ABSTRACT: In swine nutrition, little is known about vitamin requirements for reproductive processes and bone health, especially vitamin D. Supplemental vitamin D is usually added to animal feed as cholecalciferol (vitamin D3), which is transported to the liver and hydroxylated to 25-hydroxycholecalciferol (25(OH)D3), and this metabolite has become commercially available for swine nutrition. Recently, the official vitamin D requirement for gestating and lactating swine was increased from 200 to 800 IU vitamin D/kg feed. The purpose of the present paper was to review the main findings of a published study, which has contributed to the basis for this establishment, and to put them into context with the existing literature. In this study, a dose–response trial with 4 doses of both vitamin D3 and 25(OH)D3 was performed with breeding swine and consisted of 2 experiments: In Exp. 1, 160 gilts from first estrus until d 28 of gestation were fed diets containing 4 concentrations of 1 of 2 vitamin D sources (i.e., 200, 800, 1,400, or 2,000 IU/kg from cholecalciferol or corresponding levels of 5, 20, 35, or 50 μg/kg from 25(OH)D3 [Hy-D]). Concurrently in Exp. 2, the same 8 dietary treatments were fed to 160 multiparous sows from the first day of mating until weaning. Dietary treatments of ≥800 IU/kg feed showed beneficial effects for breeding swine in terms of bone mineral content and ultimate strength, decreased number of still born piglets, and greater vitamin D status in comparison with dietary treatments of 200 IU/kg of feed. In addition, using the Hy-D resulted in greater concentrations of plasma 25(OH)D3 when fed at equal amounts (weight) of vitamin D3 but depended on the level tested. Above 200 IU/kg feed, 25(OH)D3 resulted in greater concentrations in plasma than vitamin D3 and could as such been considered as an equivalent or even more advantageous dietary source of vitamin D. In conclusion, this study, together with other recently published studies, addressed the nutritional benefits of vitamin D dose and forms for gestating and lactating sows and their offspring in terms of vitamin D status, reproduction, transfer to the neonate, and bone health.

Key words: bone health, piglets, reproduction, sows, 25-hydroxycholecalciferol

INTRODUCTION

Estimation of vitamin requirements in swine nutrition has traditionally been assessed to prevent deficiency rather than estimating levels required for optimal production. From research in other animal species and humans, it is known that vitamin D has several biologically functions related to reproduction, bone health, and immune modulation, which may also be of interest for optimal swine production. With regard to sow nutrition, little information is available on the requirement for vitamin D, and former recommendations concluded that there were insufficient data to use as a basis for an estimate of the requirement of breeding pigs (NRC, 1998). The general consideration is that swine have no requirement for vitamin D when exposed to sunlight; however, because pigs housed in confinement systems often do not have access to sunlight, they are entirely dependent on dietary supplementation. The official vi-
Vitamin D requirement for gestating and lactating swine ranged from 200 (NRC, 1998) to 1,000 IU/kg feed (British Society of Animal Science, 2003). The purpose of the present paper is to review the knowledge that has formed the basis for the establishment of the recent (NRC, 2012) vitamin D requirement in swine nutrition. Hence, 800 IU vitamin D/kg feed has been recommended for gestating and lactating sows.

**SOURCES AND CIRCULATING FORMS**

**Sources of Vitamin D**

Vitamin D is closely associated with sunlight in the pig as the provitamin 7-dehydrocholesterol yields the active cholecalciferol on exposure of the skin to sunlight. However, the source is not relevant in most production systems where exposure of pigs to sunlight is lacking. The concentration of plasma vitamin D and most of its metabolites in pigs exposed to sunlight was 2.2 to 20.3 times the concentration of pigs kept in confinement (Engstrom and Littledike, 1986). Most feed for pigs, with exception of fish meal, contain little or no vitamin D, and the vitamin is generally supplied in the form of fish-liver oils or synthetic preparations. Vitamin D exists in the forms of vitamins D2 (i.e., ergocalciferol) and D3 (i.e., cholecalciferol), but in terms of pig nutrition (Horst and Littledike, 1982), vitamin D3 is the most potent one. Vitamin D3 is hydroxylated to 25-hydroxycholecalciferol (25(OH)D3) in the liver, and this metabolite is the major circulating form in the body. This form is further hydroxylated in the kidneys to the hormonally active form, 1,25-dihydroxycholecalciferol (1,25(OH)2D3), which is normally present at levels of approximately 1% of 25(OH)D3 in plasma. Tissue concentration of 25(OH)D3 in fat, liver, and intestinal mucosa was low in pigs (less than one-third of plasma levels) whereas tissue concentrations of 1,25(OH)2D3 exceeded plasma levels by a factor of 3 to 7 with adipose tissue concentrations being the greatest (Rungby et al., 1993). Furthermore, it is known that the concentration of 1,25(OH)2D3 in plasma is greater in young animals than in adult animals (Horst and Littledike, 1982). Vitamin D3 in the form of 25(OH)D3 is commercially available in a synthetic form (Hy-D; DSM Nutritional Products A/S, Basel, Switzerland), which is immediately available to the blood stream on intake. Hence, the plasma concentrations of 25-hydroxy-vitamin D are greater when Hy-D is provided in the feed rather than when fed equal amounts (by weight) of vitamin D3, and this has been demonstrated in recent reports on pigs (Witschi et al., 2011; Coffey et al., 2012), as a dramatic increase in circulating concentrations of 25(OH)D3 has been shown on supplementation of swine with the Hy-D. The mechanism for this is not completely clear; however, it has been demonstrated in young broiler chickens that 25(OH)D3 is absorbed more efficiently than vitamin D2 in the upper portion of the intestine (Bar et al., 1980, 2003). The potency, however, seems to be dependent on the amount tested. In the study by Lauridsen et al. (2010) in which levels of 200, 800, 1,400, or 2,000 IU/kg were tested, it was concluded that above 200 IU/kg feed, Hy-D resulted in greater concentrations of plasma 25(OH)D3 when fed at equal amounts (by weight) of vitamin D3.

**Assessment of Vitamin D Status**

The Institute of Medicine has concluded (IOM, 2011) that serum 25(OH)D3 levels are considered to be the most useful marker of vitamin D exposure; however, the committee was cognizant of its limitations as a biomarker of effect. As shown in previous studies on swine (Wilborn et al., 2004) and humans (Heaney et al., 2003), plasma 25(OH)D3 reflected the dietary dose of vitamin D. It has been shown for humans that for every 1 μg (i.e., 40 IU) of vitamin D intake, circulating 25(OH)D3 increased by 0.28 ng/mL over 5 mo on a given supplemental regimen (Hollis and Wagner, 2004). Vitamin D deficiency in humans is defined as values below 10 ng 25(OH)D3/mL (Mosekilde, 2005, and to obtain this plasma concentration in swine, more than 1,400 IU of vitamin D3 per kilogram of feed was necessary (Lauridsen et al., 2010). This conclusion was obtained on the basis of a dose–response trial in which dietary treatments containing 4 concentrations of 1 of the 2 different vitamin D sources (i.e., 200, 800, 1,400, or 2,000 IU/kg from cholecalciferol or corresponding to 5, 20, 35, and 50 μg/kg feed from 25(OH)D3 [Hy-D]) was supplemented to gilts and sows. However, as shown in Table 1, the plasma concentration of 25(OH)D3 was greater when Hy-D was provided. Provision of 200 IU vitamin D/kg feed, irrespective of source, provided concentrations of 25(OH)D3, which could be considered critical (Lauridsen et al., 2010).

**Deficiency and Toxicity of Vitamin D in Swine**

Vitamin D deficiency reduces retention of calcium, phosphorus, and magnesium (Miller et al., 1965), and in mature swine, a mild deficiency reduced bone mineral content (osteomalacia) whereas vitamin D deficiency in young growing pigs may result in rickets. In severe vitamin D deficiency, pigs may exhibit signs of calcium and magnesium deficiency, including tetany. In practical swine production, a plasma concentration below 10 to 15 ng 25(OH)D3/mL is considered as deficient; however, it should be underlined that there are no scientific data available to confirm that this level is actually deficient.

Vitamin D toxicity may cause hypercalcaemia (Bille et al., 1976), and in the study on acute toxicosis of vitamin D
by Long (1984), some gross necropsy findings consistently observed were hemorrhagic gastritis and diffuse interstitial pneumonia. In this study, the concentration of vitamin D$_3$ and 25(OH)D$_3$ in serum of finishing hogs and replacement gilts ingesting excessive vitamin D$_2$ inadvertently were up to 2,015 ng vitamin D$_3$/mL and 1,427 ng 25(OH)D$_3$/mL (Long, 1984). Dietary vitamin D toxicity has also been observed in a household of pot-bellied pigs (Wimsatt et al., 1998) in which 2-yr-old female pigs diagnosed with different vitamin D toxicity signs had concentrations approximately 145 ng 25(OH)D$_3$/mL plasma, which was considerably greater than the normal blood concentration of 25(OH)D$_3$ in swine given as 32 to 117 ng/mL plasma (Wimsatt et al., 1998). In weanling pigs, vitamin D toxicity can be produced when pigs are supplemented with a daily dose of 6,250 μg of vitamin D$_3$ for 4 wk (NRC, 2012). Under short-term feeding conditions (i.e., <60 d), swine can tolerate as much as 33,000 IU vitamin D$_3$/kg of diet (NRC, 1987). Feeding supranutritional concentrations (i.e., 40,000 and 80,000 IU/kg feed) of vitamin D$_3$ for 44 or 51 d before slaughter to investigate the effects on pork quality traits resulted in plasma concentrations above 100 ng/mL (Wilborn et al., 2004). That study concluded that 40,000 IU/kg feed for 44 d can be used to improve pork quality whereas 80,000 IU/kg feed may result in a decreased growth rate of the pigs.

**IMPACT OF VITAMIN D ON MECHANISMS INVOLVED IN REPRODUCTION**

Vitamin D recommendation for sows during gestation and lactation is not based on scientific reports, and, in general, very little evidence is available regarding vitamin D and its metabolites in relation to breeding of pigs. Normal implantation and placentation are critical for a successful pregnancy. In swine, up to 40% of conceptuses are lost before term (Matte and Lauridsen, 2013). Development of the so-called hyperprolific lines of pigs has resulted in a considerable increase in ovulation rate but at the expense of embryonic survival at the beginning of gestation. The loss can be attributed to essential events of implantation, which require a functional normal conceptus, a receptive endometrium, and a communication link between them. Various reproductive organs contain vitamin D receptors, and that vitamin D is important for their normal functioning. Female fertility seems to be markedly reduced in vitamin D-deficient murine models (Vigianò et al., 2003). It has been postulated that local synthesis of 1,25(OH)$_2$D$_3$ may play a role in implantation and/or placentation either through the established immunomodulatory effects of 1,25(OH)$_2$D$_3$ or via the regulation of specific target genes associated with implantation, and overall the influence of vitamin D on the reproductive capacity seems to be linked to calcium-independent mechanisms (Vigianò et al., 2003). Therefore, it is of major interest to better understand the role of vitamin D and its related metabolites on the breeding female pigs.

In a study by Lauridsen et al. (2010), the effect of the dose–response pattern of the 2 vitamin D sources, vitamin D$_3$ and 25(OH)D$_3$, with respect to early pregnancy in sows was investigated. The effect of the dietary treatments on attachment of fetuses to the uterine endometrial epithelium was determined by counting the number of ovulations assessed by counting the corpora lutea and the number of attached and free-floating fetuses. No significant influence of dietary treatment with vitamin D$_3$ or Hy·D could be seen with regard to the early pregnancy measurements of the gilts (Table 2). In this experiment, endometrial biopsies were collected from 2 different implantation sites in the uterus, and mRNA abundance of VDR (nuclear hormone receptor for vitamin D$_3$) and homeobox protein A10 (HoxA10) were analyzed because it has been shown that, in addition to its immunosuppressive actions, vitamin D may influence placental attachment by regulating such key target genes (Vigianò et al., 2003). Human endometrium shows upregulation of HoxA10 expression at the time of implantation (Taylor et al., 1998). Null-mutant mice for the VDR or for the 25-hydroxyvitamin D$_3$–1α-hydroxylase, which is the enzymes that catalyses the synthesis of 1,25(OH)$_2$D$_3$ from the major circulating form of vitamin D, 25(OH)D$_3$, had reduced fertility. Moreover, a link between vitamin D and HoxA10 has been highlighted because 1,25(OH)$_2$D$_3$ is able to stimulate a dose-dependent increase in HoxA10 expression (Rots et al., 1998; Lalwani et al., 2000). No statistical influence of the dietary vitamin D treatments was observed with regard to the relative mRNA abundance of HoxA10 and VDR in the uterus of the gilts (Fig. 1) although a tendency ($P = 0.13$) toward increased HoxA10 in gilts fed vitamin D$_3$ was observed (Lauridsen and Theil, 2011). It should be mentioned that samples from the uterus were obtained at d 28 of gestation whereas attachment of embryos in swine

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### Table 1. Concentration (ng/mL of plasma) of 25(OH)D$_3$ in the plasma of gilts and sows fed varying dose and form of vitamin D$^1$

<table>
<thead>
<tr>
<th>Swine group</th>
<th>Vitamin D$_3$, IU/kg feed</th>
<th>Hy-D$_2$, μg/kg feed</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gilts$^3$</td>
<td>200 800 1,400 2,000</td>
<td>5 20 35 50</td>
<td></td>
</tr>
<tr>
<td>Sows$^4$</td>
<td>12.1 17.9 17.4 26.3</td>
<td>12.4 29.9 50.0 62.3</td>
<td>2.7</td>
</tr>
</tbody>
</table>

$^1$Adapted from Lauridsen et al. (2010).
$^2$Hy-D (25-hydroxycholecalciferol; DSM Nutritional Products A/S, Basel, Switzerland).
$^3$Gilts ($n = 20$ treatment) were provided the dietary vitamin D treatment from mating until d 28 of gestation. Effect ($P < 0.001$) of dose and form.
$^4$Sows ($n = 20$ treatment) were provided the dietary vitamin D treatment from mating until end of lactation (d 28 after farrowing). Data are pooled among blood sampling days (d = –8, 2, 16, and 28 of lactation). Effect ($P < 0.001$) of dose, form, and blood sampling days.
occurs after d 10 after mating, and hence, eventual differences between dietary vitamin D treatments in terms of modulation of vitamin D-dependent genes related to early pregnancy may be difficult to obtain.

In Exp. 2 (Lauridsen et al., 2010), it was observed that dietary treatments of vitamin D influenced the number of stillborn pigs (Table 2) because a decreased number of stillborn piglets was obtained with the high doses of vitamin D (1,400 and 2,000 IU vitamin D yielding 1.17 and 1.13 stillborn piglets per litter, respectively) compared with the low doses of vitamin D (200 and 800 IU vitamin D giving 1.98 and 1.99 stillborn piglets per litter, respectively). In addition, number of total born was affected by a significant interaction among parity, form, and dose of vitamin D. However, no influence of dietary vitamin D was observed with regard to number of suckling piglets at birth and at weaning, and no difference between dietary vitamin D forms and dose was obtained with regard to ADG of litters (Lauridsen et al., 2010). A subsequent study (Coffey et al., 2012) compared a control diet containing 25,000 IU vitamin D3/kg diet with an experimental diet containing 500 IU/kg diet and 50 μg 25(OH)D3/kg diet (Hy·D) and concluded that feeding 25(OH)D3 to first-service gilts before and during gestation improved maternal reproductive performance in terms of a statistically significant improvement in litter size at d 90 of gestation; gilts fed 25(OH)D3 had 12.7 fetuses whereas control gilts had 10.2 fetuses, and the mean fetal weight was not decreased in gilts fed 25(OH)D3 as frequently occurs when litter size is increased. The results obtained from these studies, that is, the smaller number of stillborn piglets obtained with greater dietary doses of vitamin D and likewise the effect for vitamin D source on litter size, may be of practical importance for the swine production especially when using hyperprolific lines of pigs because these lines of swine suffer from high piglet mortality.

Table 2. Reproductive performance in gilts and sows fed varying dose and form of vitamin D1

<table>
<thead>
<tr>
<th>Item</th>
<th>Vitamin D3, IU/kg feed</th>
<th>200</th>
<th>800</th>
<th>1,400</th>
<th>2,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avg no. of fetus</td>
<td>13.4</td>
<td>13.8</td>
<td>14.7</td>
<td>14.7</td>
<td>14.7</td>
</tr>
<tr>
<td>Avg no. of corpora lutea</td>
<td>16.7</td>
<td>16.8</td>
<td>16.7</td>
<td>16.7</td>
<td>16.7</td>
</tr>
<tr>
<td>Implanted fetus, %</td>
<td>80</td>
<td>82</td>
<td>88</td>
<td>88</td>
<td>88</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Item</th>
<th>Hy-D2 μg/kg feed</th>
<th>5</th>
<th>20</th>
<th>35</th>
<th>50</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gilts3</td>
<td>Average no. fetus</td>
<td>12.6</td>
<td>13.6</td>
<td>12.3</td>
<td>12.5</td>
<td>0.75</td>
</tr>
<tr>
<td>Total born</td>
<td>1.71</td>
<td>2.23</td>
<td>1.07</td>
<td>0.65</td>
<td>2.19</td>
<td>1.76</td>
</tr>
<tr>
<td>Stillborn</td>
<td>11.7</td>
<td>10.9</td>
<td>11.1</td>
<td>11.7</td>
<td>10.1</td>
<td>11.9</td>
</tr>
<tr>
<td>No. suckling at birth</td>
<td>11.1</td>
<td>10.6</td>
<td>10.4</td>
<td>9.31</td>
<td>10.3</td>
<td>11.3</td>
</tr>
<tr>
<td>No. suckling at d 28</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10.3</td>
<td>10.0</td>
</tr>
</tbody>
</table>

1 Adapted from Lauridsen et al. (2010).
2 Hy-D (25-hydroxycholecalciferol; DSM Nutritional Products A/S, Basel, Switzerland).
3 Gilts (n = 20/treatment) were provided the dietary vitamin D treatment from mating until d 28 of gestation.
4 Sows (n = 20/treatment) were provided the dietary vitamin D treatment from mating until end of lactation (d 28 after farrowing). Significant effects were obtained on total born (interaction between parity, form, and dose, P = 0.009) and on stillborn (effect of dose, P < 0.05).

Figure 1. Relative mRNA abundance of vitamin D receptor (VDR) and homeobox protein A10 (HoxA10) in samples obtained from the uterine horn of gilts fed either vitamin D3 or 25-hydroxyvitamin D3 from mating until d 28 of gestation. Adapted according to Lauridsen and Theil (2011).
of feeding 25(OH)D₃ on maternal and fetal circulating 25(OH)D₃ concentration, 2 diets were formulated (Coffey et al., 2012): the control diet contained 2,500 IU vitamin D₃/kg diet, and the experimental diet was formulated to contain 500 IU vitamin D₃/kg diet + 50 μg 25(OH)D₃/kg diet (Hy·D). These rather high levels were used because commercial gestation diets typically contained at least 10 times the amount listed in the NRC (1998) requirement of 200 IU/kg of feed for gestating and lactating sows. The gilts fed the 25(OH)D₃ had greater concentration of 25(OH)D₃ in their plasma compared with gilts fed the control diet (Coffey et al., 2012), and the results were consistent with the results obtained by Lauridsen et al. (2010). On d 89 of gestation, gilts had 93 vs. 58 ng 25(OH)D₃/mL plasma when provided the 25(OH)D₃ or the control diet, respectively (Coffey et al., 2012). Circulating plasma 25(OH)D₃ in fetuses obtained at d 90 of gestation was also affected by the dietary treatment of the gilts (8.23 ng/mL [n = 14] fetuses vs. 6.46 ng/mL [n = 14] fetuses). The authors concluded that improving of maternal vitamin D status by feeding 25(OH)D₃ also resulted in an improvement of fetal vitamin D status (Coffey et al., 2012). A recent published study (Hines et al., 2013) based on the same initial study reported by (Coffey et al., 2012) evaluated the impact of the maternal vitamin D status on the development of fetal skeletal muscle and it was demonstrated that the increase in maternal and fetal vitamin D status by feeding gilts 25(OH)D₃ resulted in noteworthy changes in fetal skeletal muscle development characteristics and myoblast activity in fetuses obtained from the gilts at d 90 of gestation. In this context, it can be mentioned that samples of the uterine fluid at d 28 of gestation were collected in the dose–response trial reported by Lauridsen et al. (2010), but no detectable 25(OH)D₃ was found in the samples. Furthermore, the plasma concentration of 25(OH)D₃ in piglets was very low (<5 ng/mL of plasma) and was not detectable in most piglets. Unfortunately, it has not yet been possible to analyze the concentration of vitamin D in the sow milk from the study by Lauridsen et al. (2010), and presently there is generally a lack of scientific results concerning the content of vitamin D in sow milk in relation to dietary intake. Amongst the other fat-soluble vitamins (A, E, and K₃), the proportion of vitamin D₃ is much less (Csapo et al., 1996) and seems to be slightly greater in colostrum and transient milk of sows compared with mature milk. A study in humans demonstrated that because of poor penetration of vitamin D and 25(OH)D₃ into milk, breast-fed infants exclusively are at greater risk of vitamin D deficiency than are formula-fed infants (Kovacs, 2008). This may also be true for the sow and its progeny. In fact, the study by Witschi et al. (2011) showed that offspring of sows fed diets fortified with either 5 μg (i.e., low, representing the estimated requirement by NRC, 1998) or 50 μg of vitamin D₃ (i.e., normal, representing the level used in practice in Switzerland) or 50 μg of 25(OH)D₃ (Hy·D)/kg of diet had serum 25(OH)D₃ concentrations below 10 ng/mL while suckling the sow. Although the vitamin D status seemed to be greater in neonates of sows provided 25(OH)D₃ (Hy·D) rather than cholecalciferol in the recent studies (Lauridsen et al., 2010; Witschi et al., 2011; Coffey et al., 2012), the transfer of vitamin D and its metabolites across the placenta and into milk of the sows is low and does not affect the vitamin D status of the infant at birth to a level at which the risk of vitamin D deficiency is overcome assuming that a plasma concentration below 10 to 15 ng 25(OH)D₃/mL may be considered deficient for swine. Therefore, it is recommended that future research focus on other strategies in providing vitamin D to pigs while suckling. In this context, it can be mentioned that beginning in spring of 2011, investigations on farms across the United States led to the anecdotal observation that most weaned pigs tested were vitamin D deficient (Tousignant et al., 2013). Work began in practice on developing an oral supplement to be given to piglets early in life, and improvement in body weight at weaning and 1 wk after weaning and an increase in vitamin D status was observed by treatment with oral vitamin D₃ (Tousignant et al., 2013). A very recent paper (Flohr et al., 2013) also investigated the effects of varying concentrations of supplemental vitamin D₃ for nursing and weanling pigs. This study showed no benefit in any response criteria for supplementing vitamin D₃ of nursing or weanling pigs other than an increase in vitamin D status (Flohr et al., 2013). Administration of 1 of 3 oral vitamin D₃ dosages (none, 40,000, or 80,000 IU vitamin D₃) on d 1 or 2 of age increased circulating 25(OH)D₃ from 3.6 ng/mL (initial) to 14.7, 57.3, and 68.5 ng 25(OH)D₃/mL serum at d 10 for the 3 dosages, respectively, after which the response in vitamin D status according to the dietary provision was less pronounced. In that study, the authors furthermore observed that dietary concentrations of vitamin D₃ of 44,100 IU/kg feed may negatively affect feed preference of nursery pigs (Flohr et al., 2013).

**Impact of Vitamin D in Relation to Bone Health**

Vitamin D deficiency causes poor mineralization of the skeleton in most mammals (Holick, 2006). Prolonged deficiency of vitamin D can result in 2 diseases in pigs, namely rickets (in piglets and growing pigs) and osteomalacia (in sows and boars); however, these deficiency symptoms are very rare. Rickets is a disease of growing bone in which calcium and phosphorus deposition are disturbed and as a result, the bones are weak and easily broken and the legs may be bowed. Symptoms usually are enlarged joints, broken bones, stiffness of the joints, and occasionally paralysis. Osteomalacia is the reabsorption of calcium from bone that is already
Because the piglet is born with very low 25(OH)D₃, it is thus highly predisposed to vitamin D deficiency. Actually, it has been shown that kyphosis, which is an idiopathic disease characterized by abnormal, outward spinal curvature, is induced in young pigs if sows are fed reduced amount of vitamin D from breeding through lactation (Rortvedt and Crenshaw, 2012). In that study, decreased amount of calcium and phosphorus in the diet (i.e., 80% of requirement) resulted in reduction of whole body and femur bone mineral content in pigs compared with high levels of calcium and phosphorus (i.e., 120% of requirement), and pigs produced by low vitamin D sows were more severely affected (Rortvedt and Crenshaw, 2012). Active intestinal absorption of calcium in newborn piglets does not rely on vitamin D-dependent transport until the fourth week postpartum; however, alterations in concentration of vitamin D, calcium, and phosphorus in maternal and nursery diets influenced the incidence of kyphosis and bone health (Rortvedt and Crenshaw, 2012). Supplementing creep feed represents a potential approach to provide adequate amounts of vitamin D to piglets when their regulation of calcium absorption becomes vitamin D dependent. Provision of 200 IU vitamin D/kg feed for dams or through creep feed for the offspring reduced the bone-breaking strength and the cortical bone mineral content and density at the tibial midshaft of piglets when compared with provision of 50 μg/kg of feed of either cholecalciferol or 25(OH)D₃ to dams or through creep feed (Witschi et al., 2011). Overall, that study and the study of Lauridsen et al. (2010) showed that vitamin D may be a more important factor to consider rather than simply adjusting dietary calcium and phosphorus levels. In the study of Lauridsen et al. (2010), in which calcium and phosphorus was provided at levels recommended for sows during gestation and lactation, results with gilts showed that the ultimate strength of the bones and their content of ash were greater when vitamin D₃ was supplemented compared with the same amount of 25(OH)D₃ and results were maximized at 800 IU/kg feed (Table 3).

**Summary and Conclusion**

According to the NRC (1998), no studies of the vitamin D requirement of sows during gestation or lactation had been reported yet. Consequently, the vitamin D recommendation for sows during gestation and lactation of 200 IU/kg feed (NRC, 1998) was not based on scientific reports. However, research published since that time has contributed knowledge that has formed the basis of the recommendation for increased vitamin D for gestating and lactating sows to 800 IU/kg feed (NRC, 2012). In the present paper, this recent research was reviewed and put into context with previous studies. The dietary vitamin D treatments used in the recent reviewed literature was ranged from 200 to 2,500 IU/kg feed, and the studies addressed effects on vitamin D status, performance, reproduction, transfer of vitamin D to the neonate, and bone status markers of gilts and sows. In general, dietary treatments with ≥800 IU/kg feed have shown beneficial effects in terms of bone mineral content and ultimate strength, decreased number of still born piglets, and greater vitamin D status in comparison with dietary treatment of 200 IU/kg feed. It therefore seems to be a reasonable nutritional strategy to increase the vitamin D recommendation for sows and gilts to 800 IU/kg feed. However, the influence on the vitamin D status of the offspring is minor, and although the optimal plasma vitamin D level has not been assessed for swine, newborn and suckling pigs may still suffer from decreased plasma concentration of 25(OH)D₃ although gilts and sows are fed dietary levels around 800 IU/kg feed or even above as often observed in some commercial diets. Recent studies have demonstrated that it is possible to increase the vitamin D status of nursery and preweaning pigs by oral vitamin D₃ supplementation; however, it is recommended to further investigate the impact on

### Table 3. Bone status markers in gilts fed increasing doses and different sources of vitamin D¹,²

<table>
<thead>
<tr>
<th>Bone status marker</th>
<th>Vitamin D₃, IU/kg feed</th>
<th>Hy-D,³ μg/kg feed</th>
<th>SE²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>200</td>
<td>800</td>
<td>1,400</td>
</tr>
<tr>
<td>Ca, % in wet sample²</td>
<td>9.94</td>
<td>10.6</td>
<td>10.5</td>
</tr>
<tr>
<td>P, % in wet sample²</td>
<td>4.94</td>
<td>5.18</td>
<td>5.07</td>
</tr>
<tr>
<td>Ash, % in wet sample²</td>
<td>28.8</td>
<td>30.4</td>
<td>29.7</td>
</tr>
<tr>
<td>Breaking strength, kg⁶</td>
<td>1.279</td>
<td>1.533</td>
<td>1.500</td>
</tr>
<tr>
<td>Breaking point, mm</td>
<td>11.56</td>
<td>11.67</td>
<td>11.51</td>
</tr>
</tbody>
</table>

¹Adapted from Lauridsen et al. 2(010).
²Gilts (n = 20/treatment) were provided the dietary vitamin D treatment from mating until d 28 of gestation.
³Hy-D (25-hydroxycholecalciferol; DSM Nutritional Products A/S, Basel, Switzerland).
⁴Interaction between form and dose (P = 0.02).
⁵Effect of form of vitamin D (P = 0.02).
⁶Effect of form of vitamin D (P = 0.01).
biological responses other than concentration of 25(OH)D3 in serum to optimize the strategies for supplementing young piglets with vitamin D.

**LITERATURE CITED**


