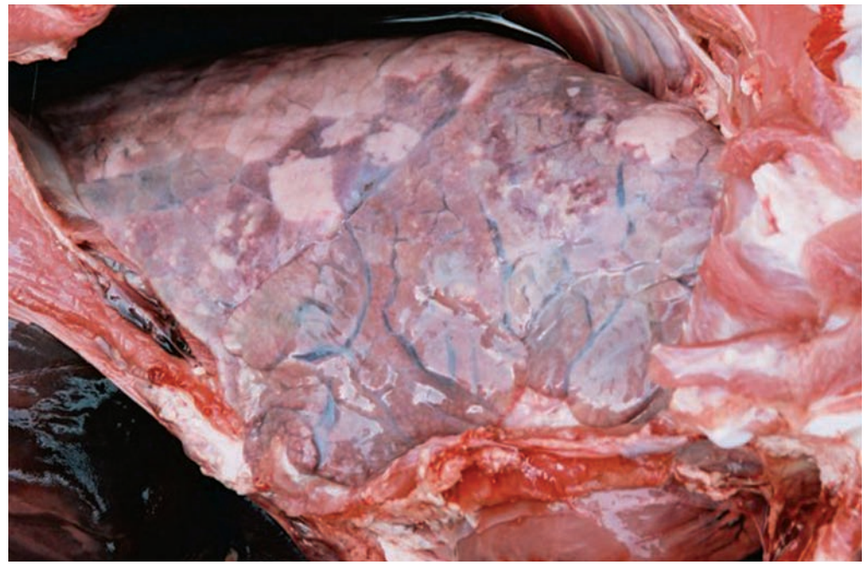
# Effective pneumonia control is an essential part of cost-effective production

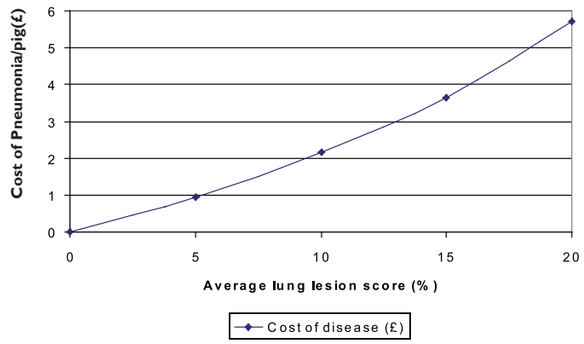
health MATTERS

How vaccines and antimicrobials can successfully tackle pneumonia in UK herds, by **David Burch**, Veterinarian, Octagon Services

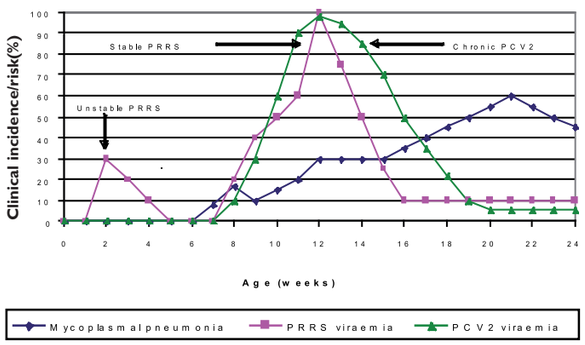


Extensive lesions of PCV2 complicated enzootic pneumonia

Back in the eighties pneumonia in pigs was relatively simple, most of it was enzootic pneumonia (EP) based *(Mycoplasma hyopneumoniae)* with a few complications. In the 1990s, PRRS virus (porcine reproductive and respiratory syndrome virus) came along and radically changed our perceptions of respiratory disease. Then, in the 2000s, we have had PMWS (post-weaning multi-systemic wasting syndrome) caused by PCV2 (porcine circovirus type 2) sweeping across our national herd and complicating matters even further.




**Graph 1. Cost of enzootic pneumonia/pig finished by average herd lung lesion score**




**Graph 2. Disease patterns (% affected) associated with mycoplasmal (enzootic) pneumonia and PRRSV and PCV2 viraemia**

Over 90% of herds are still infected with *M. hyopneumoniae*, which causes the classic, purplish lesions affecting the ends of the lung lobes. In chronically affected breeder-finisher herds, it was suspected that the initial transmission was passed from the sow to the piglet by 21 days of age and as maternally derived antibodies faded they were then vulnerable to the development of the disease, particularly at times of overcrowding and inadequate ventilation e.g. at the end of the weaner or grower stage when coughing would start. Lesions take some time to develop post infection, between 10-21 days, they peak at about 28 days when the pig's immunity starts to control them and resolution of the lesions can take several weeks. It is usually a chronic disease, which damages the tracheal lining and reduces the pig's physical and immune defences allowing secondary bacteria, such as *Pasteurella multocida* to invade the lung and double the extent of the pneumonic lesions and clinical effect. *Actinobacillus pleuropneumoniae* also played an important role in pneumonia in finishing pigs in about 30% of herds. The disease was frequently more acute than EP and the lesions were distinctive, as they affected the main body of the lung and also caused pleurisy. EP lesions largely remain small, involving on average 10% of the lung, but in severe cases it increases to 50%. An average herd lung score of 10% would reduce the growth in finishers by about 5% (see Graph 1) and on average cost about £2.15/pig finished in reduced FCE and increased mortality.

When PRRS virus hit, after the initial reproductive problems, the levels of respiratory disease increased. In the early stages respiratory signs, such as acute coughing and difficulty in breathing with high temperatures were seen in piglets as young as 16 days of age. The virus damaged the respiratory tract and the defence mechanisms and several bacteria made the most of this and attacked, in particular *Streptococcus suis* and *Haemophilus parasuis*. This still occurs in piglets when the sow herd's immunity to PRRSV is unstable either from poor gilt integration or in unvaccinated herds.

When the sows are vaccinated maternal antibodies protect the piglets for several weeks, but this wanes at the back end of the weaner or nursery stage and a full-blown infection takes place at about 8-10 weeks of age (see Graph 2).

In the UK, we seem to be lucky that the strains we have are not as severe as in the USA and we seem to cope quite well. The damage to the lung macrophages, the cells which eat up the bacteria and mycoplasma, lasts for about 28 days, so the pigs are susceptible to increased levels of bacterial attack.

The disaster associated with PMWS or PCV2 infection started in the UK in 1999. The acute phase spread around the country and caused mortality in young pigs, upto 30%. This acute phase has largely passed and we are now in the chronic phase. Most sow herds have a degree of immunity, the early reproductive effect has largely passed and we are facing the chronic effects such as immuno-compromised grower/finisher pigs with extensively damaged immune systems. In severe cases this can lead to the characteristic wasting, even death and overall reduced growth rates.

Recent Danish work has shown that piglets are infected with the virus in the first week of life colonising the nose. It does not appear to become viraemic until maternal antibodies have waned at about 8-9 weeks of age. Either the pig will control the infection and recover or the immune system is so badly damaged that it cannot produce a response to the virus and it goes on to overwhelm the pig, with the viraemia peaking at about 12 weeks of age. The pneumonia that results from a mixture of virus, mycoplasma and bacteria can involve almost 80-90% of the lung, before it dies (see Photo 1) and the average herd weaning to slaughter mortality in the UK has increased from 5.9% to 9.9% due to this disease.

Currently, enzootic pneumonia is controlled by good mycoplasma vaccines, which have reduced the average lung lesion score to about 3%, which is very successful.

Occasionally, medication is also required where environmental conditions are inadequate. Another approach has been eradication of the *M. hyopneumoniae* so that farms do not need to continue vaccination. The use of partial depopulations of the young stock (under 10 months of age) and the medication of the remaining breeding stock with such products as tiamulin (Denagard® – Novartis) have proven highly successful and popular over the years in many countries in Europe (see Table 1) and there is currently no resistance development.

There have been a lot of new strains of PRRS virus developing in the UK and some have shifted immunologically quite a long way from the original vaccine virus strain. On average, reports suggest that the modified live vaccines are still 70% effective in reducing pneumonia, but the response can be quite variable. Zonal eradication is being considered in the USA. The exciting new developments regarding PCV2 vaccines are already hitting the headlines both in the USA and Europe and their introduction, especially of the piglet vaccines, will have a major impact on mortality and pneumonia in the UK herd.

Pneumonia in pigs has been with us for a long time and although substantial advances are being made, I am sure it will remain a problem for the veterinarian and farmer for many years to come.

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**Table 1. Comparative susceptibility of 21 Belgian M. hyopneumoniae isolates to various antimicrobials (Vicca et al, 2004)**

| Antimicrobial | MIC 50% (µg/ml) | MIC 90% (µg/ml) | MIC range (µg/ml) |
|---|---|---|---|
| Denagard | 0.015 | 0.12 | 0.015-0.12 |
| Oxytetracycline | 0.12 | 1.0 | 0.03-2.0 |
| Lincomycin | 0.06 | 0.06 | 0.06->8.0 |
| Tilmicosin | 0.25 | 0.5 | 0.25->16 |
| Tylosin | 0.03 | 0.06 | 0.015->1.0 |

MIC = minimum inhibitory concentration; 50%, 90% - of isolates