Vitamin E supplementation of newly arrived feedlot calves[1,2]

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ABSTRACT: Seven hundred fifteen crossbred (primarily British) calves purchased in southern Oklahoma and northern Texas auction barns were received at the Willard Sparks Beef Research Center, Stillwater, OK, and used to study effects of duration (days) of vitamin E feeding during a 42-d receiving period on animal performance, health, and serum cholesterol and vitamin E concentrations. Upon arrival, calves were blocked by load (seven loads), sorted by BW (light, n = 4 pens per load; and heavy, n = 4 pens per load), and assigned randomly to one of four dietary treatments (n = 2 pens per load; 14 pens per treatment). Experimental diets were formulated to provide 2,000 IU·calf^{-1}·d^{-1} of supplemental vitamin E (DL-α-tocopherol acetate) for 0 (CON), 7 (E7), 14 (E14), or 28 (E28) d. Vitamin E was delivered in a pelleted supplement that was added to the basal diet in decreasing concentrations as DMI increased (2.0 kg of DMI = 6%; 4.0 kg of DMI = 4%; and 6.0 kg of DMI = 2%). Serum samples were collected on d 0, 14, 28, and 42 for determination of cholesterol, α-tocopherol (d 0, 28, and 42), and antibody (IgG) concentrations. Duration of vitamin E supplementation did not affect ADG (0.98 kg/d; $P = 0.56$) or G:F (0.189; $P = 0.87$). Serum cholesterol concentrations decreased (day effect; $P < 0.001$) for all treatments from d 0 (average = 127 mg/100 mL) to 14 (average = 62 mg/100 mL). Serum α-tocopherol decreased (day effect; $P < 0.001$) from d 0 (5.2 μg/mL) to 28 (1.8 μg/mL); however, on d 28, a greater ($P < 0.001$) serum α-tocopherol concentration was observed for E28 (3.4 μg/mL) calves than for CON (1.1 μg/mL), E7 (1.2 μg/mL), or E14 (1.5 μg/mL) calves. Respiratory disease was diagnosed in 64.6% of calves in this study. Medical costs were less ($P = 0.08$) for calves fed vitamin E for 28 d ($4.88/calf) than for calves fed the control diet ($6.29/calf). Carcass characteristics were not affected ($P = 0.19$ to 0.88) by dietary treatments. Supplemental vitamin E formulated for 2,000 IU·calf^{-1}·d^{-1} had little influence on performance and overall health status of calves under our experimental conditions; however, the increased serum concentrations of α-tocopherol when vitamin E was fed for 28 d suggests that any potential effects of vitamin E on health status might be time-dependent.

Key Words: Calves, Disease, Stress, Vitamin E

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Introduction

The nebulous mechanisms of stress in cattle caused by marketing, transit, weaning, and other management practices and its interaction with infectious disease has long been recognized, but not well understood (Breazile, 1988). Vitamin E is a potent lipid-soluble antioxidant that functions in the prevention of chronic diseases associated with oxidative stress (Cipriano et al., 1982; Eicher-Pruiett et al., 1992; Galyean et al., 1999). Vitamin E was shown to have positive effects on the immune system of young dairy calves (Cipriano et al., 1982; Eicher-Pruiett et al., 1992) and to decrease morbidity and increase performance in receiving cattle (Gill et al., 1986; Hays et al., 1987; Galyean et al., 1999). Although many studies have attempted to determine the optimal level of vitamin E required in the diet of ruminants, a definitive value remains unclear. The NRC (1996) suggested a minimum of 400 to 500 IU·calf^{-1}·d^{-1} of vitamin E for 250-kg calves during the receiving and starting period.

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Table 1. Origin, date received, calves/load, and average body weight of calves received at the Willard Sparks Beef Research Center for a vitamin E supplementation experiment

Load	Origin	Date received	Calves/load[a]	Average BW, kg ± SD
1	Texas-Oklahoma	July 15	86	234 ± 33
2	Texas-Oklahoma	August 12	85	233 ± 33
3	Texas-Oklahoma	August 20	85	227 ± 33
4	Texas-Oklahoma	September 4	130[b]	151 ± 11
5	Texas-Oklahoma	October 4	111	168 ± 33
6	Texas-Oklahoma	December 8	106	172 ± 33
7	Texas-Oklahoma	December 16	112	174 ± 33

[a]Total cattle received = 715. Not all cattle completed the 42-d receiving trial due to mortality or morbidity. In total, 21 calves were removed due to chronic morbidity or death loss (<1%).
[b]Bulls and steers; all other loads were heifers.

Decreased plasma α-tocopherol concentrations were observed in cattle in response to transit stress (Han et al., 1999) and deprivation from feed and water (Nockels et al., 1996). The decrease in plasma vitamin E concentrations suggests that supplemental vitamin E may be important for optimal function of the immune system during times of stress. Whereas previous research has attempted to quantify an optimal concentration of vitamin E fed for extended periods of time, our objective was to measure the influence of a high dose of supplemental dietary vitamin E (2,000 IU·calf^{-1}·d^{-1}) fed for either 0, 7, 14, or 28 d in a 42-d receiving period on animal performance (total gain, ADG, and G:F) and health (number of antimicrobial treatments). Additionally, serum cholesterol and α-tocopherol concentrations were quantified to report physiological responses to dietary treatments.

Materials and Methods

Animals, Design, and Processing

Seven truckloads of sale barn-origin crossbred calves (568 heifers [six loads] with an average initial BW = 197 ± 33 kg; and 126 bulls and steers [one load], with an average initial BW =151 ± 11 kg) were received at the Willard Sparks Beef Research Center, Stillwater, OK, from July to mid-December (Table 1). Calves were purchased from numerous auction barns in south central Oklahoma and northern Texas, hauled (no more than 4 h) to a facility in central Oklahoma and sorted into truckload lots. Calves were then hauled approximately 145 km to our facility. Upon arrival (d 0), calves were allowed to commingle and rest for at least 1 h in a processing facility alley before a preprocessing procedure. This procedure included visual assessment of overall health, weighing of each calf, and application of a sequentially numbered identification tag in the left ear. Calves were distributed randomly to six holding pens for no more than 36 h before initiation of the study. While in these holding pens, 0.9 kg of prairie hay and 1.4 kg of the control diet (Table 2) were fed per calf. On d 1, calves were processed at approximately 0600 before feeding. Processing included individual BW measurment; vaccination for infectious bovine rhinotracheitis/parainfluenza-3 virus/bovine viral diarrhea/bovine respiratory syncytial virus (BRSV-Vac 4, [Bayer Corp., Shawnee Mission, KS] 2 mL i.m.); vaccination for clostridial organisms (Vision-7 [Bayer Corp.] 2 mL s.c. [heifers]) or seven-way Clostridial plus tetanus (Covexin 8 [Schering-Plough Animal Health, Kenilworth, NJ] 5 mL s.c. [bulls and steers]); and treatment with anthelmintics for internal and external parasites (Ivomec-Plus, Merial Animal Health, Duluth, GA; 1.0 mL/50 kg of BW s.c.). Cattle were revaccinated with viral respira-

Table 2. Composition of diets, DM basis

Ingredient, % of diet	Control[a]	Experimental
Soybean hulls	32.5	32.5
Whole corn	27.0	27.0
Wheat middlings	17.0	15.0
Sparks 99 supplement[b]	13.5	13.5
Cottonseed hulls	10.0	10.0
B-171 supplement[c]	0.0	2.0
Nutrient, % DM basis[d]		
NE_m, Mcal/kg	1.82	1.82
NE_g, Mcal/kg	1.13	1.13
TDN, %	72.0	72.0
NDF, %	39.0	39.0
Fat, %	2.9	2.9
Crude protein, %	14.8	14.8
Ca, %	1.1	1.1
P, %	0.8	0.8
K, %	0.5	0.5

[a]Control diet provided 28.2 IU vitamin E/kg DM from added and natural sources.
[b]Contained (DM basis): cottonseed meal (55.16%); soybean meal (48% CP, 30.6%), limestone (5.4%); pellet binder (6.5%); salt (1.9%); vitamin A (30,000 IU/g, 0.15%), vitamin E-50 adsorbate (0.02%, Roche Vitamins, Nutley, NJ); Bovatec 68 (0.09%, Alpharma, Fort Lee, NJ), and Se (0.02%, 0.18%); manufactured at the Oklahoma State Univ. Feed Mill.
[c]Contained (DM basis): wheat middlings (97%) and vitamin E-50 Adsorbate (3.0%). Fed at a constant rate of 0.14 kg·calf^{-1}·d^{-1} to provide 2,000 IU of supplemental vitamin E; supplement inclusion decreased as DMI increased; manufactured at the Oklahoma State Univ. Feed Mill.
[d]Crude protein based on actual laboratory analysis; all other values based on NRC (1996).

tory vaccine on d 14. For Load 4, an equal number of bulls and steers were assigned to each pen. Bulls remained intact until after the end of the 42-d receiving period, after which they were surgically castrated before being transported to a commercial finishing yard.

For each load, calves were sorted by initial weight (d 0) into two weight classes, light and heavy, and assigned randomly within weight class to one of four dietary treatments. Treatments were assigned randomly to eight pens (10 to 16 calves per pen depending on load). Dietary treatments were represented by the number of days that the control diet was supplemented with vitamin E (DL-α-tocopherol acetate; B-171 supplement; Table 2): 0 d = **Control**; 7 d = **E7**; 14 d = **E14**; or 28 d = **E28**. The control diet was formulated to contain 28.2 IU/kg of diet DM, which is within the range (15 to 60 IU/kg of DM) suggested by NRC (1996) for young calves. The vitamin E supplement (B-171) was included in the diet to provide a constant quantity of vitamin E (2,000 IU/d) as DMI increased (2.0 kg/d of DMI = 6%; 4.0 kg/d of DMI = 4%; and 6.0 kg/d of DMI = 2%). All experimental procedures were reviewed and approved by the Oklahoma State University Institutional Animal Care and Use Committee.

After d-1 processing, calves were immediately taken to their assigned pens, and 2.3 kg/calf of the control or experimental diets were delivered into fence-line concrete feed bunks (12.2 m of linear bunk per pen). Prairie hay was fed for the first 7 d only (0.75 kg·calf^{-1}·d^{-1}). Feed was offered on an ad libitum basis. The basal diet was formulated to meet or exceed NRC (1996) requirements and contained lasalocid (Bovatec, Alpharma, Fort Lee, NJ) as a low-level coccidiostat (Table 2). Nutrient composition is shown in Table 2. Pen size was uniform across all treatments (12.2 × 30.5 m), and alternating pens shared automatic water basins. Feed was delivered once daily at approximately 0700. Feed was delivered twice daily during inclement weather to provide clean, dry feed for a majority of each day. Diets and supplements were sampled and analyzed for N (AOAC, 1996) and α-tocopherol acetate (J. Wilson, Roche Laboratories, Nutley, NJ).

Cattle were weighed individually on d 0, 14, 28, and 42 of the experiment. On d 41, cattle received half the previous day's ration, and calves were not permitted access to water from 1700 until after the final weight was obtained on d 42.

Before d 0 processing, a subsample of six calves per pen or 12 calves per dietary treatment from each load was selected randomly for whole blood sample collection. On d 0, 14, 28, and 42, 30 mL of whole blood was collected via jugular venipuncture into three 10-mL Vacutainer tubes (no anticoagulant; Becton Dickinson, Franklin Lakes, NJ). After collection, blood was allowed to clot at room temperature, stored overnight at 4°C, centrifuged (2,500 × g at 4°C for 15 min), and stored at −10°C until laboratory analyses could be conducted. Storage tubes with serum designated for vitamin E analysis were purged with N_2 gas to minimize degradation of vitamin E during storage.

Health Assessment

Cattle were closely observed each morning at approximately 0630 by or under the supervision of experienced veterinary personnel (Oklahoma State University College of Veterinary Medicine) for signs of respiratory and other diseases. Two or more clinical signs of disease were required to designate a calf as sick and thereby classify the calf as eligible for further clinical review and therapeutic antimicrobial treatment. Visual signs indicating clinical disease included depression, lack of fill, occasional soft cough, physical weakness, altered gait, and ocular or nasal discharge (R. A. Smith, Stillwater, OK, personal communication). Sick calves were removed from their pen, taken to the processing area, and restrained in a squeeze chute, where their BW was recorded and rectal temperature measured. If rectal temperature was greater than 40°C, a regimen of antimicrobial treatment therapy was followed (Table 3) and calves were returned to their home pen. Regardless of health status, all information was recorded on an individual sick card and filed by pen for future reference.

Serum Analyses

Serum cholesterol concentrations were quantified using an enzymatic procedure (total cholesterol kit No. 352, Sigma Diagnostics, St. Louis, MO). Serum samples were analyzed at the University of Florida Institute of Food and Agricultural Sciences for vitamin E concentration (Njeru et al., 1992). The procedure utilized an ABI Analytical Spectraflow 400 HPLC with an EQC 10 μSi 60A, 4.6 × 250 mm column (Whatman Int., Ltd., Maidstone, U.K.), a Hitachi L-7485 fluorescence detector (excitation wavelength = 290 nm; emission wavelength = 330 nm; Hitachi, Schaumburg, IL), a Perkin-Elmer ISS-100 autosampler, and a Perkin-Elmer LCI-100 integrator (Perkin-Elmer, Boston, MA). Standards (T-3251 DL-α-tocopherol, 95%; stock concentration = 5 mg/mL in 2-propanol) were obtained from Sigma Chemical Co.

To extract α-tocopherol, 500 μL of serum were pipetted into a 16 × 125 mm glass tube, and 1.0 mL of ethanol was added to precipitate the serum proteins. The sample was then vigorously vortexed. A double-ether extraction procedure followed. Three milliliters of petroleum ether were added to the previously described tube, which was then vortexed and centrifuged (1,500 × g for 5 min). The petroleum ether layer was decanted into a separate 16 × 125 mm glass tube already in an ice bath. Three milliliters of petroleum ether was added to the tube with the precipitated serum, and then vortexed and centrifuged as before. The petroleum ether layer was removed from this tube and deposited into the second tube in an ice bath. The second tube with

Table 3. Antimicrobial treatment protocol[a]

Treatment	Severity score[b]	Rectal temperature	Antimicrobial treatment[c]
First	One or greater	40°C or higher	Micotil
	Returned to home pen for at least 48 h before next treatment		
Second	One or greater	40°C or higher	Nuflor
	Returned to home pen for at least 72 h before next treatment		
Third	One or greater	40°C or higher	Excenel
	Repeat therapy in 48 h; may be given a third dose if remains morbid		

[a]Antimicrobial pharmaceuticals were administered under the supervision of a licensed veterinarian at label doses.
[b]Subjective severity score: 0 = healthy; 1 = slightly morbid; 2 = moderately morbid; 3 = severely morbid; and 4 = fatally morbid.
[c]Micotil = tilmicosin phosphate (10 mg/kg of BW; Elanco Animal Health, Indianapolis, IN); Nuflor = florfenicol (27 μL/kg of BW; Schering-Plough Animal Health, Union, NJ); Excenel = ceftiofur sodium (0.34 mg/kg of BW; Pharmacia Animal Health, Kalamazoo, MI).

the two deposits of petroleum ether was evaporated to dryness under a N_2 stream in a water bath (35°C). The remaining residue was then dissolved with 1 mL of mobile phase solution (90% iso-octane, 9.5% tetrahydrofuran, and 0.5% acetic acid), and then stored in sealed vials at –10°C until analyzed by HPLC.

Antibody Response to Keyhole Limpet Hemocyanin

The purpose of this test was to detect and quantify the antibody response of stressed cattle to a foreign or novel antigen (keyhole limpet hemocyanin; **KLH**). One hundred eight randomly selected animals in two loads (Load 6, n = 52 and Load 7, n = 56; Table 1) were injected s.c. on d 0 with an emulsion containing KLH (0.5 mg) and 1.0 mL of Freund's incomplete adjuvant. Serum was collected as previously described, and antibody (IgG) response to KLH was determined by ELISA (Korver et al., 1984; Pollock et al., 1991). Briefly, 96-well plates were coated with 100 μL of KLH in a concentration of 10 μg/mL in PBS. Plates were then incubated at 37°C for at least 1 h, sealed with plastic and stored overnight at 4°C. After allowing plates to return to room temperature, they were washed three times with PBS/Tween (0.05% Tween 20). Optimal dilutions of sera (100 μL; 1:500) were placed in triplicate wells and incubated at 37°C for 2 h. Plates were again washed three times in PBS/Tween. A dilute conjugated secondary antibody (1:400 BSA anti-bovine IgG in PBS/Tween) was added (100 μL), and the plate was incubated as before. After a final wash (six times), 100 μL of a color substrate consisting of *o*-phenylenediamine and hydrogen peroxide was added. Wells were allowed to color for 5 min, after which a stop reagent (6.8 mL of H_2SO_4 in 500 mL of distilled H_20) was added (50 μL) to each well. Optical density (490 nm) was determined for each well in an automated plate reader (V Max Kinetic microplate reader, Molecular Devices, Sunnyvale, CA).

Carcass Data

After completion of the 42-d receiving period, cattle from five loads were transported to commercial feedlots in either the Oklahoma or Texas panhandle for finishing. The feedlot was selected by the cooperating producer/owner who retained ownership of the cattle since inception of the study. All cattle were fed commercial high-energy finishing diets for approximately 200 d. Upon reaching their final target weights, cattle were slaughtered at a commercial facility. Carcass data (HCW, quality grade, and yield grade) were collected on only two of the original seven loads of cattle. An estimate of final live weight before slaughter was calculated by dividing HCW by the average dressing percent. Estimated ADG in the feedlot was calculated using estimated final live weight, final live weight at our facility, and the known days on feed.

Calculations and Statistical Analyses

Medical costs were calculated by multiplying the actual amount of each respective antimicrobial drug used in each animal at each clinical incident by the appropriate unit cost of the drug. Drug costs used were as follows: Micotil = $0.93/mL, Nuflor = $0.39/mL, and Excenel = $0.50/mL.

Data were analyzed as a split-plot in a randomized complete block design, where loads were blocks, weight class was the whole plot factor, and dietary treatment was the subplot treatment factor. The error term used when testing weight class in the whole plot was the interaction of weight class and load. Residual error was used when response variables were tested for dietary treatments and interactions involving dietary treatments. Pen was used as the experimental unit, except for variables measured on individual animals (e.g., incidence of respiratory disease, drug treatment costs, estimated final live weight (feedlot), carcass characteristics, serum vitamin E, serum cholesterol, response to KLH). Serum vitamin E and cholesterol concentrations were analyzed as repeated measures over days; the model included fixed effects of vitamin E and days and the appropriate two-way interaction (Littell et al., 1998). The appropriate covariance structure was determined to be autoregressive (AR1). All models were ana-

lyzed using the Mixed procedures of SAS (SAS Inst. Inc., Cary, NC). Least squares means were compared using the LSD method protected by an ($P < 0.05$) *F*-value. Regression analyses were conducted using the PROC REG procedures of SAS, with carcass quality or yield grade as dependent variables and the number of antimicrobial drug treatments per calf as the independent variable. Nonparametric variables related to health (number of antimicrobial treatments) were analyzed with the Genmod procedure of SAS. Block, weight class, and treatment were included in the model statement, and a binomial distribution specified. Results are discussed as significant if $P \leq 0.05$ and as tendencies if $P = 0.06$ to 0.10.

Results and Discussion

Supplemental Vitamin E

Our experimental diet and supplement (B-171) were analyzed for α-tocopherol acetate to determine the actual amount of vitamin E supplied (J. Wilson, Roche Vitamins, Inc., personal communication). Results of these analyses indicated that concentrations of vitamin E were 56 to 87% (average = 69%) of that calculated for the diet, and 39 to 78% (average = 62%) for the supplement. Ideally, these results would have been 85% or greater of our calculated amount. One reason for lower than calculated vitamin E concentrations might be explained by the starch matrix of the supplement and total diet occluding the analytical procedure to the extent that the results were masked (J. Wilson, Roche Vitamins, Inc., personal communication). The problematic starch matrix might have been contributed by the wheat middlings (35% starch), which comprised more than 95% (DM basis) of the B-171 supplement and 15% (DM basis) of the diet (Table 2). Interestingly, a sample collected before pelleting was determined to be 84% of the calculated value. Although analysis before pelleting was performed on only one sample, this finding might suggest that vitamin E was lost or otherwise altered during the pelleting process, possibly due to heat damage, such that assay sensitivity was decreased.

Weight Class

With few exceptions, the whole-plot factor of weight class and the subplot interaction between weight class and days of vitamin E supplementation were not significant. Therefore, results for weight class and the weight class × days of vitamin E supplementation are neither reported nor discussed.

Animal Performance

Daily gain ($P = 0.56$) was not affected by increasing days of supplemental vitamin E (Table 4). The ADG and total gain averaged 0.98 ± 0.05 kg/d and 41.3 ± 2.3 kg across treatments, respectively. During the 42-d course of feeding, average DMI was not affected ($P = 0.31$) by dietary treatment; therefore, because gains were similar, no difference (average = 0.198 ± 0.140) was evident in G:F. From d 0 to 7, DMI was less ($P = 0.04$) by calves consuming Control and E7 than by calves consuming E14 and E28. During all other periods (d 8 to 14, d 15 to 28, and d 29 to 42), DMI was not affected ($P = 0.21$ to 0.86) by diet. Because calves fed E7, E14, and E28 consumed a similar amount of vitamin E from d 0 to 7, differences in intake were most likely not related to treatment. Expressed as a percentage of BW, typical DMI by lightweight, newly received cattle has been estimated to be 1.55% of BW for the first 7 d and 1.90% of BW for the first 14 d following arrival (Hutcheson and Cole, 1986). In the present experiment, DMI averaged 1.48% of initial BW from d 0 to 7, and 1.82% of initial BW from d 0 to 14.

To calculate vitamin E intake, we used the average analyzed dietary value (69% of calculated), which resulted in an average vitamin E intake of 1,259 IU·calf^{-1}·d^{-1} for treatment calves rather than our objective of 2,000 IU·calf^{-1}·d^{-1} (Table 4). Due to relatively low initial DMI and low recovery of dietary vitamin E, calves fed the control diet received less dietary vitamin E (20.3 IU·calf^{-1}·d^{-1} from d 0 through 7, and 90.3 IU·calf^{-1}·d^{-1} from d 8 through 14) than the NRC (1996) estimated minimum requirement for stressed calves (97.0 to 291.0 IU·calf^{-1}·d^{-1} from d 0 to 7, and 119.0 to 356.0 IU·calf^{-1}·d^{-1} from d 0 to 14). Throughout the entire receiving and starting period, the NRC (1996) recommends that between 400 and 500 IU of vitamin E·calf^{-1}·d^{-1} should be fed. During the 42-d receiving period in the present experiment, calves not receiving supplemental vitamin E (B-171 Supplement) consumed an average of 275.3 to 375.3 IU·calf^{-1}·d^{-1} less vitamin E than the recommended level, whereas calves receiving supplemental vitamin E consumed an average of 758.6 to 858.6 IU·calf^{-1}·d^{-1} more vitamin E than the recommended level.

Similar to our results, Rivera et al. (2002) found no effects of vitamin E supplementation on ADG or G:F of 173-kg steer and bull calves consuming 217, 380, or 787 IU/d of vitamin E, and found a linear decrease in ADG (d 14 to 28) and G:F in 204-kg heifers consuming 335, 640, or 1,148 IU/d of vitamin E for 28 d. In contrast, Gill et al. (1986) reported increased weight gain and gain efficiency among newly received stocker cattle under geographic and environmental conditions similar to those of the present experiment. One possible explanation for the observed difference might be related to diet. Gill et al. (1986) provided ad libitum access to prairie hay and fed 0.91 kg/d of a soybean meal-based pellet fortified with an additional 800 IU of vitamin E fed for 28 d. Daily gains in their study were lower (0.43 vs. 0.53 kg/d for control and vitamin E, respectively) than those observed in the present experiment. The diet fed in our experiment was designed to achieve a daily gain of 0.91 kg/d and was fed for greater DMI for a longer period of time (28 vs. 42 d). In the present experiment, the Control diet seemed to be adequate for

Table 4. Effect of days of supplementing vitamin E on animal performance and health during the 42-d receiving period

	Treatments[a]					
Item	CON	E7	E14	E28	SEM[b]	$P > F$
No. of pens	14	14	14	14	—	—
No. of animals	183	180	178	174	—	—
ADG, kg/d	0.95	0.99	0.98	1.00	0.05	0.56
Total gain, kg	40.0	41.8	41.3	42.2	2.3	0.56
G:F	0.20	0.20	0.19	0.20	0.14	0.87
DMI, kg·calf^{-1}·d^{-1}						
d 0 to 7	2.78^{x}	2.77^{x}	2.96^{y}	2.95^{y}	0.36	0.04
d 8 to 14	4.08	4.11	4.37	4.30	0.30	0.21
d 15 to 28	4.94	5.04	5.03	4.95	0.26	0.86
d 29 to 42	5.50	5.62	5.76	5.74	0.23	0.48
d 0 to 42	4.33	4.38	4.53	4.49	0.25	0.31
Vitamin E intake, IU·calf^{-1}·d^{-1}						
d 0 to 7	20.3^{x}	941.4^{y}	1,003.6^{y}	1,002.7^{y}	97.5	0.001
d 8 to 14	90.3^{x}	91.0^{x}	1,454.5^{y}	1,432.5^{y}	73.6	0.001
d 15 to 28	112.3^{x}	115.0^{x}	114.9^{x}	1,716.8^{y}	35.1	0.001
d 29 to 42	153.7	157.1	161.1	160.4	6.6	0.47
Morbidity, %	67.8	68.3	61.8	60.3	0.07	0.22
Medical costs, $/calf	6.29	5.67	5.18	4.88	0.70	0.08
Treatments/calf	0.92	0.84	0.78	0.76	0.80	0.14
First treatment, d	3	3	3	3	0.68	0.64
Second treatment, d	9	9	9	6	1.80	0.45
Third treatment, d	20	18	24	11	3.61	0.11
Med 0, %[c]	32.3	31.7	38.2	39.7	—	0.28
Med 1, %[c]	47.5	53.9	47.2	47.7	—	0.67
Med >1, %[c]	20.2	14.4	14.6	12.6	—	0.21

[a]Least squares means; CON = no supplemental (B-171 supplement) vitamin E; E7 = vitamin E fed from d 1 through 7; E14 = vitamin E fed from d 1 through 14; and E28 = vitamin E fed from d 1 through 28.
[b]Standard error of the least squares means; n = 14 for daily gain, total gain, and DMI:ADG for all treatments; n = 183, 180, 178, and 174 for CON, E7, E14, and E28, respectively, for all other variables.
[c]Percentage of calves receiving zero (Med 0), exactly one (Med 1), or more than one (Med >1) antibiotic treatment.
[x,y]Means within a row without a common superscript letter differ, $P < 0.05$.

maintaining performance, and no benefit was realized from feeding a supplement, which provided an average of 1,259 IU of vitamin E for 7, 14, or 28 d.

Animal Health

No difference in the percentage of calves exhibiting signs of morbidity was observed among days of supplemental vitamin E ($P = 0.22$; Table 4). Medical costs ($/calf) tended ($P = 0.08$) to be decreased by increasing days of vitamin E supplementation. From Control to E28, costs incurred due to respiratory disease were decreased by 28.9% or $1.41/calf treated. During the present experiment, the cost of providing 2,000 IU (as formulated) of vitamin E in the diet for 28 d was approximately $1.00/calf. Although the number of drug treatments per calf was similar ($P = 0.14$) among our dietary treatments, the numerical decrease resulted in a tendency toward decreased ($P = 0.08$) medical costs per calf. These results are in general agreement with those of Gill et al. (1986), who reported that the addition of vitamin E to the diets of newly arrived calves tended to decrease the incidence of observed sickness. A numerical decrease in the rate of calves retreated was also noted by Rivera et al. (2002) when calves were fed 1,400 vs. 285 IU/d of vitamin E. Consistently decreasing the costs associated with respiratory disease could potentially result in a significant annual savings to the cattle feeding industry.

The number of drug treatments per calf was similar ($P = 0.14$) among dietary treatments, but numerically decreased as days of vitamin E supplementation increased (Table 4). Lofgreen et al. (1975) observed in two experiments that slightly more than half and one-third of calves treated for respiratory disease were treated initially during the first week after arrival. In our study, on average, the first drug treatment was given on d 3 ($P = 0.64$), the second on d 8 ($P = 0.45$), and all subsequent drug treatments were given on or after d 18 with the exception of E28, which occurred on d 11 ($P = 0.11$; Table 4). Because both E14 and E28 calves were fed additional vitamin E during the first 14 d, reasons for this tendency are unclear.

The percentage of calves that received zero, one, or more than one drug treatment was evaluated in the present experiment (Table 4). Although approximately 40.3, 38.4, and 60.3% fewer calves fed E7, E14, and E28, respectively, received greater than one drug treatment during the 42-d receiving period compared with Control calves, these values were not different ($P = 0.21$).

Table 5. Results of laboratory analyses for serum concentrations of cholesterol and vitamin E

Item	Treatment[a] CON	E7	E14	E28	SEM	Treatment $P > F$	Time[b] $P > F$
Animal, No.	84	84	84	84	—	—	—
Cholesterol, mg/100 mL[c]							
d 0	130.35	129.48	118.09	128.09	3.51	0.96	
d 14	61.03	61.33	60.34	63.96	3.52	0.95	*
d 28	66.24	66.65	66.69	70.30	3.52	0.97	*
d 42	78.51	80.54	76.95	82.61	3.52	0.93	*
Vitamin E, μg/mL[d]							
d 0	5.72	4.95	5.05	5.22	0.76	0.45	
d 28	1.11[e]	1.20[e]	1.48[e]	3.38[f]	0.22	0.001	*
d 42	1.32	1.46	1.47	1.67	0.16	0.19	*

[a]Least squares means; CON = no supplemental (B-171 supplement) vitamin E; E7 = vitamin E fed from d 1 through 7; E14 = vitamin E fed from d 1 through 14; and E28 = vitamin E fed from d 1 through 28.
[b]Means within a column with an asterisk differ from values on d 0, $P < 0.001$.
[c]Treatment × day interaction, $P = 0.84$.
[d]Treatment × day interaction, $P < 0.001$.
[e,f]Means within a row without a common superscript letter differ, $P < 0.05$.

Garber et al. (1996) reported that performance was not changed, but immune response was increased when a high level of vitamin E was added to the diet of steers.

Serum Cholesterol and Vitamin E

Vitamin E is a fat-soluble vitamin and as with many other biological compounds, requires a lipid carrier for transport throughout the body. Lipoproteins are molecules with variable proportions of both lipid and protein (Lehninger, 1993). We hypothesized, based on previous physiological work by Han et al. (1999) studying the mechanisms of tocopherol metabolism, that the decrease in serum antioxidant concentrations (α-tocopherol) may coincide with, or even be precluded by, a decrease in serum lipid values when animals are exposed to stress and disease. Because cholesterol is a major component of lipoproteins, laboratory methods to measure total serum cholesterol have been used as a general indicator of serum lipid concentration (Ravel, 1984).

Results of serum cholesterol and vitamin E analyses are presented in Table 5. There was no serum cholesterol × days of vitamin E supplementation interaction ($P = 0.84$). Serum cholesterol concentrations were not affected by vitamin E supplementation ($P = 0.93$ to 0.97), but they were affected ($P < 0.001$) by days on feed. Serum cholesterol concentrations on d 14, 28, and 42 were less ($P < 0.001$) than on d 0. On average, calves had cholesterol values within the "normal" range (60 to 240 mg/100 mL) for juvenile bovine on d 0 (Jenkins et al., 1988); however, by d 14, some animals in all treatments had serum cholesterol concentrations below 60 mg/100 mL. Although cholesterol concentration numerically increased on d 28 and 42 compared with d 14, final concentration on d 42 averaged 59% less ($P < 0.001$) than the concentration on d 0. In a subsequent load of heifers (Choat et al., 2001), compared with no added fat, adding 2% fat to the vitamin E-supplemented diet resulted in a smaller decrease in serum cholesterol by d 14, and serum cholesterol returned to d 0 concentrations by d 28. Nonetheless, adding fat had no effect on serum vitamin E concentrations across the 42-d receiving study (Choat et al., 2001).

Serum concentrations of α-tocopherol in cattle vary with diet (Caravaggi, 1969; McMurray et al., 1983). Adams (1982) described various levels of serum α-tocopherol status in cattle as <2.0 μg/mL = deficient, 2.0 to 3.0 μg/mL = marginal, and 3.0 to 4.0 μg/mL = minimal, but adequate. These values agree with those reported by Gill et al. (2000) from a summary of previous experiments. In our experiment, the pattern of serum vitamin E disappearance was similar to that of serum cholesterol; however, a treatment × sampling day interaction ($P < 0.001$) was observed (Table 5). Serum α-tocopherol concentrations were similar for all treatments on d 0. Serum α-tocopherol concentrations were not determined on d 7 or 14; however, E28 calves had greater ($P < 0.001$) serum α-tocopherol concentration on d 28 than other treatment groups. By d 42, serum α-tocopherol concentration of calves on treatment E28 had decreased to values similar ($P = 0.19$) to control, E7, and E14. Considering these greater serum α-tocopherol concentrations for E28, the trend toward decreasing medical treatment costs, and the numeric decrease ($P = 0.22$) in morbidity for E14 and E28 calves, it seems that vitamin E may provide some protection against at least a portion of the detrimental effects of stress and disease, and that these effects might be time dependent. Further investigation of these physiological responses during stress and disease will be required to adequately explain these patterns of change.

IgG Antibody Response to KLH

The KLH procedure was used to determine whether the immune systems of calves used in this experiment

would respond to a foreign antigen; however, response due to dietary treatments was not evaluated. Serum antibody concentrations of calves vaccinated against KLH were greater ($P < 0.001$) on d 14 and 28 than on d 0. On d 14, calves not vaccinated against KLH averaged 0.37 mg/100 mL of IgG, whereas calves vaccinated against KLH averaged 2.13 mg/100 mL of IgG ($P < 0.001$). On d 28, calves not vaccinated against KLH averaged 0.40 mg of IgG/100 mL of serum, whereas KLH-vaccinated calves averaged 1.94 mg/100 mL ($P < 0.001$). These data strongly suggest that the immune systems of the calves in this experiment were fully responsive to a foreign antigen, and would therefore be considered functional. The ADG from d 0 to 14 by calves vaccinated with KLH tended ($P = 0.11$) to be less than those not vaccinated (1.4 vs. 1.7 kg/d); however, ADG during all other time periods compared (d 0 to 28, d 14 to 28, d 0 to 42, and d 14 to 42) did not differ between vaccinated and nonvaccinated calves.

Estimated Feedlot Performance and Carcass Characteristics

Carcass data were obtained from 185 heifers (data not shown). The estimated final live weight was similar (average = 490 ± 7 kg) among all dietary treatments in our study. No differences were observed among treatments for quality grade (51% of all carcasses graded Choice or greater). Among all carcasses graded, yield grade averaged 2.4 and was not affected by dietary treatment. Stovall (2000) observed a negative relationship between marbling score and medical treatments; marbling scores were less in calves treated for respiratory disease than in calves not treated. Carcass value was decreased by nearly $20/animal when heifers in their study received more than one drug treatment. Among our data, neither model (i.e., quality grade or yield grade vs. drug treatments) was significant, nor were any linear, quadratic, or cubic relationships detected. Lack of a relationship in the present experiment might be the result of the relatively few number of carcasses graded (n = 185) compared with the more than 700 health records reported and analyzed, or a result of the relatively small differences observed in morbidity.

In summary, supplemental vitamin E (approximately 1,259 IU·calf^{-1}·d^{-1}) had little influence on performance and overall health status of calves under our experimental conditions. Nonetheless, the greater serum α-tocopherol concentrations for E28, the trend toward decreasing medical treatment costs, and the numeric decrease in morbidity and treatments/calf for E14 and E28 calves support previous literature, and suggests that the positive effects of vitamin E on health status might be time-dependent. It seems reasonable to include vitamin E at or above the stated minimum requirement in the receiving diets of feedlot calves. When feeding stressed calves, regardless of which nutrient is being studied, adequate DMI is critical in the delivery and subsequent effectiveness of the diet, especially during the first 7 to 14 d.

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