

**VLA Monthly Surveillance Report for PIGS**  
**JUNE 2008**

*Defra Food and Farming Group funds the VLA's pig surveillance work as part of the Veterinary Surveillance Strategy*

## **Enteric Diseases**

### **Neonatal scour due to *E. coli***

Low grade diarrhoea in occasional litters on a 400-sow indoor breeding unit was investigated by submission of two faeces from two to four-week-old piglets. Profuse growths of K88 antigen positive *E.coli* were obtained from both faeces making enteric colibacillosis likely, no salmonella or rotavirus was detected. The possible involvement of coccidiosis could not be determined; this requires the submission of typically affected live piglets.

### ***Brachyspira pilosicoli*, Porcine Intestinal Spirochaetosis.**

Diarrhoea and weight loss in an unspecified number of 10 to 12-week-old housed finishers on a 120-sow unit was found to be due to *Brachyspira pilosicoli* which was identified by both PCR and culture.

## **Respiratory Diseases**

### **PRRS and PCV2.**

Concurrent PCV-2 and PRRS virus infection was diagnosed as the cause of illthrift with respiratory disease affecting 20% of 2000 rearing pigs with 7 to 8% mortality. Pigs were on a

continuous nursery finisher unit receiving 150 pigs every two weeks. There were mixed findings in four pigs submitted with variable degrees of pneumonia and some with pleurisy. *Pasteurella multocida* was isolated from all and a profuse growth of a group L streptococcus was isolated from joints and internal sites of a pig with polyarthritis and septicaemia. Histopathology confirmed severe subacute bronchointerstitial pneumonias with features typical of PCV-2 activity with numerous inclusion bodies. Necrosis of alveolar tissues raised the possibility of PRRS virus infection which was confirmed by lung immunohistochemistry and PCR.

### **PRRS**

PRRS virus infection was also implicated as the cause of acute coughing in 70 to 80% of 17-week-old finishers with low mortality. Three live pigs were submitted which were all found to have cranioventral pulmonary consolidation affecting all lung lobes. *Streptococcus suis* type 1 was isolated from one and a non-typable *Streptococcus suis* from another. All three pigs were positive for PRRS virus by PCR. Histopathology on the lungs revealed severe chronic bronchointerstitial pneumonias with widespread type 2 pneumocyte hyperplasia and a dearth of alveolar macrophages in two of the pigs, typical of PRRS virus infection. No swine influenza virus was isolated.

### ***Actinobacillus pleuropneumoniae***

Two grower pig carcasses were submitted for necropsy, with a history of respiratory disease and sudden deaths on an outdoor unit. *Actinobacillus pleuropneumoniae* had previously been detected from similar production stage pigs under the same ownership, but it was hoped that transferring this stage of production to a different site would assist control. The main findings were pulmonary consolidation and milk spot lesions (migrating Ascarids) in the liver. A *pleuropneumoniae* was isolated from one of the pigs and *Mycoplasma hyopneumoniae* from the other.

### **Swine Influenza and secondary bacterial infections.**

Three live, approximately 2½-3-week-old piglets were submitted for postmortem examination to investigate the cause of respiratory signs consisting of coughing, affecting approximately 15% of the group of 100 pre-weaned piglets. Coughing had been seen in the pre-weaned piglets over a 10 week period, with signs usually starting at between 3½ and 4 weeks of age just prior to weaning at 4½-5 weeks of age. Most losses of pigs seemed to occur after weaning with growth reduction and increased coughing. Dark purple/red colouration of the lung parenchyma with areas of consolidation were observed in all pigs but varied in severity. Influenza virus, identified as an avian-like H1 (H195852), was thought to be responsible for damage to the airways with secondary infection. *Haemophilus parasuis*, *Bordetella bronchiseptica*, *Streptococcus suis* type 8 and *Mycoplasma hyorhinis* were all isolated from lung culture. This case illustrates the multitude of pathogens that can be involved in secondary respiratory infection.

### **PRRS and *Streptococcus suis* serotype 2**

Five 3½ to 6-week-old piglets were submitted from a breeding and finishing unit to investigate illthrift, malaise and respiratory signs in piglets following transfer to flat deck weaner pens. Postmortem findings consisted of pneumonia and fibrinous pericarditis. *Pasteurella multocida*, *Arcanobacterium pyogenes* and *Streptococcus suis* type 2 were isolated from lung cultures. Spleen tissue from two pigs tested positive for PRRS by PCR and one pig also had histopathological lesions indicating a current active PCV-2 infection, particularly in the lung tissue.

### **Multifactorial Pneumonia with high mortality**

The carcasses of three live pigs were submitted with a history of respiratory signs and mortality within a week of weaning. The herd previously used homeopathic nosodes for enzootic pneumonia. Post-mortem examination revealed fibrinous pleurisy in one pig, clearly demarcated purple consolidation in another pig, and some fibrinous pleurisy in the third. PRRSV was isolated from all three pigs, and histopathological evidence of the presence of PCV2 was confirmed together with an interstitial pneumonia. What was interesting was the number of pathogens also isolated from these pigs, which included *Mycoplasma hyopneumoniae*, *Mycoplasma hyorhinis*, *Streptococcus suis* type 2, *Pasteurella multocida* and *Arcanobacterium pyogenes* from the lungs, and beta-haemolytic *E. coli* from the intestines. The assumption is that the viral agents caused marked immunosuppression, allowing the other agents to take hold.

## **Systemic Diseases**

### **Septicaemia due to *Salmonella Typhimurium* and *E. coli* O9**

Eight pigs in a group of 870 19-week-old finishers were found dead over 48 hours. Three of these pigs were found dead in a hospital pen having been treated for trauma and the two pigs submitted for necropsy were found dead in their pens. The pigs were from a single source and entering the unit at 56 days and receiving live in-water *Salmonella* Typhimurium vaccine on entry. The pigs were also vaccinated against PCV-2 at weaning. Both pigs had lesions typical of a septicaemia including pericarditis and one of the pigs had a severe necrotising typhlocolitis. *Salmonella* Typhimurium U288 was isolated in septicaemic distribution from both pigs and *Escherichia coli* O9:K103, 987p (formerly P16) was isolated from the kidney and pericardium of one pig. There was no evidence of immunosuppressive disease to account for these overwhelming infections with both pigs testing negative for PRRSV by PCR and with no histological evidence of PCV-2 infection.

### **Kidney failure due to bouts of septicaemia**

Two gilts found dead outdoors from a group of 150 were submitted. One in poor condition with a rectal prolapse had intestinal haemorrhage, oesophagitis and firm cream-coloured kidneys with dark speckling, suggestive of PDNS. Urea in aqueous humour was markedly elevated consistent with a uraemia which was likely to have caused mucosal damage resulting in some of the lesions seen. However histopathology did not confirm PDNS but revealed extensive fibrosis and chronic tubulointerstitial nephritis more indicative of repeated haematogenous/systemic insults and confirming severe renal damage which would have resulted in renal failure.

### **Necrotic hepatitis due to *Cl. novyi* “Aero chocolate liver”**

Aberystwyth investigated the sudden death of a well grown eight month-old Welsh gilt that was found dead. It was one of two homebred gilts being reared for breeding out of a herd of 80. The housing allowed access to pasture. There was severe congestion of the liver with large areas of small gas bubble filled tissue. There was a significant amount of blood stained peritoneal fluid. FAT on liver tissue was positive for *Clostridium novyi*. The findings were consistent with a diagnosis of sudden death due to clostridial necrotic hepatitis and toxemia due to *Cl. novyi* infection. *Cl. novyi* infection of this type is one of a few true causes of sudden death in growing pigs. Clostridial spores are more commonly acquired by pigs with access to outdoors.

### **Meningitis due to *Streptococcus suis* serotype 2**

A live 35 kg pig was submitted for investigation into a problem of recumbency and nervous signs in an outdoor rearing unit. It was unable to support its weight on the hind legs, but was otherwise bright and alert. At post mortem, it showed an umbilical infection, peritonitis and generalised polyarthritis and meningitis, *Streptococcus suis* type 2 was isolated. There was no evidence of PCV-2 involvement, but the herd is PRRS positive

## **Musculoskeletal Diseases**

### **Limb fractures due to slippery floors**

A 6-week old weaner pig was euthanased after having been found severely lame. The piglet had only been bought in 5 days earlier in a group of 32. Post mortem examination revealed the presence of a severe comminuted fracture of the right femur. The ribs appeared slightly bendy and of slightly rubbery consistency. Mineral and ash content in the bone were determined and within the expected reference ranges. No obvious trauma site could be detected during a farm visit. However, the pigs were sliding and falling onto the concrete floor when running and rubber mats have now been put onto the floor to improve grip.

### **Arthritis due to *Mycoplasma hyosynoviae***

A fifteen week old pig was euthanased after becoming progressively lame on the hind legs despite antibiotic and anti-inflammatory treatment. It was the only one of 120 affected. Grossly there were only minor changes in several joints indicating polyarthritis, with one stifle joint containing joint fluid with white flecks. *Mycoplasma hyosynoviae* was isolated from this joint by PCR and DGGE.

