METABOLISM AND NUTRITION

The Effect of Carrier for Vitamin E on Liver Concentrations of Vitamin E and Vitamin E Excretion in Broilers\textsuperscript{1,2}

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ABSTRACT Two experiments were conducted to determine the effect of 2 carrier sources of vitamin E on growth performance and excreta and liver vitamin E concentrations of broilers. Chicks were pretested from d 0 to 7 posthatching on a corn-soybean meal diet without vitamin E supplementation, and the experiments lasted from d 7 to 19 posthatching. Each treatment in both experiments was replicated with 6 pens with 5 chicks each. Initial and final BW were 155 and 684 g in experiment 1 and 155 and 691 g in experiment 2. In experiment 1, the dietary treatments were the corn-soybean meal diet with varying concentrations (0, 30, 100, or 300 IU/kg) of supplemental vitamin E either absorbed to verxite (VE) or adsorbed to silica (SE). In experiment 2, the dietary treatments were the 0 and 30 IU/kg of vitamin E as VE or SE. In experiment 1, feed intake decreased and gain:feed increased as concentration of VE increased, but feed intake increased and gain:feed decreased as concentration of SE increased (source $\times$ concentration, $P < 0.03$). Gain, feed intake, and gain:feed were not affected ($P > 0.10$) by the vitamin E carrier source in experiment 2. The mean excreta vitamin E concentration at d 7 posthatching was 17.2 IU/kg (DM basis). The percentages of vitamin E excreted were based on analyzed vitamin E concentrations in the diet. At 100 and 300 IU/kg of supplemental vitamin E, an average of 94 and 44% of vitamin E intake from broilers fed vitamin E from VE and SE, respectively, was excreted (vitamin E source, $P < 0.01$; source $\times$ concentration, $P < 0.08$), but at 30 IU/kg of vitamin E, 49 and 45% of vitamin E intake from broilers fed vitamin E from VE and SE was excreted. In experiment 2, 52 and 43% of vitamin E intake from VE and SE was excreted (source, $P < 0.02$). Liver $\alpha$-tocopherol concentration was not different ($P > 0.10$) between the sources of vitamin E in either experiment. Increased concentrations of vitamin E increased liver $\alpha$-tocopherol concentrations ($P < 0.01$). On the basis of the results of excreted vitamin E, vitamin E adsorbed to SE was more available than vitamin E absorbed to VE, but on the basis of liver vitamin E concentration, their availabilities were similar.

Key words: vitamin E, broiler, verxite, silica, excreta

INTRODUCTION

Very little research has been conducted on the effect of carrier on the availability of vitamin E for broilers (NRC, 1994). The main function of vitamin E is to work as a biological antioxidant, but it may also function in membrane structure, prevention of heavy metal toxicity, blood clotting, and biological oxidation-reduction reactions (McDowell, 1989). Oftentimes, excess vitamin E is provided in feeds to prevent oxidation and rancidity of added fat. In addition, inclusion of higher levels of E than the requirement has been reported to increase consumer perception of poultry meat quality when stored at 4°C for 4 d (Kennedy et al., 2005).

Verxite (VE), a highly purified form of vermiculite, is approved by the Food and Drug Administration (1976) for feeds as a nonnutritive carrier or to provide bulk density, as long as it does not exceed 5% of the total weight of the finished diet. Verxite, being highly absorbent, inorganic, and nonnutritive, has physical properties that allow it to be a carrier for liquids (Grace Specialty Vermiculite, 1999).

Verxite has been shown to be a suitable carrier for tallow in dairy feeds (Jenkins and Palmquist, 1984). Feeding tallow as fatty acids alone was shown to decrease the digestibility of fiber in the rumen. When tallow was fed as either calcium soap or attached to VE as a nonnutritive carrier to replace corn, the digestion of
fiber was the same as that of control animals with no added tallow. Jenkins and Palmquist (1984), however, reported that the fatty acids from tallow carried with VE were not as digestible as fatty acids from other carriers. They concluded that fatty acids were not completely removed from VE, which caused this effect. Hurley et al. (1990) determined that Mg attached to vermiculite was as available to lambs as Mg from MgOH or MgO. They reported that the absorption site for Mg changed from the abomasum to postabomasally when attached to VE, which caused this effect. Hurley et al. (2006) fed 10, 20, and 40 IU/kg of vitamin E absorbed to VE or vitamin E adsorbed to silica (SE) and reported that broilers fed VE had lower breast muscle, liver, and plasma concentrations of vitamin E than those fed SE. They concluded that VE bound vitamin E too tightly and made it less available to the chick. The purpose of this study was to determine whether VE was a suitable carrier of vitamin E compared with SE.

**MATERIALS AND METHODS**

**General**

All methods used in these experiments were approved by the Louisiana State University Agricultural Center Animal Care and Use Committee.

<table>
<thead>
<tr>
<th>Item</th>
<th>Diet amount (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ingredient</td>
<td></td>
</tr>
<tr>
<td>Corn</td>
<td>53.825</td>
</tr>
<tr>
<td>Soybean meal, 47.5% CP</td>
<td>37.086</td>
</tr>
<tr>
<td>Corn oil</td>
<td>4.980</td>
</tr>
<tr>
<td>Monocalcium phosphate</td>
<td>1.514</td>
</tr>
<tr>
<td>Limestone</td>
<td>1.484</td>
</tr>
<tr>
<td>Salt</td>
<td>0.500</td>
</tr>
<tr>
<td>Vitamin premix (^1)</td>
<td>0.025</td>
</tr>
<tr>
<td>Mineral premix (^2)</td>
<td>0.250</td>
</tr>
<tr>
<td>ni-Met</td>
<td>0.192</td>
</tr>
<tr>
<td>t-Thr</td>
<td>0.075</td>
</tr>
<tr>
<td>Choline chloride</td>
<td>0.070</td>
</tr>
<tr>
<td>ME (kcal/kg)</td>
<td>3,200</td>
</tr>
<tr>
<td>CP (%)</td>
<td>22.36</td>
</tr>
<tr>
<td>Calcium (%)</td>
<td>1.00</td>
</tr>
<tr>
<td>P (%)</td>
<td>0.725</td>
</tr>
<tr>
<td>Nonphytate P (%)</td>
<td>0.450</td>
</tr>
<tr>
<td>Choline (mg/kg)</td>
<td>1,869</td>
</tr>
<tr>
<td>True digestible Lys (%)</td>
<td>1.133</td>
</tr>
<tr>
<td>True digestible TSAA (%)</td>
<td>0.814</td>
</tr>
<tr>
<td>True digestible Thr (%)</td>
<td>0.808</td>
</tr>
<tr>
<td>True digestible Trp (%)</td>
<td>0.237</td>
</tr>
</tbody>
</table>

\(^1\)Provided the following, per kilogram of diet: vitamin A, 15,435 IU; vitamin D<sub>3</sub>, 6,615 IU; vitamin E, 0 IU; menadione 2.68 mg; vitamin B<sub>12</sub>, 0.0265 mg; biotin, 0.1323 mg; folic acid, 1,764 mg; niacin, 77.18 mg; pantothenic acid, 19.85 mg; pyrolase, 5.513 mg; riboflavin, 13.23 mg; and thiamin, 0.0265 mg.

\(^2\)Provided the following, per kilogram of diet: copper (cupric sulfate pentahydrate), 7.00 mg; iodine (calcium iodate), 1.00 mg; iron (ferrous sulfate monohydrate), 50.00 mg; manganese (manganese sulfate monohydrate), 100.00 mg; selenium (sodium selenite), 0.15 mg; and zinc (zinc sulfate monohydrate), 75 mg.

Two experiments were conducted with male broilers (Ross × Ross 508) obtained from House of Raeford in Gibsland, Louisiana. They were housed in stainless steel Petersime starter batteries for the duration of the projects and were allowed ad libitum access to feed in mash form and water. Each treatment in both experiments had 6 replications, with 5 chicks per pen. The chicks were pre-tested from d 0 to 7 posthatching on a corn-soybean meal diet containing no supplemental vitamin E, the negative control (NC) diet. Treatments were applied on d 7, and chicks were fed until d 19 posthatching. Chicks and feathers were weighed on d 7 and 19 for determination of average daily gain (ADG), average daily feed intake (ADFI), and gain:feed (G:F). Excreta samples were collected on d 7 after being fed the diet with no vitamin E supplementation. Excreta samples also were collected and feeders were weighed on d 18 and 19. Chicks were killed via CO<sub>2</sub> asphyxiation, and liver samples were collected and frozen for later analysis.

**Dietary Treatments**

In experiment 1, dietary treatments were as follows: 1) NC; 2 and 3) NC + 100 or 300 IU/kg of VE; 4 and 5) NC + 100 or 300 IU/kg of SE; 6) NC + 30 IU/kg of VE mixed in a commercial vitamin premix; and 7) NC + 30 IU/kg of SE mixed in a commercial vitamin premix. In experiment 2, dietary treatments included 1) NC; 2) NC with 30 IU/kg of VE mixed in a commercial vitamin premix; and 3) NC with 30 IU/kg of SE mixed in a commercial vitamin premix. The commercial premix used in experiment 1 in diets 6 and 7, and in experiment 2 in diets 2 and 3 was identical to the premix described in Table 1, with the exception that it had vitamin E added at 30 IU/kg. Nutrients in all diets met or exceeded NRC (1994) recommendations except for vitamin E (Table 1). The diets were analyzed for vitamin E concentration (Table 2).

**Excreta and Liver Analysis**

Excreta were collected from d 6 to 7 and from d 18 to 19. On d 5 and 17, excreta collection pans were cleaned

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**Table 1. Composition of the negative control diet for broiler chicks fed varying concentrations of vitamin E for experiments 1 and 2**

<table>
<thead>
<tr>
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**Table 2. Calculated and analyzed concentrations of vitamin E in diets**

<table>
<thead>
<tr>
<th>Item</th>
<th>Vitamin E added (IU/kg)</th>
<th>Vitamin E analyzed (IU/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Experiment 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative control</td>
<td>0</td>
<td>17</td>
</tr>
<tr>
<td>Verxite (100 IU/kg)</td>
<td>100</td>
<td>101</td>
</tr>
<tr>
<td>Verxite (300 IU/kg)</td>
<td>300</td>
<td>266</td>
</tr>
<tr>
<td>Silica (100 IU/kg)</td>
<td>100</td>
<td>99</td>
</tr>
<tr>
<td>Silica (300 IU/kg)</td>
<td>300</td>
<td>240</td>
</tr>
<tr>
<td>Commercial verxite (30 IU/kg)</td>
<td>30</td>
<td>28</td>
</tr>
<tr>
<td>Commercial silica (30 IU/kg)</td>
<td>30</td>
<td>33</td>
</tr>
<tr>
<td>Experiment 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative control</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>Commercial verxite (30 IU/kg)</td>
<td>30</td>
<td>29</td>
</tr>
<tr>
<td>Commercial silica (30 IU/kg)</td>
<td>30</td>
<td>35</td>
</tr>
</tbody>
</table>
RESULTS

Diet Analysis

The NC diet with no vitamin E contained 17 and 13 IU/kg of vitamin E by analysis for experiment 1 and experiment 2, respectively (Table 2). Dietary analyses of the graded concentrations of vitamin E were lower than the anticipated values, but they were similar across the different carriers.

Growth Performance

In experiment 1, ADG was not affected ($P > 0.10$) by source or concentration of vitamin E (Table 3). There was a significant ($P < 0.01$) source × concentration interaction for ADFI. Daily feed intake decreased as concentration of VE increased, but ADFI increased as concentration of SE increased. There was also a significant ($P < 0.03$) source × concentration interaction for G:F. As concentration of VE increased in the diet, G:F increased, but as concentration of SE increased in the diet, G:F decreased. In experiment 2, ADG, ADFI, and G:F were not affected ($P > 0.10$) by source or concentration of vitamin E in the diets.

Excreta Analysis

The mean excreta vitamin E concentration at d 7 post-hatching was 17.2 IU/kg. Excreta samples were pooled over a 48-h collection period. Vitamin E excretion was higher ($P < 0.01$) in broilers fed VE than in those fed SE for both the 100 and 300 IU/kg concentrations in experiment 1 (Table 4). In addition, there was a significant ($P < 0.01$) source × concentration interaction. As the concentration of VE increased in the diet, the vitamin E excreted increased from 79 to 221 IU. As the concentration of SE increased in the diet, the amount of vitamin E excreted increased as well but to a lesser degree, from 0.10 of all excreta. On d 6 and 7 and on d 18 and 19, all excreta were removed from the pans. Care was taken to eliminate excreta contaminated with feathers or wasted feed. Excreta samples were frozen for later analysis. Excreta samples collected over the 2-d collection periods (d 6 and 7, d 18 and 19) were pooled and dried at 60°C for 48 h. Samples were lipid extracted and saponified in the presence of methanol and potassium hydroxide (BASF Corp., Florham Park, NJ). They were then analyzed for $\alpha$-tocopherol content with an HPLC instrument equipped with a fluorescence detector at 293-nm excitation and 326-nm emission wavelengths.

Liver samples were pooled by pen and homogenized by using a Kinematica Polytron benchtop homogenizer (Brinkman Instruments Inc., Westbury, NY) fitted with a standard generator with saw teeth. The samples were then extracted by using a typical lipid extraction method (Xu, 2002), with ultrasonic assistance applied instead of saponification to increase recovery. Lipid extract was analyzed for $\alpha$-tocopherol content with an HPLC instrument equipped with a fluorescence detector at 290-nm excitation and 330-nm emission wavelengths.

Statistical Analysis

All data were analyzed as a completely randomized design (Steel and Torrie, 1980), and the pens of chicks served as the experimental unit in both experiments. Data were analyzed by using the GLM of SAS (1990; SAS Institute Inc., Cary, NC). The liver vitamin E concentration data in experiment 1 had heterogeneous variances; therefore, the data were log transformed (Log + 1) before statistical analysis. The actual unadjusted means are shown in the tables. Orthogonal contrasts were used to determine differences between source of carrier, concentration of vitamin E, a source × concentration interaction, commercial concentrations (30 IU/kg) of vitamin E, and commercial concentrations of vitamin E compared with the NC.

<table>
<thead>
<tr>
<th>Diet</th>
<th>ADG (g)</th>
<th>ADFI (g)</th>
<th>Gain:feed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Experiment 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative control</td>
<td>44.36</td>
<td>60.96</td>
<td>0.723</td>
</tr>
<tr>
<td>Verxite (100 IU/kg)</td>
<td>45.04</td>
<td>60.96</td>
<td>0.736</td>
</tr>
<tr>
<td>Verxite (300 IU/kg)</td>
<td>44.66</td>
<td>58.73</td>
<td>0.744</td>
</tr>
<tr>
<td>Silica (100 IU/kg)</td>
<td>44.33</td>
<td>59.67</td>
<td>0.743</td>
</tr>
<tr>
<td>Silica (300 IU/kg)</td>
<td>44.19</td>
<td>63.23</td>
<td>0.700</td>
</tr>
<tr>
<td>Commercial verxite (30 IU/kg)</td>
<td>44.01</td>
<td>59.71</td>
<td>0.737</td>
</tr>
<tr>
<td>Commercial silica (30 IU/kg)</td>
<td>44.97</td>
<td>61.30</td>
<td>0.734</td>
</tr>
<tr>
<td>SEM</td>
<td>0.824</td>
<td>1.04</td>
<td>0.011</td>
</tr>
<tr>
<td>Experiment 2</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Negative control</td>
<td>44.63</td>
<td>59.78</td>
<td>0.747</td>
</tr>
<tr>
<td>Commercial verxite (30 IU/kg)</td>
<td>44.37</td>
<td>58.90</td>
<td>0.753</td>
</tr>
<tr>
<td>Commercial silica (30 IU/kg)</td>
<td>45.30</td>
<td>60.02</td>
<td>0.755</td>
</tr>
<tr>
<td>SEM</td>
<td>0.75</td>
<td>0.95</td>
<td>0.007</td>
</tr>
</tbody>
</table>

1ADG = average daily gain.
2ADFI = average daily feed intake.
3Experiment 1; source × concentration interaction, $P < 0.01$.
4Experiment 1; source × concentration interaction, $P < 0.03$.
5Data are means of 6 replications of 5 chicks each, Initial and final weights were 155 and 684 g, respectively. There were no significant differences between the control diet and the commercial verxite and commercial silica diets, or between the commercial verxite and commercial silica diets.
6Data are means of 6 replications of 5 chicks each, except the negative control and VE treatments, which had only 5 replications. Initial and final weights were 155 and 691 g, respectively.
basis (vitamin E analyzed in the diets). In experiment 1, on both a calculated and analyzed basis, percentage of vitamin E excreted was higher ($P < 0.01$) for broilers fed VE compared with those fed SE. On a calculated basis, there was a significant ($P < 0.05$) source × concentration interaction. As concentration of VE increased in the diet, percentage of vitamin E did not change, but as concentration of SE increased in the diet, percentage of vitamin E excreted increased from 42 to 30%. On an analyzed basis, there was a significant ($P < 0.08$) source × concentration interaction. As concentration of VE increased in the diet, percentage of vitamin E excreted increased from 91 to 97%, but as concentration of SE increased in the diet, percentage of vitamin E excreted decreased from 49 to 40%. On the basis of either the analyzed or calculated basis, percentage of vitamin E excretion was not different ($P > 0.10$) between commercial concentrations of VE and SE.

In experiment 2, there were no differences ($P > 0.10$) in percentage of vitamin E excreted between broilers fed commercial concentrations of VE or SE, with values of 35 and 36%, respectively, on a calculated basis. In addition, broilers fed both VE and SE had lower ($P < 0.09$) percentages of vitamin E excretion compared with those fed the NC diet (40%). On an analyzed basis, percentage of vitamin E excreted was higher ($P < 0.02$) in broilers fed VE than those fed SE, with values of 52 and 43%, respectively. Broilers fed both VE and SE had higher ($P < 0.01$) percentages of vitamin E excretion compared with those fed the NC diet (38%).

### Liver Analysis

Liver α-tocopherol concentration was not affected ($P > 0.10$) by carrier source of vitamin E in either experiment (Tables 4 and 5). However, in experiment 1 increased concentrations of vitamin E in the diet increased ($P < 0.01$) liver α-tocopherol concentrations. In addition, in experiment 2, liver α-tocopherol concentration was greater ($P < 0.01$) in chicks fed either source of vitamin E compared with broilers fed the NC.

### DISCUSSION

Daily gain was not affected by the source or concentration of vitamin E added to the diet in either experiment. Feed intake was decreased and feed efficiency was increased by increased concentrations of VE. The increase in G:F in experiment 1 was due to the decrease in ADFI, with no change in ADG. This is in contrast to research in poults, which indicated that growth performance was not affected by the type or concentration of vitamin E in the diet (Csallany et al., 1988; Applegate and Sell, 1996; Sell et al., 1997). In experiment 2, growth performance was not affected by the addition of vitamin E from either carrier.

Excreta vitamin E excretion and percentage of vitamin E excretion were higher in birds fed VE than in those fed SE at the 100 and 300 IU/kg concentrations, but not at the 30 IU/kg concentration in either experiment. On the basis of this response, it seems that the VE-bound vitamin E was less available to the bird. These data agree

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### Table 4. Vitamin E intake and wet and dry excreta weights and liver α-tocopherol concentrations for experiment 1

<table>
<thead>
<tr>
<th>Response</th>
<th>NC</th>
<th>100 VE</th>
<th>300 VE</th>
<th>100 SE</th>
<th>300 SE</th>
<th>C-VE</th>
<th>C-SE</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total feed intake (g)</td>
<td>901.9</td>
<td>869.3</td>
<td>866.2</td>
<td>890.72</td>
<td>973.8</td>
<td>893.6</td>
<td>879.7</td>
<td>31.5</td>
</tr>
<tr>
<td>Total wet excreta (g)</td>
<td>695.8</td>
<td>631.0</td>
<td>689.0</td>
<td>787.5</td>
<td>629.4</td>
<td>686.8</td>
<td>685.9</td>
<td>62.4</td>
</tr>
<tr>
<td>DM of excreta (%)</td>
<td>35.0</td>
<td>34.6</td>
<td>35.1</td>
<td>30.2</td>
<td>32.5</td>
<td>34.3</td>
<td>33.3</td>
<td>2.4</td>
</tr>
<tr>
<td>Total DM (g)</td>
<td>232.5</td>
<td>214.0</td>
<td>233.5</td>
<td>228.1</td>
<td>203.7</td>
<td>235.0</td>
<td>225.5</td>
<td>12.2</td>
</tr>
<tr>
<td>Vitamin E excreted (IU)</td>
<td>10.4</td>
<td>79.2</td>
<td>221.3</td>
<td>43.5</td>
<td>93.0</td>
<td>12.4</td>
<td>13.0</td>
<td>5.7</td>
</tr>
</tbody>
</table>

**Dietary calculated vitamin E**

- Vitamin E intake (IU) 15.3 101.7 274.6 104.2 308.7 42.0 41.3 6.6
- Vitamin E excreted (%) 68.5 78.1 81.0 41.7 30.1 29.7 31.4 3.5

**Dietary analyzed vitamin E**

- Vitamin E intake (IU) 14.9 87.7 230.0 104.2 308.7 42.0 41.3 6.6
- Vitamin E excreted (%) 70.3 90.6 96.7 49.1 39.8 49.4 45.0 4.2

<table>
<thead>
<tr>
<th>Liver α-tocopherol (IU/kg)</th>
<th>0.30</th>
<th>6.41</th>
<th>20.36</th>
<th>3.68</th>
<th>16.01</th>
<th>0.98</th>
<th>0.37</th>
<th>2.68</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-value</td>
<td>0.05</td>
<td>0.21</td>
<td>0.18</td>
<td>0.76</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.75</td>
</tr>
</tbody>
</table>

1Data are means of 6 replications of 5 chicks each. These data are for the 2-d collection period.
2NC = negative control; VE = verxite; SE = silica; C = commercial.
3Contrast values reported are $P > F$.
4Source VE (100, 300 IU/kg) vs. SE (100, 300 IU/kg).
5Concentrations 100 vs. 300 IU/kg.
6Source × concentration interaction.
7Source commercial concentrations SE vs. VE.
8Control vs. commercial concentrations of SE vs. VE.
9Data are means of 6 replicates with 6 samples per replicate. Contrast data are log transformed [log + 1].

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Table 5. Vitamin E intake and wet and dry excreta weights in grams and liver α-tocopherol concentrations for experiment 2

<table>
<thead>
<tr>
<th>Response</th>
<th>Diet</th>
<th>P-value $^3$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NC</td>
<td>C-VE</td>
</tr>
<tr>
<td>Total feed intake (g)</td>
<td>796.4</td>
<td>720.4</td>
</tr>
<tr>
<td>Collected excreta (g)</td>
<td>586.6</td>
<td>588.6</td>
</tr>
<tr>
<td>DM of excreta (%)</td>
<td>34.0</td>
<td>32.2</td>
</tr>
<tr>
<td>Total DM (g)</td>
<td>198.1</td>
<td>186.8</td>
</tr>
<tr>
<td>Vitamin E excreted (IU)</td>
<td>4.1</td>
<td>10.8</td>
</tr>
<tr>
<td>Dietary calculated vitamin E</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin E intake (IU)</td>
<td>10.4</td>
<td>31.0</td>
</tr>
<tr>
<td>Vitamin E excreted (%)</td>
<td>39.3</td>
<td>34.9</td>
</tr>
<tr>
<td>Dietary analyzed vitamin E</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin E intake (IU)</td>
<td>10.7</td>
<td>20.8</td>
</tr>
<tr>
<td>Vitamin E excreted (%)</td>
<td>38.0</td>
<td>51.9</td>
</tr>
<tr>
<td>Liver α-tocopherol (IU/kg) $^6$</td>
<td>1.07</td>
<td>3.77</td>
</tr>
</tbody>
</table>

$^1$Data are means of 6 replications of 5 chicks each, except treatment 1, which had 5 replications. These data are for the 2-d collection period.
$^2$NC = negative control; C = commercial; VE = verxite; SE = silica.
$^3$Contrast values reported are $P > F$.
$^4$Source commercial concentrations of SE vs. VE.
$^5$Negative control vs. VE as a commercial premix and silica as a commercial premix.
$^6$Data are means of 6 replicates, with 2 samples per replicate.

with Baker et al. (2006), who reported that VE was less available than SE when comparing liver, plasma, and muscle α-tocopherol concentrations. This response could be because VE binds the vitamin E more tightly than SE. Verxite has been shown to bind tightly to radioactive elements in ruminants (Hazzard, 1969; Hazzard et al., 1969). Jenkins and Palmquist (1984) reported that fatty acids from tallow absorbed to VE were not as digestible as fatty acids absorbed to other carriers. It also has been suggested that the VE may change where absorption takes place in the intestine (Hurley et al., 1990). These combined effects could have made the VE less available to the chicks, thereby causing higher excretion of the vitamin. This does not account for the commercial concentrations (30 IU/kg) of vitamin E having the same excretion percentage in both experiments. In addition, α-tocopherol concentrations in the liver were not affected by the source of vitamin E in the diet. This response indicates that although birds fed VE excreted a higher percentage of the vitamin E, the birds were able to store the same amount of α-tocopherol as birds fed SE. This seems to be especially so when using commercial concentrations of vitamin E. The route for excretion of vitamin E is through the bile (McDowell, 1989). Because of this, the excreta vitamin E excretion is less indicative of vitamin E absorption than is the liver concentration of α-tocopherol.

**Overall Conclusions**

Although birds excreted more vitamin E with VE as the carrier at high supplemental concentrations of vitamin E, the storage of vitamin E was not affected by the source of carrier. Despite the higher excretion of vitamin E from broilers fed vitamin E absorbed to VE at high concentrations, at the commercial concentrations of vitamin E in the diet, excretion of vitamin E was not different between sources of carrier. These results indicate that at commercial supplementation levels of vitamin E, VE is a comparable carrier to SE.

**REFERENCES**


