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Experience of Vaccination against Porcine Parvovirus in Pig-Breeding Herds: Serological Status and Reproductive Performance

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Einarsson, S., K. Larsson and B. Thafvelin: Experience of vaccination against porcine parvovirus in pig-breeding herds: Serological status and reproductive performance. Acta vet. scand. 1987, 28, 279–284. – Blood samples from 77 gilts were examined for HI-antibody titers to PPV, all gilts belonged to the same herd and PPV-induced reproductive failure had previously occurred in the herd. Thirty-three gilts were vaccinated twice 5 and 2 weeks before mating while 44 gilts served as non-vaccinated controls. Only 3 % of the vaccinated gilts were seronegative at the time of mating compared to 14 % of the non-vaccinated gilts and 32 % of the non-vaccinated gilts had a serum titer lower than 1:64.

The second part of the study comprised 4 herds with 50–70 sows in each herd. All of the herds had previously had reproductive problems caused by PPV infection. During the last 2 years, all gilts in these herds were vaccinated against PPV at 6.5 months of age with a revaccination 3–4 weeks later.

There was a marked variation in serum titer levels among the 4 herds. In two herds the titers were overall rather low. In the third herd all had high PPV-titers at both sampling occasions and in the fourth herd the titers varied among animals but were rather consistent within animals at the two sampling occasions. In the herd with high titer, a PPV-outbreak was confirmed during January-March 1984. During that period all sows, vaccinated as gilts, farrowed normal litters.

The results indicate that even in PPV-infected herds a large number of gilts are seronegative at the time of breeding and vaccination of gilts is therefore recommended. Furthermore it does not seem necessary to revaccinate sows, vaccinated as gilts, in herds where PPV is still present.

sow; PPV-infection; litter size; HI-titer.

Introduction

Porcine parvovirus (PPV) causes reproductive failure in swine. The virus is ubiquitous among swine throughout the world and is enzootic in most herds, shown by the serological surveys that have been conducted, using the haemaglutination inhibition (HI) technique (*Joo & Johnson* 1976). It was associated with swine reproductive failure in 1967 by *Cartwright & Huck.*

The virus is able to pass through the placenta of pregnant sows and infects the foetuses. The infection results in early embryonic death, mumification and stillbirth (*Johnson & Collings* 1971). Animals with immunotolerance to PPV, as a result of early in utero infection, have been suggested as symtomless carriers of virus (*Cartwright et al.* 1971, *Johnson et al.* 1976). The most

common route of infection for postnatal pigs is oronasal.

Pigs nursing immune dams absorb antibodies against PPV from the colostrum. These titers decrease progressively and most gilts have lost their passive immunity by 21 weeks of age, but some gilts retain low levels of PPV antibody titers until 36 weeks of age (*Johnson et al.* 1976). Thereafter the pigs are susceptible to PPV infection. As exposure to PPV is common in pig herds, a large proportion of gilts are naturally infected with PPV and develop an active immunity before conception (*Mengeling* 1972). The proportion of gilts seronegative to PPV within 1 week after mating was approximately 20 % in 2 large farms in Australia and USA respectively (*Cutler et al.* 1982). The percentage of small litters ($\leq$ 6 born alive) in the groups of pregnant gilts that seroconverted during gestation, was 36.6 % compared with 10 % in the gilts that remained seronegative throughout gestation (*Cutler et al.* 1982). Vaccination is the only way to assure that gilts develop active immunity before conception and thereby avoid intrauterine infection (e.g. *Joo & Johnson* 1977, *Mengeling et al.* 1979, *Sørensen & Askaa* 1981).

The objectives of the present field study were to:

- test the serostatus of PPV of vaccinated and non-vaccinated gilts at the time of mating in one large swine herd,
- test the serostatus to PPV in sows vaccinated as gilts,
- evaluate the effect of vaccination, for prevention of PPV-induced reproductive failure, in some swine herds.

Materials and methods

In one large herd, which earlier had experienced reproductive failure induced by PPV, blood samples from 77 gilts were examined for HI-antibody titers to PPV prior to mating. Thirty-three gilts were immunized twice subcutaneously with parvovirus vaccin after the age of 6 months approximately 5 and 2 weeks before mating using PARVO VAC©*. Forty-four gilts served as non-vaccinated controls.

Part two of this study comprised 4 herds (I, II, III and IV) varying in size between 50 and 70 sows. All reproductive and production data were carefully recorded. Before the vaccination program was started the herds suffered from more or less severe problems associated with embryonic death caused by PPV-infection (according to laboratory examinations). Since 2 years all gilts were vaccinated with a PPV-vaccine. The same vaccination program was used in this part of the study that means no vaccination before 6 months of age. No sows were vaccinated. The boars were vaccinated twice before they were allowed into the gilt/sow units. In February and July 1984 blood samples were collected from a number of randomly selected vaccinated pregnant sows in each of the herds. The sampled sows were of different ages (2nd to 5th gestation) and all samples were collected at mid-gestation. PPV antibody titers were determined by the haemagglutination inhibition test (HI). Farrowing results were collected from the records of the herds during 1984 (January to August) for all pluriparous vaccinated sows for analyses. Also data from non-vaccinated older sows belonging to herd III were collected and compared with those from vaccinated sows during a PPV-caused outbreak of reproductive failure. Foetuses and stillborn piglets were sent to the virus laboratory (National Veterinary Laboratory, Uppsala) for verifying the diagnosis.

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* PARVO VAC supplied by Norden Lab., Novo, Malmö, Sweden.

Results

The serological status in unmated gilts belonging to one large herd is presented in Table 1. Only 1 of 33 (3 %) vaccinated gilts remained seronegative (titer ≤ 1:8). Among the non-vaccinated gilts 32 % had a titer ≤ 1:64, and 14 % were seronegative.

Table 1. Antibody titers to PPV in vaccinated and non-vaccinated gilts at time of mating. 2nd vaccination given approx. 2 weeks before sampling.

HI-titer	Vaccinated	Non-vaccinated
≤ 1:8	1	6
1:16	– 3 %	1 32 %
1:32	–	4
1:64	–	3
1:128	4	3
1:256	16	3
1:512	–	5
1:1024	2	–
≥ 1:2048	10	19
Total number	33	44

The PPV-titers of sows belonging to herds I–IV are presented in Tables 2 and 3. In these herds all sampled sows had been vaccinated as gilts prior to first mating. Herds I and II had on both sampling occasions rather low serum titers to PPV. However, only a few sows were seronegative. In herd III all sows had high PPV-titers on both sampling occasions indicative of a recent PPV outbreak. All serum samples except 1 had PPV-titers exceeding or equal to 1:2000. The only blood sample with negative PPV-titer was collected from a sow which 5 months earlier (1st sampling) had a titer above 1:8000. The PPV-titers in herd IV varied between animals at both sampling

Table 2. Changes in PPV-titers occurring in pluriparous sows, vaccinated as gilts, in 4 different herds.

Herd	Month of sampling	No. of samples	Numbers of sows with antibody titers ≤ 1:64	1:128–1:256	1:512–1:1024	> 1:1024
I	Febr.	14	5	9	0	0
	July	11	0	8	3	0
II	Febr.	10	2	7	0	1
	July	9	4	3	2	0
III	Febr.	15	0	0	0	15
	July	10	1	0	0	9
IV	Febr.	13	3	5	2	3
	July	8	1	3	2	2

Table 3. PPV-titers in pregnant sows, vaccinated as non-mated gilts.

Age (litters born + one)	No. of sows	PPV-titers, % of sows ≤ 1:64	1:128–1:256	1:512–1:1024	> 1:1024
2	32	25.0	34.4	9.4	31.3
3	31	12.9	51.6	9.7	25.8
4–5	26	11.5	30.8	11.5	46.2

Table 4. Farrowing results (means ± S.D.) during 1984 (January through August) of PPV-vaccinated pluriparous sows in herds that were serologically investigated.

Herd	No. of litters	Live born	Still born	Litters ≤ 6 live born %
I	35	10.5 ± 2.5	1.6 ± 1.9	8.6
II	23	11.6 ± 4.1	1.3 ± 1.9	8.7
III	35	11.3 ± 2.3	0.4 ± 0.8	0
IV	26	9.7 ± 2.7	0.1 ± 0.3	7.7

Table 5. Farrowing results of pluriparous sows between January 15 and March 15 1984 in herd III.

Sows	No. of litters	Live born		Still born	
		Mean	Range	Mean	Range
Vaccinated	6	10.2	8–13	0.2	0–2
Non-vaccinated	17	10.1	6–14	2.5	0–12

occasions giving a very irregular pattern. The titers were almost unchanged within animals, however.

As is evident from Table 3 the PPV-titer was approximately the same in the different age classes of sows, vaccinated as unmated gilts. The tendency, if any, was an increasing PPV-titer by age.

The farrowing results from vaccinated pluriparous sows are presented in Table 4. None of the herds had a percentage of litters with less than 6 live piglets at birth exceeding 10 %.

An outbreak of PPV-infection with reproductive problems occurred in herd III between January and march 1984. Several older non-vaccinated sows farrowed increasing numbers of mumified and/or stillborn piglets. PPV antibodies were detected in stillborn piglets from non-vaccinated sows. All vaccinated sows farrowed normal litters during corresponding period (Table 5). The PPV-titers were high in non-vaccinated as well as in vaccinated sows during this period.

Discussion

A large proportion of the non-vaccinated gilts had, judging from their high antibody-titer, been naturally infected with PPV and developed an active immunity prior to mating. This result is in accordance with some earlier reports. *Mengeling* (1972) suggested that well over one-half of all gilts in areas where PPV is enzootic are infected before they are bred for the first time. Approximately 1/3 of the non-vaccinated gilts, on the other hand, had low PPV-titers or were seronegative. *Paul et al.* (1980) suggested that passively immunized pigs with HI-antibody titers of 1:80 or less, are susceptible to PPV-infection. If this is true under Swedish conditions, it means that every third gilt is susceptible to PPV-infection at breeding age. Pigs that seroconvert to PPV during gestation have a higher number of mumified foetuses and a significantly lower number of piglets born alive per litter (*Cutler et al.* 1982).

All but one of the vaccinated gilts were seropositive to PPV at sampling prior to their

first breeding. The lack of response to vaccination in that animal may be due to maternal antibody interference at the time of vaccination (*Sørensen & Askaa* 1981). There are different opinions about the minimum titer of PPV-antibody of actively immunized pigs, necessary for protection against transplacental PPV-infection. Antibody titers as low as 1:10 (HI) could according to *Mengeling et al.* (1979) inhibit transplacental foetal infection.

The majority of the sampled sows had positive PPV-titers. Recently performed comparative studies with HI-titration and the highly specific and sensible competitive ELISA using monoclonal antibodies detect specific HI-antibodies at the dilution 1:64 (*Juntti et al* 1986). The pattern on a herd basis was, however, different between the 4 herds. The reason for this might be different pressure of natural PPV-infection within the herds. General health status, management, animal contacts with other pig herds etc. will certainly influence the spreading of virus among the pigs and the maintenance of immunity. One of the herds (III) had extremely high PPV-titer on both sampling occasions indicating a high pressure of natural PPV-infection. Interesting is that older non-vaccinated sows belonging to this herd farrowed increasing number of mumified foetuses and stillborn piglets during a 2-months period, coinciding with the sampling period. The reproductive performance of the sows, vaccinated as gilts, was not affected. The PPV-titers were as high in the vaccinated as in the non-vaccinated sows. *Sørensen & Askaa* (1981) demonstrated a 2- to 8-fold increase of HI-titer in vaccinated gilts after challenge, indicating an active immune response. Transplacental infection and foetal death did no occur. It has even been speculated that once the immune system has been primed with PPV, subsequent exposure to virulent virus during gestation is unlikely to result in transplacental infection, even if antibody from vaccination is no longer detected (*Mengeling et al.* 1979).

The percentage of small litters ($\leq$ 6 born alive) should no exceed 10 % in a swine herd (*Cutler et al.* 1982). A higher frequency is proof of an infectious reproductive disease e.g. PPV-infection. The low frequency of small litters among the vaccinated sows in the present study therefore indicates a satisfactory protection against transplacental infection of PPV in all age classes of vaccinated sows. The PPV-titer was approximately the same in the different age classes of sows within herds. Judging from the reproductive performance as well as from the PPV-titers in sows vaccinated only as unmated gilts, revaccination of breeding animals seems not to be necessary in herds where PPV is still present.

Conclusions

A large proportion of gilts in PPV-infected herds are seronegative or have a too low PPV-titer at breeding age and are therefore susceptible to infection during their first gestation. Vaccination of all gilts before breeding is therefore recommended. Revaccination of breeding animals seems not to be necessary, in herds where PPV is present.

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Sammanfattning

Erfarenheter av vaccination mot porcint parvo virus i suggbesättningar: Serologiskt status och fertilitet.

Ändamålen med föreliggande undersökning var att 1) undersöka serologiskt status avseende PPV hos vaccinerade och ovaccinerade gyltor vid tiden för betäckning i en stor svinbesättning, 2) undersöka serologiskt status avseende PPV hos suggor som vaccinerats som gyltor och 3) utvärdera effekten av vaccination för förebyggande av PPV-inducerade fruktsamhetsproblem i några svinbesättningar.

Blodprover från 77 gyltor undersöktes med avseende på HI-titer mot PPV. Samtliga gyltor tillhörde en stor besättning som hade haft problem med PPV-inducerad nedsatt fertilitet. Trettiotre gyltor vaccinerades två gånger (5 och 2 veckor) före betäckning och 44 gyltor utgjorde ovaccinerade kontrolldjur. Den andre delen av undersökningen utfördes i 4 besättningar med 50–70 suggor per besättning. Samtliga besättningar hade tidigare haft problem med PPV-infektion och sedan två år vaccinerades samtliga gyltor vid 6,5 månaders ålder med omvaccination 3–4 veckor senare. I februari och juli 1984 togs blodprover, för bestämning av PPV-titrar, från ett antal slumpvis utvalda dräktiga suggor. Suggorna var av olika åldrar (2:a till 5:a dräktigheten) och proverna togs under mitten av dräktigheten. grisningsresultat från besättningarna analyserades för perioden januari–augusti 1984 för samtliga äldre vaccinerade suggor. I en besättning analyserades också grisningsresultat från äldre ovaccinerade suggor under samma period beroende på ett PPV utbrott bland dessa djur. Endast 3 % av de vaccinerade gyltorna var seronegativa vid tiden för betäckning jämfört med 14 % av de ovaccinerade och 32 % av de ovaccinerade gyltorna hade PPV-titer mindre än 1:64.

I de 4 undersökta besättningarna sågs en avsevärd variation i PPV-titrar. I två av besättningarna var samtliga titrar låga. I den tredje besättningen var samtliga titrar höga vid båda provtagningstillfällena och i den fjärde besättningen varierade titrarna mellan de undersökta djuren, men inte mellan provtagningar inom samma individ. I besättningen med genomgående höga titrar uppträdde PPV infektion, bekräftat genom laboratorieundersökningar av foster, bland äldre ovaccinerade suggor under januari–mars 1984. Samtliga vaccinerade suggor som grisade under denna period gav normala kullar.

Slutsatserna av undersökningen är att relativt många gyltor är seronegativa vid tiden för första betäckning och generell vaccination av gyltor kan därför rekommenderas. Det synes däremot inte vara nödvändigt att revaccinera äldre suggor, som vaccinerats som gyltor, i besättningar där PPV finns närvarande.

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