Effects of dietary vitamin E concentration and source on sow, milk, and pig concentrations of α-tocopherol[1,2]

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ABSTRACT: A total of 126 gilts and sows (PIC 1050) and their litters were used to determine the effects of dietary vitamin E concentration and source on sow plasma, milk, and pig concentrations of α-tocopherol. Additionally, we estimated the bioavailability of D-α-tocopheryl acetate (D-α-TAc) relative to DL-α-tocopheryl acetate (DL-α-TAc) when fed in diets containing dried distillers grains with solubles (DDGS). The 6 dietary treatments included DL-α-TAc at 44 and 66 mg/kg and D-α-TAc at 11, 22, 33, and 44 mg/kg. From breeding to d 69 of gestation, sows were fed 2.0 kg/d of a diet containing 40% DDGS, 0.30 mg/kg added Se, and no added vitamin E. Vitamin E treatments were fed from d 70 of gestation through weaning. Plasma was collected from sows on d 69 and 100 of gestation, at farrowing, and at weaning. Colostrum and milk samples were also collected. Plasma from 3 pigs per litter and heart and liver samples from 1 pig per litter were collected at weaning. Plasma, milk, and tissues from 6 litters per treatment were analyzed for α-tocopherol. Although tissue, plasma, and milk concentrations of α-tocopherol were the primary response criteria of interest, sow and litter performance were measured. As expected, treatment effects were not observed for lactation feed intake, sow BW, or backfat measurements. A trend ($P = 0.085$) for a treatment effect on average pig BW at weaning was detected, with pigs nursing sows fed 44 mg/kg DL-α-TAc weighing less because of a younger weaning age. No other differences in litter performance were observed. As D-α-TAc increased in the diet, sow plasma, colostrum, and milk, pig plasma, and pig heart concentrations of α-tocopherol increased (linear, $P < 0.03$). Sows fed diets with 44 mg/kg D-α-TAc had increased ($P < 0.03$) plasma and colostrum and pig plasma concentrations of α-tocopherol compared with sows fed 44 mg/kg of DL-α-TAc. Sows fed 66 mg/kg DL-α-TAc also had greater ($P = 0.022$) plasma α-tocopherol at weaning than sows fed 44 mg/kg DL-α-TAc. Bioavailability coefficients for D-α-TAc relative to DL-α-TAc ranged from 1.9 to 4.2 for sow and pig plasma α-tocopherol, 2.9 to 3.6 for colostrum α-tocopherol, 1.6 for milk α-tocopherol, and 1.7 to 2.0 for pig heart and liver α-tocopherol. Overall, this study indicates the bioavailability for D-α-TAc relative to DL-α-TAc varies depending on the response criteria but is greater than the standard potency value of 1.36.

Key words: α-tocopherol, bioavailability, natural vitamin E, sow

J. Anim. Sci. 2014.92:4547–4556
doi:10.2527/jas2013-7311

INTRODUCTION

Vitamin E is a collective term referring to a group of 8 compounds (α-, β-, γ-, and δ-tocopherols and α-, β-, γ-, and δ-tocotrienols) that serve as antioxidants in plant and animal tissues. The α-tocopherol compound is the most bioactive form in the lipid component of animals (Traber, 2007). The eight stereoisomers of α-tocopherol have biological activities from 21 to 100% (Weiser et

[1]Contribution no. 14-115-J from the Kansas Agric. Exp. Stn., Manhattan 66506.
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Received October 25, 2013.
Accepted July 29, 2014.

al., 1996). Synthetic vitamin E (DL-α-tocopherol; all rac-α-tocopherol) is a combination of the 8 stereoisomers, whereas natural vitamin E (D-α-tocopherol; RRR-α-tocopherol) comprises only the RRR stereoisomer.

The United States Pharmacopeia (1980) states that 1 IU of vitamin E is equivalent to 1 mg of DL-α-tocopheryl acetate (**DL-α-TAc**) or 0.735 mg of D-α-tocopheryl acetate (**D-α-TAc**). The 1.36 biopotency estimate for D-α-TAc relative to DL-α-TAc is based on a pregnant rat model (Harris and Ludwig, 1948). The value has been extrapolated for use in other species; however, research suggests the bioavailability ratio for D-α-TAc relative to DL-α-TAc is greater than 1.36 in pigs (Mahan et al., 2000; Lauridsen et al., 2002; Yang et al., 2009).

Increasing colostrum α-tocopherol concentration is important because the newborn pig is vitamin E deficient and colostrum is the only source for suckling pigs (Lauridsen et al., 2002; Mahan, 1991). Diets that contain dried distillers grains with solubles (DDGS) have increased unsaturated fatty acid concentrations compared with corn-soybean meal-based swine diets (Kim et al., 2012; Stein and Shurson, 2009). Unsaturated fatty acids negatively impact the vitamin E status of pigs (Hidiroglou et al., 1993). Therefore, adding DDGS may negatively impact the sow's and her litter's vitamin E status. The objectives of this study were to determine the α-tocopherol concentration in plasma and milk and pig body tissues when sows were fed diets supplied with D-α-TAc or DL-α-TAc. We also estimated the bioavailability of D-α-TAc relative to DL-α-TAc when included in diets containing DDGS.

MATERIALS AND METHODS

The protocol used in this experiment was approved by the Kansas State University Institutional Animal Care and Use Committee. The study was conducted at the Kansas State University Swine Teaching and Research Facility in Manhattan, KS.

Animals and Diets

A total of 126 gilts and sows and their progeny (PIC 327 × 1050; PIC, Hendersonville, TN) were used. The 6 dietary treatments were DL-α-TAc at 44 and 66 mg/kg and D-α-TAc at 11, 22, 33, and 44 mg/kg. The 44 and 66 mg/kg of DL-α-TAc were selected as treatment levels in this experiment because they reflect the estimated requirement of the sow (NRC, 1998) and the standard dietary level that is commonly used in the industry for vitamin E (PIC, 2008), respectively. The 4 levels of D-α-TAc were selected to evaluate a range of bioavailability for each DL-α-TAc level. Treatments were allotted to sows in a generalized block design with farrowing group as the blocking factor and parity was balanced across treatments. Six farrowing groups were used and farrowed from November 2010 through May 2011.

Before beginning the experiment, all gilts and sows were fed diets containing 66 mg/kg DL-α-TAc. This level of vitamin E is an industry acceptable level for use in sow diets (PIC, 2008) and the standard inclusion for this particular farm. Beginning at breeding and continuing through d 69 of gestation, gilts and sows were fed 2.0 kg of a gestation diet containing no added vitamin E. On d 70 postcoitum, gilts and sows were randomly allotted to dietary treatments and remained on their dietary vitamin E concentration and source through the end of lactation. The gestation and lactation diets were formulated to contain 0.71 and 1.10% total Lys or 0.55 and 0.94% standardized ileal digestible Lys, respectively (Table 1). All diets were also formulated to include 0.30 mg/kg added Se from sodium selenite. All other nutrients were formulated to meet or exceed the NRC (1998) requirements. Gestation and lactation diets were also formulated to contain 40 and 20% DDGS, respectively. A sample of each lot of DDGS was analyzed for S content by inductively coupled plasma atomic emission spectroscopy (AOAC, 2000), and calcium sulfate was added to DDGS to maintain a constant S concentration of 0.80% in the DDGS. Dietary α-tocopherol analysis was also performed by Europhins Scientific Inc. (Des Moines, IA) using HPLC as outlined by AOAC International procedure 971.30 (AOAC, 2000). For the first 3 d after farrowing, sows were gradually stepped up on feed, and after d 3, all sows were allowed ad libitum access to the lactation diet. Temperature in the farrowing facility was maintained at a minimum of 20°C, and supplemental heat was provided to litters through the use of heat lamps.

Data Collection and Analysis

Although they were not the primary response criteria for the experiment, sow BW and backfat thickness measurements were recorded at breeding, d 69 of gestation, postfarrowing, and at weaning. Individual pig BW, litter size, and total litter weight were recorded at birth, d 3 of lactation, d 17 of lactation, and weaning. Lactation feed intake was measured. The primary response criteria for this trial included plasma, pig tissue, and milk α-tocopherol concentrations. Blood was collected from sows via jugular venapuncture on d 69 and 100 of gestation approximately 4 h after feeding. All blood samples were collected in tubes containing sodium heparin as an anticoagulant. All blood samples were stored on ice for approximately 1 h and then centrifuged at 1,600 × *g* for 20 min at 4°C in order to isolate the plasma fraction for α-tocopherol analysis. Milk and sow blood samples were also collected at 8 to 12 h postfarrowing and at weaning.

Table 1. Composition of diets (as-fed basis)

Feed ingredient	Gestation	Lactation
Corn	51.98	51.96
Soybean meal (46.5% CP)	4.15	24.24
DDGS[1]	40.00	20.00
Monocalcium P (21% P)	0.70	1.00
Limestone	1.75	1.45
Salt	0.50	0.50
Vitamin premix[2]	0.25	0.25
Trace mineral premix[3]	0.15	0.15
L-Lys HCl	0.18	0.10
Phytase[4]	0.10	0.10
Vitamin E premix[5]	0.25	0.25
Total	100	100
Calculated analysis		
ME, kcal/kg	3,302	3,293
CP, %	17.4	21.2
Total Lys, %	0.71	1.10
SID[6] AA, %		
Lys	0.55	0.94
Thr	0.49	0.66
Met	0.28	0.32
Trp	0.11	0.20
Ile	0.51	0.74
Leu	1.67	1.79
Val	0.66	0.86
Ca, %	0.84	0.84
P, %	0.61	0.66
Available P, %[7]	0.50	0.49

[1]DDGS = dried distillers grains with solubles.

[2]Vitamin premix provided per kilogram of diet: 11,022 IU of vitamin A, 1,378 IU of vitamin D_3, 4.41 mg of vitamin K, 0.04 mg of vitamin B_{12}, 49.60 mg of niacin, 27.56 mg of pantothenic acid, 8.26 mg of riboflavin, 0.22 g of biotin, 1.65 mg of folic acid, 4.96 mg of pyridoxine, 551.14 mg of choline, 49.6 mg of carnitine, and no vitamin E.

[3]Trace mineral premix provided per kilogram of diet: 16.5 mg Cu from $CuSO_4$, 200 µg of Cr from $Cr(C_6H_4NO_2)_3$,0.30 mg I from $Ca(IO_3)_2$, 165 mg Fe from $FeSO_4$, 40 mg Mn from $MnSO_4$, 0.305 mg Se from $NaSeO_3$, and 165 mg Zn from $ZnSO_4$.

[4]Phyzyme 600 (Danisco Animal Nutrition, St. Louis, MO) provided 600 phytase units/kg of diet.

[5]Vitamin E premixes were generated for each treatment by combining appropriate amounts of DL-α-tocopheryl acetate or D-α-tocopheryl acetate and rice hulls. For the depletion diet used in gestation, the vitamin E premix was replaced with corn starch.

[6]SID = standardized ileal digestible.

[7]Phytase provided 0.11% available P to the gestation and lactation diets.

Milk samples were obtained after an intravenous injection of oxytocin, and milk was collected with a minimum of 15 mL from each functional gland. At weaning, plasma was taken from the vena cava of 3 pigs per litter, and 1 pig per litter was stunned and killed to obtain heart and liver samples. Each heart and liver was immediately flash-frozen in liquid N to limit any oxidation in the tissue. Milk, blood, and tissue samples were kept at –80°C from the time of collection until the completion of the live animal portion of the study. After all samples were collected, α-tocopherol concentrations were determined for all samples at the same time.

From each farrowing group, 1 sow and litter per dietary treatment were used for plasma, milk, and pig tissue analysis of α-tocopherol, and females from the same parity were selected for each dietary treatment within each farrowing group. In addition, litters with similar lactation lengths, number of piglets, litter weights, and sow lactation feed intakes across the 6 dietary treatments were selected for α-tocopherol analysis to limit any potential bias. Tissue samples were prepared for analysis as outlined by Willburn et al. (2008), and samples were analyzed for α-tocopherol by HPLC using the procedures of Zaspel and Csallany (1983). The samples were sent on dry ice overnight and were analyzed at The Ohio State University, Columbus, OH.

Statistical Analysis

Experimental data were analyzed initially using PROC MIXED in SAS (SAS Inst. Inc., Cary, NC). Overall treatment significance was first determined by the overall treatment *F*-test. Contrast statements also were used to test for linear and quadratic effects associated with increasing D-α-TAc and to compare the 44 mg/kg DL-α-TAc treatment separately to both the 44 mg/kg D-α-TAc and 66 mg/kg DL-α-TAc treatments. Farrowing group was used as a random effect, and sow or litter was used as the experimental unit for all data analysis. For sow performance, interactions between dietary treatments and farrowing group were found to be nonsignificant and were therefore pooled with the error variance component for each response. For sow plasma, d-69 plasma α-tocopherol was used as a covariate. Statistics were considered significant at P-values < 0.05 and tendencies at P-values < 0.10.

Coefficients for the bioavailability of D-α-TAc relative to DL-α-TAc were also calculated based on both the formulated and analyzed concentrations of both DL-α-TAc treatments. The estimation of added α-tocopherol based on analyzed concentration was calculated by subtracting an estimate for the amount of indigenous α-tocopherol from the analyzed concentration for each dietary treatment. The estimated amounts of indigenous α-tocopherol were 11.4 and 8.9 mg/kg for gestation and lactation, respectively. Analyzed dietary concentrations of α-tocopherol in the gestation diets were used to calculate bioavailability estimates based on α-tocopherol concentrations in sow plasma on d 100 of gestation, sow plasma at farrowing, and colostrum. Analyzed dietary concentrations of α-tocopherol in the lactation diets were used to calculate bioavailability estimates based on α-tocopherol concentrations in sow plasma at weaning, sow milk, and pig tissues. To calculate estimates of bioavailability, linear

regression was first conducted with PROC REG in SAS to relate the analyzed plasma, milk, and tissue concentrations of α-tocopherol to the dietary concentrations of added D-α-TAc. Based on the regression line, the D-α-TAc dietary concentration needed to achieve the same tissue concentration of α-tocopherol as each of the DL-α-TAc treatments was calculated. The ratio of the each dietary DL-α-TAc relative to the calculated D-α-TAc was used to estimate the relative bioavailability.

RESULTS

The analyzed concentrations of α-tocopherol in each treatment's gestation and lactation diet are shown in Table 2. The analyzed α-tocopherol values were similar to the expected values with the exception of the lactation diet with 66 mg/kg DL-α-TAc, gestation diet with 44 mg/kg D-α-TAc, and lactation diet with 44 mg/kg D-α-TAc, which, when analyzed, were lower than expected.

No differences were observed in sow BW or backfat thickness measurements at any of the time points (Table 3). Also, no differences were observed in total or daily lactation feed intake. No litter size, average weight, or total litter weight differences were observed for total born, live born, d 3 of lactation, or d 17 of lactation (Table 4). A trend was observed ($P = 0.085$) for a difference in average pig BW at weaning, primarily due to the numerically lower average pig BW for sows fed the diet containing 44 mg/kg DL-α-TAc compared with other concentrations or sources of vitamin E. This lower average weight may be a function of the difference ($P = 0.044$) in weaning age, in addition to a numerically lower average pig birth weight for sows on that particular treatment, which was unexpected.

Sow plasma α-tocopherol was similar ($P = 0.724$) on d 69 when all sows were fed diets containing no added vitamin E, but it increased (linear, $P < 0.01$) with increasing D-α-TAc on d 100 of gestation, postfarrowing, and at weaning (Table 5). Sow plasma α-tocopherol was greater ($P < 0.01$) for sows fed 44 mg/kg D-α-TAc compared with those of sows fed either of the 2 DL-α-TAc concentrations at each time point. Sow plasma α-tocopherol also increased ($P = 0.022$) at weaning with increased dietary concentrations of DL-α-TAc. The calculated bioavailability estimates for sow plasma α-tocopherol concentrations on d 100 of gestation were 2.1 and 2.4 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on the formulated concentrations of added DL-α-TAc and D-α-TAc (Table 6). Plasma α-tocopherol postfarrowing yielded bioavailability estimates of 4.2 and 3.0 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on the formulated concentrations of added DL-α-TAc and D-α-TAc. Estimates of bioavailability based on sow plasma α-tocopherol at weaning were 2.7 and 2.4 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on the formulated concentrations of added DL-α-TAc and D-α-TAc. Estimates of bioavailability based on sow plasma α-tocopherol in relation to analyzed dietary α-tocopherol were 1.9 to 3.9.

Table 2. Dietary concentration of α-tocopherol, mg/kg[1]

	Source of vitamin E					
	DL-α-TAc[2]		D-α-TAc[3]			
	Formulated added vitamin E mg/kg					
Item	44	66	11	22	33	44
Analyzed concentrations of α-tocopherol						
Gestation diet	54.4	85.7	23.0	33.4	46.0	45.7
Lactation diet	54.9	66.3	23.0	33.2	47.6	48.4
Estimation of added α-tocopherol based on analyzed concentration[4]						
Gestation diet	43.0	74.3	11.6	22.0	34.6	34.3
Lactation diet	46.0	57.4	14.1	24.3	38.7	39.5

[1]Samples were collected from each batch of feed manufactured. From the samples, a composite for each dietary treatment and phase (gestation and lactation) was used for analysis of α-tocopherol.

[2]DL-α-TAc = DL-α-tocopheryl acetate.

[3]D-α-TAc = D-α-tocopheryl acetate.

[4]The estimation of added α-tocopherol based on analyzed concentration was calculated by subtracting an estimate for the amount of indigenous α-tocopherol from the analyzed concentration for each dietary treatment. The estimated amounts of indigenous α-tocopherol were 11.4 and 8.9 mg/kg for gestation and lactation, respectively.

Sow colostrum and milk α-tocopherol increased (linear, $P < 0.03$) with increasing dietary D-α-TAc (Table 5). Sows fed 44 mg/kg D-α-TAc had greater ($P = 0.003$) colostrum α-tocopherol than sows fed 44 mg/kg DL-α-TAc. A numerical increase in colostrum α-tocopherol was also observed as DL-α-TAc increased in the sow's diet, but the increase was not significant ($P = 0.456$). The calculated bioavailability estimates based on colostrum α-tocopherol were 3.0 and 2.9 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on formulated concentrations of added DL-α-TAc and D-α-TAc (Table 6). The estimated bioavailability for colostrum α-tocopherol was 3.6 for both DL-α-TAc treatments when using the analyzed dietary α-tocopherol concentrations. The estimates for bioavailability based on milk α-tocopherol were 1.6 and 7.3 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on formulated concentrations of added DL-α-TAc and D-α-TAc. The estimated bioavailability for milk α-tocopherol was 1.6 and 4.0 for the 44 and 66 mg/kg DL-α-TAc treatments, respectively, based on analyzed dietary concentrations of α-tocopherol.

Heart and plasma α-tocopherol in the suckling pigs increased (linear, $P < 0.01$) as the D-α-TAc increased in the sow's diet and tended to increase (linear, $P = 0.089$) in the pig's liver (Table 5). Pigs from sows fed 44 mg/kg D-α-TAc had greater ($P < 0.05$) plasma α-tocopherol compared with pigs from sows fed 44 mg/kg DL-α-TAc. Similar to sow's milk, a numerical decrease in plasma,

Table 3. Effects of vitamin E concentration and source on sow backfat, sow BW, and lactation feed intake[1]

	Source of vitamin E									
	DL-α-TAc[2]		D-α-TAc[3]					Significance level, *P* =		
	Added vitamin E, mg/kg								D-α-TAc	
Item	44	66	11	22	33	44	SEM	Treatment	Linear	Quadratic
n	21	21	21	21	21	21				
Backfat measurements, mm[4]										
Breeding[5]	15.7	16.0	16.0	16.0	15.6	15.9	0.70	0.997	0.839	0.794
d 69 of gestation[6]	16.2	16.4	16.2	16.0	15.9	16.3	0.76	0.998	0.952	0.717
Postfarrowing	15.8	15.7	15.6	15.9	15.7	16.0	0.59	0.995	0.682	0.933
Weaning	12.5	12.3	12.4	12.0	11.9	13.0	0.57	0.778	0.466	0.194
Sow BW, kg										
Breeding[5]	187	181	192	189	189	192	7.6	0.862	0.956	0.692
d 69 of gestation[6]	207	203	209	206	209	212	7.2	0.933	0.703	0.562
Postfarrowing	213	208	218	213	214	216	6.7	0.886	0.860	0.562
Weaning	206	202	210	205	204	209	6.8	0.934	0.867	0.358
Daily lactation feed intake, kg										
d 0 to 17	6.09	5.98	5.92	5.89	5.78	5.90	0.335	0.978	0.881	0.779
d 0 to weaning	6.20	6.11	6.05	6.01	5.89	6.12	0.336	0.975	0.926	0.611

[1]A total of 126 sows and litters were used over 6 farrowing groups to determine the effects of supplemental vitamin E concentration and source on sow, milk, and pig concentrations of α-tocopherol.

[2]DL-α-TAc = DL-α-tocopheryl acetate.

[3]D-α-TAc = D-α-tocopheryl acetate.

[4]Backfat measurements were determined by averaging both sides at the P2 position (last rib and approximately 65 mm off the midline).

[5]From breeding until d 70 of gestation, all sows were fed a deficient diet containing no supplemental vitamin E.

[6]On d 70, sows were allotted to treatment diets; sows remained on the same vitamin E concentration throughout the remainder of gestation as well as lactation.

Table 4. Effects of vitamin E concentration and source on sow lactation performance[1]

	Source of vitamin E									
	DL-α-TAc[2]		D-α-TAc[3]					Significance level, *P* =		
	Added vitamin E, mg/kg								D-α-TAc	
Item	44	66	11	22	33	44	SEM	Treatment	Linear	Quadratic
n	21	21	21	21	21	21				
Litter size, *n*										
Total born	14.1	13.2	12.6	12.6	13.2	14.0	0.82	0.645	0.172	0.601
Live born	13.7	13.0	12.0	12.0	12.8	13.2	0.81	0.652	0.235	0.793
d 3	11.7	11.8	11.4	11.4	12.0	11.9	0.35	0.487	0.088	0.747
d 17	11.5	11.3	11.0	10.9	11.3	11.1	0.34	0.748	0.564	0.781
Weaning	11.5	11.3	11.0	10.8	11.3	11.1	0.34	0.651	0.460	0.913
Total litter wt, kg										
Total born	18.6	19.5	17.1	17.5	17.9	19.0	0.98	0.464	0.152	0.728
Live born	18.3	19.2	16.5	17.0	17.5	18.2	0.97	0.428	0.205	0.896
d 3	20.7	22.6	20.9	21.1	21.5	21.4	0.78	0.540	0.561	0.801
d 17	55.8	58.7	57.3	57.4	56.7	58.4	2.40	0.953	0.800	0.719
Weaning	60.1	66.2	62.6	62.6	64.9	65.6	2.62	0.523	0.326	0.883
Pig wt, kg										
Total born	1.33	1.52	1.41	1.45	1.39	1.40	0.053	0.170	0.690	0.781
Live born	1.34	1.53	1.44	1.47	1.40	1.42	0.053	0.230	0.632	0.927
d 3	1.76	1.91	1.84	1.85	1.79	1.79	0.056	0.371	0.397	0.954
d 17	4.87	5.19	5.29	5.27	4.99	5.26	0.189	0.302	0.611	0.348
Weaning	5.24	5.87	5.75	5.81	5.72	5.89	0.208	0.085	0.671	0.751
Lactation length, d	19.1	20.0	19.2	19.5	20.0	20.2	0.31	0.044	0.010	0.816

[1]A total of 126 sows and litters were used over 6 farrowing groups to determine the effects of supplemental vitamin E concentration and source on sow, milk, and pig concentrations of α-tocopherol.

[2]DL-α-TAc = DL-α-tocopheryl acetate.

[3]D-α-TAc = D-α-tocopheryl acetate.

Table 5. Effects of vitamin E concentration and source on sow plasma, milk, and pig tissue concentrations of α-tocopherol[1]

	Source of vitamin E											
	DL-α-TAc[2]		D-α-TAc[3]					Significance level, P =				
	Added vitamin E, mg/kg								D-α-TAc		44 DL-α-TAc vs.	
Item	44	66	11	22	33	44	SEM	Treatment	Linear	Quadratic	44 D-α-TAc	66 DL-α-TAc
No. of samples	6	6	6	6	6	6						
Tissue concentrations of α-tocopherol, μg/mL												
Sow plasma												
d 69 of gestation[4]	1.00	0.85	0.89	0.89	0.95	0.98	0.082	0.724	0.383	0.849	0.828	0.177
d 100 of gestation[5]	1.32	1.51	1.09	1.28	1.64	1.99	0.187	0.003	0.001	0.556	0.003	0.372
Postfarrowing[5,6]	0.72	0.87	0.75	0.86	1.01	1.19	0.120	0.018	0.003	0.717	0.002	0.289
Weaning[4]	1.41	1.88	1.15	1.75	2.02	2.53	0.139	0.001	0.001	0.739	0.001	0.022
Sow colostrum[6]	8.19	10.31	7.62	11.39	9.40	17.76	2.165	0.017	0.004	0.258	0.003	0.456
Sow milk[7]	3.25	2.51	2.36	3.22	3.75	3.63	0.458	0.145	0.030	0.260	0.524	0.232
Pig[7]												
Plasma	2.47	2.38	2.11	3.03	3.51	3.78	0.376	0.024	0.004	0.395	0.022	0.861
Heart	4.84	3.93	3.60	4.75	5.93	6.00	0.619	0.014	0.002	0.301	0.128	0.225
Liver	4.18	3.39	2.99	4.88	4.96	5.12	1.063	0.339	0.089	0.301	0.423	0.499

[1]A total of 126 sows and litters were used over 6 farrowing groups to determine the effects of supplemental vitamin E concentration and source on sow, milk, and pig concentrations of α-tocopherol.

[2]DL-α-TAc = DL-α-tocopheryl acetate (all-rac-α-tocopheryl acetate).

[3]D-α-TAc = D-α-tocopheryl acetate (RRR-α-tocopheryl acetate).

[4]Before beginning dietary treatments.

[5]Adjusted with d 69 as a covariate.

[6]Collected 8 to 12 h after the completion of farrowing.

[7]Collected at the time of weaning.

heart, and liver α-tocopherol was observed as DL-α-TAc increased in the sow's diet; however, the differences were not significant. The estimated bioavailability from the suckling pigs with 44 and 66 mg/kg DL-α-TAc concentrations were 3.0 and 5.1 for plasma, 1.8 and 5.3 for heart, and 2.0 and 7.5 for liver, respectively, based on formulated concentrations of added DL-α-TAc and D-α-TAc (Table 6). Estimated bioavailability based on analyzed dietary concentration α-tocopherol for the suckling pigs were 2.0 and 3.4 for plasma, 1.7 and 3.4 for heart, and 1.9 and 4.2 for liver based on the 44 and 66 mg/kg DL-α-TAc concentrations, respectively.

DISCUSSION

This experiment demonstrated no differences in sow or litter performance associated with varying concentrations of either D-α-TAc or DL-α-TAc. The main objective of the experiment was to determine the relative efficacy of the 2 sources of vitamin E on various biological parameters. Mahan et al. (2000) compared supplementing sow diets with 30 or 60 IU/kg of either D-α-TAc or DL-α-TAc and also observed no differences in lactation litter performance over 5 parities, so the lack of differences in our experiment were not unexpected. Several other studies have indicated that 30 to 60 IU/kg of added vitamin E is sufficient to optimize reproductive performance (Mahan, 1991, 1994; Nielsen et al., 1979).

The diets used in this experiment contained DDGS. Adding DDGS to swine diets results in increased amounts of dietary unsaturated and polyunsaturated fatty acids (Stein and Shurson, 2009). Increasing the amount of dietary PUFA in sow diets has been shown to lower the pig's vitamin E status (Hidiroglou et al., 1993). The PUFA found in DDGS are susceptible to lipid peroxidation (NRC, 1998). The amount of peroxidation is related to duration and amount of heat to which PUFA are exposed during the drying process; therefore, the amount of oxidized lipids in DDGS can vary among ethanol plants as well as individual production lots.

Adding DDGS to swine diets also increases the dietary S concentration. The S content in DDGS is greater than other ingredients due to the addition of sulfuric acid during the ethanol production process and corn protein's greater proportion of the sulfur-containing AA compared with soy protein (Kim et al., 2012). Sulfur is a component in many bioactive compounds, such as Met, Cys, and glutathione, which all have antioxidant properties (Atmaca, 2004). Sulfur also has been shown to compete with Se for absorption in ruminants (Spears, 2003). Therefore, feeding elevated sulfur amounts may have both positive and negative effects on the oxidative stress on the animal.

Table 6. Bioavailability estimates based on tissue concentrations of α-tocopherol[1]

	Calculated bioavailability of D-α-TAc[2] relative to DL-α-TAc[3]			
	Formulated synthetic vitamin E, mg/kg			
	44		66	
	Based on dietary concentrations			
Item	Formulated	Analyzed[4]	Formulated	Analyzed[4]
Sow plasma				
d 100 of gestation	2.1	2.1	2.4	2.9
Postfarrowing	4.2	3.9	3.0	3.6
Weaning	2.7	2.4	2.4	1.9
Sow colostrums	3.0	3.6	2.9	3.6
Sow milk	1.6	1.6	7.3	4.0
Piglets				
Plasma	3.0	2.5	5.1	3.4
Heart	1.8	1.7	5.3	3.4
Liver	2.0	1.9	7.5	4.2

[1]A total of 126 sows and litters were used over 6 farrowing groups to determine the effects of supplemental vitamin E concentration and source on sow, milk, and piglet concentrations of α-tocopherol.

[2]DL-α-TAc = DL-α-tocopheryl acetate (all-rac-α-tocopheryl acetate).

[3]D-α-TAc = D-α-tocopheryl acetate (RRR-α-tocopheryl acetate).

[4]Analyzed dietary concentrations of α-tocopherol for gestation diets were used to calculate bioavailability estimates based on α-tocopherol concentrations in sow plasma on d 100 of gestation, sow plasma at farrowing, and colostrum. Analyzed dietary concentrations of α-tocopherol for lactation diets were used to calculate bioavailability estimates based on α-tocopherol concentrations in sow plasma at weaning, sow milk, and piglet concentrations.

Each incoming load of DDGS was analyzed for S content, and calcium sulfate was added to achieve a constant S level of 0.80% to standardize any impacts of S in our experiment. The similar bioavailability estimates from our experiment compared with other studies that did not use DDGS (Lauridsen et al., 2002; Mahan et al., 2000) suggests that dietary DDGS inclusions did not alter the bioavailability of D-α-TAc relative to DL-α-TAc.

Hanson et al. (2013) demonstrated that feeding sows a diet containing DDGS with a high concentration of oxidized lipids resulted in a decreased concentration of α-tocopherol in the serum of weaned pigs compared with pigs nursing sows fed a diet with no DDGS and a lower concentration of oxidized lipids. In contrast, Song et al. (2013) indicated that feeding DDGS with a high concentration of oxidized lipids to pigs for 8 wk postweaning increased the α-tocopherol concentration in serum compared with pigs fed diets without DDGS and a decreased concentrations of oxidized lipids. The authors suggest that this observation is a result of a sparing effect of α-tocopherol by the elevated S-containing antioxidants associated with DDGS supplementation in the diet. One possible theory that combines the results of these 2 studies is that feeding sows diets with DDGS containing oxidized fatty acids will be more detrimental to the α-tocopherol status of the nursing pigs than to the sow, possibly due to the elevated S-containing antioxidants' ability to spare α-tocopherol in the sow but not transfer through the milk to the nursing pig. Additional research is needed to validate this hypothesis.

All sows were fed a non-vitamin-E-fortified diet for the first 69 d of the experiment. This model was used to reduce the sows' storage capacity of vitamin E and appeared effective because plasma concentrations of α-tocopherol increased across dietary treatments from d 69 of gestation to d 100. A similar model was used by Yang et al. (2009) for finishing pigs, in which increasing amounts of D-α-TAc resulted in increases in plasma concentrations of α-tocopherol. Although both the gestation and lactation diets containing the 44 mg/kg level of added D-α-TAc were, when analyzed, approximately 5 to 10 mg/kg less than expected, the numerical increase in plasma α-tocopherol associated with that particular treatment suggests that the lower analyzed levels may have been due to analytical variation. Increasing the amount of DL-α-TAc resulted in increased sow plasma concentrations of α-tocopherol at weaning. Similar increases in sow plasma α-tocopherol have been observed with increasing D-α-TAc or DL-α-TAc (Mahan, 1991; Mahan et al., 2000). The estimates of bioavailability based on plasma concentrations of α-tocopherol were 1.9 to 4.2, with similar estimates calculated using formulated and analyzed dietary levels of α-tocopherol. Lauridsen et al. (2002) administered deuterated labeled forms of D-α-TAc and DL-α-TAc to compare the bioavailability of the 2 sources by supplementing both simultaneously. Based on ratios of the isotopes in plasma, they estimated the bioequivalence of D-α-TAc to be 2 times that of DL-α-TAc, which is similar to our conclusions. Mahan et al. (2000) also demonstrated that when sow diets contained similar vitamin E concentrations from the 2 sources on an IU basis, D-α-TAc increased plasma α-tocopherol concentrations compared with DL-α-TAc.

The newborn pig is vitamin E–deficient, and colostrum is the only source of α-tocopherol for suckling pigs (Lauridsen et al., 2002; Mahan, 1991). In the present study, increasing the amount of added D-α-TAc resulted in increased α-tocopherol concentrations in both colostrum and milk. Mahan et al. (2000) demonstrated increased α-tocopherol concentrations for colostrum and milk when increasing concentrations of DL-α-TAc or D-α-TAc were provided. They also indicated that when diets contained similar concentrations of vitamin E on an IU basis, D-α-TAc increased in the colostrum and milk concentrations of α-tocopherol compared with DL-α-TAc. In our experiment, milk α-tocopherol was numerically lower for the 66 mg/kg DL-α-TAc treatment than for the 44 mg/kg treatment. This response was also observed for α-tocopherol concentrations in the plasma, liver, and hearts of suckling pigs. One explanation for

the decrease in milk and pig α-tocopherol concentrations is that sows consuming the diet containing 66 mg/kg of added DL-α-TAc did not transfer greater quantities of α-tocopherol into the milk as compared to sows consuming the diet with 44 mg/kg of added DL-α-TAc despite consuming a greater daily amount of DL-α-TAc and having greater plasma α-tocopherol concentrations at weaning. While numerically lower, the differences in milk and pig α-tocopherol concentrations were not significantly different between the DL-α-TAc treatments and could simply have been due to sampling variation. Another possible explanation for this observation might be related to the 66 mg/kg of added DL-α-TAc lactation diet, which, when analyzed, had approximately 10 mg/kg of α-tocopherol below the expected level; however, it still analyzed with a greater concentration of α-tocopherol than the 44 mg/kg of added DL-α-TAc treatment. As a result of this observation, the bioavailability estimate for milk is based on the 44 mg/kg DL-α-TAc treatment; therefore, the estimated bioavailability for D-α-TAc relative to DL-α-TAc was 3.0 for colostrum α-tocopherol and 1.6 for milk α-tocopherol based on formulated dietary α-tocopherol concentrations. Similar bioavailability estimates were calculated when using analyzed dietary α-tocopherol concentration. The estimated bioavailability based on milk α-tocopherol concentrations was similar to the estimate of 1.54 by Mahan et al. (2000). Lauridsen et al. (2002) showed a 2:1 ratio for D-α-TAc relative to DL-α-TAc based on milk concentrations of labeled vitamin E sources.

It is important that the concentrations of α-tocopherol increase in pigs while they are nursing the sow because the neonate pig is born vitamin E deficient (Mahan, 1991) and plasma α-tocopherol drops postweaning (Lauridsen and Jensen, 2005). The plasma α-tocopherol concentrations observed from all treatments in this study would be above the threshold of 0.4 to 1.0 μg/mL, which separates deficiency from sufficiency (Jensen et al., 1988; Van Vleet, 1980). Concentrations of α-tocopherol in pig plasma, livers, and hearts followed patterns similar to milk α-tocopherol concentrations. Increasing D-α-TAc resulted in increased α-tocopherol in pig plasma, hearts, and livers. Mahan et al. (2000) demonstrated increased α-tocopherol concentrations in pig livers and plasma when increasing the amount of D-α-TAc or DL-α-TAc. Similar to milk, the bioavailability estimates are based on the 44 mg/kg DL-α-TAc treatment, so the estimated bioavailability for D-α-TAc relative to DL-α-TAc was 3.0 for pig plasma α-tocopherol, 1.8 for heart α-tocopherol, and 2.0 for liver α-tocopherol based on formulated concentrations of added D-α-TAc and DL-α-TAc; similar bioavailabilities were estimated using analyzed dietary α-tocopherol concentrations.

Previous research has established that the biological activity of D-α-TAc and DL-α-TAc differ due to the differences in conformation of the stereoisomers in DL-α-TAc (Blatt et al., 2004). The potency estimate of 1.36 used in conversion of IU (United States Pharmacopeia, 1980) was based on data using a rat fetal absorption model (Harris and Ludwig, 1948). That potency value has been extrapolated for use in other livestock species as well as in humans. Recent findings related to the metabolism and transport of α-tocopherol stereoisomers suggests that the bioavailability is different in pigs and humans, particularly when dietary vitamin E concentrations are fed at the requirement. In addition, the affinity of different tissues can result in different bioavailability estimates. Understanding the metabolism associated with α-tocopherol may help explain differences from the original potency value.

The hepatic α-tocopherol transfer protein (**α-TTP**) is responsible for regulating plasma concentrations of α-tocopherol. The α-TTP–α-tocopherol complex will move α-tocopherol to the plasma membrane and release the α-tocopherol so it can be taken up by very-low-density lipoproteins and released into the blood stream (Horiguchi et al., 2003). Hepatic α-TTP has varying affinities for different forms of vitamin E; RRR-α-tocopherol is 100%, SRR-α-tocopherol is 10.5%, and other tocopherols vary from 1.5 to 38% (Hosomi et al., 1997). This suggests that the α-TTP preferentially transports stereoisomers of α-tocopherol with the 2-R conformation in comparison to the 2-S conformation (Leonard et al., 2002; Traber et al., 1990). In addition to hepatic tissue, the α-TTP also has been detected in the pregnant mouse uterus (Jishage et al., 2001). In the rat fetal absorption model, rats were depleted of vitamin E for an extended period before beginning test diets (Harris and Ludwig, 1948). Due to the depletion of vitamin E in rats, the α-TTP may have transported greater quantities of the 2-S stereoisomers of α-tocopherol to the uterus than if the rats were not in a deficient state.

Studies have also indicated differences in elimination of α-tocopherol with D-α-TAc compared with DL-α-TAc. Traber et al. (1998) dosed 150 mg of deuterated D-α-tocopherol (**D-α-T**) and DL-α-tocopherol **(DL-α-T)** and showed that plasma contained twice as much α-tocopherol from D-α-T as DL-α-T; however, the urinary metabolite 2,5,7,8-tetramethyl (2′-carboxyethyl)-6-hydroxychroman (**α-CEHC**) from DL-α-T was 3 to 4 times greater than α-CEHC from D-α-T. Clifford et al. (2006) also performed a crossover study in humans and demonstrated that the degradation and elimination of α-tocopherol from DL-α-TAc was 2 to 3 times greater than for D-α-TAc.

Meglia et al. (2006) looked at the relative proportions of α-tocopherol stereoisomers in the milk and plasma of dairy cows during late gestation and early

lactation. Essentially all of the α-tocopherol in plasma and milk were the RRR conformation when cows were fed diets containing D-α-T or D-α-TAc; however, about 90% of the α-tocopherol were in the RRR conformation in plasma and milk when cows were fed diets contained DL-α-TAc. The remaining 10% was primarily made up of the other 2-R stereoisomers (RRS, RSR, and RSS) and a small portion (1%) of the 2-S stereoisomers (SSS, SRR, SRS, and SSR). Lauridsen and Jensen (2006) performed a similar experiment with sows in late gestation and lactation and determined that when DL-α-TAc was used as the source of α-tocopherol, approximately 35% of the α-tocopherol in milk was in the RRR configuration, 55 to 65% was in the other 2-R configuration, and 5 to 8% were in the 2-S configuration. The relative proportion of different stereoisomers of α-tocopherol in suckling pig plasma was similar to that of the milk. These findings agree with hypothesis that the 2-S stereoisomers are metabolized rapidly and not circulated in the bloodstream at concentrations equal to the 2-R stereoisomers.

A great deal of controversy arises from conflicting terminology in publications. The bioavailability of a nutrient has been defined as the proportion of ingested nutrient that is absorbed in its chemical form and available for use in metabolic pathway (Ammerman et al., 1995). This can be difficult to determine because the chemical forms of D-α-TAc and DL-α-TAc are not identical (Hoppe, 2010). In a letter to the editor, Hoppe (2010) points out that the potency for D-α-TAc relative to DL-α-TAc can be determined only by measuring a physiological activity reflective of vitamin E activity as was accomplished in the rat fetal absorption model. Hoppe (2010) also states that no true potency tests have been conducted in pigs or cattle and it is therefore premature to conclude that the potency ratio is different than 1.36 as was determined in the rat fetal absorption assay (Harris and Ludwig, 1948). Hoppe (2010) claims that measurements of bioavailability based on concentrations or ratios of labeled forms of α-tocopherol in various tissues pools are not valid estimates to replace the potency value of 1.36 as determined in the rat fetal absorption model. The retention of α-tocopherol also needs to be considered because they differ greatly.

Although estimates of bioavailability presented in this experiment may not be true estimates of potency, they do provide insight into the various tissue concentrations of α-tocopherol and how vitamin E sources affect those pools of α-tocopherol. It should be noted that this experiment was performed with concentrations of DL-α-TAc either at or above the sows' requirement (NRC, 1998). True potency can be quantified only in a deficient state, and it has been suggested that the bioavailability changes with different dosages and durations (Hoppe and Krennrich, 2000; Hoppe, 2010). Therefore, the potency of D-α-TAc relative to DL-α-TAc may underestimate the sows' ability to utilize D-α-TAc relative to DL-α-TAc when concentrations at or slightly above the requirement are fed due to differences in retention and elimination of the various α-tocopherol stereoisomers.

In conclusion, this experiment demonstrated a range of bioavailability estimate coefficients. The bioavailability coefficients for D-α-TAc relative to DL-α-TAc ranged from 1.9 to 4.2 for sow and pig plasma α-tocopherol, 2.9 to 3.6 for colostrum α-tocopherol, 1.6 for milk α-tocopherol, 1.8 for heart α-tocopherol, and 2.0 for liver α-tocopherol. Overall, this study suggests that the bioavailability for D-α-TAc relative to DL-α-TAc varies depending on the response criteria but is greater than the standard potency of 1.36 when sows are fed diets close to the requirement of vitamin E.

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