The effect of maternal vitamin D source on broiler hatching egg quality, hatchability, and progeny bone mineral density and performance

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SUMMARY

Vitamin D is involved in calcium metabolism as well as bone and shell quality, and is therefore important to broiler breeders. In this research we investigated the effects of maternal dietary 25-OH vitamin D₃ on broiler breeder egg quality and hatchability, as well as on progeny bone mineral density and performance. In a field study, all hens were fed 3,000 IU of vitamin D₃ (D) per kilogram of complete feed; in addition half of the hens also received 34.5 µg of 25-OH vitamin D₃ per liter in the drinking water (25OHD). Eggs from each treatment group were incubated and hatched; chicks were fed a common diet and grown to 41 d of age. Eggs from hens in the 25OHD treatment had a nearly 30% reduction in early embryo mortality. However, a larger egg size resulted in greater chick BW for the D chicks, although this did not affect broiler production performance. Broilers from the maternal 25OHD treatment had a lower FCR during the grower phase. Unexpectedly, chick plasma 25-OH vitamin D₃ was only greater for the maternal 25OHD treatment at 4 d of age, but not at hatch, 2, 6, 8, 10, 12, or 14 d of age. Maternal vitamin D₃ source did not affect progeny 41-d bone mineral density. Maternal 25-OH vitamin D₃ had a protective effect on the growing embryo, reducing early embryonic mortality, with minimal effects on progeny performance and bone mineral density to processing at 41 d of age. The previously reported effects of 25-OH vitamin D₃ on increasing broiler performance and breast yield seem to be dependent on supplementation of the broiler diet; a carry-over effect of maternal supplementation is insufficient to achieve these effects.

Key words: broiler breeder, vitamin D₃, 25-hydroxyvitamin D₃, egg quality, embryonic mortality, broiler

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DESCRIPTION OF PROBLEM

Twenty-five hydroxycholecalciferol (25-OH vitamin D₃) is a metabolite of vitamin D₃ that is formed in the liver from vitamin D [1]. This metabolite is then hydroxylated to the active form, 1,25(OH)₂D₃ in the kidney by 25-hydroxy-D₃-1α-hydroxylase [2]. The 1,25(OH)₂D₃ is involved in intestinal Ca absorption, the deposition of skeletal Ca, as well as Ca resorption from bone tissues when plasma Ca levels are low [3]. Its role in skeletal metabolism makes vitamin D₃
and its metabolites a natural target for studies involving bone quality in poultry.

The egg yolk is the main source of nutrition for the developing embryo and newly hatched chick [4]. The vitamin D₃ level in the hens diet is positively correlated with vitamin D₃ and 25-OH vitamin D₃ contents within the egg yolk [5]. The enzyme 25-hydroxyvitamin D-1α-hydroxylase, which is responsible for the hydroxylation of 25-OH vitamin D₃ to 1,25(OH)₂D₃, is present as early as 12 d of incubation and increases in specific activity during further embryonic development [6]. The early development of vitamin D₃ metabolism signifies the importance of this nutrient to the developing embryo. However, even with diets sufficient in vitamin D activity (2,200 IU), the addition of 1,100 IU of 25-OH vitamin D₃ to the diet of turkey breeders increased egg hatchability as compared with dietary vitamin D₃ alone [7]. This may be related to the fact that 25-OH vitamin D₃ is more efficiently absorbed by the bird than vitamin D₃ [8], which may also be the case for the chick embryo.

Providing the broiler breeder hen with dietary 25-OH vitamin D₃, may increase the absorption efficiency of Ca from the gut, leading to increased eggshell quality and less reliance on bone Ca as the bird ages. In addition, maternal supplementation of 25-OH vitamin D₃, increases deposition of 25-OH vitamin D₃ into the egg [9] and may result in increased egg hatchability and chick quality, which could in turn result in greater growth and efficiency of the broiler chick. Therefore the objectives of the current research were to investigate the effects of supplementation of a liquid 25-OH vitamin D₃ water supplement to broiler breeders on egg quality and hatchability as well as progeny bone mineral density and performance. We hypothesized that maternal dietary 25-OH vitamin D₃ would increase egg hatchability, chick quality, and overall broiler performance to slaughter at 41 d of age.

MATERIALS AND METHODS

Experimental Design and Conditions

The experimental protocols were conducted according to the Canadian Council on Animal Care guidelines [10]. Cobb 500 [11] broiler hatching eggs (n = 3,530) were obtained from 2 commercial breeder flocks on the same production complex at 29 wk of age. Approximately half of the eggs (n = 1,510) came from a flock that received 3,000 IU/kg of dietary vitamin D₃ (D) during the breeder phase. The remainder of the eggs (n = 1,620) came from breeder hens that were fed the same diet as the control birds, as well as being supplemented with 34.5 µg of 25-OH vitamin D₃ (25OHD) per liter of drinking water starting 3 wk before fertile egg collection. Eggs were incubated [12] for 21.5 d at 37.5°C and 85% RH. Eggs were transferred to a hatcher at 18 d of age and placed in hatch baskets that held 18 eggs per basket (n = 84 and 88 hatch baskets for the D and 25OHD treatments, respectively). At hatch, stage of development at embryonic mortality [13], hatchability, and chick BW were assessed for each maternal dietary treatment group. Early (0 to 7 d) and late (8 to 18 d) embryonic mortality (mid embryonic mortality was combined with late embryonic mortality due to low mortality in both phases), late hatch (chicks requiring longer than 21.5 d to hatch), percent internal pip live and dead (IPL and IPD; those chicks that pipped through the shell membrane only before expiration), percent external pip live and dead (EPL and EPD; those chicks that pipped through the shell before expiration) were determined as a percentage of fertile eggs.

Egg Quality

In addition to the eggs placed in the incubator, eggs (n = 200 per treatment) were assessed for egg quality traits. Egg specific gravity was measured by the floatation method [14]. Individual eggs were weighed and the weights of yolk, albumen, and shell were recorded. Eggshell weight (with membranes attached) was measured after the eggshells were washed and air-dried overnight. Eggshell thickness was determined from the middle of the egg using a micrometer. Eggshell conductance was determined using the method described by O’Dea et al. [15] and calculations given by Ar et al. [16]. Briefly, the rate of egg weight loss (presumed to be moisture loss) was determined daily on eggs (n = 15 per treatment) that were placed in desiccators and covered in desiccant for a 9-d period. Room temperature was recorded daily for the determination of the saturation vapor pressure.
SAUNDERS-BLADIES AND KORVER: BROILER VITAMIN D SOURCE

Broiler Production, Plasma 25-OH Vitamin D₃, and Bone Quality

Chicks from each maternal treatment group were randomly allocated to 20 floor pens (n = 60 birds per pen at 17.44 birds/m²) according to the maternal dietary treatment groups (n = 1,200 per maternal dietary treatment). Due to overcrowding, 10 birds per pen were removed after weighing on d 14. The new stocking density was 14.09 birds/m². The birds were fed a nutritionally complete crumbled broiler starter ration (23% CP, 3,067 kcal/kg of ME, 1.1% Ca, 0.50% available P, and 2,500 IU of supplemental vitamin D per kilogram of diet) from 0 to 14 d of age. The grower (20% CP, 3,152 kcal/kg of ME, 0.90% Ca, 0.45% available P, and 2,500 IU of supplemental vitamin D per kilogram of diet; fed from 15 to 27 d of age) and finisher (19% CP, 3,196 kcal/kg of ME, 0.85% Ca, 0.42% available P, and 2,500 IU of supplemental vitamin D per kilogram of diet; fed from 28 to 41 d of age) rations were pelleted. All diets were wheat-based, supplemented with a commercial arabinobanolanase enzyme [17], and formulated to meet or exceed NRC [18] and primary breeder [19] nutrient recommendations. Broiler pen BW was measured at 0, 7, 14, 27, and 42 d. Feed consumption and FCR were calculated on a pen basis at 7, 14, 27, and 42 d of age.

During the first 2 wk posthatch, 10 chicks per maternal treatment were assessed for plasma 25-OH vitamin D₃ levels every 2 d. Blood samples were collected and centrifuged at 4,000 × g for 15 min at 4°C. The plasma was removed and stored at −20°C until further analysis. The 25-OH vitamin D₃ was extracted from plasma as described by Aksnes [20]. A standard curve was obtained using dilutions of a 25-OH vitamin D₃ standard [21]. Femur bone mineral density (BMD), cross-sectional area, and bone mineral content (BMC) at 41 d was measured on the right femur of male birds (n = 25 per treatment) selected at random from each maternal treatment group using quantitative computed tomography [22] using procedures and calculations outlined in Saunders-Blades et al. [23]. In addition, femur breaking strength was measured using an Instron Material Tester [24] as described by Saunders-Blades et al. [23].

Statistical Analysis

The egg was the experimental unit for the egg trait data. Each hatch basket of 18 eggs was the experimental unit for the hatch data. The pen was the experimental unit for the broiler growth data. One pen from the maternal 25OHD treatment was removed from the data set due to flooding within the pen, which affected broiler production traits. Most data, except for the percentage data [percent fertility, hatch (total eggs), hatch (fertile eggs), early and late mortality, late hatch, all the internal and external pips (live and dead) as well as the dead and culls at hatch] were analyzed as a one-way ANOVA with maternal dietary treatment as the main effect using the Mixed procedure in SAS [25]. The percentage data listed were analyzed as a Chi-squared analysis using SAS [25]. Plasma 25-OH vitamin D₃ data were analyzed using the repeated measures procedure in Proc Mixed of SAS [25]. Means were compared using LSmeans comparisons of SAS [25]. Significance was assessed at a P ≤ 0.05.

RESULTS AND DISCUSSION

Fertility, Hatchability, and Chick Quality

The increase in hatchability of total and of fertile eggs approached significance (P = 0.072 and 0.071; respectively; Table 1) as a result of a nearly 30% reduction in early embryonic mortality when broiler breeders were supplemented with 25-OH vitamin D₃ in the drinking water (P < 0.03; Table 1). In the current study, no differences were observed in late embryonic mortality, IPL, IPD, EPL, EPD, and percent dead and culls at hatch (P > 0.05; Table 1). At low levels of vitamin D₃ relative to typical commercial supplementation, the addition of 1,100 IU of 25-OH vitamin D₃ to a diet that already contained 2,200 IU of vitamin D₃ increased hatchability of fertile turkey eggs from 48-wk-old hens by 2- to 3-fold [7]. Supplementation of breeder hen diets with 3,125 ng of 25-OH vitamin D₃ per kilogram of feed reduced late, but not early, embryonic mortality as compared with 3,125 ng of vitamin D₃ per kilogram of feed [26]. According to some sources, 3,125 ng/
kg of 25-OH vitamin D₃ would provide a greater amount of vitamin D₃ bioactivity than 3,125 ng/kg of vitamin D₃ [27], which could be the cause for the different results than the current study. However, when the breeder hen dietary levels of 25-OH vitamin D₃ and vitamin D₃ were increased to 12,500 ng/kg (500 IU/kg), no difference in embryonic mortality was noted between treatments [26]. Other studies have suggested that the bioefficacy of 25-OH vitamin D₃ relative to vitamin D₃ to increase BW and reduce tibial dischondroplasia in broiler chickens is greater per unit of supplementation at levels below 1,000 IU/kg, but above this level the difference is minimal [28]. In the current study, all of the hens would have received sufficient levels of vitamin D activity, regardless of the source. Therefore, the protective effect of 25-OH vitamin D₃ observed on early embryonic mortality in the current study are not likely due simply to the level of vitamin D activity supplemented, but rather to the form of vitamin D₃ added. In broiler chicks, 25-OH vitamin D₃ is absorbed more efficiently from the gut than vitamin D₃ [8], and young chicks may have lower ability to convert vitamin D₃ to 25-OH vitamin D₃ [29]. Therefore, chicken embryos may be metabolically limited in the ability to use vitamin D₃, and provision of preformed 25-OH vitamin D₃ via maternal supplementation may provide a survival advantage to the embryo relative to vitamin D₃. In contrast, Torres et al. [30] reported no difference in hatchability in 54- and 64-wk-old Cobb 500 broiler breeder hens supplemented with up to 69 µg of 25-OH vitamin D₃ per kilogram of feed (equivalent to 2,670 IU of vitamin D₃) in addition to 2,000 or 3,400 IU of vitamin D₃ per kilogram of feed as compared with the same levels of vitamin D₃ alone.

The hatch weight of chicks from the broiler breeders on the D treatment was greater than those from the 25OHD eggs (P < 0.001; Table 2). This was a result of the greater set and transfer weight of the eggs from the D hens (P < 0.0001 and 0.0059; respectively; Table 2). Egg size is one of the main factors affecting chick weight at hatch [31]. A smaller egg mass from birds supplemented with 25-OH vitamin D₃ as compared with vitamin D₃ has also been reported in laying hens [32], although no significant difference in egg production was reported.
Egg Traits

The eggs from the D treatment lost a greater proportion of weight throughout the 21.5 d of incubation \( (P < 0.05; \text{Table 2}) \), which may be explained by the greater percentage of eggshell of the eggs from the breeders on the 25OHD treatment (Table 3). Eggshell water vapor conductance increases with egg weight [33]. In the current study, although no treatment effect on eggshell conductance was observed, any potential effect on egg size was likely eliminated because eggs used for conductance measurement were selected to be within 56.3 ± 0.5 g of egg weight (Table 2). Eggs from the breeders on the 25OHD treatment had approximately 1.7% greater eggshell as a proportion of egg weight relative to eggs from breeders on the D treatment (Table 3). No differences in egg specific gravity, shell weight, or thickness were seen between the 2 treatment groups (Table 3). This is contrary to a previous study where a strong positive correlation was found between specific gravity and percent eggshell in eggs from laying hens [34]. In the current study, although the percent shell was different between the treatments, the difference may have been small enough that no measurable effect on specific gravity could be detected. However, similar to the results of the current study, supplementation of laying hens diets with 25-OH vitamin D3 in place of vitamin D3 did not increase egg specific gravity [9, 32], nor were albumen weight and shell breaking strength affected [9]. Dietary supplementation of 1,100 IU of 25-OH vitamin D3 per kilogram of feed in addition to 2,200 IU of vitamin D3 per kilogram of feed did not affect turkey egg specific gravity in comparison with dietary vitamin D3 alone [7]. On the contrary, in older broiler breeders, egg-specific gravity was greater in eggs from hens fed 25-OH vitamin D3 than in eggs from hens fed vitamin D3 at equivalent levels [30].

Eggs from the maternal 25OHD treatment had increased yolk weight as a proportion of egg weight relative to the D treatment \( (P < 0.05; \text{Table 3}) \). An increase in the proportion of egg yolk caused by dietary 25-OH vitamin D3 has not previously been reported in the literature. In spite of the difference in yolk size, the eggs from the 25OHD hens were smaller than eggs from the D hens. No significant difference was observed between dietary treatments in percent albumen (Table 3).

Broiler Growth and FE

The greater chick BW at hatch of the D group (Table 2) did not lead to differences in chick BW, gain, feed consumption, and FCR during the starter and finisher periods (Table 4). The only maternal treatment effect on broiler pro-

<table>
<thead>
<tr>
<th>Item</th>
<th>Set egg weight</th>
<th>Transfer egg weight</th>
<th>Weight loss</th>
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<tr>
<td>D(^a)</td>
<td>56.0(^b) (1,510)</td>
<td>49.9(^a) (1,399)</td>
<td>10.98(^a) (1,399)</td>
<td>11.32 (15)</td>
<td>38.2(^b) (84)</td>
</tr>
<tr>
<td>25OHD(^b)</td>
<td>55.5(^a) (1,619)</td>
<td>49.6(^b) (1,523)</td>
<td>10.82(^b) (1,523)</td>
<td>11.76 (15)</td>
<td>37.7(^a) (88)</td>
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</table>

\(^{a,b}\) Treatment means within same column with different superscripts are significantly different \( (P < 0.05) \).

\(^1\) Means are followed by n in parentheses; n for egg data = number of eggs for each treatment, n for BW = number of hatch basket sections (18 eggs/basket).

\(^2\) Set egg weight = weight of egg when first put in incubator; transfer egg weight = weight of egg after 18 d of incubation; weight loss = percent of weight loss of the egg from 0 to 18 d of incubation; eggshell conductance = rate of water loss from egg when stored for 7 d covered with desiccant.

\(^3\) Broiler breeders fed a diet containing 3,000 IU of vitamin D\(_3\) as the sole supplemental source of vitamin D activity.

\(^4\) Broiler breeders supplemented with 34.5 µg of 25-OH vitamin D\(_3\) per liter of water starting at 26 wk of age in addition to 3,000 IU/kg of dietary vitamin D\(_3\).
production was found during the grower phase (15–27 d), when chicks from the 25OHD maternal treatment had a lower FCR than those from the D hens (Table 4). Body weight and gain during this phase were nearly significantly greater for the broilers from the maternal 25OHD treatment (P = 0.0592 and 0.0762; respectively; Table 4), with no difference in feed consumption. Maternal 25-OH vitamin D₃ supplementation did not have lasting effects on broiler performance, as no differences in final BW and overall FCR were noted. Atencio et al. [35] reported a greater BW gain of progeny from hatch to 16 d of age with maternal dietary vitamin D₃ increasing from a deficient (125 IU/kg) to a sufficient (2,000 IU/kg) level. Broiler performance past 16 d was not investigated in that study. In the current study, vitamin D₃ supplementation was well in excess of 2,000 IU/kg. When 25-OH vitamin D₃ was fed directly to broilers in previous studies, increases in BW, BW gain, and breast muscle yield were noted [28, 36, 37]. The minimal and transient effect of maternal vitamin D₃ source on broiler chick performance indicates that the effects of 25-OH vitamin D₃ on broiler performance are therefore dependent upon dietary supplementation of vitamin D₃ to diets sufficient in vitamin D₃. The broiler breeder supplementation of 25-OH vitamin D₃ to diets did not appear to influence broiler performance to 41 d of age.

Broiler Chick Plasma 25-OH Vitamin D₃ from Hatch to 14 d

Chick plasma 25-OH vitamin D₃ at hatch was greater for the maternal D₃ treatment than the D₃ treatment at 4 d of age (Figure 1). Increasing dietary vitamin D₃ in laying hen diets from 1,064 to 8,640 IU/kg and 25-OH vitamin D₃ from 30 to 122 μg/kg (equivalent to 1,200 to 4,880 IU/kg) increased both egg yolk vitamin D₃ and 25-OH vitamin D₃ [5, 9]; however, the effect of egg vitamin D₃ and its metabolites on the plasma levels in the offspring has not been previously reported. The cause of the delay in the appearance of treatment differences in plasma 25-OH vitamin D₃ is unknown. The yolk sac is intensively absorbed during the first 5 d posthatch [38], suggesting that stored 25-OH vitamin D₃ (either tissue or yolk sac) may be readily available to the fast-growing chick. It is

<table>
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<tr>
<th>Item</th>
<th>df</th>
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<th>Egg weight (g)</th>
<th>Specific gravity¹</th>
<th>Shell weight¹ (g)</th>
<th>Shell thickness¹ (mm)</th>
<th>Shell¹ (%)</th>
<th>Yolk¹ (%)</th>
<th>Albumen¹ (%)</th>
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<td>1.079</td>
<td>5.43</td>
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<td>1.080</td>
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¹Specific gravity was measured by the floatation method with a series of saline solutions of increasing specific gravity ranging from 1.060 to 1.010 in increments of 0.002. Shell weight is the weight of the washed and air-dried egg shell (with membrane). Shell thickness was determined on the egg shell from the middle of the egg using a micrometer. Percent shell, yolk, and albumen were determined as a percentage of the total egg weight.

²Broiler breeders fed a diet containing 3,000 IU/kg of vitamin D₃ as the sole supplemental source of vitamin D activity.

Table 3. Effect of maternal vitamin D source on egg quality from 29-wk-old broiler breeders

³Broiler breeders supplemented with 34.5 μg of 25-OH vitamin D₃ per liter of water starting at 26 wk of age in addition to 3,000 IU/kg of dietary vitamin D₃.

D₃ treatment had a lower FCR than those from the 25-OHD maternal treatment (Table 4). Body weight and gain from the grower phase (15–27 d) were nearly significantly greater for the offspring of the maternal 25OHD treatment than the D treatment (Table 4). Body weight and gain during this phase were nearly significantly greater for the broilers from the 25OHD maternal treatment (P = 0.0592 and 0.0762; respectively; Table 4), with no difference in feed consumption. Maternal 25-OH vitamin D₃ supplementation did not have lasting effects on broiler performance, as no differences in final BW and overall FCR were noted. Atencio et al. [35] reported a greater BW gain of progeny from hatch to 16 d of age with maternal dietary vitamin D₃ increasing from a deficient (125 IU/kg) to a sufficient (2,000 IU/kg) level. Broiler performance past 16 d was not investigated in that study. In the current study, vitamin D₃ supplementation was well in excess of 2,000 IU/kg. When 25-OH vitamin D₃ was fed directly to broilers in previous studies, increases in BW, BW gain, and breast muscle yield were noted [28, 36, 37]. The minimal and transient effect of maternal vitamin D₃ source on broiler chick performance indicates that the effects of 25-OH vitamin D₃ on broiler performance are therefore dependent upon dietary supplementation of vitamin D₃ to diets sufficient in vitamin D₃. The broiler breeder supplementation of 25-OH vitamin D₃ to diets did not appear to influence broiler performance to 41 d of age.
Table 4. Effect of maternal vitamin D source on broiler BW, gain, feed consumption, and FCR from 0 to 41 d

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<th>d 14</th>
<th>d 27</th>
<th>d 41</th>
<th>d 0–7</th>
<th>d 8–14</th>
<th>d 15–27</th>
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* Treatment means within same column with different superscripts are significantly different ($P < 0.05$).

1 Broilers from broiler breeders fed a diet containing 3,000 IU of vitamin D₃ as the sole supplemental source of vitamin D activity.

2 Broilers from broiler breeders supplemented with 34.5 µg of 25-OH vitamin D₃ per liter of water starting at 26 wk of age in addition to 3,000 IU/kg of dietary vitamin D₃.
possible the yolk sac of chicks from the 25OHD treatment hens had a greater store and, therefore, when the plasma levels of 25-OH vitamin D$_3$ start to decrease for the D chicks, sufficient yolk 25-OH vitamin D$_3$ was present to delay this decrease in the 25OHD maternal treatment.

In both treatment groups, plasma 25-OH vitamin D$_3$ decreased from hatch to 6 d of age, at which point it remained low to 10 d of age, after which chicks from both maternal treatments had increased plasma 25-OH vitamin D$_3$ levels. Chicks at hatch had similar levels of plasma 25-OH vitamin D$_3$ regardless of maternal supplementation when supplemented with adequate levels of vitamin D$_3$ activity. Although providing maternal 25-OH vitamin D$_3$ minimized the decrease in plasma 25-OH vitamin D$_3$ at 4 d of age, no further differences beyond that age were noted. This is likely related to the chick using up the yolk sac storage of the metabolite. The decrease in 25-OH vitamin D$_3$ from 2 to 6 d of age may reflect a limited ability of the chick to convert dietary vitamin D$_3$ to 25-OH vitamin D$_3$ in the liver. Our laboratory has shown that plasma 25-OH vitamin D$_3$ levels decrease after hatch when chicks are fed only vitamin D$_3$ [29]. To our knowledge, no previous reports exist on the effect of maternal dietary 25-OH vitamin D$_3$ on the chick plasma 25-OH vitamin D$_3$.

**Broiler Bone Mineral Density**

Femur total, cortical, and trabecular BMD, cross-sectional areas, BMC, and bone breaking strength of broilers at 41 d of age were not different between the 2 maternal dietary treatments ($P>0.05$; Table 5). Although broiler bone ash was not measured in the current study, BMD and bone ash are positively correlated in many species [39, 40]. Early in life, chicken bones are not well mineralized; maximum BMD is not reached until 35 wk posthatch [41]. In the

**Figure 1.** The effect of maternal vitamin D source on broiler chick plasma 25-OH vitamin D$_3$ from hatch to 14 d. The D treatment was chicks from broiler breeders fed a diet containing 3,000 IU/kg of vitamin D$_3$ as the sole supplemental source of vitamin D activity. The 25OHD treatment was chicks from broiler breeders supplemented with 34.5 µg of 25-OH vitamin D$_3$ per liter of water in addition to 3,000 IU/kg of dietary vitamin D$_3$. n = 12 birds per treatment at each sample day. Means with differing lowercase letters (a,b) are significantly different within each age ($P<0.05$). Means over time across treatment with differing uppercase letters (A–C) are significantly different over time ($P<0.05$).
current study, plasma 25-OH vitamin D₃ levels between the D and 25OHD maternal treatment groups were equivalent from 0 to 2 and from 6 to 14 d posthatch (Figure 1). Rapid bone growth and formation occurs up to 28 d of age in broilers [42], with BMD, BMC, and cross-sectional bone areas in chicks increasing rapidly from 2 to 3 wk of age [43]. Therefore, it is likely that maternal dietary supplementation of 25-OH vitamin D₃ would not have long-term effects on broiler bone formation. Increasing maternal dietary vitamin D₃ from 0 to 4,000 IU/kg of feed increased 16-d progeny broiler bone ash and reduced tibial dyschondroplasia scores [35], whereas it increased bone ash from turkey poults up to 12 d when increased in the hen diet from 300 to 2,700 IU/kg of feed [44]. In the current study, all hens were provided with vitamin D₃ greatly in excess of the NRC requirements [18], and the 25OHD hens received further vitamin D₃ activity in the form of 25-OH vitamin D₃. As intake of vitamin D₃ activity was not likely limiting to the hens’ ability to deposit sufficient vitamin D₃ and 25-OH vitamin D₃, the lack of effects on many of the bone traits measured is not surprising.

CONCLUSIONS AND APPLICATIONS

1. Supplementing breeder hens with 25-OH vitamin D₃ in addition to sufficient levels of dietary vitamin D₃ had a protective effect on the developing embryo from 0 to 7 d of incubation. This reduction in early embryonic mortality could increase overall productivity of broiler breeder flocks.

2. Based on the increase in hatchability (which approached significance), the increase in shell weight, and the protective effect against early embryonic mortality, either 25-OH vitamin D₃ has effects beyond that of a sufficient dietary vitamin D₃ level, the requirement of the hen to maximize these functions is higher than 3,000 IU/kg of feed, or both.

3. Although maternal 25-OH vitamin D₃ supplementation had a positive influence on some early chick characteristics, no consistent long-lasting effects on broiler performance were observed.
4. To achieve the previously reported effects of 25-OH vitamin D3 on broiler performance, supplementation of the broiler diets directly may be necessary.

REFERENCES AND NOTES


Acknowledgments

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