

# An evaluation of the effects of added vitamin D<sub>3</sub> in maternal diets on sow and pig performance<sup>1,2</sup>

J. R. Flohr,\* M. D. Tokach,\* S. S. Dritz,\*<sup>3</sup> J. M. DeRouchey,\* R. D. Goodband,\*<sup>4</sup> J. L. Nelssen,\* and J. R. Bergstrom†

\*Department of Animal Sciences and Industry, Kansas State University, Manhattan 66506; and †DSM Nutritional Products Inc., Parsippany, NJ 07054

**ABSTRACT:** A total of 84 sows (PIC 1050) and their litters were used to determine the effects of supplementing maternal diet with vitamin D<sub>3</sub> on sow and pig performance, serum 25-hydroxyvitamin D<sub>3</sub> (25(OH)D<sub>3</sub>), milk vitamin D<sub>3</sub>, neonatal bone mineralization, and neonatal tissue vitamin D<sub>3</sub>. After breeding, sows were randomly assigned to 1 of 3 dietary vitamin D<sub>3</sub> treatments (1,500, 3,000, or 6,000 IU/kg of complete diets). Sows were bled on d 0 and 100 of gestation and at farrowing and weaning (d 21). Pig BW was recorded at birth and weaning, and serum was collected from 2 pigs/litter at birth, on d 10 and at weaning. A total of 54 pigs (18/treatment) were euthanized at birth and necropsied to sample bones and tissues. Sow and suckling pig performance and neonatal bone ash and bone density did not differ among maternal vitamin D<sub>3</sub> treatments; however, sow 25(OH)D<sub>3</sub> and milk vitamin D<sub>3</sub> increased (linear,  $P < 0.01$ ) with increasing maternal vitamin D<sub>3</sub> supplementation. Piglet serum 25(OH)D<sub>3</sub> increased (quadratic,  $P < 0.03$ ) with increased maternal vitamin D<sub>3</sub>. Neonatal kidney vitamin D<sub>3</sub> tended (quadratic,  $P = 0.08$ ) to decrease with increasing maternal vitamin D<sub>3</sub>, but liver vitamin D<sub>3</sub> tended (linear,  $P = 0.09$ ) to increase with increasing maternal

vitamin D<sub>3</sub>. At weaning, a subsample of 180 pigs (PIC 327 × 1050) were used in a 3 × 2 split plot design for 35 d to determine the effects of maternal vitamin D<sub>3</sub> and 2 levels of dietary vitamin D<sub>3</sub> (1,800 or 18,000 IU/kg) from d 0 to 10 postweaning on nursery growth and serum 25(OH)D<sub>3</sub>. Overall (d 0 to 35), nursery ADG and G:F were not affected by either concentration of vitamin D<sub>3</sub>, but ADFI tended (quadratic,  $P < 0.06$ ) to decrease with increasing maternal vitamin D<sub>3</sub> as pigs from sows fed 3,000 IU had lower ADFI compared with pigs from sows fed 1,500 or 6,000 IU/kg. Nursery pig serum 25(OH)D<sub>3</sub> increased with increasing maternal vitamin D<sub>3</sub> (weaning) on d 0 (linear,  $P < 0.01$ ), and maternal × diet interactions ( $P < 0.01$ ) were observed on d 10 and 21 because pigs from sows fed 1,500 IU had greater increases in serum 25(OH)D<sub>3</sub> when fed 18,000 IU compared with pigs from sows fed 3,000 IU. In conclusion, sow and pig serum 25(OH)D<sub>3</sub>, milk vitamin D<sub>3</sub>, and neonatal tissue vitamin D<sub>3</sub> can be increased by increasing maternal vitamin D<sub>3</sub>, and nursery pig 25(OH)D<sub>3</sub> can be increased by increasing dietary vitamin D<sub>3</sub>; however, sow and pig performance and neonatal bone mineralization was not influenced by increasing vitamin D<sub>3</sub> dietary levels.

**Key words:** nursery pigs, sow nutrition, vitamin D, vitamin D<sub>3</sub>, 25-hydroxyvitamin D<sub>3</sub>

© 2014 American Society of Animal Science. All rights reserved.

J. Anim. Sci. 2014.92:594–603

doi:10.2527/jas2013-6792

## INTRODUCTION

Speculation has surfaced recently about serum 25-hydroxyvitamin D<sub>3</sub> (25(OH)D<sub>3</sub>) concentrations of nursery pigs reared in modern swine production facilities. This is mainly due to documented cases, in which vitamin D has been absent from premixes fed to pigs (Feedstuffs, 2010; Salas, 2011), leading to metabolic bone disease and rickets. In an attempt to prevent deficiency, veterinarians have advocated guidelines on serum 25-hydroxyvitamin D (25(OH)D) concentra-

<sup>1</sup>Contribution no. 13-322-J from the KS Agric. Exp. Stn., Manhattan 66506.

<sup>2</sup>We would like to thank DSM Nutritional Products Inc., Heartland Assays (Ames, IA), and Iowa State University College of Veterinary Medicine (Ames, IA) for providing funding, laboratory analysis, and vitamin D<sub>3</sub> supplement for these experiments.

<sup>3</sup>Present address: Food Animal Health and Management Center, College of Veterinary Medicine, Kansas State University, Manhattan 66506.

<sup>4</sup>Corresponding author: Goodband@ksu.edu

Received June 11, 2013.

Accepted November 19, 2013.

tions that are associated with deficiency and adequacy of the vitamin from similar values used in human health (IOM, 1997; Specker et al., 1992). A survey of swine indicated that a substantial proportion of pigs have serum 25(OH)D<sub>3</sub> concentrations below guidelines, especially weaned pigs (Madson, 2011). Additionally, recent research describing the presence of the vitamin D receptor in numerous soft tissues not associated with Ca and P homeostasis (Norman and Bouillon, 2010) indicates that vitamin D may play a role in other biologically important functions of the body.

Vitamin D can be supplemented to the suckling pig in synthetic forms; however, this increases production and labor cost. Goff et al. (1984) reported increased piglet serum 25(OH)D<sub>3</sub> concentrations in newborn pigs from sows dosed with vitamin D<sub>3</sub> intramuscularly, indicating that vitamin D and its metabolites are transferred transplacentally. No research has examined the influence of maternal dietary vitamin D<sub>3</sub> on vitamin D status of young pigs (measured by serum 25(OH)D<sub>3</sub>), which would represent a less expensive and less labor intensive form of supplement for the young pig. The objectives of this experiment were to evaluate whether increasing vitamin D status through maternal vitamin D<sub>3</sub> supplementation (2, 4, and 8 times requirement; NRC, 2012) affects maternal performance, milk vitamin D<sub>3</sub> concentrations, sow and piglet serum 25(OH)D<sub>3</sub> concentrations, subsequent pig growth, neonatal pig liver and kidney vitamin D<sub>3</sub> concentrations, and neonatal bone mineralization.

## MATERIALS AND METHODS

Experimental procedures and animal care were approved by the Kansas State University Institutional Animal Care and Use Committee. This experiment was conducted at the Kansas State University Swine Teaching and Research Facility (Manhattan, KS) from the months of January through August of 2012. Gestation and lactation sow diets and phase 2 and phase 3 nursery diets were prepared at the Kansas State University Animal Science Feed Mill (Manhattan, KS). Phase 1 nursery diets were prepared at the Kansas State University Grain Science Feed Mill (Manhattan, KS). All diets were formulated to meet or exceed nutrient requirement estimates (NRC, 2012).

A total of 84 sows (PIC 1050; PIC, Hendersonville, TN) and their litters were used to determine the effects of supplementing varying levels (2, 4, and 8 times requirement; NRC, 2012) of dietary vitamin D<sub>3</sub> on maternal performance, subsequent pig performance, sow and piglet serum 25(OH)D<sub>3</sub>, Ca, and P, milk vitamin D, neonatal bone mineralization, and piglet tissue vitamin D<sub>3</sub> concentrations. Following breeding, sows were randomly assigned to 1 of 3 dietary vitamin D<sub>3</sub> treatments (1,500, 3,000, or 6,000 IU/kg) within 3 consecutive far-

rowing groups. There were 28 sows per treatment and 7 to 11 replications per farrowing group. During d 0 to 110 of gestation, sows were housed in gestation stalls (2.13 by 0.61 m) and were fed 2.0 kg/d of the gestation diets. On d 110, sows were transported to the farrowing house and were housed in farrowing crates. Both the gestation and farrowing barns were totally enclosed, environmentally controlled, and mechanically ventilated buildings. The farrowing barn contained 29 farrowing crates (2.13 by 0.61 m for the sow and 2.13 by 0.96 m for the pigs) that were each equipped with a single feeder and nipple waterer. After farrowing, sows were switched to lactation diets. Gestation and lactation diets were formulated to contain 0.56 and 0.94% standardized ileal digestible Lys, respectively (Table 1). Gestation and lactation diets contained 40 and 20% dried distillers grains with solubles, respectively. For the first 3 d after farrowing, sows were gradually provided increased feed according to appetite. After d 3, all sows were allowed ad libitum access to the lactation diet. Temperature in the farrowing house was maintained at a minimum of 20°C, and supplemental heat was provided to piglets with heat lamps.

Lactation feed intake was determined by measuring feed disappearance on d 0, 7, 14, and 21 (weaning). Sow BW was measured at breeding, d 110 of gestation, within 24 h after farrowing, and at weaning to determine gestation weight gain and lactation weight loss. Sows were bled on d 0 and 100 of gestation, within 12 h after farrowing, and on d 10 and 21 (weaning) in lactation to determine serum 25(OH)D<sub>3</sub>, Ca, and P concentrations. Milk samples were collected within 12 h after farrowing and on d 10 and 21 (weaning) to determine milk vitamin D<sub>3</sub> concentrations. Milk samples were obtained after an intravenous injection of oxytocin (1 mL; Agrilabs, St. Joseph, MO), and milk was collected from all functional glands. At birth, all piglets were weighed individually and ear tagged for identification. The second and fifth pigs born within each litter were bled before suckling on d 0 and 10 and at weaning to determine piglet serum 25(OH)D<sub>3</sub>, Ca, and P. The seventh pig born from 54 litters (18 pigs per treatment and 6 replications per farrowing group) was euthanized before suckling and necropsied for bone and tissue sample analysis to determine neonatal pig bone ash content, bone density, and tissue vitamin D<sub>3</sub> concentrations. Mummified and stillborn pigs were recorded to calculate total born and live born piglets. Although minimal, cross-fostering was conducted within 24 h postfarrowing to help standardize litter size within vitamin D<sub>3</sub> dietary treatments. Pigs were weighed after fostering to measure fostered litter weight. At weaning, piglet weights and piglet counts were recorded to determine individual and litter weight gains along with survivability.

**Table 1.** Composition of sow diets (as-fed basis)<sup>1,2</sup>

Item	Gestation	Lactation
Ingredient, %		
Corn	52.95	52.19
Soybean meal (46.5% CP)	2.99	23.88
Dried distillers grains with solubles	40.00	20.00
Monocalcium P (21% P)	0.65	0.90
Limestone	1.90	1.60
Salt	0.50	0.50
Vitamin premix <sup>3</sup>	0.50	0.50
Trace mineral premix <sup>4</sup>	0.15	0.15
L-Lys HCl	0.23	0.15
Phytase <sup>5</sup>	0.13	0.13
Total	100	100
Calculated analysis		
ME, kcal/kg	3,289	3,281
CP, %	17.0	21.1
Total Lys, %	0.72	1.13
Standardized ileal digestible Lys, %	0.56	0.97
Ca, %	0.88	0.88
P, %	0.59	0.64
Available P, <sup>6</sup> %	0.50	0.48

<sup>1</sup>A total of 84 sows and litters were used to determine the effects of supplemental vitamin D<sub>3</sub> on maternal performance, subsequent pig performance, sow and piglet serum 25-hydroxyvitamin D<sub>3</sub>, Ca, and P, milk vitamin D, neonatal bone mineralization, and piglet tissue vitamin D concentrations.

<sup>2</sup>Vitamin D<sub>3</sub> premixes were mixed to contain 2,024,620 IU vitamin D<sub>3</sub>/kg of premix by blending vitamin D<sub>3</sub> with rice hulls. Premix replaced a percentage of corn to achieve the desired treatment vitamin D<sub>3</sub> concentrations.

<sup>3</sup>Vitamin premix provided 11,023 IU vitamin A, 1,378 IU vitamin D<sub>3</sub>, 44 IU vitamin E, 4.41 mg menadione, 8.27 mg riboflavin, 27.56 mg pantothenic acid, 49.60 mg niacin, 38.5 µg vitamin B<sub>12</sub>, 551 mg choline, 0.22 mg biotin, 1.65 mg folic acid, and 4.96 mg pyridoxine per kilogram of the complete diet.

<sup>4</sup>Trace mineral premix provided 39.68 mg Mn as manganous oxide, 151.84 mg Fe ferrous sulfate, 151.84 mg Zn as zinc oxide, 15.18 mg Cu copper sulfate, 0.30 mg I as potassium iodide, and 0.30 mg Se as sodium selenite per kilogram of the complete diet.

<sup>5</sup>Natuphos 600 (BASF, Florham Park, NJ). Provided 752 phytase units/kg of diet.

<sup>6</sup>Phytase provided 0.12% available P to gestation and lactation diets.

At weaning, a subsample of 180 multisex pigs (PIC 327 × 1050; PIC) from the first sow group were used in a 3 × 2 split plot design for 35 d to determine the effects of maternal vitamin D<sub>3</sub> concentration and 2 levels of dietary vitamin D<sub>3</sub> (1,800 or 18,000 IU/kg from d 0 to 10 postweaning) on growth performance and serum 25(OH)D<sub>3</sub>, Ca, and P. At weaning, pigs were allotted to pens based on their previously administered maternal vitamin D<sub>3</sub> treatments to maintain the integrity of weaning weights consistent with maternal vitamin D<sub>3</sub> effects. Pens were then randomly assigned to dietary vitamin D<sub>3</sub> treatments. There were 6 pigs/pen and 5 pens/treatment. Dietary vitamin D<sub>3</sub> treatments were provided from d 0 to 10 in the nursery and were fed in a pellet form (Table 2). Common phase 2 and 3 diets were provided to pigs from d 10 to 21 and d 21 to 35, respectively. Common diets were formulated to contain 1,800 IU vitamin D<sub>3</sub>/kg. All pens (1.2 by 1.5 m) had woven wire flooring, one 3-hole, dry self-feeder, and a nipple waterer to allow for ad libi-

**Table 2.** Composition of nursery diets (as-fed basis)<sup>1,2</sup>

Item	Phase 1 <sup>3</sup>	Phase 2 <sup>4</sup>	Phase 3 <sup>5</sup>
Ingredient, %			
Corn	39.56	44.72	65.76
Soybean meal (46.5% CP)	17.33	23.41	30.67
Dried distillers grains with solubles	5.00	15.00	–
Spray-dried porcine plasma	5.00	–	–
Spray-dried blood cells	1.25	–	–
Spray-dried whey	25.00	10.00	–
Select menhaden fish meal	–	4.50	–
Soybean oil	3.00	–	–
Monocalcium P (21% P)	0.85	0.15	1.03
Limestone	1.00	0.70	0.98
Salt	0.30	0.30	0.35
Zinc oxide	0.39	0.25	–
Trace mineral premix <sup>6</sup>	0.15	0.15	0.15
Vitamin premix <sup>7</sup>	0.25	0.25	0.25
L-Lys HCl	0.20	0.28	0.36
DL-Met	0.13	0.05	0.13
L-Thr	0.05	0.05	0.13
L-Ile	0.10	–	–
Phytase <sup>8</sup>	0.13	0.17	0.17
Acidifier <sup>9</sup>	0.20	–	–
Vitamin E, 20,000 IU	0.05	–	–
Choline chloride 60%	0.04	–	–
Vitamin D premix <sup>10</sup>	0.02	0.02	0.02
Total	100	100	100
Calculated analysis			
ME, kcal/kg	3,415	3,320	3,314
CP, %	21.2	23.1	20.4
Standardized ileal digestible Lys, %	1.35	1.30	1.25
Ca, %	0.80	0.70	0.68
P, %	0.71	0.63	0.61
Available P, <sup>11</sup> %	0.63	0.50	0.42

<sup>1</sup>A total of 180 pigs (initially 21 d of age; PIC 327 × 1050; PIC, Hendersonville, TN) were used in a 3 × 2 split plot design for 35 d to determine the effects of maternal vitamin D<sub>3</sub> and early nursery dietary vitamin D<sub>3</sub> on nursery growth performance and serum 25-hydroxyvitamin D<sub>3</sub> concentrations.

<sup>2</sup>Vitamin D<sub>3</sub> premixes were mixed to contain 2,024,620 IU vitamin D<sub>3</sub>/kg of premix by blending vitamin D<sub>3</sub> with rice hulls. Premix replaced a percentage of corn to achieve the desired treatment vitamin D<sub>3</sub> concentrations.

<sup>3</sup>Phase 1 diets were fed from d 0 to 10 and were formulated to contain either 1,800 or 18,000 IU vitamin D<sub>3</sub>/kg of the complete diet.

<sup>4</sup>Common phase 2 diets were fed from d 10 to 24 and were formulated to contain 1,800 IU vitamin D<sub>3</sub>/kg of the complete diet.

<sup>5</sup>Common phase 3 diets were fed from d 24 to 35 and were formulated to contain 1,800 IU vitamin D<sub>3</sub>/kg of the complete diet.

<sup>6</sup>Trace mineral premix provided 39.68 mg Mn as manganous oxide, 151.84 mg Fe as ferrous sulfate, 151.84 mg Zn as zinc oxide, 15.18 mg Cu as copper sulfate, 0.30 mg I as potassium iodide, and 0.30 mg Se as sodium selenite per kilogram of the complete diet.

<sup>7</sup>Vitamin premix provided 11,023 IU vitamin A, 1,378 IU vitamin D<sub>3</sub>, 44 IU vitamin E, 4.41 mg menadione, 8.27 mg riboflavin, 27.56 mg pantothenic acid, 49.60 mg niacin, and 38.5 µg of vitamin B<sub>12</sub> per kilogram of the complete diet.

<sup>8</sup>Natuphos 600 (BASF, Florham Park, NJ). Provided 780, 1,021, and 1,021 phytase units/kg of the complete diet for phase 1, 2, and 3 diets, respectively.

<sup>9</sup>Kem-Gest: phosphoric, fumaric, lactic, and citric acid (Kemin Industries Inc., Des Moines, IA).

<sup>10</sup>Vitamin D<sub>3</sub> premixes were mixed to contain 2,024,620 IU/kg of premix by blending vitamin D<sub>3</sub> (Rovimix D, DSM Nutritional Products, Parsippany, NJ) with rice hulls. Premix replaced a percentage of corn to achieve the desired treatment vitamin D<sub>3</sub> concentrations.

<sup>11</sup>Phytase provided 0.12, 0.13, and 0.12% available P for phase 1, 2, and 3 diets, respectively.

tum access to feed and water. All pigs and feeders were weighed on d 0, 5, 10, 17, 21, 28, and 35 after weaning to determine ADG, ADFI, and G:F. Blood samples were collected from 10 pigs/treatment on d 0, 10, 24, and 35 to determine serum 25(OH)D<sub>3</sub>, Ca, and P.

### ***Feed and Premix Vitamin D<sub>3</sub> Analysis***

Vitamin D<sub>3</sub> supplement (Rovimix D<sub>3</sub>, 500,000 IU/g; DSM Nutritional Products, Parsippany, NJ) was mixed with rice hulls to achieve a premix formulated to contain 2,204,620 IU vitamin D<sub>3</sub>/kg. Premix was then added to the control diet (1,378 IU vitamin D<sub>3</sub>/kg) by replacing corn to achieve desired dietary treatments. Vitamin premixes and complete diet samples were collected during feed manufacturing. These samples were pooled by specific diet type or premix and were subsampled. Subsamples were analyzed for vitamin D<sub>3</sub> by using a combination of HPLC and mass spectrometry (DSM Nutritional Products; Schadt et al., 2012).

### ***Serum 25-Hydroxyvitamin D<sub>3</sub>, Milk and Tissue Vitamin D, and Serum Ca and P Analysis***

All blood, milk, and tissue sample analyses were conducted by a commercial laboratory (Heartland Assays, Ames, IA). Blood samples were collected via jugular venipuncture using 25-mm (neonatal and nursery pigs) and 38-mm (sows) × 20-gauge needles and 10-mL blood collection tubes containing a gel separator to determine circulating serum 25(OH)D<sub>3</sub>, Ca, and P concentrations. Six hours after collection, blood was centrifuged (1,600 × *g* for 25 min at 2°C) and serum was harvested and stored at -20°C until analysis. Serum 25(OH)D<sub>3</sub> concentrations were determined by using a previously described RIA (Hollis et al., 1993), and serum Ca concentrations were determined by spectrophotometry with a commercial kit (Pointe Scientific, Canton, MI) using a method described by Pointe Scientific (2009a). Serum P was determined by spectrophotometry with a commercial kit (Pointe Scientific) using a method described by Pointe Scientific (2009b). Milk and whole tissue samples were frozen at -20°C until analysis. Analysis was conducted using a combination of HPLC and mass spectrometry previously described by Schadt et al. (2012).

### ***Necropsies, Bone and Tissue Sampling, and Analysis***

Necropsies were performed onsite and in compliance with the college's standard operating procedures. Pigs were euthanized with an intravenous overdose of sodium pentobarbital (Fatal Plus; Vortech Pharmaceuticals, Dearborn, MI). Right femurs and second ribs were collected to determine bone ash content and left second

ribs were used to determine bone density. Whole liver and kidney tissues were collected and frozen immediately at -20°C until samples were prepared for specific analysis.

Bone densities were determined at the Iowa State University College of Veterinary Medicine (Ames, IA). All left second ribs were stripped to the periosteum, submerged in water for 4 h under 83 kPa vacuum, and blotted dry before recording bone weight. Bone volume was determined using weight in air minus weight under water according to Archimedes principle (Keenan et al., 1997). Bone density values were then expressed as grams of bone per milliliter volume.

Bone ash analysis, which was performed on the right femurs and right second rib, was conducted at the Kansas State University Swine Nutrition Laboratory (Manhattan, KS). Bones were cleaned to the periosteum and were split perpendicular to the long axis of the diaphysis. Fat was extracted by placing bones in cellulose thimbles and inserting thimbles into the main chambers of Soxhlet extractors. The extraction solvent was petroleum ether. At the completion of the 7-d extraction period, bone samples were dried in a forced-air oven at 100°C until a consistent dry weight was achieved. Then, bones were ashed at 600°C for 24 h. Ash weights were recorded and expressed as a percentage of dry fat-free bone.

### ***Statistical Analysis***

Data were analyzed using the MIXED procedure in SAS (SAS Inst. Inc., Cary, NC). Treatment means were analyzed using the LSMEANS statement, and preplanned contrasts were used to determine the linear and quadratic effects of increasing vitamin D<sub>3</sub>. Unequally spaced linear and quadratic contrasts were derived using the IML procedure in SAS. Maternal performance data were analyzed with sow as the experimental unit, treatment as a fixed effect, and farrowing group as a random effect. Nursery performance was analyzed as a 3 × 2 split plot design and the pen was the experimental unit. Additional preplanned contrasts were used to determine the effects of early nursery vitamin D<sub>3</sub> treatments and the interaction of maternal vitamin D<sub>3</sub> and early nursery dietary vitamin D<sub>3</sub> treatments. Serum 25(OH)D<sub>3</sub>, Ca, and P and milk vitamin D<sub>3</sub> data were analyzed using the REPEATED function of SAS to determine the effects of treatment variables over time, and the individual pig was the experimental unit. Bone ash, bone density, and tissue vitamin D concentrations were analyzed using the individual pig as the experimental unit. Differences among treatments were considered significant with  $P \leq 0.05$  and trends if  $P > 0.05$  and  $P \leq 0.10$ .

## RESULTS

Analysis of vitamin D<sub>3</sub> concentrations in the diets were presented in Table 3. The results verified that they were within acceptable analytical error of formulated dietary values.

### Maternal Performance

Supplementation of vitamin D<sub>3</sub> at levels used in this study did not influence sow lactation ADFI (Table 4) or sow BW throughout gestation and lactation. Additionally, the levels of maternal vitamin D<sub>3</sub> within the experiment did not affect litter size criteria or suckling pig performance. Four sows were removed from experimental treatments during the study. A first parity sow from the 3,000 IU vitamin D<sub>3</sub>/kg group was removed due to later term abortion before entry into the farrowing house, and another first parity sow from the 3,000 IU vitamin D<sub>3</sub>/kg treatment was euthanized and a caesarean section was performed due to farrowing complications. One fifth parity sow from the 6,000 IU vitamin D<sub>3</sub>/kg treatment group was removed due to a uterine infection after parturition. A fourth parity sow from the 6,000 IU vitamin D<sub>3</sub>/kg group was removed due to lingering structural issues that caused lameness within 3 wk after being administered onto the treatment diets. None of the removed sows showed signs associated with the onset of hypervitaminosis D, which would include weight loss, lethargic behavior, or feed refusal.

### Nursery Performance

During the nursery portion of the study, no interactions of maternal vitamin D<sub>3</sub> and dietary vitamin D<sub>3</sub> on growth performance were observed (Table 5). Overall (d 0 to 35), ADFI tended (quadratic,  $P = 0.06$ ) to decrease with increasing vitamin D<sub>3</sub>, with pigs from sows fed 3,000 IU having lower ADFI than pigs from sows fed either 1,500 or 6,000 IU/kg.

### Sow Serum 25-Hydroxyvitamin D<sub>3</sub>, Ca, and P and Milk Vitamin D<sub>3</sub>

A maternal  $\times$  day interaction ( $P < 0.01$ ; Table 6) was observed for sow serum 25(OH)D<sub>3</sub> because serum 25(OH)D<sub>3</sub> was not different among sows on d 0 of gestation; however, increasing maternal vitamin D<sub>3</sub> increased serum 25(OH)D<sub>3</sub> on d 100 of gestation, at farrowing, and at weaning. A day effect ( $P < 0.01$ ; Fig. 1) was observed for serum Ca. Serum Ca tended (linear,  $P = 0.07$ ) to be greater on d 0 of gestation for sows assigned to the 6,000 IU vitamin D<sub>3</sub> treatment compared with sows assigned to lower maternal vitamin D<sub>3</sub> treatments.

**Table 3.** Analyzed dietary vitamin D<sub>3</sub> concentrations (as-fed)<sup>1</sup>

Item	Maternal diets, IU/kg <sup>2</sup>			Nursery diets, IU/kg <sup>3</sup>			
				Phase 1 <sup>4</sup>	Phase 2 <sup>5</sup>	Phase 3 <sup>6</sup>	
Formulated	1,500	3,000	6,000	1,800	18,000	1,800	1,800
Analyzed	–	–	–	1,870	19,300	1,855	1,911
Gestation	1,505	3,370	8,025	–	–	–	–
Lactation	1,475	3,390	6,210	–	–	–	–

<sup>1</sup>Vitamin D<sub>3</sub> analysis was performed by DSM Nutritional Products (Parsippany, NJ), and values represent the average of 2 pooled samples per diet.

<sup>2</sup>A total of 81 sows and litters were used to determine the effects of supplemental vitamin D<sub>3</sub> on maternal performance, subsequent pig performance, sow and piglet serum 25-hydroxyvitamin D<sub>3</sub> (25(OH)D<sub>3</sub>), Ca, and P, milk vitamin D, neonatal bone mineralization, and piglet tissue vitamin D concentrations.

<sup>3</sup>A total of 180 pigs (PIC 327  $\times$  1050; initially 21 d of age) were used in a 3  $\times$  2 split plot design for 35 d to determine the effects of maternal vitamin D<sub>3</sub> and early nursery dietary vitamin D<sub>3</sub> on nursery growth performance and serum 25(OH)D<sub>3</sub> concentrations.

<sup>4</sup>Phase 1 diets were fed from d 0 to 10.

<sup>5</sup>Common phase 2 diets were fed from d 10 to 24.

<sup>6</sup>Common phase 3 diets were fed from d 24 to 35.

On d 100 of gestation, increasing maternal dietary vitamin D<sub>3</sub> tended to decrease ( $P = 0.09$ ) serum Ca. Serum P concentrations were not influenced by maternal vitamin D<sub>3</sub> treatments or by sampling day (Fig. 1). Milk vitamin

**Table 4.** The effects of high maternal vitamin D<sub>3</sub> on sow and litter performance<sup>1,2</sup>

Item	Vitamin D <sub>3</sub> , IU/kg			SEM	P-value	
	1,500	3,000	6,000		Linear	Quadratic
Sows, <i>n</i>	28	26	26			
ADFI, kg						
d 0 to wean (d 21)	5.65	5.88	5.98	0.34	0.27	0.63
Sow BW, kg						
Gestation						
d 0	193.1	194.1	192.1	8.8	0.91	0.89
d 110	231.4	235.2	237.1	6.0	0.52	0.80
Change	+38.3	+41.1	+45.0	5.4	0.24	0.92
Lactation						
d 0	221.9	227.6	224.1	6.0	0.89	0.50
d 21 (weaning)	212.3	220.3	217.4	7.5	0.67	0.42
Change	–9.6	–7.3	–6.7	2.8	0.24	0.43
Piglets						
Litter size, no.						
Mummies	0.3	0.2	0.3	0.1	0.88	0.86
Stillborn	0.6	0.4	0.8	0.3	0.60	0.37
Total born alive	13.0	12.5	13.2	0.9	0.74	0.57
Fostered	12.3	12.1	13.0	0.7	0.50	0.48
Weaned	11.2	10.8	11.5	0.7	0.48	0.32
Survivability, <sup>3</sup> %	91.2	89.2	88.5	2.0	0.88	0.58
Piglet BW, kg						
Birth	1.31	1.36	1.34	0.04	0.63	0.47
Weaning	5.31	5.55	5.52	0.17	0.43	0.42

<sup>1</sup>A total of 84 sows (PIC 1050; PIC, Hendersonville, TN) and their litters were used. There were 2 sows removed from the 3,000 IU/kg vitamin D<sub>3</sub> treatment because of lameness and illness. There were 2 sows removed from the 6,000 IU/kg vitamin D<sub>3</sub> treatment because of late-term abortion and farrowing complications.

<sup>2</sup>Sow group was used as a random effect in the statistical model.

<sup>3</sup>Survivability was calculated by dividing the weaned litter size by the fostered litter size.

**Table 5.** The effects of maternal and early nursery vitamin D<sub>3</sub> supplementation on nursery pig growth performance (d 0 to 35)<sup>1</sup>

Item	Maternal vitamin D <sub>3</sub> , IU/kg						SEM	P-value			
	1,500		3,000		6,000			Maternal			Diet
Vitamin D <sub>3</sub> <sup>2</sup> :	1,800	18,000	1,800	18,000	1,800	18,000	Maternal × diet	Linear	Quadratic	Diet	
ADG, g	417	419	391	399	416	396	13	0.56	0.56	0.12	0.75
ADFI, g	597	618	577	565	602	576	17	0.39	0.47	0.06	0.69
G:F	0.70	0.68	0.68	0.71	0.69	0.69	0.01	0.17	0.96	0.71	0.90

<sup>1</sup>A total of 180 mixed-sex pigs (initially 21 d of age; PIC 327 × 1050; PIC, Hendersonville, TN) were weaned from the first sow group and used in a 3 × 2 split plot design for 35 d to determine the effects of maternal and early nursery dietary vitamin D<sub>3</sub> on growth performance.

<sup>2</sup>Early dietary vitamin D<sub>3</sub> treatments (IU/kg). Phase 1 diets were fed from d 0 to 10. Common phase 2 and 3 diets were fed from d 10 to 21 and d 21 to 35, respectively. Common diets were formulated to contain 1,800 IU/kg vitamin D<sub>3</sub>. Treatments are expressed as IU/kg of the complete diet.

D<sub>3</sub> concentrations were not influenced by sampling day; however, milk vitamin D<sub>3</sub> increased (linear,  $P < 0.01$ ) with increasing maternal dietary vitamin D<sub>3</sub> at farrowing, on d 10 of lactation, and at weaning.

**Table 6.** Effects of high maternal vitamin D<sub>3</sub> on serum 25-hydroxyvitamin D<sub>3</sub> (25(OH)D<sub>3</sub>), Ca, P, milk vitamin D, neonatal bone ash content, and bone density<sup>1,2</sup>

Item	Maternal vitamin D <sub>3</sub> , IU/kg				P-value	
	1,500	3,000	6,000	SEM	Linear	Quadratic
<b>Sow</b>						
25(OH)D <sub>3</sub> , ng/mL						
d 0	30.1	26.2	32.0	4.7	0.54	0.27
d 100	33.2	36.5	57.9	4.7	<0.01	0.23
Farrowing	30.1	35.4	56.9	4.7	<0.01	0.38
Weaning	39.3	52.5	66.3	4.7	<0.01	0.31
Milk vitamin D <sub>3</sub> , ng/g						
Farrowing	1.02	2.33	3.97	0.31	<0.01	0.37
d 10	0.78	2.33	3.73	0.31	<0.01	0.13
Weaning	1.02	1.98	3.53	0.31	<0.01	0.73
<b>Piglet</b>						
25(OH)D <sub>3</sub> , ng/mL						
Birth	4.5	5.9	9.4	0.8	<0.01	0.03
d 10	4.4	6.2	10.6	0.8	<0.01	<0.01
Weaning	5.6	8.0	14.0	0.8	<0.01	<0.01
Bone ash content, %						
Second rib	43.6	43.6	43.5	0.8	0.95	0.96
Femur	44.9	44.5	44.8	0.6	0.76	0.66
Bone density, g/mL						
Second rib	1.30	1.30	1.31	0.02	0.64	0.56
Tissue vitamin D <sub>3</sub> , ng/g						
Kidney	1.68	0.10	1.37	0.84	0.99	0.09
Liver	0.04	0.04	0.19	0.05	0.08	0.16

<sup>1</sup>A total of 84 sows (PIC 1050, PIC, Hendersonville, TN) and their litters were used to determine the effects of high maternal vitamin D<sub>3</sub> on sow and pig performance, serum 25(OH)D<sub>3</sub>, Ca, P, milk vitamin D<sub>3</sub>, neonatal bone mineralization, and tissue vitamin D<sub>3</sub>.

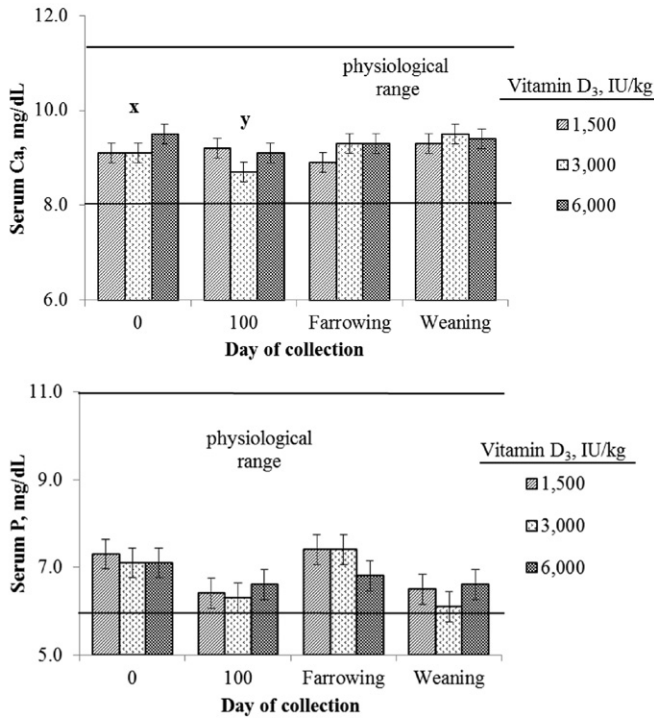
<sup>2</sup>Day effects were  $P < 0.01$ ,  $P = 0.56$ , and  $P < 0.01$  for sow 25(OH)D<sub>3</sub>, milk vitamin D<sub>3</sub>, and piglet 25(OH)D<sub>3</sub>, respectively. Maternal × day interactions were  $P < 0.01$ ,  $P = 0.87$ , and  $P = 0.13$  for sow 25(OH)D<sub>3</sub>, milk vitamin D<sub>3</sub>, and piglet 25(OH)D<sub>3</sub>, respectively.

### Piglet Serum 25-Hydroxyvitamin D<sub>3</sub>, Ca, and P and Neonatal Bone Ash and Density and Tissue Vitamin D

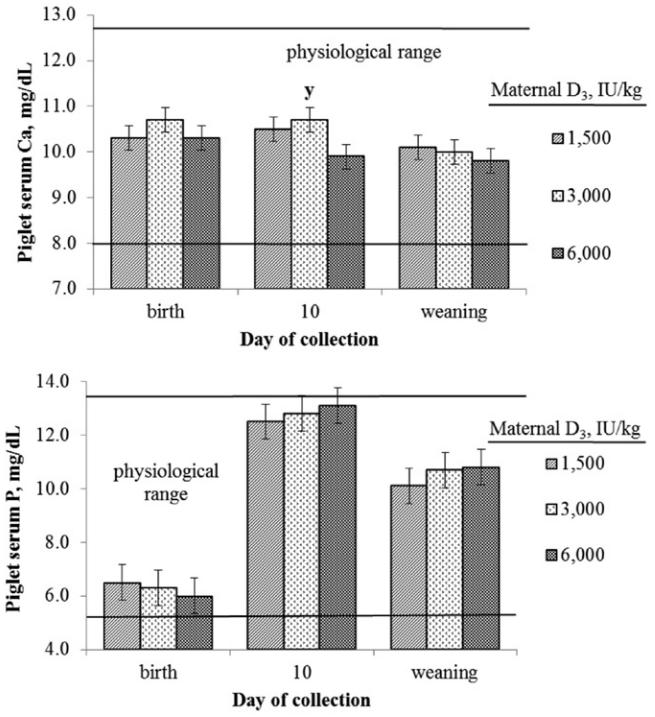
A day effect was observed ( $P < 0.01$ ; Table 6) for piglet serum 25(OH)D<sub>3</sub> as serum concentrations increased over time from birth to weaning, and serum 25(OH)D<sub>3</sub> increased (quadratic,  $P = 0.03$ ) with increasing maternal vitamin D<sub>3</sub> at birth, on d 10 in lactation, and at weaning. A day effect was observed ( $P < 0.01$ ; Fig. 2) for serum Ca concentrations. Additionally, serum Ca tended to decrease ( $P = 0.08$ ) with increasing maternal vitamin D<sub>3</sub> on d 10 of lactation. Serum P concentrations (Fig. 2) were not influenced by maternal vitamin D<sub>3</sub> treatments, but they tended to differ based on day of sampling ( $P = 0.08$ ). No differences in bone ash values were observed for femurs or second ribs. Rib bone density was not influenced by maternal vitamin D<sub>3</sub> concentrations. Kidney vitamin D<sub>3</sub> concentrations (quadratic,  $P = 0.09$ ) tended to decrease with increasing maternal vitamin D<sub>3</sub> because pigs from sows fed 3,000 IU vitamin D<sub>3</sub> had much lower tissue vitamin D<sub>3</sub> concentrations than pigs from sows fed 1,500 or 6,000 IU/kg. Liver tissue vitamin D<sub>3</sub> concentrations tended to increase (linear,  $P = 0.08$ ) with increased maternal dietary vitamin D<sub>3</sub>.

### Nursery Pig Serum 25-Hydroxyvitamin D<sub>3</sub>, Ca, and P

A day effect ( $P < 0.01$ ; Table 7) was observed for nursery pig serum 25(OH)D<sub>3</sub>. At weaning (d 0), pig serum 25(OH)D<sub>3</sub> was increased (linear,  $P < 0.01$ ) with increasing maternal vitamin D<sub>3</sub>. Maternal × diet interactions ( $P < 0.01$ ) were observed on d 10 and 21 because pigs from sows fed 1,500 IU vitamin D<sub>3</sub>/kg had greater increases in serum 25(OH)D<sub>3</sub> when fed 18,000 IU vitamin D<sub>3</sub>/kg compared with pigs from sows fed 3,000 IU vitamin D<sub>3</sub>/kg. On d 35, serum 25(OH)D<sub>3</sub> concentrations were not different among maternal treatments, but a diet effect ( $P = 0.04$ ) was observed with increased nursery diet vitamin D<sub>3</sub> increasing serum 25(OH)D<sub>3</sub>. Serum Ca and P concentrations were not influenced by maternal or nursery vitamin D<sub>3</sub> concentrations, except for a tendency (quadratic,  $P = 0.08$ ; Fig. 3) for P concentrations to in-



**Figure 1.** Serum Ca and P concentrations (mg/dL) at d 0 and 100 of gestation, farrowing, and weaning (d 21 of lactation) in sows fed diets formulated to supply 1,500, 3,000, or 6,000 IU vitamin D<sub>3</sub>/kg of the complete diet. Superscripts denote tendencies (0.05 < P ≤ 0.10): <sup>x</sup>linear dietary effects and <sup>y</sup>quadratic dietary effects. Physiological range based on Friendship et al. (1984).



**Figure 2.** Serum Ca and P concentrations (mg/dL) at birth, and weaning (d 21) in pigs from sows fed diets formulated to supply 1,500, 3,000, or 6,000 IU vitamin D<sub>3</sub>/kg of the complete diet. Superscripts denote tendencies (0.05 < P ≤ 0.10): <sup>y</sup>quadratic maternal diet effect. Physiological range based on Friendship et al. (1984).

crease within increasing maternal vitamin D<sub>3</sub>. In addition, a day effect for serum P was observed (P < 0.01).

**DISCUSSION**

The concept of supplementing levels of vitamin D in excess of the requirement is often associated with hypervitaminosis or toxicity. This is due to previous studies describing soft tissue mineralization and even death as a result of toxicity (Chineme et al., 1976; Kamio et al., 1977; Long, 1984). However, in these observed toxicity incidents, supplemented levels were more than 1,000 times the animal’s requirement (NRC, 2012). The NRC

(1987) presumed the upper safe levels in swine to be 2,200 IU and 33,000 IU per kilogram of the diet when exposure time is greater than 60 or less than 60 d, respectively. These values are 10 to 150 times the growing pig’s requirement and 3 to 40 times the sow’s requirement (NRC, 2012). Despite this, most diets in the swine industry are formulated to contain 6 to 9 times the growing pig’s requirement and 2 times the sow’s requirement for vitamin D<sub>3</sub> (Reese and Hill, 2010). To maintain industry applicability, this study was developed to examine the effects of supplementing vitamin D<sub>3</sub> through maternal diets at concentrations above typically formulated levels rather than established requirement levels. To our

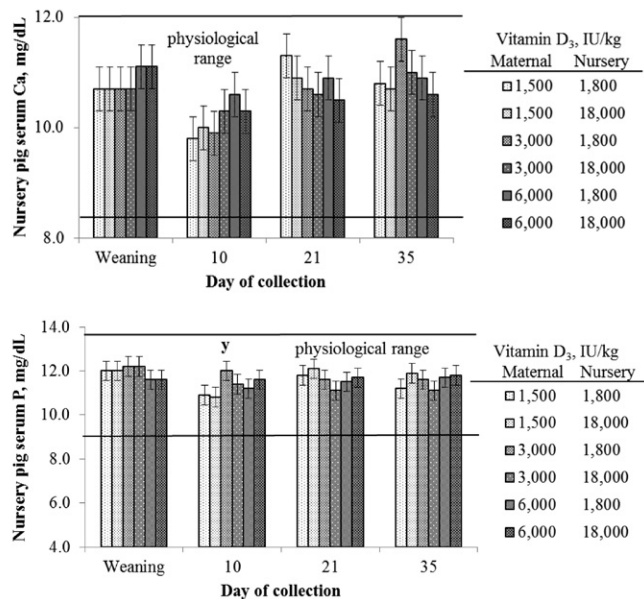
**Table 7.** The effects of maternal and early nursery dietary vitamin D<sub>3</sub> on nursery pig serum 25-hydroxyvitamin D<sub>3</sub> (25(OH)D<sub>3</sub>; ng/mL)<sup>1,2</sup>

Item	Maternal vitamin D <sub>3</sub> , IU/kg						SEM	P-value			
	1,500		3,000		6,000			Maternal × diet	Maternal		Diet
	1,800	18,000	1,800	18,000	1,800	18,000			Linear	Quadratic	
d 0	6.3		10.5		17.6		3.1		<0.01	0.91	
d 10	20.0	53.5	21.9	49.6	24.0	60.9	2.2	0.01	<0.01	0.04	<0.01
d 21	13.2	26.7	13.6	23.9	14.4	31.6	2.2	0.01	0.16	0.15	<0.01
d 35	16.7	18.0	14.5	19.3	14.9	19.5	2.2	0.42	0.94	0.83	0.04

<sup>1</sup>A total of 180 mixed-sex pigs (initially 21 d of age; PIC 327 × 1050; PIC, Hendersonville, TN) were weaned from the first sow group and used in a 3 × 2 split plot design for 35 d to determine the effects of maternal and early nursery dietary vitamin D<sub>3</sub> on growth performance. Ten pigs per treatment were bled to determine serum 25(OH)D<sub>3</sub>.

<sup>2</sup>Ten pigs per treatment were bled to determine serum 25(OH)D<sub>3</sub>. Day effect, P < 0.01, and maternal × diet × day interaction, P = 0.32.

<sup>3</sup>Early vitamin D<sub>3</sub> treatments (IU/kg). Phase 1 diets were fed from d 0 to 10. Common phase 2 and 3 diets were fed from d 10 to 21 and d 21 to 35, respectively. Common diets were formulated to contain 1,800 IU/kg vitamin D<sub>3</sub>.



**Figure 3.** Serum Ca and P concentrations (mg/dL) at weaning, d 10, 21, and 35 in nursery pigs from sows fed diets formulated to supply either 1,500, 3,000, or 6,000 IU vitamin D<sub>3</sub>/kg of the complete diet, and fed diets formulated to either 1,800 or 18,000 IU vitamin D<sub>3</sub>/kg from weaning until d 10. Superscripts denote tendencies ( $0.05 < P \leq 0.10$ ): <sup>y</sup>quadratic maternal diet effect. Physiological range based on Friendship et al. (1984).

knowledge, this is the first study to evaluate vitamin D<sub>3</sub> supplementation up to 6,000 IU vitamin D<sub>3</sub>/kg of the complete diet.

No differences were observed in the current study with regards to sow BW change or lactation ADFI from supplementing vitamin D<sub>3</sub> at concentrations between 1,500 and 6,000 IU/kg of the complete diet. Similarly, in a study conducted by Lauridsen et al. (2009), no influence of vitamin D on gilt BW change was observed during the first 28 d of gestation when gilts were fed 4 levels (200, 800, 1,400, or 2,000 IU/kg) of vitamin D from 2 sources (vitamin D<sub>3</sub> or 25(OH)D<sub>3</sub>). The authors also reported no influence of dose or form of vitamin D supplementation on BW changes of multiparous sows between 2 and 5 parities throughout gestation or lactation. Interestingly, the investigators discussed an interaction for total feed intake as feed intake decreased with increasing doses of vitamin D<sub>3</sub>, which was observed mainly in parity 4 and 5 sows, but for sows fed 25(OH)D<sub>3</sub>, the largest decrease in feed intake was observed with increased vitamin D supplementation from 200 to 800 IU. However, the authors pointed out the limitations of the results because of the complexity of the interaction.

Viganò et al. (2003) described the potential role of vitamin D in implantation because of its ability to increase expression of calbindin, an intracellular protein involved in Ca metabolism, and *HoxA* genes, which are shown to affect the viability of pre-implantation embryos. In the current study, no differences in the number of stillborns or live born pigs were observed as a

result of supplementing vitamin D<sub>3</sub> between 1,500 and 6,000 IU/kg of the complete diet. Additionally, vitamin D<sub>3</sub> supplementation in maternal diets did not influence birth weight or suckling pig performance to weaning. Lauridsen et al. (2009) reported no effect of dietary form or dose of vitamin D on early reproduction in terms of the number of implanted fetuses in gilts or litter size of sows, but the authors reported reductions in the number of stillborns with increased vitamin D doses of 1,400 and 2,000 IU compared with 200 and 800 IU. Coffey et al. (2012) reported an increased number of developed fetuses in reproductive tracts harvested from first-service gilts when supplemented with 25(OH)D<sub>3</sub> compared with vitamin D<sub>3</sub> at the same supplementation level of 2,500 IU/kg of the complete diet. The authors speculated that this increase in the number of developed fetuses may have been due to the increased efficiency of absorption of 25(OH)D<sub>3</sub> compared with vitamin D<sub>3</sub> in the upper portion of the intestine, which has been observed in poultry (Bar et al., 1980). Research determining the efficiency of absorption of vitamin D<sub>3</sub> and 25(OH)D<sub>3</sub>, specifically in swine, has not been conducted, and commercial use of 25(OH)D<sub>3</sub> has not been approved for use in swine in the United States. Ultimately, to evaluate the economic incentive of increasing supplemental vitamin D<sub>3</sub> on the basis of sow productivity, large-scale commercial studies with increased sample sizes will be needed to increase sensitivity and reduce the experimental error that is associated with sow reproduction measurements.

Compared with presumed upper safety guidelines established by NRC (1987), sows in the current experiment were supplemented vitamin D<sub>3</sub> at rates 2 to 3 times the recommended level (2,200 IU/kg of the complete diet) for exposure times greater than 60 d with no adverse effects on feed intake, sow BW, or sow productivity. This may indicate that supplementation rates up to 6,000 IU/kg of complete feeds are safe to use for sows; however, sows in this study were not followed through subsequent parities to determine potential long-term effects. In addition, because of the absence of improvement in maternal performance within this study, increasing vitamin D<sub>3</sub> supplementation above 1,500 IU/kg of complete diet appears to carry no benefit.

Nursery pig performance within the current experiment was not adversely influenced by vitamin D<sub>3</sub> supplementation in maternal diets or in early nursery diets when fed up to 18,000 IU vitamin D<sub>3</sub>/kg of the complete diet. Flohr et al. (2013) observed decreased intake of diets supplemented with 44,100 IU/kg vitamin D<sub>3</sub>. Also, serum 25(OH)D<sub>3</sub> concentrations in pigs supplemented 18,000 IU of vitamin D<sub>3</sub> ranged from approximately 50 to 60 ng/mL on d 10, which is below previously described concentrations experienced in periods of vitamin D intoxication (Littledike and Horst, 1982). The minimal impact of vitamin D<sub>3</sub> supplementation on nursery pig performance may



be interpreted similarly to previous studies conducted by Wahlstrom and Stolte (1958) and Combs et al. (1966) although vitamin D<sub>3</sub> was supplemented at much greater levels in the current study. Previous research observing improvements in growth performance, as a result of vitamin D<sub>3</sub> supplementation, have been reported consistently when pigs were fed marginal Ca and P, and vitamin D deficiency also was established (Johnson and Palmer, 1939; Bethke et al., 1946; Rortvedt and Crenshaw, 2012). A depletion period was not performed before the initiation of the current study to maintain industry applicability.

Results from this study show that increasing supplementation of vitamin D<sub>3</sub>, either through maternal or nursery diets, resulted in increased serum 25(OH)D<sub>3</sub> in sows, neonatal pigs, and nursery pigs. This agrees with previous research conducted by Goff et al. (1984), Lauridsen et al. (2009), Witschi et al. (2011), and Coffey et al. (2012). The observed increase in sow and pig 25(OH)D<sub>3</sub> at parturition supports previous conclusions reported by Goff et al. (1984) who described a strong correlation of sow 25(OH)D<sub>3</sub> and piglet 25(OH)D<sub>3</sub> at birth because of transplacental transfer of 25(OH)D<sub>3</sub>. Additionally, the increases in maternal vitamin D<sub>3</sub> supplementation resulted in increases in milk vitamin D<sub>3</sub> concentrations, which agrees with work conducted in dairy cows (Hollis et al., 1983). Additionally, this increase in milk vitamin D<sub>3</sub> concentration contributed to increased piglet serum 25(OH)D<sub>3</sub> from birth (before suckling) to weaning because of milk intake. Nursery pig serum 25(OH)D<sub>3</sub> increased as a result of an interaction between maternal and phase 1 dietary vitamin D<sub>3</sub> supplementation; however, by d 35, nursery pigs were similar in serum 25(OH)D<sub>3</sub> concentrations, confirming the half-life of 25(OH)D<sub>3</sub> is between 10 d and 3 wk, which has been described previously in human research (IOM, 1997).

Liver vitamin D<sub>3</sub> concentrations in the current study tended to increase with increased maternal vitamin D<sub>3</sub>, and kidney vitamin D<sub>3</sub> concentrations decreased with increasing maternal vitamin D<sub>3</sub>. In general, however, tissue concentrations were low, which agrees with previous research that describes 25(OH)D as the circulating and stored metabolite of the vitamin. Research in rats by Clements and Fraser (1988) showed that vitamin D was transferred transplacentally, especially in the last trimester, and it was stored in fetal muscle, most notably as 25(OH)D<sub>3</sub>. Interestingly, research by Schröder et al. (1993) concluded that newborn piglets do not rely on vitamin D-dependent Ca transport until the fourth week postpartum. This may indicate that although newborn pigs have stored vitamin D in the form of 25(OH)D<sub>3</sub>, it is not needed in early life Ca absorption. However, more research quantifying 25(OH)D<sub>3</sub> presence in a variety of tissues along with hydroxylating enzyme levels in the newborn pig will help to better understand if vitamin D is useful for preweaning Ca and P absorption during the suckling period.

To our knowledge, previous research quantifying bone mineralization in newborn pigs has not been conducted. Vitamin D supplementation has been shown to influence fetal bone development in the human fetus when mothers are vitamin D deficient (Morley et al., 2006). In the current experiment, bone ash content of ribs and femurs of pigs euthanized at birth was not influenced by maternal vitamin D<sub>3</sub> treatment. Percentage bone ash of ribs and femurs were 15% lower than previously referenced bone ash values of nursery pigs 2 mo of age (Crenshaw et al., 1981). This is probably a function of age, which has been described as a predominant factor in bone mineral content (Crenshaw et al., 1981). In addition, as discussed by Reinhard et al. (1976), differences in ash content of bones depend on skeletal function, and the current study agrees with these results, as rib bone ash percentage was 1% lower than femur bone ash percentage. Bone densities were not different among sampled ribs, which is similar to the result described by Witschi et al. (2011) who reported similar bone mineral content and bone mineral density for pigs (35 d postpartum) from sows fed 200 IU/kg vitamin D or 2,000 IU/kg vitamin D from supplemented 25(OH)D<sub>3</sub>. Rortvedt and Crenshaw (2012) reported decreased mineral content and density of femurs in pigs at 9 wk of age, which were fed diets with no supplemental vitamin D and marginal Ca and P concentrations. The decreases in mineral content and density were exacerbated to a greater degree when pigs were from sows fed vitamin D<sub>3</sub>-depleted diets. This indicates that dietary Ca and P influences bone mineralization responses of the nursery pig to maternal vitamin D.

Serum Ca and P were not adversely influenced by supplemental vitamin D<sub>3</sub> in maternal diets or in phase 1 nursery diets. All reported concentrations were within normally described physiological ranges (Friendship et al., 1984). In the current study, Ca and P were supplied in excess of the animal's requirements (NRC, 2012), which indicates that vitamin D<sub>3</sub> supplementation above 1,500 IU in maternal diets or 1,800 IU in the nursery did not influence circulating Ca and P. This conclusion is similar to results described by Witschi et al. (2011) who supplemented the complete diet with 200 or 2,000 IU vitamin D/kg using 25(OH)D<sub>3</sub>; however, decreases in serum Ca and P have been associated with deficient supplementation rates of vitamin D<sub>3</sub> in growing pigs (Hagemoser et al., 2000).

Overall, the results of this study indicate that supplementing high concentrations of vitamin D<sub>3</sub> to sows can increase sow and piglet serum 25(OH)D<sub>3</sub>, milk vitamin D<sub>3</sub> concentrations, and neonatal tissue vitamin D<sub>3</sub> concentrations. Maternal vitamin D<sub>3</sub> and dietary vitamin D<sub>3</sub> supplementation can increase nursery pig serum 25(OH)D<sub>3</sub>; however, vitamin D<sub>3</sub> supplementation to either maternal or nursery diets beyond concentrations typically used in commercial diets failed to affect sow or pig performance, neonatal bone mineralization, or serum Ca

and P. This result indicates that supplementing vitamin D<sub>3</sub> levels above 1,500 IU for sows and 1,800 IU to nursery pigs has no beneficial effect.

### LITERATURE CITED

- Bar, A., M. Sharvit, D. Noff, S. Edelstein, and S. Hurwitz. 1980. Absorption and excretion of cholecalciferol and of 25-hydroxycholecalciferol and metabolites in birds. *J. Nutr.* 110:1930–1934.
- Bethke, R. M., W. Burroughs, O. H. M. Wilder, B. H. Edgington, and W. L. Robinson. 1946. The comparative efficiency of vitamin D from irradiated yeast and cod liver oil for growing pigs, with observations on their vitamin D requirements. *Ohio Agric. Exp. Stn. Bull.* 667:1–29. Ohio Agric. Exp. Stn., Wooster, OH.
- Chineme, C. N., L. Krook, and W. G. Pond. 1976. Bone pathology in hypervitaminosis D. An experimental study in young pigs. *Cornell Vet.* 66:387–412.
- Clements, M. R., and D. R. Fraser. 1988. Vitamin D supply to the rat fetus and neonate. *J. Clin. Invest.* 81:1768–1773.
- Coffey, J. D., E. A. Hines, J. D. Starkey, C. W. Starkey, and T. K. Chung. 2012. Feeding 25-hydroxycholecalciferol improves gilt reproductive performance and fetal vitamin D status. *J. Anim. Sci.* 90:3783–3788.
- Combs, G. E., T. H. Berry, H. D. Wallace, and R. C. Crum Jr. 1966. Influence of supplemental vitamin D on gain, nutrient digestibility and tissue composition of young pigs. *J. Anim. Sci.* 25:48–51.
- Crenshaw, T. D., E. P. Peo Jr., A. J. Lewis, B. D. Moser, and D. Olson. 1981. Influence of age, sex and calcium and phosphorus levels on the mechanical properties of various bones in swine. *J. Anim. Sci.* 52:1319–1329.
- Feedstuffs. 2010. Kent feeds recalls certain swine feeds. [http://fdsmagissues.feedstuffs.com/fds/PastIssues/FDS8225/fds02\\_8225.pdf](http://fdsmagissues.feedstuffs.com/fds/PastIssues/FDS8225/fds02_8225.pdf) (Accessed 4 April 2011.)
- Flohr, J. F., M. D. Tokach, S. S. Dritz, J. M. DeRouche, R. D. Goodband, J. L. Nelssen, S. C. Henry, L. M. Tokach, M. L. Potter, J. P. Goff, N. J. Koszewski, R. L. Horst, E. L. Hansen, and E. D. Fruge. 2013. Effects of supplemental vitamin D<sub>3</sub> on serum 25(OH)D<sub>3</sub> and growth of preweaning and nursery pigs. *J. Anim. Sci.* (in press).
- Friendship, R. M., J. H. Lumsden, I. McMillan, and M. R. Wilson. 1984. Hematology and biochemistry reference values for Ontario swine. *Can. J. Comp. Med.* 48:390–393.
- Goff, J. P., R. L. Horst, and E. T. Littledike. 1984. Effect of sow vitamin D status at parturition on the vitamin D status of neonatal piglet. *J. Nutr.* 114:163–169.
- Hagemoser, W. A., J. P. Goff, T. P. Sanderson, and J. S. Haynes. 2000. Osteopenic disease in growing pigs: Diagnostic methods using serum and urine calcium and phosphorus values, parathormone assay, and bone analysis. *J. Vet. Diagn. Invest.* 12:525–534.
- Hollis, B. P., J. Q. Kamerud, S. R. Selvaag, J. D. Lorenz, and J. L. Napoli. 1993. Determination of vitamin D status by radioimmunoassay with an 125I-labeled tracer. *Clin. Chem.* 39:529–533.
- Hollis, B. W., P. W. Lambert, and R. L. Horst. 1983. Factors affecting the antirachitic sterol content of native milk. In: M. F. Holick, T. K. Gray, and C. S. Anast, editors, *Perinatal calcium and phosphorus metabolism*. Elsevier, New York, NY. p. 157.
- Institute of Medicine (IOM). 1997. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. *Natl. Acad. Press*, Washington, DC.
- Johnson, D. W., and L. S. Palmer. 1939. Individual and breed variations in pigs on rations devoid of vitamin D. *J. Agric. Res.* 58:929–940.
- Kamio, A., F. A. Kummerow, and H. Imai. 1977. Degeneration of aortic smooth muscle cells in swine fed excess vitamin D<sub>3</sub>. *Arch. Pathol. Lab. Med.* 101:378–381.
- Keenan, M. J., M. Hegsted, K. L. Jones, J. P. Delany, J. C. Kime, L. E. Melancon, R. T. Tulley, and K. D. Hong. 1997. Comparison of bone density measurement techniques: DXA and Archimedes' principle. *J. Bone Miner. Res.* 12:1903–1907.
- Lauridsen, C., U. Halekoh, T. Larsen, and S. K. Jensen. 2009. Reproductive performance and bone status markers of gilts and lactating sows supplemented with two different forms of vitamin D. *J. Anim. Sci.* 88:202–213.
- Littledike, E. T., and R. L. Horst. 1982. Vitamin D<sub>3</sub> toxicity in dairy cows. *J. Dairy Sci.* 65:749–759.
- Long, G. G. 1984. Acute toxicosis in swine associated with excessive intake of vitamin D. *J. Am. Vet. Med. Assoc.* 184:164–170.
- Madson, D. M. 2011. Metabolic bone disease in swine: A diagnostic dilemma. In: *Proc. 52nd Ann. George A Young Swine Health and Management Conf.*, Sioux City, NE. p. 8–18.
- Morley, R., J. B. Carlin, J. A. Pasco, and J. D. Wark. 2006. Maternal 25-hydroxyvitamin D and parathyroid hormone concentrations and offspring birth size. *J. Clin. Endocrinol. Metab.* 91:906–912.
- Norman, A. W., and R. Bouillon. 2010. Vitamin D nutritional policy needs a vision for the future. *Exp. Biol. Med.* 235: 1034–1045.
- NRC. 1987. Vitamin tolerance of animals. G. F. Combs Jr., editor. *Natl. Acad. Press*, Washington, DC.
- NRC. 1998. Nutrient requirements of swine. 10th rev. ed. *Natl. Acad. Press*, Washington, DC.
- NRC. 2012. Nutrient requirements of swine. 11th rev. ed. *Natl. Acad. Press*, Washington, DC.
- Pointe Scientific. 2009a. Calcium reagent set. *Tech. Bull. No. P803–C7503–01*. Rev. ed. Pointe Scientific, Canton, MI.
- Pointe Scientific. 2009b. Inorganic phosphorus reagent set (UV). *Tech. N. P803–P7516–01*. Rev. ed. Pointe Scientific, Canton, MI.
- Reese, D. E., and G. M. Hill. 2010. Trace minerals and vitamins for swine diets. In: D. J. Meisinger, editor, *National Swine nutrition guide*. U.S. Pork Center of Excellence is the publisher, Ames, IA. p. 37–52.
- Reinhard, M. K., D. C. Mahan, B. L. Workman, J. H. Cline, A. W. Fetter, and A. P. Grifo Jr. 1976. Effect of increasing dietary protein level, calcium and phosphorus on feedlot performance, bone mineralization and serum mineral values with growing swine. *J. Anim. Sci.* 43:770–780.
- Rortvedt, L. A., and T. D. Crenshaw. 2012. Expression of kyphosis in young pigs is induced by a reduction of supplemental vitamin D in maternal diets and vitamin D, Ca, and P concentrations in nursery diets. *J. Anim. Sci.* 90:4905–4915.
- Salas, E. 2011. Case report: Hypovitaminosis D in a swine herd. In: *Proc. 52nd Ann. George A Young Swine Health and Management Conf.* Sioux City, NE. p. 1–5.
- Schadt, H. S., R. Gössl, N. Seibel, and C. P. Aebischer. 2012. Quantification of vitamin D<sub>3</sub> in feed, food, and pharmaceuticals using high-performance liquid chromatography/tandem mass spectrometry. *J. AOAC Int.* 95:1487–1494.
- Schröder, B., R. Kaune, C. Schlumbohm, G. Breves, and J. Harmeyer. 1993. Evidence for vitamin D during pregnancy or lactation? *Am. J. Clin. Nutr.* 59:484S–490S.
- Specker, B. L., M. L. Ho, A. Oestreich, T. A. Yin, Q. M. Shui, X. C. Chen, and R. C. Tsang. 1992. Prospective study of vitamin D supplementation and rickets in China. *J. Pediatr.* 120:733–739.
- Viganò, P., S. Mangioni, F. Pompei, and I. Chiodo. 2003. Maternal-conceptus cross talk—A review. *Placenta* 24:S56–S61.
- Wahlstrom, R. C., and D. E. Stolte. 1958. The effect of supplemental vitamin D in rations for pigs fed in the absence of direct sunlight. *J. Anim. Sci.* 17:699–705.
- Witschi, A.-K. M., A. Liesegang, S. Gebert, G. M. Weber, and C. Wenk. 2011. Effect of source and quantity of dietary vitamin D in maternal and creep diets on bone metabolism and growth in piglets. *J. Anim. Sci.* 89:1844–1852.