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Effect of a respiratory disease challenge on nitrogen retention, IGF-I, organ weight and carcass characteristics in growing pigs

Abstract

Forty-seven growing pigs (initially 65 ű 2 lb) were used in a metabolism study to determine the effects of a single respiratory disease challenge on nitrogen retention, plasma insulin-like growth factor-I (IGF-I), organ weight, and carcass characteristics. Thirty pigs were challenged with Actinobacillus pleuropneumonia, and 7 pigs were assigned to an ad libitum fed nonchallenged control group. Ten additional nonchallenged pigs were pair-fed the feed intake of a A. pleuropneumonia-challenged counterpart. There were five 4 d collection periods (d 4 to 7, d 8 to 11, d 12 to 15, d 16 to 19, and d 22 to 25), and the A. pleuropneumonia challenge occurred on d 8. Plasma IGF-I concentrations decreased on d 9 in the disease challenged pigs compared to those in both nonchallenge treatments. Nitrogen retention was decreased during the immune challenge period and only partially recovered by the end of the experiment on d 25. Final organ weights and carcass characteristics were similar among treatments. These results suggest that a single acute respiratory disease challenge is accompanied by partial long-term compromises in protein metabolism and lean growth rate.; Swine Day, Manhattan, KS, November 18, 1999

Keywords

Swine day, 1999; Kansas Agricultural Experiment Station contribution; no. 00-103-S; Report of progress (Kansas State University. Agricultural Experiment Station and Cooperative Extension Service); 841; Swine; Pigs; Nitrogen retention; Respiratory disease challenge

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EFFECT OF A RESPIRATORY DISEASE CHALLENGE ON NITROGEN RETENTION, IGF-I, ORGAN WEIGHT AND CARCASS CHARACTERISTICS IN GROWING PIGS

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Summary

Forty-seven growing pigs (initially $65 \pm$ 2 lb) were used in a metabolism study to determine the effects of a single respiratory disease challenge on nitrogen retention, plasma insulin-like growth factor-I (IGF-I), organ weight, and carcass characteristics. Thirty pigs were challenged with Actinobacillus pleuropneumonia, and 7 pigs were assigned to an ad libitum fed nonchallenged control group. Ten additional nonchallenged pigs were pair-fed the feed intake of a A. pleuropneumonia-challenged counterpart. There were five 4 d collection periods (d 4 to 7, d 8 to 11, d 12 to 15, d 16 to 19, and d 22 to 25), and the A. pleuropneumonia challenge occurred on d 8. Plasma IGF-I concentrations decreased on d 9 in the disease challenged pigs compared to those in both nonchallenge treatments. Nitrogen retention was decreased during the immune challenge period and only partially recovered by the end of the experiment on d 25. Final organ weights and carcass characteristics were similar among treatments. These results suggest that a single acute respiratory disease challenge is accompanied by partial longterm compromises in protein metabolism and lean growth rate.

(Key Words: Pigs, Nitrogen Retention, Respiratory Disease Challenge.)

Introduction

Previous research by our laboratory (KSU 1998 Swine Day, Report of Progress 819) indicated that although short-term decreases in nitrogen (N) balance were evident, long-term effects of an enteric disease challenge by *Salmonella typhimurium* were negligible. Because economically important disease challenges in growing swine are primarily enteric or respiratory, we were curious if a similar immune response pattern in N balance would be evident when *Actinobacillus pleuropneumonia* was used as the respiratory disease agent.

As a continuation of the experiment reported in the preceding article, our objective was to characterize the effects of an acute respiratory disease challenge in pigs by measuring changes in protein metabolism using N balance techniques. Furthermore, we wanted to characterize the changes in insulin-like growth factor-I (IGF-I), organ weights, and carcass characteristics resulting from an acute respiratory disease challenge.

Procedures

The experimental protocol used in this study was approved by the KSU Institutional Animal Care and Use Committee. Forty-seven nonlittermate high-health barrows (PIC C22 × L326; initially 65 ± 2 lb) were obtained from the university swine herd after serological testing to ensure *A. pleuropneumonia* negative status. Pigs were blocked by

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weight and time and allotted to one of three experimental treatments similar to those described in the preceding article.

Daily care, feeding, disease challenge, and diet are all described in the preceding article. There were five 4-d collection periods (d 4 to 7, d 8 to 11, d12 to 15, d 16 to 19, and d 22 to 25) with the *A. pleuropneumonia* challenge occurring on d 8. These periods correlated to prechallenge, challenge, recovery (8 d), and postchallenge. The latter period was selected to be at least 14 d postchallenge.

All pigs were housed in two similar environmentally controlled rooms based upon health status and were kept in adjustable individual stainless steel metabolism cages (5 ft \times 2 ft) that allowed separate collection of feces and urine. The marker to marker method (.5% ferric oxide in the first meal and eighth subsequent meal) was used to determine the beginning and end of feces collection for a period. Feces were collected twice daily and stored at 20°F. At the end of each period, feces were autoclaved to kill pathogenic activity before being homogenized and subsampled. The fecal subsamples then were analyzed for N and dry matter (DM). Urine was collected daily in polypropylene bottles containing 75 mL of 6 NHCl. Ten percent of the daily urine volume was subsampled and stored at 20°F until laboratory analysis. Urine was centrifuged at $2000 \times g$ to remove particulate matter and then was analyzed for total N. Feed samples were ground through a 1mm screen before analysis of N and DM. Feed, urine, and feces were analyzed for N on an as-is basis to minimize any loss of gaseous ammonia before analysis.

Plasma samples were harvested from blood samples drawn via jugular venipuncture on d 5, 9, 13, 17, and 23 at least 2 h after feeding and analyzed for IGF-I.

All pigs were euthanized humanely on d 27 by intravenous sodium pentobarbitol administration and transported to the KSU veterinary medicine diagnostic laboratory. Each pig was eviscerated, and individual weights were collected for the heart, lungs, stomach, liver, spleen, kidneys, and small intestine. Additionally, 10th rib fat depth and *longissimus* muscle area (LMA) were measured. These data were collected to help determine changes in body composition after recovery from the disease challenge.

All data were analyzed as a randomized incomplete block design using a mixed model procedure with repeated measures. Pigs were blocked by initial weight and time with individual pig as the experimental unit. Periodic samples by pig were used for the repeated measures. Carcass characteristics and organ weights were adjusted covariately for final body weight. Linear and quadratic polynomial contrasts were used to determine the effects of A. pleuropneumonia challenge over time on all response criteria. Within the challenge treatment, the challenge pigs that were paired with a control were separated to compare results of disease challenge directly between pigs of similar feed-intake levels.

Table 1. Diet Composition

Ingredient	Percent
Corn	64.04
Soybean meal, 46.5% CP	32.89
Monocalcium phosphate	1.22
Limestone	1.10
Salt	.35
Vitamin premix	.25
Trace mineral premix	.15

^aDiet was formulated to contain 1.15% lysine, .75% Ca, and .65% P.

Results and Discussion

A disease status effect and a linear time effect were observed for DM digestibility (P<.01; Table 2). A disease status effect and a quadratic time effect were observed for N digestibility (P<.05). The disease status

effect is a result of greater DM and N digestibilities for A. pleuropneumonia-challenged pigs compared to control ad libitumfed pigs from d 8 to 11 and d 22 to 25 (P<.05) and pair-fed control pigs from d 8 to 11 and d 12 to 15 (P<.05). These periodic differences in DM and N digestibilities contributed to the overall d 8 to 25 increased DM and N digestibilities of the A. pleuropneumonia-challenged pigs versus both control treatments (P < .01). These increases appear to result from more than changes in DM and N intakes. If feed intake were the primary cause, then pair-fed pigs should have greater digestibility than the ad libitum-fed controls. Because this was not the case, the differences in DM and N digestibilities appear directly associated with changes in immune system activity and most likely are results of physiological changes caused by increased levels of proinflammatory cytokines associated with the immune response.

A disease status by time interaction was observed for N retained (P<.05; Table 3). The interaction resulted from decreased N retention during the acute challenge period and both subsequent recovery periods (d 8 to 19), for both pair-fed controls and the A. pleuropneumonia-challenged pigs compared to the ad libitum-fed control pigs. A disease status effect and linear and quadratic time effects were observed for N intake, fecal N, urinary N, and retained N (P < .01). The disease status effect for N intake was due to decreased N intake in the A. pleuropneumonia and pair-fed controls versus that of the ad libitum-fed controls (P<.05) from d 8 to 11, 12 to 15, and d 16 to 19. The fecal N effect was due primarily to the decreased intake N from d 8 to 25 for the A. pleuropneumonia-challenged pigs (P < .01) and pair-fed pigs (P < .01) compared to that for the ad libitum-fed control pigs. In addition, increased DM digestibility of the A. pleuropneumonia-challenged pigs likely contributed to lower fecal N levels compared to that of the ad libitum-fed control pigs.

Urinary N differences resulted from pairfed pigs having the lowest urinary N levels compared to either the ad libitum-fed controls from d 12 to 25 (P<.04) or the A. pleuropneumonia-challenged pigs during d 8 to 11 (P<.04), d 16 to 19 (P<.01), and d 8 to 25 (P<.01). Tendencies for lower urinary N levels of the pair-fed controls compared to the A. pleuropneumonia-challenged pigs were observed both from d 12 to 15 (P < .07) and d 22 to 25 (P<.09). Lower urinary N levels of the pair-fed controls compared to the ad libitum-fed controls were associated with decreased N intake in healthy pigs. Although intake N was lower in A. pleuropneumonia-challenged pigs compared to ad libitum-fed controls, urinary N was not different (P<.48). This lack of difference indicates that the disease challenge increased muscle breakdown above that associated with inadequate feed intake. The loss of muscle during an immune challenge typically is associated with requirements for amino acids above those supplied by feed intake. The increased amino acids are utilized for increased hepatic protein synthesis to support an immune response to the disease challenge.

Greater N retention was observed for the ad libitum-fed control pigs compared to both A. pleuropneumonia-challenged pigs from d 8 to 11 and 12 to 15 (P<.001) and pair-fed controls from d 8 to 11, 12 to 15, and 16 to 19 (P<.03). A tendency for decreased N retention also was observed from d 16 to 19 for A. pleuropneumonia-challenged pigs compared to the ad libitum-fed controls (P<.06). The decreased N retention of the different periods after challenge resulted in lower N retention for both A. pleuropneumonia-challenged pigs and pair-fed pigs compared to that of the ad libitum-fed pigs from d 8 to 25 (P<.01). Although most of the decreased N retention was a result of decreased N intake, the similarity of d 8 to 25 N retentions between the pair-fed pigs and the A. pleuropneumonia-challenged pigs, but different N intakes, further indicate that increased muscle loss likely occurred in the disease-challenge treatment.

A tendency for a quadratic time effect was observed for the percentage of absorbed N retained (P < .08; Table 4). From d 12 to 15, N retention efficiency, both as percentage of

N intake and percentage of absorbed N, was worse for pair-fed pigs (P<.01), with intermediate efficiency for the A. pleuropneumonia-challenged pigs (P<.15) versus control ad libitum-fed pigs. Pair-fed control pigs also tended to be worse than A. pleuropneumonia-challenged pigs from d 12 to15 (P<.10). The lower N retention efficiency from d 12 to 15 was not apparent when pairfed controls were compared only to their disease-challenged counterparts (P<.88). The lack of long-term differences in N retention efficiency indicate that changes in N retention are due primarily to differences in N intake. In addition, the increased shortterm losses in urinary N associated with the muscle loss from the disease challenge did not appear to be great enough to affect N retention efficiency after challenge.

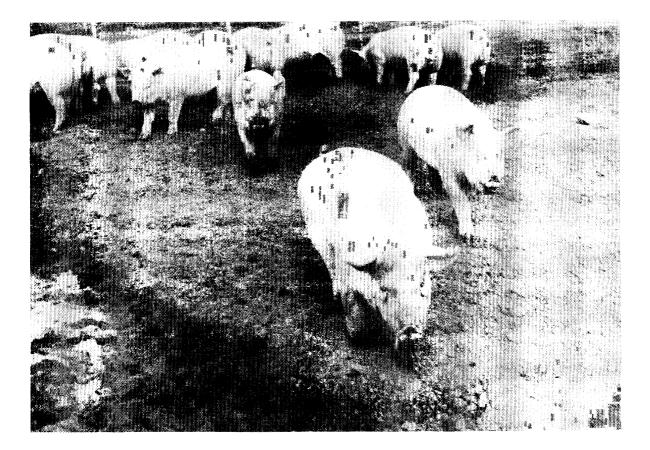
A disease challenge effect and a quadratic time effect were observed for IGF-I concentrations (P<.03; Table 5). Insulin like growth factor-I levels were lowest for A. pleuropneumonia-challenged pigs on d 9 compared to ad libitum-fed control pigs (P<.003) and pair-fed control pigs (P<.001). Insulin like growth factor-I levels of A. pleuropneumonia-challenged pigs tended to remain lower than those of ad libitum-fed control pigs through d 17 (P<.10). Pair-fed control pigs tended to have intermediate IGF-I levels, except on d 17, when they tended to be lower than those of the ad libitum-fed controls (P<.10). Insulin like growth factor-I is a growth mediator that is dependent upon several factors for maximum expression, including feed intake. The decreased IGF-I levels of the A. pleuropneumonia-challenged pigs and the pair-fed pigs were consistent with decreased feed intake and subsequent decreased N retention associated with the disease challenge and recovery. However, IGF-I levels were not different among treatments by d 23. This indicates that although the A. pleuropneumonia challenge did decrease metabolic growth signals during the challenge and recovery, once the disease challenge was overcome, hormonal growth signals likely returned to normal levels. Although IGF-I did return to nonchallenged control levels, the A. pleuropneumonia-challenged pigs had different levels of N retention or N intake at d 23, which indicated that they were still recovering from the effects of the disease challenge.

Carcass characteristics and total organ weights were not affected by treatment, except for stomach weight (P<.03; Table 6). Stomach weight was greater for pair-fed control pigs compared to ad libitum-fed controls (P<.01) and A. pleuropneumoniachallenged pigs (P < .02). Lung weights were greater for A. pleuropneumonia-challenged pigs compared to pair-fed control pigs (P < .05). Higher lung weights were results of some disease-challenge pigs having residual lesions and fluid remaining at the termination of the experiment. Heart weight tended to be greater for ad libitum-fed control pigs compared to A. pleuropneumonia-challenged pigs (P < .10). Both liver weight and total organ weight tended to be greater for the ad libitum-fed control pigs compared to the pair-fed control pigs (P < .09).

A major portion of the differences in N balance between A. pleuropneumonia-challenged pigs and ad libitum-fed control pigs was due to N intake differences; however, a significant portion also was due to nutrient repartioning to the immune response. The differences in N retention between the pairfed controls and ad libitum-fed controls indicate the differences in protein metabolism related to N intake. Differences in N intake and urinary N associated with the lack of difference in N retention for the A. pleuropneumonia-challenged pigs compared to the pair-fed controls indicate the differences associated with repartitioning nutrients to immune response. The changes in plasma IGF-I are also consistent with the results observed for N retention.

The results of this experiment are consistent with those of previous studies and indicate that protein metabolism, as indicated by N retention, is affected negatively by immune activation. They also indicate that an intranasal dose of 5×10^7 to 1×10^8 cfu of *A. pleuropneumonia* is sufficient to produce an acute immune response. These results further indicate that most of the N balance effects from an acute respiratory immune challenge on protein metabolism are due to decreased N intake.

In contrast to typical field conditions, the pigs used in this experiment were maintained in a near ideal environment with minimal outside stress and received a similar infectious dose. The lack of additional stresses such as social interaction with pen mates, reinfection from other pigs, and competition for feed and water likely contributed to the vigorous recovery rate for the survivors in this experiment. But even in these near ideal conditions, the pigs were not able to completely recover from the effects of the acute respiratory challenge. This indicates that an acute challenge using *A. pleuropneumonia* will produce effects still apparent 17 d after challenge, which is longer than the period we observed using a *S. typhimurium* challenge model. Finally, changes in N balance from an acute respiratory disease challenge are due to both reductions in N intake and increased muscle loss during nutrient repartitioning for the immune response.



	Nonchallenged		A. pleurop	A. pleuropneumonia			Probability $(P <)$			
	Ad Libitum	Pair Fed	Ad Libitum ^c	Ad Libitum						
Digestibility ^b , %	Trt 1	Trt 2	Trt 3	Trt 4	1 vs. 2	2 vs. 3	2 vs. 4	1 vs. 4		
Pigs per treatment	7	10	10	30						
D 4 to 7										
Dry matter	87.4 ± 1.2	88.±11.1	87.51 ± .2	86.8. ± 9	.56	.61	.17	.61		
Nitrogen	81.6 ± 1.5	82.6 ± 1.3	82.6 ± 1.5	82.1 ± 1.0	.54	.92	.66	.74		
D 8 to 11										
Dry matter	85.9 ± 1.2	85.8 ± 1.1	87.4 ± 1.2	87. ± 9.9	.92	.28	.03	.06		
Nitrogen	83.0 ± 1.5	83.4 ± 1.3	86.5 ± 1.5	86.6 ± 1.0	.84	.08	.008	.01		
D 12 to 15										
Dry matter	86.1 ± 1.2	85.1 ± 1.1	86.4 ± 1.2	87.0. ± 9	.41	.41	.05	.44		
Nitrogen	83.7 ± 1.5	83.1 ± 1.3	85.5 ± 1.5	85.7 ± 1.1	.72	.18	.04	.15		
D 16 to 19										
Dry matter	85.6 ± 1.2	85.7 ± 1.1	86.6 ± 1.2	86.9.±9	.93	.59	.22	.24		
Nitrogen	$83.21 \pm .5$	84.4 ± 1.3	85.7 ± 1.5	85.3 ± 1.1	.47	.48	.44	.13		
D 22 to 25										
Dry matter	84.2 ± 1.2	85.4 ± 1.1	$86.81 \pm .2$	87.0. ± 9	.35	.35	.09	.02		
Nitrogen	81.81 ± .5	83.8 ± 1.3	86.1 ± 1.5	85.7 ± 1.1	.21	.21	.12	.006		
D 8 to 25										
Dry matter	85.5. ± 8	$85.6 \pm .7$	$86.8 \pm .9$	87.0. ± 7	.93	.09	.002	.004		
Nitrogen	82.9 ± 1.1	83.6 ± 1.0	$85.91 \pm .1$	85.9. ± 9	.38	.01	.001	.001		

 Table 2. Effects of Actinobacillus pleuropneumonia Challenge and Feeding Regimen on Dry Matter and Nitrogen Digestibility in 65-to 120-lb Pigs^a

^aForty seven pigs were used in a randomized incomplete block design with individual pig as the experimental unit. Effects were analyzed using a mixed model with repeated measures.

^bA challenge effect, and a linear time effect were observed for DM digestibility (P<.01). A challenge effect and a quadratic time effect were observed for N digestibility (P<.05).

^cTreatment 3 is a subset of challenged pigs from treatment 4 that were paired with a pair-fed pig in treatment 2.

	Nonchal	lenged	A. pleura	Probability (P <)				
	Ad Libitum	Pair Fed	Ad Libitum ^c	Ad Libitum				
Item ^b , g/d	Trt 1	Trt 2	Trt 3	Trt 4	1 vs. 2	2 vs. 3	2 vs. 4	1vs. 4
Pigs per treatment	7	10	10	30				
D 4 to 7								
N intake	53.4 ± 5.4	51.4 ± 4.6	53.5 ± 5.3	$55.73 \pm .1$.76	.39 .31	.75	.70 .77
Fecal N	9.7 ± 1.1	8.9 ± .95	9.3 ± 1.1	$10.0 \pm .58$.61	.31	.79	.77
Urine N	12.9 ± 2.4	12.9 ± 2.1	15.2 ± 2.2	14.7 ± 1.6	.99	.35	.44	.41
N retained	30.8 ± 3.4	29.5 ± 2.9	28.8 ± 3.1	31.0 ± 2.0	.74	.63	.99	.97
D 8 to 11								
N intake	$60.25 \pm .4$	44.8 ± 4.6	48.5 ± 5.6	44.8 ± 3.2	.02	.99	.60	.008
Fecal N	10.0 ± 1.1	$7.3. \pm 95$	$6.61 \pm .2$	$6.1 \pm .61$.07	.29	.69	.003
Urine N	16.2 ± 2.4	12.8 ± 2.1	18.8 ± 2.2	17.2 ± 1.7	.19	.03	.04	.67
N retained	33.9 ± 3.4	24.7 ± 2.9	23.0 ± 3.2	21.6 ± 2.1	.03	.33	.81	.001
D 12 to 15	55.5 - 5.1	21.7 - 2.9	<u></u>	21.0 - 2.1	.05			
N intake	66.6 ± 5.4	39.1 ± 4.6	43.2 ± 5.3	47.6 ± 3.3	.001	.10	.56	.001
Fecal N	10.7 ± 1.1	$6.2 \pm .95$	$6.21 \pm .1$	$6.8 \pm .62$.002	.57	.98	.003
Urine N	17.7 ± 2.4	12.5 ± 2.1	17.8 ± 2.2	17.4 ± 1.7	.04	.01	.07	.89
N retained	38.1 ± 3.4	20.4 ± 2.9	17.0 ± 2.2 19.2 ± 3.1	23.4 ± 2.1	.001	.34	.89	.001
D 16 to 19	JU.1 - J.4	20.4 - 2.7	17.2 - 5.1	23.4 ± 2.1	.001		.07	.001
N intake	71.1 ± 5.4	50.8 ± 4.6	58.2 ± 5.3	59.8 ± 3.3	.003	.08	.30	.05
Fecal N	11.7 ± 1.1	$8.2 \pm .95$	9.0 ± 1.1	$9.1 \pm .63$.02	.42	.62	.04
Urine N	22.5 ± 2.4	15.5 ± 2.1	22.5 ± 2.2	20.7 ± 1.7	.006	.008	.01	.43
N retained	22.5 ± 2.4 36.8 ± 3.4	27.1 ± 2.9	26.7 ± 3.1	30.0 ± 2.2	.000	.36	.98	.06
D 22 to 25	50.0 ± 5.4	21.1 + 2.7	20.7 ± 5.1	JU.U - 2.2	.02	.50	./0	.00
N intake	79.6 ± 5.4	66.3 ± 4.6	74.9 ± 5.3	78.4 ± 3.3	.05	.01	.23	.84
Fecal N	14.3 ± 1.1	$10.9 \pm .95$	10.8 ± 1.1	$11.4 \pm .63$.02	.64	.98	.07
Urine N	29.6 ± 2.4	23.6 ± 2.1	28.3 ± 2.2	26.9 ± 1.7	.02	.10	.09	.02 .23
N retained	35.6 ± 3.4	31.7 ± 2.9	35.63.1	40.2 ± 2.2	.35	.009	.60	.21
D 8 to 25	55.0 ± 5.4	51.7 ± 2.9	55.05.1	40.2 ± 2.2		.007	.00	.2.1
N intake	69.2 ± 3.7	50.1 ± 3.2	56.3 ± 3.3	57.3 ± 2.3	.001	.18	.05	.004
Fecal N	$11.7 \pm .7$	30.1 ± 3.2 8.1 ± .6	30.3 ± 3.3 8.2 ± .7	37.3 ± 2.3 8.3 ± .4	.001	.18	.82	.004
Urine N	11.7 ± 1.7 21.5 ± 1.8	16.1 ± 1.7	$8.2 \pm .7$ 21.9 ± 1.5	$3.3 \pm .4$ 20.4 ± 1.4	.001	.94	.002	.48
	21.5 ± 1.8 36.1 ± 2.4	10.1 ± 1.7 26.0 ± 2.1		20.4 ± 1.4 28.6 ± 1.7	.002	.002	.002	.48
N retained	30.1 ± 2.4	20.0 ± 2.1	26.2 ± 1.8	20.0 ± 1.7	.001	.70	.41	.002

Table 3. Effect of Actinobacillus pleuropneumonia Challenge and Feeding Regimen on Nitrogen Balance in 65- to 120-lb Pigs^a

^aForty seven pigs were used in a randomized incomplete block design with individual pig as the experimental unit. Effects were analyzed using a mixed model with repeated measures. ^bA challenge effect and both linear and quadratic time effects were observed for N intake, fecal N, urine N, and retained N (P<.01). A challenge × time interaction was observed for retained N (P<.01). ^cTreatment 3 is a subset of challenged pigs from treatment 4 that were paired with a pair-fed pig in treatment 2.

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	Noncha	llenged	A. pleuropneumonia		Probability (P <)			
	Ad Libitum	Pair Fed	Ad Libitum ^c	Ad Libitum				
Efficiency ^b , %	Trt 1	Trt 2	Trt 3	Trt 4	1 vs. 2	2 vs. 3	2 vs. 4	1vs. 4
Pigs per treatment	7	10	10	30				
D 4 to 7								
% of ADFI	57.71 ± 1.8	57.7 ± 9.9	54.4 ± 13.9	55.6 ± 6.2	.99	.88	.85	.87
% of absorbed	70.6 ± 15.5	69.7 ± 13.0	65.8 ± 18.4	67.9 ± 8.0	.96	.90	.90	.87
D 8 to 11								
% of ADFI	56.4 ± 11.8	53.3 ± 9.9	45.2 ± 14.6	43.8 ± 6.5	.84	.71	.41	.34
% of absorbed	67.8 ± 15.5	63.8 ± 13.0	52.3 ± 19.4	50.9 ± 8.5	.84	.68	.40	.33
D 12 to 15								
% of ADFI	57.3 ± 11.8	18.8 ± 9.9	19.0 ± 13.9	38.1 ± 6.6	.01	.97	.10	.15
% of absorbed	68.5 ± 15.5	16.0 ± 13.0	19.3 ± 18.4	43.3 ± 8.6	.009	.88	.08	.15
D 16 to 19								
% of ADFI	52.2 ± 11.8	50.7 ± 9.9	45.0 ± 13.9	50.1 ± 6.8	.92	.79	.96	.87
% of absorbed	62.6 ± 15.5	60.1 ± 13.0	52.9 ± 18.4	59.0 ± 8.8	.90	.80	.94	.84
D 22 to 25			-					
% of ADFI	45.0 ± 11.8	48.1 ± 9.9	48.7 ± 13.9	$51.86 \pm .8$.84	.95	.75	.61
% of absorbed	55.0 ± 15.5	57.2 ± 13.0	56.6 ± 18.4	60.6 ± 8.8	.91	.99	.82	.75
D 8 to 25								
% of ADFI	52.8 ± 7.1	42.8 ± 6.1	39.5 ± 8.4	45.8 ± 4.5	.24	.79	.66	.35
% of absorbed	63.6 ± 9.2	. 49.4 ± 7.9	45.3 ± 11.0	53.2 ± 5.7	.21	.81	.67	.30

Table 4. Effect of Actinobacillus pleuropneumonia Challenge and Feeding Regimen on Nitrogen Retention Efficiency in 65- to 120-lb Pigs^a

^aForty seven pigs were used in a randomized incomplete block design with individual pig as the experimental unit. Effects were analyzed using a mixed model with repeated measures.

^bA tendency for a quadratic time effect was observed for efficiency of absorbed nitrogen (P<.08).

^cTreatment 3 is a subset of challenged pigs from treatment 4 that were paired with a pair-fed pig in treatment 2.

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Item	Nonchallenged		A. pleuropneumonia		Probability (P <)			
	Ad Libitum Trt 1	Pair Fed Trt 2	Ad Libitum ^c Trt 3	Ad Libitum Trt 4	1 vs. 2	2 vs. 3	2 vs. 4	1vs. 4
Pigs per treatment	7	10	10	30				
D 5								
IGF-I, ng/mL	2383 ± 7	274 ± 34	238 ± 41	2402 ± 2	.45	.41	.34	.98
D9								
IGF-I, ng/mL	274 ± 37	275 ± 34	187 ± 39	155 ± 22	.96	.05	.001	.003
D 13								
IGF-I, ng/mL	239 ± 37	221 ± 34	156 ± 39	173 ± 23	.70	.13	.17	.10
D 17								
IGF-I, ng/mL	306 ± 37	228 ± 34	238 ± 41	236 ± 24	.10	.99	.84	.08
D 23								
IGF-I, ng/mL	288 ± 37	270 ± 34	247 ± 39	263 ± 24	.70	.54	.85	.54

Table 5. Effect of Actinobacillus pleuropneumonia Challenge and Feeding Regimen on Plasma IGF-I and Serum Acute Phase Proteins in 65-
to 120-lb Pigs^a

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^aForty seven pigs were used in a randomized incomplete block design with individual pig as the experimental unit. Effects were analyzed using a mixed model with repeated measures.

^bA challenge effect and a quadratic time effect were observed for IGF-I (P<.03).

^cTreatment 3 is a subset of challenged pigs from treatment 4 that were paired with a pair-fed pig in treatment 2.

Item ^b	Nonchallenged		A. pleurop	Probability (P <)				
	Ad Libitum Trt 1	Pair Fed Trt 2	Ad Libitum ^c Trt 3	Ad Libitum Trt 4	1 vs. 2	2 vs. 3	2 vs. 4	1vs. 4
Pigs per treatment	5	7	7	17				
D 27 10 th rib BF, in	.45 ± .05	.38 ± .05	.36 ± .07	.38 ± .03	.30	.87	.93	.20
D 27 LMA, in ²	4.96 ± .21	5.04 ± .18	4.52 ± .18	4.80 ± .11	.78	.10	.24	.48
Organ wt, g								
Heart	332 ± 32	280 ± 28	296 ± 22	277 ± 21	.19	.55	.90	.10
Liver	1605 ± 98	1440 ± 90	1564 ± 123	1516 ± 78	.09	.13	.30	.29
Spleen	204 ± 35	190 ± 30	164 ± 34	169 ± 22	.75	.52	.53	.36
Kidneys	315 ± 22	313 ± 18	337 ± 14	322 ± 12	.94	.26	.66	.77
Lungs	685 ± 87	596 ± 72	788 ± 72	768 ± 47	.45	.07	.05	.41
Stomach	335 ± 16	386 ± 16	359 ± 7	372 ± 12	.01	.19	.36	.02
Small Intestine	1284 ± 95	1370 ± 86	1306 ± 63	1387 ± 73	.38	.78	.81	.22
Total Organ Wt.	4891 ± 190	4441 ± 177	4798 ± 175	4727 ± 100	.09	.16	.17	.45
Organs as % BW	9.35 ± .63	9.46 ± .62	10.1 ± .56	9.79 ± .54	.84	.41	.49	.36

 Table 6. Effect of Actinobacillus pleuropneumonia Challenge and Feeding Regimen on Carcass Characteristics and Organ Weights in 65- to 120-lb Pigs^a

^aForty seven pigs were used in a randomized incomplete block design with individual pig as the experimental unit. Effects were analyzed using a mixed model.

^bA challenge effect was observed for stomach weight (P<.03).

^cTreatment 3 is a subset of challenged pigs from treatment 4 that were paired with a pair-fed pig in treatment 2.