 8-15%) associated with respiratory disease and wasting in each three-weekly batch. The pigs were sourced from a single breeding herd and were vaccinated for porcine circovirus 2 (PCV2), Mycoplasma hyopneumoniae and PRRSv. All three submitted pigs were in poor body condition with the heaviest being 5.4kg at five-weeks-old and they had watery small intestinal contents from which monophasic Salmonella 4,12:i:- phage type 120 was isolated and no rotavirus, porcine epidemic diarrhoea (PED) or enteropathogenic Escherichia coli was found. PRRSv was detected in pooled serum from the pigs and sequencing revealed it was a field strain which showed a high degree of similarity to the viruses in two submissions to Bury St Edmunds in February and October 2014, and to the PRRSv strain detected on the breeding unit supplying this farm. Lung histopathology was suggestive of PRRS and the poor body condition of the piglets and detection of PRRSv early post-weaning suggest that the PRRSv was playing a role in the clinical disease prior to weaning, possibly prior to vaccination, although immunohistochemistry did not detect PRRSv in the lungs.

Subsequently another submission from this unit also diagnosed PRRSv infection, this time with immunohistochemistry confirming involvement of PRRSv in the pneumonia, with sequencing identifying the same virus strain as in the earlier submissions from this unit and the source breeding unit. On this occasion, all three pigs submitted had polyarthritis but no bacteria were isolated, probably due to prior antimicrobial treatment. The small intestine of one pig was thin-walled and distended with watery fluid and F4 antigen-positive E. coli was isolated in heavy pure growth from this pig, consistent with enteric colibacillosis. As in the previous submission, the five-week-old pigs submitted were significantly underweight with the heaviest being 4.4kg.

Monophasic Salmonella variants and S. Typhimurium detected in growers and finishers
Salmonellosis was possibly involved in a limited outbreak of acute diarrhoea in about 40 of 200 outdoor finishing pigs with monophasic Salmonella 4,5,12:i:- phage type 193 isolated by direct culture from all of four faeces submitted. No Brachyspira species or PED virus were detected. The pigs were unusually old to show clinical signs associated with Salmonella infection although farm staff had noted large starling populations which could have increased challenge or introduced a new Salmonella serotype, and no pigs
died to allow fuller investigation. The clinical disease resolved rapidly following inclusion of citric acid to bring the pH of their drinking water down to 4, it is unclear what role this intervention played in the resolution of disease but no antimicrobials were administered.

Two cases of monophasic *Salmonella Typhimurium* were identified from faeces samples submitted to Starcross from pigs with diarrhoea. In the first, seven-week-old pigs born outdoors were affected and the monophasic variant 4,5,12:i:- phage type 193 was isolated showing resistance to four antimicrobials on the antimicrobial sensitivity testing panel. The second case was diagnosed in three-month-old indoor pigs, and yielded the monophasic variant 4,12:i:-, also phage type 193, with resistance to three of the tested antimicrobials.

Six-week-old post-weaned pigs were submitted to Thirsk for post-mortem examination from a commercial unit of 900 sows. The farmer reported a problem with diarrhoea over the previous three weeks; however young pigs had not been doing well generally over the past few months. Circular, red ulcers and diphtheresis were visible on the large intestinal mucosa and colitis due to salmonellosis involving *Salmonella Typhimurium* Copenhagen was confirmed by culture.

**Respiratory Disease**

**Complex disease with underlying PRRS and mixed infections**

Multiple diseases with underlying PRRS were diagnosed in five-week-old pigs on a single-source indoor nursery-finisher receiving pigs weekly. Sneezing, weight loss and diarrhoea had developed within a week of arrival in the previous three batches of weaners and, in the batch from which pigs were submitted to Bury St Edmunds, 10% were affected and nine pigs had died from a group of 600. Pigs were vaccinated for PCV2 and *M. hyopneumoniae* but not for PRRSv which was detected by PCR and confirmed as causing respiratory disease by lung immunohistochemistry. In addition, *Streptococcus suis* 8 was isolated from the lung, a likely secondary pathogen in one pig and *Staphylococcus aureus* was involved in long-standing joint infections in two pigs, likely secondary to trauma while on the breeding unit. Interestingly, although not detected by PCR in the three submitted pigs, swine influenza was detected in an oral fluid sample from the affected pigs tested elsewhere, demonstrating the value of the wider sampling enabled through oral fluid collection. The continuous nature of this large unit, and the weekly introduction of significant numbers of weaners, both favour the persistence of viral and other infections from batch to batch.

**Sudden deaths and respiratory disease due to *Actinobacillus pleuropneumoniae***

Two plucks were submitted from eight-week-old pigs, four of which had died suddenly with a few others showing respiratory signs in a batch of 1000 on an indoor-nursery-finisher unit. About half of the lung mass was dark red and consolidated, with large focal areas of consolidation in the dorsal parts of both plucks and overlying fibrinous pleurisy suggestive of *Actinobacillus pleuropneumoniae* (APP) infection which was confirmed by culture, together with *Streptococcus suis* 2 from both plucks. PRRSv was also detected in one pluck, although immunohistochemistry did not confirm involvement of PRRSv in the pneumonia, the pig was not vaccinated and field challenge was likely. There was no evidence of PCV2 or swine influenza involvement.

**Swine influenza outbreak in late finishers**

Swine influenza was diagnosed when samples were submitted to Bury St Edmunds from 20-week-old housed finishers with respiratory disease and increasing mortality, in which PRRS was suspected. No PRRS was detected but histopathology revealed a bronchiolitis and immunohistochemistry confirmed swine influenza. The cause of death following swine influenza infection is usually a secondary bacterial infection, rather than the influenza virus itself; histopathology revealed an interstitial pneumonia which may have been due to systemic disease but no samples were submitted for culture.

**Severe outbreaks of pasteurellosis with PRRS**

Severe pneumonic pasteurellosis and PRRS were confirmed in two of three pigs submitted to Bury St Edmunds to investigate 13 sudden deaths in a 24-hour period from a group of 1000 nineteen-week-old pigs with others off their food with low level coughing and cyanotic or reddened skin. The pigs were on an all-in, all-out nursery-finisher unit and were vaccinated for *M. hyopneumoniae* and PCV2 but not for PRRSv. A third pig submitted died of massive haemorrhage from deep ulceration of the pars
oesophagea. The pigs with PRRS had marked fibrinous pleurisy and consolidated areas of lung were swollen and firm as illustrated in Figure 1. It is likely that the PRRS was exacerbating clinical disease due to the pasteurellosis and the virus detected showed close similarity to viruses in two submissions to Bury St Edmunds in February and March 2015.

Figure 1 Fibrinous pleurisy and pneumonia due to pasteurellosis and PRRS

Glässer’s outbreaks in weaner-growers
Glässer’s disease was diagnosed in a nine-week-old pig submitted to investigate lameness, recumbency and respiratory disease affecting about 30% of growers in outdoor tents with three deaths. There was a severe fibrinous polyserositis and pneumonia and *Haemophilus parasuis* was isolated, no viral involvement was detected. Interestingly, the isolate was resistant to potentiated sulphonamide which was present in feed.

The Royal Veterinary College investigated a problem of malaise and lethargy affecting about 10% of weaners purchased a few days earlier. Several batches of weaners were reported to have been affected since the farm stopped breeding its own piglets and started bringing in weaners from a single source. Two live piglets were submitted; one had fibrinous pericarditis, pleurisy, polyarthrits and pneumonia and was in poor body condition while the other piglet was markedly depressed but showed no gross findings. *Haemophilus parasuis* was isolated from both piglets confirming Glässer’s disease in one and *H. parasuis* septicaemia in the other. In-feed tetracycline was not reported to have produced a good clinical response, this may in part reflect poor feed intake by affected piglets as the *H. parasuis* isolate showed *in vitro* sensitivity to tetracycline. There was no evidence of viral involvement.

Multiple viral and bacterial infections involved in respiratory disease and mortality
A recurrent problem of ill thrift in weaned piglets was investigated by Thirsk. On-farm post-mortem examinations had revealed pneumonias and some pigs also had nasal discharges and occasional sneezing. Post-weaning mortality up to 30kg was around 10%. The piglets were vaccinated against PCV2 and *Mycoplasma hyopneumoniae*. Six and nine-week-old pigs were submitted which had reddened, mucus-covered nasal turbinates, cranioventral lung consolidation and varying degrees of pleuritis. Inclusion body rhinitis (cytomegalovirus) was confirmed by histopathology and swine influenza virus was detected in a six-week-old pig; while both PRRSv and APP were detected in a nine-week-old pig. Lung histopathology was also suggestive of underlying mycoplasmal involvement and DGGE confirmed the presence of *M. hyopneumoniae*. 
Systemic Disease

Streptococcal disease causing outbreaks of meningitis, endocarditis, arthritis and septicaemia

*Streptococcus suis* type 14 infection was identified as the cause of polyarthritis and septicaemia in seven-week-old pigs in which 5% were reported to be affected with lameness, some nervous signs and sudden deaths with a poor response to doxycycline. The organism was isolated from joints and liver and was sensitive to penicillin but resistant to tetracycline, explaining the lack of response seen on farm.

Three sudden deaths of well-grown 14-week-old pigs on an outdoor organic unit prompted their submission. Two different causes were found; one pig had vegetative endocarditis of the atrophic ventricular valve due to erysipelas (confirmed by isolation of *Erysipelothrix rhusiopathiae*) while the other two had *Streptococcus suis* type 2 meningitis confirmed when the organism was detected by FAT in smears from the meninges which appeared reddened with prominent blood vessels.

Large cauliflower-like vegetative endocarditis lesions were also found in two seven-week-old pigs submitted from a unit where 70% were described as affected with respiratory disease and wasting with 9% mortality on an outdoor nursery unit. *S. suis* type 2 was isolated from the heart lesions of one pig with *S. suis* also isolated from the second pig but the isolate remained acapsulate and untypeable. The third pig which was younger had died due to haemorrhage from a ruptured liver, possibly secondary to a septicaemia. No underlying viral disease was detected, however the clinical history on the unit suggests that the submitted pigs, which were all found dead and in fair or good body condition, were representative of sudden deaths, but probably not the wider respiratory disease.

Sudden deaths were reported in three-week-old piglets and cases were submitted to Thirsk. Meningitis was suspected and confirmed together with fibrinous peritonitis, pleuritis and pericarditis. Cultures from peritoneum, meninges and spleen yielded pure growths of *Streptococcus suis* type 1 which is more common in preweaned pigs like these than *S. suis* type 2.

Musculoskeletal Disease

Osteomyelitis in limb bones causing unresponsive lameness

An interesting case of pigs affected with septic arthritis and osteomyelitis due to *Trueperella pyogenes* infection (in association with *Fusobacterium necrophorum* at one site) was diagnosed, possibly occurring as a sequel to tail biting. Two 18-week-old pigs were submitted to Bury St Edmunds to investigate lameness and, in some pigs, lethargy and apparent ataxia occurring after the growers moved to the finishing unit at 12 to 15-weeks-old. The response to injectable antimicrobial treatments was variable and mainly unrewarding. Severe chronic suppurative joint infections were present in carpal and tarsal joints in both pigs but, in addition, both had foci of osteomyelitis not directly related to the joint infections and illustrated in Figures 2 and 3.

Figures 2 and 3 showing osteomyelitis lesions in long bones of forelimbs of pigs (arrowed)
Interestingly, both pigs had bitten tails and although the length of the tails made them appear that they had been docked, it was subsequently reported that they had not been docked on-farm, confirming that significant tail biting had occurred. The tail lesions may well have been the origin of infection through haematogenous spread of bacteria and this was highlighted to the attending practitioner. There were no other gross lesions to indicate that underlying metabolic bone disease was playing a predisposing role. Antimicrobial treatment of osteomyelitis is not usually feasible and affected pigs should be culled and the predisposing factors, in this case, likely tail biting, controlled or prevented.

**Ill thrift and lameness in growers associated with deficient home-mix diet**

A severe osteochondropathy was diagnosed in 16-week-old growing pigs from a small indoor breeder-finisher unit submitted to Penrith to investigate ill thrift and lameness, some with swollen joints. The pigs were fed a commercial diet with some home-mix feed for about two months after weaning and then changed to just the home-mix including soya and home-grown barley without mineral-vitamin supplementation. It was after this feed change that problems developed. A typical case was euthanased for submission and the significant gross findings included poor body condition and polyarthropathy with articular cartilage fissures affecting humeri, and the mandible broke with minimal manipulation. The pig had a low serum calcium concentration but bone analysis (bone ash, calcium, phosphorus and magnesium) was, surprisingly, unremarkable. Histopathology revealed severe osteochondropathy with osteoclasis, presumptive osteochondritis dissecans and growth plate arrest. The combination of lesions present was highly suggestive of metabolic bone disease which would include mineral, trace element and vitamin deficiency/imbalance. Further biochemistry revealed hypocuprosis and suboptimal selenium status in this pig and also in two others tested. The attending veterinary surgeon has advised appropriate dietary changes to address the problem and will review the progress of the next batches of growers.

Figure 4: Appearance of pig with lameness due to severe osteochondropathy (image kindly provided by the attending practitioner)

This is the third GB case over a six-month period relating to inadequate supplementation of home-mix diets for pigs and emphasises the need to ensure the diets of rapidly-growing pigs are suitably supplemented and provide adequate vitamins, minerals and trace elements.