Effects of feeding diets containing highly peroxidized distillers dried grains with solubles and increasing vitamin E levels to wean –finish pigs on growth performance, carcass characteristics, and pork fat composition

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Effects of feeding diets containing highly peroxidized distillers dried grains with solubles and increasing vitamin E levels to wean–finish pigs on growth performance, carcass characteristics, and pork fat composition[1]

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ABSTRACT: Lipid peroxidation in animal feed can negatively affect growth performance and meat quality. Weanling pigs ($n = 432$; BW = 6.6 ± 0.4 kg) were used to evaluate the effects of feeding peroxidized distillers dried grains with solubles (DDGS) with 3 levels of vitamin E (α-tocopheryl acetate) on growth performance, carcass composition, fatty acid composition of pork fat, and lipid peroxidation in LM. The DDGS source used in this study contained the highest thiobarbituric acid reactive substances (TBARS) value, peroxide value, and total S content (5.2 ng malondialdehyde/mg oil, 84.1 mEq/kg oil, and 0.95%, respectively) among 30 DDGS sources sampled. Pens within blocks were assigned randomly to 1 of 6 diets in a 2 × 3 factorial arrangement of treatments with 8 pens per treatment and 9 pigs per pen. Pigs were fed a corn–soybean meal (CON) or 30% peroxidized DDGS (Ox-DDGS) diets with 3 levels of vitamin E: none supplemented (No-E), NRC (1X-E), or 10X NRC (10X-E). Compared to CON, inclusion of 30% Ox-DDGS in diets reduced ($P < 0.001$) final BW (110 vs. 107 kg), overall ADG (0.76 vs. 0.74 kg/d), and G:F (0.39 vs. 0.37). Increasing dietary vitamin E concentrations improved G:F ($P = 0.03$) of pigs fed 10X-E and 1X-E vs. No-E diets (0.39 and 0.39 vs. 0.38, respectively). Hot carcass weight, dressing percentage, backfat depth, and LM area were reduced ($P < 0.01$) in pigs fed Ox-DDGS compared to CON, but percentage of fat-free carcass lean was not affected. Feeding Ox-DDGS increased ($P < 0.001$) PUFA concentration, particularly linoleic acid ($P < 0.001$), and iodine value ($P < 0.001$) in belly fat and backfat compared to pigs fed CON. Dietary vitamin E levels did not affect fatty acid profiles in belly or back fat. Loin muscle TBARS were measured to determine the lipid peroxidation level in pork loins. Although pigs were fed a Ox-DDGS source in this study, TBARS in LM were similar between Ox-DDGS and CON treatments. There was no interaction between Ox-DDGS and dietary vitamin E concentration in LM TBARS. Alpha-tocopherol concentration in LM was greater ($P < 0.001$) in 10X-E than No-E or 1X-E dietary treatments. Compared to CON, feeding Ox-DDGS increased α-tocopherol concentration in LM of pigs fed No-E (1.0 vs. 3.1 mg/kg; $P = 0.005$) but not in those fed 1X-E or 10X-E. These results indicate that feeding diets containing 30% Ox-DDGS to wean–finish pigs may negatively affect growth performance, but supplementation of additional vitamin E in the diet did not counteract these effects.

Key words: corn distillers dried grains with solubles, growth performance, lipid peroxidation, wean–finish pigs, vitamin E

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INTRODUCTION

The use of distillers dried grains with solubles (**DDGS**) in swine diets has increased dramatically in recent years because of its cost competitiveness compared with corn and soybean meal (Stein and Shurson, 2009). Currently, the long-term sustainability of using high levels of DDGS (up to 40%) in grower–finisher swine diets is in jeopardy because of concerns related to potential negative effects of lipids in DDGS on pork

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quality. The lipid fraction of corn DDGS is made up largely of PUFA, particularly linoleic acid (NRC, 1998), which is prone to lipid peroxidation. Lipid peroxidation is a free-radical chain reaction that produces oxidized lipids and a series of toxic aldehydes (Blokhina et al., 2003). Additionally, during drying, DDGS are heated at relatively high temperatures that may accelerate lipid peroxidation by oxidizing unsaturated fatty acids.

Oxidative damage of lipids in feed ingredients represents a substantial economic loss because they can negatively affect animal health and growth performance (Miller and Brzezinska-Slebodzinska, 1993; Pfalzgraf et al., 1995). Furthermore, changes in body composition, pork quality, and reduced shelf life of pork may occur when pigs consume oxidatively damaged feed ingredients, which decreases consumer acceptance of the pork produced (Fernández-Dueñas, 2009). Additionally, toxic secondary lipid peroxidation products may be present in meat products from pigs consuming diets containing high amounts of peroxidized lipids, which may cause human health concerns (Esterbauer et al., 1988).

Supplemental vitamin E in diets can be deposited in fat associated with muscle tissue as α-tocopherol (Jensen et al., 1998). Vitamin E is the most important antioxidant that protects against lipid peroxidation and improves pork shelf life (Jensen et al., 1998). Therefore, the objective of this study was to evaluate the effects of feeding diets containing highly peroxidized DDGS (**Ox-DDGS**) with increasing levels of vitamin E on pig growth performance from weaning to harvest and to determine carcass characteristics, fatty acid composition of belly and backfat, and LM oxidation of pork carcasses.

MATERIALS AND METHODS

All animal care and use procedures used in this experiment were approved by the University of Minnesota Institutional Animal Care and Use Committee.

Animals and Housing

Mixed-sex pigs ($n = 432$; initial BW = 6.6 ± 0.4 kg) produced from crossbred sows (Landrace × Yorkshire; TOPIGS, Winnipeg, MB, Canada) mated to Duroc boars (Compart's Boar Store, Nicollet, MN) were used in this experiment conducted at the University of Minnesota's West Central Research and Outreach Center (Morris, MN). Pigs were housed in an environmentally controlled confinement nursery facility from weaning to d 44 postweaning and then transferred to grower–finisher facility until harvest on d 136. Pigs were blocked by initial BW at weaning, and 9 pigs (either 5 gilts and 4 barrows or 4 gilts and 5 barrows) within block were grouped together in a pen. Pens within block were assigned randomly to 1 of 6 dietary treatments in a 2 × 3 factorial arrangement, resulting in 8 pens per treatment. Each pen (1.6 by 4.5 m) was equipped with 1 nipple drinker and one 4-space self-feeder, with totally slatted floors. Pigs were fed corn–soybean meal (**CON**) or corn–soybean meal–30% Ox-DDGS diets containing 1 of 3 levels of vitamin E (α-tocopheryl acetate): not supplemented (**No-E**), NRC (1998) recommended concentration of vitamin E (**1X-E**), or 10 times the NRC (1998; **10X-E**). Pigs were offered diets in a 7-phase feeding program throughout the 136 d wean-to-finish feeding period based on target BW for each diet phase of 6 to 9 kg, 9 to 16 kg, 16 to 27 kg, 27 to 45 kg, 45 to 68 kg, 68 to 91 kg, and 91 to 113 kg, respectively. Pigs had ad libitum access to their assigned dietary treatments and water throughout the experiment.

Diet Composition and Distillers Dried Grains with Solubles Source

Ingredient composition of Phase 1, 2, and 3 is shown in Table 1. Ingredient composition of Phase 4, 5, and 6 diets is not shown because they consisted of the same ingredients as those used in Phase 3 diets. Analyzed nutrient content of each diet phase is presented in Table 2. All diets were fed in meal form and were formulated on a standardized ileal digestible AA and available P basis. Nutrient concentration of all diets met or exceeded NRC (1998) recommended nutrient requirements for pigs with 350 g of fat-free lean gain/d, except for vitamin E concentration in the No-E treatments. Vitamin E was supplemented in the form of DL-α-tocopheryol acetate in 1X-E and 10X-E treatments. The high Ox-DDGS source used in this study was selected from 31 corn DDGS sources produced by U.S. ethanol plants (Song and Shurson, 2013). This DDGS source that contained the highest thiobarbituric acid reactive substances (**TBARS**) value, peroxide value (**PV**), and total S content (5.2 ng malondialdehyde [**MDA**]/mg oil, 84.1 mEq/kg oil, and 0.95%, respectively) among the other 30 DDGS sources sampled (mean values = 1.8 ng MDA/mg oil, 11.5 mEq/kg oil, and 0.50%, respectively).

Growth Performance

Pigs were weighed individually on the day dietary treatments were imposed and every 2 wk or every week if a diet-phase change was needed during the experiment. Individual BW of the pigs within pens was used to calculate ADG on a pen basis. On each weigh day, feed disappearance was measured to calculate ADFI of pigs on a pen basis. Pen ADG and ADFI were used to calculate G:F.

Table 1. Composition of Phase 1, 2 and 3 experimental diets[1]

	CON[2]			DDGS[3]		
Ingredient, %	No-E[4]	1X-E[4]	10X-E[4]	No-E	1X-E	10X-E
Phase 1						
Corn	43.84	43.81	43.52	23.40	23.37	23.09
Soybean meal (46.5% CP)	23.13	23.13	23.13	13.75	13.75	13.75
DDGS	–	–	–	30.00	30.00	30.00
Fish meal, menhaden	10.00	10.00	10.00	10.00	10.00	10.00
Whey powder	20.00	20.00	20.00	20.00	20.00	20.00
Limestone	0.89	0.89	0.89	1.27	1.27	1.27
Monocalcium phosphate	0.70	0.70	0.70	–	–	–
NaCl	0.25	0.25	0.25	0.25	0.25	0.25
L-Lys HCl	–	–	–	0.17	0.17	0.17
DL-Met	0.05	0.05	0.05	–	–	–
L-Trp	–	–	–	0.03	0.03	0.03
L-Thr	0.01	0.01	0.01	–	–	–
Vitamin–trace mineral premix[5]	0.50	0.50	0.50	0.50	0.50	0.50
Denagard[6]	0.18	0.18	0.18	0.18	0.18	0.18
Chlortetracycline	0.40	0.40	0.40	0.40	0.40	0.40
Zinc oxide	0.05	0.05	0.05	0.05	0.05	0.05
Vitamin E[7]	–	0.032	0.32	–	0.032	0.32
Total	100.00	100.00	100.00	100.00	100.00	100.00
Phase 2						
Corn	59.16	59.13	58.87	38.95	38.92	38.66
Soybean meal (46.5% CP)	37.64	37.64	37.64	28.00	28.00	28.00
DDGS	–	–	–	30.00	30.00	30.00
Limestone	0.95	0.95	0.95	1.34	1.34	1.34
Monocalcium phosphate	1.31	1.31	1.31	0.61	0.61	0.61
NaCl	0.35	0.35	0.35	0.35	0.35	0.35
L-Lys HCl	0.07	0.07	0.07	0.25	0.25	0.25
DL-Met	0.02	0.02	0.02	–	–	–
Vitamin–trace mineral premix[5]	0.50	0.50	0.50	0.50	0.50	0.50
Vitamin E[7]	–	0.029	0.29	–	0.029	0.29
Total	100.00	100.00	100.00	100.00	100.00	100.00
Phase 3						
Corn	60.12	60.09	59.85	40.49	40.46	40.23
Soybean meal (46.5% CP)	37.08	37.08	37.08	26.82	26.82	26.82
DDGS	–	–	–	30.00	30.00	30.00
Limestone	0.91	0.91	0.91	1.30	1.30	1.30
Monocalcium phosphate	1.01	1.01	1.01	0.31	0.31	0.31
NaCl	0.35	0.35	0.35	0.35	0.35	0.35
L-Lys HCl	0.03	0.03	0.03	0.23	0.23	0.23
Vitamin–trace mineral premix[5]	0.50	0.50	0.50	0.50	0.50	0.50
Vitamin E[7]	–	0.026	0.26	–	0.026	0.26
Total	100.00	100.00	100.00	100.00	100.00	100.00

[1]Phase 4 to 7 experimental diets consisted of the same ingredients as Phase 3 diets and are not shown.

[2]CON = corn–soybean meal based control diet.

[3]DDGS = diets contained 30% peroxidized corn distillers dried grains with solubles.

[4]No-E = no supplemental vitamin E; 1X-E = NRC (1998) level of supplemental vitamin E; and 10X-E = 10 times the NRC (1998) recommended level of supplemental vitamin E. Vitamin E was supplied as α-tocopheryl acetate.

[5]Vitamin–trace mineral premix (without vitamin E) provided the following nutrients per kilogram of diet: 17,637 IU of vitamin A as retinyl acetate, 3,307 IU of vitamin D_3 as cholecalciferol, 6.16 mg of vitamin K as menadione dimethylpyrimidinol bisulfite, 11.02 mg of riboflavin, 66.14 mg of niacin, 44.09 mg of pantothenic acid as D-calcium pantothenate, 0.07 mg of vitamin B_{12}, 0.60 mg of iodine as ethylenediamine dihydroiodide, 0.60 mg of selenium as sodium selenite, 110.23 mg of zinc as a polysaccharide encapsulated complex of zinc ([**SQM**]; QualiTech, Inc., Chaska, MN), 66.14 mg of iron as a polysaccharide encapsulated complex of iron (SQM), 7.72 mg of copper as a polysaccharide encapsulated complex of copper (SQM), and 11.02 mg of manganese as a polysaccharide encapsulated complex of manganese (SQM).

[6]Denagard 10 Medicated Premix (Novartis Animal Health U.S., Inc., Greensboro, NC) containing 2,200 Tiamulin (as hydrogen fumarate).

[7]Vitamin E was supplied as α-tocopheryl acetate with concentration of 44,092 IU/kg.

Table 2. Energy and nutrient composition of experimental diets (as-fed basis)[1]

	CON[2]			DDGS[3]		
Item	No-E[4]	1X-E[4]	10X-E[4]	No-E	1X-E	10X-E
Phase 1						
Calculated ME,[5] kcal/kg	3,276	3,275	3,265	3,288	3,287	3,277
CP, %	22.9	–	–	24.7	–	–
Crude fat, %	2.48	–	–	3.78	–	–
Crude fiber, %	2.0	–	–	3.0	–	–
Lys, %	1.54	–	–	1.52	–	–
Met, %	0.38	–	–	0.42	–	–
Thr, %	0.95	–	–	1.00	–	–
Trp, %	0.27	–	–	0.28	–	–
Ca, %	1.32	–	–	1.35	–	–
P, %	0.92	–	–	0.91	–	–
Calculated available P, %	0.62	–	–	0.62	–	–
Calculated α-tocopherol,[6] IU/kg	0.00	14	141	0.00	14	141
α-tocopherol, IU/kg	<10	13	137	<10	19	127
Phase 2						
Calculated ME, kcal/kg	3,282	3,281	3,272	3,293	3,292	3,283
CP, %	21.7	–	–	23.3	–	–
Crude fat, %	2.20	–	–	3.67	–	–
Crude fiber, %	2.7	–	–	3.6	–	–
Lys, %	1.37	–	–	1.35	–	–
Met, %	0.33	–	–	0.39	–	–
Thr, %	0.84	–	–	0.89	–	–
Trp, %	0.27	–	–	0.25	–	–
Ca, %	0.96	–	–	0.97	–	–
P, %	0.70	–	–	0.72	–	–
Calculated available P, %	0.36	–	–	0.36	–	–
Calculated α-tocopherol, IU/kg	0.00	13	128	0.00	13	128
α-tocopherol, IU/kg	<10	12	117	<10	13	124
Phase 3						
Calculated ME, kcal/kg	3,296	3,295	3,287	3,306	3,306	3,298
CP, %	20.6	–	–	23.0	–	–
Crude fat, %	2.41	–	–	3.82	–	–
Crude fiber, %	2.5	–	–	3.4	–	–
Lys, %	1.32	–	–	1.30	–	–
Met, %	0.32	–	–	0.38	–	–
Thr, %	0.83	–	–	0.88	–	–
Trp, %	0.27	–	–	0.24	–	–
Ca, %	0.71	–	–	0.81	–	–
P, %	0.62	–	–	0.64	–	–
Calculated available P, %	0.29	–	–	0.29	–	–
Calculated α-tocopherol, IU/kg	0.00	11	115	0.00	11	115
α-tocopherol, IU/kg	<10	16	129	<10	16	118
Phase 4						
Calculated ME, kcal/kg	3,317	3,317	3,309	3,329	3,328	3,321
CP, %	16.5	–	–	19.3	–	–
Crude fat, %	2.61	–	–	3.79	–	–
Crude fiber, %	2.2	–	–	3.3	–	–
Lys, %	1.14	–	–	1.12	–	–
Met, %	0.28	–	–	0.35	–	–
Thr, %	0.70	–	–	0.77	–	–
Trp, %	0.22	–	–	0.20	–	–
Ca, %	0.72	–	–	0.72	–	–
P, %	0.55	–	–	0.54	–	–
Calculated available P, %	0.24	–	–	0.24	–	–
Calculated α-tocopherol, IU/kg	0.00	11	110	0.00	11	110
α-tocopherol, IU/kg	<10	12	105	<10	15	124

(Continued)

Table 2. Continued

Item	CON[2]			DDGS[3]		
	No-E[4]	1X-E[4]	10X-E[4]	No-E	1X-E	10X-E
Phase 5						
Calculated ME, kcal/kg	3,327	3,326	3,319	3,340	3,339	3,332
CP, %	12.1	–	–	17.9	–	–
Crude fat, %	2.84	–	–	3.91	–	–
Crude fiber, %	2.0	–	–	3.6	–	–
Lys, %	0.97	–	–	0.96	–	–
Met, %	0.25	–	–	0.33	–	–
Thr, %	0.61	–	–	0.71	–	–
Trp, %	0.18	–	–	0.18	–	–
Ca, %	0.61	–	–	0.65	–	–
P, %	0.52	–	–	0.56	–	–
Calculated available P, %	0.22	–	–	0.22	–	–
Calculated α-tocopherol, IU/kg	0.00	11	110	0.00	11	110
α-tocopherol, IU/kg	<10	12	126	<10	12	106
Phase 6						
Calculated ME, kcal/kg	3,331	3,330	3,323	3,344	3,343	3,336
CP, %	12.1	–	–	16.6	–	–
Crude fat, %	2.51	–	–	4.22	–	–
Crude fiber, %	2.0	–	–	3.2	–	–
Lys, %	0.83	–	–	0.82	–	–
Met, %	0.23	–	–	0.31	–	–
Thr, %	0.54	–	–	0.64	–	–
Trp, %	0.15	–	–	0.15	–	–
Ca, %	0.61	–	–	0.56	–	–
P, %	0.53	–	–	0.51	–	–
Calculated available P, %	0.21	–	–	0.21	–	–
Calculated α-tocopherol, IU/kg	0.00	11	110	0.00	11	110
α-tocopherol, IU/kg	<10	12	107	<10	16	112
Phase 7						
Calculated ME, kcal/kg	3,335	3,334	3,326	3,348	3,348	3,340
CP, %	11.2	–	–	15.5	–	–
Crude fat, %	2.48	–	–	4.20	–	–
Crude fiber, %	2.2	–	–	3.0	–	–
Lys, %	0.70	–	–	0.69	–	–
Met, %	0.21	–	–	0.29	–	–
Thr, %	0.46	–	–	0.57	–	–
Trp, %	0.13	–	–	0.13	–	–
Ca, %	0.55	–	–	0.54	–	–
P, %	0.50	–	–	0.50	–	–
Calculated available P, %	0.21	–	–	0.21	–	–
Calculated α-tocopherol, IU/kg	0.00	11	110	0.00	11	110
α-tocopherol, IU/kg	<10	14	102	<10	11	109

[1]Analyzed values unless otherwise indicated. Except for α-tocopherol, nutrient concentrations were only analyzed for No-E and CON and No-E and DDGS treatments because values were assumed to be similar to their corresponding 1X-E and 10X-E treatments.

[2]CON = corn–soybean meal based control diet.

[3]DDGS = diets contained 30% peroxidized corn distillers dried grains with solubles.

[4]No-E = no supplemental vitamin E; 1X-E = NRC (1998) level of supplemental vitamin E; and 10X-E = 10 times NRC (1998) recommended level of supplemental vitamin E. Vitamin E was supplied as α-tocopheryl acetate.

[5]ME values were calculated using NRC (1998) values for corn and soybean meal without hulls (46.5% CP), and 3,416 kcal/kg for DDGS, which was the average value of 10 DDGS samples analyzed by Pedersen et al. (2007).

[6]Indicates calculated concentration of supplemental α-tocopherol (IU/kg).

Ultrasound and Carcass Measurements

Ultrasound was used to determine 10th rib backfat thickness and LM area 2 d before harvest when the average BW of all pigs reached 113 kg. Scanning was accomplished by a trained technician using a real-time ultrasonic machine (ALOKA 500V; Corometrics Medical Systems,

Wallingford, CT) fitted with a 12.5-cm long, 3.5-MHz linear array transducer. Ultrasound measurements were taken along the dorsal midline at the 10th rib. The transducer was aligned perpendicular to the spine at the 10th rib. Digitized images were processed using software (Quality Evaluation and Prediction Software; Iowa State University, Ames, IA) specifically developed to measure linear distance and area of digitized images and matriculate results to a data file. Tenth rib backfat depth was measured at a point three-fourths of the distance of the LM, curvilinear from the spine and perpendicular to the LM surface.

All pigs were harvested on the same day (Hormel Foods Corporation, Austin, MN). Pigs were weighed individually 1 d before harvest to obtain a final live BW. Hot carcass weight was measured on carcasses immediately after harvest. Live BW of pigs along with HCW were used to calculate dressing percentage using the following equation: dressing, % = (HCW/live BW) × 100. Percentage of fat-free carcass lean was calculated using the following equation according to the National Pork Producers Council (NPPC, 2000): fat-free carcass lean, % = {[2.620 + (0.401 × HCW, kg) – (3.358 × ultrasound 10th rib backfat depth, cm) + (0.306 × ultrasound 10th rib LM area, cm^2) + (0.456 × sex of pig) (barrow = 1, gilt = 2)]/[HCW, kg]} × 100.

Pork Fat Composition of Belly and Backfat

Forty-eight gilts (1 from each pen) weighing closest to the mean BW of their pen were selected to determine fatty acid composition of pork fat. Twenty-four hours after carcasses were chilled at 1.7 to 4.4°C, 2.54-cm cores from the belly and backfat were collected. The belly tissue cores were collected at the midline opposite the last rib, and the backfat cores were collected at the 10th-rib location on the right side of the carcass. Core samples of belly and backfat were stored at –20°C after collection and sent to University of Missouri Agricultural Experiment Station Chemical Laboratories (Columbia, MO) for analysis of fatty acid profile using gas chromatography according to the American Oil Chemists' Society (AOCS, 1998) method (Ce 1-62). The iodine value (**IV**) of fat was calculated using the following equation (AOCS, 1998): IV = (C16:1 × 0.95) + (C18:1 × 0.86) + (C18:2 × 1.732) + (C18:3 × 2.616) + (C20:1 × 0.785) + (C22:1 × 0.723).

Lipid Peroxidation in LM

To evaluate the level of lipid peroxidation and α-tocopherol concentration in LM, samples from the 48 selected carcasses were collected at the 10th rib from the right side 24 h after chilling. Each LM sample was separated into 2 equal halves and stored at –80°C immediately. One section of LM was sent to Michigan State University Diagnostic Center for Population & Animal Health (Lansing, MI) for analysis of α-tocopherol concentration using ethanol and hexane extraction followed by quantification via HPLC (Separation Module 2690; Waters, Milford, MA; Katsanidis and Addis, 1999). Another section of LM was used to determine TBARS concentration following the method described by Animal Models of Diabetic Complications Consortium (AMDCC, 2005). Specifically, 50 mg of LM sample was weighed into a 2-mL flat-bottom centrifuge tube containing 500 μL PBS and 1% Triton X-100 (Sigma-Aldrich, St. Louis, MO). The mixture was homogenized on ice for 15 s followed by centrifugation at 10,000 × *g* for 5 min at 4 °C to collect the supernatant. One hundred microliters supernatant and standards of malonaldehyde (catalog number AC14861-1000; Fisher Scientific, Pittsburgh, PA) were mixed with 200 μL ice cold 10% trichloroacetic acid (Sigma-Aldrich) and centrifuged at 2,200 × *g* for 15 min at 4º C. Two hundred microliters of supernatant were removed and incubated with an equal volume of 0.67% (w/v) thiobarbituric acid (Sigma-Aldrich) for 10 min in a boiling water bath. The mixture was cooled to room temperature and read at 532 nm using a spectrophotometer (SpectraMax 250; Molecular Device, Sunnyvale, CA). This assay was conducted in 4 batches with duplicate samples and a standard. The intra-assay CV was 7.4% and the interassay CV was 4.6%.

Statistical Analysis

All data were analyzed using the MIXED procedure of SAS (SAS Inst. Inc., Cary, NC). The pen was used as the experimental unit for all responses. The statistical model included the fixed effects of Ox-DDGS, vitamin E concentration, and Ox-DDGS × vitamin E interactions and random effect of block. Live BW before harvest was used as the covariate in analysis of carcass characteristics if the effect of live BW was significant ($P < 0.05$). Repeated measures in time were used to analyze growth performance data throughout the 7 dietary phases. The unstructured option was used to fit a variance–covariance matrix in the model for repeated measures in time. The slice option of SAS was used to separate main effects within different phases. All results are reported as least squares means. Multiple comparisons among treatments were performed using PDIFF and adjusted by Tukey option for multiple comparisons of means. The significance level chosen was $\alpha = 0.05$. Treatment effects were considered significant if $P < 0.05$ whereas values between 0.05 and 0.10 were considered statistical trends.

RESULTS

During the experiment, 11 pigs (CON and No-E = 1, CON and 10X-E = 4, Ox-DDGS and No-E = 1, Ox-

Table 3. Effects of dietary distillers dried grains with solubles (DDGS) and increasing levels of vitamin E on overall growth performance and carcass and ultrasound measurements[1]

	CON[2]			DDGS[3]				*P*-value		
Item	No-E[4]	1X-E[4]	10X-E[4]	No-E	1X-E	10X-E	SE	DDGS	E	DDGS× E
Growth performance										
No. of pens	8	8	8	8	8	8				
No. of pigs	71	72	68	71	69	70				
Initial BW, kg	6.6	6.6	6.6	6.6	6.6	6.6	0.35	1.00	0.69	0.33
Final BW, kg	111.4	109.5	109.8	105.5	107.2	106.9	1.71	<0.001	1.00	0.21
ADG, kg	0.77	0.76	0.76	0.73	0.74	0.74	0.01	<0.001	1.00	0.21
ADFI, kg	1.99	1.90	1.91	1.99	1.96	1.91	0.04	0.23	0.10	0.66
G:F	0.39	0.40	0.40	0.37	0.38	0.38	0.13	<0.001	0.03	0.99
Carcass ultrasound measurements										
No. of pens	8	8	8	8	8	8				
No. of pigs	68	63	62	54	56	59				
HCW,[5] kg	89.2	89.3	89.2	87.4	87.9	88.3	0.4	<0.001	0.43	0.36
Dressing, %	78.2	78.1	78.1	76.4	76.9	77.2	0.003	<0.001	0.47	0.31
Ultrasound backfat depth,[5] cm	2.37	2.45	2.45	2.28	2.27	2.27	0.09	0.008	0.85	0.72
Ultrasound LM area,[5] cm^2	39.4	38.5	38.1	36.4	36.1	35.8	0.6	<0.001	0.26	0.81
Fat-free carcass lean, %	48.5	47.6	47.5	47.9	47.8	47.7	0.004	0.83	0.26	0.49

[1]Values are least square means of 8 replicate pens per dietary treatment.

[2]CON = corn–soybean meal based control diet.

[3]DDGS = diets contained 30% peroxidized corn distillers dried grains with solubles.

[4]No-E = no supplemental vitamin E; 1X-E = NRC (1998) level of supplemental vitamin E; and 10X-E = 10 times NRC (1998) recommended level of supplemental vitamin E. Vitamin E was supplied as α-tocopheryl acetate.

[5]Values were adjusted using live BW as a covariate because live BW in the model was statistically significant ($P < 0.05$). If not adjusted for live BW, *P*-values did not change between significant ($P < 0.05$) and a trend ($P < 0.10$).

DDGS and 1X-E = 3, and Ox-DDGS and 10X-E = 2) were removed for reasons unrelated to dietary treatments ($\chi^2 = 4.17$, df = 2, $P = 0.12$). Fifty-nine out of 421 pigs (CON/ and No-E = 3, CON and 1X-E = 9, CON and 10X-E = 6, Ox-DDGS/No-E = 17, Ox-DDGS/ and 1X-E = 13, and Ox-DDGS and 10X-E = 11; $\chi^2 = 90.42$, df = 2, $P < 0.001$) were excluded from collection of carcass data because they were too light to fit specifications of the processor and could not be slaughtered on the same day as contemporaries.

Growth Performance

No interaction between dietary Ox-DDGS and vitamin E supplementation level was observed for overall growth performance responses (Table 3). There were time effects for ADG ($P < 0.001$), ADFI ($P < 0.001$), and G:F ($P < 0.001$) across phases but no time × Ox-DDGS × vitamin E interactions. For the overall period, initial BW at weaning was similar among dietary treatments. However, compared with pigs consuming CON, ADG (0.74 vs. 0.76 kg; $P < 0.001$) and final BW at harvest (106.6 vs. 110.2 kg; $P < 0.001$) were lower in pigs consuming Ox-DDGS. There were no effects of feeding Ox-DDGS on ADFI, but G:F was lower (0.37 vs. 0.39; $P < 0.001$) in pigs fed Ox-DDGS compared with those fed CON. Increasing dietary vitamin E concentrations had no effect on BW and ADG but tended to decrease ADFI ($P = 0.10$), resulting in an improved G:F ($P = 0.03$) of pigs fed 10X-E and 1X-E vs. No-E diets (0.39 and 0.39 vs. 0.38, respectively).

Ultrasound and Carcass Characteristics

No effects of vitamin E supplementation or interaction between Ox-DDGS and vitamin E were detected for any carcass measurements (Table 3). Because of reduced growth rate, pigs fed Ox-DDGS had a lower live BW (113.3 vs. 115.2 kg; $P = 0.04$) and HCW (87.9 vs. 89.2 kg; $P < 0.001$) compared with those fed CON. Dressing percentage was also decreased in pigs consuming Ox-DDGS diets (76.9 vs. 78.2%; $P < 0.001$) compared with those fed CON. Additionally, ultrasound 10th rib backfat depth was less in pigs fed Ox-DDGS than those fed CON (2.3 vs. 2.4 cm; $P = 0.008$). Ultrasound LM area was also smaller when comparing pigs fed Ox-DDGS with those fed CON (36.1 vs. 38.7 cm^2; $P < 0.001$). However, the fat-free carcass lean calculated as the percentage of HCW was unaffected by dietary treatments.

Fatty Acid Composition and Iodine Value of Belly and Backfat

Changes in fatty acid composition of belly fat and backfat resulting from feeding Ox-DDGS and increasing levels

Table 4. Effects of dietary distillers dried grains with solubles (DDGS) and increasing levels of vitamin E on fatty acid profile of belly fat and backfat samples[1]

	CON[2]			DDGS[3]				*P*-value		
Item	No-E[4]	1X-E[4]	10X-E[4]	No-E	1X-E	10X-E	SE	DDGS	E	DDGS × E
Belly fat sample										
Fatty acid, % of total fat										
C14:0	1.44	1.39	1.39	1.35	1.38	1.35	0.04	0.11	0.77	0.51
C16:0	25.13	24.74	25.13	22.74	22.97	22.67	0.33	<0.001	0.97	0.51
C16:1n-7	3.12	3.23	2.66	2.60	2.53	2.70	0.15	0.003	0.36	0.05
C18:0	11.88	11.21	13.10	10.40	10.53	10.25	0.37	<0.001	0.08	0.02
C18:1n-9	46.32	48.37	46.80	43.29	43.28	43.27	0.46	<0.001	0.08	0.08
C18:2n-6	8.48	7.25	7.20	15.27	15.03	15.36	0.63	<0.001	0.46	0.52
C18:3n-3	0.44	0.41	0.41	0.59	0.57	0.57	0.02	<0.001	0.48	0.95
C20:0	0.20	0.18	0.22	0.18	0.20	0.19	0.01	0.18	0.23	0.03
C20:1n-9	0.82	0.80	0.91	0.83	0.85	0.85	0.04	0.98	0.32	0.37
C20:3n-6	0.04	0.05	0.06	0.08	0.08	0.08	0.01	<0.001	0.31	0.65
C20:4n-6	0.22	0.20	0.20	0.28	0.27	0.29	0.02	<0.001	0.35	0.58
SFA	39.02	37.92	40.18	35.15	35.51	34.92	0.57	<0.001	0.31	0.04
MUFA	50.69	52.88	50.78	47.22	47.13	47.31	0.52	<0.001	0.11	0.05
PUFA	9.23	7.94	7.90	16.26	15.99	16.34	0.66	<0.001	0.45	0.54
IV[5]	59.27	58.90	57.02	68.31	67.82	68.52	0.95	<0.001	0.53	0.28
Backfat sample										
Fatty acid, % of total fat										
C14:0	1.38	1.35	1.35	1.26	1.29	1.27	0.04	0.01	0.98	0.72
C16:0	25.58	25.04	26.09	23.32	23.52	23.01	0.50	<0.001	0.84	0.25
C16:1n-7	2.44	2.74	2.13	2.05	1.89	2.16	0.14	0.001	0.48	0.01
C18:0	13.92	12.64	15.83	12.13	12.56	11.44	0.64	<0.001	0.24	0.004
C18:1n-9	42.33	44.28	42.54	39.62	38.55	39.83	0.69	<0.001	0.80	0.04
C18:2n-6	9.98	9.36	8.12	16.87	17.42	16.93	1.05	<0.001	0.36	0.38
C18:3n-3	0.52	0.50	0.45	0.64	0.64	0.66	0.03	<0.001	0.61	0.20
C20:0	0.23	0.20	0.27	0.20	0.22	0.20	0.01	0.01	0.19	0.003
C20:1n-9	0.83	0.79	0.92	0.82	0.81	0.80	0.04	0.25	0.27	0.21
C20:3n-6	0.07	0.07	0.08	0.08	0.08	0.09	0.00	<0.001	0.08	0.83
C20:4n-6	0.20	0.19	0.18	0.27	0.26	0.28	0.02	<0.001	0.87	0.40
SFA	41.55	39.71	43.94	37.46	38.12	36.50	1.07	<0.001	0.41	0.02
MUFA	46.03	48.32	45.96	42.97	41.69	43.31	0.79	<0.001	0.80	0.03
PUFA	10.83	10.16	8.86	17.91	18.44	18.03	1.09	<0.001	0.37	0.37
IV[5]	58.58	59.65	54.66	67.69	67.79	68.93	1.68	<0.001	0.44	0.11

[1]Values are least square means of 8 pigs (1 pig/pen) per dietary treatment.

[2]CON = corn–soybean meal based control diet.

[3]DDGS = diets contained 30% peroxidized corn distillers dried grains with solubles.

[4]No-E = no supplemental vitamin E; 1X-E = NRC (1998) level of supplemental vitamin E; and 10X-E = 10 times NRC (1998) recommended level of supplemental vitamin E. Vitamin E was supplied as α-tocopheryl acetate.

[5]IV = iodine value.

of vitamin E are presented in Table 4. Compared with pigs fed CON, pigs fed Ox-DDGS had 10% lower SFA content (39.04 vs. 35.19%; $P < 0.001$) and 8% lower MUFA content (51.45 vs. 47.22%; $P < 0.001$) in belly fat. Specifically, pigs fed Ox-DDGS had lower content of palmitic acid (**C16:0**; $P < 0.001$), stearic acid (**C18:0**; $P < 0.001$), palmitoleic acid (**C16:1n-7**; $P = 0.003$), and oleic acid (**C18:1n-9**; $P < 0.001$) than those fed CON. Conversely, pigs fed Ox-DDGS diets had higher PUFA content (16.20 vs. 8.36%; $P < 0.001$), including linoleic acid (**C18:2n-6**; 15.22 vs. 7.64%; $P < 0.001$), linolenic acid (**C18:3n-3**; 0.58 vs. 0.42%; $P < 0.001$), eicosatrienoic acid (**C20:3n-6**; $P < 0.001$), and arachidonic acid (**C20:4n-6**; $P < 0.001$) than pigs fed CON. Because of the increase in the concentration of PUFA, the IV of belly fat was increased when comparing Ox-DDGS vs. CON treatments (68.2 vs. 58.4; $P < 0.001$). There were no effects of vitamin E supplementation level on fatty acid profile of belly fat. However, an interaction between Ox-DDGS and vitamin E was observed for SFA ($P = 0.04$) and MUFA ($P = 0.05$). In pigs fed 30% Ox-DDGS diets, there were no effects of vitamin E on SFA or MUFA concentration in belly fat. However, in pigs fed

Table 5. Influence of dietary distillers dried grains with solubles (DDGS) and vitamin E supplementation on thiobarbituric acid reactive substance (TBARS) and α-tocopherol concentration in LM[1]

	CON[2]			DDGS[3]				*P*-value		
Item	No-E[4]	1X-E[4]	10X-E[4]	No-E	1X-E	10X-E	SE	DDGS	E	DDGS × E
TBARS, mg MDA[5]/kg	0.44	0.43	0.44	0.45	0.44	0.43	0.01	0.93	0.27	0.31
α-tocopherol (as-is), mg/kg	1.03	2.03	7.99	3.10	2.46	8.87	0.89	0.005	<0.001	0.02

[1]Values are least square means of 8 pigs (1 pig/pen) per dietary treatment.

[2]CON = corn–soybean meal based control diet.

[3]DDGS = diets contained 30% peroxidized corn distillers dried grains with solubles.

[4]No-E = no supplemental vitamin E; 1X-E = NRC (1998) level of supplemental vitamin E; and 10X-E = 10 times NRC (1998) recommended level of supplemental vitamin E. Vitamin E was supplied as α-tocopheryl acetate.

[5]MDA = malondialdehyde.

CON, supplementing vitamin E at the NRC level reduced SFA concentration but increased MUFA concentration compared with supplementing at 10X NRC level (37.92 vs. 40.18% and 52.88 vs. 50.78%, respectively).

Similar to belly fat, pigs fed Ox-DDGS had approximately 11% less SFA content (37.36 vs. 41.73%; $P < 0.001$) and 9% less MUFA content (42.66 vs. 46.77%; $P < 0.001$) in backfat than those fed CON. Specifically, feeding Ox-DDGS resulted lower content of myristic acid (**C14:0**; $P = 0.01$), C16:0 ($P < 0.001$), C18:0 ($P < 0.001$), arachidic acid (**C20:0**; $P = 0.01$), C16:1n-7 ($P < 0.001$), and C18:1n-9 ($P < 0.001$) compared with feeding CON. In contrast, feeding Ox-DDGS increased PUFA content of back fat compared to CON by approximately 82% (18.13 vs. 9.95%; $P < 0.001$) , including C18:2n-6, C18:3n-3, C20:3n-6, and C20:4n-6 ($P < 0.001$). In addition, IV of backfat was increased when comparing Ox-DDGS with CON treatments (68.1 vs. 57.6; $P < 0.001$). There were no effects of dietary vitamin E level on fatty acid composition of backfat. However, there were interactions between Ox-DDGS and vitamin E level for SFA ($P = 0.02$) and MUFA ($P = 0.03$). Compared with supplementing vitamin E at 10X NRC level, providing vitamin E at 1X NRC level reduced SFA (43.94 vs. 39.71%) and increased MUFA (45.96 vs. 48.32%) concentrations in pigs fed CON but increased SFA (36.50 vs. 38.12%) and reduced MUFA (43.31 vs. 41.69%) concentrations in pigs fed Ox-DDGS.

Lipid Peroxidation in LM

There were no effects of Ox-DDGS, vitamin E supplementation level, or their interaction on TBARS values on fresh LM (Table 5). An interaction between Ox-DDGS and vitamin E concentration was detected ($P = 0.02$) for α-tocopherol concentration in LM. Specifically, pigs fed Ox-DDGS without vitamin E supplementation (No-E) had a greater concentration of serum α-tocopherol compared with those fed CON and No-E (3.10 vs. 1.03 mg/kg; $P = 0.005$). However, when vitamin E was supplemented at NRC or 10X NRC levels, LM α-tocopherol concentration was similar for pigs fed Ox-DDGS and CON diets. Additionally, α-tocopherol concentration of LM was greater when dietary vitamin E was provided at 10X NRC level than 1X-E and No-E in both Ox-DDGS and CON treatments (8.43 vs. 2.25 and 2.06 mg/kg, respectively; $P < 0.001$). However, no differences were observed between No-E and 1X-E treatments for α-tocopherol concentration of LM.

DISCUSSION

With expansion of the U.S. ethanol industry in the past decade, the use of corn co-products, such as DDGS, in swine feeds has increased dramatically because of increased availability and cost competitiveness compared with corn and soybean meal. However, limits on dietary DDGS inclusion rates may occur because of reduced growth performance and pork fat quality when high dietary levels of DDGS (>20%) are fed to growing–finishing pigs. Results from some experiments showed that feeding diets containing 20 or 30% DDGS results in similar growth performance (Cook et al., 2005; DeDecker et al., 2005; Gaines et al., 2007) whereas other experiments (Whitney et al., 2006; Linneen et al., 2008) showed reduced ADG and ADFI in pigs fed diets containing DDGS compared with those fed standard corn–soybean meal diets. The reduction in growth performance may be caused by several factors, such as poor AA balance and digestibility, low NE level, antinutritional factors, toxins, or peroxidized lipids in DDGS or all of these (Stein and Shurson, 2009).

The lipid content in corn DDGS is approximately 10% and primarily consists of PUFA, particularly C18:2n-6 (NRC, 1998), that are vulnerable to lipid peroxidation. The secondary lipid peroxidation products produced during the peroxidation process are highly reactive and potentially cause damage to lipids, proteins, and nucleic acids (Logani and Davies, 1979; Comporti, 1993) and thus impair animal health and growth performance (Dibner et al., 1996; DeRouchey et al., 2004; Harrell et al., 2010). Additionally, drying temperatures used by ethanol plants vary substantially, and increased

drying time and temperature during the production process of DDGS may accelerate lipid peroxidation by oxidizing unsaturated fatty acids in DDGS. In the current study, a DDGS source containing a high amount of peroxidized lipids was selected according to a recent survey conducted in our laboratory (Song and Shurson, 2013).

Growth performance has been evaluated previously in swine when feeding different types of peroxidized lipids, but the responses were inconsistent. For example, reduced ADG was reported in pigs fed 5% peroxidized corn oil (Fernández-Dueñas, 2009; Harrell et al., 2010) while other studies reported no differences in growth rate and feed intake (Mitchaothai et al., 2007; Fernández-Dueñas, 2009). The lack of negative effects on animal performance may be due to insufficient dietary oxidative challenge. Peroxide value, which measures hydroperoxides, is a commonly used indicator of lipid peroxidation (Gray, 1978). DeRouchey et al. (2004) suggested that there appears to be a threshold for peroxidation, above which growth performance is decreased. Those authors reported that a PV for peroxidized lipids (6% dietary inclusion rate) of less than 40 mEq/kg, which is about equal to a dietary PV less than 2.4 mEq/kg (2.4 mEq/kg = 40 mEq/kg × 6%), might not result in decreased growth performance in nursery pigs. The Ox-DDGS source used in the current study contained 9.66% crude fat and a PV of 84.1 mEq/kg oil. Therefore, by including 30% Ox-DDGS in the diet, the PV of the diet was 2.4 mEq/kg (84.1 mEq/kg oil × 9.66% crude fat × 30% inclusion rate), which is at the threshold level suggested by DeRouchey et al. (2004). To our knowledge, no study has evaluated the influence of feeding peroxidized lipids in DDGS on pig growth performance. In the current study, compared with pigs fed CON, those fed Ox-DDGS showed a 3% reduction in overall ADG, 3% reduction in final BW, and 5% reduction in overall G:F, which were in agreement with the negative effects reported from feeding peroxidized oil on pig growth performance by Fernández-Dueñas (2009) and Harrell et al. (2010). However, because we did not compare performance relative to a low Ox-DDGS source in the present study, it is not possible to determine if the depressed growth performance was due to the peroxidized lipids in DDGS or due to some other factors, such as overestimation of Lys digestibility in the DDGS source used, reduced NE content, or other factors.

The effects of supplementing antioxidants to animals under a dietary peroxidative challenge on growth performance have been inconsistent. Harrell et al. (2010) reported that supplementing a blend of antioxidants (AGRADO PLUS; Novus International Inc., St. Charles, MO) to diets containing 5% peroxidized corn oil (PV = 7.5 mEq/kg of diet) improved ADG, ADFI, and BW but not G:F in nursery pigs. In contrast, growth performance was not improved in finishing pigs fed 5% peroxidized corn oil (PV = 9 mEq/kg of diet; Fernández-Dueñas, 2009) when up to 132 mg/kg of ethoxyquin was added to the diet. The reasons for these inconsistent responses are unclear but may be due to different types and concentrations of supplemental antioxidants used in these studies or differences in metabolic oxidation status of the animals. In the present study, compared to pigs fed diets without supplementation of vitamin E, feeding pigs α-tocopheryl acetate at either NRC (11 mg/kg of feed) or 10X NRC (110 mg/kg of feed) levels from weaning until harvest improved G:F but had no effect on ADG, ADFI, and final BW. Additionally, no differences were observed between 1X-E and 10X-E treatments for G:F. Therefore, increasing the dietary vitamin E concentrations above those recommended by NRC (1998), when feeding a highly Ox-DDGS source, may not be necessary.

In addition to growth performance, feeding high dietary levels of DDGS has been studied extensively relative to pig carcass characteristics. The reduction in HCW and dressing percentage of pigs fed DDGS diets observed in the current study is consistent with responses reported from several previous studies (Fu et al., 2004; Cook et al., 2005; Whitney et al., 2006). Our observation that dietary Ox-DDGS reduced 10th rib backfat depth disagree with some previous studies (Fu et al., 2004; Cook et al., 2005; Whitney et al., 2006), in which backfat depth was unaffected when feeding 30% DDGS to pigs. However, in a more recent study conducted by Xu et al. (2010), using pigs from the same genetic lines as used in the current study, last-rib backfat depth was reduced linearly with increasing dietary DDGS from 0 to 30%. The authors explained that the reduction in backfat depth might be due to the reduction in ADFI, which resulted in a reduction in total daily energy intake and thus provided less energy for fat deposition. In the current study, however, ADFI was not affected by feeding Ox-DDGS, indicating that there may be other reasons for the reduction of backfat.

Feeding thermally oxidized oil or fat to pigs (Luci et al., 2007) alters in vivo lipid metabolism by activating the peroxisome proliferator-activated receptor (**PPARα**), a nuclear receptor that regulates its target genes, such as acyl CoA oxidase, catalase, and carnitine palmitoyltransferase-1. The transcription factor PPARα controls the expression of fatty acid oxidative metabolism (Cabrero et al., 2001), and PPARα activation stimulates catabolic pathways of fatty acids (Desvergne and Wahli, 1999). Therefore, upregulation of the target genes in PPARα involved in the oxidation of fatty acids is expected to enhance β-oxidation capacity in peroxisomes and reduce concentrations of lipids in liver and plasma (Sulzle et al., 2004). Considering these biological relationships, one may reasonably speculate that feeding the Ox-DDGS source may have resulted in increased β-oxidation through the activation of PPARα and thus reduced the amount of fatty acids available for deposition in

adipose tissue. Indeed, Fernández-Dueñas (2009) reported reduction in both 10th-rib and last-rib backfat depth in pigs fed 5% peroxidized corn oil, which indicates that the peroxidized lipid or its components in DDGS, may have led to reduced backfat depth observed in the current study. Further studies focusing on PPARα gene expression in liver and adipose tissue of pigs fed diets containing Ox-DDGS are necessary to better determine if this is a contributing factor to reduced backfat thickness observed in some studies. Regardless of the effect of Ox-DDGS on carcass characteristics, our results are in agreement with Asghar et al. (1991), Cannon et al. (1996), and Waylan et al. (2002), who reported that vitamin E supplementation did not affect carcass characteristics in pigs.

Fatty acid composition in animal tissues depends on 1) de novo fatty acid synthesis and 2) fatty acid composition in the diet (Wiseman and Agunbiade, 1998). Particularly in pigs, de novo fatty acid synthesis and direct deposition of synthesized fatty acids in adipose tissue is inhibited effectively by dietary fat, especially PUFA (Farnworth and Kramer, 1987; Chilliard, 1993). Thus, the fatty acid composition of adipose tissue triacylglycerols, particularly essential PUFA such as C18:2n-6 and C18:3n-3, which cannot be synthesized by animals, is influenced substantially by the composition of dietary fat (Gatlin et al., 2002). Corn DDGS contains about 10% corn oil, which is high in unsaturated fatty acids (81% of total) and C18:2n-6 (54% of total; Xu et al., 2010). In the current study, including 30% Ox-DDGS in the diet increased the concentration of PUFA in belly fat and backfat by 94 and 82%, respectively, and increased the concentration of C18:2n-6 of belly fat and backfat by 99 and 87%, respectively, compared with CON. Our results support previous findings that fatty acid composition can be manipulated by the dietary fat consumed (Gatlin et al., 2002).

Iodine value is a measure of the degree of unsaturation of fatty acids in lipid and is defined as the number of grams of iodine absorbed by 100 g of fat (Xu et al., 2010). Because the concentration of PUFA in belly fat and backfat increased when 30% Ox-DDGS was included in the diets, the IV of belly fat and backfat increased by about 17 and 18%, respectively. Similarly, Xu et al. (2010) reported an increase of about 14 and 11%, respectively, in IV of belly fat and backfat of pigs fed diets containing 0 to 30% DDGS. Although belly firmness was not measured in the present study, the increased IV in pork fat, together with increased concentration of PUFA, indicate that pigs fed Ox-DDGS had reduced belly firmness. No effects of dietary vitamin E level on fatty acid composition or IV of backfat or belly fat were observed in the current study, which is consistent with results reported by Fernández-Dueñas (2009). These authors also reported no effects of dietary antioxidants on fatty acid profile of belly and backfat in pigs fed diets containing peroxidized corn oil.

The susceptibly of PUFA to peroxidation is the main reason for reduced shelf-life stability of meat, increased rancid flavor, and meat discoloration during storage, as observed in several studies when pigs were fed diets with vegetable oil (Leszczynski et al., 1992; Ahn et al., 1996; Leskanich et al., 1997) or DDGS (Leick et al., 2010) that contain high levels of PUFA. Concentrations of α-tocopherol and TBARS in LM were determined in this study to evaluate the effect of feeding diets containing 30% Ox-DDGS on LM peroxidation. The TBARS measurement represents saturated aldehydes (i.e., MDA), 2-enals, and 2-dienals produced in lipid peroxidation (Palmquist and Jenkins, 2003). Measurement of TBARS is one of the most frequently used methods for determining lipid peroxidation because of its simplicity and relatively short assay time. However, unlike using HPLC or liquid chromatography–mass spectrometry, the TBARS value obtained from the color reaction should not be interpreted to represent absolute levels of peroxidation. In the present study, the TBARS value in fresh LM was not different in pigs fed Ox-DDGS compared with CON diets, and pigs fed Ox-DDGS even exhibited about 2 times higher concentrations of α-tocopherol in LM than those fed CON when no additional vitamin E was added to the diets. These results are consistent with results from a recent study conducted by Song et al. (2013), in which serum TBARS values were not different, and serum concentrations of α-tocopherol were greater in pigs of the same genetic source and fed the same source of Ox-DDGS compared with those fed the same CON dietary treatments.

Song et al. (2013) discovered that the Ox-DDGS source used in that study as well as in the current study not only contained the highest amount of peroxidized lipids but also contained a high total S concentration (0.95%). They explained that the unchanged TBARS and increased α-tocopherol concentration could be due to the increase in S-containing antioxidants (glutathione, Met, taurine, and glutathione peroxidase) from feeding the high S DDGS source, which protected against the lipid peroxidation of PUFA and led to a vitamin E-sparing effect. As expected, increasing dietary vitamin E to 10X-NRC increased the α-tocopherol concentration in LM, which is in agreement with results from several studies (Monahan et al., 1990; Leskanich et al., 1997; Boler, 2008). However, because increasing dietary vitamin E levels did not overcome the negative effect on growth performance and carcass characteristics from feeding Ox-DDGS, and it did not change the fatty acid composition in pork fat, it may not be necessary to increase the vitamin E levels greater than those recommended by NRC (1998) when feeding diets containing a high level of Ox-DDGS to pigs from weaning to harvest.

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