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## EC01-219 2001 Nebraska Swine Report

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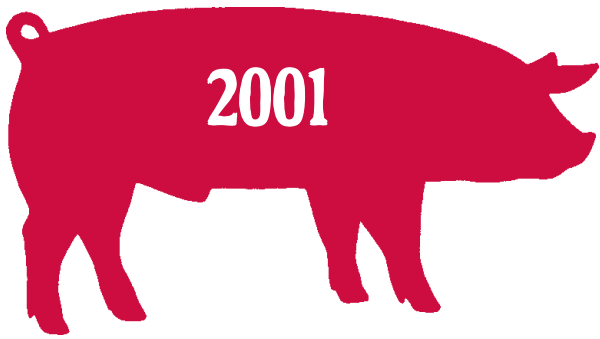
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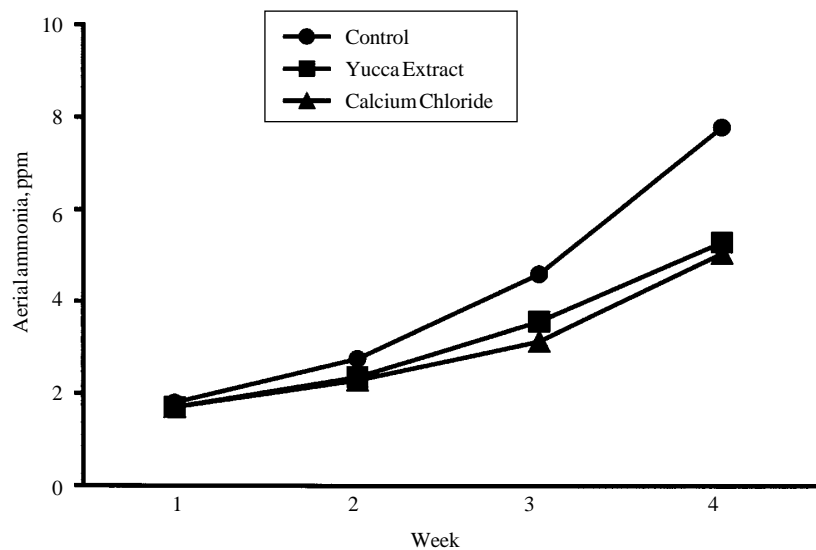
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# NEBRASKA SWINE REPORT

- Nutrition
- Genetics
- Legal Issues



Web site:

[www.ianr.unl.edu/pubs/swine/pigpdf.htm](http://www.ianr.unl.edu/pubs/swine/pigpdf.htm)

Prepared by the staff in Animal Science and cooperating Departments for use in Extension, Teaching and Research programs.

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### Nebraska Swine Report Acknowledgments for 2001

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### Cover Illustration:

Aerial ammonia concentrations measured by ammonia aspiration tubes (SEM=.54) in rooms with pigs fed a control, *Yucca schidigera* extract (De-Odorase), or calcium chloride diet (major study).

### From:

“Dietary Manipulation to Reduce Ammonia Concentration in Nursery Pig Facilities,” page 7.

*The 2001 Nebraska Swine Report was compiled by Duane Reese, Associate Professor, Department of Animal Science.*

### 2001 Nebraska Swine Report

**Editor:** Marcia Oetjen  
**Typesetting & Design:** Anne Moore



This edition of the University of Nebraska-Lincoln's annual Swine Report is dedicated to Dr. D. Murray Danielson who passed away in 2000. Dr. Danielson was a distinguished member of the Animal Science Department for 33 years.

During his career, Dr. Danielson served as a scientific advisor for the American Grain Sorghum Association, the U.S. Feeds Grain Council in China and the American Soybean Association in Mexico, Guatemala, Honduras, Costa Rica and Panama, as well as a consultant on Swine Nutrition in Belgium, Netherlands, Austria, France, Switzerland, England, Scotland, Spain, West Germany, South Korea and Brazil. He authored more than 200 technical publications and was a regular contributor to the Nebraska Swine Report.

Dr. Danielson's commitment to research and teaching was recognized with the University of Nebraska Livestock Service Award and the American Society of Animal Science Animal Management award.

In 1991, Dr. Danielson retired from the University of Nebraska. He is fondly remembered by his colleagues and former students.



# Phytase in Swine Diets

Michael C. Brumm<sup>1</sup>

## Summary and Implications

*An experiment was conducted to determine whether phytase additions to swine growing-finishing diets improved the availability of lysine, calcium, energy and phosphorus in corn and soybean meal-based diets. Diets investigated for growing-finishing barrows of high-lean-gain potential included: 1) University of Nebraska recommended diet formulations; 2) diets formulated to contain 85% of the lysine recommended; 3) lysine-deficient diets formulated with phytase; 4) phytase-formulated diets with phytase deleted; and 5) phytase-formulated diets with phytase deleted and with additional calcium and phosphorus. Pigs fed diets formulated to 85% of the recommended lysine level had slower growth, slower daily lean gain, poorer feed conversion, and less carcass lean. There was no effect of phytase addition or deletion on growth, feed efficiency, or carcass lean. There was no effect of phytase addition or deletion on bone strength or bone ash. These results do not agree with the large body of evidence regarding the improvement in availability of phosphorus in corn and soybean meal with the addition of phytase to swine diets and may be related to the growth impairment associated with the lysine deficiency.*

## Introduction

Phosphorus is a key mineral required by pigs for growth of body tissues. Feeding diets deficient in phosphorus results in reduced growth, reduced lean tissue accretion and reduced bone development.

In corn and soybeans, the major feed grain and protein supplement in

swine diets in Nebraska, the majority of the phosphorus occurs as an organic complex called phytate. Because pigs secrete very limited amounts of the enzyme phytase, they are unable to use most of the phosphorus in these feed grains. For example, while corn and 44% protein soybean meal contain .28% and .60% total phosphorus on an as-fed basis, only .04% and .20%, respectively, are available. Thus, swine diets are typically formulated using inorganic phosphorus sources such as dicalcium phosphate to meet the pigs' requirements. In addition to being relatively unavailable, there is evidence that phytate binds some of the calcium and other minerals in swine diets and reduces their availability.

It is now possible to add the enzyme phytase to swine diets. The result is an increase in the availability of phytate-phosphorus and a resulting decrease in the amount of inorganic phosphorus addition necessary to meet the needs of the pig and a decrease in the amount of undigestible phosphorus excreted in the manure. Experimental evidence from poultry and swine suggests that in addition to increasing phytate phosphorus availability, phytase additions to the diet also increase the calcium, lysine and energy digestibility of feed grains. The purpose of the following experiment was to evaluate the possibility that the use of a commercial phytase source in corn-soybean meal diets would result in increased calcium, lysine and energy availability, in addition to improved phosphorus utilization.

## Methods

The experiment was conducted at the University of Nebraska's Haskell Ag Lab at Concord. Pigs were housed in partially slatted pens measuring 6 ft x 15 ft with 13 pigs per pen

(6.9 ft<sup>2</sup>/pig). In Trial 1, which began in March, the facilities were mechanically ventilated. Pigs in Trial 2 (began in May) and Trial 3 (began in November) were housed in naturally ventilated facilities. Sprinklers were used for summer heat relief with sprinkling set to begin at 80°F.

In each of three trials, 260 cross-bred PIC barrows were allocated at arrival based on weight outcome groups (light and heavy) to experimental dietary treatments (Tables 1 and 2). The experimental treatments were:

- A) University of Nebraska recommended diets with added fat (**UNL**).
- B) Lysine at 85% of UNL. All other nutrients at same level of addition (**85**).
- C) Lysine at 84% of UNL with phytase added based on credits for lysine, energy, Ca and P per phytase recommended equivalencies (**PHY**).
- D) PHY without phytase (**NEG**).
- E) NEG with P and Ca added to same level as UNL and 85 (**MIN**).

The UNL diets were formulated to contain 1.00%, 0.88%, 0.73% and 0.60% lysine with diets switched on the week individual pens of pigs weighed 80, 130 and 190 pounds. Using corn and soybean meal, UNL diets were formulated for lysine, calcium, total phosphorus and available phosphorus according to the 1995 edition of the *Nebraska and South Dakota Swine Nutrition Guide* (EC95-273). Fat was added to treatments UNL and 85 to increase the metabolizable energy content by the amount credited to phytase in treatment PHY. Phytase diets were formulated using the nutrient availability matrix of the phytase supplier. When added at 500 FTU (phytase units)/kg, the matrix estimated that phytase



**Table 1. Experimental diet composition, 40 to 130 pound pigs.**

Item	40 to 80 lb					80 to 130 lb				
	Treatment <sup>a</sup>					Treatment				
	UNL	85	PHY	NEG	MIN	UNL	85	PHY	NEG	MIN
<b>Ingredient, lb/ton</b>										
Com	1352	1468	1503.3	1505	1494	1448	1551	1581.3	1583	1572
Soybean meal, 44% CP	580	465	455	455	455	486	385	380	380	380
Fat	19	16				19	16			
Dicalcium phosphate, 18.5% P	26	28	15	15	28	23	24	11	11	24
Limestone	13	13	15	15	13	14	14	16	16	14
Salt	6	6	6	6	6	6	6	6	6	6
Vit/TMmix <sup>b</sup>	4	4	4	4	4	4	4	4	4	4
Phytase <sup>c</sup>			1.7					1.7		
<b>Composition</b>										
ME, kcal/lb	1508 <sup>d</sup>	1509	1497	1500	1491	1514	1514	1502	1505	1497
Lysine, %	1.00(.97 <sup>e</sup> )	.85(.88)	.84(.83)	.84(.83)	.84(.80)	.88(.87)	.75(.76)	.74(.74)	.74(.73)	.74(.75)
Calcium, %	.69(.80)	.70(.70)	.58(.63)	.58(.61)	.70(.64)	.66(.74)	.66(.71)	.54(.58)	.54(.52)	.66(.65)
Phosphorus, %	.60(.58)	.60(.55)	.48(.44)	.48(.47)	.59(.58)	.55(.57)	.54(.55)	.43(.44)	.43(.40)	.54(.52)
Total available phosphorus, %	.34	.34	.34	.22	.34	.30	.30	.30	.18	.30
Particle size, microns <sup>e</sup>						818	846	851	852	842
Phytase activity, FTU/kg <sup>c</sup>			467					445		

<sup>a</sup>UNL = University of Nebraska recommended; 85 = Lysine at 85% of UNL; PHY = Phytase to 85 diet with credits for lysine, energy, calcium, and phosphorus; NEG = PHY diet with phytase deleted; MIN = NEG diet with calcium and phosphorus added.

<sup>b</sup>Provided the following per pound of complete diet: Zn, 90 ppm; Fe, 80 ppm; Mn, 32 ppm; Cu, 10 ppm; I, 0.4 ppm; Se, 0.3 ppm; Vitamin A, 2500 IU; Vitamin D 500 IU; Vitamin E, 11 IU; Vitamin K, 1 mg; Choline, 30 mg; Niacin, 12 mg; D-pantothenic acid, 8 mg; Riboflavin, 2 mg; Vitamin B<sub>12</sub>, .024 mg.

<sup>c</sup>Natuphos 600, BASF, Inc., Mt. Olive, NJ 07828.

<sup>d</sup>Calculated composition.

<sup>e</sup>Analyzed composition, Ward Labs, Kearney, NE 68848.

**Table 2. Experimental diet composition, 130 pounds to slaughter.**

Item	130 to 190 pounds					190 pounds to slaughter				
	Treatment <sup>a</sup>					Treatment				
	UNL	85	PHY	NEG	MIN	UNL	85	PHY	NEG	MIN
<b>Ingredient, lb/ton</b>										
Com	1569	1651	1690.3	1692	1681	1672	1743	1778.3	1780	1769
Soybean meal, 44% CP	370	290	275	275	275	270	200	190	190	190
Fat	18	15				18	16			
Dicalcium phosphate, 18.5% P	19	20	7	7	20	16	16	3	3	16
Limestone	14	14	16	16	14	14	15	17	17	15
Salt	6	6	6	6	6	6	6	6	6	6
Vit/TMmix <sup>b</sup>	4	4	4	4	4	4	4	4	4	4
Phytase <sup>c</sup>			1.7					1.7		
<b>Composition</b>										
ME, kcal/lb	1521 <sup>d</sup>	1521	1510	1513	1505	1528	1528	1516	1519	1511
Lysine, %	.73(.71 <sup>e</sup> )	.62(.62)	.61(.61)	.61(.60)	.61(.62)	.60(.58)	.51(.51)	.50(.50)	.50(.51)	.50(.50)
Calcium, %	.60(.89)	.60(.82)	.48(.44)	.48(.47)	.59(.62)	.55(.55)	.56(.64)	.44(.42)	.44(.53)	.55(.65)
Phosphorus, %	.49(.50)	.49(.52)	.37(.36)	.37(.36)	.49(.48)	.45(.42)	.44(.47)	.32(.31)	.32(.33)	.44(.42)
Available phosphorus, %	.25	.25	.25	.13	.25	.21	.21	.21	.09	.21
Particle size, microns <sup>e</sup>	792	828	812	832	823	882	851	827	843	859
Phytase activity, FTU/kg <sup>c</sup>			495					533		

<sup>a</sup>UNL = University of Nebraska recommended; 85 = Lysine at 85% of UNL; PHY = Phytase to 85 diet with credits for lysine, energy, calcium, and phosphorus; NEG = PHY diet with phytase deleted; MIN = NEG diet with calcium and phosphorus added.

<sup>b</sup>Provided the following per pound of complete diet: Zn, 90 ppm; Fe, 80 ppm; Mn, 32 ppm; Cu, 10 ppm; I, 0.4 ppm; Se, 0.3 ppm; Vitamin A, 2500 IU; Vitamin D 500 IU; Vitamin E, 11 IU; Vitamin K, 1 mg; Choline, 30 mg; Niacin, 12 mg; D-pantothenic acid, 8 mg; Riboflavin, 2 mg; Vitamin B<sub>12</sub>, .024 mg.

<sup>c</sup>Natuphos 600, BASF, Inc., Mt. Olive, NJ 07828.

<sup>d</sup>Calculated composition.

<sup>e</sup>Analyzed composition, Ward Labs, Kearney 68848.

supplied .12% calcium and available phosphorus, .01% lysine and 12 kcal/lb metabolizable energy.

Pigs were vaccinated at arrival and revaccinated two weeks later for H

parasuis, M hypopneumonia and Erysipelas. All pigs that died during the experiment were examined by a consulting veterinarian for cause of death. Pen sizes were not adjusted in the

event of pig death or removal.

Individually identified pigs were slaughtered at SiouxPreme Packing Co. at Sioux Center, IA, on the week

(Continued on next page)



they weighed 240 pounds or more. Carcass lean was estimated by TOBEC (total body electrical conductivity) at the slaughter house. Daily lean gain was calculated based on the individual carcass lean estimate and the formulas of the National Pork Producers Council.

To further clarify the response of the pigs to the experimental diets, one front foot from two pigs from each pen of pigs (10 pigs/diet) in Trial 2 was collected at slaughter and frozen for later analysis. The feet were sent to Dr. Merlin Lindemann at the University of Kentucky for metacarpal bone breaking strength and ash content.

The pen of pigs was the experimental unit. The model included treatment, trial, weight block and all two- and three-way interactions. Means were separated based on preplanned contrasts. The contrasts chosen were:

1. UNL vs 85. This examined whether the 85 diet was deficient in lysine.
2. 85 vs PHY. This examined whether diets containing phytase and formulated according to the phytase suppliers recommendation gave equivalent performance to diets formulated without phytase.
3. PHY vs NEG. This examined whether diets containing phytase improved performance compared to diets without phytase but formulated for the phytase matrix.
4. PHY vs MIN. This examined whether diets containing phytase improved performance over diets that had the same level of total P and Ca, but were lower in energy and lysine by the amount credited to phytase by the supplier.

## Results and Discussion

Tables 1 and 2 include the laboratory analysis of the pooled diet samples. As documented in the tables, the diets as sampled contained the formulated amounts of calcium, phosphorus, and lysine. While the diets were formu-

**Table 3. Main effects of experimental treatments on pig performance.**

Item	Treatment <sup>a</sup>					SE	P-value			
	UNL	85	PHY	NEG	MIN		Contrasts			
	1	2	3	4	5		1v2	2v3	3v4	3v5
Number pens	12	12	12	12	12					
<b>Pig wt., lb</b>										
Initial	56.1	55.6	56.3	55.7	56.3	.2				
First mkt (M) <sup>b</sup>	220.8	215.3	208.0	207.3	211.6	2.4	.0005	.043	NS	NS
Final (F)	247.0	241.8	244.1	239.8	237.9	2.1	.025	NS	NS	.0511
CV, % <sup>c</sup>	8.0	9.9	10.7	9.5	8.7	.8	NS	NS	NS	.0706
<b>Daily gain, lb/d</b>										
0 to M	1.72	1.60	1.56	1.60	1.60	.02	.0001	NS	NS	NS
0 to F	1.75	1.63	1.60	1.63	1.64	.02	.0001	NS	NS	NS
<b>Daily feed, lb/d</b>										
0 to M	4.87	4.77	4.74	4.72	4.83	.05	.049	NS	NS	NS
0 to F	5.13	5.08	5.23	5.14	5.26	.05	NS	.045	NS	NS
<b>Feed/gain</b>										
0 to M	2.84	2.98	3.04	2.95	3.04	.03	.0051	NS	.0418	NS
0 to F	2.94	3.13	3.27	3.16	3.22	.03	.0001	.0007	.0092	NS
Carcass % lean <sup>d</sup>	52.0	50.4	50.3	50.6	50.6	.3	.0021	NS	NS	NS
Daily lean gain, lb/d <sup>d</sup>	.67	.60	.59	.60	.60	.01	.0001	.137	NS	.137
No. dead/removed	4	10	6	2	7					

<sup>a</sup>UNL = University of Nebraska recommended; 85 = Lysine at 85% of UNL; PHY = Phytase to 85 diet with credits for lysine, energy, calcium, and phosphorus; NEG = PHY diet with phytase deleted; MIN = NEG diet with calcium and phosphorus added.

<sup>b</sup>Average pen weight when first pig removed for slaughter.

<sup>c</sup>Coefficient of variation of within pen weight when first pig removed for slaughter.

<sup>d</sup>Containing 5% fat.

**Table 4. Metacarpal bone strength and ash (least squares means) - Trial 2.**

Item	Treatment <sup>a</sup>					SE
	UNL	85	PHY	NEG	MIN	
Bone strength, kg/cm <sup>2</sup>	199	208	204	195	197	7
Bone ash, %	58.3	57.8	56.7	57.7	57.8	.5

<sup>a</sup>UNL = University of Nebraska recommended; 85 = Lysine at 85% of UNL; PHY = Phytase to 85 diet with credits for lysine, energy, calcium, and phosphorus; NEG = PHY diet with phytase deleted; MIN = NEG diet with calcium and phosphorus added.

lated to contain 500 FTU/kg phytase, they ranged from 445 to 533 FTU.

The main effects of the experimental diets on pig performance are given in Table 3. Decreasing the lysine to 85% of the UNL recommended level while maintaining energy, calcium and phosphorus at similar levels (85 vs UNL) resulted in a decrease in daily gain (1.63 vs 1.75 lb/d; P<.0001), a worsening in feed:gain (3.13 vs 2.94; P<.0001), carcasses with a lower lean percentage (50.4 vs 52.0%; P<.0021), and a decrease in the rate of daily lean gain (.60 vs .67; P<.0001).

There was no effect of experimental diet when comparing 85 vs PHY, PHY vs NEG, and PHY vs MIN for overall average daily gain. A trial ×

treatment effect was observed for daily gain and feed conversion, demonstrating the treatment variation among the three trials. However, in all trials, the performance of UNL pigs was the best.

Pigs fed the PHY treatment ate more feed than 85 pigs (5.23 vs 5.08 lb/d; P<.045) with no difference in feed intake for the PHY vs NEG or MIN treatments.

As a consequence of the difference in feed intake with no difference in daily gain, the PHY pigs had a worse feed conversion when compared to the 85 pigs (3.27 vs 3.13; P<.0007). Feed conversion for the PHY treatment was also worse than for NEG (3.27 vs 3.16; P<.0092), and not different from MIN.

There was no difference between



PHY and 85, NEG or MIN for carcass lean percentage or daily lean gain. There was no effect of experimental treatments on the number of pigs that died or were removed for poor performance during the experiment. Overall death loss and removal was 3.7%.

In this experiment, all treatments except UNL were designed to have lysine as a growth limiting nutrient. The claim for a .01% increase in lysine availability due to phytase addition was not supported as evidenced by the lack of improvement in performance for the MIN vs PHY treatments or the 85 vs PHY treatments.

Furthermore, the lysine limitation in the PHY treatment appears to have

been severe enough to prevent any response of phytase in improving calcium and phosphorus availability. The lack of a response to the experimental diets on bone breaking strength and bone ash (Table 4), both considered sensitive indicators of phosphorus availability, supports this conclusion. The fact that MIN was not superior to NEG for any of the traits reported further supports the conclusion that the lysine limitation was severe enough to limit the possible phytase response.

### Conclusion

These results document the impact of inadequate lysine on growth

and carcass lean. They do not agree with the large body of evidence regarding the improvement in corn and soybean meal phosphorus availability with the addition of phytase to swine diets. They do suggest a limited, if any, response to phytase additions in diets in which lysine is limiting performance. The results were also unable to document the improvement in calcium, energy and lysine availability previously reported in poultry experiments from the addition of phytase to corn and soybean meal based diets.

<sup>1</sup>Michael C. Brumm is Professor and Extension Swine Specialist at the Northeast Research and Extension Center, Concord, Nebraska.

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## Dietary Manipulation To Reduce Ammonia Concentration in Nursery Pig Facilities

Janeth J. Colina  
Austin J. Lewis  
Phillip S. Miller  
Robert L. Fischer<sup>1</sup>

### Summary and Implications

Five trials were conducted to determine the effects of *Yucca schidigera* extract or calcium chloride addition to the diet on aerial ammonia concentration and growth performance in nursery pigs. Trials were divided into two groups: preliminary studies (two trials) and major study (three trials). Pigs were fed one of three diets in separate, environmentally regulated rooms: 1) Control, containing 23% CP; 2) Control diet plus 125 ppm of *Yucca schidigera* extract; and 3) Control plus 1.95% calcium chloride. Average daily gain (ADG), average daily feed intake (ADFI), and ADG/ADFI were recorded weekly. Aerial ammonia concentration was measured daily using aspiration detector tubes and during the last week of each trial using diffusion tubes. Blood samples were collected at the end of each trial to determine plasma

urea concentration. There were no differences in ADG, ADFI, and ADG/ADFI between pigs fed the control diet and pigs fed the *Yucca schidigera* diet. In all trials, pigs fed the calcium chloride diet had lower ADG and ADG/ADFI than pigs fed the other two diets ( $P < .05$ ). In the preliminary studies (Trials 1 and 2), aerial ammonia concentration tended to be greater in the rooms in which pigs were fed the control diet than in the rooms with pigs fed the yucca extract diet ( $P < .08$ ) or calcium chloride diet ( $P < .11$ ). In the major study (Trials 3, 4, and 5), aerial ammonia concentration increased as the experiment progressed ( $P < .001$  in all rooms. In the fourth week, ammonia concentrations were greater ( $P < .001$ ) in the rooms that housed pigs fed the control diet than in the rooms in which the other two diets were fed. Dietary treatment did not affect plasma urea concentration ( $P > .10$ ). This research has shown that ammonia concentration in nursery pig facilities can be reduced by dietary manipulation such as the addition of *Yucca schidigera* extract or calcium salts.

### Introduction

Ammonia is one of the gases of most concern in swine buildings and is a major source of indoor air contamination. The large variation in aerial ammonia concentration is influenced by the bacterial activity and the presence of ideal fermentation conditions. Ammonia volatilization is a process that depends on factors such as concentration of aerial ammonia, air speed in the building, ammonia and dry matter content in the manure, pH of manure, and slurry temperature.

In addition to objectional odors, there also is concern about the health problems that ammonia exposure may produce in animals and animal caretakers. We reported a review of the ammonia issue and pork production in the Nebraska Swine Report (1999) and identified a clear need to continue to evaluate methods to reduce and control odor from livestock enterprises, especially pork production units. These methods include reducing of ammonia concentration using certain additives in growing pig diets such as *Yucca*

(Continued on next page)





*schidigera* extract or calcium salts. These additives have been successful in reducing ammonia concentrations in growing pig facilities by 26 and 33%, respectively. By adding calcium salts such as calcium chloride to the diet, the dietary electrolyte balance can be reduced. Dietary mineral balance may be expressed as Na+K-Cl (hereafter referred to as electrolyte balance) which considers the monovalent mineral ions in the diet. Diets with high levels of protein and amino acids that are typical for nursery-age pigs can increase the amount of nitrogen excreted in urine and feces, and this can increase ammonia production. Based on these observations, we designed a study to determine the effect of *Yucca schidigera* extract and calcium chloride addition to nursery diets on aerial ammonia concentration.

### Procedures

This research consisted of five trials: two preliminary four-week trials (preliminary study) and three, five-week trials (major study). The objective of the preliminary trials was to establish the ammonia concentration in our environmentally regulated nursery pig facilities. Information about each trial is presented in Table 1. In all trials, pigs were blocked on initial weight and allotted to one of three environmentally controlled rooms such that the average initial weight within each room was similar. In the major study, the three trials, three rooms, and three dietary treatments constituted a Latin square design such that each treatment was assigned in turn to each room.

Pigs were weighed and feed disappearance was recorded weekly to determine average daily gain (ADG), average daily feed intake (ADFI), and feed efficiency (ADG/ADFI). During the first week of each trial, all pigs were fed the same standard commercial prestarter diet (23.5% CP, 1.75% lysine, .75% Ca, and .70% P, with apramycin [as apramycin sulfate, 150 g/T]), to allow them to adapt to the

**Table 1. Details of the five trials conducted to determine the effects of the addition of *Yucca schidigera* extract or calcium chloride to nursery diets.**

Trial	Purpose	Duration wk <sup>a</sup>	Pigs per pen (barrows, gilts) <sup>b</sup>	Weaning age, d	Weaning wt, lb
1	Preliminary	1 + 3	6,0	15	11.4
2	Preliminary	1 + 3	6,4	13	6.6
3	Major	1 + 4	7,3	13	7.7
4	Major	1 + 4	6,4	15	13.0
5	Major	1 + 4	5,5	14	10.5

<sup>a</sup>Each trial consisted of a 1-wk initial period followed by a 3- or 4-wk experimental period.

<sup>b</sup>Each room with five pens, 6.7 ft<sup>2</sup>/pig (Trial 1) and 4.03 ft<sup>2</sup>/pig (Trials 2, 3, 4, and 5).

**Table 2. Composition and analysis of diets<sup>a</sup>.**

Item	Control	<i>Yucca schidigera</i>	Calcium chloride
<b>Ingredient, %</b>			
Corn	30.33	30.32	28.38
Soybean meal (46.5% CP)	20.50	20.50	20.50
Dried whey	30.00	30.00	30.00
Spray-dried plasma protein	6.00	6.00	6.00
Menhaden fishmeal	5.00	5.00	5.00
Corn oil	5.00	5.00	5.00
Dicalcium phosphate	1.55	1.55	1.55
Vitamin premix <sup>b</sup>	1.00	1.00	1.00
Trace mineral premix <sup>c</sup>	.20	.20	.20
Zinc oxide (72% zinc)	.42	.42	.42
<i>Yucca schidigera</i> extract <sup>d</sup>	—	.013	—
Calcium chloride <sup>e</sup>	—	—	1.95
<b>Nutrient composition</b>			
Crude protein <sup>f</sup> , %	23.20	23.30	22.90
Lysine <sup>g</sup> , %	1.58	1.58	1.58
Ca <sup>f</sup> , %	1.13	1.12	1.76
P <sup>f</sup> , %	.91	.98	.97
Na + K - Cl, mEq/kg <sup>h</sup>	345	345	-7

<sup>a</sup>As-fed basis.

<sup>b</sup>Supplied per kilogram of diet: retinyl acetate, 4,409 IU; cholecalciferol, 551 IU; all-rac- $\alpha$ -tocopheryl acetate, 22 IU; menadione (as menadione sodium bisulfite complex), 3.3 mg; riboflavin, 5.6 mg; d-pantothenic acid (as d-calcium pantothenate), 22 mg; niacin, 33 mg; choline (as choline chloride), 110 mg; cyanocobalamin, 22  $\mu$ g.

<sup>c</sup>Supplied (mg/kg of diet): Cu (as CuSO<sub>4</sub>•5H<sub>2</sub>O), 22; I (as Ca(IO<sub>3</sub>)<sub>2</sub>), 44;

Fe (as FeSO<sub>4</sub>•H<sub>2</sub>O), 220; Mn (as MnO), 44; Se (as Na<sub>2</sub>SeO<sub>3</sub>), 0.6; Zn (as ZnO), 220.

<sup>d</sup>*Yucca schidigera* extract (De-Odorase®, Alltech, Nicholasville, KY) added at the rate of 125 ppm (4 oz/ton).

<sup>e</sup>Calcium chloride (36.1% Ca; 63.9% Cl).

<sup>f</sup>Analyzed composition.

<sup>g</sup>Calculated composition.

<sup>h</sup>dEB (dietary electrolyte balance) = Na + K - Cl.

initial stress of weaning. For the next three weeks (Trials 1 and 2) and four weeks (Trials 3 to 5), three experimental diets were fed. All diets were formulated to meet or exceed the nutrient requirements of nursery pigs (NRC, 1998). Diets (Table 2) used were 1) Control, 2) Control plus 125 ppm of *Yucca schidigera* extract (De-Odorase, Alltech, Nicholasville, KY), and 3) Control plus 1.95% calcium chloride.

There were three identical pig nursery rooms. Each room had five pens and all pens had plastic-coated wire flooring; one nipple waterer; and one, three-hole, stainless steel feeder. Pigs had *ad libitum* access to feed and water throughout the trial. Heat lamps and comfort boards were provided during the first week after weaning.

In each room, relative humidity (maintained at 60%) and temperature



**Table 3. Growth performance, plasma urea concentration and ammonia concentration measured by diffusion tubes of pigs fed either a control diet, a diet with *Yucca schidigera*<sup>a</sup> extract, or calcium chloride diet<sup>b</sup> during the preliminary studies (Trials 1<sup>c</sup> and 2<sup>d</sup>).**

Item	Control	<i>Yucca schidigera</i>	Calcium chloride	SEM <sup>e</sup>	P-Value <sup>f</sup>
ADG, lb	.765 <sup>g</sup>	.765 <sup>g</sup>	.565 <sup>h</sup>	.019	.027
ADFI, lb	1.111	1.157	1.031	.043	.320
ADG/ADFI	1.511 <sup>g</sup>	1.452 <sup>g</sup>	1.166 <sup>h</sup>	.040	.045
Urea, mg/100 mL	35.68	35.59	29.35	1.200	.100
Aerial ammonia, ppm	8.00	5.60	7.30	2.028	.711

<sup>a</sup>*Yucca schidigera* extract (De-Odorase®, Alltech, Nicholasville, KY) added at the rate of 125 ppm.

<sup>b</sup>1.95% calcium chloride added.

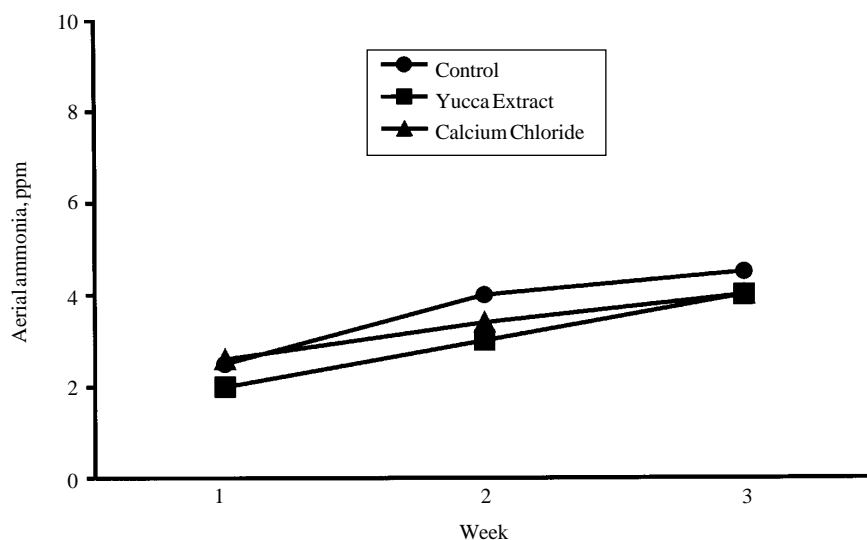
<sup>c</sup>Each room with five pens with six barrows per pen. Initial weight 11.4 lb; final weight 31.2 lb. 28-d experiment; 21-d experimental diets.

<sup>d</sup>Each room with five pens with ten pigs (six barrows and four gilts) per pen. Initial weight 6.6 lb; final weight 20.3 lb. 28-d experiment; 21-d experimental diets.

<sup>e</sup>Standard error of means.

<sup>f</sup>Significance of main effect.

<sup>g,h</sup>Within a row, means without a common superscript letter differ ( $P < .05$ ).



**Figure 1. Aerial ammonia concentrations measured by ammonia aspiration tubes (SEM=.16) in rooms with pigs fed a control, *Yucca schidigera* extract (De-Odorase), or calcium chloride diet (preliminary studies).**

(maintained at 77°F) were monitored continuously using temperature and humidity recorders. Airflow in the exhaust duct from each room (maintained at 920 ft<sup>3</sup>/min) was measured two times per day (8:00 am and 2:00 pm) using an air velocity meter. Aerial ammonia concentration was measured every day at 8:00 am by using Sensidyne aspiration tubes. Air was sampled in the center of the left and right sides of

the rooms approximately 3.3 ft above floor level. Ammonia was also measured using 8-hour Dräger diffusion tubes distributed throughout each room at approximately 1.6 ft above the floor three times during the last week of each trial. Five tubes were used per room to measure differences in ammonia concentration in different places in the rooms.

Feces and urine that collected

below the pens were allowed to accumulate throughout the trials. Manure (feces, urine, spilled feed and spilled drinking water) that drained from the pens was maintained in a collection pit located at the end of each room. These pits were emptied weekly because their capacity was limited. Water was not added when pits were emptied. Blood was collected from the jugular vein of each pig at the end of the trial to determine plasma urea concentration.

## Results

**Preliminary Studies:** There were no differences in ADG, ADFI, and ADG/ADFI of pigs fed the control diet and the diet containing *Yucca schidigera* extract diet (Table 3). However, there was a reduction in ADG and ADG/ADFI ( $P < .05$ ) of pigs fed the calcium chloride diet compared with pigs fed the other two diets. There were no differences in ADFI among pigs fed the three diets. There were no differences in plasma urea concentration among treatments (Table 3).

There was no effect of diet on aerial ammonia measured by either Sensidyne aspiration tubes (Figure 1) or Dräger diffusion tubes (Table 3). However, a tendency for increased aerial ammonia was observed in rooms with pigs fed the control diet compared with rooms in which pigs were fed the *Yucca schidigera* extract diet ( $P = .08$ ) and the calcium chloride diet ( $P = .10$ ). Aerial ammonia concentration increased as the experiment progressed ( $P < .001$ ) in all rooms.

**Major Study:** There were no differences in ADG, ADFI, and ADG/ADFI of pigs fed the control diet and the pigs fed the diet containing *Yucca schidigera* extract (Table 4). There were no differences in ADFI among pigs fed any of the three diets. There was a significant reduction in ADG and ADG/ADFI ( $P < .05$ ) of pigs fed the calcium chloride diet compared with pigs fed the other two diets. There were no differences in plasma urea concentration among treatments.

(Continued on next page)



There was no effect of diet on aerial ammonia measured by Dräger diffusion tubes during the final week of the experiment (Table 4). The aerial ammonia concentrations measured by Sensidyne aspiration tubes increased weekly ( $P < .001$ ) in all treatments (Figure 2). There was a diet x week ( $P < .001$ ) interaction, with a greater increase of aerial ammonia in rooms with pigs fed the control diet compared with the other rooms. Differences among treatments were evident during the third week ( $P = .07$ ) and clear during the fourth week ( $P < .001$ ), when ammonia concentration was 2.5 and 2.7 ppm higher in rooms with pigs fed the control diet compared with the concentration in rooms with pigs fed the yucca extract and the calcium chloride diets, respectively (Figure 2).

### Discussion

In this experiment, there was no effect of yucca extract additive on growth performance in nursery pigs. Lowering the dietary electrolyte balance with calcium chloride resulted in reduced ADG and ADG/ADFI compared with pigs fed the control or yucca extract diets. This was probably due to a metabolic acidosis produced by the increased plasma chloride, which is the mechanism by which calcium chloride generally decreases feed intake and weight gain. In this research, feed intake was reduced by 7% to 10% in pigs fed the calcium chloride versus the control diet in the preliminary studies, but this difference was not statistically significant.

Ammonia concentrations were relatively low, probably because of the small size of the pigs and the high ventilation rate in our research facility. The lower weight gain in pigs fed the calcium chloride diet may have been at least partly responsible for the decreased ammonia concentration in rooms in which pigs were fed this treatment.

Ammonia concentration measured by diffusion tubes was not different among the three rooms. However, lower

**Table 4. Growth performance, plasma urea concentration and ammonia concentration measured by diffusion tubes of pigs fed either a control diet, a diet with *Yucca schidigera*<sup>a</sup> extract, or calcium chloride diet<sup>b</sup> during the major study (Trials 3<sup>c</sup>, 4<sup>d</sup>, and 5<sup>e</sup>).**

Item	Control	<i>Yucca schidigera</i>	Calcium chloride	SEM <sup>f</sup>	P-Value <sup>g</sup>
ADG, lb	.968 <sup>h</sup>	.965 <sup>h</sup>	.732 <sup>i</sup>	.023	.028
ADFI, lb	1.364	1.339	1.229	.073	.456
ADG/ADFI	1.579 <sup>h</sup>	1.621 <sup>h</sup>	1.291 <sup>i</sup>	.041	.050
Urea, mg/100 mL	29.29	27.73	26.48	1.170	.400
Aerial ammonia, ppm	8.92	8.28	5.75	1.220	.348

<sup>a</sup>*Yucca schidigera* extract (De-Odorase®, Alltech, Nicholasville, KY) added at the rate of 125 ppm (4 oz/ton).

<sup>b</sup>1.95% calcium chloride added.

<sup>c</sup>Each room with five pens with ten pigs (seven barrows and three gilts) per pen. Initial weight 7.7 lb; final weight 33.44 lb, 35-d experiment; 28-d experimental diets.

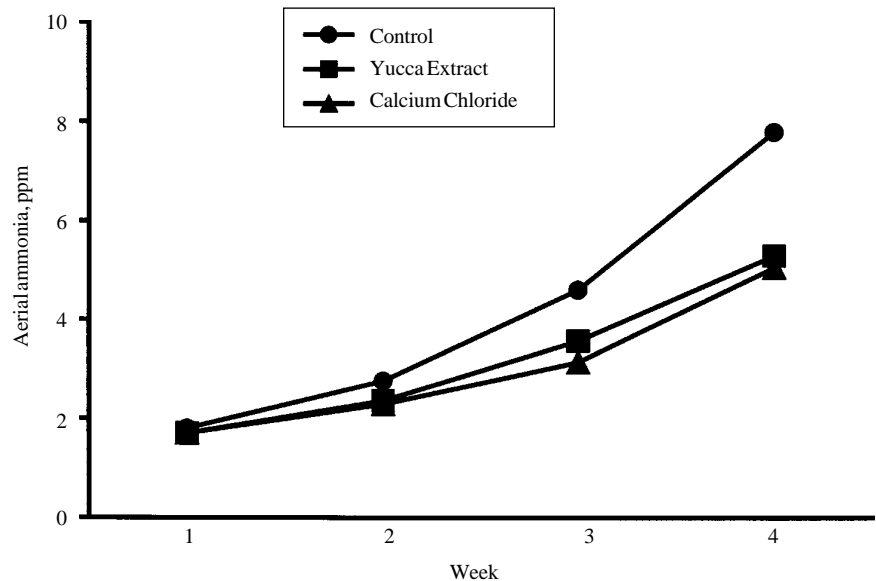
<sup>d</sup>Each room with five pens with ten pigs (six barrows and four gilts) per pen. Initial weight 13.0 lb; final weight 41.5 lb; 35-d experiment; 28-d experimental diets.

<sup>e</sup>Each room with five pens with ten pigs (five barrows and five gilts) per pen. Initial weight 10.5 lb; final weight 38.4 lb; 35-d experiment; 28-d experimental diets.

<sup>f</sup>Standard error of means.

<sup>g</sup>Significance of main effects.

<sup>h,i</sup>Within a row, means without a common superscript letter differ ( $P < 0.05$ ).



**Figure 2. Aerial ammonia concentrations measured by ammonia aspiration tubes (SEM=.54) in rooms with pigs fed a control, *Yucca schidigera* extract (De-Odorase), or calcium chloride diet (major study).**

concentrations were measured by aspiration tubes in rooms with pigs fed yucca extract or calcium chloride compared with the control diet. There was considerable variation in ammonia concentration among samples obtained from different locations in the rooms. Ammonia concentrations were highest when the tubes were placed over the

manure maintained below the pens. Lower concentrations of ammonia were measured when samples were taken from the right side of the room (few feces on the floor) compared with the left side (feces on the floor) or when measured over the collection pit.

In this study, manure collection pits were emptied weekly because the



capacity to store feces, urine, waste feed and spilled drinking water was limited. This may have limited aerial ammonia concentration. In spite of the relatively low concentration of aerial ammonia, these results show that *Yucca schidigera* extract and calcium chloride diets can be used to reduce ammonia concentration from nursery pig facilities. These effects were most evident during the third and fourth weeks of the major study.

Plasma urea concentration was not affected by diet. In the present study, crude protein levels and ADFI were similar among treatments. Apparently the changes in ammonia concentration caused by the dietary treatments were not reflected in changes in plasma urea.

### Conclusions

The results from these studies indicate that ammonia concentrations in nursery pig facilities can be reduced by using feed additives such as *Yucca schidigera* extract and calcium chloride. Aerial ammonia concentrations increased steadily as the trials progressed, but never reached excessive concentrations. However, under commercial conditions, where the air exchange rate is lower and the density of nursery pigs is greater than those used in this study, aerial ammonia concentrations may be higher. The different response of growth performance between pigs fed the calcium chloride diet and the pigs fed the control diet can be attributed to alterations in the dietary electrolyte balance in pigs fed calcium chloride. Further research is needed to determine the optimal concentration of this calcium salt that must be added to nursery diets to reduce ammonia concentration without reducing growth performance.

<sup>1</sup>Janeth J. Colina is a graduate student in animal science, Austin J. Lewis is a professor of animal science, Phillip S. Miller is an associate professor of animal science, and Robert L. Fischer is a graduate student and technician in animal science.

# Plasma Urea Concentrations of Pigs on Commercial Operations

Robert L. Fischer  
Phillip S. Miller  
Austin J. Lewis<sup>1</sup>

## Summary and Implications

*Research was conducted on commercial swine operations to determine whether plasma urea concentrations could be used as an indicator of the protein requirement of growing-finishing pigs. The research consisted of a 30-question survey and an on-farm visit to collect blood and feed samples. The survey included questions about genetics, nutrition, housing and health. Results showed that when plasma urea concentrations were analyzed across all phases of production, barrows had greater plasma urea concentrations than gilts. Plasma urea concentrations varied between the different phases of production, with nursery pigs having the lowest plasma urea concentrations, followed by growing and finishing pigs, respectively. An increase in dietary crude protein resulted in an increase of plasma urea in barrows and gilts in all phases of production. The comparison of dietary crude protein concentrations and age of the pigs at the time of blood collection indicates that the majority of the diets were over-formulated for crude protein. The effects of sex, crude protein, and phase of production on plasma urea concentrations in pigs raised on commercial operations were similar to those in a research setting. These results suggest that within an individual swine operation, plasma urea is a useful indicator of the protein requirement of growing-finishing pigs.*

## Introduction

The main goal of pork producers is to produce a high-quality product at the least cost. For producers to attain this goal and remain competitive in a

changing industry, they must keep an open mind about changes that will improve the efficiency of their operations. To operate an efficient swine enterprise, producers must stay informed about new technologies in the areas of genetics, nutrition, management practices, facilities, and disease management. Implementing new technology from any one of these areas, or in combination, may alter the nutrient requirements of pigs in the operation. To monitor the protein requirement of pigs on a regular basis, a quick and reliable indicator of a pig's protein requirement would be an effective diagnostic tool for the producer and (or) nutritionist. This type of diagnostic tool would enable producers to formulate diets that accurately provide a pig with its dietary protein requirement and allow pigs to achieve their genetic potential for lean growth. The net result of a more precise feeding program would be decreased costs, because of an increase in the efficiency of nutrient use by the pig.

Diets that supply crude protein in excess of the requirements for maintenance and protein accretion are inefficient because excess protein nitrogen is excreted in the urine in the form of urea. Pigs have little ability to store excess amino acids independent of muscle protein. Thus, amino acids in excess of the requirement are catabolized (used for energy or fat deposition), and the nitrogen ( $\text{NH}_3$ ) is converted to urea, causing in some instances a sharp rise in plasma urea concentrations. Researchers have reported that dietary lysine concentrations above the requirement result in an increase in plasma urea concentration in growing and finishing pigs. A similar plasma urea pattern has been observed in pigs when dietary crude protein is supplied in excess of the requirement. These data suggest that feeding crude protein

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**Table 1. Average concentrations of plasma urea, dietary crude protein concentrations, and age of pigs at the time of blood collection for each farm.**

Item	Farm											
	A	B	C	D	E	F	G	H	I	J	K	L
<b>Nursery barrows</b>												
PUC <sup>a</sup> , mg/dL	29.47	20.82	28.00	—	—	—	19.52	—	19.71	16.26	31.72	—
CP <sup>b</sup> , %	20.78	20.46	22.69	—	—	—	16.11	—	21.48	17.15	22.14	—
Age, weeks	9	4	8	—	—	—	7	—	6	8	8	—
<b>Nursery gilts</b>												
PUC, mg/dL	33.90	17.76	23.00	—	—	—	18.70	—	21.39	16.79	25.28	—
CP, %	20.78	20.46	22.69	—	—	—	16.11	—	21.48	17.15	22.14	—
Age, weeks	9	4	8	—	—	—	7	—	6	8	8	—
<b>Growing barrows</b>												
PUC, mg/dL	31.41	31.77	—	22.12	28.60	44.74	32.10	39.37	30.53	21.04	21.86	31.66
CP, %	17.77	17.78	—	20.21	17.78	18.99	15.39	18.38	17.54	14.91	13.72	19.99
Age, weeks	17	14	—	13	12	13	15	11	15	17	16	10
<b>Growing gilts</b>												
PUC, mg/dL	32.71	31.89	—	24.45	35.63	34.29	26.85	30.21	31.01	18.42	23.17	27.94
CP, %	16.69	17.85	—	20.21	19.67	18.89	17.09	18.67	17.90	14.91	15.55	19.99
Age, weeks	17	14	—	13	12	13	15	11	15	17	16	10
<b>Finishing barrows</b>												
PUC, mg/dL	38.47	36.47	30.94	37.51	41.95	42.44	—	—	39.46	28.49	20.11	25.23
CP, %	15.79	16.05	12.54	17.66	19.36	15.46	—	—	14.71	13.31	12.76	14.55
Age, weeks	24	22	24	20	19	22	—	—	24	21	20	23
<b>Finishing gilts</b>												
PUC, mg/dL	37.67	30.57	28.02	31.82	32.73	36.66	—	—	36.91	22.97	24.46	24.98
CP, %	16.47	16.05	12.54	17.66	18.75	15.26	—	—	15.51	13.31	16.01	14.55
Age, weeks	24	22	24	20	19	22	—	—	24	21	20	23

<sup>a</sup>PUC = plasma urea concentration.

<sup>b</sup>CP = dietary crude protein.

levels greater than the requirement result in an excess of amino acids that must be catabolized and removed from the body.

Plasma urea concentrations have been used by researchers as an indicator of a pig's protein and amino acid requirements. Research projects conducted at the University of Nebraska-Lincoln have demonstrated that changes in plasma urea concentrations in response to changes in the dietary protein level can be used to determine the protein requirement of pigs that possess different genetic potentials to deposit lean. These studies have shown that plasma urea concentrations could also be used to determine the time point at which the protein level should be changed throughout the growing-finishing period. The plasma urea concentration response agreed well with the growth and carcass data, and it seems that plasma urea concentrations may reflect changes in the pig's protein requirement more precisely than do standard growth and carcass data. Therefore, the use of plasma urea concentrations as a tool to help producers precisely formulate diets throughout

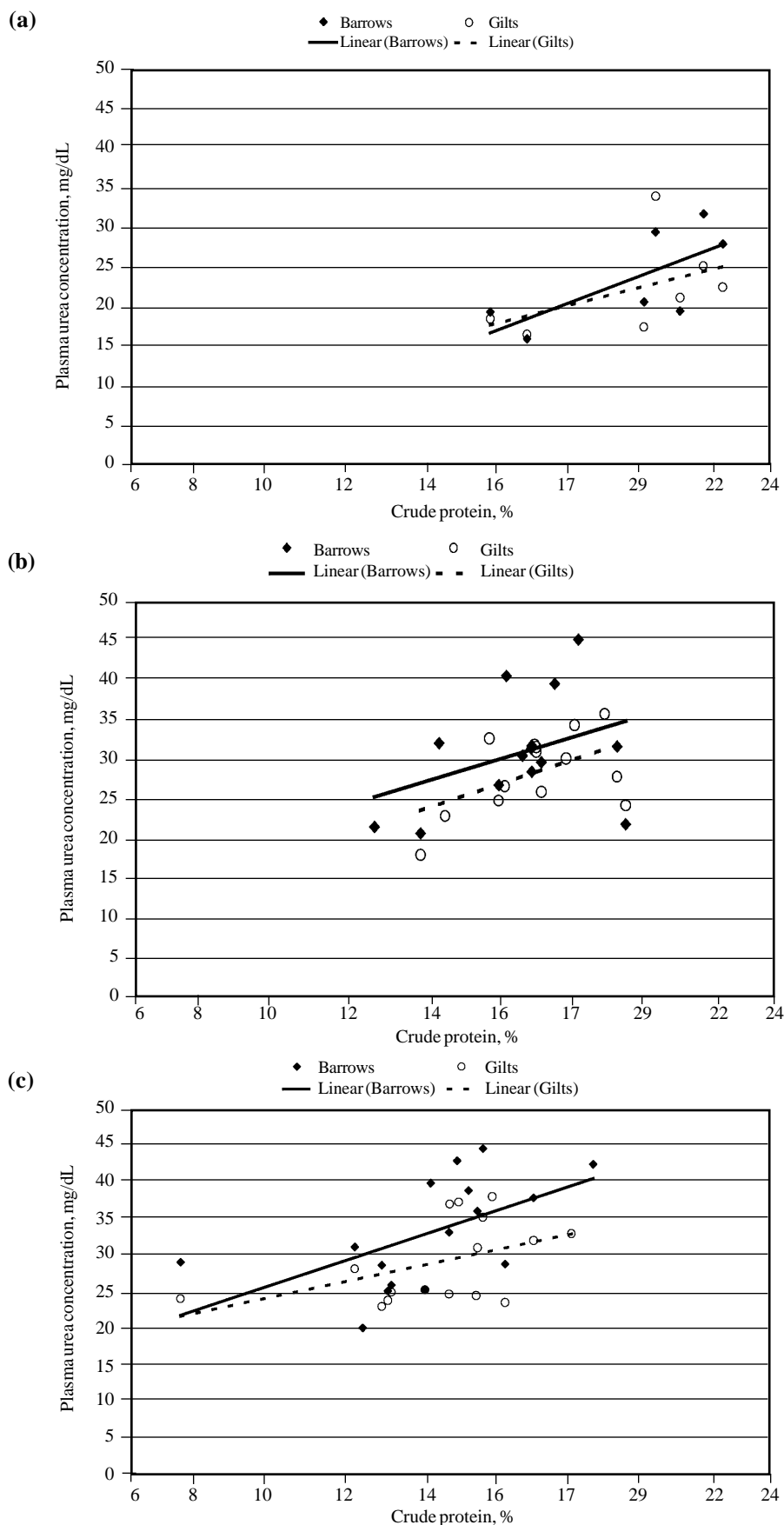
the growing-finishing period provides the pork industry an alternate and simple method of determining requirements for dietary protein. Thus, the current research was designed to investigate whether plasma urea concentrations can be used as an on-farm index of the protein requirements of different populations of pigs.

### Procedures

The research included two parts. Part one was a 30-question survey that was completed by the producer. The survey included four major sections with questions about genetics, nutrition, housing, and health. The genetics section asked questions about seedstock suppliers, replacement gilts, determination of nutrient requirements, and lean gain potential. This section also inquired about carcass data and production records. Information acquired included backfat depth, loin depth, percent yield and lean, hot carcass weight, average weight at the beginning of the growing-finishing period, average slaughter weight, and days from start of the growing-finishing

period to slaughter. From the carcass and production data, an average fat-free lean gain was calculated for each operation. Nutrition questions included protein and lysine concentrations fed in each diet, the amount of each diet provided, separate-sex feeding, and type and amount of antibiotics used in the diets. The housing section questions pertained to type of facilities (confinement or outdoors), ventilation, space/pig, type of feeders, and space/pig for feeders and waterers. The health section included questions about facility biosecurity, pig flow, pig grouping, facilities cleaning, antigen exposure, visual symptoms of illness, and percent death loss.

The second part of this research was an on-farm visit. During the on-farm visit any questions that the producer had about the questionnaire were answered. Blood samples (plasma and serum) were collected from 10 barrows and 10 gilts within each growth phase (nursery, growing, and finishing). A diet sample was collected for each group of pigs sampled. The diet sample was analyzed for crude protein and plasma samples were analyzed for urea con-



**Figure 1.** The response of plasma urea concentration to changes in dietary crude protein; a) nursery; b) growing; c) finishing. Each data point represents an individual operation; a) nursery, n=7; b) growing, n=14; c) finishing, n=16.

centration.

Data were analyzed using the GLM procedure of SAS (1996). The main effects in the statistical model were sex (barrows and gilts) and phase (nursery, growing, and finishing). Regression equations for plasma urea concentration on dietary crude protein concentration for barrows and gilts during each phase of production were analyzed to determine the amount of variation in plasma urea concentration that was accounted for by a change in dietary crude protein. In all statistical analyses, farm was the experimental unit.

## Results

Plasma urea concentration, dietary crude protein concentration, and age of the pigs at the time of blood collection are shown in Table 1. Plasma urea concentrations were greater ( $P < 0.05$ ) in barrows than in gilts in all phases of production. There was an effect of phase of production ( $P < 0.01$ ), with the finishing pigs having the greatest plasma urea concentration followed by the growing and nursery pigs, respectively. An increase in the concentration of dietary crude protein increased ( $P < 0.01$ ) the concentration of plasma urea (Figure 1). Gilts consistently had lower plasma urea concentrations than barrows, especially when dietary protein concentrations were high. This lower plasma urea concentration indicates that gilts have better utilization of protein for lean muscle deposition compared to barrows. The 1998 NRC model was used to predict the crude protein requirement of pigs from each farm and phase of production using data shown in Table 2. These predicted crude protein requirements were compared to the analyzed crude protein concentrations in the diet. During the late nursery phase, six to eight weeks of age, some diets were providing approximately two percentage units more crude protein than required by the pig. Analyzed crude protein concentrations indicated that many pigs in the growing, nine to 17 weeks of age, and finishing, 17 to 24 weeks of age, (Continued on next page)



**Table 2. Production data from each farm surveyed and sampled.**

Item	Farm											
	A	B	C	D	E	F	G	H	I	J	K	L
Number of pigs sold/year	2,400	2,400	1,300	2,400	1,250	1,200	1,200	25,000	2,300	9,000	8,000	285,000
Backfat, in	.75	.72	.76	.74	.82	.77	.79	.74	.70	.66	.89	.74
Loin depth, in	2.50	2.60	2.68	2.43	2.60	2.47	2.51	2.50	2.65	2.27	—	2.3
Percent lean	54.00	55.20	54.00	55.00	54.00	54.00	53.90	55.00	54.50	55.50	—	53.50
Percent yield	75.00	76.80	75.90	75.40	76.10	75.00	75.09	75.00	75.50	76.60	74.74	75.85
Hot carcass weight, lb	180	199	204	200	214	190	198	193	201	192	197	183
Starting weight, lb	11	11	11	11	11	11	65	11	11	12	12	45
Slaughter weight, lb	240	260	272	265	279	253	266	257	266	243	253	242
No. of days in finishing period	172	164	168	182	175	170	112	166	163	161	—	122
Fat-free lean gain, lb/day <sup>b</sup>	.53	.63	.62	.56	.62	.56	.70	.60	.63	.62	—	.64
Separate-sex feeding?	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	No
Space/pig <sup>a</sup> (Nursery)	3.5	—	4.0	3.0	—	—	3.0	—	3.5	3.0	2.5	2.8
Space/pig (Growing)	6.0	—	8.0	5.0	8.4	8.0	7.8	—	8.0	3.5	12.0	7.8
Space/pig (Finishing)	6.0	—	8.0	10.0	8.4	8.0	7.8	—	8.0	4.0	12.0	7.8
Percent death loss (Nursery)	2.5	1.0	2.5	2.5	4.5	—	1.0	—	2.5	2.5	1.0	2.5
Percent death loss (Growing/Finishing)	4.5	4.5	2.5	2.5	2.5	2.5	2.5	—	2.5	2.5	2.5	4.5

<sup>a</sup>Space/pig = ft<sup>2</sup>/pig.

<sup>b</sup>FFLG was calculated by using the equation:

$$\frac{(\text{Final carcass fat-free lean, lb}) - (\text{Initial carcass fat-free lean, lb})}{\text{Days from initial to final weight}}$$

Final carcass fat-free lean (lb) = 0.95 x (percent lean, 5% fat basis x hot carcass weight, lb)

Initial fat-free lean (lb) = 0.95 x [-3.65 + (.418 x live weight, lb)]

age, phases were potentially overfed protein by as much as three percentage units.

Regression equations (Table 3) for the effect of crude protein on plasma urea concentration (depicted in Figure 1) were calculated for each sex and phase of production. Results show that the change in crude protein accounted for 14 to 50% of the variation in plasma urea concentration.

Survey results from each farm are shown in Table 2. Production data from the operations showed that approximately 28,000 pigs/year (range: 1,200 to 285,000 pigs/year) were sold per farm. Lean gain potential on most farms was considered to be in the high category (> .72 lb/d). Data from the kill sheets show that the average backfat depth was .76 inches and loin depth was 2.50 inches. The average percent lean was 54.4, yield was 75.6 %, and hot carcass weight was 195 lb. Weight at the beginning of the growing-finishing period averaged 19 lb, average weight at slaughter was 258 lb, and the number of days from the start of the finishing period to slaughter was 160. Fat-free lean gain calculated from the production and carcass data averaged .61 lb/

**Table 3. Regression equation of plasma urea concentration on dietary crude protein for barrows and gilts during each phase of production, equations are represented in Figure 1.**

Item	Phase of Production and Gender <sup>a</sup>					
	N-G	N-B	G-G	G-B	F-G	F-B
a <sup>b</sup>	1.1243	1.6727	1.5929	1.4255	.9877	1.5419
b	-0.2587	-10.0030	.0485	5.9710	14.1490	10.1990
R square	.23	.50	.27	.14	.22	.32

<sup>a</sup>N-G = nursery gilts; N-B = nursery barrows; G-G = growing gilts; G-B = growing barrows; F-G = finishing gilts; F-B = finishing barrows.

<sup>b</sup>Equation: Y = ax + b; Y = plasma urea concentration (mg/dL); x = dietary crude protein concentration (%).

d. Separate sex feeding was used on most farms. The majority of pig flow was all-in-all-out in the nursery and growing-finishing facilities, and the majority of pigs were raised in mechanically ventilated confinement buildings. Space per pig averaged 3.16 ft<sup>2</sup> in the nursery and 7.75 ft<sup>2</sup> in the growing-finishing phase. All facilities were routinely high-pressure washed and disinfected between pig groups. Mycoplasma hyopneumonia and Porcine Respiratory and Reproductive Syndrome (PRRS) were the two main disease concerns. Death loss in the nursery and growing-finishing periods averaged 2.5%.

Production and carcass data acquired from the producers indicate they

are producing a lean pork product. The use of separate sex feeding on most farms allows producers to accurately provide the nutrients required to maximize lean growth without over-formulating the diet. The space per pig in the nursery and growing-finishing facilities was adequate, therefore growth (lean tissue gain) was not affected by crowding. The low mortality rate can be attributed to good management, all-in-all-out pig flow, and the cleaning and disinfecting of facilities between pig groups. A major concern is the difference between the lean gain potential as estimated by individual producers and the calculated fat-free lean gain of the pigs. The difference between the lean gain potential (> .72



lb/d) and the average calculated fat-free lean gain (.61 lb/d) is 15%. Therefore, if the producers were formulating their diets based on the lean gain potential of their pigs and not the actual fat-free lean gain the diets would be over-formulated for dietary crude protein. This may explain why some diets were over-formulated for dietary crude protein by as much as three percentage units. In addition, research has shown that when pigs are fed a corn-soybean meal diet with no crystalline amino acids to meet the pig's crude protein requirement the plasma urea concentration should be approximately 25 mg/dL. In many cases, the plasma urea concentrations shown in Table 1 exceed 25 mg/dL, further supporting the finding that most diets were over-formulated for crude protein.

### Conclusions

Results from this on-farm study indicate that the relationship between plasma urea concentration and dietary crude protein is similar to the relation-

ship established in our research facilities. Plasma urea concentrations have the potential to be used as a means of selecting replacement boars and gilts with a high potential for lean growth. Animals would be selected for a low plasma urea concentration when fed a diet formulated to meet their dietary protein requirement. By using this technology, a producer would select animals with a more efficient utilization of dietary protein and an increase in lean muscle accretion. This technology also has the ability to help producers make management decisions on their farms. Producers could use this technology to determine if the environment, diet, facility or a management practice is limiting the lean growth potential of their pigs. These results also indicate that plasma urea concentration has the potential to be used as an indicator of the protein requirement of growing-finishing pigs. The use of plasma urea concentrations to determine the protein requirement would be less costly and time consuming than the traditional feeding and carcass analysis experiments used to identify

protein requirements for growing-finishing pigs. However, nutrient requirements for each phase of production on each operation are not the same because of differences in genetics, management, disease, and facilities between operations. Therefore, this approach may assist producers in determining the correct time point to change the nutrient density in the diet to meet the protein requirement more accurately. Also, this methodology may help producers and nutritionists clearly identify instances where excess dietary protein is provided.

A special thank-you goes out to all of the producers involved in this experiment. We appreciated your willingness to participate in this experiment, taking time to fill out the survey, and allowing us to bleed pigs on your operation. Without you this experiment would not have been possible.

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<sup>1</sup>Robert L. Fischer is a research technologist and graduate student, Phillip S. Miller is an associate professor, and Austin J. Lewis is a professor in the Department of Animal Science.

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# Replacing Conventionally Processed Soybean Meal with Extruded/Expelled Soybean Meal in Swine Diets

**Andrea M. Tucker**  
**Phillip S. Miller**  
**Austin J. Lewis**  
**Duane E. Reese<sup>1</sup>**

### Summary and Implications

*The effects of extruded/expelled soybean meal (ESBM) on growth performance and carcass composition of pigs from weaning to slaughter were investigated. Two experiments were conducted. In the first experiment, weaned pigs were fed a diet containing either conventional solvent-extracted soybean meal or ESBM. Average daily gain and feed efficiency were greater*

*in pigs fed the control diet. The second experiment was designed to determine whether nursery diet influenced performance during the growing-finishing period. In the second experiment, half of the pigs from Experiment 1 were assigned to either a control or ESBM diet and were fed until slaughter. Average daily gain and feed efficiency of pigs fed the control diet were slightly greater than those of pigs fed the ESBM diets. Differences in performance of pigs fed the two diets were greater during the nursery phase than during the growing-finishing phase. These results support our previous research in that ESBM offers no advantage in swine growth performance*

*over conventional solvent-extracted soybean meal.*

### Introduction

Extruded/expelled soybean meal (ESBM) is produced by mechanical friction creating a high temperature for a short time period. The temperature and the time spent at a given temperature directly affects the quality and nutritional value of the product. Extruded/expelled soybean meal has the potential to be a high-quality protein and oil source if processed correctly. After extrusion, soybeans (~18% fat) are expelled (pressed) to remove  
(Continued on next page)





the oil. Extruded/expelled soybean meal (the final product) has about 7% fat compared to <1% fat in conventional soybean meal. Therefore, ESBM has a greater energy content than conventional soybean meal and has the potential to be an excellent feed ingredient. Extruded/expelled soybean meal is a convenient way to include fat in swine diets for purposes such as dust reduction, in addition to its nutritional value.

In the 1998 Nebraska Swine Report, results of a study comparing conventional solvent-extracted soybean meal to ESBM fed to early-weaned pigs were presented. In that study, pigs fed ESBM grew more slowly and were less efficient than pigs fed conventional soybean meal. In contrast, recent work completed at Kansas State University (KSU) suggests that pigs fed ESBM perform similarly to pigs fed conventional solvent-extracted soybean meal. The ESBM used at KSU was from a different source than that used in the Nebraska study. An article in the 2000 Nebraska Swine Report concluded that there is considerable variation in the quality of ESBM fed to pigs and that affects its economic value.

The primary objective of this research was to investigate the effects of soybean meal type on pig performance in pigs fed either a diet containing conventional soybean meal or ESBM.

### Procedures

Two experiments were conducted. Experiment 1 was a 28-day nursery trial and Experiment 2 was a 119-day growing-finishing trial.

#### Experiment 1

Four-hundred-eighty crossbred pigs weaned at 11 to 14 days of age (initial body weight 9.05 lb) were used. Pigs were housed in an environmentally controlled nursery with heat lamps for supplemental heat and had continuous fluorescent lighting throughout the trial. Pigs were allotted to pens with 20 pigs/pen (10 barrows and 10 gilts), and the pen was the experimental unit. Pens were allocated to either a control diet

**Table 1. Composition of experiment 1 diets, % (as fed basis).**

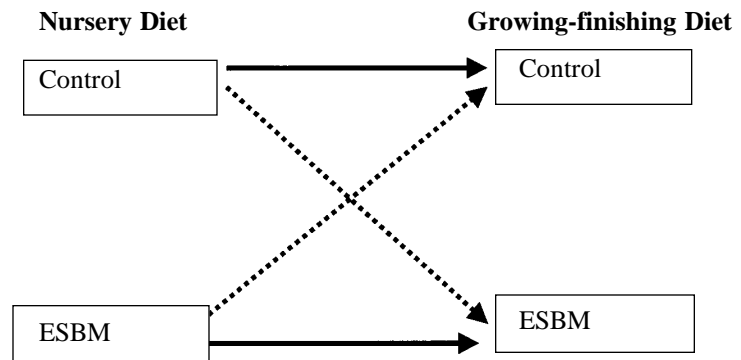
Ingredient, %	Phase 1 <sup>a</sup>		Phase 2 <sup>a</sup>	
	Control	ESBM <sup>b</sup>	Control	ESBM
Corn	39.15	40.40	50.00	50.65
Soybean meal (47.5% CP <sup>c</sup> )	15.75	—	25.75	—
ESBM (43% CP)	—	16.50	—	27.10
Dried whey	27.00	27.00	14.00	14.00
Plasma protein	5.00	5.00	—	—
Menhaden fishmeal	7.00	7.00	4.00	4.00
Corn oil	2.00	—	2.00	—
Dicalcium phosphate	1.15	1.15	1.25	1.25
Limestone	.15	.15	.20	.20
Mineral premix	.10	.10	.10	.10
Vitamin premix	1.00	1.00	1.00	1.00
Salt	.30	.30	.30	.30
Mecadox-50	1.00	1.00	1.00	1.00
Zinc oxide	.40	.40	.40	.40
Calculated composition				
CP, %	22.26	21.98	20.57	20.04
Lysine <sup>d</sup> , %	1.34	1.34	1.09	1.09
ME, kcal/lb <sup>c</sup>	1,514	1,495	1,513	1,512

<sup>a</sup>Phase 1 diets fed from d 0 to 14, Phase 2 diets fed from d 14 to 28.

<sup>b</sup>ESBM = Extruded/expelled soybean meal.

<sup>c</sup>CP = Crude protein; ME = metabolizable energy.

<sup>d</sup>Apparent digestible basis.



**Figure 1. Experiment 2 pig allotment.**

**Table 2. Composition of experiment 2 diets, % (as fed basis).**

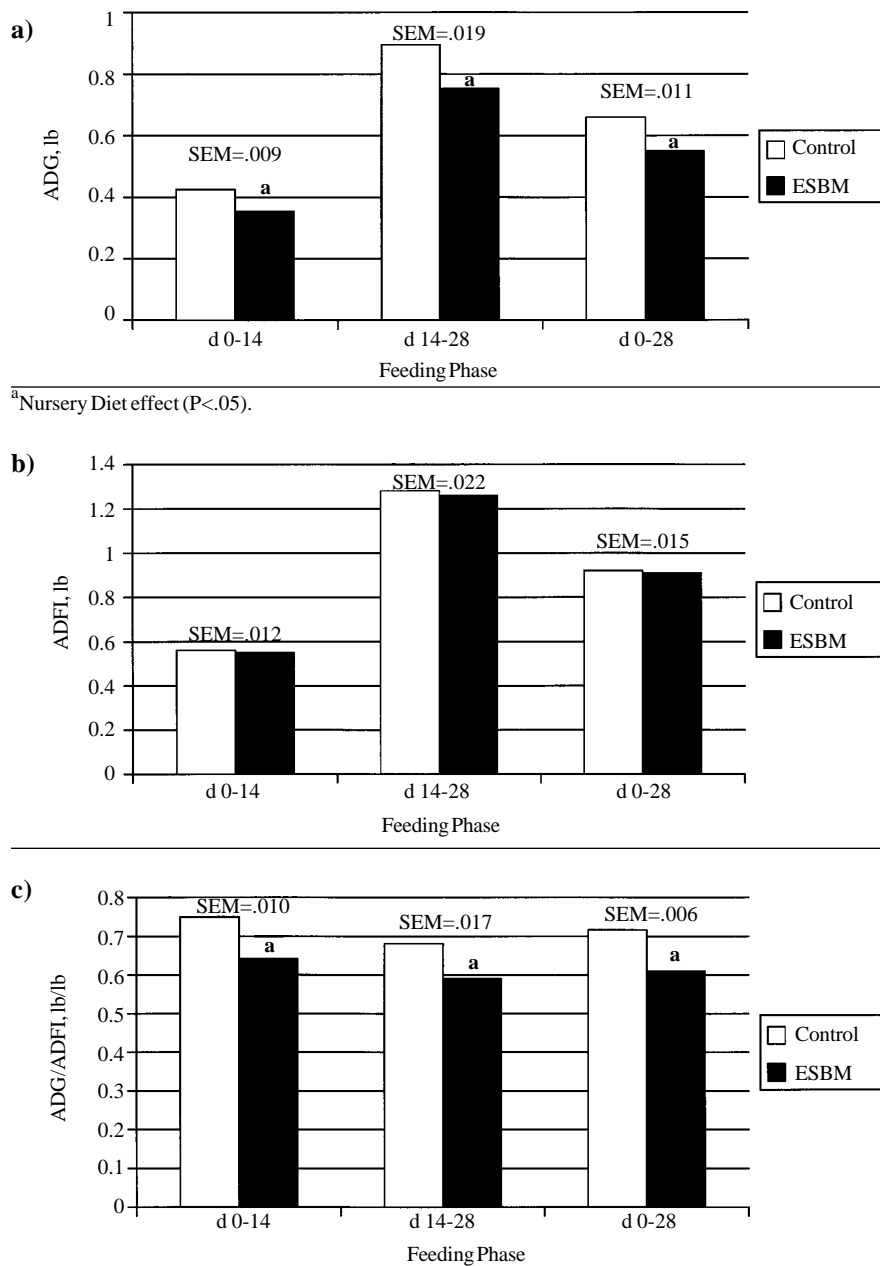
Ingredient, %	Phase 1 <sup>a</sup>		Phase 2 <sup>a</sup>		Phase 3 <sup>a</sup>	
	Control	ESBM <sup>b</sup>	Control	ESBM	Control	ESBM
Corn	68.15	68.65	76.15	76.90	80.30	81.59
Soybean meal (47.5% CP <sup>c</sup> )	27.25	—	19.25	—	15.25	—
ESBM (43% CP)	—	28.75	—	20.50	—	15.96
Tallow	2.00	—	2.00	—	2.00	—
Dicalcium phosphate	1.10	1.10	1.10	1.10	.95	.95
Limestone	.40	.40	.40	.40	.40	.40
Mineral premix	.10	.10	.10	.10	.10	.10
Vitamin premix	.70	.70	.70	.70	.70	.70
Salt	.30	.30	.30	.30	.30	.30
Calculated Composition						
CP, %	18.60	18.06	15.46	15.20	13.91	13.63
Lysine <sup>d</sup> , %	1.00	1.00	.78	.78	.67	.67
ME, kcal/lb <sup>c</sup>	1,547	1,556	1,549	1,544	1,552	1,539

<sup>a</sup>Phase 1 diets fed from 27 to 110 lb (d 0 to 56), Phase 2 diets fed from 110-180 lb (d 56 to 91), Phase 3 fed diets from 180-240 lb (d 91 to 119).

<sup>b</sup>ESBM = Extruded/expelled soybean meal.

<sup>c</sup>CP = Crude protein; ME = metabolizable energy.

<sup>d</sup>Apparent digestible basis.



<sup>a</sup>Nursery Diet effect ( $P < .05$ ).

<sup>a</sup>Nursery Diet effect ( $P < .05$ ).

**Figure 2.** The response of a) average daily gain (ADG), b) average daily feed intake (ADFI), and c) ADG/ADFI to extruded/expelled soybean meal (ESBM) in Experiment 1.

( $n = 12$ ) or an experimental diet ( $n = 12$ ). Pigs were given ad libitum access to feed and water throughout the 28-day feeding trial.

Diets were formulated to contain similar percentages of digestible lysine and were corn-soybean meal based containing either conventional soybean meal (Control) or ESBM (Table 1). Pigs were fed their respective treatment diet for 28 days. There were two feeding phases during Experiment 1 to meet the changing nutritional require-

ments of the pigs. Phase 1 diets were fed from day 0 to 14 (1.34% digestible lysine) and phase 2 diets were fed from day 14 to 28 (1.09% digestible lysine).

Pig and feeder weights were recorded weekly and blood samples were collected on days 0, 14, and 28 of the trial for analysis of plasma urea nitrogen.

#### Experiment 2

Two-hundred-forty pigs from Experiment 1 were selected immedi-

ately after the nursery trial to continue on in the grower-finisher phase of the study. We selected an equal number of pigs from each Experiment 1 diet (120 pigs fed Control and 120 pigs fed ESBM) to achieve a common initial pig body weight (27 lb) for all pens in Experiment 2. Pigs were housed in a modified-open-front building 10 pigs/pen (5 barrows and 5 gilts), and pen was the experimental unit. Pens were assigned either a Control diet ( $n = 12$ ) or an ESBM diet ( $n = 12$ ). Pigs were assigned to a pen based on the dietary treatment fed during the nursery phase such that all pigs in a pen were fed the same diet during Experiment 1. This created four possible Experiment 1-Experiment 2 diet combinations: Control-Control, Control-ESBM, ESBM-Control, and ESBM-ESBM, respectively (Figure 1).

Diets were formulated on an equal digestible lysine basis (Table 2). Experiment 2 was divided into three feeding phases. Phase 1 diets were fed from days 0 to 56 (27 to 110 lb). Phase 2 diets were fed from days 56 to 91 (110 to 180 lb), and Phase 3 diets were fed from days 91 to 119 (180 to 240 lb). The diets contained 1.00%, 0.78% and 0.67% lysine, respectively.

Pigs and feeders were weighed approximately every two weeks to make phase changes close to target weights. Blood samples were collected on days 56, 91 and 119. On day 119, backfat (BF) and longissimus muscle area (LMA) were measured using real-time ultrasound by a trained technician. Pigs were transported to a slaughter facility and TOBEC (total body electrical conductivity) measurements were recorded for each carcass.

## Results

### Experiment 1

Average daily gain (ADG), average daily feed intake (ADFI), and feed efficiency (ADG/ADFI) are shown in Figures 2 a, b, and c, respectively. Overall, and from days 0-14 and days 14-28, ADG was greater ( $P < .05$ ) in the Control pigs than the ESBM pigs.

(Continued on next page)



There were no significant differences between the treatment groups in ADFI at any point during the nursery trial. From days 0-14, days 14-28 and days 0 to 28, ADG/ADFI was greater ( $P < .05$ ) in the Control pigs than the pigs fed ESBM. On days 14 and 28, plasma urea concentrations (PUC) were greater ( $P < .05$ ) for pigs fed ESBM compared to the Control group (data not shown).

### Experiment 2

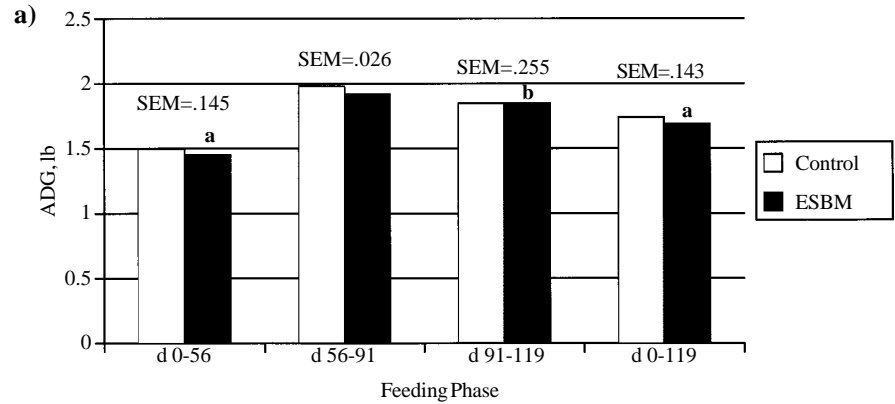
Average daily gain, average daily feed intake, and ADG/ADFI data are shown in Figure 3 a, b, and c, respectively. From days 0-56 and for the entire growing-finishing period (days 0 to 119), ADG was greater ( $P < .05$ ) for the Control pigs than for ESBM-fed pigs. From days 91-119 there was a nursery x growing-finishing diet interaction ( $P < .05$ ) for ADG. Average daily feed intake was greater ( $P < .05$ ) for Control pigs from days 56-91. Control pigs had a greater ( $P < .05$ ) ADG/ADFI from days 0-56. There were no significant differences in plasma urea concentration (data not shown) between the treatment groups.

Carcass data are shown in Table 3. There were no differences in BF and LMA measurements between treatments. Hot carcass weight (HCW) and total pounds of primal cuts were greater ( $P < .05$ ) for Control pigs than for pigs fed ESBM.

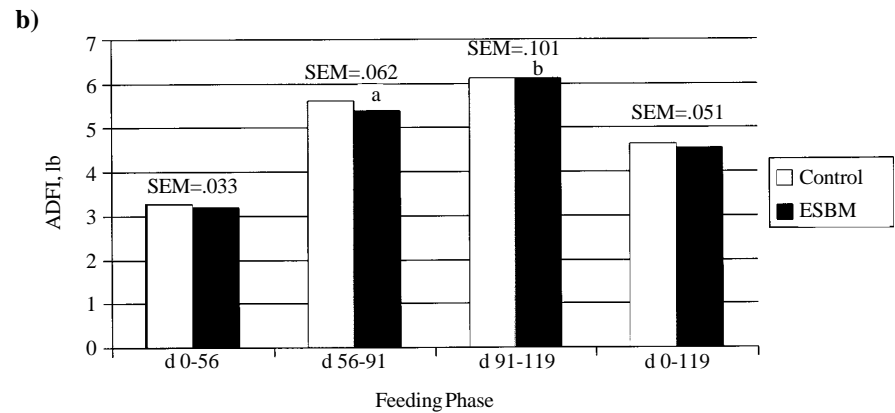
Considering only the 120 pigs that were maintained on either the Control diet or the ESBM diet from weaning till slaughter, there was a trend ( $P < .10$ ) for greater ADG and ADG/ADFI for the Control pigs vs. ESBM-fed pigs (ADG: 1.52 vs. 1.47 lb, respectively; ADG/ADFI: 1.00 vs. 0.98, respectively).

### Conclusions

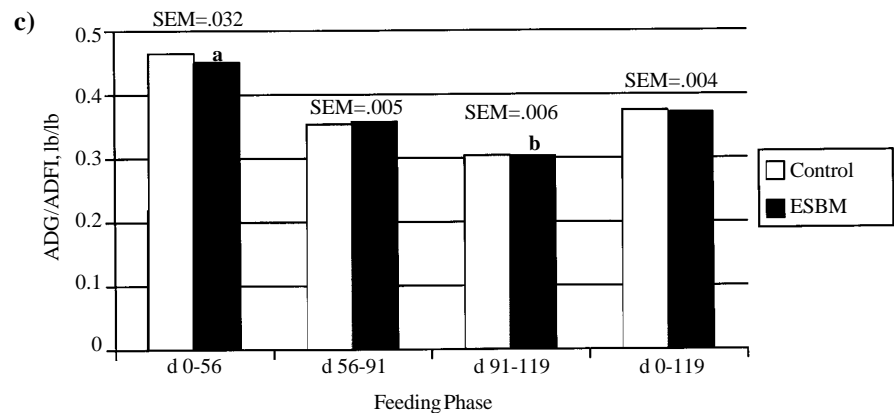
During the nursery trial, pigs fed ESBM had reduced ADG and ADG/ADFI versus pigs fed the Control diet. The greater PUC for the ESBM-fed



<sup>a</sup>Growing-finishing diet effect ( $P < .05$ ).  
<sup>b</sup>Growing-finishing diet x nursery diet interaction ( $P < .05$ ).



<sup>a</sup>Growing-finishing diet effect ( $P < .05$ ).  
<sup>b</sup>Growing-finishing diet x nursery diet interaction ( $P < .05$ ).



<sup>a</sup>Growing-finishing diet effect ( $P < .05$ ).  
<sup>b</sup>Nursery diet ( $P < .05$ ).

**Figure 3. The response of a) average daily gain (ADG), b) average daily feed intake (ADFI), and c) ADG/ADFI to extruded/expelled soybean meal (ESBM) in Experiment 2.**



**Table 3. Effects of soybean meal type on carcass composition.**

	GF <sup>a</sup> Diet		P-value
	Control	ESBM	GF Diet
HCW <sup>b</sup>	178.88	173.81	<.05
Ham, lb	21.52	20.97	NS
Loin, lb	24.65	24.23	NS
Shoulder, lb	25.89	25.32	NS
Total pounds lean <sup>c</sup>	90.23	87.40	<.05
Primal cut <sup>d</sup> , %	40.28	40.57	NS
Total lean <sup>e</sup> , %	50.44	50.28	NS
Backfat, in	.70	.70	NS
LMA <sup>f</sup> , in <sup>2</sup>	16.00	15.58	NS

<sup>a</sup>GF = growing-finishing diet; Control = conventional soybean meal; ESBM = extruded/expelled soybean meal.

<sup>b</sup>HCW = hot carcass weight.

<sup>c</sup>Total pounds lean = pounds of boneless ham, loin, shoulder, belly, and trimmings.

<sup>d</sup>Primal cut, % = pounds of boneless ham, loin, and shoulder/HCW.

<sup>e</sup>Total lean, % = total pounds of boneless lean/HCW.

<sup>f</sup>LMA = longissimus muscle area.

<sup>g</sup>NS = Not significant (P > .05).

pigs suggests that protein quality and(or) amino acid availability may be compromised in ESBM. During the growing-finishing trial, growth performance differences between the Control group

and the ESBM-fed pigs were reduced. This observation could be related to age of the pig. If the quality of the ESBM was poor (damaged protein and(or) presence of antinutritional fac-

tors), the deleterious affects would be reduced as the pig matured. Considering the whole period from weaning to finishing, there was a trend for pigs fed the Control to have a slight advantage over pigs fed ESBM.

Extruded/expelled soybean meal may be a satisfactory ingredient in swine diets when fed either during the growing-finishing period or from weaning to finishing. The variation in ESBM from different processing plants and questions about quality control and nutrient availability in ESBM need to be explored.

<sup>1</sup>Andrea M. Tucker is a graduate student, Phillip S. Miller is an associate professor, Austin J. Lewis is a professor, and Duane E. Reese is an associate professor in the Department of Animal Science.

# Economic Value of Ractopamine (Paylean™) for Finishing Pigs

Duane E. Reese  
Larry L. Bitney<sup>1</sup>

## Summary and Implications

*Ractopamine, a feed additive which improves feed efficiency, daily gain and several carcass characteristics recently became available to pork producers. An economic feasibility analysis on the feeding of 4.5, 9.0, and 18.0 g/ton ractopamine to finishing pigs fed a 16% crude protein (0.82% lysine) corn-soybean meal diet from 150 to 240 lb was conducted. The analysis was performed in two stages: 1) an economic benefit for ractopamine was calculated from cost savings due to improved feed efficiency and daily gain, and 2) the amount of carcass premium needed per pig to recover the added cost of feeding ractopamine was calculated for each dietary level of ractopamine. We assumed one pound of Paylean™,*

*containing 9 grams of ractopamine per pound, cost \$26. As expected, the economic benefit (considering improved feed efficiency and daily gain) of feeding ractopamine increases as corn and soybean meal prices increase. However, its use cannot be justified economically through improved feed efficiency and daily gain alone (corn = \$2.00/bu; soybean meal = \$200/ton). A producer would need to earn carcass premiums averaging \$.41, \$1.85, or \$4.97 per pig in order to recover the cost of feeding 4.5, 9.0, and 18.0 g/ton ractopamine, respectively. From the standpoint of costs and returns and assuming carcass premium is based on 10th rib backfat, it appears easier to justify feeding 9 g/ton ractopamine compared to 4.5 or 18 g/ton, because the first 9 grams of ractopamine resulted in the biggest reduction in 10th rib backfat (.09 inches), while an additional 9 g/ton (total of 18 g/ton) reduced backfat another .04 inches*

*only. However, if carcass premium is based on a measure of loin eye area, feeding 4.5 g/ton ractopamine may be the best choice. We conclude that a consistent carcass premium is necessary to justify feeding ractopamine economically and that producers supplement published research information on responses to feeding ractopamine with data generated on their own pigs.*

## Introduction

Pork producers have the opportunity to use a new feed additive, ractopamine, in finishing pig diets. Ractopamine (Paylean™; Elanco Animal Health) belongs to a class of compounds known as beta-agonists. These compounds are similar in structure and pharmacological properties to epinephrine (adrenaline), a hormone secreted by the adrenal gland. Beta-agonists alter how nutrients that pigs

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consume are used for growth; more nutrients are used for muscle deposition and less are used for fat synthesis. Ractopamine was recently approved by the Food & Drug Administration (FDA) for increased rate of gain, improved feed efficiency, and increased carcass leanness in finishing pigs fed a complete diet containing at least 16% crude protein from 150 to 240 lb. The additive can be included in a finisher diet at 4.5, 9.0, or 18.0 g/ton.

As new technologies become available, it is important that producers carefully evaluate their value. In this paper we intend to provide producers a tool to estimate the economic feasibility of feeding ractopamine to finishing pigs. Experience feeding ractopamine under current conditions in the pork industry is very limited, thus this is a progress report.

### Performance Results

A summary of several studies conducted to determine the effect of feeding ractopamine to finishing pigs is shown in Tables 1 and 2. Daily gain and feed efficiency increased by 7 and 8%, respectively, when pigs were fed diets containing 4.5 g/ton ractopamine (Table 1). Further additions of ractopamine only slightly improved gain and efficiency. Adding 14.5 g/ton more ractopamine (total of 18.0 g/ton) improved daily gain and feed efficiency by 3 and 5%, respectively, over the 4.5 g/ton response. Thus, the total response observed from 18.0 g/ton of ractopamine for daily gain and feed efficiency was 10 and 13%, respectively. Adding 4.5 g/ton of ractopamine also significantly reduced feed intake, but the overall response was much less than that observed for daily gain and feed efficiency.

The effect ractopamine had on carcass characteristics varied depending on the trait measured (Table 2). Dressing percent was improved at all levels of ractopamine. Midline backfat at the last rib, average midline backfat, and 10th rib fat depth were not affected when 4.5 g/ton ractopamine was added to the diet. However, 10th rib backfat

**Table 1. Effect of ractopamine on finisher pig growth performance<sup>a</sup>.**

Item	Ractopamine, g/ton			
	0	4.5	9.0	18.0
No. of pens	84	84	84	82
No. of pigs	479	488	486	469
Daily gain, lb	1.84	1.97 <sup>b</sup>	1.99 <sup>b</sup>	2.02 <sup>b</sup>
Daily feed, lb	6.60	6.50 <sup>c</sup>	6.42 <sup>b</sup>	6.34 <sup>b</sup>
Feed/gain	3.62	3.33 <sup>b</sup>	3.25 <sup>b</sup>	3.16 <sup>b</sup>

<sup>a</sup>Adapted from Elanco Paylean<sup>TM</sup> Technical Summary. Average beginning and final body weights were 150 and 240 lb, respectively. A 20-trial summary. Dietary protein and lysine = 16 and .82%, respectively.

<sup>b</sup>Different from the control diet ( $P < .01$ ).

<sup>c</sup>Different from the control diet ( $P < .05$ ).

**Table 2. Effect of ractopamine on finisher pig carcass measurements<sup>a</sup>.**

Item	Ractopamine, g/ton			
	0	4.5	9.0	18.0
Slaughter weight, lb	232	233	233	232
Dressing percent	73.3	73.7 <sup>b</sup>	74.1 <sup>c</sup>	74.4 <sup>c</sup>
Midline last rib backfat, in	.99	1.00	.98	.97
Avg midline backfat, in	1.21	1.23	1.19	1.17 <sup>b</sup>
10th rib fat depth, in	1.08	1.06	.99 <sup>c</sup>	.95 <sup>c</sup>
10th rib loin eye area, in <sup>2</sup>	5.08	5.51 <sup>c</sup>	5.68 <sup>c</sup>	5.80 <sup>c</sup>

<sup>a</sup>Adapted from Elanco Paylean<sup>TM</sup> Technical Summary. Dietary protein and lysine = 16 and .82%, respectively.

<sup>b</sup>Different from the control diet ( $P < .05$ ).

<sup>c</sup>Different from the control diet ( $P < .01$ ).

**Table 3. Benefit (\$ per pig) from feeding 4.5 g/ton ractopamine at alternative corn and soybean meal prices<sup>a</sup>.**

44% CP soybean meal, \$/ton	Corn, \$/bushel			
	1.50	2.00	2.50	3.00
150	1.06	1.24	1.42	1.60
200	1.21	1.38	1.56	1.74
250	1.35	1.53	1.71	1.88
300	1.49	1.67	1.85	2.03

<sup>a</sup>Calculated from feed efficiency values in Table 1.

**Table 4. Benefit (\$ per pig) from feeding 9.0 g/ton ractopamine at alternative corn and soybean meal prices<sup>a</sup>.**

44% CP soybean meal, \$/ton	Corn, \$/bushel			
	1.50	2.00	2.50	3.00
150	1.36	1.58	1.81	2.04
200	1.54	1.77	1.99	2.22
250	1.72	1.95	2.18	2.41
300	1.91	2.13	2.36	2.59

<sup>a</sup>Calculated from feed efficiency values in Table 1.

**Table 5. Benefit (\$ per pig) from feeding 18.0 g/ton ractopamine at alternative corn and soybean meal prices<sup>a</sup>.**

44% CP soybean meal, \$/ton	Corn, \$/bushel			
	1.50	2.00	2.50	3.00
150	1.69	1.97	2.26	2.54
200	1.92	2.20	2.48	2.77
250	2.15	2.43	2.71	3.00
300	2.37	2.66	2.94	3.22

<sup>a</sup>Calculated from feed efficiency values in Table 1.



depth was significantly reduced at the 9.0 g/ton inclusion rate and at 18.0 g/ton ractopamine seemed to reduce backfat slightly further. A significant reduction in average midline backfat was not observed until 18 g/ton ractopamine was included in the diet, while there was no change detected in last rib midline backfat. Ractopamine increased loin eye area, especially at the 4.5 g/ton level.

### Estimated Value

To estimate the economic value of ractopamine in a finishing pig diet, four 16% crude protein (0.82% lysine), corn-soybean meal diets were formulated (This level of lysine is about 0.1% units higher than the 0.72% that we normally recommend for 150 to 240 lb finishing pigs that are not fed ractopamine. Because ractopamine reduces feed intake and increases lean gain, dietary amino acid level should be increased.). All the diets contained 44% crude protein soybean meal as the sole source of supplemental protein and the same level of energy, amino acids, vitamins and minerals. Diets were formulated to contain 0, 4.5, 9.0, and 18.0 g/ton of ractopamine. Ractopamine replaced corn in the diet.

### Feed Efficiency

The responses for feed efficiency shown in Table 1 were applied to the diets containing ractopamine. The cost savings realized from improved feed conversion were attributed to ractopamine. (Note - The control diet for calculating the cost savings was a 16% protein (0.82% lysine) diet, not the typical 0.72% lysine diet recommended for pigs in this weight range.) The feed cost savings per pig is the benefit from feeding 4.5 (Table 3), 9.0 (Table 4), and 18.0 g/ton ractopamine (Table 5). This benefit is of course higher at higher corn and soybean meal prices.

Approximately two-thirds of the total benefit from increased feed efficiency is realized at the 4.5 gram per ton level. For example, at a corn price

of \$2.00/bu and soybean meal price of \$200/ton, the benefit from using 4.5 grams/ton is \$1.38 per pig, while the added benefit from using another 4.5 grams/ton (9 grams/ton) is only \$0.39 per pig (\$1.77-\$1.38). The added benefit from feeding 18 grams/ton vs 9 grams/ton is \$0.43 per pig (\$2.20-1.77). The marginal benefits of feeding ractopamine from improvements in feed efficiency decrease at the 9 and 18 g/ton level, because of the diminishing response observed in feed efficiency as the dietary level of the additive increased (Table 1).

### Average Daily Gain

This is a difficult benefit to quantify. Based on data in Table 1, pigs receiving diets containing 4.5, 9.0, and 18.0 g/ton of ractopamine would reach market weight 3.2, 3.7, and 4.3 days sooner than those not receiving ractopamine. These changes are not of a magnitude to justify changing the number of turns per year in a facility, but there could be other sources of benefit. For example, if all pigs were sold from a facility a few days early, some savings in interest, utilities and repairs might be realized. Or, the manager may choose to feed the pigs the "normal" time, and realize a benefit in extra pounds sold, less added feed cost. Still another source of benefit could be fewer lightweight pigs when the facility is completely emptied, resulting in less sort loss.

We chose the most conservative estimate, that of interest, utility and repair savings due to pigs going to market earlier. This was credited at the rate of \$.05 per pig per day, resulting in benefits of \$.16, \$.18, and \$.22 per pig for the 4.5, 9.0, and 18.0 g/ton of ractopamine.

### Carcass Premiums

Given the current cost of ractopamine used in our budgeting process, its use cannot be justified economically by increased feed efficiency and average daily gain alone. However, ractopamine increases loin eye

area and may reduce carcass backfat (Table 2). That could generate additional income for the producer. The question is whether current packer carcass merit buying programs fully reward the producer for the investment in ractopamine. To generate a carcass premium, the technology applied to the pig must change carcass merit enough to move its carcass into a better pricing category. The percentage of pigs in a group that would be shifted to a better pricing category would depend on the size of the range for the carcass trait(s) measured within each pricing category and how much the technology changes carcass merit.

Because of the large variation in genetics, production systems and differences in packer buying grids and how carcasses are evaluated, it is difficult to develop estimates of the benefit a producer would receive in carcass premiums. The approach we have taken is presented in Figure 1. The cost of ractopamine at each of the inclusion levels is represented by a bar in the graph. We assumed Paylean™, containing 9 grams of ractopamine per pound, cost \$26 per pound. The benefits from increased feed efficiency and average daily gain are shown, as is the carcass premium that would be needed per pig in order to recover the added cost of feeding ractopamine. According to our calculations, a producer would need to earn carcass premiums averaging \$.41, \$1.85, and \$4.97 per pig in order to recover the cost of feeding 4.5, 9.0, and 18.0 g/ton ractopamine, respectively. If a producer considers it highly likely to obtain a larger average premium than that shown in Figure 1, it would be profitable to feed ractopamine. When considering possible premiums for carcass merit, note that it is likely that not all carcasses from a group of pigs fed ractopamine will be shifted into a higher carcass pricing category and earn a premium. Thus, carcasses from pigs that earned a premium must pay for the ractopamine consumed by pigs that did not earn a carcass premium.

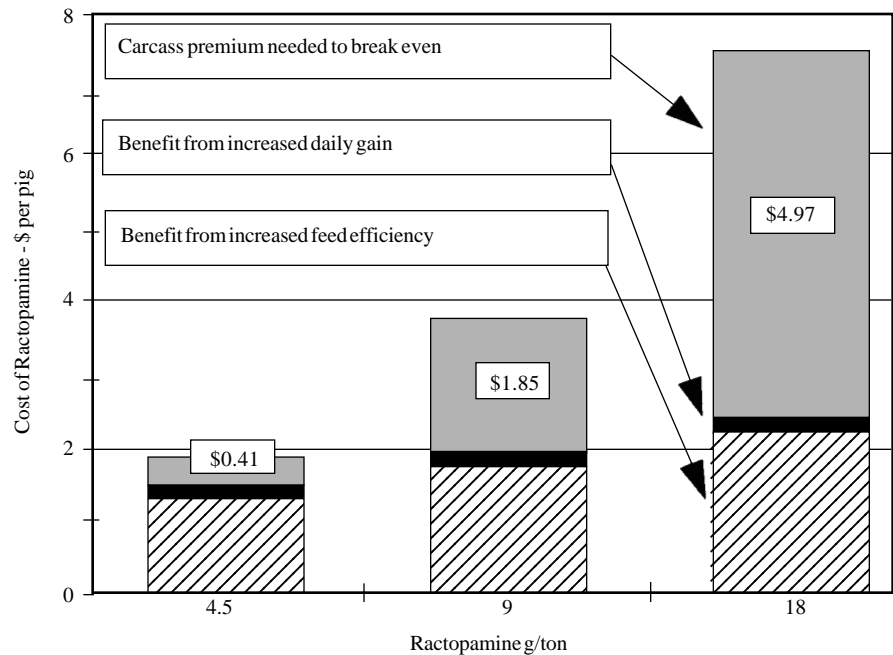
The price of Paylean™ also will affect the size of the carcass premium



needed per pig. For each \$2/lb change in the price of Paylean™, the carcass premium required changes by approximately \$.15, \$.30, and \$.60 per pig for the 4.5, 9.0, and 18.0 g/ton levels, respectively. For example, if Paylean™ cost \$24/lb (we used \$26 in our analysis), the carcass premium required to break even feeding 4.5, 9.0 and 18.0 g/ton ractopamine would be \$.26, \$1.55, and \$4.37, respectively.

The carcass premium necessary to justify feeding 4.5 g/ton ractopamine is much lower than for the higher inclusion rates of the additive. However, according to the data in Table 2, 4.5 g/ton of ractopamine does not reduce backfat. Therefore, to justify feeding 4.5 g/ton ractopamine, all the benefit would need to come from improved feed efficiency and daily gain if the carcass merit program was based on backfat only. Corn or soybean meal prices would need to rise above \$3/bu or \$300/ton, respectively, for that to occur or the price of Paylean™ would need to be about \$20/lb. If, however, the carcass premium is based on a measure of loin eye area, feeding 4.5 g/ton ractopamine may be easy to justify under our input price assumptions. Assuming pigs are evaluated on 10th rib backfat, the greater economic potential may be for feeding 9 g/ton ractopamine compared to 4.5 or 18 g/ton. The first 9 g of ractopamine resulted in the biggest reduction in 10th rib backfat (.09 inches); an additional 9 g/ton (total of 18 g/ton) reduced backfat another .04 inches only.

The cost of Paylean™ per pig may be understated in Figure 1. The costs shown reflect adding Paylean™ to an existing 16% protein (0.82% lysine) diet. Producers who are currently following UNL swine nutrient recommendations (available at <http://ianrwww.unl.edu/pubs/swine/ec273.htm>), feeding a 15% protein (0.72% lysine) diet, would incur a cost of \$0.82 per pig (\$2/bu corn and \$200/ton soybean meal) to switch to the 16% protein diet, so that they could feed Paylean™. If no improvement in feed efficiency resulted from the switch to a 16% protein diet, the \$0.82 would be



**Figure 1.** Estimated benefit from feeding 4.5, 9, or 18 g/ton ractopamine to finishing pigs (150 to 240 lb) considering improvements in feed efficiency and daily gain. The difference between the cost of ractopamine per pig and the benefits shown represents the amount of carcass premium required to cover the cost of ractopamine consumed. Selected ingredient prices: Paylean™, containing 9 grams ractopamine per lb, \$26/lb; corn \$2.00/bu; 44% soybean meal \$200/ton.

an added cost per pig of feeding Paylean™, and the carcass premium required (Figure 1) from feeding the 9 g/ton level would be \$2.67 instead of \$1.85 per pig (\$1.85+0.82). An decrease of 0.18 lb feed per pound of gain would be required to offset the cost of changing from a 15% to a 16% crude protein diet.

### Conclusion

Practical experience with feeding ractopamine in today's pork industry is limited. Therefore, it is important that producers calculate the costs and benefits of ractopamine for themselves and supplement that with published research data. Three key variables affecting the level of carcass merit premium required are the prices of Paylean™, corn and soybean meal.

It may be very useful for producers to collect data from their own pigs fed ractopamine. The data we used in this analysis (Table 1 and 2) was generated during the late 1980s and early 1990s. Improvements have been made in the

genetic merit of pigs since then that could affect the response to ractopamine. In addition, further research may indicate that the response to ractopamine could be different when diets contain more than 16% crude protein or when ractopamine is fed for shorter lengths of time than over the 150 to 240 lb range that we modeled. Moreover, one would obtain specific information from the packer which would help decide if the carcass premiums we calculated are likely to be obtained. Guidelines for conducting on-farm feed research trials are available in the University of Nebraska publication, *Conducting Pig Feed Trials on the Farm (EC 92-270)* available at county extension offices in Nebraska or on the Internet at <http://www.ianr.unl.edu/pubs/swine/ec270.htm>.

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# Valine, Isoleucine, and Histidine Supplementation of Low-Protein, Amino Acid-Supplemented Diets for Growing Pigs

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## Summary and Implications

Previous experiments have shown that when the crude protein of a growing diet is reduced from 16 to 11% there is reduced growth performance of pigs, even though the diets are supplemented with lysine, tryptophan, threonine, and methionine. To determine which amino acid was next limiting in a corn-soybean meal, low-protein (11%), amino acid-supplemented diet, we conducted three experiments. In each of the three experiments, 36 growing gilts were individually penned and fed one of six diets. The three first diets were used in all three experiments: 1) 16% crude protein (CP) positive control, 2) 12% CP neutral control, and 3) 11% CP negative control. In Experiment 1, the 11% CP diet was supplemented with: 4) isoleucine, 5) valine, and 6) isoleucine + valine. In Experiment 2, the 11% CP diet was supplemented with: 4) histidine, 5) histidine + valine, and 6) histidine + valine + isoleucine. In Experiment 3, the 11% CP diet was supplemented with: 4) valine, 5) valine + histidine, and 6) valine + isoleucine. All low-protein diets were supplemented with lysine, tryptophan, threonine, and methionine. The supplementation of isoleucine or histidine alone reduced feed intake, daily gain, feed/gain, daily lean gain, longissi-

*mus muscle area, and backfat thickness. Valine supplementation improved growth performance, but the combination of valine with isoleucine or histidine increased growth performance to levels similar to those of gilts fed the 16% CP diet. Plasma urea nitrogen decreased as the crude protein decreased from 16 to 11%. These results suggest that dietary crude protein can be reduced from 16 to 11% with little or no effect on pig performance if amino acids such as valine and isoleucine or valine and histidine are added. This reduction in dietary crude protein will reduce nitrogen excretion in feces and urine.*

## Introduction

Previous experiments reported in Nebraska Swine Reports have indicated that the crude protein (CP) content of diets for growing pigs can be reduced if they are supplemented with crystalline amino acids (lysine, tryptophan, threonine, and methionine). However, a reduction in protein content from 16 to 11% caused a reduction in other amino acids, and, because of that, growth performance was reduced. Amino acids that may be limiting in an 11% CP diet are valine, isoleucine, and histidine.

Other researchers have reported improvements in pig performance with supplementation of both valine and isoleucine to an 11% CP diet, but they did not obtain an improvement in growth performance with either valine or isoleucine supplementation alone. In fact,

isoleucine decreased pig performance, and valine improved average daily gain (ADG) but not feed efficiency.

## Materials and Methods

Three experiments were conducted to determine the fifth and sixth limiting amino acids in a low-protein (11% CP), corn-soybean meal, amino acid-supplemented diet for growing pigs. In all experiments, 36 crossbred gilts weighing 46 lb were penned individually and fed one of six diets for 35 days. The diets (Tables 1, 2, and 3) were a standard corn-soybean meal diet with 16% CP and two low-CP amino acid-supplemented (lysine, tryptophan, threonine, and methionine) diets with 12 and 11% CP. In addition, in Experiment 1, the 11% CP diet was supplemented with isoleucine, valine, or isoleucine + valine; in Experiment 2, the 11% CP diet was supplemented with histidine, histidine + valine, or histidine + valine + isoleucine; and in Experiment 3, the 11% CP diet was supplemented with valine, valine + histidine, or valine + isoleucine to make up the other three diets in each experiment. All low-protein diets were supplemented with lysine, tryptophan, threonine, and methionine to have the same total concentration as in the standard (16% CP) diet. The supplements of isoleucine, valine, and histidine were also to approximately the same levels as in the 16% CP diet. Average daily gain (ADG) and average daily feed intake (ADFI) were measured weekly.

(Continued on next page)





Fat-free lean gain was calculated from backfat thickness and longissimus muscle area (obtained on the first and the last day of the experiment using real-time ultrasound) using the National Pork Producers Council equation, and plasma urea and plasma amino acid concentrations were determined in blood collected on the last day of each experiment.

## Results and Discussion

### Growth Performance

In general, ADG was higher in gilts fed 16 or 12% CP diets than in gilts fed 11% CP diets, even when supplemented with valine, isoleucine, histidine, or various combinations (Tables 4, 5, and 6). Supplementation of isoleucine or histidine alone decreased ADG, whereas supplementation of valine alone increased ADG compared with the 11% CP diet. However, the combination of valine and isoleucine; valine and histidine; or valine, histidine, and isoleucine in an 11% CP diet increased ADG to levels similar to those of gilts fed the 12% CP diet or even the 16% CP diet.

There was no effect of dietary CP concentration on ADFI. Supplementation of isoleucine or histidine alone reduced ADFI compared with the supplementation of valine alone or the control diets. However, gilts fed the 11% CP diet with the combination of valine and isoleucine had higher ADFI than gilts fed the 12 and 16% CP diets.

Feed efficiency (ADFI/ADG) was affected by dietary CP concentration, with better values in gilts fed 16 and 12% CP diets than in gilts fed the 11% CP diets. Supplementation of isoleucine or histidine alone had a negative effect on feed efficiency. Supplementation of both isoleucine and valine or histidine and valine resulted in better feed efficiency than the 11% CP diet.

Fat-free lean gain was affected by dietary CP concentration, with higher values in gilts fed the 16 or 12% CP diets than in gilts fed the 11% CP diets. Supplementation of either isoleucine or histidine alone decreased lean gain

**Table 1. Composition of diets<sup>a</sup>, Exp. 1.**

Item	Dietary protein concentration, %					
	16	12	11	11+I	11+V	11+IV
Ingredient, %						
Corn	71.36	82.41	85.22	84.95	84.95	84.67
Soybean meal (46.1% CP)	23.70	11.50	8.50	8.50	8.50	8.50
Tallow	2.00	2.00	2.00	2.00	2.00	2.00
Dicalcium phosphate	1.17	1.46	1.47	1.47	1.47	1.47
Limestone	0.68	0.60	0.60	0.60	0.60	0.60
Salt	0.30	0.30	0.30	0.30	0.30	0.30
Vitamin premix <sup>b</sup>	0.70	0.70	0.70	0.70	0.70	0.70
Trace mineral premix <sup>c</sup>	0.10	0.10	0.10	0.10	0.10	0.10
L-Lysine•HCl	—	0.48	0.57	0.57	0.57	0.57
L-Tryptophan	—	0.08	0.095	0.095	0.095	0.095
L-Threonine	—	0.23	0.27	0.27	0.27	0.27
DL-Methionine	—	0.15	0.175	0.175	0.175	0.175
L-Isoleucine	—	—	—	0.275	—	0.275
L-Valine	—	—	—	—	0.275	0.275
Calculated nutrient composition, %						
Crude protein	16.00	12.00	11.00	11.20	11.20	11.50
Lysine	0.92	0.92	0.92	0.92	0.92	0.92
Tryptophan	0.20	0.20	0.20	0.20	0.20	0.20
Threonine	0.67	0.67	0.67	0.67	0.67	0.67
Methionine + cystine	0.60	0.60	0.60	0.60	0.60	0.60
Isoleucine	0.69	0.47	0.42	0.69	0.41	0.69
Valine	0.79	0.57	0.52	0.52	0.79	0.79
Calcium	0.61	0.61	0.61	0.61	0.61	0.61
Phosphorus	0.56	0.57	0.57	0.57	0.57	0.57
Available phosphorus	0.32	0.32	0.32	0.32	0.32	0.32
Metabolizable energy, Mcal/lb	1.52	1.52	1.52	1.51	1.51	1.51
Net energy, Mcal/lb	1.06	1.07	1.07	1.07	1.07	1.07

<sup>a</sup>As-fed basis. 11+I means 11% CP supplemented with isoleucine; 11+V, 11% CP diet and valine; 11+IV, 11% CP and isoleucine and valine.

<sup>b</sup>Supplied per kilogram of diet: retinyl acetate, 3,858 IU; cholecalciferol, 386 IU; all-rac- $\alpha$ -tocopheryl acetate, 19.3 IU; menadione (as menadione sodium bisulfite complex), 2.3 mg; folic acid, 1.54 mg; niacin, 23 mg; riboflavin, 3.9 mg; cyanocobalamin, 15.4  $\mu$ g; d-pantothenic acid (as d-calcium pantothenate), 15.4 mg; D-biotin, 77  $\mu$ g; choline (as choline chloride), 386 mg.

<sup>c</sup>Supplied (mg/kg of diet): Cu (as  $\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$ ), 11; I (as  $\text{Ca}[\text{IO}_3]_2 \cdot \text{H}_2\text{O}$ ), 22; Fe (as  $\text{FeSO}_4 \cdot \text{H}_2\text{O}$ ), 110; Mn (as MnO), 22; Se (as  $\text{Na}_2\text{SeO}_3$ ), 0.3; Zn (as ZnO), 110.

**Table 2. Composition of diets<sup>a</sup>, Exp. 2.**

Item	Dietary protein concentration, %					
	16	12	11	11+H	11+HV	11+HVI
Ingredient, %						
Corn	71.56	83.15	85.94	85.73	85.45	85.19
Soybean meal (44.8% CP)	23.50	10.75	7.75	7.75	7.75	7.75
Tallow	2.00	2.00	2.00	2.00	2.00	2.00
Dicalcium phosphate	1.16	1.45	1.47	1.47	1.47	1.47
Limestone	0.68	0.60	0.60	0.60	0.60	0.60
Salt	0.30	0.30	0.30	0.30	0.30	0.30
Vitamin premix <sup>b</sup>	0.70	0.70	0.70	0.70	0.70	0.70
Trace mineral premix <sup>c</sup>	0.10	0.10	0.10	0.10	0.10	0.10
L-Lysine•HCl	—	0.47	0.57	0.57	0.57	0.57
L-Tryptophan	—	0.08	0.10	0.10	0.10	0.10
L-Threonine	—	0.23	0.29	0.29	0.29	0.29
DL-Methionine	—	0.15	0.19	0.19	0.19	0.19
L-Histidine•HCl•H <sub>2</sub> O	—	—	—	0.21	0.21	0.21
L-Valine	—	—	—	—	0.28	0.28
L-Isoleucine	—	—	—	—	—	0.28
Calculated nutrient composition, %						
Crude protein	16.00	12.00	11.00	11.20	11.30	11.50
Lysine	0.91	0.91	0.91	0.91	0.91	0.91
Tryptophan	0.20	0.20	0.20	0.20	0.20	0.20
Threonine	0.67	0.67	0.67	0.67	0.67	0.67
Methionine + cystine	0.60	0.60	0.60	0.60	0.60	0.60
Histidine	0.45	0.32	0.29	0.45	0.45	0.45
Isoleucine	0.69	0.46	0.40	0.40	0.40	0.69
Valine	0.79	0.56	0.50	0.50	0.79	0.79
Calcium	0.61	0.61	0.61	0.61	0.61	0.61
Phosphorus	0.57	0.57	0.57	0.57	0.57	0.57
Available phosphorus	0.29	0.32	0.32	0.32	0.32	0.32
Metabolizable energy, Mcal/lb	1.52	1.52	1.52	1.52	1.51	1.51
Net energy, Mcal/lb	1.06	1.07	1.07	1.07	1.07	1.07

<sup>a</sup>As-fed basis. 11+H means 11% CP diet supplemented with histidine; 11+HV, 11% CP and histidine and valine; 11+HVI, 11% CP and histidine, valine, and isoleucine.

<sup>b</sup>Supplied per kilogram of diet: retinyl acetate, 3,858 IU; cholecalciferol, 386 IU; all-rac- $\alpha$ -tocopheryl acetate, 19.3 IU; menadione (as menadione sodium bisulfite complex), 2.3 mg; folic acid, 1.54 mg; niacin, 23 mg; riboflavin, 3.9 mg; cyanocobalamin, 15.4  $\mu$ g; d-pantothenic acid (as d-calcium pantothenate), 15.4 mg; D-biotin, 77  $\mu$ g; choline (as choline chloride), 386 mg.

<sup>c</sup>Supplied (mg/kg of diet): Cu (as  $\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$ ), 11; I (as  $\text{Ca}[\text{IO}_3]_2 \cdot \text{H}_2\text{O}$ ), 22; Fe (as  $\text{FeSO}_4 \cdot \text{H}_2\text{O}$ ), 110; Mn (as MnO), 22; Se (as  $\text{Na}_2\text{SeO}_3$ ), 0.3; Zn (as ZnO), 110.



**Table 3. Composition of diets<sup>a</sup>, Exp. 3.**

Item	Dietary protein concentration, %					
	16	12	11	11+V	11+VH	11+VI
<b>Ingredient, %</b>						
Corn	70.99	82.35	85.19	84.91	84.71	84.64
Soybean meal (44.8% CP)	24.10	11.55	8.50	8.50	8.50	8.50
Tallow	2.00	2.00	2.00	2.00	2.00	2.00
Dicalcium phosphate	1.16	1.45	1.47	1.47	1.47	1.47
Limestone	0.65	0.60	0.60	0.60	0.60	0.60
Salt	0.30	0.30	0.30	0.30	0.30	0.30
Vitamin premix <sup>b</sup>	0.70	0.70	0.70	0.70	0.70	0.70
Trace mineral premix <sup>c</sup>	0.10	0.10	0.10	0.10	0.10	0.10
L-Lysine•HCl	—	0.47	0.57	0.57	0.57	0.57
L-Tryptophan	—	0.08	0.10	0.10	0.10	0.10
L-Threonine	—	0.23	0.29	0.29	0.29	0.29
DL-Methionine	—	0.15	0.19	0.19	0.19	0.19
L-Isoleucine	—	—	—	—	—	0.28
L-Valine	—	—	—	0.28	0.28	0.28
L-Histidine•HCl•H <sub>2</sub> O	—	—	—	—	0.21	—
<b>Calculated nutrient composition, %</b>						
Crude protein	16.00	12.00	11.00	11.10	11.30	11.30
Lysine	0.93	0.92	0.92	0.91	0.91	0.91
Tryptophan	0.20	0.20	0.20	0.20	0.20	0.20
Threonine	0.68	0.68	0.68	0.68	0.68	0.67
Methionine + cystine	0.60	0.61	0.61	0.60	0.60	0.60
Isoleucine	0.70	0.47	0.42	0.41	0.41	0.69
Valine	0.80	0.57	0.52	0.79	0.79	0.79
Histidine	0.46	0.33	0.30	0.30	0.51	0.30
Calcium	0.60	0.61	0.61	0.61	0.61	0.61
Phosphorus	0.57	0.57	0.57	0.57	0.56	0.56
Available phosphorus	0.29	0.32	0.32	0.32	0.32	0.32
Metabolizable energy, Mcal/lb	1.52	1.52	1.52	1.51	1.51	1.51
Net energy, Mcal/lb	1.06	1.07	1.07	1.07	1.07	1.07

<sup>a</sup>As-fed basis. 11+V means 11% CP diet supplemented with valine; 11+VH, 11% CP and valine and histidine; 11+VI, 11% CP and valine and isoleucine.

<sup>b</sup>Supplied per kilogram of diet: retinyl acetate, 3,858 IU; cholecalciferol, 386 IU; all-rac- $\alpha$ -tocopheryl acetate, 19.3 IU; menadione (as menadione sodium bisulfite complex), 2.3 mg; folic acid, 1.54 mg; niacin, 23 mg; riboflavin, 3.9 mg; cyanocobalamin, 15.4  $\mu$ g; d-pantothenic acid (as d-calcium pantothenate), 15.4 mg; D-biotin, 77  $\mu$ g; choline (as choline chloride), 386 mg.

<sup>c</sup>Supplied (mg/kg of diet): Cu (as CuSO<sub>4</sub>•5H<sub>2</sub>O), 11; I (as Ca[IO<sub>3</sub>]<sub>2</sub>•H<sub>2</sub>O), 22; Fe (as FeSO<sub>4</sub>•H<sub>2</sub>O), 110; Mn (as MnO), 22; Se (as Na<sub>2</sub>SeO<sub>3</sub>), 0.3; Zn (as ZnO), 110.

**Table 4. Effect of protein concentration and amino acid supplementation on growth performance of growing gilts<sup>ab</sup> (Exp. 1).**

Item	Dietary protein concentration, % <sup>c</sup>						SEM <sup>d</sup>
	16	12	11	11+I	11+V	11+IV	
<b>Growth performance</b>							
ADG <sup>e</sup> , lb	1.72	1.59	1.47	1.12	1.50	1.58	0.0297
ADFI, lb	3.55	3.50	3.44	2.89	3.55	3.66	0.0574
ADFI/ADG	2.064	2.201	2.340	2.580	2.367	2.317	0.0109
FFLG <sup>g</sup> , lb/d	0.69	0.64	0.58	0.48	0.56	0.60	0.0111
<b>Carcass traits</b>							
Backfat, in	0.40	0.40	0.40	0.33	0.44	0.45	0.0551
LMA <sup>h</sup> , in <sup>2</sup>	3.32	3.34	3.06	2.95	2.91	3.18	0.6857
<b>Blood metabolites, mg/dL</b>							
Urea	20.49	4.97	4.89	6.46	5.84	4.31	0.8544
Lysine	1.29	4.15	5.15	4.71	4.34	5.28	0.7434
Tryptophan	1.05	1.20	1.43	1.24	1.26	1.32	0.1358
Threonine	2.41	4.91	8.35	7.33	5.16	6.83	0.7525
Methionine	0.50	1.09	1.33	1.20	1.06	1.44	0.1830
Valine	3.10	0.93	0.83	0.62	6.99	6.85	0.2840
Isoleucine	1.68	0.69	0.54	2.45	0.64	3.20	0.1979
Histidine	1.34	0.63	0.61	0.54	0.61	0.57	0.0606

<sup>a</sup>The average initial weight was 43 lb.

<sup>b</sup>Number of observations per treatment = 6.

<sup>c</sup>11+I means 11% CP supplemented with isoleucine; 11+V, 11% CP diet and valine; 11+IV, 11% CP and isoleucine and valine.

<sup>d</sup>SEM = Standard error of mean.

<sup>e</sup>ADG = Average daily gain.

<sup>f</sup>ADFI = Average daily feed intake.

<sup>g</sup>FFLG = Fat-free lean gain.

<sup>h</sup>LMA = Longissimus muscle area.

compared with supplementation of valine. In addition, the combination of isoleucine and valine or histidine and valine added to the 11% CP diet was more effective in restoring fat-free lean gain than was the single supplementation of either amino acid. Amino acid supplements restored lean gain to values that were between those of the 11 and 12% CP diets.

### Carcass Traits

Backfat thickness increased with the supplementation of isoleucine plus valine to the 11% CP diet. This effect seemed to be due to the single supplementation of valine compared with isoleucine supplementation, because the single supplementation of isoleucine reduced backfat thickness. Supplementation of either histidine or valine with isoleucine tended to increase backfat thickness of gilts to levels similar to those of the control diet (Table 5), but in Experiment 3 (Table 6) there was no difference among gilts fed any diet.

Longissimus muscle area was affected by dietary CP concentration. It was similar in gilts fed 16 and 12% CP diets but lower in gilts fed 11% CP. There were only minor differences in longissimus muscle area among gilts fed 11% CP diets. Supplementation of valine alone was not enough to improve longissimus muscle area. Supplementation of isoleucine or histidine alone decreased longissimus muscle area, but the combination of valine with isoleucine or histidine tended to increase longissimus muscle area. However, this increase was not large enough to reach levels similar to those of gilts fed 16% CP diets. Supplementation of isoleucine to the diet with histidine and valine did not increase longissimus muscle area further.

### Blood Metabolites

There was a large reduction in plasma urea concentration from the 16% CP diet to all other low-protein diets. Generally, plasma urea concentrations of gilts fed 11% CP diets were

(Continued on next page)



**Table 5. Effect of protein concentration and amino acid supplementation on growth performance of gilts<sup>ab</sup> (Exp. 2).**

Item	Dietary protein concentration, % <sup>c</sup>						SEM <sup>d</sup>
	16	12	11	11+H	11+HV	11+HVI	
Growth performance							
ADG <sup>e</sup> , lb	1.94	1.67	1.50	1.40	1.80	1.68	0.2256
ADFI <sup>f</sup> , lb	3.94	3.88	3.59	3.42	4.00	3.76	0.0695
ADFI/ADG	2.031	2.323	2.393	2.443	2.222	2.238	0.0131
FFLG <sup>g</sup> , lb/d	0.752	0.644	0.567	0.540	0.666	0.615	0.0121
Carcass traits							
Backfat depth, in	0.45	0.43	0.41	0.46	0.51	0.45	0.0888
LMA <sup>h</sup> , in <sup>2</sup>	3.92	3.64	3.41	3.43	3.69	3.48	0.8824
Blood metabolites, mg/dL							
Urea	27.42	8.94	8.29	8.50	6.97	6.61	1.3360
Lysine	1.92	4.26	5.26	5.73	4.56	5.38	0.8811
Tryptophan	1.26	1.32	1.51	1.50	1.24	1.25	0.1241
Threonine	2.60	3.90	5.56	6.06	5.29	5.88	0.6364
Methionine	0.56	1.0	1.28	1.34	1.10	1.08	0.0360
Valine	3.26	0.94	0.69	0.92	6.39	6.25	0.0616
Isoleucine	1.67	0.52	0.42	0.51	0.49	2.66	0.0156
Histidine	1.44	0.48	0.59	1.83	1.69	1.67	0.0214

<sup>a</sup>The average initial weight was 48.3 lb.

<sup>b</sup>Number of observations per treatment = 6.

<sup>c</sup>11+H means 11% CP diet supplemented with histidine; 11+HV, 11% CP and histidine and valine; 11+HVI, 11% CP and histidine, valine, and isoleucine.

<sup>d</sup>SEM = Standard error of mean.

<sup>e</sup>ADG = Average daily gain.

<sup>f</sup>ADFI = Average daily feed intake.

<sup>g</sup>FFLG = Fat-free lean gain.

<sup>h</sup>LMA = Longissimus muscle area.

lower than those of gilts fed 12% CP. The supplementation of valine, histidine, isoleucine, or various combination did not change plasma urea concentrations.

The plasma concentrations of amino acids that were supplemented to all diets (lysine, tryptophan, threonine, and methionine) all responded in a similar manner, increasing as dietary CP was reduced.

Supplementation of valine with or without isoleucine, histidine, or the combination of these two amino acids dramatically increased plasma valine concentration. Values were higher than in pigs fed the control 16% CP diet, suggesting that valine-supplemented diets contained more available valine than the control diet. Similar effects were observed when either isoleucine or histidine were supplemented. The addition of these two amino acids either alone or in combinations resulted in higher plasma concentrations than in pigs fed the control diet. These effects were anticipated because diets were formulated to the same total amino acid concentrations and it is known that crystalline amino acids are more readily bioavailable than those in intact protein sources, such as soybean meal and corn.

These results suggest that valine and isoleucine may be the fifth and sixth limiting amino acids, respectively, in a low-protein, amino acid-supplemented, corn-soybean meal diet for growing pigs. Further research may provide additional insight about the optimal amounts of crystalline amino acids that should be supplemented to improve the performance and carcass traits of growing pigs.

**Table 6. Effect of protein concentration and amino acid supplementation on growth performance of growing gilts<sup>ab</sup> (Exp. 3).**

Item	Dietary protein concentration, % <sup>c</sup>						SEM <sup>d</sup>
	16	12	11	11+V	11+VH	11+VI	
Growth performance							
ADG <sup>e</sup> , lb	1.86	1.79	1.62	1.76	1.82	1.90	0.0355
ADFI <sup>f</sup> , lb	3.89	3.67	3.53	3.85	3.83	3.91	0.0681
ADFI/ADG	2.091	2.050	2.179	2.188	2.104	2.058	0.0077
FFLG <sup>g</sup> , lb/d	0.717	0.706	0.613	0.631	0.672	0.688	0.0105
Carcass traits							
Backfat depth, in	0.44	0.50	0.58	0.52	0.48	0.50	0.0812
LMA <sup>h</sup> , in <sup>2</sup>	3.65	3.69	3.43	3.48	3.49	3.59	0.2014
Blood metabolites, mg/dL							
Urea	22.92	17.85	10.31	8.51	6.66	9.41	2.9701
Lysine	2.36	3.72	3.80	4.66	4.44	4.25	0.4245
Tryptophan	1.26	1.49	1.18	2.08	1.50	2.03	0.3328
Threonine	2.99	3.71	4.94	4.74	4.37	5.30	0.4758
Methionine	0.54	1.00	1.19	1.00	1.08	1.05	0.0955
Valine	3.46	1.35	0.87	4.52	5.12	5.30	0.2930
Isoleucine	1.72	0.77	0.56	0.57	0.55	2.40	0.1017
Histidine	1.30	0.74	0.58	0.49	1.75	0.44	0.0918

<sup>a</sup>The average initial weight was 43 lb.

<sup>b</sup>Number of observations per treatment = 6.

<sup>c</sup>11+V means 11% CP diet supplemented with valine; 11+VH, 11% CP and valine and histidine; 11+VI, 11% CP and valine and isoleucine.

<sup>d</sup>SEM = Standard error of the mean.

<sup>e</sup>ADG = Average daily gain.

<sup>f</sup>ADFI = Average daily feed intake.

<sup>g</sup>FFLG = Fat-free lean gain.

<sup>h</sup>LMA = Longissimus muscle area.

<sup>1</sup>Jose L. Figueroa is a graduate student, Austin J. Lewis is a professor, Phillip S. Miller is an associate professor, and Robert L. Fischer is a graduate student in the Department of Animal Science.



# Dietary Conjugated Linoleic Acid (CLA) and Body Fat Changes

Jess Miner  
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Xiaoli Chen  
Clifton Baile<sup>1</sup>

## Summary and Implications

*Feeding mixed isomers of linoleic acid with conjugated double bonds (CLA) reduces body fat in several species, including pigs. To learn about the mechanism involved, we fed CLA to sexually mature mice at 0, 1 and 2% of the diet for 5, 12 and 14 days. Dietary CLA reduced body fat by nearly 50% but did not reduce body weight. Mice fed CLA also experienced programmed death (apoptosis) of fat cells. This implicates a new mechanism (fat cell death) by which CLA reduces body fat without reducing lean. The effectiveness of feeding CLA for as few as five days may indicate that benefits could be obtained in pigs by feeding CLA for a short duration at the conclusion of the finishing phase.*

## Introduction

Conjugated linoleic acids (CLA) are a group of polyunsaturated fatty acids that differ from linoleic acid only by position of double bonds. Some CLA are produced naturally by anaerobic ruminal metabolism and subsequent animal metabolism. Consequently, ruminant animal fats contain substantial CLA (~.5%). Inclusion of CLA in pig diets has consistently improved belly firmness, often reduced backfat and improved feed efficiency, and sometimes improved lean growth rate (*Nebraska and South Dakota Swine Nutrition Guide*, EC 95-273). The backfat reduction and growth rate responses to CLA have been variable.

We believe that by understanding the mechanism by which CLA causes these benefits, it will be possible to control the variability in response.

Our hypothesis was that CLA can trigger a specific cellular mechanism which leads to an organized death of adipocytes (fat cells) known as programmed cell death, or apoptosis. The research reported here was designed to test this hypothesis in a mouse model. Several mouse experiments can be conducted with the resources required for one experiment using pigs. Subsequent mouse studies will aim to determine the mechanism by which CLA causes fat cell death. Our hope is that an understanding of this biology in mice will allow development of methods to reduce fatness in other mammals such as pigs, cattle, or humans. The specific objective of this study was to determine the effect of CLA on fat depots, feed intake, energy expenditure and apoptosis (programmed death), of fat cells.

## Procedures

### Diets

Conjugated linoleic acid was mixed into a purified base diet of corn starch, casein, soy oil, sucrose, cellulose, vitamins and minerals (AIN-93G). Soy oil was replaced (1:1) with CLA to create diets containing 0, 1 and 2% CLA. All diets were equal in fat content. This CLA was a mixture of isomers with approximately 44% being the type (cis-9/trans-11) that predominates in ruminant fats, and 41% was trans-10/cis-12 which is a major component of commercially synthesized CLA. The remainder of the added CLA was linoleic acid and several additional conjugated isomers.

### Experiment 1

Ninety 10- to 12-week-old male mice were housed individually at 22° C and randomly assigned to one of the three experimental diets (0, 1 and 2% CLA). Feed disappearance and body weight were measured daily. Direct calorimetry was used to measure heat loss during a 4-hour period beginning at 1700 h (5 p.m.) on day nine. On the day of calorimetry, feed was unavailable from 1200 until 1900 h. Thus, heat loss was determined in the fasted and in the refed state for each animal. Heat loss was determined at one-minute intervals and collected every 30 minutes for two hours in each state. Water was not available in the calorimeter chambers. Three days after calorimetry, between 0800 and 1000, mice were sacrificed by CO<sub>2</sub> asphyxia. Brown, epididymal and retroperitoneal fat pads, and livers were removed and weighed. Twenty-one retroperitoneal fat pads were analyzed for apoptosis (programmed cell death).

### Experiment 2

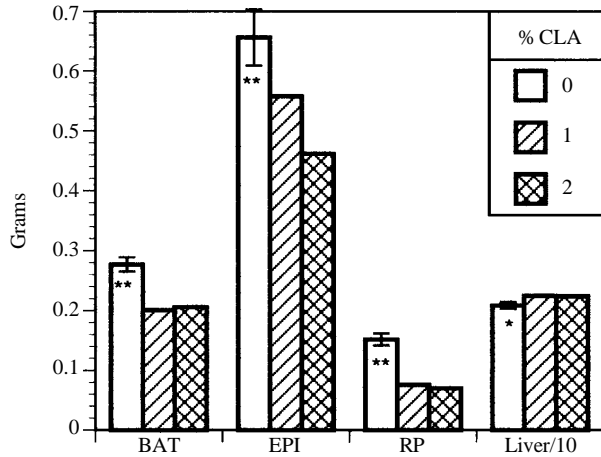
Twenty obese 26- to 30-week-old male mice were randomly assigned to one of three CLA diets: 1) 0% for 12 days; 2) 2% for 14 days; and 3) 0% for nine days followed by 2% for five days. Body fat, body weight, and apoptosis were assayed as in Experiment 1.

## Results

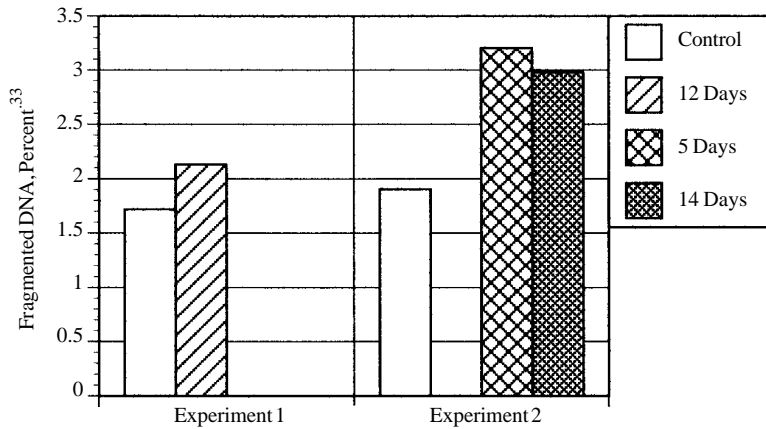
### Experiment 1

Feed intake (g/day) by mice fed 0, 1 and 2% CLA for 12 days was 5.0, 4.7, and 4.4 (SE=.11; P<.05), respectively. Despite consuming less feed, the mice fed 1 and 2% CLA expended as much energy as the controls; heat loss was

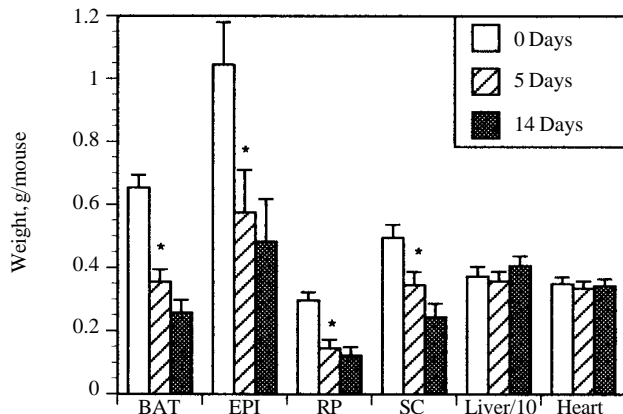
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**Figure 1.** Experiment 1 weight in grams of brown (BAT), epididymal (EPI), and retroperitoneal (RP) fat pads, and of liver (scaled to 10% of actual weight). \*\*CLA effect ( $P < .01$ ). \*CLA effect ( $P < .10$ ). Error bars represent SEM;  $N = 30$  per diet group. Body weight was not influenced by CLA (not shown).



**Figure 2.** Apoptosis in retroperitoneal fat pads. Fragmentation of DNA is a hallmark of apoptosis. Pooled effect of CLA ( $P < .01$ ). From left to right, bars represent data from 9, 12, 7, 6 and 7 animals. Pooled SEM is .19.



**Figure 3.** Experiment 2 weight of subcutaneous (SC), epididymal (EPI), retroperitoneal (RP), and brown (BAT) fat pads, liver (scaled to 10% of actual weight) and heart. \*\*CLA effect ( $P < .01$ ). Error bars represent SEM;  $N = 7$  per diet group.

317, 320, and 308 kcal/kg/day for 0, 1 and 2% CLA, respectively ( $SEM = 12.5$ ;  $P > .5$ ). After consuming CLA for 12 days, mice had up to 50% less body fat than contemporaries which were not fed CLA (Figure 1). The fat cells from mice fed 2% CLA presented more apoptosis as indicated by DNA fragmentation than cells from control mice (Figure 2).

### Experiment 2

Consumption of 2% CLA diet for either five or 14 days caused a significant loss of body fat in all of the depots measured (Figure 3;  $P < .01$ ); however, total body weight increased in animals fed CLA versus control ( $P < .05$ ). Consistent with the results of Experiment 1, analysis of retroperitoneal fat pads indicated that dietary CLA caused programmed cell death in fat cells (Figure 2).

### Discussion

Feeding mixed isomers of conjugated linoleic acid to mice caused a rapid (within five days) loss of body fat, no loss of body weight, and apoptosis of cells in adipose tissue. Perhaps this apoptosis mediates the specific loss of body fat caused by CLA consumption. We speculate that the apoptosis may be mediated by a specific isomer of CLA which has been shown by others to activate a nuclear receptor that regulates gene expression. Perhaps the responses to CLA depend on the amount of this isomer in various sources of CLA. Further work with mice will be directed at identifying which isomer(s) of CLA cause the greatest fat cell apoptosis and reduction of body fat. This should allow more efficient design of treatments to influence composition of growth in swine.

<sup>1</sup>Chris Cederberg was an undergraduate student at UNL. Some of the information in this paper is from his honors research thesis. Xiaoli Chen is a graduate research assistant at the University of Georgia. Clifton Baile is a faculty member at the University of Georgia. Merlyn Nielsen and Jess Miner are faculty of the UNL animal science department.



# The Effect of Compensatory Growth on Organ Weights and Carcass Composition in Growing Gilts

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## Summary and Implications

An experiment was conducted to examine the effects of compensatory growth and amino acid supply on organ weights and carcass characteristics in growing gilts. Gilts were fed either a corn-soybean meal diet or a corn-soybean meal diet supplemented with crystalline lysine. Pigs were randomly allotted to either a 21-day ad libitum eating period or a 42-day restricted-realimentated feeding period. The restricted-realimentated (RR) feeding period consisted of a 21-day restriction period and a 21-day ad libitum eating period (realimentation). During the restriction period, pigs were fed to maintain body weight. Results indicated that during the restriction period, gilts had a decrease ( $P < 0.01$ ) in the weight of the liver, kidneys and small intestine. During the first week of ad libitum eating, organ weights of gilts in the RR group increased dramatically. Weights of the liver and stomach of gilts in the RR group were greater ( $P < 0.05$ ) than the ad libitum (AL) fed gilts during week one of ad libitum eating. In addition, weights of the kidneys, small intestine and mesentery

were not different between feeding regimens after the first week of ad libitum eating. Carcass and ultrasound measurements taken before and after the restriction period showed a numerical decrease in tenth-rib backfat and an increase in longissimus muscle area during the 21-day restriction period. These measurements are consistent with the decrease ( $P < 0.01$ ) in the percentage of carcass fat and an increase ( $P < 0.05$ ) in carcass protein percentage caused by restricted feeding. Although the carcass protein percentage was greater in the RR gilts at the start of the ad libitum eating period, carcass protein accretion was greater ( $P < 0.01$ ) in the AL gilts versus the RR gilts during weeks one and two of ad libitum eating. Gilts in the RR group exhibited compensatory organ growth during the first week of the ad libitum eating period. Also, during a restriction period, growing gilts are able to use fat stores and repartition visceral protein to maintain lean muscle deposition.

Growing pigs often face environmental and health challenges which limit energy and nutrient intake. This research has identified that protein from liver and other visceral depots can be used to help provide amino acids for muscle growth during prolonged feed restriction. Also, the weight and composition of visceral organs are restored quickly when refeeding

commences. Because these tissues account for a significant portion of pig's daily energy requirement, fundamental knowledge documenting how key organs respond to energy and nutrient intake will ultimately help provide insight into how pigs will adapt to specific nutritional regimens. Additional research is needed to see how organs and muscle adaptations change as the pig progresses through the growing and finishing phases.

## Introduction

When a growing pig's development is restricted by a period of environmental stress, such as reduced nutrient availability, severe temperature extremes, or disease, it exhibits a period of decreased growth. Upon removal of such stresses, the animal exhibits an accelerated and more efficient rate of growth than that which is normal for animals of the same chronological age. Bohman (1955) termed this abnormally rapid growth relative to age "compensatory growth." Carcass composition, organ size and metabolic activities are altered during a restriction-realimentation period. Animals are able to recover from periods of undernutrition by prolonging the growth period, increasing appetite and rate of gain, reducing their maintenance energy requirement, and

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increasing efficiency of energy use for body gain. The maintenance energy requirement is reduced in restricted fed animals because of a decrease in basal metabolism rate. Thus, during the early stages of realimentation a greater fraction of the net energy of the diet is available for productive processes in animals exhibiting compensatory growth.

The primary objective of this research was to investigate the effects of feed restriction and realimentation on the response of plasma urea concentration in gilts fed a traditional corn-soybean meal diet or a lysine-supplemented, corn-soybean meal diet. The second objective was to examine organ adaptations and the gilt's ability to deposit lean tissue after a period of feed restriction. The restriction-refeeding model has been used in order to: 1) investigate how organs adapt (provide nutrients) during a period of feed restriction (e.g., with disease and (or) high environmental temperatures); and 2) examine how growth rate (often accelerated upon refeeding after feed restriction) is related to and (or) controlled by changes in visceral organ metabolic activity.

### Procedures

Forty-six crossbred gilts with an initial weight of 73 lb were used. Four gilts were randomly selected for an initial slaughter group to determine initial organ weights and carcass composition. Eighteen gilts were allocated to have ad libitum access to either a corn-soybean meal diet or a corn-soybean diet with supplemental lysine. Within this group, six pigs, three from each diet treatment, were slaughtered on weeks one, two and three of the experiment. Twenty-four gilts were offered a maintenance level of feed for 21 days. Feed allotments were adjusted every three days to minimize weight loss or gain. At the end of the 21-day feed restriction period, the restricted pigs weighed 73 lb. On day 21, six restricted gilts were randomly selected for slaughter. The remaining 18 gilts were allowed ad libitum access to

**Table 1. Ingredient and calculated composition of diets, as-fed basis.**

Item	Corn-soybean meal	Corn-soybean meal + lysine
Ingredient, %		
Corn	74.02	77.85
Soybean meal (46.5% CP)	21.40	17.25
Tallow	2.00	2.10
Lysine	—	.15
Dicalcium phosphate	1.05	1.15
Limestone	.43	.40
Salt	.30	.30
Vitamin premix <sup>a</sup>	.70	.70
Trace mineral premix <sup>b</sup>	.10	.10
Calculated nutrient content		
Crude protein, %	16.30	14.30
ME <sup>c</sup> , Mcal/lb	1.55	1.55
Lysine, %	.89	.89
Calcium, %	.65	.65
Phosphorus, %	.55	.55

<sup>a</sup> Supplied per kilogram of diet: retinyl acetate, 3,088 IU; cholecalciferol, 386 IU;  $\alpha$ -tocopherol acetate, 15 IU; menadione sodium bisulfite, 2.3 mg; riboflavin, 3.9 mg; d-pantothenic acid, 15.4 mg; nicacin, 23.2 mg; choline, 77.2 mg; vitamin B<sub>12</sub>, 15.4  $\mu$ g.

<sup>b</sup> Supplied per kilogram of diet: Zn (as ZnO), 110 mg; Fe (as FeSO<sub>4</sub>•H<sub>2</sub>O), 110 mg; Mn (as MnO), 22 mg; Cu (as CuSO<sub>4</sub>•5 H<sub>2</sub>O), 11 mg; I (as Ca(IO<sub>3</sub>)•H<sub>2</sub>O), .22 mg; Se (as Na<sub>2</sub>SeO<sub>3</sub>), .3 mg.

<sup>c</sup> Metabolizable energy.

either the corn-soybean meal or the lysine-supplemented diet until slaughter. Within this group, six pigs, three from each diet treatment were slaughtered on weeks four, five and six of the experiment. All pigs were individually penned in an environmentally controlled room.

Diets were corn-soybean meal-based and formulated to contain one of two crude protein percentages (16.3 or 14.3%; Table 1). All other nutrient concentrations were equal to, or in excess of, NRC (1998) requirements. During the feed restriction period gilts were fed the 16.3% CP corn-soybean meal diet. Daily feed allotments during the feed restriction period were based on each pig's maintenance energy requirement. Because nutrient densities were not adjusted during the restriction period, daily intakes of all nutrients were less than NRC (1998) requirements for growth.

Pig weights were recorded weekly during the ad libitum period and every three days during the restriction period. Feed consumption was measured weekly for the ad libitum (AL) groups and daily during the realimentation period for the restricted-realimented (RR) groups. Blood samples

were collected weekly for both feeding groups and daily during the first week of ad libitum feeding. Ultrasound scan measurements of backfat and loin area at the 10th rib were made weekly by a certified technician. Carcass measurements and organ weights were collected at slaughter. Gastrointestinal contents were removed to determine empty body weight (live weight minus gastrointestinal content weight). The right half of each carcass was ground and a subsample was obtained to determine the percentage of protein, fat, water and ash in each carcass.

Data were analyzed as a completely randomized design using the GLM procedure of SAS (1996). The main effects in the statistical model were feeding regimen (AL or RR) and diet (corn-SBM or corn-SBM + lysine). The data were analyzed within week of ad libitum eating. Therefore, comparisons were made between gilts that had ad libitum access to feed for an equal amount of time. The comparisons that were analyzed were between gilts slaughtered on weeks one and four, weeks two and five, and weeks three and six. In addition, gilts slaughtered at the start of the trial were compared to gilts slaughtered after the restriction period



**Table 2. Organ weights, carcass measurements, and carcass composition of gilts slaughtered on day 0 versus gilts slaughtered after a 21-d restriction period.**

Item	d 0 <sup>a</sup>	d 21 <sup>b</sup>	FR <sup>c</sup>
Body Weight, lb	73.32	72.77	NS
<b>Organs</b>			
Heart, lb	.36	.33	NS
Liver, lb	1.34	1.04	0.01
Kidney, lb	.32	.22	0.01
Pancreas, lb	.15	.14	NS
Lungs, lb	.90	.86	NS
Stomach, lb	.55	.49	0.05
Small intestine, lb	2.28	1.56	0.01
Large intestine, lb	1.14	1.07	NS
Mesentery, lb	.82	.64	0.05
<b>Carcass measurements</b>			
First-rib backfat, in	.33	.65	0.05
Tenth-rib backfat, in	.25	.23	NS
Last-rib backfat, in	.22	.25	NS
Last-lumbar backfat, in	.20	.13	0.05
Longissimus muscle area, in <sup>2</sup>	3.15	4.13	0.01
Carcass length, in	22.63	22.83	NS
Hot carcass weight, lb	45.18	52.46	0.01
Empty body weight, lb	71.51	71.71	NS
<b>Carcass Percentage</b>			
Protein, %	17.70	18.97	0.05
Fat, %	15.89	9.43	0.01
Water, %	62.17	67.96	0.01
Ash, %	2.18	2.56	0.01

<sup>a</sup>d 0 = gilts slaughter at the start of the trial.

<sup>b</sup>d 21 = gilts slaughter after a 21-d restriction period.

<sup>c</sup>FR = feeding regimen P-value and NS = nonsignificant effect, P > 0.10.

**Table 3. Organ weights of gilts fed a corn-soybean meal or lysine-supplemented, corn-soybean meal diet during two different feeding regimens.**

Item	Diets		Corn-soybean meal		Corn-soybean meal+lysine		P-Value <sup>b</sup>		
	Feeding regimen <sup>a</sup>		AL	RR	AL	RR	FR	D	FR x D
<b>Week 1</b>									
Body weight, lb			90.78	97.02	93.27	92.54	NS	NS	NS
Heart, lb			.40	.38	.41	.39	NS	NS	NS
Liver, lb			1.86	1.94	1.66	1.90	< 0.05	< 0.05	NS
Kidney, lb			.39	.41	.37	.33	NS	< 0.05	NS
Stomach, lb			.74	.84	.66	.82	< 0.05	< 0.05	NS
Small intestine, lb			3.00	2.76	2.75	2.69	NS	NS	NS
Large intestine, lb			1.70	1.74	1.40	1.59	NS	NS	NS
Mesentery, lb			1.09	1.29	1.14	.92	NS	NS	< 0.05
<b>Week 2</b>									
Body weight, lb			109.30	106.57	105.11	108.55	NS	NS	NS
Heart, lb			.46	.48	.43	.50	NS	NS	NS
Liver, lb			1.93	1.94	1.85	2.13	< 0.05	NS	< 0.05
Kidney, lb			.44	.45	.42	.44	NS	NS	NS
Stomach, lb			.82	.90	.76	.99	< 0.05	NS	NS
Small intestine, lb			2.56	2.77	2.48	2.86	NS	NS	NS
Large intestine, lb			1.83	1.92	1.74	1.82	NS	NS	NS
Mesentery, lb			1.23	1.16	1.27	1.29	NS	NS	NS
<b>Week 3</b>									
Body weight, lb			117.90	131.13	116.14	132.81	< 0.01	NS	NS
Heart, lb			.50	.49	.62	.53	NS	< 0.05	NS
Liver, lb			2.35	2.34	2.05	2.38	NS	NS	NS
Kidney, lb			.48	.51	.48	.50	NS	NS	NS
Stomach, lb			.93	.91	.79	.98	NS	NS	< 0.05
Small intestine, lb			2.85	2.81	2.93	2.95	NS	NS	NS
Large intestine, lb			2.00	2.11	1.95	2.14	NS	NS	NS
Mesentery, lb			1.71	1.68	1.54	1.54	NS	< 0.05	NS

<sup>a</sup>AL = ad libitum group and RR = restricted-realimentated group.

<sup>b</sup>FR = feeding regimen; D = diet; and NS = nonsignificant effect, P > 0.10.

to examine the effects of restriction on body composition. The model used was as follows:  $Y_{ijk} = \mu + \alpha_i + \beta_j + (\alpha\beta_{ij}) + e_{ijk}$  where  $Y_{ijk}$  is the observed value for a dependent variable on the  $k$ th pig ( $k = 1, 2, \dots, 46$ ),  $\mu$  is the overall mean,  $\alpha_i$  is the effect of the  $i$ th feeding regimen ( $i = 1, 2$ ),  $\beta_j$  is the effect of the  $j$ th diet ( $j = 1, 2$ ), and  $e_{ijk}$  is the random error term. In all statistical analyses, pig was the experimental unit.

## Results

Growth performance, plasma urea concentrations, and ultrasound measurement data from this experiment were reported in the 2000 Nebraska Swine Report.

Organ weights, carcass measurements, and carcass composition of the initial slaughter groups are shown in Table 2. During the restriction period, weights of the liver, kidney, stomach, and the small intestine were decreased ( $P < 0.01$ ) in the restricted gilts compared with gilts at the start of the trial. Carcass measurements showed a decrease ( $P < 0.05$ ) in last-lumbar backfat, and an increase in first-rib backfat ( $P < 0.05$ ), longissimus muscle area ( $P < 0.01$ ), and hot carcass weight ( $P < 0.01$ ) during the restriction period. During the restriction period, gilts were able to increase the percentage of protein ( $P < 0.05$ ), water ( $P < 0.01$ ), and ash ( $P < 0.01$ ). During this same period there was a decrease ( $P < 0.01$ ) in the percentage of carcass fat in the restricted gilts compared with the initial slaughter group.

Organ weights during the three weeks of ad libitum eating are shown in Table 3. Liver weight of the RR gilts increased dramatically during the first week of ad libitum feeding and was greater ( $P < 0.05$ ) than that of the AL gilts throughout the ad libitum eating period. During week one, liver weights were heavier ( $P < 0.05$ ) in gilts fed the corn-soybean meal diet versus the lysine-supplemented diet. There was a feeding regimen  $\times$  diet interaction ( $P < 0.05$ ) observed during week two of the ad libitum feeding period for liver

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weight. This interaction seems to be a result of the increased metabolic demands of the liver because of the increase in feed intake of the RR gilts fed the lysine-supplemented diet. Kidney weights were greater ( $P < 0.05$ ) in gilts fed the corn-soybean meal diet versus the lysine-supplemented diet during week one. Stomachs of the RR gilts were heavier ( $P < 0.05$ ) than those of AL gilts during weeks one and two and were heavier ( $P < 0.05$ ) in gilts fed the corn-soybean meal versus the lysine supplemented diet during week 1. There was a feeding regimen  $\times$  diet interaction ( $P < 0.05$ ) observed during week three of the ad libitum feeding period for stomach weight. This interaction seems to be a result of differences in feed intake, gilts in the AL group fed the corn-soybean meal diet consumed more feed than did gilts fed the lysine-supplemented diet, however, the opposite effect was seen in the RR group. The weight of the small intestine in the RR gilts almost doubled during the first week of ad libitum eating (Table 2 and 3), but there were no differences between feeding or diet treatments during the ad libitum eating period. Heart and large intestinal weights showed no differences between initial slaughter group, or between feeding treatments during the ad libitum eating period.

Carcass accretion of protein, fat, water, and ash are shown in Table 4. There were no differences between feeding or diet treatments for carcass fat accretion during the entire ad libitum eating period. Carcass protein, water, and ash accretion were greater ( $P < 0.05$ ) in the AL versus RR gilts during weeks one and two of the ad libitum eating period. There was no effect of

**Table 4. Carcass accretion in gilts fed a corn-soybean meal or lysine-supplemented, corn-soybean meal diet during two different feeding regimens.**

Item	Diets Feeding regimen <sup>a</sup>	Corn-soybean meal		Corn-soybean meal+lysine		P-Value <sup>b</sup>		
		AL	RR	AL	RR	FR	D	FR $\times$ D
Week 1								
Body weight, lb		90.78	97.02	93.27	92.54	NS	NS	NS
Protein, lb/d		.37	.24	.47	.23	< 0.01	NS	NS
Fat, lb/d		.41	.47	.38	.40	NS	NS	NS
Water, lb/d		1.65	.81	1.83	.95	< 0.01	NS	NS
Ash, lb/d		.06	.03	.09	.02	< 0.01	NS	NS
Week 2								
Body weight, lb		109.30	106.57	105.11	108.55	NS	NS	NS
Protein, lb/d		.30	.25	.34	.25	< 0.01	NS	NS
Fat, lb/d		.35	.42	.46	.38	NS	NS	NS
Water, lb/d		1.24	.83	1.30	.87	< 0.01	NS	NS
Ash, lb/d		.05	.03	.05	.03	< 0.01	NS	NS
Week 3								
Body weight, lb		117.90	131.13	116.14	132.81	< 0.01	NS	NS
Protein, lb/d		.28	.27	.28	.30	NS	NS	NS
Fat, lb/d		.52	.52	.44	.51	NS	NS	NS
Water, lb/d		.93	1.02	1.01	1.14	NS	NS	NS
Ash, lb/d		.04	.04	.04	.03	NS	NS	NS

<sup>a</sup>AL = ad libitum group and RR = restricted-reimplemented group.

<sup>b</sup>FR = feeding regimen; D = diet; and NS = nonsignificant effect,  $P > 0.10$ .

feeding or diet treatment during week 3 for carcass accretion of protein, water or ash.

These results indicate that organs exhibit a compensatory growth response after a period of feed restriction. This is best illustrated by the dramatic increase in organ weights during the first week of the ad libitum eating period. During the 21-day restriction period, gilts in the RR group were able to increase the protein percentage in the carcass, but during the ad libitum eating period carcass protein accretion was lower compared to the AL gilts. This decrease in carcass protein accretion and increase in organ weights during weeks one and two of the ad

libitum eating indicates that the majority of the protein intake was being used for organ growth. This may suggest that after a restriction period the amino acid concentration and/or pattern may need to be adjusted to support organ growth and carcass protein accretion. Further research is needed to explore the metabolic pathways by which pigs are able to use fat stores and deposit lean muscle tissue during a period of feed restriction.

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# Factors Affecting Small Intestine Development in Weanling Pigs

Steven J. Kitt  
Phillip S. Miller  
Austin J. Lewis<sup>1</sup>

## Summary and Implications

*The pig faces significant biological and environmental challenges after weaning. A great deal of information is available on behavior, environment, health, and nutrition of the newly weaned pig; however, newly weaned pigs still suffer a growth lag. The pig's small intestinal structure and function is altered during the days that follow weaning. As a consequence, the digestive and absorptive capacity of weanling pigs is decreased during this period and this may be partially responsible for the post weaning growth lag. Additionally, health benefits may be associated with an improved small intestinal structure and function. The goal of this article is to review some of the potential causes of changes in small intestinal structure and outline some potential nutritional modifications that have been suggested to attenuate the negative changes in small intestinal structure and function.*

## Small Intestinal Changes

The lumen (inside) lining of the small intestine is comprised of very small finger-like projections called villi. Microvilli are “micro” finger-like projections attached to the villi. The villi and microvilli function to increase surface area that augment the absorptive efficiency of the small intestine. Crypts of Lieberkuhn (or simply “crypts”) are tubular depressions found between the villi and are the source of new cells that migrate to the villi. A portion of the digestive and all of the absorptive capacity of the small intestine occurs near and around the villi

and crypts. In the weanling pig, villi atrophy (digress) and crypts undergo hyperplasia (increased number of cells). When the villi are digressed, the cells associated with the crypt attempt to begin to rebuild the villi. This is appropriate because the cells residing on the periphery of the villi originate from the crypt. This regeneration phenomenon is thought to cause a temporary (until the structure is rebuilt) decrease in digestive and absorptive capacity.

These changes may affect the growth and(or) health status of the newly weaned pig. Therefore, a means to alter the villi digression and crypt hyperplasia may be advantageous to both growth and health of newly weaned pigs. The remainder of this article will highlight several factors known to be associated with the changes in intestinal morphology and function and will outline some proposed nutritional modifications that may improve the intestinal maladies following weaning.

## Factors Associated With Small Intestinal Changes

### *Pathogenic Bacteria*

Enteric bacteria colonized in the small intestine have a profound influence on the structure and digestive/absorptive capacity. Typically haemolytic *E. coli* is one of the most prevalent bacterial species and these bacteria are suggested to be a causative factor of villi atrophy and crypt hyperplasia. In experimental conditions, the presence of *E. coli* resulted in shorter villi, deeper crypts, and reduced carbohydrate digestive enzyme activity. Additionally, the absorption of fluid and electrolytes has shown to be decreased in pigs inoculated with *E. coli*. The presence of pathogenic bacteria in the

small intestine does affect villus height and crypt depth (and associated digestion/absorption); however, this does not entirely explain the decreased nutrient absorption in weanling pigs. For example, Figure 1 illustrates the effect of weaning on both *E. coli*-inoculated and control pigs. Clearly, the *E. coli*-inoculated pigs absorbed less fluid; however, decreased absorption was also observed for weaned animals compared to unweaned animals. Additionally, changing the diet of germ-free pigs from milk to a dry diet has been shown to decrease villus height, crypt depth, and carbohydrate digestive enzyme activities, suggesting that enteric pathogens are not the only causative factor changing gut morphology. Moreover, it is not known whether the presence of pathogens in the small intestine is a cause or effect of changes in small intestinal morphology.

### *Stressors of Weaning*

Because the lag in performance and changes in gut morphology occur after weaning, some researchers have suggested that the psychological stress of weaning (e.g., displacement from sow, new environment, new pen mates) causes gut morphological changes. This theory is plausible, but not all of the observed changes in small intestinal morphology have been associated with weaning stress. For example, there seems to be no relationship between plasma cortisol (an indicator of acute stress) concentrations and poor growth rate and (or) decreased villus height in weaned pigs. Also, research suggests that weaned pigs fed sow milk have similar small intestinal morphology as their unweaned counterparts. This implies that the act of weaning may be less important than changes in diet-related factors. However, it should

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be noted that the proposed theory of psychological stress at weaning (contributing to changes in small intestinal morphology) may be confounded with other factors at weaning (e.g., pathogen exposure, diet, low feed intake, etc.). Therefore, it is difficult to sort out the potential of psychological stress contributing to the changes in gut morphology.

#### Feed Intake and Adaptation to Solid Food

Decreased feed intake is usually observed in newly weaned pigs. To illustrate this point, Figure 2 summarizes several data sets showing typical voluntary energy intake of newly weaned pigs. Note that metabolizable energy intake is not equal to preweaning intake until at least 10 days postweaning. Obviously, pigs need to consume nutrients to grow, but these changes in nutrient intake may also contribute directly to changes in morphology and function of the small intestine. The presence of feed in the small intestine is a potent stimulus of cell division and growth. There is a positive relationship between dry matter intake and villus height. Similarly, it has been shown that restricted feeding and intravenous feeding results in villus atrophy. This suggests that “feeding” the small intestine is important in maintaining a viable small intestine morphology and function. Additionally, other factors associated with gastrointestinal changes are often confounded with the occurrence of low feed intakes.

#### Dietary Factors

Specific components of certain feedstuffs may contribute to the negative changes in small intestinal morphology. There is general agreement that soybean meal causes negative changes in small intestinal morphology. Proteins found in soybean meal have been shown to cause an immunogenic reaction or a “gut allergy” in pigs exposed to soybean meal. This

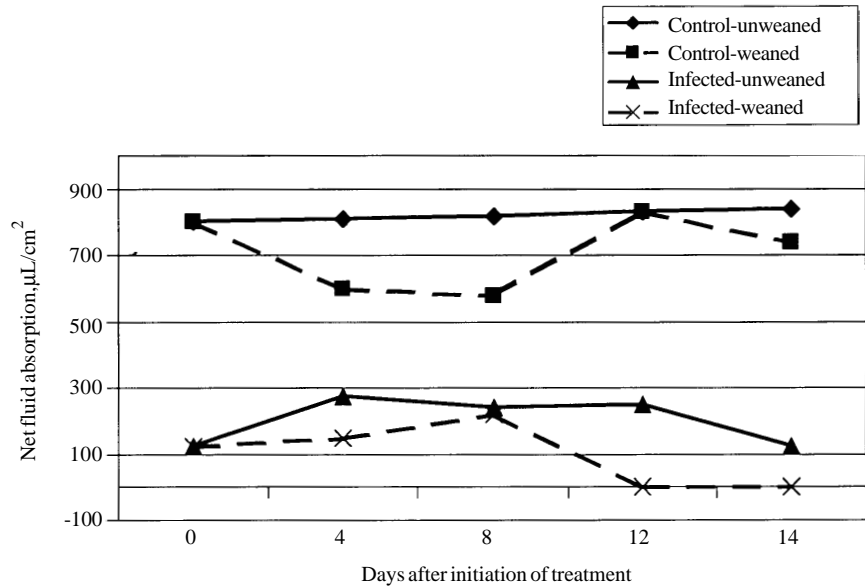


Figure 1. Fluid absorption of control and infected, and weaned and unweaned pigs.

Res. Vet. Sci. 56:379-385.

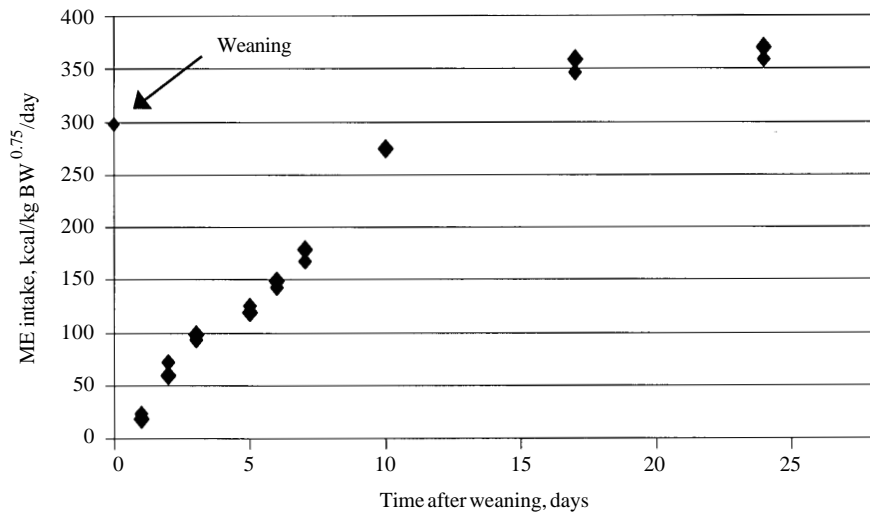


Figure 2. Energy intake of weaned pigs.

Livest. Prod. Sci. 38:79-90.

“gut allergy” has been associated with villus atrophy. For this reason, nutritionists often limit the amount of soybean meal in weaned pig diets. However, some debate exists whether the initial research (linking soybean meal and villus atrophy) is confounded by low feed intakes after weaning because low feed intakes may contribute to the atrophy of villus and therefore a depression in growth. Other antinutritional

compounds such as lectins and tannins have been implicated as factors related to villus atrophy after weaning.

Sow milk (and colostrum) is a nearly perfect food for young pigs, therefore the compounds and nutrients in sow milk serve as excellent references as we formulate dry feed diets for weanling pigs. For example, it has been known for some time that weanling pigs require a high dietary con-



centration of lactose because sow milk contains high concentrations of lactose. Other nutrients and growth factors/hormones found in milk could be responsible for maintaining the integrity of the small intestinal morphology and function. The lack of these compounds or reduction in their concentration could be responsible for the changes observed in the small intestine after weaning.

Hormones found in sow milk may contribute to small intestine morphology integrity. For these hormones to influence the integrity of the small intestine, at least four criteria must be met. First, the hormone of interest must be present in sow milk. Secondly, the receptor (i.e., site where the hormone binds to initiate a physiological change) for the hormone must be present in the lumen of the small intestine. Thirdly, the presence of the hormone must elicit a physiological response (e.g., increased villus height, increased enzyme activity, etc.). Lastly, the digestive processes must not alter the hormone.

Several hormones have been reported to influence small intestinal morphology. Epidermal growth factor (EGF), insulin-like growth factors (IGF-I and IGF-II), and insulin are hormones that influence growth of tissues, including the gastrointestinal tract. All of these hormones have been found in sow milk. These hormones seem to have a positive influence on the small intestinal morphology. Additionally, research on investigating the resistance of the hormones to digestive processes has been initiated. For example, recent research suggests that one-half to two-thirds of the EGF exposed to weanling pig digestion is still intact, and a significant portion of that may be biologically active at the site of the small intestine. There is little doubt that these hormones are important for gut development while pigs are suckling, and they may be useful for implementing in postweaning pig diets in the future.

Other compounds present in milk have been associated with improved gastrointestinal morphology. Polyamines (e.g., putrescine, spermine, spermidine) are compounds that are important for cellular proliferation and differentiation. Both the enzyme responsible for synthesizing polyamines and polyamines themselves have been shown to increase in concentration before and during an increased proliferation of cells. There has been limited discussion about whether the pig synthesizes an adequate supply of polyamines. Therefore, some researchers have attempted to answer whether supplementing polyamines in the diet improves gastrointestinal morphology and (or) growth. In chicks, spermidine supplementation seems to improve growth; however, large doses may be toxic. Young swine and preruminant calves have shown some improvement in small intestinal proliferation when fed supplemental polyamines. Polyamines are natural compounds and typical feedstuffs contain polyamines; however, the concentration of these compounds in feedstuffs is unknown and (or) their effects on pig metabolism have not been extensively studied. Considering the limited amount of data, more research regarding whether dietary polyamines improve small intestinal morphology is needed before any conclusions can be drawn.

Recently, glutamine has received attention as a modifier of gastrointestinal growth. Glutamine is considered a nonessential amino acid for swine. However, glutamine has been recognized by the human health community to help maintain gastrointestinal growth during intravenous feeding and after gastrointestinal surgery. Rapidly dividing cells, including the absorptive and immune cells of the small intestine, prefer glutamine (compared to glucose) as an energy source. Additionally, it seems that free (unbound to protein) glutamine is the most abundant amino acid in sow milk, particularly in late lactation (tested on day 22

and 29). The addition of 1% crystalline glutamine to a corn-soybean meal diet has been reported to partially prevent villous atrophy in the jejunum (mid portion of the small intestine) on the seventh day after weaning. Other recent research has confirmed that supplemental glutamine improves small intestinal morphology in pigs. However, previous research used crystalline glutamine. This form of glutamine is expensive and basically unavailable to the feed industry at this time. Therefore, there is a need to identify whether glutamine from intact protein from typical feedstuffs (e.g., soybean meal, spray-dried porcine plasma, fish meal, dried skim milk, etc.) is as effective in stimulating a response as is crystalline glutamine. Glutamine appears to be an effective way to help optimize the growth of the small intestine after weaning; however, a more applicable method to analyze and include glutamine in the diet must be pursued.

## Conclusions

Many of the factors that are associated with changes in the small intestinal morphology may be inter-related. Understanding how these factors alter the growth of the small intestine of weanling pigs may lead to developments that improve growth and (or) pig health. This is particularly important with increasing pressure to limit the use of antibiotics/growth promotants in pig diets. Future experiments at the University of Nebraska will be investigating the importance of the integrity of the gastrointestinal tract relative to overall growth and potentially develop methods to improve the integrity of the weaned pig's small intestine after weaning.

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<sup>1</sup>Steven J. Kitt is a graduate student, Phillip S. Miller is an associate professor, and Austin J. Lewis is a professor in the Department of Animal Science.



# Two-stage Selection for Ovulation Rate and Litter Size in Swine — An Effective Procedure to Increase Reproductive Rate

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## Summary and Implications

Litter size continues to be an important economic variable in pig production. Two determinants of litter size are ovulation rate and uterine capacity, where uterine capacity is defined as the maximum number of pigs a female can carry to parturition. When the number of fertilized ova exceeds uterine capacity embryo/fetal losses during gestation reduce litter size to that sow's uterine capacity. This experiment tested whether litter size can be increased by direct selection for ovulation rate and uterine capacity. It was accomplished by selecting for ovulation rate (OR) and number of fully formed pigs at birth (FF) in two stages. All gilts from 50% of the largest litters were selected in Stage 1, and then 50% of these gilts were selected on OR in Stage 2. Litter size at birth in gilts with high ovulation rate was considered a measure of their uterine capacity. Selected gilts were mated to boars selected from the upper one third of the litters for FF. Selection in each of two lines for eight generations was practiced. One of the lines, designated IOL, was started from a line previously selected for increased ovulation rate and embryonic survival,

and thus began with a base of high litter size. The other line, designated COL, began from an unselected population. Responses in these lines were compared to those in a randomly selected control line (Line C). The genetic increases per generation for OR and FF were  $.27 \pm .07$  ova/generation ( $P < .01$ ) and  $.35 \pm .06$  pigs/generation ( $P < .01$ ) in line IOL and  $.30 \pm .06$  ova/generation ( $P < .01$ ) and  $.29 \pm .05$  pigs/generation ( $P < .01$ ) in line COL. In previous experiments, only 25 to 50% of the increase in OR was realized as a pig at birth. In this experiment OR and FF increased equally, indicating that increased litter size resulted in approximately equal increases in both ovulation rate and uterine capacity. Furthermore, the responses were similar in both lines indicating that the selection procedure will work effectively in populations with varying base levels of ovulation rate, uterine capacity and litter size. The selection procedure is still not practical in most breeding programs because the surgical procedure of laparotomy or laparoscopy is used for accurate measurement of OR. However, if a noninvasive procedure to measure OR is developed, this procedure can be effectively applied in industry breeding programs. Other changes that occurred as a result of this selection were decreased age at puberty, increased number of pigs born alive, and increased number of still-

born and mummified pigs. Two-stage selection for FF and OR is an effective procedure to improve litter size in swine.

## Introduction

Ways to enhance selection response in litter size is a goal of pig breeders because litter size is an important economic variable in pig production. In females with high ovulation rate, litter size at birth is expected to closely represent uterine capacity. The hypothesis tested in this experiment was that selection with emphasis on ovulation rate (OR) and uterine capacity would cause litter size to increase. The objective was to quantify direct and correlated responses in ovulation rate, litter size and other production traits to two-stage selection for these traits.

## Animals and Selection Procedure

Three genetic lines were used. Selection lines IOL and COL were derived from the Index and Control lines, respectively, developed at the University of Nebraska. The population is a composite developed from a Large White-Landrace base. Line IOL originated from the Index line that had previously been selected eight generations for increased ovulation rate and embryonic survival. Line COL was derived from the randomly selected



**Table 1. Number of observations (n) and unadjusted phenotypic means for ovulation rate (ova) and number of fully formed pigs by line-generation**

Generation	Line IOL		Line COL		Line C	
	n	Mean	n	Mean	n	Mean
	Ovulation rate					
1	57	17.1	66	12.7	66	12.7
2 <sup>a</sup>	101	17.3	92	13.0	—	—
3 <sup>a</sup>	84	17.4	96	13.3	—	—
4 <sup>b</sup>	—	—	—	—	—	—
5	83	17.0	92	13.7	35	12.5
6	96	18.5	88	14.2	52	13.0
7	87	18.4	97	14.7	41	12.3
8	90	19.0	99	15.1	51	12.9
	Number of fully formed pigs					
0 <sup>c</sup>	42	13.4(11.3) <sup>c</sup>	36	9.8(8.1) <sup>c</sup>	36	9.8(8.1) <sup>c</sup>
1 <sup>d</sup>	44	11.1	38	10.2	41	9.4
2	52	11.8	56	10.3	36	9.0
3	43	12.2	45	9.8	36	9.3
4	43	11.8	42	10.8	45	8.8
5	41	12.7	43	10.4	39	8.5
6	48	13.4	44	11.5	35	8.9
7	43	13.4	45	11.8	37	9.6
8	42	12.5	42	10.3	35	7.4

<sup>a</sup>Ovulation rate was not measured in Generations 2 and 3 in line C.

<sup>b</sup>Ovulation rate was not measured in Generation 4 gilts.

<sup>c</sup>Mean number of fully formed pigs in 2nd parity sows. Means for these same sows at first parity are in parenthesis.

<sup>d</sup>Beginning in Generation 1 number of fully formed pigs was recorded in gilts.

control line (line C). Line IOL started from a base with greater ovulation rate and litter size than Line COL. Line C was continued with random selection to serve as a control for both selection lines.

Lines IOL and COL underwent eight generations of two-stage selection. In Stage 1 all gilts born in 50% of the largest litters were selected. Laparotomy was performed on these gilts ten days after their second estrus to measure OR by counting number of corpora lutea. Approximately 50% of these gilts were selected on OR in Stage 2. Boars in each line were selected from the highest ranking 15 litters for number of fully formed pigs (FF). Although we did not know each gilt's uterine capacity, it was believed that the number of fertilized ova in gilts selected on OR exceeded their uterine capacity. Then during gestation, embryo/fetal losses due to insufficient uterine capacity reduced the number of fetuses for that sow. Thus, litter size at birth was considered a

measure of uterine capacity in the gilts selected on OR. In line C at least one gilt per litter and one boar per half-sib family were randomly selected. Each line had approximately 40 litters per generation.

#### Traits Measured and Analyzed

Number of corpora lutea (OR) present during the second estrous cycle in gilts of lines IOL and COL was measured in all generations except the third one. In line C gilts, OR was recorded only in the first and in the last four generations. Prenatal loss (PL) was calculated as the difference between OR and FF. Number of pigs born alive (BA), number of stillborn pigs (SB), number of mummified pigs (M), and individual birth weight (BW) and litter birth weight (LBW) were recorded at birth. Number of pigs weaned (NW), individual weaning weight (IWW), and litter weaning weight (LWW) were recorded at weaning. Age at puberty (AP) was recorded when the

gilt first stood immobile to back pressure in the presence of a boar. Weight of gilts was measured on average at 125 d of age (W1). Weight of boars and gilts was recorded approximately at 178 d of age (W2). Backfat was recorded when average weight of pigs in the pen was approximately 209 lb.

#### Data Analysis

Genetic parameters and direct and correlated responses were estimated with an animal model that calculated the estimated breeding values for each pig. Depending on the trait, models included the fixed effects of generation and sex, and the random additive direct and maternal genetic effects. Direct and correlated responses were estimated with regressions of estimated breeding values on generation number to estimate responses per generation.

## Results and Discussion

### Generation means

Mean ovulation rate and number of fully formed pigs per litter are shown by line and generation in Table 1, and means for number of live, stillborn and mummified pigs are in Table 2. Development of these lines from the Index selection lines began with litters from second parity sows. Thus, litter size data for Generation 0 females are for these second parity sows. Data for these same females as first parity sows are in parentheses. All litter size data for Generations 1 through 8 are for first parity sows. Ovulation rate and number of fully formed pigs increased steadily in both lines IOL and COL, but remained relatively unchanged in line C. Number of live pigs per litter increased in the selection lines, but both number of stillborn and mummified pigs per litter also increased.

### Genetic trends

Estimated genetic trends in ovulation rate and litter size are illustrated

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in Figure 1. Genetic responses (Table 3) for number of pigs per litter were substantial in both line IOL (.35±.06 pigs/generation, P<.01) and line COL (.29±.05 pigs/generation, P<.01). Genetic responses for ovulation rate were .27±.07 ova/generation (P<.01) in line IOL and .30±.06 ova/generation (P<.01) in line COL.

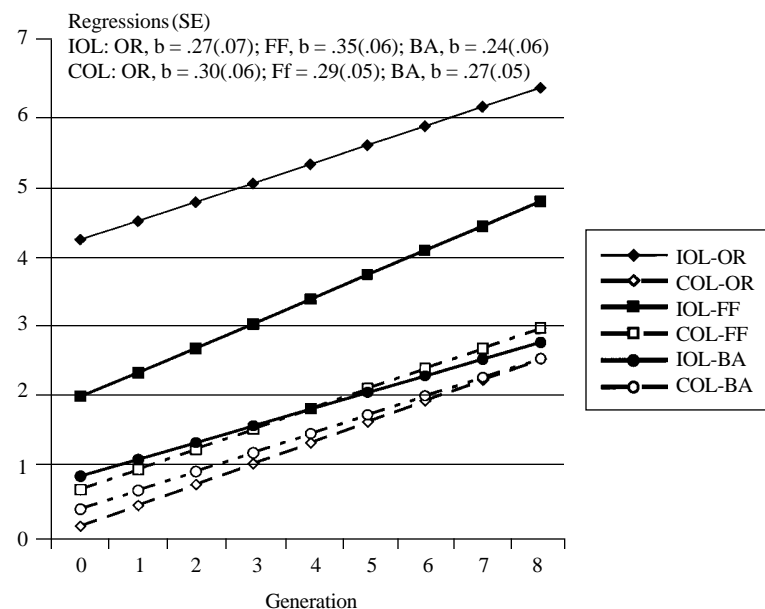
In line COL, number of pigs per litter increased 97% as rapidly as ovulation rate. In line IOL, litter size increased 130% compared with ovulation rate, although this greater rate of response in line IOL was not significantly more than that in line COL. The control line from which line COL was derived had no antecedents of selection, neither for component traits nor directly for litter size. Line IOL already had increased ovulation rate (4.2 ova) and litter size (2.0 fully formed pigs) at Generation 0 of two stage selection due to previous selection. Responses for ovulation rate, number of fully formed pigs, and number born alive were comparable in both selection lines (Figure 1). Thus, rate of change in ovulation rate and uterine capacity did not depend on mean genetic level of the line. Similar responses can be expected in other genetic lines.

The estimated total genetic responses at Generation 8 in ovulation rate in line COL relative to line C were 2.32±.74 ova and 2.16±.57 fully formed pigs at birth. Responses in line IOL relative to line C were 2.08±.79 ova and 2.64±.62 fully formed pigs. Responses at Generation 8 in number of live pigs per litter were 1.84±.57 pigs in line COL and 1.60±.62 pigs in line IOL. These comparisons suggest that at Generation 0, uterine capacity was more limiting in line IOL than in line COL because the number of fully formed pigs increased more rapidly than ovulation rate. If that was true, selection for fully formed pigs in line IOL should have resulted in a greater increase in uterine capacity than selection for number of fully formed pigs in line COL. Workers at USDA MARC have clearly shown that increased litter size will occur from changes in the most limit-

**Table 2. Number of observations (n) and unadjusted phenotypic means for number of pigs born alive, stillborn, and mummified by line-generation.**

Generation	Line IOL		Line COL		Line C	
	n	Mean	n	Mean	n	Mean
Number of pigs born alive						
0	42	11.7(9.2) <sup>a</sup>	36	9.1(7.6) <sup>a</sup>	36	9.1(7.6) <sup>a</sup>
1	44	9.6	38	9.7	41	9.0
2	52	10.8	56	9.7	36	8.4
3	43	10.2	45	9.1	36	8.9
4	43	9.7	42	9.8	45	8.1
5	41	11.0	43	9.6	39	8.1
6	48	9.5	44	10.6	35	8.6
7	43	11.1	45	10.8	37	9.0
8	42	10.6	42	9.7	35	6.6
Number of stillborn pigs						
0	42	1.7(2.0) <sup>a</sup>	36	0.7(0.5) <sup>a</sup>	36	0.7(0.5) <sup>a</sup>
1	44	1.4	38	0.5	41	0.3
2	52	1.0	56	0.6	36	0.6
3	43	2.0	45	0.7	36	0.4
4	43	2.1	42	0.9	45	0.7
5	41	1.7	43	0.8	39	0.4
6	48	3.9	44	1.0	35	0.6
7	43	2.3	45	1.1	37	0.6
8	42	1.8	42	0.6	35	0.6
Number of mummified pigs						
0	42	0.3(1.7) <sup>a</sup>	36	0.2(.5) <sup>a</sup>	36	0.2(0.5) <sup>a</sup>
1	44	0.3	38	0.2	41	0.2
2	52	0.4	56	0.3	36	0.2
3	43	0.5	45	0.4	36	0.1
4	43	0.3	42	0.5	45	0.2
5	41	0.5	43	0.6	39	0.2
6	48	0.6	44	0.5	35	0.2
7	43	0.5	45	0.5	37	0.2
8	42	1.2	42	1.2	35	1.0

<sup>a</sup>Generation means are for 2nd parity sows. Means for these same sows at their first parity are in parenthesis.



**Figure 1. Genetic changes in lines IOL (solid lines) and COL (dashed lines) relative to line C for ovulation rate (OR) and number of fully formed (FF) and live (BA) pigs per litter.**



**Table 3. Coefficients (b) and standard errors (SE) of regressions of mean estimated breeding value on generation number by trait and line**

Trait <sup>a</sup>	Line IOL		Line COL		Line C	
	b	SE	b	SE	b	SE
OR	0.27**	0.07	0.30**	0.06	0.01	0.07
FF	0.35**	0.06	0.29**	0.05	0.02	0.05
BA	0.24**	0.06	0.27**	0.05	0.04	0.05
M	0.04*	0.01	0.03*	0.01	0.00	0.01
SB	0.10**	0.03	0.03	0.02	0.01	0.02
AP	-2.37*	0.70	-2.03*	0.67	-0.00	0.72
BF, in	0.004	0.003	0.12**	0.003	-0.002	0.003
BW, lb	0.024**	0.007	0.013†	0.004	-0.002	0.004
LBW, lb	0.80**	0.15	0.42*	0.13	-0.07	0.13
LWW, lb	0.02	0.33	-0.09	0.31	0.13	0.29
NW	0.07	0.04	0.15**	0.03	-0.01	0.03
PL	0.15*	0.05	0.18**	0.05	0.00	0.05
W1, lb	1.19*	0.44	-0.26	0.42	-0.18	0.40
W2, lb	2.47**	0.53	-0.29	0.51	-0.40	0.53
IWW, lb	0.07†	0.02	-0.00	0.02	0.00	0.02

<sup>a</sup>OR=ovulation rate, FF=number of fully formed pigs, BA=number of pigs born alive, M=number of mummified pigs, SB=number of stillborn pigs, AP=age at puberty, BF=backfat thickness at 209.5 lb., BW=pig birth weight, LBW=litter birth weight, LWW=litter weaning weight adjusted for age weaned and to a standard number nursed, NW=number weaned per litter adjusted for age weaned and to a standard number nursed, PL=prenatal loss, W1=weight at 125 d, W2=weight at 178 d, and IWW=pig weaning weight.

\*\* P<.01, \* P<.05, † P<.10.

**Table 4. Estimates of heritabilities ( $h^2$ ), phenotypic variances ( $\sigma^2$ ), genetic correlations ( $r_g$ ), and phenotypic correlations ( $r_p$ ).**

Trait <sup>a</sup>	$h^2$	$\sigma^2$	$r_g$		$r_p$		
			OR	FF	OR	FF	
OR <sup>b</sup>	0.42±0.06	8.29	—	—	—	—	
FF	0.18±0.08	10.76	0.52	—	.16	—	
BA	0.23±0.06	9.81	0.14	0.83	.05	0.88	
M	0.17±0.05	0.80	-0.11	0.79	.01	-0.08	
SB	0.29±0.05	2.54	0.62	0.20	.23	0.33	
AP	0.73±0.05	675.36	0.07	-0.41	.07	-0.12	
BF, in	0.49±0.04	.018	-0.09	0.24	-.07	0.03	
BW, lb	D <sup>b</sup>	0.04±0.03	0.44	0.22	.11	-0.05	
	M <sup>b</sup>	0.43±0.03	—	-0.26	-0.95	—	
LBW, lb	D	0.30±0.12	56.7	0.40	0.73	.08	0.85
	M	0.04±0.06	—	-0.14	0.19	—	—
LWW, lb	0.16±0.05	355.7	-0.24	0.06	-.07	-0.04	
NW	0.24±0.06	4.54	-0.22	0.62	-.11	0.34	
PL	0.12±0.09	14.83	0.83	-0.04	.59	-0.69	
W1, lb	D	0.36±0.10	359.3	0.08	-0.03	.12	0.03
	M	0.21±0.05	—	-0.01	-0.37	—	—
W2, lb	0.58±0.04	597.5	0.02	-0.05	.14	0.07	
IWW, lb	D	0.15±0.04	3.89	-0.18	0.18	.06	0.04
	M	0.25±0.03	—	0.11	-0.51	—	—

<sup>a</sup>See Table 3 for definition of traits.

<sup>b</sup>D=direct heritability, M=maternal heritability; heritability is direct for traits without D or M designation.

ing component and that when ovulation rate and litter size are in balance, changes in litter size will occur from uniform changes in both traits (Bennett and Leymaster, 1990, J. of Anim. Sci. 68:969). Our results are consistent with their findings.

In previous work at Nebraska (Cassady et al., 1999, J. Anim. Sci. 78:1430), we found that selection for increased plasma concentration of FSH can be used as an indirect selection criteria for ovulation rate. Although the genetic correlation between plasma

FSH and ovulation rate was only moderate, indirect selection was nearly as effective (93%) in changing ovulation rate as direct selection because plasma concentration of FSH can be measured in both sexes. In our experiment, two-stage selection for ovulation rate and number of fully formed pigs was effective because litter size in gilts with increased ovulation rate was a good measure of uterine capacity. However, this procedure still requires laparotomy to record ovulation rate, a procedure that may not be practical in most breeding herds. A strategy to improve litter size could be to select for uterine capacity through litter size in a first stage and on plasma concentration of FSH in a second stage. Thus, the difficult task of measuring ovulation rate by surgical procedures is avoided. The efficacy of this selection procedure to enhance rate of response in litter size has not been tested directly.

#### Genetic parameters

Estimates of genetic parameters (heritabilities and genetic correlations) are needed to develop multi-trait selection programs. Heritability is the relative contribution of genetic effects (the heritable component) to total phenotypic variation that is due to both genetic and environmental effects. This heritable component of variation can be due to genes of the pig in which the trait was measured (direct heritability) or to genes of the dam for maternal effects on the pig's performance (maternal heritability). For some traits, such as ovulation rate and backfat thickness, the pig's own genes are responsible for genetic differences among animals and there is almost no effect of genes of the dam. But for other traits, such as pig birth and weaning weight, the pig's record is due to the effect of its own genes and to the effect of its dam on its development. Some of the dam's effect is due to her genes and is heritable.

Knowledge of the relative value of these heritabilities allows breeders to

(Continued on next page)





predict the response expected from selection for a trait. However, single-trait selection is almost never practiced because many traits contribute to economics of pig production. Therefore, it is also important to know genetic and phenotypic correlations among traits to predict correlated responses in other traits and to develop selection indexes to jointly improve all economic traits. Positive genetic correlations mean that selection for one trait will cause the other trait to increase in value while a negative correlation means that selection to improve one trait will cause the other trait to decrease. The strength of the association is determined by how close these correlations are to 1 or -1. When values of correlations are undesirable, such that single-trait selection to improve one trait will cause an undesirable change in another trait, selection indexes can be constructed to simultaneously improve both traits.

Estimates of genetic and phenotypic correlations and direct ( $h_d^2$ ) and maternal ( $h_m^2$ ) heritabilities are presented in Table 4. Most of the traits had considerable genetic variation. Direct heritability estimates ranged from  $.04 \pm .03$  for birth weight to  $.73 \pm .05$  for age at puberty. Maternal heritability estimates ranged from  $.04 \pm .06$  for litter birth weight to  $.43 \pm .03$  for individual pig birth weight.

The important genetic correlations in this study are those between ovulation rate and number of fully formed pigs, with other production traits. Many of these correlations were close to zero,

indicating at most very weak associations with ovulation rate and litter size.

Correlated responses per generation in the other traits measured were also estimated. Averaged across selection lines, number of mummified pigs per litter increased by .035 per generation and number of stillborn pigs per litter increased by .065 per generation ( $P < .05$ ). These responses explain the somewhat lower response in number of live pigs per litter (average of .255 across lines) compared with number of fully formed pigs per litter (average of .32). The increase in number of mummified pigs occurred because of its genetic correlation of .79 with number of fully formed pigs. The correlation between ovulation rate and mummified pigs was very low. On the other hand, the increase in number of stillborn pigs occurred because of its moderately high correlation of .62 with ovulation rate, although it was also positively correlated with number of fully formed pigs.

Prenatal loss, ova not represented by a pig at birth, increased at the rate of .165 embryos/fetuses per generation ( $P < .05$ ). It was due entirely to its high genetic correlation (.83) with ovulation rate. Age at puberty decreased at the rate of 2.2 days per generation.

The selection practiced in this experiment significantly increased ovulation rate and litter size. Associated with these changes were greater numbers of stillborn and mummified pigs per litter. However, the increase in total born was sufficiently large to

offset these changes so number of live pigs per litter increased significantly. The correlated decrease in age at puberty was a desirable change as there are economic benefits to pork producers from decreased age at puberty in gilts. The selection applied did not cause significant correlated responses in other production traits.

## Conclusions

Two-stage selection was effective in improving ovulation rate and litter size. Approximately 97% of the increase in ovulation rate was realized as more pigs in line COL. In line IOL, previously selected for increased OR, litter size increased 130% more than OR, although the extra rate of response was not significant. Two-stage selection can be used to improve litter size in populations varying greatly in ovulation rate and litter size. Application would be enhanced by a non-invasive procedure to record ovulation rate. Because number of mummified and stillborn pigs increased along with increased litter size, selection criteria to increase litter size should include number and/or weight of live pigs rather than number of fully formed pigs at birth.

<sup>1</sup>Agustín Ruíz-Flores was a graduate student in animal genetics and is now a professor at the University of Chapingo, Mexico. Rodger Johnson is Professor of Animal Science.





# Supreme Court Rules in Progress Pig Case

J. David Aiken<sup>1</sup>

Article 8 §12 of the Nebraska constitution (Initiative 300) establishes several requirements for corporations to legally qualify as family farm or ranch corporations. Under one provision, a majority of the family farm or ranch corporation's shareholders must be family members, "at least one of whom is a person residing on or actively engaged in the day-to-day labor and management of the farm or ranch." In *Hall v Progress Pig Inc.*, 259 Neb 407 (May 12, 2000) (*Progress Pig II*) the Nebraska Supreme Court ruled that where no family member resides on the farm or ranch, a family member must perform daily physical labor on the farm or ranch for the corporation to legally qualify as a family farm or ranch corporation.

Progress Pig Inc. is an Otoe county farrow-to-finish swine operation, with David Zahn the sole shareholder. Zahn, who lives on a farm three miles from the Progress Pig site, handles the operation's finance, management and marketing and works with production consultants. The Progress Pig production manager and other employees care for the swine. Zahn was physically onsite one to three days per week.

Zahn contended that the I300 daily labor requirement included production activities in addition to physical labor, such as bookkeeping, marketing, etc. The district court judge concluded that Zahn did provide labor and management for the farming operation, but ruled that Zahn's labor was insufficient to qualify as the *daily* labor and management required by I300. The Supreme Court, in contrast, ruled that Zahn's activities were primarily management, and that he provided only minimal physical labor (less than one hour per month). The Court ruled that Zahn did not provide the daily labor required for non-resident corporate owners by I300.

Under I300, Zahn will have to begin providing daily physical labor at the swine facility, sell the corporation within two years, or restructure the operation as a sole proprietorship or general partnership. If Zahn could prove that he had *previously* met the daily labor and management requirement and therefore qualified for family farm corporation status, Zahn might now qualify for the 50-year requalification provision under I300 so long as his family retained a majority interest in the corporation.

The district court judge noted that daily labor requirements would vary depending on whether the farm was a crop operation or a livestock operation. Livestock would require daily care, while crop operations might require physical labor only seasonally (e.g. at planting or harvesting). This issue was not addressed by the Supreme Court. However, future litigation seems inevitable regarding whether a non-resident corporate owner has provided sufficient daily physical labor to qualify for family farm corporation status, particularly where an older farmer is phasing out his or her physical labor contribution to the operation.

*Progress Pig II* was an important victory for family farm proponents. The lawsuit was originally filed in 1993, and plaintiffs (who include leaders of the Farmers Union and the Women Involved in Farm Economics) won an important procedural victory when the Nebraska Supreme Court ruled in *Hall v Progress Pig Inc.*, 254 Neb 150, 575 NW2d 369 (1998) (*Progress Pig I*) that the farmer-plaintiffs could enforce I300 under its citizen suit provision even after the county attorney had declined to bring suit. Nebraska Attorney General Stenberg earlier disqualified his office in the case as he had prepared incorporation documents for Progress Pig Inc. while in private practice law prior to his election.

*Progress Pig II* has important implications particularly for swine production in Nebraska, where family farm corporate owners providing management and non-family employees providing the physical labor is common. The owners of these operations face the same choices as Mr. Zahn.

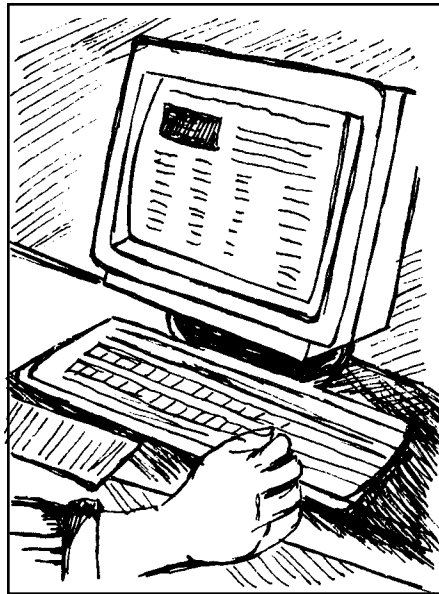
*Family farm estate planning implications.* Often families may incorporate the farm or ranch when the parents finally consult an attorney to establish their estate plan. In many cases this may not occur until the parents are getting older. One estate planning recommendation stemming from *Progress Pig II* is that if the family wishes to establish a family farm corporation, it should do so while the parents (i.e. the current operators) can still meet the I300 family farm requirements by either residing on the farm or else by providing daily labor and management. If, for example the parents move off the farm, then they (or other family members) must meet the daily labor and management requirement (or else move onto the farm) in order to qualify for family farm corporation status under I300. And, the older the parents become, the less likely it is that they will be providing daily labor and management. So the bottom line is that families should establish a family farm or ranch corporation while the parents either reside on the farm or actively participate in daily labor and management. Once a family farm corporation has been legally established, the family will then have a 50-year grace period to requalify if they fall out of compliance with I300 (e.g. when the parents move into town). However, if the family cannot initially qualify under I300 when the family farm corporation is first established, they may have difficulty doing so (if they can at all) later on.

<sup>1</sup>J. David Aiken is a water and ag law specialist in the agricultural economics department.

# Explanation of Statistics Used in This Report

Pigs treated alike vary in performance due to their different genetic makeup and to environmental effect we cannot completely control. When a group of pigs is randomly allotted to treatments it is nearly impossible to get an “equal” group of pigs on each treatment. The natural variability among pigs and the number of pigs per treatment determine the expected variation among treatment groups due to random sampling.

At the end of an experiment, the experimenter must decide whether observed treatment differences are due to “real” effects of the treatments or to random differences due to the sample of pigs assigned to each treatment. Statistics are a tool used to aid in this decision. They are used to calculate the probability that observed differences between treatments were caused by the luck of the draw when pigs were assigned to treatments. The lower this probability, the greater confidence we have that “real” treatment effects exist. In fact when this probability is less than .05 (denoted  $P < .05$  in the articles), there is less than a 5% chance (less than 1 in 20) that observed treatment differences were due to random sampling. The conclusion then is that the treatment effects are “real” and caused different performance for pigs on each treatment. But bear in mind that if the experimenter obtained this result in each of 100 experiments, 5 differences would be declared to be “real” when they were really due to chance. Sometimes the probability value calculated from a statistical analysis is  $P < .01$ . Now the chance that random




sampling of pigs caused observed treatment differences is less than 1 in 100. Evidence for real treatment differences is very strong.

It is commonplace to say differences are significant when  $P < .05$ , and highly significant when  $P < .01$ . However, P values can range anywhere between 0 and 1. Some researchers say that there is a tendency that real treatment differences exist when the value of P is between .05 and .10. Tendency is used because we are not as confident that differences are real. The chance that random sampling caused the observed differences is between 1 in 10 and 1 in 20.

Sometimes researchers report **standard errors of means (SEM)** or **standard errors (SE)**. These are calculated from the measure of variabil-

ity and the number of pigs in the treatment. A treatment mean may be given as  $11 \pm .8$ . The 11 is the mean and the .8 is the SEM. The SEM or SE is added and subtracted from the treatment mean to give a range. If the same treatments were applied to an unlimited number of animals the probability is .68 (1 = complete certainty) that their mean would be in this range. In the example the range is 10.2 to 11.8.

Some researchers report **linear (L)** and **quadratic (Q)** responses to treatments. These effects are tested when the experimenter used increasing increments of a factor as treatments. Examples are increasing amounts of dietary lysine or energy, or increasing ages or weights when measurements are made. The L and Q terms describe the shape of a line drawn to describe treatment means. A straight line is linear and a curved line is quadratic. For example, if finishing pigs were fed diets containing .6, .7, and .8% lysine gained 1.6, 1.8 and 2.0 lb/day, respectively we would describe the response to lysine as linear. In contrast, if the daily gains were 1.6, 1.8, and 1.8 lb/day the response to increasing dietary lysine would be quadratic. Probabilities for tests of these effects have the same interpretation as described above. Probabilities always measure the chance that random sampling caused the observed response. Therefore, if  $P < .01$  for the Q effect was found, there is less than a 1% chance that random differences between pigs on the treatments caused the observed response. 



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