Porcine Reproductive and Respiratory Syndrome virus reduces feed efficiency, digestibility, and lean tissue accretion in grow-finish pigs[1]

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ABSTRACT: Porcine reproductive and respiratory syndrome (PRRS) virus is a major swine virus that causes reproductive impairment in sows, as well as respiratory disease, reduction in growth rates, and mortalities in all ages of pigs. The objective of this study was to quantify the impact PRRS has on grower-finisher pig feed efficiency and tissue accretion rates. Thirty PRRS naïve, littermate pairs of maternal line Choice Genetics gilts (33.6 ± 0.58 kg BW) were selected and pairs split across 2 barns consisting of 5 pens (n = 6 pigs/pen per barn). Pigs in both barns were fed corn-soybean-DDGS diets ad libitum. All pigs in one barn were inoculated (CHAL) via an i.m. injection of a live PRRS strain isolated from the region (0 d post inoculation, dpi), while pigs in the other barn were given a saline control injection (CONT). Pig performance (ADG, ADFI, G:F) was assessed from 35 kg BW until each group reached market BW (128 kg). Additionally, longitudinal apparent total tract digestibility (ATTD) and body composition was assessed using Dual-energy X-ray absorptiometry (DXA) post inoculation (dpi) to estimate lean, protein, fat and bone accretion rates. Serological data from CHAL pigs showed that PRRS titers peaked 7 dpi and these pigs seroconverted by 35 dpi. According to both genomic and protein PRRS titers, CONT pigs were naïve to CHAL throughout the study. The PRRS infection reduced ($P < 0.001$) ATTD of dry matter, energy and nitrogen by 3 to 5% at 21 dpi and the reduction in ATTD persisted after 65 dpi. Compared to the CONT, CHAL pigs had decreased ADG (0.89 vs. 0.80 kg/d, $P < 0.001$), ADFI (2.05 vs. 1.93 kg/d, $P < 0.001$), and G:F (0.44 vs. 0.41 kg/d, $P < 0.001$) over the entire test period. The CHAL pigs also had attenuated DXA predicted whole body accretion of lean (547 vs. 633 g/d, $P = 0.001$), protein (109 vs. 126 g/d, $P = 0.001$) and fat (169 vs. 205 g/d, $P = 0.001$) compared to their CONT counterparts from dpi 0 to 80. Based on carcass data at slaughter (and consistent with the DXA data), CHAL pigs had leaner carcasses and reduced yields. These data clearly demonstrate that PRRS infection reduces digestibility, feed efficiency and protein accretion rates in grower-finisher pigs.

Key words: digestibility, feed efficiency, pig, Porcine Reproductive and Respiratory Syndrome, tissue accretion

Transl. Anim. Sci. 2017.1:480–488
doi:10.2527/tas2017.0054

INTRODUCTION

Porcine reproductive and respiratory syndrome (PRRS) virus is a major swine virus that causes reproductive impairment in sows and respiratory distress in all ages of pigs. This virus is estimated to cost the pork industry $664 million annually, mainly stemming from loss of production (Holtkamp et al., 2013). Despite numerous reports on the impact of PRRS virus

[1]This work was funded by the National Pork Board grant #12–163 and Choice Genetics USA; and partial support from the USDA National Institute of Food and Agriculture, Agriculture and Food Research Initiative Competitive Grants no. 2011–68004–30336 (to J. F. Patience) and the Animal Health and Disease Research Capacity Grant Program, by State of Iowa fund.

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Received August 7, 2017.
Accepted September 8, 2017.

on growth rates and immunity in grow-finish pigs, information on how PRRS disrupts tissue accretion rates, digestibility, and feed efficiency is surprisingly limited.

Continuous immune stimulation and inflammatory responses can disturb anabolic growth factors and suppress muscle growth (Johnson, 1997; Spurlock, 1997; Broussard et al., 2003). Recent reports indicate that serum pro-inflammatory cytokine concentrations are elevated early after PRRS infection (Lunney et al., 2010). In addition, Che et al. (2012) observed that PRRS-infected pigs experienced increases in pro-inflammatory cytokines (e.g., tumor necrosis factor α) as well as acute phase proteins (e.g., C-reactive protein and haptoglobin) at d 7 and 14 post-infection.

Pigs with reduced health status often exhibit reduced appetite and feed intake, and have altered nutrient utilization in a tissue-specific manner (Johnson, 2002). Collectively, it is assumed that the associated growth depression and diversion of nutrients away from tissue accretion ensures that adequate energy and nutrients are available for high priority immunological and homeostatic pathways. This is exemplified by the fact that pigs raised in suboptimal environmental conditions and exposed to inflammatory stimuli have decreased protein deposition rates (Williams et al., 1997c). Research conducted by Escobar et al. (2004) is unique in that it is the only reported study which showed PRRSV infection reduced tissue accretion rates in nursery pigs. Therefore, the objective of this study was to determine the longitudinal impact of PRRS on growing-finishing pig's energy and nutrient digestibility, whole body tissue accretion rates, and feed efficiency. Additionally, the economic impact of grower pigs infected with PRRS was evaluated.

MATERIALS AND METHODS

All animal procedures were approved by the Iowa State University Institutional Animal Care and Use Committee (IACUC# 8–12–7427-S). This work was conducted in central Iowa, USA in the fall of 2012 through winter of 2013.

Animals, Housing, and Experimental Design

Sixty, PRRS naïve Choice Genetics maternal line gilts (33.6 ± 0.58 kg BW) were assigned to 1 of 2 barns, and grouped into five pens of 6 pigs each. The 2 barns were retrofitted conventional finishing barns with pen space set at ≥ 0.84 m^2 per pig (grow-finish) and they were located 25 miles apart. Both barns had fully slotted concrete flooring with curtain sided, natural ventilation. Pens in each barn had the same feeder (Boar Feeder, Hog Slat, Inc., Newton Grove, NC), feeder space allowance, pen area, and the same genetic configuration (i.e., sow offspring pairing, 2 gilts per sow, in which one went to the control barn and the other to the challenged barn). Throughout the study pigs were allowed ad libitum access to water and feed. Both barns were fed a corn-soybean-DDGS diets in five phases that met or exceeded NRC (2012) nutrient requirements (Table 1). All diets were manufactured in the same feed mill at the same time.

After a 2 wk acclimation period of common performance, pigs in one barn (CHAL; n = 30 gilts, 5 pens/treatment) were inoculated i.m. with 1 mL of inoculum containing 1,000 genomic units of a live field strain of PRRS virus (ORF5-RFLP 1–18–4) common to northern Iowa. Pigs in the second barn served as control pigs (CONT; n = 30 gilts, 5 pens/treatment) and received 1 mL of saline i.m. Body weight was measured immediately prior to PRRS virus inoculation [0 d post inoculation (dpi)], at 7, 14, 21, 28, 35, 42, 56, 70 dpi and when CONT and CHAL treatments reached market BW (~128 kg BW, dpi 105 and 119, respectively). Pen feed intake was recorded weekly and pen feed efficiency was calculated. The growth study ended when the average pig BW within a treatment (i.e., in each barn) reached ~128 kg. Thereafter, all pigs were slaughtered and carcass data collected from the slaughter plant (Tyson Foods, Storm Lake, IA). Shipping and pre-slaughter handling were

Table 1. Diet composition, as fed basis

Ingredients, %	Phase 1	Phase 2	Phase 3	Phase 4	Phase 5
Corn	53.63	56.72	60.90	64.58	71.03
Soybean meal, 48% CP	23.00	20.00	16.00	12.50	6.25
Corn DDGS	20.00	20.00	20.00	20.00	20.00
Soybean oil	1.00	1.00	1.00	1.00	1.00
Limestone	0.85	0.85	0.85	0.85	0.83
L-Lysine·HCl	0.63	0.60	0.55	0.50	0.43
Sodium chloride	0.35	0.35	0.35	0.35	0.35
Commercial VTM[1]	0.10	0.10	0.10	0.08	0.08
Heat Stable Optiphos 2000	0.02	0.02	0.01	0.01	0.01
L-Threonine	0.23	0.20	0.14	0.10	0.04
Methionine HMTBa	0.20	0.16	0.09	0.05	–
Titanium dioxide	0.40	–	0.40	–	–
Formulated composition					
CP, %	20.1	19.1	17.7	16.5	14.3
ME, Mcal/kg	3.39	3.39	3.39	3.40	3.40
Lys, SID %	1.30	1.20	1.05	0.95	0.75
Met+Cys, SID %	0.78	0.72	0.64	0.58	0.45
SID Lys/ME, g/Mcal	3.83	3.53	3.10	2.79	2.21
STTD Phosphorus, %	0.31	0.30	0.24	0.23	0.22
Chemical composition					
Crude Protein, %	20.9	–	19.9	–	–
Gross energy, Mcal/kg	4.16	–	4.09	–	–

[1]VTM = Vitamin-trace mineral premix, which supplied per kilogram of diet: vitamin A, 8,820 IU; vitamin D_3, 1,653 IU; vitamin E, 33.1 IU; vitamin K, 4.4 mg; riboflavin, 6.6 mg; niacin, 38.9 mg; pantothenic acid, 22.1 mg; vitamin B_{12}, 0.04 mg; I, 1.1 mg as potassium iodide; Se, 0.30 mg sodium selenite; Zn, 60.6 mg as zinc oxide; Fe, 36.4 mg as ferrous sulfate; Mn, 12.1 mg as manganous oxide; and Cu, 3.6 mg as copper sulfate.

constant for each group. Hot carcass weight (HCW), fat depth and loin muscle depth were collected at the slaughter plants as previously described (Arkfeld et al., 2015).

Whole Body Composition Analysis

Following an overnight fast, on –1, 42, and 80 dpi, pigs were scanned using dual-energy X-ray absorptiometry (DXA; Hologic Discovery A, Bedford, MA) as described in detail elsewhere (Suster et al., 2003). Briefly, pigs were weighed, anesthetized with an i.m. injection of telazol:ketamine:xylazine (4.4:2.2:2.2 mg/kg BW) and placed on the scan table in ventral recumbency with hind legs and fore legs extended. Each pig was then scanned and thereafter transported back to its home pen. Measurements made by DXA included total mass, lean mass, fat mass and percentage, and bone mineral density and content. Data from the raw DXA output was then adjusted to account for blood volume and gut fill using calibration curves that were built as previously described by Curry et al. (2017). These longitudinal scan data were then used to calculate tissue accretion rates (g/d) from corrected DXA scan results using the following formula:

$$\text{g/d} = \frac{\text{(corrected final scan measurement - corrected initial scan measurement)}}{\text{days between scans}}$$

Nutrient Digestibility

At 14 to 21 and 62 to 75 dpi, 0.4% titanium dioxide was placed in the diets as a digestibility marker. Feces were collected and pooled within pig from 15 pigs per treatment at 19 to 22 dpi and again at 65 to 70 dpi. Fecal matter was stored at approximately –20°C until further analysis. The stored fecal matter from each pig was thawed and homogenized. Feed or fecal matter were placed into an aluminum tray to be dried at 75°C in a mechanical convection oven for approximately 1 wk. Dried feed and fecal weights were recorded and used to determine moisture and dry matter content. The dried feces and feed were then ground in a Willey Mill using a 0.8 mm screen and placed in a desiccator until further analysis.

Feed and fecal nitrogen (N) content were determined using a TruMac Nitrogen Analyzer (Leco Corporation, St. Joseph, MO) according to method 990.03 (AOAC, 2007). The GE was determined using an isoperbolic bomb calorimeter (Model 6200, Parr Instrument Company, Moline, IL). Titanium dioxide content in feed and feces was determined according to Leone (1973). Dry matter, N and energy apparent total tract digestibility (ATTD) were measured and calculated to determine differences between treatments as previously described (Oresanya et al., 2008).

Blood Sampling and Analysis

Ten mL of blood were collected via jugular venipuncture into serum tubes prior to virus inoculation to confirm the pigs were PRRS naïve and again at 7, 21, 35, and 70 dpi. After clotting, serum was separated by centrifugation (2,000 × *g* for 10 min at 4°C) and aliquots were stored at –80°C until analysis or sent to the Iowa State University Veterinary Diagnostic Laboratory for PRRS virus detection.

Serum samples from 5 pens per treatment (3 CONT pens and 6 CHAL pens) were assayed for PRRS virus antibodies. The PRRSV antibodies were assessed using a commercial indirect ELISA (PRRSX3 antibody test, IDEXX Laboratories, Inc., Westbrook, ME) performed according to the manufacturer's instructions. Serum PRRS virus load was also determined by routine quantitative PCR protocol for serum. Briefly, nucleic acid extraction from serum samples was performed using a commercial RNA extraction kit (Ambion MagMax-96 Viral RNA isolation kit, Applied Biosystems, Foster City, CA). Real-time PCR was performed with commercial reagent sets (TaqMAN NA and EU PRRS Reagents, and TaqMAN NA and EU PRRS Controls, Applied Biosystems) using the following cycling conditions: 1 cycle at 45°C for 10 min, 1 cycle at 95°C for 10 min, 40 cycles of: 97°C for 2 s, 60°C for 40 s. Eight 10-fold serially-diluted (10° to 10^7 copies/μL), plasmid-derived commercial standards (TaqMAN NA and EU PRRS RNA controls, Applied Biosystems) were run on each PCR plate and their Ct values used to derive a standard curve. Samples were quantified as genome equivalents per μL (geq/μL) by fitting the sample Cts to the standard curve using the AB7500 Fast System SDS Software (Applied Biosystems) and $\log_{10}$ transformed.

Serum obtained from pigs at 21 dpi was also assessed for insulin, glucose, and cytokine concentrations. A porcine Insulin ELISA kit (Mercodia AB, Uppsala, Sweden) and Wako Diagnostics Autokit Glucose (Wako Chemical USA Inc., Richmond, VA) were employed to assess insulin and glucose, respectively, per manufacturer's instructions. The ELISA plates were read with a Synergy 4 plate reader using Gen 5 software (BioTek Instruments Inc., Winooski, VT). The ProcartaPlex Porcine Cytokine and Chemokine Panel 1 (eBioscience, Inc., San Diego, CA) was used to evaluate circulating concentrations of interleukin (IL)-12p40, interferon (IFN) α, IL-6, IL-8, and TNFα. ProcartaPlex Porcine Immunoassays use the Luminex technology (multi-analyte profiling beads) to enable the detection and quantitation of multiple protein targets simultaneously in serum.

Statistical Analysis

Data were analyzed using the PROC MIXED procedure of SAS version 9.2 (SAS Inst. Inc., Cary, NC) to study the effects of treatment. Pen (5 pens/treatment) was considered the experimental unit for ADG, ADFI, G:F, and tissue accretion rates. Pen was considered the experimental unit for ATTD and all blood analyses. Three pigs per pen where sampled and data averaged to create pen values. Residual correlations between serum metabolites, cytokines and tissue accretion rates were analyzed using PROC CORR to generate Pearson correlations. All data are reported as least squares means ± SEM and considered significant if $P \leq 0.05$ and a trend if $P \leq 0.10$.

RESULTS

PRRS Infection and Growth Performance

Prior to inoculation (dpi -14 to 0), initial BW, growth rate and feed intake were similar across barns (data not shown).

The efficacy of the infection model was verified by examining both PRRS viremia load and antibody response over time. All pigs were PRRS naïve prior to the study and as anticipated, the 30 CONT gilts remained PRRS negative (qRT-PCR and antibody) throughout the study. However, as expected, the CHAL pigs had increased virus titers from 0 to 7 to 21 dpi (0, 4.6 and 5.9 PRRS virus titer, $\log_{10}$ genomic units/mL, respectively; Table 2); CHAL gilts seroconverted by 35 dpi.

In the first 42 dpi CHAL gilts had reductions ($P < 0.05$; Table 3) compared to CONT gilts for ADG (0.93 vs. 0.75 kg/d), ADFI (1.87 vs. 1.68 kg/d) and G:F (0.50 vs. 0.44). In the second period (43 to 70 dpi), ADG and ADFI did not differ between treatments ($P > 0.05$); however, there was a tendency ($P = 0.077$) for G:F to be reduced in CHAL gilts compared to CONT gilts (0.35 and 0.30, respectively). Overall, CHAL gilts grew slower (0.80 kg/d vs. 0.90 kg/d; $P = 0.002$) and ate less feed (1.93 kg/d vs. 2.06 kg/d; $P = 0.032$) than CONT gilts; however G:F remained unaffected ($P > 0.05$) for the overall test period. As a result of PRRS infection, growth was impeded and resulted in an extra 14 d on feed to achieve the same market BW of 128 kg as the CONT gilts (Fig. 1). It was deemed important to terminate the experiment at a constant weight, rather than constant time, to support the most effective comparison between treatments of growth performance and tissue accretion rates.

Apparent Total Tract Nutrient Digestibility (ATTD)

During the first period, ATTD coefficients of DM, N and energy were reduced by 3.0, 5.5, and 4.0%, respectively, due to PRRS infection (Table 4; $P < 0.001$). Reductions ($P < 0.05$) in ATTD coefficients persisted in CHAL gilts but were smaller in magnitude (65 to 70 dpi) compared to the CONT for DM (81.5 vs. 79.8%), N (80.1 vs. 78.0%) and energy (78.1 vs. 76.3%), respectively.

Table 2. PRRS viremia and antibody titer serology

Parameter	CONT[1]	CHAL[1]
PRRS viremia[2]		
dpi 0	0.0 (neg.)	0.0 (neg.)
dpi 7	0.0 (neg.)	4.6 (pos.)
dpi 21	0.0 (neg.)	5.9 (pos.)
dpi 35	0.0 (neg.)	0.0 (neg.)
dpi 70	0.0 (neg.)	0.0 (neg.)
PRRS antibody titer[3]		
dpi 0	0.00 (neg.)	0.00 (neg.)
dpi 7	0.00 (neg.)	0.20 (pos.)
dpi 21	0.02 (neg.)	0.20 (pos.)
dpi 35	0.01 (neg.)	1.20 (pos.)
dpi 70	0.01 (neg.)	0.87 (pos.)

[1]n = 5 pens/treatment. neg. = Negative; pos. = Positive.

[2]QPCR viremia *(Log+1 genomic copies/mL).*

[3]PRRSX3 EIA titer.

Table 3. The impact of PRRS on growth performance of grow-finish pigs

Parameter	CONT[1]	CHAL[1]	SEM	*P*-value
0–42 dpi				
ADG, kg	0.93	0.75	0.028	< 0.001
ADFI, kg	1.87	1.68	0.038	0.002
G:F	0.50	0.44	0.017	0.010
43–70 dpi				
ADG, kg	0.81	0.73	0.065	0.260
ADFI, kg	2.31	2.41	0.087	0.301
G:F	0.35	0.30	0.052	0.077
Overall, 0 dpi to market BW 128 kg (~114 dpi)				
ADG, kg	0.90	0.80	0.021	0.002
ADFI, kg	2.06	1.93	0.049	0.032
G:F	0.44	0.41	0.017	0.227

[1]n = 5 pens/treatment.

Whole Body Composition, Tissue Accretion, and Carcass Data

From 0 to 80 dpi, CONT gilts had greater fat (230 vs. 184 g/d), lean (657 vs. 568 g/d) and protein (131 vs. 112 g/d) deposition rates compared to CHAL gilts ($P < 0.01$; Table 5); there was no difference in bone mineral accretion rates ($P > 0.10$). Interestingly, these reductions in daily tissue accretion rates were most profound between 0 and 42 dpi where lean, protein, fat, and whole body gain were reduced by 23, 24, 30, and 24%, respectively ($P < 0.01$). From 43 to 80 dpi, only lean was still

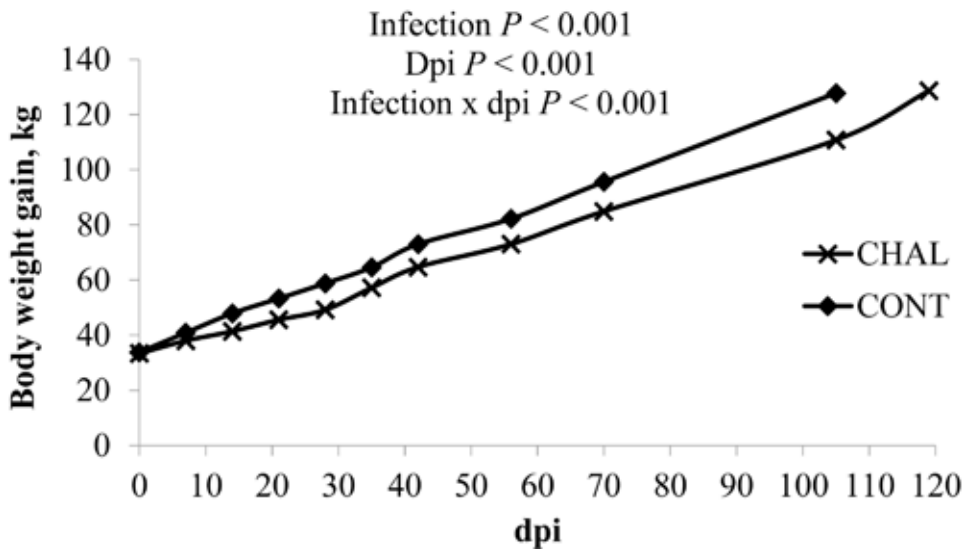

Figure 1. Growth differences in of PRRS infected (CHAL) and PRRSV-naïve (CONT) gilts. Gilts were placed on test at 33.6 kg BW and taken to a target market BW of 128 kg. At dpi 0, the CHAL gilts were inoculated i.m. with a live PRRSV strain. The CONT gilts received a saline vehicle.

Table 4. PRRS reduces apparent total tract digestibility of dry matter, nitrogen and energy

Parameter	CONT[1]	CHAL[1]	SEM	*P*-value
19–22 dpi ATTD coefficients, %				
Dry matter	83.9	81.3	0.54	< 0.001
Nitrogen	81.8	77.3	0.84	< 0.001
Gross Energy	81.0	77.8	0.64	< 0.001
65–70 dpi ATTD coefficients, %				
Dry matter	81.5	79.8	0.62	0.008
Nitrogen	80.1	78.0	0.82	0.018
Gross Energy	78.1	76.3	0.76	0.020

[1]*n* = 5 pens/treatment.

Table 5. The impact of PRRS on whole body tissue accretion and carcass

Parameter[1]	CONT	CHAL	SEM	*P*-value
0–42 dpi[1]				
Lean, g/d	635	491	14.5	< 0.001
Protein, g/d	123	94	2.61	< 0.010
Fat, g/d	155	108	8.41	< 0.001
BMC, g/d	24.6	21.5	1.07	0.006
Whole body gain, kg/d	0.83	0.63	0.02	< 0.001
43–80 dpi[1]				
Lean, g/d	683	664	30.1	0.54
Protein, g/d	141	135	6.36	0.38
Fat, g/d	326	280	16.6	0.008
BMC, g/d	31.9	31.5	1.47	0.78
Whole body gain, kg/d	0.94	0.89	0.02	0.17
Overall (0–80 dpi)[1]				
Lean, g/d	657	568	13.9	< 0.001
Protein, g/d	131	112	2.89	< 0.001
Fat, g/d	230	184	9.22	< 0.001
BMC, g/d	27.8	26.0	0.94	0.063
Whole body gain, kg/d	0.92	0.78	0.019	< 0.001
Carcass data[2]				
Live weight, kg	123.0	126.4	1.74	0.21
HCW, kg	94.5	95.3	1.67	0.64
Yield, %	76.7	75.3	0.44	0.063
Lean. %[3]	55.4	56.3	0.25	0.034
Back fat depth, cm	1.85	1.50	0.065	0.005
Loin depth, cm	7.04	7.05	0.079	0.96

[1]*n* = 30 gilts/treatment.

[2]Carcass data means of 5 pens/treatment.

[3]Calculated as 58.86 – [fat depth (mm) × 0.61] + [loin depth (mm) × 0.12].

significantly reduced in CHAL gilts ($P < 0.01$; Table 5). Reductions in tissue accretion rates were reflected in the carcass composition at 128 kg live BW (Table 5). Increased lean (56.3 vs. 55.4%, $P = 0.034$) and decreased back fat depth (1.50 vs. 1.85 cm, $P < 0.01$) were seen in CHAL gilts, compared to CONT gilts. Hot carcass weight was not affected by PRRS infection but carcass yield tended to be decreased ($P = 0.063$) in CHAL gilts compared to CONT (75.3 vs. 76.7%, respectively).

Blood Metabolite and Inflammation Markers

At 21 dpi, the CONT and CHAL gilts had identical blood glucose concentrations (Table 6) even though feed intake was significantly reduced. However, blood insulin concentrations were reduced by 50% in the CHAL pigs ($P < 0.05$; Table 6) resulting in a significantly greater ($P < 0.05$; Table 6) degree of insulin sensitivity in the CHAL pigs compared to the CONT. No differences in the pro-inflammatory cytokines IL-6, IL-8 and IL-12p40 were reported over the first 21 dpi among the 2 treatment groups (Table 6). Nevertheless, PRRS infection tended ($P = 0.094$) to increase TNF-α and increased ($P < 0.001$) IFN-α in the first weeks after PRRS inoculation compared to the CONT gilts.

Table 6. Blood metabolite and inflammatory markers at dpi 21 post PRRS virus inoculation

Parameter[1]	CONT	CHAL	SEM	*P*-value
Glucose, mg/dL	115	113	5.1	0.618
Insulin, ng/L	134	64	23.9	0.005
Glucose:Insulin	1.3	2.8	0.32	0.016
Interferon α, pg/mL	1.1	2.8	0.76	0.032
Interleukin 6, pg/mL	340	144	152.7	0.216
Interleukin 8, pg/mL	169	271	66.0	0.140
Interleukin 12p40, pg/mL	898	1178	256.2	0.289
Tumor necrosis factor α, pg/mL	0	63	35.6	0.094

[1]*n* = 5 pens/treatment.

Residual correlations were made using 21 dpi serum metabolite and cytokine concentrations and 0 to 42 dpi tissue accretion rates (Table 7). TNF-α, IL-8, IL-12p40, insulin and glucose showed little to no correlation. However, IFN-α tended to have a negative correlation with protein accretion rates (–0.54, P = 0.107) and whole body gain (–0.57, P = 0.086) while IL-6 tended to be positively correlated with protein (0.59, P = 0.072), lean (0.62, P = 0.057) and whole body (0.58, P = 0.078) accretion rates.

Economic Assessment of PRRS Infection

Two economic analyses were conducted based on the data generated in this study (Table 8). Parameters included BW of grow-finisher gilts from 35 to 128 kg body weight, feed prices set at $245/MT, standardized base price $61.59/cwt, and a barn cost of $0.10 per pig per day. If feeder space was not limited and a producer could take pigs to the designated market weight of 128 kg, then the economic impact of PRRS infection was conservatively estimated at $3.47 per head. However, if feeder space was limited, then the loss was estimated to be $10.49 per head. No mortality was observed in this study, and our economic model did not account for mortalities and medication costs or packing plant grid penalties.

Table 8. Economic assessment of a grow-finish pigs challenged with PRRS virus[1]

Assumptions	CONT	CHAL	Difference
1) Assumption if taken to same live weight			
ADFI, kg	2.05	1.93	
Feed days	105	119	+14 d
Total feed, kg	215	229	+14.5
Feed cost @ $245/metric t	$52.68	$56.11	-$3.43
Barn cost @ $0.10/pig per d housing	$10.50	$11.90	-$1.40
Carcass wt, kg	94	95	+1.0
Carcass value	$127.66	$129.02	+$1.36
Estimated cost of PRRS per pig			-$3.47
2) Assumption if same age			
Feed cost @ $245/metric t and 105 d	$52.74	$49.65	+$3.09
Body weight, kg	123	112	
Carcass weight, kg	94	84	
Carcass value	127.66	$114.08	-$13.58
Estimated cost of PRRS per pig			-$10.49

[1]Calculated using 5-yr average (2012–2017) prices: Corn = $4.52/bu; Soybean Meal = $370/t; DDGS = $175/t; Lean hog = $61.59/cwt (USDA-NASS, 2017).

DISCUSSION

The objective of this research was to determine the longitudinal impact of PRRS on grow-finisher pig in a commercial industry field setting. In agreement with Escobar et al. (2004), we report a significant reduction in ADG, ADFI, and G:F in pigs experimentally infected with PRRS. However, as the previous work (Escobar et al., 2004) demonstrated reduced short-term performance (i.e., a 14 d period) in PRRS challenged pigs, the experiment described herein clearly demonstrates a persistent negative impact on growth rate and feed intake all the way to market BW (approximately 128 kg). Additionally, the work in pigs involving PRRS infection by Escobar et al. (2004) and immune stimulation by Williams et al. (1997a, 1997b, 1997c) classically demonstrates the profound adverse effects of health status and immune stimulation on protein accretion. Particularly in

Table 7. Residual correlations between Period 1 (0–42 dpi) tissue accretion and 21 dpi serology[1]

Parameter	INFα	IL6	IL8	TNFα	IL12p40	Insulin	Glucose
Protein, g/d	-0.54	0.59	-0.28	-0.03	-0.09	-0.02	-0.35
	0.107	*0.072*	*0.426*	*0.943*	*0.804*	*0.965*	*0.327*
Lean, g/d	-0.52	0.62	-0.31	-0.06	-0.08	0.0008	-0.35
	0.126	*0.057*	*0.384*	*0.870*	*0.825*	*0.998*	*0.324*
Fat, g/d	-0.50	0.37	0.16	0.33	0.09	-0.06	-0.04
	0.142	*0.293*	*0.669*	*0.390*	*0.808*	*0.877*	*0.902*
BMC, g/d	-0.54	0.30	-0.20	-0.04	0.20	0.02	-0.14
	0.362	*0.398*	*0.571*	*0.910*	*0.573*	*0.953*	*0.672*
Whole body, g/d	-0.57	0.58	-0.18	0.07	-0.02	-0.01	-0.27
	0.086	*0.078*	*0.614*	*0.849*	*0.956*	*0.976*	*0.456*

[1]Upper row = residual correlations. Bottom row = *P*-values.

the first week of PRRS infection, pig whole body protein accretion has been reported to be reduced by up to 60% compared with naïve control pigs (Escobar et al., 2004). Our longitudinal assessment (i.e., applying DXA technology in the same animal over time), indicates that protein accretion was reduced by 24% in the first 42 dpi but was not different in the following 38 d due to PRRS infection in grower pigs. Overall, this resulted in a 10% reduction in protein accretion over an 80 dpi period. Similar results were observed with whole body lipid accretion rates with PRRS infection resulting in a 20% lower accretion rate compared to the CONT pigs. Thus, pigs challenged with PRRS in the current study are leaner at market weight and have reduced carcass yields. This is consistent with observations by Williams et al. (1997c) and Rakhshandeh and de Lange (2012) whereby reduced carcass yields and higher offal and organ weights were reported in immune stimulated pigs.

In previous experimental PRRS-challenge studies, a decrease in tissue accretion rates has only been observed in nursery pigs (Escobar et al., 2004). Similarly, non-pathogenic lipopolysaccharide (LPS) challenge models have been shown to acutely decrease muscle protein synthesis in vivo and in vitro through impaired phosphorylation of both eIF4E-binding protein 1 and ribosomal protein S6 kinase 1 in the mammalian target of the rapamycin (mTOR) pathway, which is a key determinant of translation initiation and protein synthesis in pigs (Kimball et al., 2002; Kimball et al., 2003; Orellana et al., 2007a; Orellana et al., 2007b). This inhibition of the translation initiation process seems to be a direct effect of the inflammatory response, but the subsequent increase in pro-inflammatory cytokines may augment the depression in protein synthesis events and rates (Lang et al., 2002; Lang et al., 2003; Lang and Frost, 2007; Lang et al., 2007).

The residual correlations between serum cytokine concentrations and tissue accretion rates over the first 6 wk of a PRRS challenge showed positive correlations between IL-6, protein, lean and whole body accretion rates. Increased concentrations of IL-6 have been reported in contracting muscle fibers (Febbraio et al., 2004) which can lead to increased muscle mass by satellite cell activation (Serrano et al., 2008). Also, IFN-α was correlated to protein and whole body accretion rates, albeit negatively. Increasing IFN-α concentrations suggest an increased antiviral activity by increasing the surface expression of CD14 on porcine alveolar macrophages (Amadori, 2007). Sustained levels of IFN-α also lead to increases in pro-inflammatory cytokines which may decrease protein synthesis, as mentioned previously.

The impact of immune stimulation, particularly using industry applicable pathogens, on nutrient and energy digestibility has been poorly described. Lipopolysaccharide has been extensively used to study the inflammatory response in swine. More recently, repeated challenges with LPS have been used to mimic pathogenic challenges like PRRS (Rakhshandeh and de Lange, 2012; Rakhshandeh et al., 2012). These studies have shown that inflammation reduces ATTD of organic matter, energy, and nitrogen. Similarly, at 21 dpi we clearly reported ATTD of nitrogen, dry matter and energy to be reduced due to PRRS infection. Possibly, even more important is the fact that this antagonism of total tract digestibility persists even up to or beyond 70 dpi. We can only speculate this to be a result of intestinal microbial population alterations as a result of PRRS infection.

Meta-analysis of pig microarray studies revealed mitochondrial dysfunction and dysfunction of oxidative phosphorylation pathways, innate immune response, apoptosis and cell homeostasis pathways to be regulated under PRRS infection (Badaoui et al., 2013). Based on this meta-analysis and our observed reduction in performance measures, innate and adaptive responses to intense, prolonged and poorly-contained immunological stimuli are most likely responsible. This may partially be evident in the blood glucose and insulin data at 21 dpi. For blood glucose to be maintained under reduced feed intake at 21 dpi in the CHAL pigs, presumable mobilization of amino acids from skeletal muscle is required for gluconeogenesis and the generation of energy. Additionally, these amino acids support the increase in protein synthesis of immune cells and molecules. Therefore, switches in bioenergy intimately links metabolism with immune function and inflammation to restore homeostasis in the face of PRRS infection.

Altogether, these data clearly demonstrate that PRRS infection reduces digestibility in the short and long term of pigs. Feed efficiency and whole protein accretion rates are also profoundly impacted by PRRS infection in grow-finisher pigs. The research described in this paper reported on the interaction between PRRS infection and lifetime tissue accretion, performance and feed efficiency in grow-finisher pigs. Besides the recent outbreaks and prevalence of Porcine Endemic Diarrhea (PED), PRRS is arguably one of the most economically important pathogens to the U.S. swine industry, estimated to cost the pork industry $664 million annually, mainly stemming from loss of production (Holtkamp et al., 2013). Herein, based on our own phenotype data and feed efficiency numbers, we estimated that PRRS infection in grower pigs cost the producer a conservative $3 to 11 per head with extra housing, feed costs, lighter weight and lower carcass value significantly contributing to this estimate.

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