REPRODUCTIVE DISEASES

PRRS abortions
PRRSV was detected by PCR in serum from aborting sows submitted to Thirsk to investigate abortions on a large weaner-producer herd. Late abortions, early farrowing and some non viable piglets were occurring. This is highly suggestive that PRRS was the cause of the reproductive failure, especially in the face of typical clinical signs. To confirm the diagnosis of PRRS, it is necessary to detect the virus in aborted, stillborn or non viable piglets.

Antibodies to swine influenza in sows
Similar to cases described previously, a problem of increased regular and irregular returns affecting 20 sows of a range of parities on an indoor 400 sow unit was investigated by submission of sera from returning sows. High antibody titres were detected to pandemic H1N1 2009 influenza virus. Whilst single serology does not provide evidence confirming the involvement of influenza infection in the infertility, as the timing of infection is unknown, the seropositive status demonstrates previous exposure of the sows to this virus.

DISEASES OF THE DIGESTIVE SYSTEM

Swine dysentery outbreaks
Swine dysentery outbreaks were confirmed on four units. Swine dysentery was confirmed in faeces submitted to Bury St Edmunds from growing pigs on an indoor unit with brown to bloody mucoid diarrhoea in 10% of pigs between eight and 22 weeks old. The history suggested that disease was likely to be from recrudescence of endemic infection on farm rather than from newly introduced infection. Swine dysentery was also confirmed by Langford when Brachyspira hyodysenteriae was isolated from a group of growing pigs on a large breeder-finisher unit.

Two swine dysentery outbreaks were diagnosed by Shrewsbury. In one of these, scour and mortality was reported in 16 week old pigs, with 20 deaths over four weeks from 60 pigs. Post mortem examination revealed colitis with marked thickening of the proximal large intestinal mucosa and areas of mucosal discolouration. There were severe Pasteurella multocida pneumonias which probably accounted for the high mortality. In the other, a fattening pig was submitted following an outbreak of scour in the farrowing sows and weaned pigs. Approximately half the pigs on the farm were affected with scour and reduced appetite for about three to four weeks before slowly recovering. Post-mortem examination revealed a thickened large intestine with mucoid contents and the isolation of B. hyodysenteriae confirmed swine dysentery.

RESPIRATORY DISEASES

Swine influenza in preweaned pigs
Active swine influenza infection was detected in pre-weaned pigs on a 100-sow closed indoor breeder-finisher. Nasal swabs were collected from piglets that had been seen to cough or sneeze in the previous five days and influenza virus was detected by PCR in two of the 11 swabs submitted. Selecting recently affected cases, preferably in the first two to three days of clinical signs, gives the best chance of detecting the virus which is excreted for approximately seven days. A spate of regular returns had occurred at the same time in sows over a four to five week period and titres to pandemic H1N1 2009 influenza virus were detected in breeding pigs, prompting the submission of nasal swabs for testing free of charge under the Defra-funded swine influenza surveillance project. For more details see http://vla.defra.gov.uk/science/docs/sci_si_info.pdf
Concurrent swine influenza and PRRS
Three eight to ten–week-old pigs were submitted to investigate a sudden upsurge in coughing and mortality on an indoor fully-slatted grower producer unit. Two pigs had severe respiratory pathology comprising generalised pleurisy and cranioventral consolidation affecting 30–40% of the lung fields. PRRSV and pandemic H1N1 2009 swine influenza virus were both detected by PCR. *Streptococcus suis* serotypes 7 and 8 were isolated from affected lung; likely secondary to the viral infections.

**SYSTEMIC & MISCELLANEOUS DISEASES**

**Klebsiella species septicaemia in outdoor pigs**
*Klebsiella pneumoniae* subspecies *pneumoniae* septicaemia was diagnosed in two submissions to Bury St Edmunds, each time in good condition pre-weaned pigs three to four weeks old found dead on outdoor units. One was diagnosed from swabs submitted from on-farm post mortem examinations, the other from dead pigs submitted for post mortem examination from a unit where deaths were only occurring in gilt litters with approximately 10% of litters affected and one to three piglets dying per affected litter. *Klebsiella pneumoniae* subspecies *pneumoniae* is a recognised cause of mastitis in sows and occasionally causes other infections in individual pigs, however it is unusual to find it causing outbreaks as on these units. Pure and profuse growths of the organism were obtained from multiple sites in several pigs. Various risk factors are being considered. Increased environmental levels of the organism could occur in moist or contaminated bedding, and contaminated wallows (hot weather increasing wallowing); ammonia favours growth of the organism and it would thrive in urine-contaminated wallows and bedding. Pig risk factors include the possibility that gilts have inadequate immunity to a resident strain, the presence of *Klebsiella* mastitis or high levels of vaginal carriage in gilts. No concurrent disease was detected in the submitted pigs. Investigations continue.

**Erysipelas in an unvaccinated herd**
Erysipelas was the cause of septicaemia and death of a one month old piglet in a small herd of four sows. The piglet was one of a litter of 15 and another pig in the group was very lethargic. They were in a field during the day and housed at night. At necropsy at Carmarthen, the spleen was very enlarged and there were renal haemorrhages. *Erysipelothrix rhusiopathiae* was isolated from multiple sites, confirming the septicaemia. The herd was not vaccinating sows against erysipelas and it was suggested that colostral immunity from sow vaccination would help control disease in younger piglets.

**Clostridial disease**
*Clostridium novyi* was diagnosed as the cause of sudden death of a sow. Despite having only been found dead a few hours before submission, the carcase was severely autolysed, as is often the case in clostridial disease. The liver parenchyma had the appearance of “Aero” chocolate (see Figure 1 Cross section of liver from a sow with *C. novyi* hepatitis.

Image courtesy of RVC/AHVLA surveillance centre
Figure 1). Fluorescent antibody testing confirmed the diagnosis. Where deaths due to *C. novyi* become a problem, vaccination can be implemented.

**Multiple outbreaks of disease due to *Streptococcus suis***

There were several incidents of disease involving *Streptococcus suis* types 2 and 1/2 in weaned pigs. Factors that precipitate *Streptococcus suis* sepsicaemia include transport, overcrowding, poor ventilation, high humidity and dust levels, intercurrent disease and mixing of pigs. In a typical case, six of 50 weaners aged 5-6 weeks old died. Gross findings were non specific and *S. suis* type 2 was isolated from brain, lung and meninges consistent with streptococcal meningitis and sepsicaemia.

Streptococcal meningitis was also diagnosed in a 17-week old pig submitted from a breeder-finisher unit with an increased incidence of nervous disease. Twelve pigs had died in the past month, some found dead, others seen recumbent with convulsions. In the nervous system there was marked engorgement of the meningeal vessels and an excess of cerebrospinal fluid. *S. suis* type 2 was detected by FAT and a heavy pure growth of *S. suis* type 2 was obtained.

A nine-week-old pig was presented, the second death in a group of three pigs bought in at five weeks of age from one source. The skin of the ventral abdomen and thorax was reddened, multiple joints contained purulent material, and there was fibrinous peritonitis and pericarditis, pneumonia and congested meninges. *S. suis* type 2 was isolated in septicaeic distribution.

**CARDIOVASCULAR DISEASES**

**Mulberry heart disease**

Mulberry heart disease was diagnosed in several submissions. In a typical example of these, three five-week-old pigs in good body condition were found dead from a group of 700. Post-mortem examination showed evidence of heart failure with marked excesses of pleural and pericardial fluids with fibrin stranding, lung oedema, and prominent haemorrhages with patchy discolouration of the heart. These findings were suggestive of Mulberry heart disease, no significant organisms were cultured and histopathology revealed moderate peracute necrotising and haemorrhagic cardiomyopathy with Mulberry heart disease being the most likely cause. Mulberry heart disease in the rapidly growing pig may be associated with dietary vitamin E and/or selenium deficiency but it is not uncommon to find liver or serum vitamin E and selenium values within reference ranges. It has been suggested that disease may also occur as the result of altered metabolism, for example increased detoxification demands or rapid growth; or following a period of greater requirement for vitamin E or selenium, for example, during activation of the immune system in infectious disease outbreaks.

**NEUROLOGICAL DISEASES**

**Congenital tremor type A2**

A 250-sow breeding herd had several litters affected by congenital tremor. 40 out of 100 piglets were affected, and ten had died. There were no significant gross findings on post mortem examination of a one-day-old piglet submitted to Preston but a deficiency of stainable myelin was demonstrated in the spinal cord, consistent with congenital hypomyelination, of which porcine congenital tremor A2 is the commonest cause. Brain histopathology revealed cerebellar cortical degeneration characterised by Purkinje cell necrosis. This type of cerebellar lesion is unusual in porcine congenital tremor and further piglets are being examined. The cerebellar lesions were not like those seen in CSFV-associated cerebellar dysgenesis.

**Bowel oedema**

Three dead four- to six-week-old weaners were submitted to Thirsk to investigate nervous signs, deaths and conjunctivitis in a 1,800-pig place nursery where accommodation was mainly in straw yards. A previous outbreak had responded moderately well to amoxicillin treatment. Pigs were
reported to stagger, 30 pigs had died and 50 were affected. All three pigs showed blotchy red cutaneous erythema covering up to 30% of the skin surface and conjunctivae were swollen. Culture of faeces yielded a profuse and predominant growth of a beta-haemolytic *E. coli* which was identified as *E. coli* O138: K81 (E57) while cultures from brains did not reveal any significant pathogens and an FAT for *Streptococcus suis* type 2 was negative. On this basis, a diagnosis of bowel oedema was made. A change in diet was suspected to have triggered the clinical signs.