The Vitamin D Conundrum[1] [2]

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Summary

Vitamin D is an important nutrient for pigs, necessarily required to maintain bone formation and development in association with calcium and phosphorus. However, pigs in confinement housing are born with low serum vitamin D concentrations, which may result in vitamin D deficiency and related problems. Some of the nursery death loss in routine production the past 2 years has responded to oral vitamin D supplementation leading to the postulate that modern production practices result in vitamin D-deficient pigs at weaning. Therefore, the ability to enhance the vitamin D status of pigs during lactation could potentially prevent some nursery problems related to vitamin D status of the pigs. Three experiments were conducted to investigate the effect of vitamin D, as well as vitamins A and E, administration to sows or newborn pigs on serum vitamin status. In Experiments 1 and 2, newborn pigs were administered vitamin D (with variable vitamin A and E addition) by oral gavage or intramuscular injection. In Experiment 3, gestating sows were injected with a vitamin D/A/E product at 2 week pre-partum. Serum vitamin concentrations of sows and piglets were then monitored in all experiments. In both Experiment 1 and 2, when administrated vitamin D by oral gavage or intramuscular injection, pigs had increased serum 25-hydroxycholecalciferol (25-OH D_3) concentration ($P < 0.01$). Additionally, the injectable administration was more efficient to enhance vitamin D status of pigs than oral administration in both studies ($P < 0.05$). Regarding oral administration of vitamin D, serum 25-OH D_3 concentration was higher in the pigs treated with vitamin D_3 only compared to those treated with vitamin D_3 and vitamin A and/or E (always numerically, statistically [$P < 0.01$] in Experiment 1). In Experiment 3, when sows were injected with the vitamin D product pre-partum, serum 25-OH D_3 concentrations of sows at farrowing ($P < 0.01$), and their progeny at birth ($P < 0.01$) and weaning ($P < 0.05$), were increased. In conclusion, these results clearly demonstrated that vitamin D_3 supplementation to newborn pigs can improve vitamin D status by increasing serum 25-OH D_3 concentration regardless of administration routes and an improvement of maternal vitamin D status by injecting pre-partum affected that status of their offspring. If continued research demonstrates that the serum levels of 25-OH D_3 are critical in weanling pigs, a variety of means to increase those levels are available to producers.

Introduction

Vitamin D, the "sunshine vitamin", is one of the fat soluble vitamins. It is considered a steroid hormone, and plays important roles in bone strength and structure, immune regulation, and hormonal responses in the animal body. In its involvement in growth and bone development, not only is it related to calcium and phosphorous metabolism but also regulation of their absorption and homeostasis.

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Vitamin D has not been a major issue in the swine industry for several decades but in the past few years attention on vitamin D and its status of pigs on the farm has risen with the increasing occurrence of metabolic bone diseases in some production settings. The vitamin D status of pigs is clearly affected by access to sunshine (Table 1) and it is postulated that modern confinement production systems in which pigs do not have access to sunshine may be predisposing pigs to vitamin D deficiency. The main symptoms of vitamin D deficiency are metabolic bone diseases such as rickets, osteomalacia, and osteoporosis which are related to failure in bone mineralization and/or loss of bone mineral in pigs of various ages. Recently, low levels of serum vitamin D have been identified in peri-weaning failure to thrive syndrome (PFTS), noted by veterinarians and researchers since 2008 (Huang et al., 2011). Therefore, means to prevent pigs from metabolic bone diseases, prevent mortality, and maintain normal growth and bone development of pigs are of interest.

Vitamin D for Pigs

Vitamin D requirement for pigs (requirement, deficiency, and toxicity)

NRC (1998) has provided the vitamin D requirement estimates for pigs with the amount of vitamin D_3 for each growth phase or reproductive cycle. The vitamin D requirements ranged from 220 IU/kg diet and 200 IU/kg diet for nursing and weaning pigs, respectively, to 150 IU/kg diet for growing-finishing pigs. For all breeding pigs including gestating and lactating sows as well as boars, the requirement estimate was 200 IU/kg diet. In the recently released NRC (2012) the requirement estimate for gestating and lactating sows was raised to 800 IU/kg diet.

Historically, the major symptom of vitamin D deficiency in young and growing pigs has been rickets (a failure in adequate bone mineralization which results in bowed legs and, at times, spinal deviances) and in adult pigs has been osteomalacia (a demineralization of formed bone which can eventually lead to bone fractures).

When pigs are supplied with vitamin D in excess of requirement through either long- or short-term feeding, toxic symptoms of vitamin D are observed such as reduced feed intake, growth rate, and liver weight as well as hypercalcemia and soft tissue calcification of heart, kidney and lung (NRC, 1987; NRC, 1998). Toxicity of vitamin D_3 (cholecalciferol) is higher than that of vitamin D_2 (ergocalciferol; NRC, 1987). According to the NRC (1987), the maximum safe level is 33,000 IU/kg diet for an exposure duration of less than 60 days, but 2,200 IU/kg diet is the maximum tolerable level for over 60 days of exposure. However, vitamin D toxicity occurs rarely in pigs.

Sources of vitamin D for pigs

Because it is fat soluble, vitamin D absorbed is stored in body fat or transported into liver. Rungby et al. (1993) reported the plasma and tissue distribution of hydroxylated vitamin D metabolites (1,25-$(OH)_2D_3$, 25-OH D_3 and 25-OH D_2) in 50 kg pigs fed 1,000 IU of cholecalciferol/kg diet from weaning to slaughter. In this study, tissue concentrations of 1,25-$(OH)_2D_3$ in the liver, kidney, and adipose tissues were higher than plasma concentration whereas 25-OH D_3 concentration in tissues were lower than in plasma. These results mean the circulating metabolite of vitamin D is 25-OH D_3 but the stored or active form of vitamin D is 1,25-$(OH)_2D_3$. Vitamin D can be provided to pigs by any of the various forms of vitamin D_3 (cholecalciferol or 25-OH D_3) but the effectiveness of the forms should be demonstrated for improving vitamin D status of pigs. In a recent study of Witschi et al. (2011), when dietary vitamin D was supplemented in maternal and creep diets, the piglets from sows fed the diet supplemented with 25-OH D_3 showed remarkably improved serum vitamin D status. Another study of vitamin D sources reported the supplementation of 25-OH D_3 to the gestating and lactating

sows was more efficient than that of vitamin D_3 supplementation (Lauridsen et al., 2010). Based on these results, 25-OH D_3 may be a more efficient source to improve vitamin D status of sows and pigs than vitamin D_3. On the other hand, Jakobsen et al. (2007) reported the equal efficiency between both vitamin D_3 and 25-(OH) D_3 when growing-finishing pigs were fed diets containing either vitamin D source or a mixture of vitamin D_3 and 25-OH D_3. However, 25-OH D_3 supplementation did not produce vitamin D_3 in the body tissues of pigs such as liver, longissimus dorsi muscle (loin) and subcutaneous fat of loin because the reaction to hydroxylate vitamin D_3 to 25-OH D_3 by 25-hydroxylase is not reversible. Finally, while vitamin D_3 and its metabolites such as 25-OH D_3 and 1,25-$(OH)_2D_3$ are possible to use as supplemental vitamin D to pigs, 1,25-$(OH)_2D_3$ is not useful due to a short plasma half-life of about 4 to 8 hours compared to other sources (vitamin D_3: 5 to 7 days; 25-OH D_3: 20 to 30 days, McDowell, 2000).

Effects of Vitamin D Supplementation to Pigs

Weaning, and growing-finishing pigs

Studies have been conducted to observe the effects of vitamin D (25-OH D_3, vitamin D_3, and vitamin D_2) supplementation in swine diets in association with calcium and phosphorous (Combs et al., 1966; Foley et al., 1990; Li et al., 1998; O'Doherty et al., 2010). Based on these results, vitamin D supplementation to pig diets did not affect the growth performance but had partially positive influences on calcium digestibility.

Sows and offspring

Abbott and Madson (2012) suggested a vitamin D reference range for pigs and they also provided actual serum vitamin D (25-OH D_3) values of pigs in several settings. Lower serum vitamin D concentrations were observed in pigs raised indoors compared to the reference range but not in pigs housed outdoors (Table 1). The impact of ultraviolet exposure on serum values of pigs in the outdoor housing environment compared to that of pigs raised indoors was marked.

Piglets have the lowest level of serum vitamin D at birth (Horst and Littledike, 1982). Nursing pigs in outdoor settings can acquire vitamin D via two sources - one synthesized by sunlight and the other from sow's milk, whereas pigs in confinement housing can acquire vitamin D through only sow's milk. However, sow's milk contains little vitamin D and placental transport of vitamin D is poor. Nonetheless, because fetal and nursing pigs are provided vitamin D via placental transfer and sow's milk, the maternal vitamin D status is extremely important to supply an adequate amount of vitamin D for piglets. Goff et al. (1984) therefore suggested that the parenteral vitamin D_3 treatment before parturition by intramuscular injection of vitamin D_3 to sows at 20 day pre-partum was an effective method for enhancing vitamin D status of piglets because the vitamin D status of sows is closely correlated to that of fetus and neonatal piglets.

Several recent studies have been conducted to investigate vitamin D supplementation/ administration to sows and piglets (Lauridsen et al., 2010; Witschi et al., 2011; Flohr et al., 2012; Rortvedt et al., 2012). The supplementation of vitamin D to gestation and lactation diets was demonstrated to improve serum vitamin D concentration of sows (Lauridsen et al., 2010). Similarly, Witschi et al. (2011) reported that piglets from sows that received 25-OH D_3 had higher serum 25-OH D_3 concentration at d 21, d 33, and d 77 postpartum. Regarding vitamin D supplementation of piglets, when the newborn piglets were supplemented with vitamin D via oral administration at 40,000 and 80,000 IU of vitamin D_3, linear increases of serum 25-OH D_3 concentration were detected on d 10 and 20 of age but there were no significant differences on growth performance or

bone measures (Flohr et al., 2012). Similar results were reported by Rortvedt et al. (2012) wherein pigs orally administrated vitamin D_3 at 40,000 IU had higher serum 25-OH D_3 concentrations than those without vitamin D_3 administration but growth performance and bone mineralization were not affected. Based on these results, vitamin D status of piglets can be improved via several efficient methods such as oral administration to piglets, supplementation to maternal feed and injection to sows. However, the relationship of that improved serum status to performance or bone measures is not always present.

The objective of the current research effort was to evaluate the effects of administration routes and a variety of supplemental products administered to young pigs as well as the efficacy of vitamin D_3 injection to sows pre-partum.

Materials and Methods

Experiment 1: A total of 32 pigs (Yorkshire × Duroc) were used from 4 litters of pigs (i.e., 8 pigs/sow). Within each litter, 2 pigs were assigned to 4 treatments. Treatments were: 1) control: no supplemental vitamin D_3, 2) oral administration of 0.8 ml of a vitamin complex (EMCELLE NEWBORN EAD containing 500 IU of vitamin E, 50,000 IU of vitamin A, and 50,000 IU of vitamin D_3 per ml), 3) oral administration of 1.0 ml of a vitamin D product (WEAN-D containing 40,000 IU of vitamin D_3 per ml), and 4) intramuscular injection of 0.8 ml vitamin complex (VITAL E-NEWBORN containing 500 IU of vitamin E, 50,000 IU of vitamin A, and 50,000 IU of vitamin D_3 per ml). All pigs in Treatments 2 - 4 were administered 40,000 IU of vitamin D_3. The commercial products used were from Stuart Products Inc. (Bedford TX; the EMCELLE NEWBORN EAD and VITAL E-NEWBORN products) and from GlycoMyr Inc. (Ames IA; the WEAN-D product).

Experiment 2: A total of 45 pigs (Yorkshire × Duroc) were used from 4 litters of pigs. Within each litter, pigs were assigned to 7 treatments; and following the initial assignment of pigs, any remaining pigs in each litter were allotted to Treatments 1 and 2. Treatments were: 1) control: no supplemental vitamin D_3, 2) intramuscular injection of 1.0 ml vitamin complex (VITAL E-NEWBORN containing 500 IU of vitamin E, 50,000 IU of vitamin A, and 50,000 IU of vitamin D_3 per ml) 3) oral administration of 1.25 ml vitamin D (WEAN-D containing 40,000 IU of vitamin D_3 per ml), 4) oral administration of 0.60 ml of vitamin D_3 (EMCELLE D3 containing 84,500 IU of vitamin D_3), 5) oral administration of 1.66 ml vitamin D and E complex (EMCELLE ED3 containing 30,000 IU of vitamin D_3 and 500 IU of vitamin E), 6) oral administration of 1.66 ml vitamin E (EMCELLE E containing 500 IU of vitamin E), 7) oral administration of 1.00 ml vitamin complex (EMCELLE NEWBORN E-A-D containing 500 IU of vitamin E, 50,000 IU of vitamin A, and 50,000 IU of vitamin D_3 per ml,). All pigs receiving supplemental vitamin D received about 50,000 IU of vitamin D_3. The products used were from Stuart Products Inc. (Bedford TX; the VITAL E-NEWBORN, EMCELLE D3, EMCELLE ED3, EMCELLE E, and EMCELLE NEWBORN EAD products) and from GlycoMyr Inc. (Ames IA; the WEAN-D product).

In both experiments, the gestation and lactation diets contained the following per kg diet: vitamin A, 6,600 IU; vitamin D_3, 880 IU; vitamin E, 44 IU; vitamin K (menadione sodium bisulfite complex), 6.6 mg; riboflavin, 8.8 mg; d-pantothenic acid, 22 mg; niacin, 44 mg; vitamin B_6, 4.4 mg; vitamin B_{12}, 33 ug; d-biotin, 220 ug; and folic acid, 1,320 ug. All sows with pigs were kept in individual farrowing crates in an environmentally controlled farrowing facility without windows. Sows were provided 1.8 – 2.5 kg of the gestation diet before being brought to the farrowing rooms at about d 112 of gestation. Sows were provided the lactation diet *ad libitum* and water was freely available from a water nipple throughout the experimental period. All pigs were processed at birth (within 15 hr) and assigned to a treatment. Processing of the piglets involved weighing, ear-notching, needle teeth clipping, and iron injection with 100 mg Fe as Fe dextran. With regard to administration of treatments, pigs were

administered vitamin D by either injection or oral gavage. Injectable products were provided to each pig in the neck muscle on the opposite side of where the iron injection was given. Orally administered treatments were provided through a plastic tube attached to a 3 mL syringe into which the proper dosage had been drawn. The body weights of the pigs were also recorded about d 10, at weaning and 14 d postweaning to calculate growth performance. In addition, the body weights of sows were obtained at farrowing and weaning to determine weight change of sows during lactation.

Blood samples of sows were taken from the anterior vena cava at farrowing and weaning Blood samples of pigs were collected from the anterior vena cava at d 0 (before administration of any treatments), about d 10 post-administration of treatments, and weaning. Blood samples were centrifuged for 15 minutes at 4°C; serum samples were then aliquoted into microtubes and stored at -20°C until analysis. Serum samples were sent to the Iowa State University Veterinary Diagnostic Laboratory for vitamin assay.

Experiment 3: A total of 24 PIC sows (average parity: 5.2) was used in this experiment. Treatments were divided into a control group without injection and a group that received a 5 ml injection of a vitamin D_3 product (VITAL E-Hi A+D, Stuart Products Inc., Bedford, Texas, USA) containing 100,000 IU of vitamin D_3, 300 IU of vitamin E, and 200,000 IU of vitamin A per ml at 2 week pre-partum.
All sows with pigs were housed in individual farrowing crates in an environmentally controlled farrowing facility. The sows were provided a common gestation diet containing 2,750 IU/kg of vitamin D_3, as well as a common lactation diet that also contained 2,750 IU/kg of vitamin D. Water was freely available throughout the experimental period. The injectable product was administered into a neck muscle and sows were bled immediately preceding administration of the product and again at parturition. Two piglets were selected from each litter at birth for blood sampling; one of the two selected piglets was bled again at weaning. Blood samples were centrifuged for 15 minutes; serum samples were put into microtubes and submitted to the Iowa State University Veterinary Diagnostic Laboratory Serum for analysis of 25-OH D_3 concentrations.

All data analyses were conducted with the GLM procedure of SAS (SAS Inst. Inc., Cary, NC) with individual pig as the experimental unit. Least square mean separations utilized the PDIFF option.

Results and Discussion

The effect of vitamin D_3 supplementation to newborn pigs in Experiment 1 on serum 25-OH D_3, retinol, and α-tocopherol concentrations is shown in Table 2. At d 10 after administration, all groups treated with a vitamin D_3 product had higher serum 25-OH D_3 concentration than the control group ($P < 0.01$). Additionally, when the pigs received the injectable product, the serum 25-OH D_3 concentration was the highest among treatments ($P < 0.01$). At weaning, serum values for all treatments had declined from d 10 but serum values for two of the three treatments remained elevated relative to serum values for the control pigs. The decline in all values from d 10 to weaning demonstrates that sow milk is not a good source of vitamin D and serum values are presumably diluted with the increase in blood volume associated with the rapid growth of the pigs. It should be noted that serum values for control pigs were higher at d 10 than at birth which suggests that the colostral contribution to the pig is significant. With regard to serum retinol and α-tocopherol concentrations, there were no significant differences except at d 10 when serum α-tocopherol concentrations of pigs in the injection group was the highest among all treatments ($P < 0.01$).
In Experiment 2 (Table 3), the effect of vitamin D and variable vitamin A and E administration to newborn pigs on serum 25-OH D_3 concentrations demonstrated again that the d 10 serum concentrations can be increased by either oral or injectable administration, that values decline from d 10 to weaning regardless of treatment, and that values at weaning can remain higher than those of

control pigs in which no treatment is administered. As would be expected, the injectable product resulted in the highest serum values but the results were not significantly higher than several of the oral administration treatments. In the case of α-tocopherol, the vitamin E injection to piglets was also more effective to increase serum α-tocopherol concentration at d 10 after administration ($P < 0.01$). However, at weaning the highest numerical value was actually from one of the oral products and it, as well as the injectable product, was significantly higher than that of the control pigs. These results, collectively, demonstrate that the vitamin D_3 status of pigs can be improved by administration of vitamin D_3 by a variety of methods. And the results agree with those of recent studies (Flohr et al., 2012; Rortvedt et al., 2012).

Comparing the results between oral and injectable methods in the present experiments, it was demonstrated that the intramuscular injection of vitamin D and E was more efficient to enhance those statuses than oral administration. There have been few studies to compare the efficacy of different vitamin administration routes to pigs. In the case of sheep, ewes that received vitamin D_3 by intramuscular injection had higher plasma 25-OH D_3 concentration and more lasting effects than those by oral administration (Hidiroglou et al., 1984). The parenteral administration of vitamin D_3 does not require the process of intestinal absorption that oral administration of vitamin D_3 requires. It has been documented that only 50% of orally administered vitamin D is absorbed (McDowell, 2000). Therefore the process of intestinal absorption of vitamins probably affected the difference of serum 25-OH D_3 and α-tocopherol concentration in this study.

The effects of the treatments on body weight and growth in these experiments are provided in Tables 5 and 6. No significant differences among treatments were detected on body weight and average daily gain of pigs during suckling and nursery periods in either experiment which is undoubtedly a function of the limited number of observations in each experiment. However, these results again agree with some recent research (Flohr et al., 2012). In contrast, Rortvedt et al. (2012) reported a slight improvement of ADG in pigs administered a single oral mega-dose of vitamin D_3 at birth.

In Experiment 3, a vitamin D_3 injection to gestating sows increased serum 25-OH D_3 concentrations and its change from pre-administration values ($P < 0.01$, Table 4) as would be expected. Piglets from sows that received the pre-partum vitamin D_3 injection had higher serum 25-OH D_3 concentrations than those in the control group ($P < 0.01$) at birth and this improvement was maintained until weaning ($P < 0.05$). This result means the maternal vitamin D status affects the status of offspring at birth and it may result from the placental transportation which agrees with the observations of Goff et al. (1984) who reported that when sows received vitamin D_3 by intramuscular injection at 20 day pre-partum, vitamin D status of their progeny was improved. Even though the difference in the serum 25-OH D_3 levels of piglets between treatments was maintained until weaning there was no significant difference in the magnitude of that treatment difference (or the change from birth to weaning) of 25-OH D_3 concentration in serum of piglets which means the improvement of vitamin D_3 status of gestating sows probably did not influence sow milk content. It may be noted that weaning serum values of control piglets in Experiment 3 were double that of the values at birth and that sow supplementation values were about tripled that of sows in Experiments 1 and 2. Therefore, while milk may not be one of the best means of supplying vitamin D3, it certainly would seem adequate to increase serum values of piglets if sows are supplemented at an appropriate level.

Summary

This study clearly demonstrated that vitamin D_3 supplementation to newborn pigs can improve vitamin D status by increasing serum 25-OH D_3 concentrations regardless of administration routes. The vitamin A status of pigs was not affected by any of the means of administration whereas the

vitamin E status was affected by both routes of administration but more so by injection. The improvement of vitamin D status of pregnant sows can result in the enhancement of its status of their offspring. Thus, several products and modes of administration exist to modify vitamin D status of piglets if that is a matter of concern for a swine operation. Further studies are needed, however, to determine if this altered vitamin D status has value in different production settings.

Table 1. Reference range of serum vitamin D concentration (ng/mL) of pigs.

Phase of production	Reference range	Jan Inside	June Inside	Outdoor
Newborn	5-15	-	-	-
10 day old pig	8-23	-	-	-
3-4 weeks old	25-30	8.42	13.75	58.54
Grower	30-35	21.80	18.04	61.03
Finisher	30-35	27.66	28.18	85.98
Mature animals	35-70	35.70	45.42	-
Pregnant sows	35-100	-	-	-

Source: Abbott and Madson, 2012.

Table 2. Effect of vitamin administration to pigs on serum 25-OH D_3, retinol, and α-tocopherol concentration (Exp. 1).

		Treatments[1]					P-value
Criteria	Sow	Control	Oral vit. ADE	Oral vit. D	Inj. vit. ADE	SEM[2]	Trt
25-OH D_3, ng/ml							
Birth	21.23	2.60	2.65	2.51	2.67	0.11	0.741
d 10		9.24^{d}	37.71^{c}	60.56^{b}	81.86^{a}	4.81	<0.0001
Weaning	31.60	5.97^{b}	9.99^{b}	22.47^{a}	30.72^{a}	3.57	0.0004
Retinol, ng/ml							
Birth		0.16	0.14	0.12	0.13	0.01	0.081
d 10		0.37	0.27	0.32	0.37	0.05	0.470
Weaning		0.35	0.19	0.24	0.20	0.05	0.186
α-tocopherol, ng/ml							
Birth		2.25	1.83	2.23	1.83	0.32	0.662
d 10		6.85^{b}	3.76^{b}	7.10^{b}	15.20^{a}	1.66	0.007
Weaning		3.93	3.74	4.55	5.93	0.82	0.288

[1] See text for more complete description of treatments.
[2] Standard error of mean.
[a-d] Means without a common superscript differ (P<0.01).

Table 3. Effect of vitamin administration to pigs on serum 25-OH D_3, retinol, and α-tocopherol concentration (Exp. 2).

		Treatment[1]								P-value
Criteria	Sow	Control	Inj. AD_3E	Oral D_3 (1)	Oral D_3 (2)	Oral D_3E	Oral E	Oral AD_3E	SEM[2]	Trt
25-OH D_3, ng/ml										
Birth	26.45	4.41	4.47	4.33	4.38	5.45	5.10	4.40	0.80	0.954
d 10		8.25^{b}	110.74^{a}	91.43^{a}	86.20^{a}	84.68^{a}	6.47^{b}	77.17^{a}	16.21	<0.001
Weaning	32.65	5.02^{c}	40.24^{a}	34.50^{ab}	30.88^{ab}	27.30^{ab}	5.10^{c}	26.10^{b}	5.02	<0.001
Retinol, ng/ml										
Birth	0.16	0.04	0.07	0.12	0.10	0.08	0.10	0.11	0.02	0.132
d 10		0.23	0.25	0.36	0.26	0.21	0.28	0.25	0.05	0.588
Weaning	0.27	0.18	0.16	0.22	0.11	0.16	0.18	0.17	0.05	0.850
α -tocopherol, ng/ml										
Birth	1.23	3.24	2.86	3.05	2.70	4.30	1.43	3.68	1.49	0.952
d 10		4.78^{b}	11.26^{a}	4.68^{b}	4.80^{b}	6.93^{b}	5.80^{b}	5.37^{b}	1.54	0.001
Weaning	3.13	3.86^{b}	7.16^{a}	4.38^{b}	3.85^{b}	7.33^{a}	4.80^{ab}	5.93^{ab}	0.92	0.004

[1] See text for more complete description of treatments.
[2] Standard error of mean.
[a, b, c] Means without a common superscript differ (P<0.01).

Table 4. Effect of vitamin administration to sows on serum 25-OH D_3 concentration of sows and piglets (Exp. 3).

Criteria	Control		Vitamin D_3 Injection		SEM[1]	P-value
Sows, ng/ml						
Initial[2]	42.07	(n=11)	37.10	(n=13)	2.612	0.1923
At farrowing	43.10	(n=11)	58.60	(n=13)	2.861	0.0009
Change	1.03	(n=11)	21.50	(n=13)	3.192	0.0002
Piglets, ng/ml						
At birth	4.00	(n=22)	6.15	(n=26)	0.357	0.0001
At weaning	9.67	(n=11)	12.05	(n=12)	0.807	0.0498
Change	5.67	(n=11)	5.53	(n=12)	0.798	0.9029

[1] Standard error of mean.
[2] Before injection at 2 week prepartum.

Table 5. Effect of vitamin D_3 administration to pigs on growth performance (Exp. 1)

		Treatment[1]					P-value
Criteria	Sow	Control	Oral vit. ADE	Oral vit. D	Inj. vit. ADE	SEM[2]	Trt
Body weight, kg							
Birth	231.03	1.71	1.47	1.60	1.61	0.19	0.317
d10		4.16	4.02	3.82	4.00	0.47	0.560
Weaning	242.02	6.26	6.15	5.67	5.98	0.75	0.513
14 d post-weaning		11.13	10.61	10.12	10.33	1.04	0.321
ADG, kg/d							
Birth to d10		0.21	0.22	0.19	0.20	0.03	0.608
d10 to weaning		0.27	0.28	0.24	0.26	0.04	0.780
Birth to weaning		0.23	0.24	0.21	0.22	0.04	0.540
14 d in nursery		0.35	0.32	0.32	0.31	0.04	0.355

[1] See text for more complete description of treatments.
[2] Standard error of mean.

Table 6. Effect of vitamin D_3 administration to pigs on growth performance (Exp. 2).

		Treatment[1]						
	Sow	Control	Inj. AD_3E	Oral $D_3(1)$	Oral $D_3(2)$	Oral D_3E	Oral E	Oral AD_3E	SEM[2]
Body weight, kg									
Initial	229.5	1.56	1.55	1.52	1.58	1.60	1.83	1.51	0.169
d 10		3.51	3.27	3.78	3.87	3.05	4.37	3.84	0.453
Weaning	248.2	5.53	5.12	5.99	6.44	4.12	6.90	5.84	0.728
ADG, kg									
Birth to d 10		0.17	0.16	0.21	0.21	0.14	0.24	0.19	0.030
d 10 to weaning		0.25	0.23	0.26	0.31	0.14	0.28	0.25	0.041
Birth to weaning		0.20	0.19	0.23	0.25	0.14	0.26	0.21	0.033

[1] See text for more complete description of treatments.
[2] Standard error of mean.

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NOTES

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