2009

Selection for Immune Responses to Porcine Circovirus (PCV2) to Decrease Incidence of Porcine Circovirus Associated Disease (PCVAD)

Jared S. Bates
University of Nebraska-Lincoln

Roman Moreno
University of Nebraska-Lincoln

Alan R. Doster
University of Nebraska - Lincoln, adoster1@unl.edu

Rodger Johnson
University of Nebraska - Lincoln, rjohnson5@unl.edu

Selection for Immune Responses to Porcine Circovirus (PCV2) to Decrease Incidence of Porcine Circovirus Associated Disease (PCVAD)

Selection for 65-day weight along with PCV2 viremia and antibody titers offers great potential to decrease incidence of PCVAD.

Jared S. Bates
Roman Moreno
Alan R. Doster
Rodger K. Johnson[1]

Summary

Genetic and environmental effects on incidence of Porcine Circovirus Associated Disease (PCVAD) and immune responses to Porcine Circovirus 2 (PCV2), and their relationships with body weights were studied in 3,440 pigs of the Nebraska litter size selection lines. Pigs were weighed at birth, weaning, 65, and 180 days of age and scored for symptoms of PCVAD every 10 days from 70 to 180 days of age. Necropsies were performed to confirm accuracy of scoring. PCV2 viremia, and antibodies to PCV2, Porcine Reproductive and Respiratory Syndrome Virus, Mycoplasma hyopneumoniae and Actinobacillus pleuropneumoniae were measured in serum from blood samples drawn at various ages from live pigs and in tissues of pigs necropsied. PCV2b genotype was confirmed to be the pathogen causing PCVAD; other pathogens studied were not involved. Pigs with no symptoms of PCVAD had significantly greater weights (0.22, 1.12, 6.6, and 46.0 lb, at birth, weaning, 60 d, and 180 d, respectively) than affected pigs. Heritability of PCVAD score was 16% ± 4%. The location in which pigs were raised accounted for 22% of the variation in PCVAD score. Nearly all pigs were non-viremic until 90 days of age, but many had antibody titers at weaning and at 60 days of age. These maternal antibodies appeared to protect pigs from PCVAD until approximately 90 days of age. Heritability of viremia level at 90 days of age was greater than at 125 days of age (38 ± 11% vs 11 ± 8%). Genetic variation existed for antibody titers at 90 (h^2 = 55 ± 21%) and 125 days of age (h^2 = 10 ± 8%). Incidence of PCVAD was correlated genetically with body weights, PCV2 viremia level, PCV2 antibody titers, and body weights. Expected response to direct selection for reduced PCVAD score was very low (-0.89% in one generation), whereas expected response to index selection for 65-day weight and PCV2 viremia and antibody titers at 90 days of age was -8.0%, 998% greater than direct selection. Genomic selection for decreased incidence of PCVAD is feasible.

Introduction

Porcine Circovirus Associated Disease (PCVAD), caused by Porcine Circovirus 2 (PCV2), causes high economic losses to pork producers. Symptoms of PCVAD in the University of Nebraska–Lincoln swine research herd were first observed in 2002. Not all pigs on the farm were infected and the incidence rate varied depending on the genetic makeup of the pigs, their location at the farm, and season of the year. The incidence rate in crossbred pigs was very low, but a significant number of pigs of the UNL lines selected for increased litter size were affected. Some pigs seemed to be highly sensitive to the disease whereas others in the same pen remained healthy and showed no symptoms. Usually, only one or two pigs in a pen were affected, but it was common for a high percentage of pigs within some litters to be affected, even when raised in different locations.

These observations pointed to underlying genetic variation in the immune response of pigs to the PCV2 virus. A study of PCVAD conducted at another institution supports this hypothesis as the incidence rate was greater in some breed crosses than others. However, sample size in that experiment was too small to determine the degree of genetic variation (heritability) in incidence rate of PCVAD and in immune responses to PCV2 virus. If sufficient genetic variation exists, then greater resistance to PCV2 and reduced incidence of PCVAD through selection are possible. When practiced in nucleus breeding populations, greater resistance achieved through selection can be transmitted through the breeding pyramid to commercial producers, possibly reducing the need for vaccination.

We therefore conducted a study in which pigs were systematically scored for PCVAD, weighed and bled to create a database for genetic analyses. Because secondary pathogens are often thought to be involved in expression of PCVAD, pigs were also characterized for Porcine Reproductive and Respiratory Syndrome Virus (PRRVS), *Mycoplasma hyopneumoniae* and *Actinobacillus pleuropneumoniae* to determine whether these pathogens were involved along with PCV2 in expression of PCVAD.

(Continued on next page)

Population

Pigs were from generations 24 to 26 of UNL selection lines, Lines 2 and 45, and control lines, Lines 16 and 61, that were derived from a Large White-Landrace composite population formed in 1979. Lines 2 and 45 have been selected for increased litter size (generations 1 to 20) and increased litter size, increased growth, and decreased backfat, generations 21 to 27. Lines 16 and 61 were randomly selected. Each line is maintained with 40 to 45 litters per generation. Line 2 and 16 litters were born in July and August (denoted contemporary group 1, CG1) and Line 45 and 61 litters were born in January and February (CG2).

Pig Management, PCVAD Scoring, and Serum Sampling

Within two days of farrowing, fostering of pigs among sows both within and between lines was practiced. Fostering could not be accomplished uniformly in all litters. Total number and number of live pigs per litter, pig birth (n = 3,440) and weaning weights (n = 3,438), and number after foster and weaned per foster dam (NW) were recorded.

Pigs were weaned at approximately 17 days and grouped by age in nursery pens of 30 pigs per pen. At 60-65 days of age, four boars from each of the 15 largest litters and four to five females from each of the largest 20 litters in Lines 2 and 45 were identified as candidates for selection as breeders. Two sons per sire and one to two gilts per litter were randomly selected as candidates for breeding in Lines 16 and 61. Final selections occurred at 180 days of age.

All breeder candidates were placed in one of six rooms, eight pens per room and 10 pigs per pen, in a confined, mechanically ventilated building denoted as Location 1. Remaining pigs were placed in one of three locations. Location 2 was a confined building, 25 pens of 10 pigs per pen, with natural ventilation regulated by thermostatically controlled curtains over windows. Location 3 was a confined building, 23 pens of 10 pigs per pen, with natural ventilation controlled manually by adjusting doors over windows. Location 4 was five outdoor lots containing a small hoop structure with straw bedding. There were 50 to 60 pigs per lot. Pigs in CG1 were in Locations 1, 2, and 3; pigs in CG2 were in Locations 1, 2, and 4.

Pigs with symptoms of PCVAD were first observed in 2002 (generation 21). Observations in subsequent years led us to a protocol that produced data for genetic analyses. During Generations 24, 25, and 26 pigs were systematically weighed, scored for PCVAD, and blood samples were drawn. Weight at 65 days was recorded for 2,646 pigs (some pigs were not weighed) when they were placed in finishing pens and weight at 180 days (n = 3,115) were recorded. Beginning 7 days after pigs were placed in finishing pens, they were scored for symptoms of PCVAD once every 7 to 10 days, approximately 10 scores per pig, until 180-day weight was recorded. Pigs with no symptoms received a score of 0, pig with minor symptoms a score of 1, and pigs with definitive symptoms a score of 2. Scores were based on degrees of muscle wasting, growth retardation, rough hair coat, diarrhea, and respiratory distress. A score of 1 was used only to identify pigs for more careful future observation. Only pigs receiving one or more score of 2 were considered positive for PCVAD.

Blood was collected at 60, 90, and 125 days of age from all pigs from Generation 25, CG2, and Generation 26, CGs 1 and 2. Blood was collected from pigs at weaning in Generation 25, CG2 and from sows in Generation 26, CG1 when their pigs were weaned. PCV2 viremia, a measure of the pigs ability to replicate virus, was measured in serum of all pigs with PCVAD score of 2, in serum from a randomly selected pen mate with scores of only 0, in samples of a full sib from another pen, and in samples from two pigs drawn randomly from each birth litter in which no pigs were positive for PCVAD. Serum was sent to Iowa State University Veterinary Diagnostic Laboratory, Ames, Iowa, where Porcine Circovirus II C-ELISA PCR and PCV2 Quantitation (qPCR) were performed to obtain PCV2 viremia and antibody levels.

ELISA was used to test subsamples of serum for M*ycoplasma hyopneumoniae* (MH), *Actinobacillus pleuropneumoniae* (APP), and Porcine Reproductive and Respiratory Virus (PRRSV). Samples collected at 90 (n = 261) and 125 days (n = 228) from pigs from generation 25, CG2, were tested for MH; samples collected at 125 days of age from generation 25, CG2 (n = 228) and Generation 26, CG1 and CG2 (n = 511) were tested for APP. The UNL swine herd is free of PRRSV. To confirm this status, serum from 52 pigs from generations 24 to 26 was tested for PRRSV.

Necropsies were performed on samples of pigs with a PCVAD score of 2 from generation 24, CG1 (n = 10) and CG2 (n = 11), and generation 25, CG2 (n = 17) and in 11 randomly selected pigs with PCVAD score of 0 from generation 25, CG2. Pigs were from all locations and only one pig from any one litter was selected for necropsy. Immunohistochemistry and RT-PCR for PCV2 were performed in lung, cervical lymph node, mesenteric lymph node, tonsil, kidney, and ileum of these pigs. Nasal swabs for RT-PCR testing were collected from five pigs; two of these pigs had no lesions suggestive of PCVAD. Necropsies and RT-PCR were done at the Veterinary Diagnostic Center of the University of Nebraska Department of Veterinary and Biomedical Sciences. These pigs were also tested for PRRSV antibodies by ELISA. Serum from three pigs that were necropsied and that were diagnosed with PCVAD were submitted to the Veterinary Diagnostic Laboratory at Iowa State University, Ames, Iowa where Porcine Circovirus II C-ELISA PCR-PCV2 Quantitation in which the virus was sequenced to determine the specific PCV2 transcript in this herd.

Statistical Procedures

Data were analyzed with procedures appropriate for genetic analyses. Two traits had binomial distributions as the outcome was either yes or no, coded as 0 or 1. These were PCVAD score (1 = positive, 0 = negative) and

Table 1. Number of observations for each trait by generation and contemporary group.

Generation	Group[2]	Trait[1]				
		BWT	WWT	W65	W180	PCVAD score
24	1	669	669	—	659	669
24	2	649	649	543	622	649
25	1	281	281	281	262	281
25	2	511	510	510	431	511
26	1	629	629	624	507	629
26	2	701	698	688	634	701
		Vsows	VW	V60	V90	V125
25	2	—	279	287	261	228
26	1	77	—	292	294	244
26	2	—	—	—	217	211
		IgGsows	IgGWIg	G60Ig	G90Ig	G125
25	2	—	280	287	271	229
26	1	75	—	301	294	244
26	2	—	—	211	217	210

[1]BWT = birth weight; WWT = weaning weight; W65 = weight at 65 d; W180 = weight at 180 d; Vsows = sow viremia at weaning; VW = pig viremia at weaning; V60, IgG60, V90, IGg90, V125 and IgG125 = PCV2 viermia and antibody, respectively, at 60, 90, and 125 days of age.
[2]Group1 = Lines 45 and 61 (CG2, winter litters), Group 2 = lines 1 and 2 (CG1, summer litters).

Table 2. Distribution of pigs with combinations of PCVAD score, virema, and IgG levels at days 90 and 125 days within generation (G) and contemporary group (CG) and overall.

	PCVAD score[1]	Viremic[2]	IgG[3]	G25CG1	G26CG1	G26CG2Total
Day 90						
1	+	+	11	16	0	27
1	+	S	48	64	0	112
1	+	-	27	44	0	71
1	-	+	4	4	14	22
1	-	S	11	3	8	22
1	-	-	7	0	3	10
0	+	+	36	59	1	96
0	+	S	55	68	0	123
0	+	-	18	21	1	40
0	-	+	15	10	103	128
0	-	S	19	5	57	81
0	-	-	11	0	30	41
Day 125						
1	+	+	59	80	9	148
1	+	S	8	3	3	14
1	+	-	3	0	0	3
1	-	+	12	2	0	14
1	-	S	1	0	1	2
1	-	-	0	0	4	4
0	+	+	104	145	78	327
0	+	S	12	3	17	32
0	+	-	0	0	12	12
0	-	+	29	11	13	53
0	-	S	0	0	19	19
0	-	-	0	0	55	55

[1]0 = negative and 1 = positive score for PCVAD.
[2]+ indicates PCV2 viremia level > 0, - indicates PCV2 viremia = 0.
[3]+ = IgG titer ≥ 0.5, S = 0.2 ≤ titer < 0.5, - = titer < 0.2.

whether a pig was viremic (0 = no viral replication, 1 = viral replication, veremia level greater than 0). For those pigs that were viremic (coded score of 1) the observed viremia (genomic copies per ml) were expressed as $\log_{10}$ to normalize the distribution. PCV2 antibody titers and body weights were normally distributed.

Table 1 contains a description of traits and numbers of records. Table 2 contains the joint distributions of PCVAD scores, PCV2 viremia, and PCV2 antibody titers. Genetic analyses used a pedigree file containing 12,032 pigs, all those with phenotypes in the present study and all parent animals tracing back to the base generation. Traits analyzed were PCVAD Score, birth weight, weaning weight, 65-day weight, 180-day weight, viremia scores at 90 days and 125 of age (the 0 or 1 code), $\log_{10}$ of viremia level at 90 days and 125 days of age in pigs with positive viremia scores, and PCV2 antibody titers at 60, 90, and 125 days of age. Antibody titers .5 and greater are considered to be positive, evidence that the pig had been exposed to virus, titers of .2 to .49 are in the suspect range, and those below .2 are considered to be negative.

Results

Observations and Fixed Effects

Overall, 14.4% of pigs had at least one positive PCVAD score, but the incidence varied greatly across generations (Figure 1). Mortality rate of pigs with positive score was 35.4%. Genetic lines did not differ significantly in incidence of PCVAD.

Nearly all serum samples collected at 60 days of age (94.6 %) were negative for PCV2 viremia; therefore viremia at 60 days of age was not analyzed. All but two serum samples collected at 90 days of age from pigs in generation 26, CG2 had negative viremia (Table 2); therefore, data from that group were deleted from analyses of 90-day viremia level. All but two serum samples collected from weaned pigs were negative for PCV2 viremia, but 37 of 77 of their dams were positive. Average antibody titers for sows and

(Continued on next page)

progeny were 1.07 ± 0.15 and 0.90 ± 0.16, respectively.

Antibody titers for PCV2 at 60, 90, and 125 days of age for each contemporary group are shown in Figure 2. Titers at 60 and 90 days were similar in generation 25, CG2, and generation 26, CG1, greatest in Generation 26.

Frequency of viremic pigs varied greatly across generations, contemporary groups, and ages (Figure 3). In generation 26, CG2, 53.9% of the pigs were non-viremic at 90 days of age but had antibody titers greater than 0.5 (Table 2). Only 5.9% of the pigs in other groups were non-viremic and had high antibody titers. Non-viremic pigs at 125 days of age with antibody titers less than 0.2 occurred in 60.8% of pigs from generation 26 CG2, but in only 10.2% of the pigs in the other groups.

Incidence of PCVAD was greater in males than females and males had greater ($P < 0.05$) PCV2 antibody titers at 60 (0.03) and 90 days (0.05). The probability of being viremic at 90 days of age was less for females than males (-0.30, $P < 0.05$).

Pigs with 0 PCVAD score weighed more ($P < 0.0001$) at birth, weaning, 65 d, and 180 days (0.22, 1.12, 6.6, and 46.0 lb, respectively) than pigs with score of 1. They also had lower PCV2 viremia levels at 90 (0.26 ±0.15) and 125 days (0.85 ± 0.12) and greater antibody titers (0.04 ± 0.01 and 0.05 ± 0.02, respectively).

PCV2 Sequence

All three pigs whose PCV2 mRNA was sequenced were positive for PCV2b genotype. The sequence for one pig was 100% identical to a PCV2 isolate previously characterized and described in the National Center for Biotechnology Information database. The sequences for the other two pigs were not 100% identical to any sequence in the database. One of them had a single base change at position 116 (G to A); the other one had two base changes, one at position 116 (G to A) and one at position 465 (C to G). These findings confirm that PCV2b was the causative virus for PCVAD in this population but that mutations had occurred causing slightly different nucleotide sequences from those previously characterized.

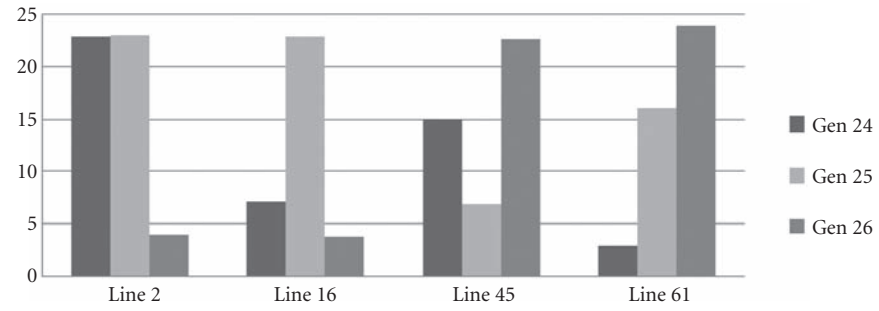

Figure 1. Percentage of pigs scored positive for PCVAD.

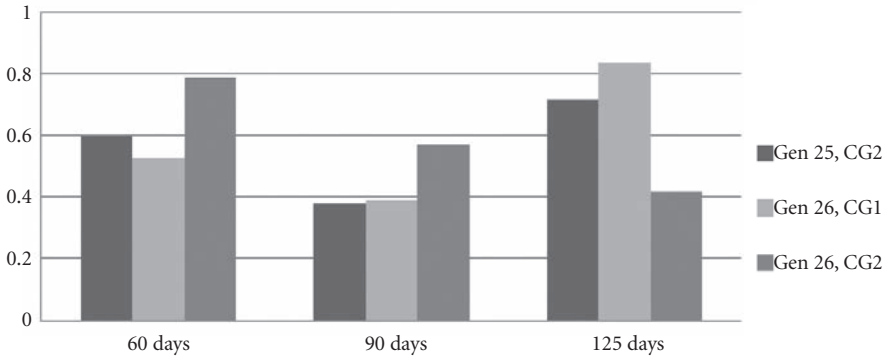

Figure 2. Mean PCV2 antibody titers.

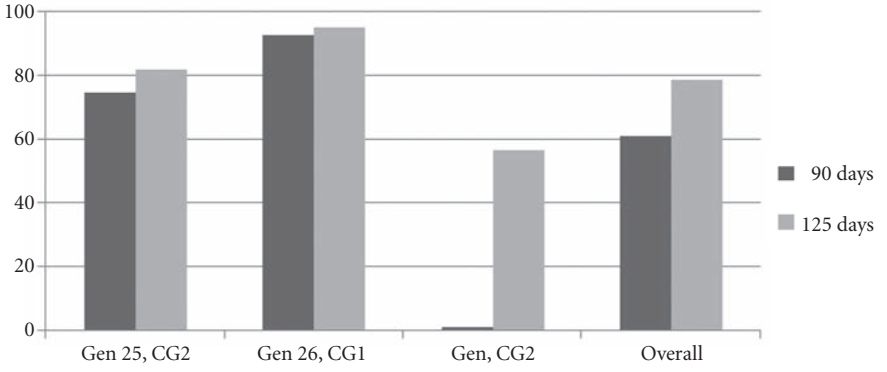

Figure 3. Viremic pigs, %, at 90 and 125 days.

Necropsy Findings.

Tissue samples from 36 of the 38 pigs that were positive for PCVAD were also positive for PCV2. Tissues of the other two were negative, but their nasal swabs were positive. Tissues from all 11 pigs scored as negative for PCVAD were negative for PCV2. Pigs with PCVAD had severe wasting, weight loss, rough hair coat, enlarged mesenteric lymph nodes, lesions indicative of pneumonia, and chronic colitis. Lymphocyte depletion within mesenteric lymph nodes and giant cells in lymphoid follicles, splenic follicles, and Peyer's Patches, and thymic atrophy due to lymphocyte depletion were observed. Nineteen pigs had symptoms of *Mycoplasma hyopneumoniae*; 18 had symptoms of

Table 3. Percentage of variation in PCVAD score and body weights due to different sources.

Source[1]	PCVAD score	BWT, lb	WWT, lb	65-day wt, lb	180-day wt, lb
		Genetic effects			
Pigs genes (h_d^2)	16.0± 4.0	26.7 ±7.0	16.0 ±6.0	23.0 ± 6.02	3.0 ± 6.0
Dam's genes (h_m^2)		18.3			11.0
		Environmental effects			
Birth litter	10.0	11.0			
Weaning litter	27.5	12.0			
Contemporary group	11.2	1.0	0.2	8.0	7.0
Location	22.0	3.0			
Pen	5.1	5.0			
Residual	47.2	43.3	57.1	47.0	51.0
Phenotypic standard deviation	1.46	0.11	0.43	1.97	6.4

[1]h_d^2 = direct heritability, h_m^2 = maternal heritability (± standard error of estimate of h_d^2).

Table 4. Percentage of variance of PCV2 viremia score[1], vermia level[2], and antibody titers[3] at 90 and 125 days of age.[1]

Parameter[4]	VS90	VS125	Viremia90	Viremia125	IgG90	IgG90[2]	IgG125
Pigs genes (h_d^2)			38 ± 11	11 ± 8		55 ± 21	10 ± 8
Birth litter	5.0	4.9			33.3	7.5	5.6
Contemporary group	77.0			18.0	16.7		18.9
Location	2.0	24.0		2.1			33.3
Room		26.2	1.9				6.7
Residual	15.0	44.9	60.2	68.4	50.0	37.5	25.6
σ_p^2	2.57	1.50	1.5	0.97	0.24	0.2	0.3

[1]VS90 and VS125 = viremia score (positive vs 0).
[2]Viremia90 and Viremia125 = $\log_{10}$ (PCV2 genomic copies/mL) for pigs with positive scores.
[3]IgG90 and IgG125 = PCV2 antibody titer.
[2]Generation 26, CG2 data deleted.
[4]h_d^2 = direct heritability ± standard error.

Streptococcus suis, and four had symptoms of *Lawsonia intracellularis.*

ELISA Screening

All serum samples tested for PRRSV and for *Mycoplasma hyopneumoniae* were negative. Thirty two of the 228 serum samples collected at 125 days of age from generation 25, CG2 pigs were positive for APP. Twenty one of 513 serum samples collected at 90 days of age from pigs of generation 26 were positive for APP, 16 had titers in the suspect range, and the rest were negative. Eight pigs that were positive for APP and five with titers in the suspect range had positive PCVAD scores. However, 142 pigs with positive PCVAD scores were negative for APP. Thus, PRRSV and M*ycoplasma hyopneumoniae* can be ruled out as secondary pathogens, and APP was not likely a secondary pathogen, involved in expression of PCVAD in this population.

Genetic and Environmental Parameters

Percentages of total variation due to genetic and environmental effects for PCVAD score and body weights are in Table 3 and those for PCV2 viremia and antibody titers are in Table 4. Direct heritability, a measure of the relative importance of genes of the pig, of PCVAD score was 16 ± 4%. Heritabilities of body weights ranged from 16% for weaning weight to 27% for birth weight. Genes of the dam, maternal heritability, were important for birth and 180-day weights. The location in which pigs were raised accounted for the most variation (22%) in PCVAD score whereas several environmental sources of variation contributed to variation in body weights. For pigs with positive viremia score (levels greater than zero), heritability estimate of viremia level at 90 days of age was greater than at 125 days of age (38 ± 11% vs 11 ± 8%). Genetic variation existed for antibody titers at 125 days of age (h^2 = 10 ± 8%), but not at 90 days of age when all data were included in analyses. However, when data for generation 26, CG2 were deleted, genetic variation in PCV2 antibody titer at 90 days of age was high (h^2 = 55 ± 21%). A large percentage of the variation in viremia score at 90 and 125 days of age was due to either the location or the room in which pigs were raised. These sources of variation were relatively small for viremia level and antibody titers.

Genetic and residual correlations among traits were calculated but are not presented here. The important ones were that PCVAD score was quite highly correlated genetically with day 90 viremia (r_g = 0.75) and antibody level (r_g = -0.67) and moderately correlated with 65-day weight (r_g = -0.53). Neither birth weight nor weaning weight were significantly correlated genetically with viremia or antibody levels. However, weaning weight was highly correlated genetically with viremia at 125 days of age (r_g = -0.73) and 180-day weight was negatively correlated with viremia at both 90 and 125 days of age and positively correlated with antibody titers at 90 days of age. Viremia level at 90 and 125 days of age were positively correlated genetically (r_g = 0.59) and viremia and antibody titers at 90 days of age were negatively correlated (r_g = -0.51). Antibody titers at 90 and 125 days of age were positively correlated (r_g = 0.59).

Although several environmental correlations among traits were significant, none were especially strong. Most notable were that environmental effects on score for PCVAD and for body weights through 65 days of age were correlated ($-0.47 \leq r_e \leq -0.39$), environmental effects on viremia and antibody levels at 90 days of age were correlated (r_e = -0.38), and viremia at 90 days of age was negatively correlated with body weights ($-0.26 \leq r_e \leq -0.16$).

Discussion

We conclude from this study and others in the literature that genetic variation in incidence of PCVAD

(Continued on next page)

and measures of immune responses to PCV2 exists. The heritability of PCVAD score was 16% and heritabilities of PCV2 viremia and antibody levels at 90 days of age were 38% and 55%, respectively. Genetic variation in viremia and antibody levels at 125 days of age were less and were not significant, perhaps because death of pigs with PCVAD between 90 and 125 days resulted in fewer records and decreased the genetic variance in remaining pigs.

Viremia and antibody levels at 90 days of age and 65-day weight were quite highly correlated genetically with PCVAD score. Thus selection for these traits may be an effective way to decrease incidence of PCVAD. Expected selection responses were calculated for different selection strategies. These were 1) direct selection — selection of breeders only from pigs that never displayed symptoms of PCVAD, 2) single-trait selection for weight, viremia, or antibody levels, and 3) index selection for correlated traits or PCVAD score plus correlated traits.

Even though PCVAD score is heritable, direct selection was relatively ineffective, resulting in a reduction of incidence of PCVAD of only -.8% in the first generation. This result occurred because 85% of the pigs, those with score of 0, were candidates for selection, resulting in a very low selection rate. The other traits are continuously distributed so pigs vary across the entire range of the distribution. Assuming 10% of the males and 30% of the females are selected for high PCV2 antibody titers at 90 days resulted in the greatest correlated expected response to single-trait selection (generation 1 response = -6.5%). Greatest expected response was for a three-trait index including 65-day weight and PCV2 viremia and antibody titers at 90 days of age (-8.0%). A four-trait index of these traits and PCVAD score produced the same expected response per generation. Expected selection responses were 414 to 998% greater when correlated traits were used than from direct selection.

Necropsy results confirmed that PCV2 was the main causative agent of PCVAD in this population. Genotyping PCV2 revealed that PCV2b was likely the causative agent. However, at least three allelic forms, and possibly more, existed. Thus, mutations have occurred in the PCV2b genome, but the consequence is not known.

Porcine Reproductive and Respiratory Virus, M*ycoplasma hyopneumoniae*, and *Actinobacillus pleuropneumoniae* were not secondary pathogens involved in expression of PCVAD. In other work, co-infection of pigs with PCV2 and Gram-negative bacteria induced viral replication of PCV2. Diagnostics for these pathogens were not performed, but infection of pigs with pathogens such as *Escherichia coli* and *Haemophilus influenzae*, Gram-negative bacteria known to be present in this population, may have increased the risk of PCVAD.

A strong relationship between PCV2 viremia level at 90 days of age and incidence of PCVAD across contemporary groups existed. Incidence of positive PCVAD Scores was 3.9% in generation 26, CG2, and only 1% of these pigs were viremic at 90 days of age. The incidences of PCVAD in generation 25, CG2, and Generation 26, CG1, were greater than 20%; 75% and 93% of the pigs in these respective groups were viremic.

Pigs with PCVAD differed in birth weight, weaning weight, 65-day weight, and 180-day weight, even though some of these weights were recorded well before pigs expressed symptoms of PCVAD. The genetic correlations of PCVAD score with birth and weaning weights were positive, but not significant. However, environmental correlations were negative and significant, indicating that the phenotypic relationship was largely due to environmental effects that reduced early body weights and increased risk of PCVAD.

Mean PCV2 antibody titers in pigs at weaning was high, most likely due to maternal PCV2 antibodies because all these pigs were non-viremic. Antibody titers decreased from weaning to 60 days of age, but 94.6% of pigs were still non-viremic at 60 days of age. Antibody titers continued to decrease from 60 to 90 days of age, suggesting that maternal antibodies were deteriorating. Effects associated with the birth litter were a major source of variation in antibody titers at 60 and 90 days of age, providing further evidence for significant variation associated with maternal antibodies. Other work has shown that high titers of maternal PCV2 antibodies are generally protective against PCV2, whereas low titers are not; however, maternal antibodies do not fully prevent PCV2 infections. Thus, pigs in our study were likely at least partially protected from PCV2 by maternal antibodies to at least 60 days of age.

Implications

Immune responses to PCV2 are heritable. Thus, genetic selection could be a useful tool to reduce incidence of PCVAD. Even though progress is permanent, several generations of selection will be required to greatly reduce the incidence and this selection must be practiced in nucleus herds and then transmitted through the breeding pyramid to commercial populations. Consequently, it would take considerable time for such a strategy to significantly reduce the incidence of PCVAD in commercial herds. Furthermore, response to selection for PCVAD scores, viremia, and antibody levels will occur only if all pigs in nucleus populations are exposed to PCV2 so that variation reflects genetic variation in the traits. Marker assisted or genomic selection may be more effective as once marker panels with known relationships with response variables are available, selection can be practiced in any population without exposure of pigs to PCV2. Thus genomic selection for resistance to PCV2 and decreased incidence of PCVAD may be the most effective long term selection strategy.

[1]Jared S. Bates was a graduate student, Roman Moreno is a graduate student and research technologist in the Animal Science Department; Alan R. Doster is a professor in the Department of Veterinary and Biomedical Science; and Rodger K. Johnson is a professor in the Animal Science Department.