The solution for the whole herd

MERIAL ANIMAL HEALTH

Merial's François Joisel opened his company's symposium at the IPVS congress with a presentation on PCV2 vaccination and reproductive improvement in sows. The first report of reproductive failure came from a farm in western Canada in 1999. PCV2 was isolated from a litter of aborted piglets in a farm that was experiencing late term abortions and stillbirths in which concurrent infections with PPV, PRRSV, EMCV and enterovirus had been ruled out.

It was shown that PCV2 inoculated into foetuses at different stages of pregnancy in PCV2 seropositive sows resulted in stillborn, mummified, weak and live born piglets depending upon the stage of pregnancy at the time of infection. PCV2 was shown to be able to multiply in foetal tissues, especially the heart. Since then many other studies have confirmed PCV2 as a major aetiological agent in foetal deaths.

Experimental intranasal infections of pregnant sows have resulted in abortions, premature births and stillbirths.

Whether the virus can cross the placenta is still debated. In some trials abortions followed elevated body temperature without PCV2 being found in foetal tissues, whereas in other trials detection of viral protein and DNA in tissues of stillborn and live piglets suggested that PCV2 may be capable of crossing the placenta.

An increasing number of reports of PCV2 in boar semen are being published and inoculation of sows with PCV2 via an AI catheter resulted in the sows becoming seropositive to PCV2 and two of them aborting. So, it was concluded that sows and foetuses could be infected via semen, seroconvert and experience a transient hyperthermia that may result in abortions.

Against this backcloth Francois went on to consider vaccination of sows with Circovac. He cited a large German field trial whose results are summarised in Table 1 and are self-explanatory.

Edison Bordin from Merial Brazil then reflected on the use of Circovac in piglets in various countries including Brazil and these results are summarised in Tables 2 and 3.

Tanja Opriessnig then compared piglet and sow vaccination. The main advantage of sow vaccination is a reduction in labour and costs and possibly earlier protection in the piglets. She then highlighted a recent study

Continued on page 16

Index	Before vaccination	After vaccination
Farrowing rate (%)	81.1	83.2
Return to heat (%)	14.4	11.5
Abortions (%)	1.95	1.38
Total born per litter	11.8	12.3
Live born per litter	10.9	11.4
Dead born per litter	1.39	1.29
Weaned per litter	9.4	9.9
Weaned per sow per year	21.2	22.4

Table 1. Summary of results of large German field trial with Circovac.

Table 2. Pig performance in the Brazilian trial.

Treatment	Mortality (%)	Weaning weight (kg)	Nursery exit weight (kg)	Nursery DWG (kg)	Nursery FCR	Final weight (kg)	Total DWG (kg)	Total FCR
Circovac	3.16	5.222	20.829	0.354	1.504	90.565	0.688	2.242
Vaccine X	3.33	5.243	20.306	0.342	1.574	88.580	0.672	2.244
Vaccine Y	2.50	5.233	20.905	0.356	1.511	90.226	0.685	2.270

Treatment	Collection time (days)				
	21	36	66	96	126
Circovac	61.9	289.4	263.5	281.2	353.7
Vaccine X	89.8	168.5	184.6	281.0	212.6
Vaccine Y	48.1	203.0	129.8	212.3	159.4

Table 3. Serology results (mean antibody titres) from the Brazilian trial.

	Progeny from sows before Circovac vaccination	Progeny from sows after Circovac vaccination
Birth to slaughter ADG (g per day)	540	567
Killing out (%)	76.4	77.4
Medication costs (£ per pig per quarter)	0.36	0.03
Pleurisy at slaughter (%)	34	3
Viral lesions at slaughter (%)	32	0
M. hyopneumoniae consolidation score	6.5	2.6

Table 4. UK case study 1 – performance benefits of sow vaccination.

	Control (No vaccination)	Sows vaccinated
Birthweight (kg)	1.53	1.54
Weight at one week (kg)	2.84	2.94
Weight at three weeks (kg)	6.07	6.32
Mortality to weaning (%)	15.6	12.3

Table 5. UK case study 2 – pre-weaning piglet performance.

Continued from page 15
of hers at Iowa State University in which two different vaccinations (sow and piglet) were compared. In addition, the efficacy of vaccinating piglets with the same vaccine that was used in the dams was evaluated in PCV2 naive sows that were vaccinated at 28 and 93 days of gestation using 2ml of Circovac or were not vaccinated. It was shown that non-vaccinated piglets born from vaccinated sows demonstrated significant protection. Passive transfer of immunity was proven, since these piglets showed levels of ELISA and seroneutralising antibodies upon challenge comparable to the piglets vaccinated at three weeks of age. After challenge, the level of PCV2 viraemia was significantly reduced compared to control piglets.

After challenge, they displayed more than 80% reduction of PCV2-specific microscopic lesions compared to control piglets. Therefore, vaccination of the sow with Circovac was able to protect piglets against a heterologous PCV2 challenge up to eight weeks of age.

Jake Waddilove, a practising veterinarian from the UK, then focused on the sow option and cited two cases studies from the UK whose outcomes are detailed in Tables 4 and 5. In concluding the symposium Catherine Charreyre from Merial highlighted what we know, what we think we know and what we still do not know about PCV 2 (see inset, right). ■

What we know:

- PCV2, the species, is an homogeneous viral population infecting swine and only swine, the world over.
- Infection with PCV2 is not always associated with disease; a lot of pigs are infected without symptoms. Some co-factors inducing immunostimulation are necessary for the clinical expression of the disease.
- Early exposure to PCV2 can lead to PMWS and associated diseases in pigs at anytime in the pig's life.
- The main known pathogenic effect of PCV2 infection is impairment of dendritic cells (NIPC) functions.
- Protection against PCV2 associated diseases can be linked to passive and active antibodies and cell mediated immunity against PCV2.
- In field situations, a low level of maternal immunity is consistently associated with disease.
- Current vaccination programs are efficacious and beneficial.

What we think we know:

- We have a scientifically established system to classify the various PCV2 isolates into sub-groups, but it could evolve.
- We have established a first link with some particular genes and disease, but this remains to be confirmed and clarified.
- We have ways to establish a PCVD diagnosis in single pig cases and even in herds, but they are cumbersome, costly and often debatable.
- It is unsure whether a high PCV2 virus load in organs is the cause of severe clinical cases or rather a secondary consequence of immune dysfunctions.
- Infection of pregnant females with PCV2 induces reproductive failures, but we have not clarified the underlying mechanism behind those symptoms.

What we still do not know:

- We do not know where PCV2 came from and why it 'suddenly' emerged.
- We do not know how to induce disease after PCV2 infection in animals older than 6-8 weeks.
- We do not know whether different breeds are actually sensitive to PCV2 infection in different ways.
- We cannot predict whether and how a pig infected with PCV2, and even less a pig herd, will eventually get diseased.
- Although impairment of dendritic cells functions indeed affects the core of the pig immune system, it is unclear how PCV2 infection further produces the devastating tissues lesions seen in severe cases.
- We do not know how to evaluate immune profiles in field conditions.
- We cannot predict how PCV2 epidemiology will evolve in the next few years.