RESEARCH REPORT
ANIMAL SCIENCE

**Title:** Evaluation of dietary soybean meal replacement on grow-finisher pig performance when challenged with PRRS and Mycoplasma – NPB #15-148

Revised

**Investigator:** Nicholas Gabler

**Institution:** Iowa State University

**Date Submitted:** December 8th, 2016

**Industry Summary:** The amino acids requirements of growing-finishing pigs are met by utilizing a variety of protein rich ingredients, including soybean meal (SBM) and distillers dried grains with solubles, as well as synthetic amino acids. As nutritionists have become more comfortable with synthetics, this has obviously reduced the demand for SBM. However, there is growing suspicion that SBM may provide protective benefits to pigs experiencing disease challenges, especially in mid-late finishing phase of production. Anecdotal and unpublished data suggest that limiting the levels of synthetic amino acids in disease-challenged pigs can result in improved growth and financial performance. Therefore, our objectives were: 1) To determine whether dietary soybean meal displacement by crystalline amino acids increases the severity of a health challenge in finisher pigs; and 2) To determine the effect of a late breaking health challenge on finisher pig growth and carcass performance. To test these objectives, two experiments where conducted using two groups of 96 and 90 finisher pigs. In both experiments, pigs were stratified by BW across either a high SBM (HSBM) or low SBM (high synthetic Trp; LSBM) diet. Both diets were formulated to contain 0.70% TID Lys and were isocaloric; only crude protein was different between HSBM and LSBM diets (15.9 vs. 13.5%, respectively). In both experiments, all pigs were co-inoculated intratracheally with Mycoplasma *hyopneumonia* (MHP) and intramuscularly with a field strain of PRRSV, weekly pig performance and serology, and subsequent carcass characteristics were assessed over a 28 or 49 day test period (Exp1 and Exp2, respectively). For each experiment, pen was considered the experimental unit with Exp1 having 8 pens, 6 pigs per pen per treatment and Exp2 having 9 pens, 5 pigs per pen per treatment. Summary of results:

During the pre-challenge period, no performance differences were detected between the LSBM and HSBM fed pigs.

- As expected, antibody titers for PRRSV and MHP increased post inoculation. However, change in PRRSV antibody titers from 0 to 28 days post inoculation tended to be increased in LSBM verses HSBM pigs.
- MHP antibody titers and lung lesion scores did not differ between dietary treatments.
- No difference in ADG, ADFI or G:F between pigs challenges with PRRSV and MHP fed either a LSBM or HSBM diets.
- There was also no difference in hot carcass weight, yield percentage or muscle depth. However, the LSBM pigs had an increase in carcass fat depth compared to the HSBM pigs.
- Altogether, these data indicate that diets with increased synthetic AA or decreased SBM do not alter pig performance during a late breaking respiratory health challenge.

**Keywords:** finisher pig, *Mycoplasma hyopneumoniae*, performance, PRRS, soybean meal

**Scientific Abstract:** Porcine reproductive and respiratory syndrome virus (PRRSV) and Mycoplasma hyopneumoniae (MHP) are two significant respiratory pathogens in finishing pigs, often found in tandem. Recent anecdotes have implied that increasing soybean meal content of diets can be beneficial in pathogen-challenged pigs, while other reports suggest similar benefits by increasing the use of synthetic AA, specifically Trp. The objective of this study was to determine if increased synthetic Trp replacing a portion of SBM would impact performance of late finishing pigs dual-challenged with PRRSV and MHP. To test this, two experiments were conducted. In the initial experiment (Exp1), ninety-six mixed sex pigs (48 gilts and 48 barrows, PIC 359 boar x PIC sows) were randomly assigned to one of two dietary treatments. The dietary treatments included a high SBM (HSBM) or low SBM (LSBM) diet. Both diets were formulated to contain 0.70% TID Lys and were isocaloric; only crude protein was different between HSBM and LSBM diets (15.9 vs. 13.5%, respectively). These diets were also formulated so that the low SBM diet provided higher amounts of synthetic Lys, Thr and Try, while keeping the Dig Thr/lys and Dig Tryp/lys ratios similar across both diets. Pigs were penned in groups of six with eight pens per treatment. After 96 days on feed and at 120.5 ± 1.42 kg BW, all pigs where health challenged for 28 days. All pigs were inoculated intratracheally with Mycoplasma *hyopneumonia* (MHP) and intramuscularly with a field strain of PRRSV. In the second experiment (Exp2), ninety barrows (89.3 ± 0.94 kg BW, PIC 359 boar x PIC

sows), naïve to PRRSV, were assigned to one of two treatments for 49 d. Dietary treatments were the same as Exp1 and pigs were penned in groups of five with nine pens per treatment. On dpi 0, all pigs were dual inoculated with PRRSV and MHP as in Exp1. In both experiments individual body weight and pen feed intake were recorded weekly and pen feed efficiency was calculated until market (28 and 49 dpi, respectively for Exp1 and 2). PRRS and MHP serology were assessed weekly and pig carcass performance was assessed at slaughter. During the 96 day pre-challenge period (Exp1), no performance differences were detected between the two dietary treatments. As expected for both experiments, antibody titers for PRRSV and MHP increased post inoculation. However, change in PRRSV antibody titers from 0 to 28 dpi tended ($P = 0.09$) to be increased in LSBM verses HSBM pigs. MHP antibody titers and lung lesion scores did not differ between treatments. For the 28 or 49 day post-challenge period, there was no difference in ADG, ADFI or G:F due to diet ($P > 0.10$). Additionally, no diet difference in hot carcass weight, yield percentage or muscle depth were reported in either Exp 1 or 2 ($P > 0.10$). However, compared to the HSBM pigs, the LSBM pigs tended to have an increase in carcass fat depth ($P = 0.078$, Exp1), while loin muscle depth tended to be increased in Exp2 ($P = 0.087$). Altogether, these data indicate that diets with increased synthetic AA or decreased SBM do not alter pig performance or health during a late breaking respiratory health challenge.

**Introduction:** In pork production, feed costs generally account for 60-70% of total production costs. To combat this, there has been an increase in the use of synthetic amino acid, especially in finishing pig diets. Crystalline amino acids can be used to replace soybean meal (**SBM**) without altering performance in healthy pigs (Madrid et al., 2013). Recently, attention has been given to SBM as it may be beneficial to feed when pigs face a health challenge (Rochell et al., 2015). Although the mechanism is unclear, it is speculated that nonnutritive, anti-inflammatory compounds in SBM like isoflavones may be involved (Greiner et al., 2001b, a). Interestingly, few study have address this occurrence under a controlled multifactorial heath challenge in finisher pigs.

Respiratory infections are the most common health challenges reported in grow/finish pigs, with 75% of grow/finish death loss related to respiratory problems (NAHMS, 2009; USDA, 2015). Several respiratory pathogens have been identified to however, two common pathogens in finishing pigs are porcine reproductive and respiratory syndrome virus (**PRRSV**) and *Mycoplasma hyopneumoniae* (**MHP**); 53% and 58% of grow/finish sights reported disease problems with PRRSV and MHP, respectively (USDA, 2015). Clinical signs of MHP include chronic, nonproductive coughing, which appears about 21 days after infection, but the incubation time varies greatly under

field conditions. *Mycoplasma hyopneumoniae* modifies the immune system of the respiratory tract allowing infection to persist for several months (Sørensen et al., 1997; Pieters et al., 2009). Consequently, pigs infected with MHP are predisposed to concurrent infections with other respiratory bacteria and/or viruses, like PRRSV (Thacker et al., 1999; Bush et al., 2003).

The current study was conducted to determine whether dietary SBM displacement by crystalline AA increases the severity of a dual infection with PRRSV and MHP in late-finishing pigs. A secondary objective of the study was to determine the effect of a late breaking health challenge on finisher pig growth performance and carcass characteristics.

**Objectives:** The cost to production and economics of a late breaking disease challenges in finishing pigs can be very high. The economic losses attributed to Porcine Reproductive and Respiratory Syndrome virus (PRRS) infection are estimated to cost the U.S. swine industry more than $664 million annually (Holtkamp et al., 2013). While significant advances have been made through research efforts to enhance our understanding of PRRS at the animal health and genomic level, this disease still remains a significant issue in the U.S. swine industry. It has been widely suggested that high soybean meal (SBM) levels may significantly reduce the negative effect of diseases such as PRRS and PCV2 on ADG and G:F under natural outbreaks. Although, the mechanism is unclear, it is speculated that the SBM isoflavones with their anti-inflammatory properties may be involved. Interestingly, few study have address this phenomenon under a controlled dual heath challenge in finisher pigs and in which tryptophan:lysine ratio are about 17%. Although we clearly know that PRRSV attenuates ADG, reduces whole body protein and fat accretion, nutrient and energy digestibility and subsequent carcass quality (NPB 12-162), it is possible that high crystalline amino acids substitution in place of SBM may exacerbate this health challenge. Therefore, our objectives were:

i. To determine whether dietary soybean meal displacement by crystalline amino acids increases the severity of a health challenge in finisher pigs.

ii. Determine the effect of a late breaking health challenge on finisher pig growth and carcass performance.

**Materials & Methods:** Two experiments were conducted to determine whether feeding a high SBM or low SBM, high crystalline amino acid diet in the late finishing phase improves performance during a multifactorial health challenge. All animal work was approved by the Iowa State University

Institutional Animal Care and Use Committee (IACUC# 11-14-7885-S) and adhered to the ethical and humane use of animals for research.

***Animals, Housing Experimental Design, and Diets***

In the initial experiment (Exp1), ninety-six mixed sex pigs (48 gilts and 48 barrows, 120.5 ± 1.42 kg BW, PIC 359 boar x PIC sows) were randomly assigned to one of two treatments for 28 d, when pigs reached market weight. Treatments included a high SBM (HSBM) or low SBM (LSBM) diet. Pigs were penned in groups of six with eight pens per treatment. Both diets were formulated to meet or exceed NRC (NRC, 2012) requirements for amino acids, vitamins, and minerals (Table 1). Pigs had ad libitum access to feed and water at all times. Prior to the experimental period, pigs were naturally exposed to a wild-type strain of PRRSV (strain ORF5 RFLP 1-7-4). On days post inoculation (dpi) 0, all pigs were dual inoculated with 3.6 million infective virions PRRSV (strain ORF5 RFLP 1-18-4) intramuscularly and 10,000 color changing units (CCU) of Mycoplasma *hyopneumonia* (LI32, strain 232) intratracheally.

In the second experiment (Exp2), ninety barrows (89.3 ± 0.94 kg BW, PIC 359 boar x PIC sows), naïve to PRRSV, were assigned to one of two treatments for 49 d, when pigs reached market weight. Treatments were the same as Exp1. Pigs were penned in groups of five with nine pens per treatment. On dpi 0, all pigs were dual inoculated with 450,000 infective virions PRRSV (strain ORF5 RFLP 1-18-4) intramuscularly and with 10,000 CCU of MHP (LI32, strain 232) intratracheally.

In both experiments individual body weight and pen feed intake were recorded weekly and pen feed efficiency was calculated. Two pigs per pen were bled weekly via jugular venipuncture into BD Vacutainer serum tubes (BD, Franklin Lakes, NJ), while pigs were snare restrained. Serum was separated by centrifugation (2000 x g for 15 min at 4°C), pooled by pen, and submitted to the Iowa State University Veterinary Diagnostic Laboratory (ISU VDL, Ames, IA) for analysis. In Exp1, serum PRRSV and MHP antibodies were determined. In Exp2, PRRSV quantitative real-time RT-PCR (qPCR) as well as PRRSV and MHP antibodies were analyzed. Testing for PRRSV was performed with commercial reagents (VetMAX NA and EU PRRSV real-time RT-PCR, Thermo Fisher Scientific, Waltham, MA). Commercial ELISA kits were used to detect anti-PRRSV and anti-MHP antibodies per manufacturer's instructions.

At the end of each experiment (28 dpi Exp1 and 49 dpi Exp2, respectively), pigs were sent to a commercial slaughter facility (Tyson Foods, Storm Lake, IA) where carcass data was collected. Hot carcass weight (HCW), fat depth and loin muscle depth were collected off the midline of the posterior portion of the loin. Carcass yield was determined from HCW and final BW. Percent lean was

estimated by applying the following equation: 58.86 – [fat depth (mm) x 0.61] + [loin depth (mm) x 0.12].

***Calculations and Statistical Analysis***

All data were analyzed using the MIXED procedure of SAS version 9.4 (SAS Inst. Inc., Cary, NC), and pen was considered the experimental unit for all measures. In Exp1, 0 dpi PRRSV antibody titer was used as a covariate for dpi 14 and 28 titers as well as the change in antibody from dpi 0-28; the same was done for MHP titers. Also in Exp 1, the main effects of treatment, sex, dpi and their interaction was determined for antibody titers. Growth performance data for Exp1 were analyzed as repeated measures. In Exp2, all data were analyzed as repeated measures. All data are reported as least squares means ± pooled SEM and are considered significant if $P \leq 0.05$ and a trend if $P \leq 0.10$.

**Results:**

***Viremia and Antibody Response***

In Exp1 pigs were naturally exposed to PRRSV at ~36 kg BW which lead to existing PRRSV antibodies prior to experimental infection with a different strain of PRRSV at 120.5 kg BW (Figure 1). Therefore, at 0 dpi HSBM pigs had higher serum antibody titer than LSBM pigs (1.55 vs 1.12 S/P ratio, $P = 0.011$). At 14 and 28 dpi there were elevated PRRSV antibodies detected from serum compared to 0 dpi; however, no treatment differences were reported in serum PRRSV antibodies ($P > 0.10$; Figure 1). The change in PRRSV antibody titer (28 dpi titers) was not different ($P = 0.698$) when adjusted for 0 dpi PRRSV titers, which was a significant covariate ($P = 0.050$). There was no difference in MHP antibody titers between HSBM and LSBM diets ($P > 0.10$, Figure 2). Evaluation of lung lesions at slaughter approximately 28 dpi showed that there were no difference between the HSBM and LSBM diets (11.45 vs. 15.89%, respectively, $P = 0.180$).

In Exp2, all pigs were naïve to PRRSV prior to inoculation based on serum qPCR analysis (Figure 3). After inoculation on 0 dpi, PRRS virus was detected from serum and peaked at dpi 7 and the virus became undetectable by dpi 49, regardless of dietary treatment (Trt x dpi $P = 0.875$, Figure 3). Similarly, PRRS and MHP antibodies were detected after inoculation, peaking at 14 and 28 dpi, respectively (Figure 4 and 5). These data lead to a significant dpi effect on PRRS viremia and antibody and MHP antibody ($P < 0.001$). However, there was no effect of dietary treatment or day by treatment interaction for PRRS viremia or PRRSV and MHP antibody titers ($P > 0.10$).

***Growth Performance and carcass data***

The effect of HSBM and LSBM on BW changes, ADG, ADFI and G:F are summarized in Table 2 and Figure 6 for Exp1 and Table 3 and Figure 7 for Exp2. Growth performance was reduced after pigs were infected with PRRSV and MHP, leading to a significant dpi effect ($P < 0.001$) in both studies. In Exp1, there was no performance differences between diets reported in the 96 day pre-challenge period, even though the pigs were naturally exposed to PRRS (data not shown). Further, in Exp1, there were no dietary treatment differences or treatment by day interactions for ADG, ADFI, or G:F ($P > 0.10$, Table 2). In combination with the pathogen challenge, the noted performance decrease at dpi 0-7 in Exp1 may also be attributed to an out of water event for 24 hours due to a broken water pump. However, feed intake over this period was not greatly impacted. In Exp2, similarly, there were no treatment or treatment by day interactions were observed for ADG or G:F ($P > 0.10$, Table 3). However, there was a tendency ($P = 0.066$) for a treatment by day interaction for ADFI, in which there was a greater reduction in LSBM fed pigs at dpi 14 and 21, but an ADFI increased in LSBM pigs at dpi 28 compared to the HSBM fed finisher pigs (Table 3).

When pigs reached market weight (average 137.7 and 120.8 kg BW for Exp. 1 and 2, respectively), all pigs were sent to a commercial slaughter facility where carcasses were evaluated (Table 4). There were no dietary treatment differences in HCW, carcass yield, or percent lean in either experiment after 28-49 days after the health challenge ($P > 0.10$). In Exp1, loin muscle depth was not different between dietary treatments ($P = 0.739$); however, in Exp2 there was a tendency for LSBM to increase loin muscle depth (7.12 vs 6.89 cm, $P = 0.087$). The LSBM diet fed pigs tended to have increased back fat depth compared to the HSBM pigs in Exp1 (1.44 vs 1.24 cm, $P = 0.078$). However, no back fat depth differences were observed between diets in Exp2 ($P > 0.10$, Table 4).

**Discussion:** Porcine reproductive and respiratory syndrome virus and Mycoplasma hyopneumoniae are two prevalent respiratory diseases, often found in tandem, that cause morbidity and mortality in grow-finish pigs. Pigs dual challenged with PRRSV and MHP exhibited a classical PRRS viremia response compared with pigs challenged with PRRSV alone, with viremia peaking at 7 dpi and decreasing thereafter (Greiner et al., 2000; Zimmerman et al., 2012). Similarly, co-infected pigs demonstrated a classical PRRSV and MHP antibody response compared to pigs challenged with PRRSV or MHP alone (Sheldrake et al., 1990; Zimmerman et al., 2012; Bourry et al., 2015). There is evidence that PRRSV can be potentiated by MHP infection (Thacker et al., 1999); however, this was not reported in the current study. Furthermore, no treatment differences in viremia or antibody responses were reported in the current study due to HSBM verses LSBM diets. This is in opposition to

a recent study where nursery pigs fed a HSBM diet had reduced PRRS viremia at 14 dpi (Rochell et al., 2015). Our study was a coinfection model in late-finishing pigs, whereas Rochell et al. used a PRRSV only infection model in nursery pigs, which may have led to their reported differences.

In finishing diets, crystalline AA are used extensively to reduce diet cost and crude protein utilization. These diets do not reduce performance in healthy pigs (Madrid et al., 2013), and our data reports no adverse effects of feeding crystalline AA during health challenges compared to a HSBM diet. In contrast, performance benefits have been reported in health challenged pigs fed an increased SBM diet, although most reports have been anecdotal (Johnston et al., 2010; Rocha et al., 2013; Boyd and Zier-Rush, 2014), with one study published (Rochell et al., 2015). Our data reports no difference between HSBM and LSBM diets for growth performance or feed conversion in either experiment. At the conclusion of Exp1, ADG and G:F appeared to diverge, with pigs fed HSBM diets having higher performance parameters. The duration of Exp1 was only four weeks, so it was proposed that if diets were fed for a longer period during the PRRS-MHP challenge, differences would be observed between treatments. No differences in growth performance were reported in Exp2. However, differences in initial PRRSV status between Exp1 and Exp2 may have caused different results.

Reduced growth performance has been previously reported in pigs co-infected with PRRSV and MHP (Roberts and Almond, 2003; Bourry et al., 2015), and our study agrees with these findings; however, the current study utilized late-finishing pigs compared to nursery or grow-finish pigs in the aforementioned studies. Attenuated growth performance is reflected in the whole-body composition, and thereby carcass characteristics, of PRRSV infected pigs. Pigs infected with PRRSV had reduced amount of lipid. Interestingly though, pigs co-infected with MHP and PRRSV had no differences in whole-body composition compared to pigs infected with PRRSV alone (Escobar et al., 2004). In the current study, there were generally no treatment differences in carcass characteristics; however, LSBM diets tended to increase backfat depth in Exp1 and loin muscle depth in Exp2. This may be explained by the crude protein content of the diets (15.7 vs 13.6%, HSBM and LSBM, respectively). Increasing CP in the diet traditionally leads to a decrease in backfat thickness (Hansen and Lewis, 1993). Increasing CP in diets generally also leads to an increased loin muscle depth, which was not reported in this study.

Altogether, these data indicate that there is minimal benefit to increasing SBM in diets of finisher pigs dual challenged with PRRSV and MHP. No differences in disease status were reported based on serum PRRS viremia and antibody or MHP antibody response. There were no differences

reported in growth performance or feed efficiency during the challenge period. Similarly, there were no significant differences in carcass characteristics.

## REFERENCES


Bourry, O., C. Fablet, G. Simon, and C. Marois-Créhan. 2015. Efficacy of combined vaccination against Mycoplasma hyopneumoniae and porcine reproductive and respiratory syndrome virus in dually infected pigs. Veterinary Microbiology 180: 230-236.

Boyd, R. D., and C. E. Zier-Rush. 2014. Managing systemic disease stress in commercial pig production: cost and possible nutritional practices to reduce performance loss. In: ASAS-ADSA Midwest Meetings, Des Moines, IA

Bush, E. J., E. L. Thacker, and S. L. Swenson. 2003. National seroprevalence of PRRS, mycoplasma, and swine influenza virus. In: 46th Annual Meeting of the American Association of Swine Practitioners. p 491-494.

Escobar, J., W. G. Van Alstine, D. H. Baker, and R. W. Johnson. 2004. Decreased Protein Accretion in Pigs with Viral and Bacterial Pneumonia Is Associated with Increased Myostatin Expression in Muscle. The Journal of Nutrition 134: 3047-3053.

Greiner, L. L., T. S. Stahly, and T. J. Stabel. 2000. Quantitative relationship of systemic virus concentration on growth and immune response in pigs. Journal of Animal Science 78: 2690-2695.

Greiner, L. L., T. S. Stahly, and T. J. Stabel. 2001a. The effect of dietary soy daidzein on pig growth and viral replication during a viral challenge. Journal of animal science 79: 3113-3119.

Greiner, L. L., T. S. Stahly, and T. J. Stabel. 2001b. The effect of dietary soy genistein on pig growth and viral replication during a viral challenge. Journal of animal science 79: 1272-1279.

Hansen, B. C., and A. J. Lewis. 1993. Effects of dietary protein concentration (corn:soybean meal ratio) on the performance and carcass characteristics of growing boars, barrows, and gilts: mathematical descriptions. Journal of animal science 71: 2122-2132.

Johnston, M. E., R. D. Boyd, C. E. Zier-Rush, and C. E. Fralick. 2010. Soybean meal level modifies the impact of high immune stress on growth and feed efficiency in pigs. Journal of Animal Science 88 (E-Suppl. 3).

Madrid, J., S. Martínez, C. López, J. Orengo, M. J. López, and F. Hernández. 2013. Effects of low protein diets on growth performance, carcass traits and ammonia emission of barrows and gilts. Animal Production Science 53: 146-153.

NAHMS. 2009. PRRS Seroprevalence on U.S. Swine Operations, National Animal Health Monitoring Service.

NRC. 2012. Nutrient Requirements of Swine: Eleventh Revised Edition. The National Academies Press.

Pieters, M., C. Pijoan, E. Fano, and S. Dee. 2009. An assessment of the duration of Mycoplasma hyopneumoniae infection in an experimentally infected population of pigs. Veterinary Microbiology 134: 261-266.

Roberts, N. E., and G. W. Almond. 2003. Infection of growing swine with porcine reproductive and respiratory syndrome virus and Mycoplasma hyopneumoniae — Effects on growth, serum metabolites, and insulin-like growth factor-I. The Canadian Veterinary Journal 44: 31-37.

Rocha, G. C., R. D. Boyd, J. A. S. Almeida, Y. Liu, T. M. Che, R. N. Dilger, and J. E. Pettigrew. 2013. Soybean meal level in diets for pigs challenged with porcine reproductive and respiratory syndrome (PRRS) virus. Journal of Animal Science 92 (E-Suppl. 2).

Rochell, S. J., L. S. Alexander, G. C. Rocha, W. G. Van Alstine, R. D. Boyd, J. E. Pettigrew, and R. N. Dilger. 2015. Effects of dietary soybean meal concentration on growth and immune response of pigs infected with porcine reproductive and respiratory syndrome virus. Journal of Animal Science 93: 2987-2997.

Sheldrake, R. F., I. A. Gardner, M. M. Saunders, and L. F. Romalis. 1990. Serum antibody response to Mycoplasma hyopneumoniae measured by enzyme-linked immunosorbent assay after experimental and natural infection of pigs. Australian Veterinary Journal 67: 39-42.

Sørensen, V., P. Ahrens, K. Barfod, A. A. Feenstra, N. C. Feld, N. F. Friis, V. Bille-Hansen, N. E. Jensen, and M. W. Pedersen. 1997. Mycoplasma hyopneumoniae infection in pigs: Duration of the disease and evaluation of four diagnostic assays. Veterinary Microbiology 54: 23-34.

Thacker, E. L., P. G. Halbur, R. F. Ross, R. Thanawongnuwech, and B. J. Thacker. 1999. Mycoplasma hyopneumoniae Potentiation of Porcine Reproductive and Respiratory Syndrome Virus-Induced Pneumonia. Journal of Clinical Microbiology 37: 620-627.

USDA. 2015. Swine 2012 Part II: Reference of Swine Health and Health Management in the United States, 2012, USDA-APHIS-VS-CEAH-NAHMS, Fort Collins, CO.

Zimmerman, J. J., D. A. Benfield, S. A. Dee, M. P. Murtaugh, T. Stadejek, G. W. Stevenson, and M. Torremorell. 2012. Chapter 31 - Porcine Reproductive and Respiratory Syndrome Virus (Porcine Arterivirus). In: J. J. Zimmerman, L. A. Karriker, A. Ramirez, K. J. Schwartz and G. W. Stevenson (eds.) Diseases of Swine (Tenth Edition). p 461-486. John Wiley and Sons, Inc.

**Table 1.** Experimental diet composition, as fed basis

| **Ingredients, %** | **HSBM** | **LSBM** |
|---|---|---|
| Corn | 63.46 | 70.05 |
| Corn DDGS | 20.00 | 20.00 |
| Soybean Meal, 46% | 11.35 | 4.45 |
| Choice White Grease | 3.35 | 3.28 |
| Limestone | 0.95 | 0.93 |
| Salt | 0.44 | 0.44 |
| Lysine-Sulphate | 0.34 | 0.64 |
| Commercial VTM[1] w/o Phytase | 0.12 | 0.12 |
| Optiphos 1000 | 0.01 | 0.01 |
| L-Threonine | 0.00 | 0.07 |
| L-Tryptophan | 0.00 | 0.04 |
| *Calculated composition* | | |
| ME Swine, kcal/lb | 1573 | 1573 |
| Crude Protein, % | 15.9 | 13.5 |
| True Dig. Lys, % | 0.70 | 0.70 |
| Dig lys/ME, g/kCal | 2.02 | 2.02 |
| Dig TSAA/lys, % | 0.58 | 0.58 |
| Dig Thr/lys, % | 0.65 | 0.65 |
| Dig Tryp/lys, % | 0.18 | 0.18 |
| Calcium, % | 0.44 | 0.41 |
| Phosphorus, % | 0.36 | 0.34 |

[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.

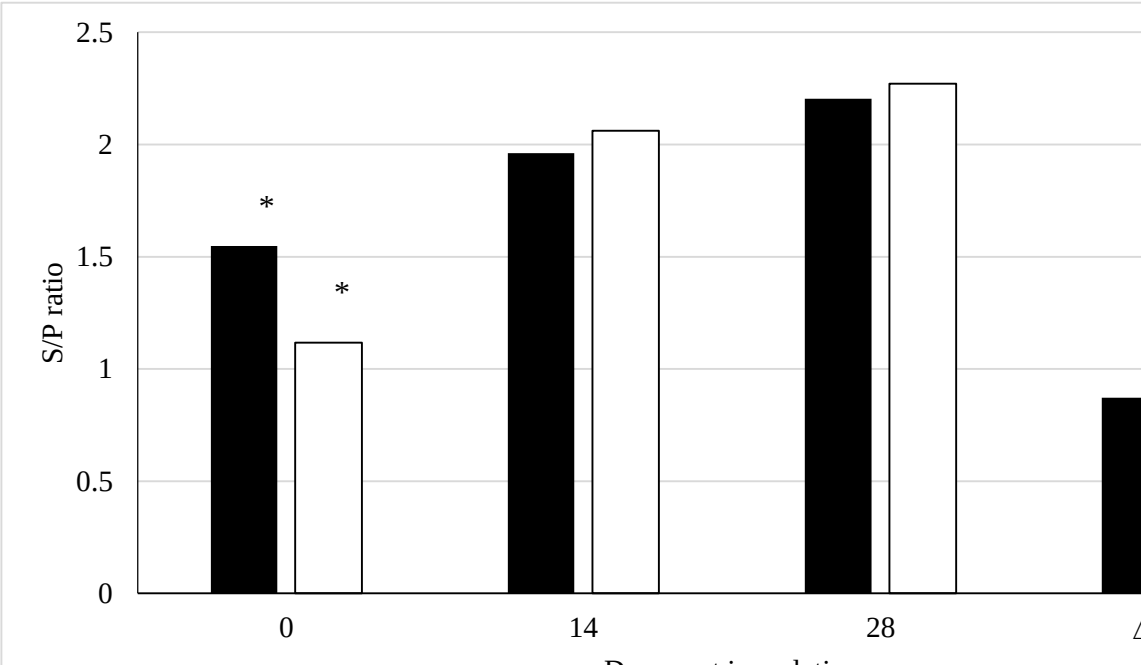


**Figure 1.** PRRS serum antibody titer differences of pigs fed HSBM or LSBM diets when challenged with PRRSV and MHP. Values represent least squares means of 8 pens of 6 pigs (Exp1).

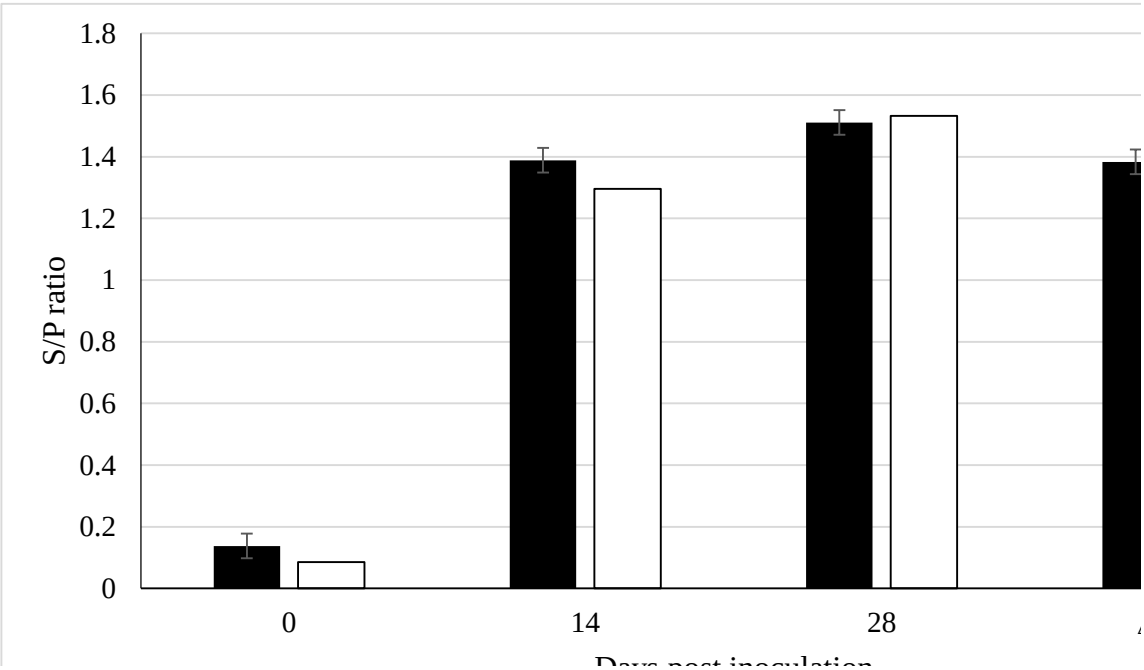


**Figure 2.** *Mycoplasma hyopneumoniae* serum antibody titer differences of pigs fed HSBM or LSBM diets when challenged with PRRSV and MHP. Values represent least squares means of 8 pens of 6 pigs (Exp1).

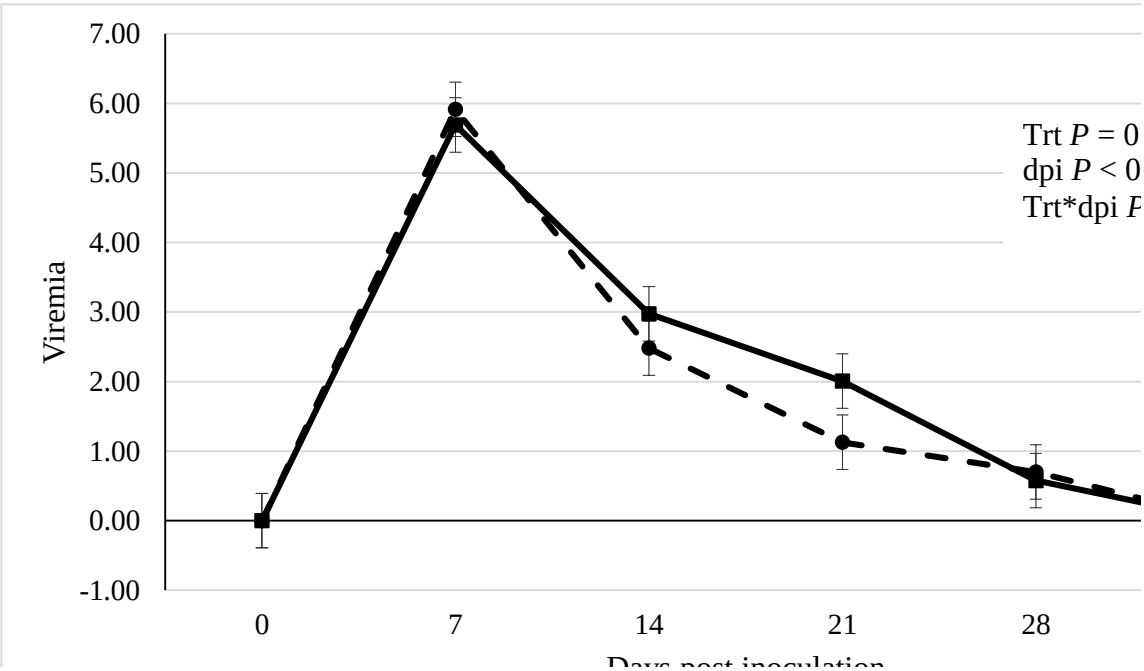


**Figure 3.** PRRSV serum viremia differences of pigs fed HSBM or LSBM diets when challenged with PRRSV and MHP. Values represent least squares means of 9 pens of 5 pigs (Exp2).

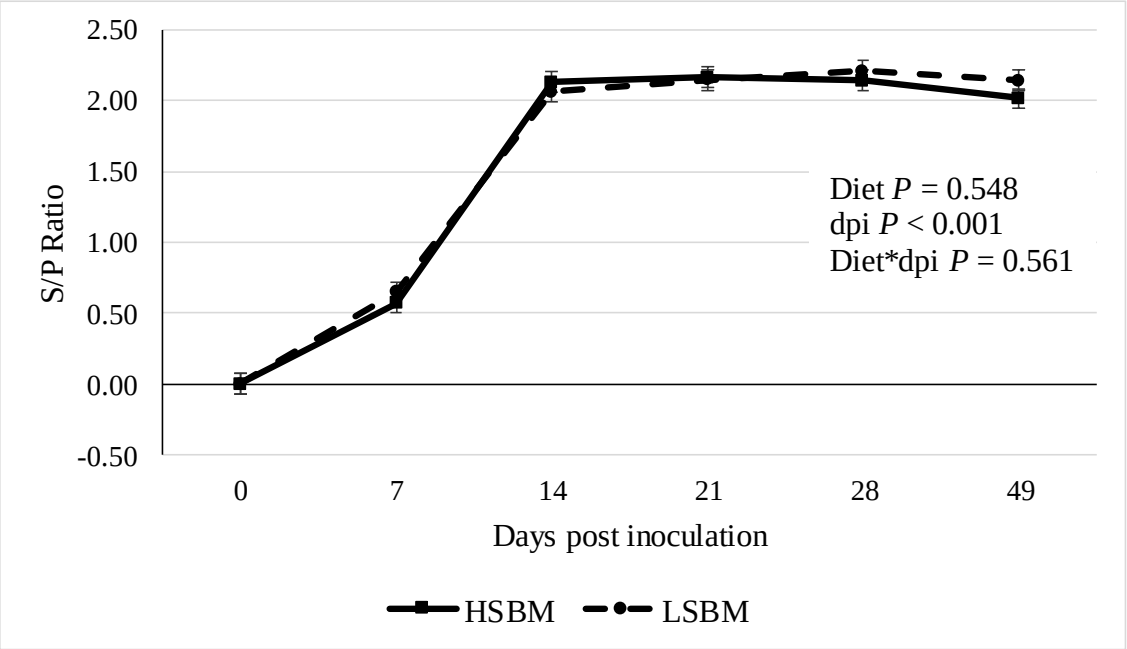


**Figure 4.** PRRS serum antibody titer differences of pigs fed HSBM or LSBM diets when challenged with PRRSV and MHP. Values represent least squares means of 9 pens of 5 pigs (Exp2).

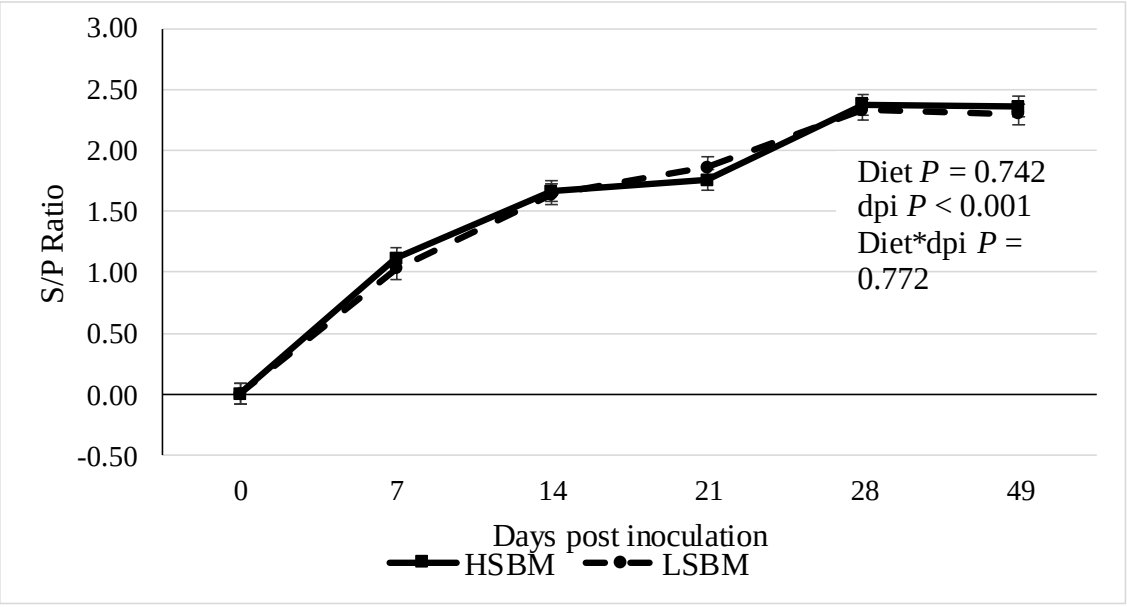


**Figure 5.** *Mycoplasma hyopneumoniae* serum antibody titer differences of pigs fed HSBM or LSBM diets when challenged with PRRSV and MHP. Values represent least squares means of 9 pens of 5 pigs (Exp2).

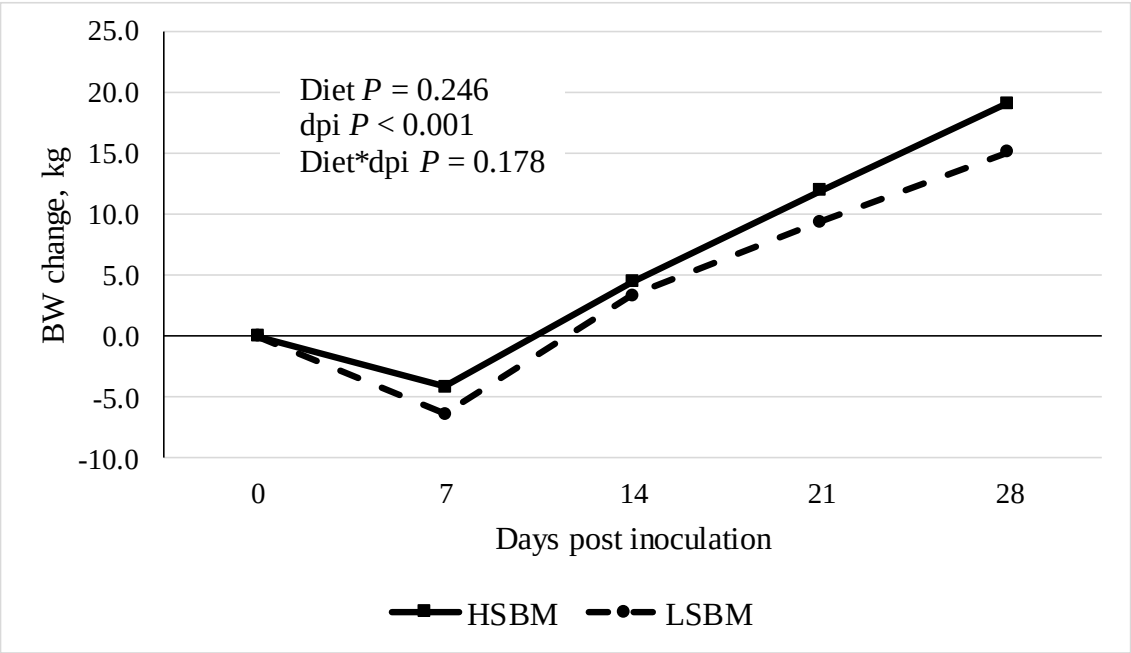


**Figure 6.** Growth differences of pigs fed HSBM or LSBM diets when challenged with PRRSV and MHP. Values represent least squares means of 8 pens of 6 pigs (Exp1).

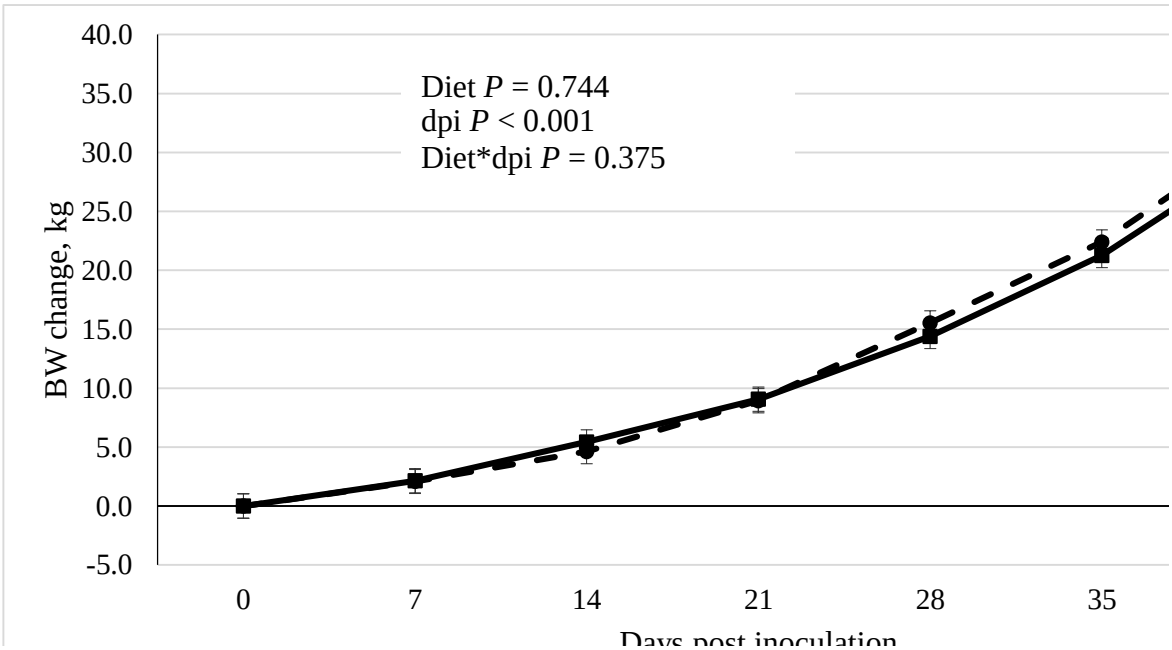


**Figure 7.** Growth differences of pigs fed HSBM or LSBM diets when challenged with PRRSV and MHP. Values represent least squares means of 9 pens of 5 pigs (Exp2).

**Table 2.** Effects of dietary SBM, PRRSV and MHP infection on growth performance (Exp1).

| Parameter[1] | HSBM | LSBM | SEM | *P*-value | | |
|---|---|---|---|---|---|---|
| | | | | ***Diet*** | ***dpi*** | ***Diet *dpi*** |
| *Average Daily Gain, kg* | | | | | | |
| Dpi -7-0 | 0.62 | 0.94 | 0.171 | 0.291 | <0.001 | 0.362 |
| Dpi 0- 7 | -0.60 | -0.92 | | | | |
| Dpi 8-14 | 1.23 | 1.39 | | | | |
| Dpi 15-21 | 0.94 | 0.76 | | | | |
| Dpi 22-28 | 1.19 | 0.94 | | | | |
| *Average Daily Feed Intake, kg* | | | | | | |
| Dpi -7-0 | 3.70 | 3.72 | 0.100 | 0.651 | <0.001 | 0.924 |
| Dpi 0- 7 | 2.08 | 2.04 | | | | |
| Dpi 8-14 | 2.26 | 2.16 | | | | |
| Dpi 15-21 | 2.65 | 2.58 | | | | |
| Dpi 22-28 | 3.21 | 3.13 | | | | |
| *Gain:Feed* | | | | | | |
| Dpi -7-0 | 0.16 | 0.26 | 0.072 | 0.237 | <0.001 | 0.245 |
| Dpi 0- 7 | -0.27 | -0.47 | | | | |
| Dpi 8-14 | 0.54 | 0.65 | | | | |
| Dpi 15-21 | 0.35 | 0.30 | | | | |
| Dpi 22-28 | 0.38 | 0.30 | | | | |

n=8 pens/trt

**Table 3.** Effects of SBM, PRRSV and MHP infection on growth performance (Exp2).

| Parameter | HSBM | LSBM | SEM | *P*-value | | |
|---|---|---|---|---|---|---|
| | | | | ***Diet*** | ***dpi*** | ***Diet*dpi*** |
| *Average Daily Gain, kg* | | | | | | |
| Dpi 0-7 | 1.07 | 1.09 | 0.070 | 0.490 | <0.001 | 0.447 |
| Dpi 8-14 | 0.30 | 0.30 | | | | |
| Dpi 15-21 | 0.55 | 0.42 | | | | |
| Dpi 22-28 | 0.51 | 0.62 | | | | |
| Dpi 29-35 | 0.76 | 0.94 | | | | |
| Dpi 36-42 | 0.98 | 0.97 | | | | |
| Dpi 43-49 | 0.68 | 0.71 | | | | |
| *Average Daily Feed Intake, kg* | | | | | | |
| Dpi 0-7 | 3.26 | 3.27 | 0.099 | 0.744 | <0.001 | 0.066 |
| Dpi 8-14 | 1.93 | 1.84 | | | | |
| Dpi 15-21 | 2.14 | 1.97 | | | | |
| Dpi 22-28 | 2.77 | 2.97 | | | | |
| Dpi 29-35 | 2.49 | 2.77 | | | | |
| Dpi 36-42 | 2.62 | 2.65 | | | | |
| Dpi 43-49 | 2.63 | 2.61 | | | | |
| *Gain:Feed* | | | | | | |
| Dpi 0-7 | 0.33 | 0.34 | 0.029 | 0.764 | <0.001 | 0.872 |
| Dpi 8-14 | 0.15 | 0.15 | | | | |
| Dpi 15-21 | 0.25 | 0.20 | | | | |
| Dpi 22-28 | 0.18 | 0.20 | | | | |
| Dpi 29-35 | 0.31 | 0.34 | | | | |
| Dpi 36-42 | 0.37 | 0.37 | | | | |
| Dpi 43-49 | 0.26 | 0.27 | | | | |

n=9 pens/trt

**Table 4.** Effects of dietary SBM, PRRSV and MHP infection on carcass characteristics.

| **Parameter** | **HSBM** | **LSBM** | **SEM** | ***P-value*** |
|---|---|---|---|---|
| *Experiment 1*[1] | | | | |
| Live weight, kg | 138.38 | 137.00 | 2.14 | 0.656 |
| HCW, kg | 104.45 | 104.01 | 1.65 | 0.854 |
| Yield, % | 75.49 | 75.91 | 0.25 | 0.245 |
| Lean, % | 57.55 | 57.11 | 0.23 | 0.199 |
| Back fat depth, cm | 1.24 | 1.44 | 0.07 | 0.078 |
| Loin depth, cm | 7.39 | 7.44 | 0.10 | 0.739 |
| Lung Lesions, % | 11.45 | 15.89 | 2.20 | 0.180 |
| | | | | |
| *Experiment 2*[2] | | | | |
| Live weight, kg | 119.82 | 121.84 | 2.27 | 0.537 |
| HCW, kg | 88.53 | 90.73 | 1.84 | 0.401 |
| Yield, % | 73.88 | 74.44 | 0.32 | 0.233 |
| Lean, % | 56.01 | 56.21 | 0.16 | 0.396 |
| Back fat depth, cm | 1.54 | 1.60 | 0.05 | 0.355 |
| Loin depth, cm | 6.89 | 7.12 | 0.09 | 0.087 |

[1]n=8 pens/trt

[2]n=9 pens/trt