

Control of PCV2 associated disease through vaccination

by David Burch, director, Octagon Services Ltd, The Round House, The Friary, Old Windsor, Berkshire SL4 2NR, UK.

Coming from a background of mainly clinical development rather than basic research, I have often been confused over the years by the scientific reports on the relationship of porcine circovirus type 2 (PCV2) and the two major disease syndromes associated with the infection, namely, post-weaning multisystemic wasting syndrome (PMWS) and porcine dermatitis and nephropathy syndrome (PDNS).

The 'x' factor

The pathologists and virologists could see the infection in the tissues and isolate the virus. They could put it back into pigs and sometimes cause the disease, but usually not, and kept on talking about factor 'x', which may be another virus or something else.

Other 'co-factors' such as porcine parvovirus (PPV), porcine reproductive and respiratory syndrome virus (PRRSV), immuno-stimulation from vaccines, and immuno-suppression from stress all seemed to help express the disease.

There were no vaccines being pro-

duced commercially and people were resorting to serum therapy from 'immune pigs' or autogenous vaccines from ground up lymph nodes, in sheer desperation.

Many producers had tried to introduce the 'Madec Principles' of good husbandry and stress reduction and to a variable degree had been quite successful after the initial sweep through of infection with accompanying high mortality. Subsequently, immunity built up in the herd and the levels of disease fell, in some cases almost back to normal.

So, what had happened? Many authors said the same virus had been in pigs for 20 years or more, yet it had spread across the UK and the world like a brand new infection.

There has been some progress on strain identification but these have not provided the complete answer yet.

Sow group in the UK

My frustration with the disease arose when I worked closely with a 12,500 sow group in the UK and subsequently tried to reduce their average finisher mortality/cull rate from 9% and wasting pigs of a further 9% over a 20 month period (see Fig. 1).

The sows were kept outdoors; the

	Before	After	Difference
Mortality (%)	6.95	4.15	2.80
Hospitalised (%)	10.58	8.66	1.92
Total (%)	17.53	12.80	4.73
Lung lesion score	11.5	3.7	7.8 (-68%)

Table 1. Effect of EP vaccination on mortality, pigs hospitalised and lung lesion scores based on 20,000 finisher pigs.

piglets were weaned at four weeks of age into straw based nurseries in approximate groups of 100 pigs and then went on to straw based finisher sheds, which housed approximately 2,200 in pens of 100 and were taken through to approximately 120kg liveweight.

The difficulties involved in reducing this problem were enormous, with pig (over)flow problems, stress, overcrowding, solid floors/scrape-through dunging areas and other diseases.

The nursery problems were minor with mortality regularly under 1%, but in the finisher, in spite of extensive cleaning and hygiene methods between batches, the infection pressure and resulting disease due to PMWS was very high and difficult to control.

A number of attempted control methods were introduced, such as, PRRS vaccination, split sex rearing (males are uncastrated in the UK), disease resistant breeds and slatted

dunging areas, but to no avail.

Enzootic pneumonia (EP) vaccination had a moderate affect reducing mortality by 2.8% and hospitalisations by 1.9% and reducing lung lesions scores at slaughter by 68% (Table 1).

Efficacy in finishers

I was not completely convinced of the efficacy in finishers by the original reports on sow vaccination coming from France, in before and after studies, involving 4,800 sows as the finisher mortality appeared to remain high.

The problem we saw was mainly in the finisher pigs (>10 weeks of age) and the nursery pigs had only a mortality of 1%. I thought by this stage that maternally derived antibodies (MDAs) would have waned and were no longer protective and this appeared to be the case (Fig. 2).

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Fig. 1. Typical mortality/cull and hospitalisation observed in the finishing shed.

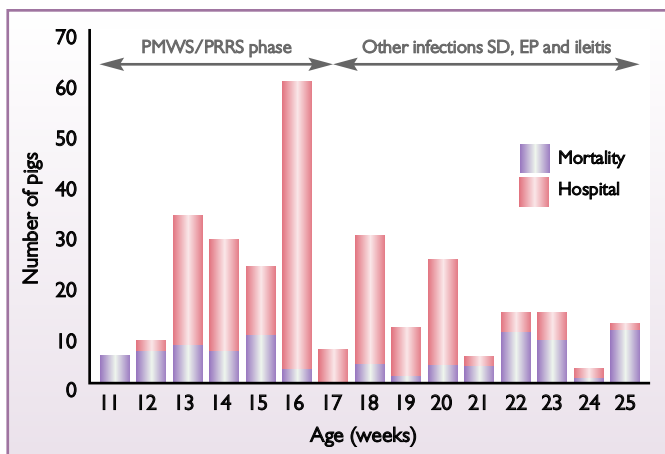
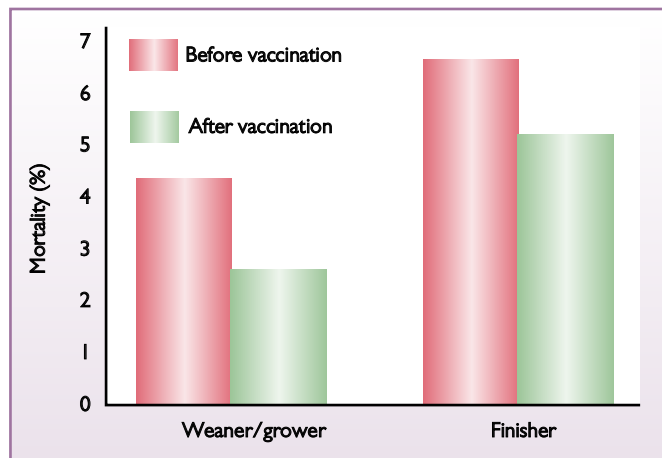


Fig. 2. Effect of sow vaccination on weaner/grower and finisher mortality (Auvigne and others, 2006).



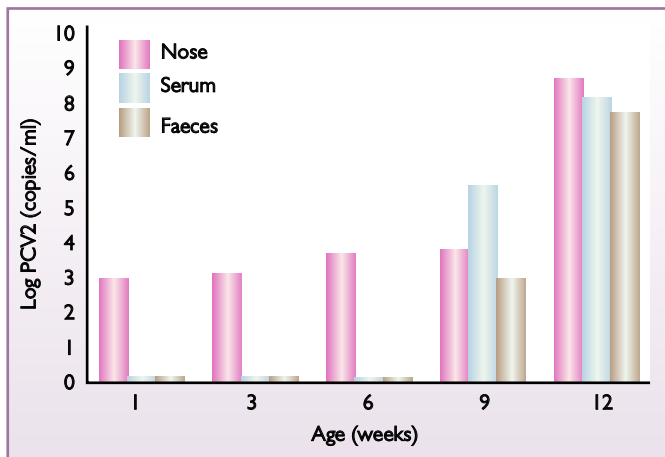


Fig. 3. PCV2 colonisation and spread in the nose, serum and faeces in PMWS affected pigs (Hjulsager and others, 2007).

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Work by Hjulsager and others, 2007, showed that in naturally occurring PCV2 infections, once antibodies decline, a viraemia usually takes place and if the challenge is sufficient an overwhelming viraemia (week 12), which leads to PMWS, can occur (Fig. 3).
In pigs which did not go down with PMWS much of the colonisation was the same but the viraemia was controlled and falling at this time.

Early spread of infection

This work is also important in that it highlights the early spread of the PCV2 infection to piglets via the nose and presumably the control of the infection systemically by MDAs but the battleground is when they decline and the pig has developed sufficient immunity to combat the infection.

Increasingly, I am coming across herds which suffer a growth check, presumably at the time of viraemia (8-12 weeks of age) and frequently this is associated with an increase in diarrhoea, in spite of medication for ileitis or colitis, which normally occurs at this time and some of the

pigs go on to develop PMWS and related problems.
It was, however, a revelation on a visit to a trial site in 2006, to see the impact of piglet vaccination with a PCV2 piglet vaccine (Ingelvac CircoFLEX – Boehringer Ingelheim) in a closely monitored UK trial carried out to Good Clinical Practice (GCP) standards, where the weaning to slaughter mortality was reduced from 14.3 to 4.6%.

Two consecutive week batches of pigs comprising approximately 770 pigs per batch were balanced according to bodyweight, litter and sex and were equally distributed between the two treatment groups, the vaccinated and the placebo injected controls, which was given at three weeks of age (Study week 0) before weaning at four weeks of age.

Table 2. Comparative mortality/culls (%) in vaccinated and control groups (Desrosiers, and others, 2007).

	Controls (%)	Vaccinated (%)	Difference (%)
Barn 1	9.6	3.0	6.6
Barn 2	8.1	2.1	6.0
Barn 3	10.6	2.8	7.8
Barn 4	7.6	0.4	7.2
Total	9.5	2.4	7.1 (p = <0.001)

Fig. 5. Number of pigs in the vaccinated and the control groups surviving during the trial.

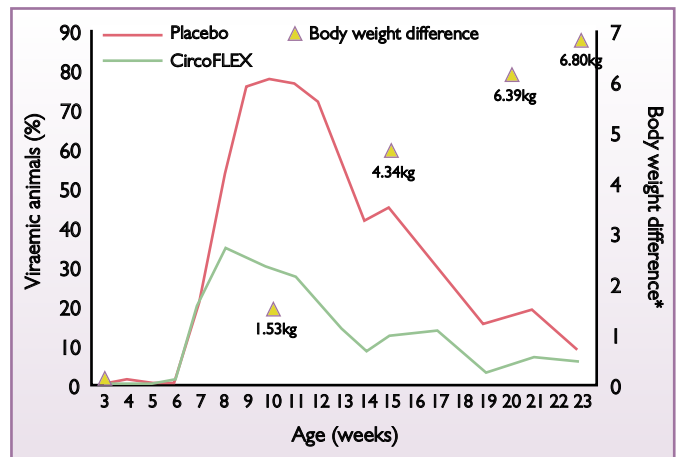
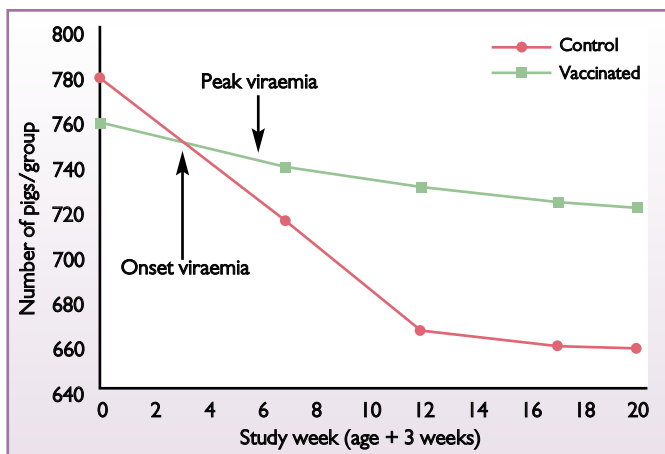


Fig. 4. Comparison of mean viraemic levels and weight gain difference in the vaccinated and control pigs (*vaccinated - placebo).

They were then transferred to the nursery and finisher site which was separate from the breeding herd. The herd was free from PRRSV and Mycoplasma hyopneumoniae so that the effect of other diseases on mortality and performance could be excluded. The pigs from both treatment groups were kept in the same pens so that they were exposed to the same level of infection and environmental conditions.

Weekly examinations

The pigs were weighed on Study week 7, 12, 17 and 20 and examined clinically on a weekly basis. Mortality was recorded and individual and dead and culled pigs were necropsied. Blood samples were taken from a representative group on a

weekly basis until week 12 and every other week thereafter for virus examination by quantitative PCR. The PCV2 viraemia started at about Study week 4-5 and peaked at 5-6 (Fig. 4).

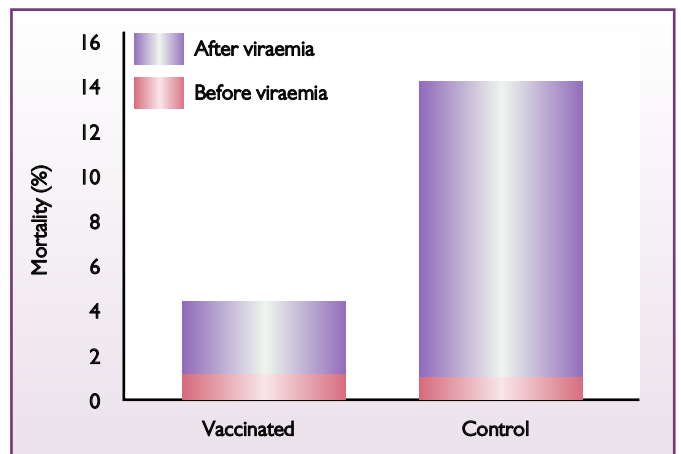
The viraemia started at the same time and the vaccinated pigs had a lower mean level and peaked approximately a week earlier than the controls. The vaccine primes the immune system and this responds more quickly when the viraemia starts.

The mean level is lower throughout the viraemic phase in comparison with the unvaccinated pigs and the number of pigs with a high level of viraemia (>10⁶GEs), which is associated with clinical disease.

The number of pigs surviving in each group is shown in Fig. 5 and the mortality before and after the onset of viraemia is shown in Fig. 6.

A dramatic reduction in mortality was seen in the vaccinated animals after the onset of viraemia. A difference in growth rate was also noticed following the onset of the viraemia (Fig. 7). There was a mean difference in liveweight at Study week 20 of 6.6kg in the favour of the vaccinated pigs, but there were statistically significant differences from

Fig. 6. Mortality in vaccinated and controls before and after the onset of viraemia.



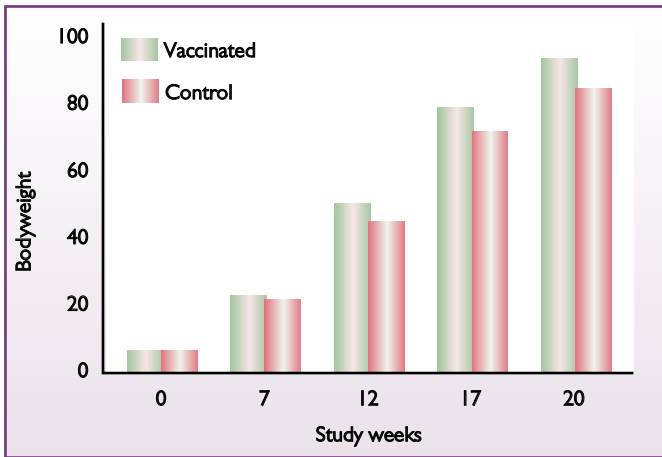


Fig. 7. Bodyweight of vaccinated and control pigs at Study week 0, 7, 12, 17 and 20.

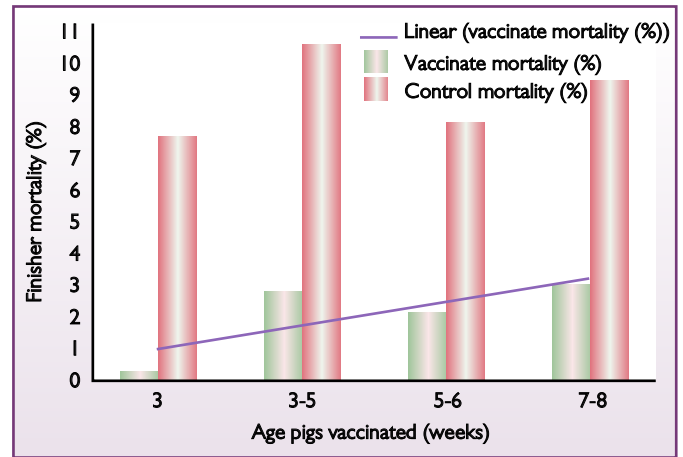


Fig. 8. Comparative mortality/culls (%) in vaccinated and control groups (Desrosiers, and others, 2007).

week seven. These are exceptional results in the face of a severe PCV2 challenge and clearly demonstrate the value and efficacy of the piglet vaccine for protection throughout the finishing period.

Further field studies have also been reported from North America where PMWS has more recently become a problem and they confirmed the UK study results and also demonstrate a consistency of protection right throughout the finishing period to slaughter. A trial was carried out on a 1,300 sow multi-site

unit, which had had PMWS for 18 months. The losses usually started in the finishing barns at three to four weeks after placement, which was normally at nine weeks of age. Four barns of decreasing ages were used in the trial.

Barn one pigs were 45-59 days of age; Barn two pigs were 38-45 days old; Barn three pigs were 22-36 days old and Barn four pigs were 19-22 days of age. The trial involved 3,850 pigs, which were divided into two groups, those that were vaccinated and those that were given a placebo,

as a control, but the groups were kept in separate pens.

The farm was also PRRSV and EP negative, so the effects of vaccination were primarily on the PCV2 infection. The results are summarised in Table 2 and Fig. 8.

There was a significant reduction in mortality from 9.5-2.4%, which could be considered a normal mortality rate for finishing pigs.

Piglets vaccinated at 19-22 days of age were protected at least as well as pigs vaccinated at a later age.

Maternal antibodies did not appear

to interfere with the vaccine. There were no reported adverse reactions to the vaccine and the incidence of PDNS subsided.

In spite of the early confusion of the pathogenesis of PMWS and PDNS, it now can be clearly seen that PCV2 was responsible for causing the overwhelming infection, increased mortality and decreased growth rate and these can be effectively blocked by early piglet vaccination. ■

References available on request