The objective of this study was to determine the effects of maternally supplemented natural- or synthetic-source vitamin E on suckling calf performance and immune response. In a 2-yr study, one hundred fifty-two 2- and 3-yr-old, spring-calving, Angus-cross beef cows were blocked by age, BW, and BCS into 1 of 3 isocaloric, corn-based dietary supplements containing 1) no additional vitamin E (CON), 2) 1,000 IU/d of synthetic-source vitamin E (SYN), or 3) 1,000 IU/d of natural-source vitamin E (NAT). Maternal supplementation began approximately 6 wk prepartum and continued until the breeding season. Colostrum from cows and blood from calves was collected 24 h postpartum for analysis of IgG concentration as an indicator of passive transfer and circulating α-tocopherol concentration. At 19 d of age, blood was collected from calves to determine the expression of CD14 and CD18 molecules on leukocytes. At 21 and 35 d of age, humoral immune response was measured by a subcutaneous injection, in the neck, with ovalbumin (20 mg; OVA) and blood samples collected weekly until d 63 of age to determine antibodies produced against OVA. At d 63 of age, calves were administered an intradermal injection of OVA (1 mg) in the neck to assess cell-mediated immunity, which was determined on d 65 of age by measuring nodule size with calipers. Circulating α-tocopherol concentrations were increased at both 24 h \( (P = 0.001) \) and at the day of initial OVA challenge \( (P < 0.001) \) in SYN and NAT compared with CON calves. No differences were detected \( (P > 0.05) \) for calf birth BW, ADG, or weaning BW. There were no differences \( (P > 0.05) \) in calf serum total IgG or cow colostrum total IgG at 24 h or presence of CD14 and CD18 receptors at d 19 of age. The NAT calves had a greater antigen response to OVA at d 63 than SYN calves \( (P = 0.01; \text{treatment } \times \text{ day interaction}) \). As an indicator of cell-mediated immunity to OVA, nodule size at 65 d of age was not affected \( (P = 0.92) \) by maternal dietary supplementation. In conclusion, calves suckling cows supplemented with natural- and synthetic-source vitamin E had increased circulating concentrations of α-tocopherol at 24 h, which appeared to continue throughout maternal supplementation; however, calf immune function and performance were not affected.

Key words: beef calf, immune function, performance, vitamin E

INTRODUCTION

A goal of commercial cow-calf producers is to optimize mass of calf weaned per cow exposed. Immune status can have a significant effect on calf growth and lifelong health (Wittum and Perino, 1995). Decreasing calf morbidity and mortality, as well as implementing methods to improve calf performance, are imperative to improve producer profitability.

Vitamin E supplementation enhances the immune system (Pepkowski et al., 1980; Reddy et al., 1986; Nemec et al., 1994) and is involved in humoral and cell-mediated immunity (Tanaka et al., 1979). Short-term supplementation of increased concentrations of vitamin E in sheep has been reported to enhance immune function (Hatfield et al., 2002), with a vitamin E deficiency resulting in impairment of cell-mediated and humoral immunity (Moriguchi and Muraga, 2000). Vitamin E requirement for beef cows has not been well established.
due to its interrelationships with other dietary components. However, dairy cow recommendations are to supplement 1.6 IU·kg of BW⁻¹·d⁻¹ of vitamin E, which is sufficient to decrease the incidence of nutritional myopathy (NRC, 2001).

Natural- and synthetic-source vitamin E differ in their chemical and physical composition. Natural-source vitamin E (RRR-α-tocopherol) is the most bioavailable tocopherol at 1.36 mg/1U, whereas synthetic-source vitamin E (all-rac-α-tocopherol) exhibits a bioavailability of 1 mg/1U. Supplementation of vitamin E to livestock commonly utilizes the synthetic-source vitamin E ester because of its decreased cost (Meglia et al., 2006; Wiltburn et al., 2008). We hypothesized that supplementation of 1,000 IU of vitamin E per day to beef cows would increase circulating concentrations of α-tocopherol and improve performance and immune function in suckling beef calves; therefore, our objectives were to evaluate the effects of maternal natural- or synthetic-source vitamin E supplementation on suckling calf immune function and performance.

**MATERIALS AND METHODS**

All protocols for this study were approved by the Purdue Animal Care and Use Committee.

**Experimental Design**

In a 2-yr study, 152 (2-yr-old, n = 78; 3-yr-old, n = 74) spring-calving Angus-cross beef cows [n = 77 in yr 1, 75 in yr 2; initial BW (±SEM) = 584 ± 11 kg; initial BCS = 5.3 ± 0.13; 1 = emaciated, 9 = obese; Wagner et al., 1988] were blocked by age (2- and 3-yr-old) and randomly assigned to 1 of 9 pens (3 pens per treatment). Cows were allotted so that each block across treatments was similar in BW and BCS. Beginning an average of 6 wk prepartum, cows were fed 1.6 kg·cow⁻¹·d⁻¹ of corn silage [7.8% CP, 69.5% TDN; 10.1 IU·kg⁻¹ of α-tocopherol; Diagnostic Center for Population and Animal Health (DCPAH), Lansing, MI], or 1,000 IU of natural-source vitamin E·cow⁻¹·d⁻¹ (NAT; Vitamin E 405 Natural Source, d-α-tocopheryl acetate, ADM Alliance Nutrition Inc., Quincy, IL), or 1,000 IU of natural-source vitamin E·cow⁻¹·d⁻¹ (NAT; Vitamin E 405 Natural Source, d-α-tocopheryl acetate, ADM Alliance Nutrition Inc.). Cows were maintained in a dry lot and allowed ad libitum access to native grass hay (19.3 IU·kg⁻¹ of α-tocopherol; Se >0.5 mg·kg⁻¹; DCPAH). All nutrients are expressed on a DM basis.

**Table 1. Effects of maternal supplementation of natural- or synthetic-source vitamin E on α-tocopherol concentrations in bovine colostrum or suckling calf serum**

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>Synthetic vitamin E²</th>
<th>Natural vitamin E²</th>
<th>SEM¹</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colostrum,³ µg·mL⁻¹</td>
<td>2.15⁺</td>
<td>3.08⁺</td>
<td>3.35ᵇ</td>
<td>0.15</td>
<td>0.03</td>
</tr>
<tr>
<td>Calf serum at 24 h, µg·mL⁻¹</td>
<td>0.68⁺</td>
<td>0.89ᵇ</td>
<td>1.11ᵇ</td>
<td>0.06</td>
<td>0.01</td>
</tr>
<tr>
<td>Calf serum at 21 d, µg·mL⁻¹</td>
<td>1.19⁺</td>
<td>1.54ᵇ</td>
<td>1.73ᵇ</td>
<td>0.10</td>
<td>0.02</td>
</tr>
</tbody>
</table>

*Means within a row with unlike superscripts differ.
²Greatest SEM are presented.
³Vitamin E provided in a corn-based supplement offered to dams and was formulated to contain 1,000 IU of vitamin E-cow⁻¹·d⁻¹; NAT supplement contained 2.5 g of natural-source vitamin E (ADM, Decatur, IL; 405 IU·g⁻¹); SYN supplement contained 2.00 g of synthetic-source vitamin E (ADM; 500.4 IU·g⁻¹); CON supplement contained no additional vitamin E. Cows were offered a free-choice mineral supplement that contained 32.7% trace mineralized salt, 32.7% dicalcium phosphate, 32.7% calcium carbonate, 1.7% vitamin A (30,000 IU·g⁻¹), and 0.10% vitamin D (350 kIU·kg⁻¹). Cows were fed corn silage [1.6 kg·cow⁻¹·d⁻¹; 10.1 IU·kg⁻¹ of α-tocopherol; Se >0.5 mg·kg⁻¹; DCPAH], and ad libitum access to native grass hay (19.3 IU·kg⁻¹ of α-tocopherol; Se >0.5 mg·kg⁻¹; DCPAH). All nutrients are expressed on a DM basis.
⁴Blood samples were taken via jugular venipuncture at 6 h postpartum.
⁵Blood samples were taken via jugular venipuncture at the day of initial ovalbumin challenge.
total antibodies produced to OVA as a measure of humoral immunity. At d 63 of age, calves were administered an intradermal injection of OVA (1 mg) in the neck to assess cell-mediated immunity, which was determined on d 65 of age by measuring nodule size with calipers. Calves were weighed at weaning, and weaning BW were adjusted (BIF, 1976) to a 205-d adjusted weaning weight to normalize performance.

**Antigen Preparation**

Ovalbumin was prepared at 20 mg·mL⁻¹ as described by Lake et al. (2006). For 100 mL of final solution, 25 mL of crystallized albumin (Sigma A-5503, St. Louis, MO) at 80 mg·mL⁻¹ was diluted in 80 mL of distilled H₂O, and 90 mL of 10% potassium alum was added. The pH was adjusted to 6.5 using 5 N NaOH; the mixture was centrifuged at 2,290 × g for 20 min at 4°C, and the supernatant was removed. The pellet was washed with 200 mL of 1:10,000 merthiolate saline, centrifuged, and washed as described 2 more times. The remaining pellet was brought to 100-mL volume with 1:10,000 merthiolate saline and stored at 5°C. A 5-mL dose was administered subcutaneously in the neck at d 21 and 35 of age.

**Intradermal Antigen Preparation**

Ovalbumin (1 mg) was suspended in 0.01 M phosphate buffer diluted in physiological saline and prepared as adapted from Lake et al. (2006). For 100-mL of 0.01 M phosphate buffer, 2.8 mL of solution A (1.36 g of KH₂PO₄ dissolved in 100 mL of distilled H₂O) was combined with 7.2 mL of solution B (1.42 g of Na₂HPO₄ dissolved in 100 mL of distilled H₂O), and 90 mL of 10% protein was added. Fifty milliliters were aliquoted as a control injection, and 0.5 g of OVA was added to the remaining 50 mL as the antigen. A 200-μL dose of each control and OVA were injected intradermally into the neck in 2 separate locations.

**Sample Collection**

Blood samples were collected via the jugular vein into 5-mL vacuum tubes with no additive (Becton, Dickinson and Co., Franklin Lakes, NJ) 24 h after birth for analysis of α-tocopherol concentration and total IgG concentration, and weekly beginning at 21d of age through 63 d of age for analysis of anti-OVA antibodies produced against the OVA antigen. Blood samples were immediately refrigerated for 8 h, centrifuged at 1,000 × g for 20 min at 4°C, and serum was collected and stored at −20°C. Colostrum was collected via hand-stripping 6 h after parturition and stored at −20°C for analysis of total IgG and α-tocopherol concentrations. At 19 ± 2 d of age, whole blood samples were collected from calves via the jugular vein into 10-mL vacuum tubes containing acid citrate dextrose and immediately analyzed for CD14 and CD18 total fluorescence.

**Sample Analyses**

Total IgG concentrations were measured at 24 h of age using the Quantitative Bovine IgG ELISA kit (Bethyl Laboratories Inc., Montgomery, TX). One microliter of capture antibody (sheep anti-bovine IgG-heavy chain antibody affinity purified, Bethyl Laboratories Inc.) was diluted in 100 μL of coating buffer (0.05 M carbonate-bicarbonate, pH 9.6) and placed in each well of a 96-well plate (Nunc MaxiSorp C bottom well Modules and Frames, Rochester, NY). The plate was coated for 1 h at room temperature, the contents of the wells were aspirated and washed with the wash solution 3 times (50 mM Tris, 0.14 M NaCl, 0.05% Tween 20, pH 8.0), and 200 μL of postcoat solution (50 mM Tris, 0.14 M NaCl, 0.05% Tween 20, pH 8.0) was added to each well and incubated for 30 min at room temperature. Plates were washed as described previously. Standards were diluted in sample diluent (50 mM Tris, 0.14 M NaCl, 0.05% Tween 20, pH 8.0) as described in the kit with serum samples diluted to 1:400,000 and colostrum samples to 1:900,000. One hundred microliters of each sample and standard were plated and incubated at room temperature for 60 min. The plate was washed 5 times with wash solution. The HRP Conjugate (Sheep anti-Bovine IgG-heavy chain Antibody Alkaline Phosphatase Conjugated, Bethyl Laboratories Inc.) was diluted 1:75,000 in sample diluent, and 100 μL was pipetted into each well. The plate was incubated for 60 min in the dark and washed 5 times as described previously. One hundred microliters of enzyme substrate solution (equal volumes of TMB Peroxidase Substrate and Peroxidase Solution B, Bethyl Laboratories Inc.) was added to each well, incubated for 10 to 12 min in the dark, and the reaction was stopped by adding 100 μL of 2 M H₂SO₄ to each well. Absorbance was determined at 450 nm on a microplate reader (Dynex MRX-Revelation Microplate Absorbance Reader, Dynex Technologies Inc., Chantilly, VA) and results were verified with an MRX Verification Plate (Dynex Technologies Inc.). Concentration of IgG was determined from the 4 parameter logistic curve-fit created from the standards.

Serum samples at 24 h and 21 d of age and colostrum samples were analyzed for vitamin E as α-tocopherol by HPLC as described by Horn et al. (2010). Serum samples collected at 21, 28, 35, 42, 49, 56, and 63 d of age were analyzed for total anti-OVA specific antibodies produced against the OVA challenge as described by Lake et al. (2006). Briefly, serum was thawed and mixed with serum conjugate diluent (diluted to 1:40). Fifty microliters of OVA carbon-coating buffer was added to each well of a 96-well plate (Immulon 2, Dynex Tech., Chantilly, VA) at 4°C for 24 h. The plates were rinsed 3 times with Tween buffer solution and patted dry. The plates were incubated with 100 μL of 10% fetal bovine...
serum carbon-coating buffer, incubated for 2 h at room temperature, and rinsed 3 times as described. Serum samples were added (50 μL) to each well and incubated at 37°C for 1 h. The plates were rinsed 3 times as described previously and incubated with 50 μL of rabbit anti-bovine IgG-horseradish peroxidase conjugate for 30 min at 37°C. After rinsing 3 times as described previously, 50 μL of enzyme substrate was allowed to develop for 3 to 4 min, and the reaction was terminated with the addition of 50 μL of 2 M H2SO4. Total antibody production in response to OVA challenge was determined by a dual absorbance (490 and 595 nm) microplate reader (Dynex MRX-Revelation Microplate Absorbance Reader, Dynex Technologies Inc.) verified with an MRX Verification Plate (Dynex Technologies Inc.). The dual absorbance reading was used to reduce optical variation. Specifically, the longer wavelength was used to subtract the optical variation that was introduced from the bottom of the plate. Data presented represent absorbance of total anti-OVA-specific antibodies (inter- and intraassay CV = 16.5 and 10.4, respectively).

Flow cytometry was utilized to measure the expression of CD14 and CD18 molecules on leukocytes at d 19 of age. Whole blood samples in acid citrate dextrose tubes were placed in a 37°C water bath for 1 h. Five hundred microliters of whole blood was added to two 12 × 75-mm polypropylene tubes labeled as cells only (containing no antibody to serve as control tube) and CD14/18 (containing CD14 and CD18 antibody to measure protein expression). Ten microliters of phycoerythrin-conjugated monoclonal mouse anti-human CD14, clone TUK4 (Dako, Glostrup, Denmark) and 20 μL of monoclonal mouse anti-human LFA-1 β-chain fluorescein isothiocyanate CD18, clone MHM23 antibodies (Dako) were added to the CD14/18 tube. The cells-only tube remained without antibody to serve as an unstained control and allowed gating of auto fluorescence. Tubes were incubated in a 37°C water bath for 1 h. After incubation, cells were lysed by hypotonic lysis for 1 min with 900 μL of cold, sterile water. One hundred microliters of 10X Hanks’ balanced salt solution (HBSS) was added to restore isotonicity, and tubes were centrifuged at 2,000 × g for 5 min at 4°C. The supernatant was removed and cells were lysed and centrifuged for a second time as described above. The supernatant was removed and 2 mL of 1X HBSS was added and mixed via pipetting, and centrifuged as described above. The supernatant was removed and the pellet was resuspended in 1 mL of 2% paraformaldehyde suspended in HBSS. The sample was diluted with 1× HBSS and analyzed on the CoulterEpics XL-MCL Flow Cytometer and System II software (Beckman Coulter Inc., Miami, FL). The flow cytometer used a 488-nm air-cooled argon laser for excitation, a 525-band pass filter for detection of fluorescein isothiocyanate emissions and a 575-band pass filter for detection of phycoerythrin emission. For each sample, a total of 10,000 cells were analyzed.

In yr 2 only, cell-mediated immunity was assessed at 63 d of age by a 100-μL intradermal antigen injection of 1 mg of OVA suspended in 0.01 M phosphate buffer diluted in physiological saline in the neck. The response to the intradermal injection was determined at 48 h (d 65 of age) by measuring the diameter of the nodule formed around the injection site with calipers.

### Statistical Analyses

Calf birth BW, weaning BW, ADG, CD14 and CD18 total fluorescence, serum and colostrum IgG, and node size were analyzed as a 1-way ANOVA (GLM procedure, SAS Inst. Inc., Cary, NC). Pen was the experimental unit for growth performance and blood analysis data. The model included the effects of maternal dietary supplement, year, pen (when applicable), cow age, and all possible interactions. Significance was declared at P ≤ 0.05. Titer concentrations for the OVA challenge were analyzed as repeated measures using a mixed model (MIXED procedure). The model included the effects of maternal dietary supplement, day of sampling, and the supplement × day interaction. Calf within maternal dietary supplement was considered random, and day of sampling was the repeated variable. Using likelihood ratio testing, an autoregressive order 1 structure was deemed most appropriate for the within-subject effects (the effects associated with days of sampling; Littell et al., 2000). When the main effects were significant (P ≤ 0.05), means were compared using the Tukey-Kramer adjustment.

### Results

The effects of maternal supplementation of natural- or synthetic-source vitamin E on serum and colostrum concentrations of α-tocopherol are presented in Table 1. Colostral α-tocopherol concentration was greater (P = 0.03) in NAT and SYN cows compared with CON cows. Alpha-tocopherol concentrations were also greater at 24 h (P = 0.01) and 21 d of age (P = 0.02) in the SYN and NAT calves compared with CON calves.

The effects of maternal supplementation of natural- or synthetic-source vitamin E on calf performance are presented in Table 2. Maternal supplementation of synthetic- or natural-source vitamin E did not affect calf birth BW (P = 0.85), weaning BW (P = 0.57), or ADG (P = 0.71).

Colostrum IgG concentrations (P = 0.94) at calving (32.62, 30.22, and 27.93 mg·mL⁻¹ in CON, SYN, and NAT cows, respectively), and calf serum IgG concentrations (P = 0.62) at 24 h of age (15.15, 15.83, and 16.57 mg·mL⁻¹ in CON, SYN, and NAT calves, respectively) did not differ due to dietary supplement.

At d 19 ± 2 d of age, CD14 (P = 0.88; 10.12, 7.56, and 9.38 in CON, SYN, and NAT calves, respectively) and CD18 (P = 0.75; 15.22, 10.65, and 14.31 in CON, SYN, and NAT calves, respectively) were not affected by maternal dietary vitamin E supplementation.
The effects of maternal supplementation of natural- or synthetic-source vitamin E on total anti-OVA antibodies produces against OVA are presented in Figure 1. On d 21 of age, an OVA challenge was initiated. A maternal supplement × day of sampling interaction was detected ($P = 0.01$) for total anti-OVA antibodies produced against OVA where NAT calves responded greater than SYN calves at d 63. Dietary supplement did not affect nodule size ($P = 0.92$; 1.76, 1.82, and 1.77 cm in CON, SYN, and NAT calves, respectively) for the intradermal challenge at d 65 of age in response to OVA at 1 mg.

There were no interactions ($P < 0.05$) for all other variables; however, differences due to year existed in the study. In yr 1 of the study, α-tocopherol concentration in calves at 21 d of age was greater ($P < 0.001$) than yr 2. Calf birth BW ($P < 0.0001$), weaning BW ($P < 0.001$), and ADG ($P < 0.001$) were lighter in yr 1 compared with yr 2 calves. Calf serum IgG concentrations ($P < 0.001$), cow colostral IgG concentrations

Table 2. Effects of maternal supplementation of natural- or synthetic-source vitamin E on calf performance

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>Synthetic vitamin E^2</th>
<th>Natural vitamin E^2</th>
<th>SEM^1</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth BW, kg</td>
<td>36.35</td>
<td>36.49</td>
<td>36.85</td>
<td>1.10</td>
<td>0.85</td>
</tr>
<tr>
<td>Weaning BW,^3 kg</td>
<td>273.56</td>
<td>277.07</td>
<td>278.40</td>
<td>5.68</td>
<td>0.57</td>
</tr>
<tr>
<td>ADG,^4 kg</td>
<td>0.64</td>
<td>0.65</td>
<td>0.65</td>
<td>0.03</td>
<td>0.71</td>
</tr>
</tbody>
</table>

^1Greatest SEM are presented.

^2Vitamin E provided in a corn-based supplement offered to dams and was formulated to contain 1,000 IU of vitamin E ·cow−1·d−1; NAT supplement contained 2.5 g of natural-source vitamin E (ADM, Decatur, IL; 405 IU·g−1); SYN supplement contained 2.00 g of synthetic-source vitamin E (ADM; 500.4 IU·g−1); CON supplement contained no additional vitamin E. Cows were offered a free-choice mineral supplement that contained 32.7% trace mineralized salt, 32.7% dicalcium phosphate, 32.7% calcium carbonate, 1.7% vitamin A (30,000 IU·g−1), and 0.10% vitamin D (350 kIU·kg−1). Cows were fed corn silage [1.6 kg·cow−1·d−1; 10.1 IU·kg−1 of α-tocopherol; Se >0.5 mg·kg−1; Diagnostic Center for Population and Animal Health (DCPAH), Lansing, MI], corn (0.91 kg·cow−1·d−1; 8.3 IU·kg−1 of α-tocopherol; Se >0.5 mg·kg−1; DCPAH), and ad libitum access to native grass hay (19.3 IU·kg−1 of α-tocopherol; Se >0.5 mg·kg−1; DCPAH). All nutrients are expressed on a DM basis.

^3205-d adjusted weaning BW (BIF, 1976).

^4ADG calculated at weaning: (actual weaning BW – birth BW) ÷ days of age.
gested that serum concentrations less than 2 μg·mL−1 were greater in yr 1 compared with yr 2 calves.

**DISCUSSION**

Tocopherols may pass through placental membranes as well as mammary glands; therefore, the diet of the dam may directly influence vitamin E concentrations in nursing calves. Small amounts of vitamin E are transferred from dam to young in utero due to the decreased amount of lipids that are transferred through the placenta, although this increases during the last third of pregnancy (Debier and Laronelle, 2005), but less than 2% of dietary vitamin E is transferred from feed to milk (McDowell, 1989). The increased α-tocopherol concentration in calves at birth agrees with Mahan (1991), who reported that supplementation of vitamin E to sows increased α-tocopherol concentrations in neonatal pigs.

In agreement with previously reported data, vitamin E supplementation increased the α-tocopherol in colostrum (Weiss et al., 1994; Meglia et al., 2006). Colostrum contains more vitamin E than milk due to the accumulation of fat globules within the colostrum. For example, Weiss et al. (1990) reported an increase in the vitamin E content of colostrum from 5.3 to 7.5 μg·mL−1 when dairy cows were supplemented with 1,000 IU·d−1 of vitamin E. Likewise, Zobell et al. (1995) reported increased concentrations of tocopherols in beef cow colostrums due to supplementation of 1,000 IU of vitamin E. Calves that receive colostrum after 24 h of age will have decreased concentrations of vitamin E than those receiving colostrum before 24 h of age (Debier and La- ronelle, 2005).

Hidiroglou et al. (1992) and Wichtel et al. (1996) suggested that serum concentrations less than 2 μg·mL−1 are deficient and those greater than 4 μg·mL−1 are adequate; therefore, the present study suggests that all calves were deficient in vitamin E, even though NAT and SYN dams were supplemented with additional vitamin E; however, calves in the current study did not exhibit symptoms of vitamin E deficiency. The d 21 concentrations presented suggest similarities for synthetic- and natural-source vitamin E and is contrary to other studies (Hidiroglou et al., 1992, 1995), which present differing serum concentrations depending on vitamin E source in beef cows. Concentrations of α-tocopherol on d 21 were greater in yr 1 compared with yr 2, although concentration of α-tocopherol in cows at calving did not differ by year (data not shown).

Vitamin E supplementation did not affect calf birth BW, ADG, or weaning BW in the current study, which agrees with previously reported data (Hidiroglou et al., 1995; Rivera et al., 2002; Carter et al., 2005). In contrast, Pehrson et al. (1991) reported improved growth in young calves supplemented vitamin E; however, calves in that study not receiving supplementation were deficient in vitamin E (serum α-tocopherol concentration <1 μg·mL−1), which likely created differences between treatments. Calves in the current study are all considered deficient in vitamin E (serum α-tocopherol concentration <2 μg·mL−1), even when NAT and SYN were maternally supplemented with 1,000 IU·d−1; therefore, this may explain the lack of differences in ADG and weaning BW between treatments.

Calves in yr 1 had lighter birth BW, as well as less ADG and weaning BW compared with yr-2 calves. The decreased birth BW in yr 1 calves helps explain the concurrent decrease in ADG and weaning BW (Vaccaro and Dillard, 1966). Interestingly, dams of calves in yr 1 had increased BW and BCS compared with cows in yr 2 (data not shown), but their calves displayed decreased growth compared with cows in yr 2. McMorris and Wilton (1986) reported increases in calf birth BW, weaning BW, and a trend for ADG to increase as cows increase in BW, although Fiss and Wilton (1993) reported no effect of cow BW on calf ADG or weaning BW, but did report a positive correlation between cow BW and calf birth BW.

Passive transfer of IgG did not differ in calf serum or cow colostrum due to maternal vitamin E supplementation in the current study. Likewise, Lacetera et al. (1996) reported no differences in colostrum IgG content when dairy cows were supplemented vitamin E during late pregnancy, and Bass et al. (2001) reported similar calf IgG serum concentration in beef cows whose dams were supplemented 1,000 IU of vitamin E·d−1 compared with nonsupplemented cows. When sows were supplemented with vitamin E, piglet serum IgG and sow colostrum IgG did not differ due to vitamin E supplementation (Nemec et al., 1994).

Cell-surface expression of CD14 and CD18 proteins did not differ due to vitamin E supplementation. Leukocyte function is an important component to health of the animal, and CD14 and CD18 are crucial in controlling infections caused by gram-negative bacteria (Paape and Capuco, 1997). To our knowledge, no studies have been published reviewing the presence of CD14 and CD18 receptors in beef calves; however, beef cows injected with vitamin E and Se exhibited increased neutrophil phagocytic activity compared with deficient cows (Gyang et al., 1984). The lack of differences between treatments in the current study may be due to the overall good health of the calves or the lack of stimulus before receptor measurement. Further research investigation the expression these molecules on leukocytes would aid in increased understanding of activation of innate immunity in relation to vitamin E supplementation.

Total anti-OVA antibodies produced against OVA did not differ between maternal vitamin E supplementation treatments. This is contrary to previously reported data where dairy calves orally supplemented 125 IU of vitamin E·d−1 demonstrated a tendency to produce more antibodies to bovine herpesvirus-1 vaccine compared with nonsupplemented calves. Reddy et al. (1987) reported that supplementation of vitamin E increased B and T cell response in dairy calves supplemented up to
500 IU·d⁻¹, whereas Tengerdy et al. (1983) reported an increase in humoral immunity due to synthetic-source vitamin E supplementation in sheep. Weaned lambs supplemented vitamin E had increased antibody titers to Clostridium perfringens toxoid D compared with nonsupplemented lambs. Lack of overall response in the current study may be confounded by α-tocopherol status of all calves, where NAT and SYN calves had increased concentrations compared with CON but were still considered deficient because concentrations were less than 2 μg·mL⁻¹. Increased anti-OVA antibody production in response to OVA at d 63 in NAT compared with SYN calves may be the result of increased humoral immunity due to natural-source supplementation of vitamin E, suggesting a prolonging effect of vitamin E on antibody response.

As an in vivo measure of cell-mediated immunity, an intradermal challenge was implemented using OVA (Lake et al., 2006). There were no differences due to maternal vitamin E supplementation at 48 h postchallenge; however, 48 h postchallenge may not have offered enough time for an adequate response. Geriatric dogs receiving vitamin E supplementation with dietary n-6 and n-3 fatty acids exhibited a greater nodule-size response between 72 and 96 h postchallenge compared with 24 h postchallenge, and vitamin E increased the immune response in dogs receiving treatments of less and more α-tocopherol and less n-3 fatty acids (Hall et al., 2003). In conclusion, calves suckling cows supplemented with natural- and synthetic-source vitamin E had increased circulating concentrations of α-tocopherol at 24 h and appeared to continue throughout maternal supplementation regardless of source of vitamin E.

**LITERATURE CITED**


