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Effect of feeding peroxidized dried distillers grains with solubles to sows and progeny on growth performance and metabolic oxidative status of nursery pigs[1]

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ABSTRACT: This experiment evaluated the effects of including peroxidized corn dried distillers grains with solubles (DDGS) in diets for sows and nursery pigs on growth performance, vitamin E (VE), and Se status, and the incidence of mulberry heart disease (MHD) of nursery pigs. Sows ($n = 12$) were fed corn-soybean meal diets (C-SBM) or C-SBM diets with DDGS (40% and 20% in gestation and lactation, respectively) for 3 parities. In the third parity, 108 weaned pigs (BW = 6.6 ± 0.36 kg) were blocked by BW within litter, assigned to pens (2 pigs/pen; 5 and 4 pens per litter for groups 1 and 2, respectively), and pens were assigned 1 of 3 nursery diets: 1) corn-soybean meal (CON), 2) 30% peroxidized DDGS (Ox-D), and 3) 30% Ox-D with 5 × NRC (1998) level of VE (Ox-D+5VE) for 7 wk, in a 2 × 3 factorial arrangement of sow and nursery diets ($n = 9$ pens/treatment). The peroxidized DDGS source in nursery diets contained concentrations of thiobarbituric acid reactive substances (TBARS) and peroxide values that were 25 and 27 times greater than a reference corn sample. Sow colostrum, milk, and serum, as well as pig serum and liver samples, were analyzed for α-tocopherol and Se concentrations. Pig serum was analyzed for glutathione peroxidase activity (GPx), TBARS, and sulfur-containing AA (SAA). Pig hearts were evaluated for gross and histopathological lesions indicative of MHD, but none were detected. Pigs from sows fed DDGS tended to have reduced ($P = 0.07$) VE in serum during lactation and reduced VE at weaning ($P < 0.01$; 5.6 vs. 6.7 ± 0.1 μg/mL) compared with pigs from sows fed C-SBM. Inclusion of DDGS in sow diets reduced the VE status of pigs during lactation, but not in the nursery when MHD can be a concern. Pigs fed Ox-D+5VE ($P = 0.08$) tended to have, and those fed Ox-D ($P = 0.04$) had greater ADFI than pigs fed CON, but ADG was not affected ($P > 0.1$) by nursery diet. Feeding Ox-D or Ox-D+5VE increased ($P < 0.05$) serum α-tocopherol compared with CON (2.5, 2.8, and 3.4 ± 0.09 μg/mL, respectively), but TBARS and GPx were not affected by nursery diet. Serum concentration of SAA was 40% to 50% greater ($P < 0.01$) for pigs fed Ox-D or Ox-D+5VE compared with those fed C-SBM, which was likely due to greater ($P < 0.01$) SAA intake for pigs fed Ox-D. The antioxidant properties of SAA may have spared VE and Se and masked any effect of Ox-D on metabolic oxidation status. Therefore, increasing the dietary VE concentration was unnecessary in nursery diets containing Ox-D.

Key words: dried distillers grains with solubles, growth performance, Mulberry Heart Disease, pigs, sows, vitamin E

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INTRODUCTION

Lipid peroxidation is a complex process that produces and degrades a variety of toxic compounds associated with cellular damage (Bartosz and Kolakowska, 2011). Peroxidation is accelerated with increasing

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concentrations of unsaturaturated fatty acids in lipids and exposure to heat, light, moisture, and oxygen, but antioxidants impede peroxidation (Belitz et al., 2009). Linoleic acid and other PUFA in corn dried distillers grains with solubles (**DDGS**) can be readily peroxidized depending on temperature and duration of drying (Song and Shurson, 2013). Song and Shurson (2013) reported that DDGS may contain thiobarbituric acid reactive substances (**TBARS**) values and peroxide values (**PV**) more than 25-fold greater than those in corn, but concentrations varied, depending on the source of DDGS.

Dietary inclusion of peroxidized lipids has been shown to reduce growth (DeRouchey et al., 2004; Boler et al., 2012; Liu et al., 2012b) and vitamin E content of serum and tissues (Yuan et al., 2007; Boler et al., 2012; Liu et al., 2012a), potentially impairing the metabolic antioxidant system. A deficiency of vitamin E or Se is associated with mulberry heart disease (**MHD**), which typically results in the sudden death of fast growing nursery pigs (AASV, 2009). Weaver (2010a,b) reported an increased incidence of MHD in the U.S. pork industry and suggested that feeding diets containing DDGS was a contributing factor. Recently, Song et al. (2013) reported improved antioxidant status in pigs fed diets with 30% DDGS, but the impact of including DDGS in sow diets on antioxidant status of their offspring has not been determined.

We hypothesized that feeding high levels of DDGS to sows during gestation and lactation over 3 parities, and subsequently feeding a highly peroxidized source of DDGS to their progeny after weaning, would reduce growth and antioxidant status of nursery pigs leading to an increased incidence of MHD, and that supranutritional supplementation of vitamin E would mitigate these effects.

MATERIALS AND METHODS

The experimental design and procedures of this study were reviewed and approved by the University of Minnesota Institutional Animal Care and Use Committee.

Animal Management

This experiment was conducted at the University of Minnesota Southern Research and Outreach Center Swine Research Facility in Waseca. Two farrowing groups of 6 (n = 12) third-parity sows (Landrace × Yorkshire; Topigs, Winnipeg, Manitoba, Canada) mated to Duroc boars (Compart Boar Store, Nicollet, MN) and their progeny (n = 108) were used. On d 109 of gestation, sows were moved into environmentally controlled farrowing rooms and placed in individual farrowing stalls (2.13 m long × 0.97 m high × 0.66 m wide) with fully slatted floors. Each farrowing stall was equipped with a feeder, heat pad, heat lamp, and nipple waterers for sows and piglets. Piglets were identified with ear notches at birth. When necessary, pigs were cross-fostered within sow dietary treatment before 24 h of age (target litter size = 10 to 11 piglets per sow). Pigs were weaned on d 19.3 ± 1.3 of age and housed in pens (1.2 m × 1.2 m) where they were provided ad libitum access to water and experimental diets for 7 wk.

Dietary Treatments

Sows were fed 1 of 2 gestation and lactation diets: corn-soybean meal diets (**C-SBM**) or diets containing DDGS (40% and 20% in gestation and lactation, respectively) for 3 successive parities (Table 1) as part of a larger experiment (Li et al., 2014). In the third parity, 108 weaned pigs (n = 60 and 48 for groups 1 and 2, respectively), half from each sow dietary treatment were selected, and 2 littermate pigs of similar BW were placed in nursery pens (5 and 4 pens from each litter for groups 1 and 2, respectively). Each pen was assigned to 1 of 3 nursery diets (**ND;** Table 2): 1) Control (**CON**), 2) 30% oxidized DDGS (**Ox-D**), and 3) **Ox-D+5VE** (as Ox-D with 5 × NRC [1998] recommended concentration of vitamin E as dl-α-tocopheryl acetate). This design resulted in a 2 × 3 factorial arrangement of sow diets and nursery diets with 9 replications for each treatment.

Diet Composition

Diet composition and nutrient profiles of sow and nursery diets are presented in Tables 1 and 2, respectively. Diets were formulated to meet or exceed NRC (1998) requirements for sows and nursery pigs and were provided in meal form. Nursery diets were fed over 3 phases with intervals of 1, 2, and 4 wk for phases 1, 2, and 3, respectively. Within each phase, nursery diets were formulated to contain similar concentrations of standardized ileal digestible AA and available P.

The DDGS source used to formulate sow diets contained a concentration of TBARS that was representative of that typically observed among DDGS sources (1.6 ng malondialdehyde [**MDA**] Eq/mg oil; Song and Shurson, 2013). The DDGS source used to formulate nursery diets was selected based on evaluation of 31 corn DDGS sources produced by U.S. ethanol plants (Song and Shurson, 2013) and contained the greatest thiobarbituric acid reactive substances (**TBARS**) value (5.2 ng MDA Eq/mg oil) and peroxide value (**PV;** 84.1 mEq O_2/kg oil) relative to 30 other DDGS sources (mean values = 1.8 ng MDA Eq/mg oil and 11.5 mEq O_2/kg oil, respectively). Coincidentally, this source also contained the highest total S concentration (0.95%) compared with 7 other DDGS sources evaluated (mean = 0.50%).

Sow and Litter Performance

Throughout gestation, sows were fed approximately 2.04 kg/d of their assigned experimental diets, which was adjusted to achieve a body condition score of 3 at farrowing (scale: 1 = thin, 5 = obese; Coffey et al., 1999). Sows were fed 2.25 kg/d of their assigned lactation diet from d 109 of gestation to farrowing. After parturition, feeding level was steadily increased to allow ad libitum consumption from d 5 to 19 of lactation. Sow feed disappearance during lactation, pig mortality, and sow and pig weights at farrowing and weaning were recorded. After weaning, pigs were weighed individually at each dietary phase change, and pen feed disappearance were recorded. These data were used to calculate ADFI, ADG, and G:F of each pen.

Milk, Blood, and Tissue Collection

Colostrum (20 to 30 mL) was collected (without exogenous oxytocin) by manual expression of functional glands during farrowing. On d 7 and d 19 of lactation, milk was collected within 10 min after injection of 20 USP of oxytocin (Bimeda-MTC–Animal Health Inc., Cambridge, Ontario, Canada). Samples were frozen (−20°C) until further analysis.

Focal pigs (at least 2 per litter, total = 27 pigs) were selected from each litter at birth based on the criteria of being the first apparently healthy pigs born regardless of sex. On d 0, blood samples were collected from pigs before initial suckling and from sows (< 24 h postfarrowing) via jugular venipuncture using serum separator vacutainer tubes coated with silicone and micronized silica particles (Becton Dickson, Franklin Lakes, NJ). Blood was again collected from sows and pigs on d 7 and 19. In the nursery, blood samples were obtained from the same focal pigs, but additional focal pigs were selected randomly to achieve 1 focal pig per pen (n = 54 focal pigs). On d 19 (weaning), 47, and 68 of age, blood samples were collected as previously described. After collection, blood was placed on ice, stored at 4°C, and centrifuged at 1,400 × g for 10 min at room temperature. Serum was transferred into microcentrifuge tubes and frozen at −20°C.

Focal pigs were euthanized with sodium pentobarbital (> 100 mg/kg BW) on d 68 to obtain heart and liver samples. Livers were placed immediately on ice and frozen at 20°C. A veterinarian blinded to treatments evaluated each intact heart and assigned a score for gross heart lesions (0 = normal heart; 1 = abnormal heart, without characteristics of MHD; 2 = abnormal heart with mild characteristics of MHD; and 3 = abnormal heart with severe characteristics of MHD). Heart tissue was then excised from right and left ventricles and septum and placed in 10% neutral buffered formalin. Heart sections were trimmed, embedded in paraffin, mounted onto a slide (1 slide/focal pig), and stained with hematoxylin and eosin according to procedures described by Carson and Hladik (2005). A veterinary pathologist, blinded to treatments, evaluated heart sections histologically for lesions characteristic of MHD. Slides were initially scanned at a 100 × magnification and subsequently evaluated at 200 and 400 × for evidence of histologic lesions.

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

	Gestation		Lactation	
Item	C-SBM[1]	DDGS[2]	C-SBM	DDGS
Ingredient, %				
Corn	74.45	54.35	61.55	51.90
Dried distillers grains with solubles	0.00	40.00	0.00	20.00
Soybean meal (46.5% CP)	18.80	0.00	30.00	20.00
Choice white grease	2.00	0.50	3.70	3.00
Dicalcium phosphate	1.90	0.80	2.40	1.90
Limestone	1.40	2.30	1.30	1.80
Salt	0.35	0.35	0.35	0.35
Vitamin mineral premix[3]	0.50	0.50	0.50	0.50
Biotin[4]	0.20	0.20	0.20	0.20
Choline chloride (50%)	0.10	0.20	0.00	0.10
L-Lys HCl	0.00	0.40	0.00	0.20
L-Trp	0.05	0.10	0.00	0.05
L-Thr	0.15	0.20	0.00	0.00
DL-Met	0.10	0.10	0.00	0.00
Total	100.00	100.00	100.00	100.00
Analyzed composition				
ME, kcal/kg[5]	3,341	3,351	3,413	3,417
CP, %	15.7	14.9	17.3	19.3
Crude fat, %	4.9	6.6	6.3	7.5
NDF, %	7.2	13.6	5.3	10.5
ADF, %	3.0	5.0	3.3	4.1
Ca, %	1.3	1.2	1.2	1.3
P, %	0.7	0.6	0.8	0.8
Lys, %	0.8	0.9	0.9	1.0
Met+Cystine, %	0.4	0.5	0.5	0.5
Thr, %	0.6	0.6	0.7	0.7
Trp, %	0.2	0.2	0.2	0.3
S, %	0.2	0.3	0.2	0.3
Se, mg/kg	0.6	0.5	0.6	0.7
Vitamin E, IU/kg	69.0	67.0	65.0	60.0

[1]C-SBM = corn-soybean meal diet.

[2]DDGS = dried distillers grains with solubles diet.

[3]Supplied the following per kilogram of diet: 12,114 IU of vitamin A (retinyl acetate); 2,753 IU of vitamin D (cholecalciferol); 66 IU of vitamin E (dl-**α**-tocopheryl acetate); 4.4 mg of vitamin K; 1 mg thiamine; 10 mg of riboflavin; 55 mg of niacin; 33 mg of pantothenic acid; 2.2 mg of pyridoxine; 1.6 mg of folic acid; 0.06 mg of vitamin B12; 0.5 mg of Iodine (ethylenediamine dihydriodide); 0.3 mg of Se (sodium selenite); 548 mg of choline (chloride); 125 mg of Zn (zinc sulfate, Seaquestra Minerals [SQM; Qualitech, Lakeville, MN]); 125 mg of Fe (iron sulfate, SQM); 40 mg of Mn (manganese sulfate, SQM); and 15 mg of Cu (copper sulfate, SQM).

[4]Supplied 0.51 mg of biotin (JBS United Inc., Sheridan, IN) per kg of diet.

[5]ME values were calculated using NRC (1998) values.

Table 2. Composition of nursery diets (as-fed basis)

	Phase 1 (d 19 to d 25)[1]			Phase 2 (d 26 to d 40)			Phase 3 (d 41 to d 68)		
Item	C[2]	Ox-D[3]	Ox-D +5VE[4]	C	Ox-D	Ox-D +5xE	C	Ox-D	Ox-D +5xE
Ingredient, %									
Corn	43.44	26.42	26.29	63.48	42.67	42.56	67.86	47.52	47.41
Dried distillers grains with solubles	0.00	30.00	30.00	0.00	30.00	30.00	0.00	30.00	30.00
Soybean meal (46.5% CP)	23.48	10.40	10.40	32.00	23.00	23.00	29.00	19.51	19.51
Fish meal, menhaden	10.00	10.00	10.00	0.00	0.00	0.00	0.00	0.00	0.00
Whey powder	20.00	20.00	20.00	0.00	0.00	0.00	0.00	0.00	0.00
Limestone	0.96	1.37	1.37	1.20	1.59	1.59	0.94	1.33	1.33
Monocalcium phosphate	0.68	0.00	0.00	1.44	0.73	0.73	0.96	0.26	0.26
Salt	0.25	0.25	0.25	0.35	0.35	0.35	0.35	0.35	0.35
Vitamin mineral premix[5]	0.50	0.50	0.50	0.50	0.50	0.50	0.50	0.50	0.50
Antibiotic[6]	0.50	0.50	0.50	0.50	0.50	0.50	0.00	0.00	0.00
Zinc oxide	0.05	0.05	0.05	0.00	0.00	0.00	0.00	0.00	0.00
a-tocopheryl acetate[7]	0.03	0.03	0.16	0.03	0.03	0.15	0.03	0.03	0.13
L-Lys HCl	0.03	0.32	0.32	0.31	0.47	0.47	0.24	0.42	0.42
DL-Met	0.06	0.04	0.04	0.09	0.03	0.03	0.06	0.00	0.00
L-Trp	0.00	0.05	0.05	0.01	0.03	0.03	0.00	0.03	0.03
L-Thr	0.02	0.07	0.07	0.10	0.09	0.09	0.07	0.07	0.07
Total	100.00	100.00	100.00	100.00	100.00	100.01	100.01	100.01	100.00
Analyzed composition									
ME, kcal/kg[8]	3,274	3,322	3,318	3,241	3,296	3,292	3,291	3,345	3,342
CP, %	23.3	23.9	24.6	20.6	22.8	23.3	19.3	20.4	21.5
Crude fat, %	2.7	3.7	3.7	2.4	3.5	3.6	2.2	3.9	3.9
NDF, %	4.7	10.6	11.7	8.0	12.2	10.4	6.5	10.3	13.0
ADF, %	2.8	5.2	5.5	2.8	5.7	5.6	2.9	5.5	6.0
Ca, %	1.6	1.5	1.4	1.0	1.1	1.2	0.8	0.6	0.6
P, %	1.0	0.9	0.9	0.7	0.7	0.8	0.6	0.6	0.6
Lys, %	1.38	1.60	1.45	1.18	1.30	1.39	1.25	1.13	1.30
Met+Cystine, %	0.66	0.78	0.75	0.57	0.66	0.66	0.60	0.63	0.66
Thr, %	0.86	0.95	0.95	0.75	0.83	0.84	0.76	0.79	0.80
Trp, %	0.26	0.28	0.28	0.24	0.26	0.27	0.21	0.24	0.20
S, %	0.3	0.6	0.7	0.2	0.5	0.6	0.2	0.4	0.5
Se, mg/kg	0.7	0.7	0.8	0.6	0.6	0.5	0.5	0.4	0.4
Vitamin E, IU/kg	14.0	19.0	60.0	11.0	15.0	67.0	12.0	13.0	44.0

[1]19 d of age = weaning.

[2]CON = corn-soybean meal diets.

[3]Ox-D = diet containing peroxidized dried distillers grains with solubles (thiobarbituric acid reactive substances = 5.2 ng MDA Eq/mg oil and peroxide value = 84.1 mEq O_2/kg oil).

[4]Ox-D+5VE = diet containing peroxidized dried distillers grains with solubles (thiobarbituric acid reactive substances = 5.2 ng MDA Eq/mg oil and peroxide value = 84.1 mEq O_2/kg oil) and 5 × the NRC (1998) recommended level of vitamin E as dl-α-tocopheryl acetate.

[5]Premix supplied the following nutrients per kilogram of diet: 11,023 IU of vitamin A (retinyl acetate); 2,756 IU of vitamin D3; 4.41 mg of vitamin K (menadione dimethylpyrimidinol bisulfite); 9.92 mg of riboflavin; 55.11 mg of niacin; 33.07 mg of pantothenic acid as D-calcium pantothenate; 496.03 mg of choline as choline chloride; 0.06 mg of vitamin B12; 2.20 mg of pyridoxine; 1.65 mg of folic acid; 1.10 mg of thiamine; 0.22 mg of biotin; 2.20 mg of iodine (ethylenediamine dihydroiodide); 0.30 mg of selenium (sodium selenite); 90.39 mg of zinc (zinc oxide, Seaquestra Minerals [SQM®; Qualitech, Lakeville, MN]); 55.11 mg of iron (ferrous sulfate, SQM); 5.51 mg of copper (copper sulfate, SQM); and 17.64 mg of manganese (manganese oxide, SQM).

[6]Mecadox (Carbadox 5.51 g/kg), Phibro Animal Health, Teaneck, NJ.

[7]Concentration: 44,090 IU vitamin E/kg.

[8]ME values were calculated using NRC (1998) values for corn and soybean meal and 3559 kcal/kg was used for DDGS (Pedersen et al., 2007).

Laboratory Analysis

Diet nutrient composition. Feed samples were retained, frozen at −20°C, and analyzed for DM (method 930.15; AOAC, 2005), crude fat (method 920.39; AOAC, 2005), NDF (method 2002.04; AOAC, 2005, and Methods 5.1 and 5.2; NFTA, 1993), ADF (method 973.18; AOAC, 2005, modified according to Tecator Application Note 3429), ash (method 942.05; AOAC, 2008), N (method 990.03; AOAC, 2005), Ca (method 985.01; AOAC, 2005), P (method 985.01; AOAC, 2005), S (method D4239; ASTM, 2011), Se, and

α-tocopherol at Minnesota Valley Testing Laboratories (New Ulm). After digestion in nitric acid, Se was analyzed according to procedures described by Wahlen et al. (2005) using an Inductively Coupled Plasma Mass Spectrometer (model 7500ce; Agilent Technologies Inc., Santa Clara, CA). The α-tocopherol content of feeds was measured using a modified AOAC method (971.30; AOAC, 2006) with high-performance liquid chromatography (**HPLC**) and a fluorescence detector. Amino acids (method 982.30; AOAC, 2006) were analyzed at the University of Missouri Agricultural Experiment Station Chemistry Laboratory (Columbia).

Milk, Liver, and Serum Se and α-tocopherol Concentrations. The Se and α-tocopherol concentrations in sow serum and milk and in pig serum and liver samples were analyzed at the Michigan State University Diagnostic Center for Population and Animal Health (East Lansing). One gram of liver tissue was digested overnight in 2 mL nitric acid, and Se concentrations were determined according to the procedure of Wahlen et al. (2005) using an Inductively Coupled Plasma Mass Spectrometer (model 7500ce; Agilent Technologies Inc., Santa Clara, CA). For α-tocopherol analysis, liver samples were weighed and homogenized in distilled, deionized water (1:4 w/v). Serum samples and liver homogenates were mixed with equal volumes of hexane and a solution of butylated hydroxytoluene in ethanol (10% w/v). Mixtures were centrifuged at 1,900 × g for 10 min, and a known aliquot of the hexane layer was removed and dried under vacuum. Samples were dissolved in a chromatographic mobile phase (7:2:1, acetonitrile, methylene chloride, methanol) and analyzed by HPLC (Separation Module 2690) using an analytical column (Waters Symmetry C18, 3.5 mm, 4.6 × 75 mm) with detection by UV absorbency at 292 nm (Waters, Milford, MA). Trans-β-APO-8"-carotenal was used as an internal standard.

Serum TBARS. Pig serum was analyzed for TBARS concentration according to methods adapted from the Animal Models of Diabetic Complications Consortium (Feldman, 2004). Briefly, 100 μL serum samples and standards of malonaldehyde (catalog number AC14861-1000; Fisher Scientific, Pittsburgh, PA) were mixed with 200 μL ice cold 10% trichloroacetic acid (Sigma-Aldrich, St. Louis, MO) and centrifuged at 12,000 × *g* for 15 min at 4°C. Two hundred microliters of supernatant were removed and incubated with an equal volume of 0.67% (w/v) thiobarbituric acid (Sigma-Aldrich, St. Louis, MO) for 10 min in a dry block heater maintained at 100°C. Each vial was then cooled in an ice bath, and an aliquot was read at 532 nm using a spectrometer (SpectraMax 250; Molecular Devices, Sunnyvale, CA).

Hepatic glutathione (GSH). Total GSH concentrations in liver were analyzed using a commercial kit (catalog number CS0260; Sigma-Aldrich, St. Louis, MO). Fifty milligrams of liver was homogenized in 500 μL of 5% 5-sulfosalcyclic acid followed by centrifugation at 10,000 × *g* for 10 min at 4°C, and duplicate 10-μL aliquots of supernatant and standards were analyzed according to kit instructions. Briefly, GSH was measured indirectly by a coupled reaction with glutathione reductase after adding 5',5'-dithio-bis(2-nitrobenzoic acid) and NADPH. Formation of 2-Nitro-5-thiobenzoic acid was monitored colorimetrically using a spectrometer (SpectraMax 250; Molecular Devices, Sunnyvale, CA) at 412 nm for 5 min.

Serum glutathione Peroxidase Activity. Glutathione peroxidase (**GPx**) activity in pig serum was determined using a commercial kit (catalog number 703102; Cayman Chemical, Ann Arbor, MI). Briefly, GPx activity was measured indirectly by a coupled reaction with GSH reductase after the addition of cumene hydroperoxide to duplicate serum samples and standards. Oxidation of NADPH was measured colorimetrically using a spectrometer (SpectraMax 250; Molecular Devices, Sunnyvale, CA) at 340 nm for 5 min. Glutathione peroxidase activity was expressed as μmol/min/mL of serum and compared to a bovine erythrocyte GPx standard curve.

Serum Sulfur-Containing Amino Acids. Concentrations of Met, Cystine, and Tau were determined in pig serum on d 68 of age by liquid chromatography–mass spectrometry (LC-MS). Briefly, 5 μL of samples and standards were mixed with 5 μL of 100 μ*M* *p*-chlorophenylalanine (internal standard), 50 μL of 10 m*M* sodium carbonate, and 100 μL of dansyl chloride (3 mg/mL in acetone). The mixture was incubated at 25°C for 15 min and centrifuged (18,000 × *g*) for 10 min. Five microliters of supernatant were injected and separated in a column (Acquity BEH C18; Waters, Milford, MA) by a gradient of mobile phase ranging from water to 95% aqueous acetonitrile containing 0.1% formic acid for 10 min. The eluent was introduced into a quadrupole time-of-flight mass spectrometer (SYNAPT; Waters, Milford, MA) for mass detection. Mass chromatograms and spectral data were acquired and processed by MassLynx software (Waters, Milford, MA) in centroid format. The concentrations of Met, Cystine, and Tau in serum were determined by calculating the ratio between their peak areas and the peak area of *p*-chlorophenylalanine using QuanLynx software (Waters, Milford, MA).

Statistical Analyses

The MIXED procedure of SAS (SAS Inst. Inc., Cary, NC) was used to evaluate the effect of sow dietary treatment on the measures obtained during the lactation

Table 3. Main effects of maternal diet (MD) and nursery diet (ND) on growth performance of pigs

	Maternal diet[1]		Nursery diet[2]			PSEM[3]	*P*-value		
Item	C-SBM	DDGS	CON	Ox-D	Ox-D +5VE		MD	ND	MD × ND
Overall[4]									
Weaning wt., kg	6.6	6.7	6.7	6.6	6.6	0.4	0.91	0.64	0.25
Final BW, kg	28.7	29.1	28.2	29.2	29.3	1.4	0.65	0.33	0.07
ADFI, g	794	793	734[a,x]	830[b]	816[y]	82	0.97	0.03	0.44
ADG, g	456	469	456	463	468	28	0.47	0.81	0.28
Gain:Feed	0.58	0.60	0.63[x]	0.57[y]	0.58[x,y]	0.03	0.37	0.06	0.75

[1]C-SBM = corn-soybean meal sow gestation and lactation diets; DDGS = sow gestation and lactation diets contained 20 and 40% dried distillers grains with solubles, respectively.

[2]CON = corn-soybean meal nursery diets; Ox-D = nursery diets containing 30% peroxidized DDGS; Ox-D+5VE = nursery diets containing 30% peroxidized DDGS and 5 times the recommended (NRC, 1998) level of vitamin E as dl-**α**-tocopheryl acetate.

[3]Pooled standard error of the mean.

[4]Over 7 wk nursery period.

[a,b]Within a row, means within an effect without a common superscript differ ($P < 0.05$).

[x,y]Within a row, means within an effect without a common superscript differ ($P \leq 0.1$).

period using farrowing group as a random effect. Sow (or litter) was the experimental unit for measures from the lactation phase, so data collected during the lactation phase were averaged within litter. A separate ANOVA was used to analyze data collected in the nursery period using the MIXED procedure of SAS (SAS Inst. Inc., Cary, NC) in a 2 × 3 factorial arrangement of sow and nursery pig diet in a split-plot design. Sow was the whole plot and nursery pen was the subplot. Pen was used as the experimental unit for data from the nursery phase. Group and nursery BW block were included as random factors in the model. The repeated measures option was used to evaluate the effect of time and its interactions. Normality of model residuals was evaluated using the UNIVARIATE procedure of SAS. The association of gross heart lesion score with dietary treatment was evaluated using chi-square analysis. Results are reported as least squares means. Comparisons were performed using the PDIFF option of SAS with the Tukey–Kramer adjustment. Treatment effects were significant at $P < 0.05$, whereas values between $0.05 \leq P \leq 0.10$ were considered statistical trends.

RESULTS AND DISCUSSION

Six pigs were removed from the experiment for reasons unrelated to dietary treatment. One pig died of intestinal torsion, and 5 pigs were euthanized because of substantial body weight loss resulting from post-weaning feed refusal. Two of these pigs were from DDGS/Ox-D, 2 pigs were from C-SBM/CON, and 2 pigs were from DDGS/CON sow and nursery dietary treatments, respectively. No pigs developed MHD during the course of this experiment, and gross heart lesion score was not associated with dietary treatment.

Growth Performance

A summary of the sow and litter performance results is available elsewhere (Li et al., 2014). The primary focus of the current work was on the antioxidant status and growth of nursery pigs.

Including DDGS in sow diets for 3 reproductive cycles did not affect ADG, ADFI, and gain:feed of nursery pigs (Table 3). Few have investigated the effect of sow diet on the growth performance of progeny, but our results suggest long-term dietary inclusion of DDGS for sows does not affect the growth performance of their offspring in the nursery phase.

The concentration of lipids in DDGS can be as much as threefold greater than corn, and linoleic acid is the predominant fatty acid (NRC, 2012). Linoleic acid and other PUFA are highly susceptible to lipid peroxidation, a process which is accelerated by heat and moisture (Bartosz and Kolakowska, 2011). During drying, DDGS is typically exposed to temperatures as high as 500°C (Rosentrater et al., 2012), but temperatures may vary from 371°C to 593°C (Song and Shurson, 2013). Such extreme and variable temperatures contribute to substantial ranges in the concentrations of lipid peroxidation products in DDGS. Analysis of PV and TBARS in 31 DDGS samples revealed ranges of 4.2 to 84.1 mEq O_2/kg oil and 1.0 to 5.2 ng MDA Eq/kg of oil, respectively (Song and Shurson, 2013). The DDGS source in the nursery diets of the current experiments had the greatest concentrations of PV and TBARS among 31 samples evaluated previously, but the DDGS in sow diets had intermediate concentrations of these commonly used indicators of peroxidation. Feeding lipids containing peroxidation compounds has been shown to cause damage to organs and cells (Griffiths, 2005; Bartosz and Kolakowska, 2011) and also to contribute to a

metabolic deficiency of antioxidants known to cause oxidative stress (Lykkesfeldt and Svendsen, 2007).

When dietary peroxidized lipids are fed to swine (DeRouchey et al., 2004; Boler et al., 2012; Liu et al., 2012b), poultry (Dibner et al., 1996), and rats (Liu and Huang, 1995; Eder, 1999), ADG often declines. Conversely, we observed no negative effects of Ox-D in nursery diets on ADG. DeRouchey et al. (2004) suggested a threshold of 2.4 mEq O_2/kg diet, above which growth performance is compromised. In the current experiment, nursery diets contained 1.7 mEq O_2/kg of diet (30% inclusion of DDGS [6.9% crude fat] with 84.1 mEq O_2/kg of oil). Therefore, it is possible that our nursery diets had insufficient concentrations of peroxidation products to reduce pig growth. Furthermore, other researchers evaluated concentrated lipid sources (e.g., corn oil, fish oil, canola oil) containing peroxidation products (DeRouchey et al., 2004; Boler et al., 2012; Liu et al., 2012b), but lipids represent < 12% of the total chemical composition in DDGS. Our findings are in agreement with others indicating that dietary DDGS does not affect ADG (Whitney and Shurson, 2004; Spencer et al., 2007; Barbosa et al., 2008), but our results are not directly comparable to the results from these studies because they fed transition diets without DDGS for 1 to 3 wk after weaning. Our experimental diets were first offered on the day after weaning. Pigs fed Ox-D had greater ($P = 0.04$) ADFI compared with pigs fed CON, and pigs fed Ox-D+5VE tended to have ($P = 0.08$) greater ADFI than those fed CON (Table 3). Therefore, gain:feed tended to improve ($P = 0.08$) by feeding CON compared to Ox-D, but not ($P > 0.1$) Ox-D+5VE. The effect of feeding diets containing DDGS on ADFI in the current study was likely related to overestimation of the ME content of the DDGS for nursery pigs because ADG was not affected. Many factors are involved in the production of DDGS and vary among ethanol plants, contributing to variable energy content of DDGS (Shurson and Alhamdi, 2008). Dietary energy density is related inversely to feed intake (Ellis and Augspurger, 2001). Furthermore, fibrous feedstuffs like DDGS contribute to expansion of the gastrointestinal tract and increased feed intake (Pond et al., 1988). In the current study, nursery diets containing Ox-D had 70% higher concentrations of NDF (mean weighted by duration of phase) than CON. Therefore, in the current study, increased ADFI for pigs fed DDGS may be related to caloric density and fiber content.

Metabolic Oxidative Balance

Compared to saturated fat sources, lipids containing high concentrations of PUFA increase the metabolic demand for vitamin E in swine (Ullrey, 1981) and other species (Horwitt, 1960; Harris and Embree, 1963) because of the increased potential for peroxidation or reduced absorption of vitamin E (Hollander, 1981). Research results suggest that dietary sources of PUFA like corn oil (Malm et al., 1976) and fish oil (Hidiroglou et al., 1993; Farnworth et al., 1995) reduce the α-tocopherol concentrations in serum of sows and fetuses compared to other dietary lipids. Concentrations of α-tocopherol in sow serum or milk were not affected by dietary inclusion of DDGS for sows ($P > 0.1$; Table 4) in the current study. However, pigs suckling sows fed DDGS tended to have reduced ($P = 0.07$) α-tocopherol (Table 4) concentration in serum during lactation compared with those from sows fed C-SBM. The calculated linoleic acid content was 11% (gestation) and 6% (lactation) greater for DDGS diets compared to C-SBM, indicating that PUFA content was greater in DDGS diets. Furthermore, sow diets containing DDGS had 3% to 8% less analyzed vitamin E than C-SBM. Research results reported by Mahan et al. (2000) suggest that dietary vitamin E concentrations for sows affects the α-tocopherol content of pig serum. Therefore, in the current study, the effects of DDGS in sow diets on the vitamin E status of pigs was likely related to the greater dietary PUFA and reduced dietary vitamin E content compared with those fed C-SBM. Others have reported no effect of feeding DDGS on the vitamin E content of sow milk and plasma (Crowder and Johnston, 2011).

Selenium is a component of GPx, which is an integral enzyme for metabolic antioxidant defense. Sows fed DDGS had reduced ($P = 0.05$) concentrations of Se in serum compared to C-SBM. The Se content of colostrum from sows fed DDGS was reduced ($P < 0.05$) compared to sows fed C-SBM, and this difference existed regardless of sampling day ($P < 0.05$; Table 4). However, analyzed Se concentration of sow diets was not equal across treatments in the present study. Gestation diets containing DDGS had 18% lower analyzed Se concentration compared to C-SBM diets. While feed disappearance was not recorded during gestation, sows were projected to receive similar daily allowances of their ration, regardless of dietary treatment. Perhaps the reduced dietary Se concentration in gestation contributed to the 25% reduction in the Se content of colostrum. Others have shown that the dietary concentration of Se in late gestation is positively associated with the concentration of Se in colostrum, milk, and serum (Mahan, 2000). Similarly, during lactation, the Se content of serum from pigs from sows fed DDGS was reduced ($P < 0.001$) relative to pigs from sows fed C-SBM (Table 4), and this may be related to milk concentration of Se. Sow diet did not affect ($P > 0.1$) GPx activity in serum of nursing pigs (data not shown). Therefore, differences in the content of Se

Table 4. The effect of maternal diet (MD) and MD × day interactions on metabolites in serum and milk from sows and serum from nursing pigs[1]

	Maternal diet[2]			*P*-value	
Item	C-SBM	DDGS	PSEM[3]	MD	MD × d
Sow serum α-tocopherol, ug/mL[4]					
Farrowing[a]	1.51	1.35	0.26		
d 7[b]	2.53	2.55	0.26		
Weaning[b]	2.95	2.87	0.26		
Mean[5]	2.27	2.17	0.15	0.62	0.87
Milk α-tocopherol, ug/mL[4]					
Farrowing[a]	5.13	5.01	1.00		
d 7[b]	3.25	1.75	1.00		
Weaning[b]	1.76	1.63	1.00		
Mean	3.23	2.60	0.58	0.41	0.66
Pig serum α-tocopherol, ug/mL[4]					
Farrowing[a]	0.41	0.36	0.49		
d 7[b]	6.39	5.28	0.49		
Weaning[b]	6.47	5.43	0.49		
Mean	4.42	3.69	0.31	0.07	0.46
Sow serum Se, ppm[4]					
Farrowing[a]	0.21	0.19	0.01		
d 7[b]	0.24	0.22	0.01		
Weaning[b]	0.24	0.23	0.01		
Mean	0.23	0.21	0.01	0.05	0.86
Milk Se, ppm[4]					
Farrowing[a]	0.40[d]	0.30[e]	0.008		
d 7[b]	0.11	0.09	0.008		
Weaning[b]	0.11	0.09	0.008		
Mean	0.20[d]	0.16[e]	0.004	< 0.01	< 0.01
Pig serum Se, ppm[4]					
Farrowing[a]	0.07	0.06	0.003		
d 7[b]	0.10	0.09	0.003		
Weaning[c]	0.12	0.11	0.003		
Mean	0.094[d]	0.09[e]	0.002	< 0.01	0.85
Pig serum thiobarbituric acid reactive substances, μ*M* malondialdehyde Eq[4]					
Farrowing[a]	8.96	6.49	1.23		
d 7[b]	1.22	1.39	1.23		
Weaning[b]	0.66	1.17	1.28		
Mean	3.62	3.02	0.83	0.53	0.37

[1]Values represent the least squares means of 6 litters/dietary treatment.

[2]C-SBM = corn-soybean meal sow diets; DDGS = sow gestation and lactation diets containing 40 and 20% DDGS, respectively.

[3]Pooled standard error of the mean.

[4]Time effect ($P < 0.05$).

[5]Main effect mean of maternal diet, regardless of day.

[a–c]Within each variable, means for a sample day without a common superscript differ ($P < 0.05$).

[d,e]Within a row, means without a common superscript differ ($P < 0.05$).

in milk or serum from sows in this study were likely affected by daily consumption of Se during gestation rather than dietary inclusion of DDGS. In support of this hypothesis, others have reported no effect of feeding DDGS on the Se content of sow milk and plasma (Crowder and Johnston, 2011). The concentrations of α-tocopherol and Se increased ($P < 0.05$) in serum and decreased ($P < 0.05$) in milk after parturition (Table 4). Similar results have been reported by others for Se (Mahan, 1994, 2000) and vitamin E (Loudenslager et al., 1986; Mahan, 2000; Mahan et al., 2000). In the current study, diminished demand for vitamin E and Se for lactogenesis in late lactation may have led to increased concentrations of these nutrients in sow serum. Despite declining concentrations in milk, α-tocopherol and Se concentrations increased ($P < 0.05$) in pig serum after farrowing (Table 4), and this response is consistent with other reports (Loudenslager et al., 1986; Mahan, 1991) where this increase is likely related to low reserves of vitamin E of pigs at birth (Mahan and Vallet, 1997).

The TBARS assay measures MDA, a highly reactive product of lipid peroxidation and commonly used as an indicator of metabolic oxidative stress (Griffiths, 2005). Interestingly, we found no influence of sow diet on the TBARS in serum from nursing pigs despite a 17% reduction in the α-tocopherol content of pig serum when feeding DDGS diets to sows (Table 4). Therefore, our results suggest adding DDGS to sow diets does not induce oxidative stress in nursing pigs.

Oxidative stress can be of great concern in nursery pigs because they may develop antioxidant deficiency signs, such as those observed for MHD, during the initial wk postweaning (Ullrey, 1981). However, no signs of MHD were observed in this experiment. Pallarés et al. (2002) suggested that several factors predispose pigs to developing MHD such as stress, genetics, and pathogenic infection. However, there were no known pathogenic challenges, and environmental stressors were minimal in the current study. Serum concentrations of α-tocopherol declined by 82% in pigs after the first month postweaning (effect of time, $P < 0.05$; Table 5), which is consistent with results reported by others showing reduced circulating vitamin E concentrations within the first few wk postweaning (Chung et al., 1992; Moreira and Mahan, 2002; Lauridsen, 2010). These changes are likely related to metabolic and environmental changes associated with weaning. For example, dl-α-tocopheryl acetate is commonly supplemented in nursery diets, but d-α-tocopherol is the predominant form of vitamin E in sow milk (Mahan, 1994; Lauridsen and Jensen, 2007). Chung et al. (1992) reported that supplementing d-α-tocopherol improved retention of serum vitamin E postweaning compared to dl-α-tocopheryl acetate, suggesting greater bioavailability of the alcohol form. Varying bioavailability among vitamin E sources is related to inefficient removal of the acetate group by carboxyl ester hydrolase before absorption in the intestinal lumen (Chung et al., 1992) because the activity of carboxyl ester hydrolase declines after weaning (Jensen et al., 1997). Furthermore, stressors at weaning may

Table 5. Effect of maternal diet (MD) × nursery diet (ND) interactions over time on Se and thiobarbituric acid reactive substances (TBARS) in serum from nursery pigs[1]

	Maternal diet[2]: C-SBM			Maternal diet: DDGS				*P*-value				
Nursery diet[3]	CON	Ox-D	Ox-D +5xE	CON	Ox-D	Ox-D + 5VE	PSEM[4]	MD	ND	MD × d	ND × d	MD × ND
Item												
Pig serum α-tocopherol, ug/mL[5,6]												
Wean, d 19[a]	6.48[d]	6.52[d]	6.96[d]	5.42[e]	6.08[d,e]	5.37[e]	0.19					
d 47[b]	0.96	0.90	1.35	0.81	0.95	1.49	0.18					
d 68[c]	0.91	1.29	2.57	0.69	1.28	2.58	0.18					
Mean[7]	2.78[d]	2.90[e]	3.62[f]	2.30[d]	2.77[e]	3.15[f]	0.12	0.02	< 0.01	< 0.01[8]	< 0.01[9]	0.12
Pig serum Se, ppm[5]												
Wean, d 19[a]	0.115	0.115	0.105	0.105	0.108	0.103	0.006					
d 47[b]	0.149	0.148	0.136	0.135	0.156	0.133	0.006					
d 68[c]	0.163	0.151	0.156	0.164	0.168	0.156	0.006					
Mean	0.142[d,e]	0.138[d,e]	0.132[d,e]	0.135[d,e]	0.144[d]	0.131[e]	0.005	0.84	< 0.01	0.10	0.27	0.09
Pig serum TBARS, μ*M* malondialdehyde Eq[5]												
Wean, d 19[a]	0.68	0.85	1.09	1.38	0.93	0.74	0.14					
d 47[b]	0.53	0.73	0.58	0.75	0.66	0.71	0.15					
d 68[b]	0.64	0.67	0.73	0.94	0.76	0.80	0.15					
Mean	0.61[d]	0.75[d,e]	0.80[d,e]	1.02[e]	0.78[d,e]	0.75[d,e]	0.10	0.19	0.80	0.92	0.88	0.01

[1]Values represent the least squares means of 9 pens/dietary treatment.

[2]C-SBM = corn-soybean meal sow diets; DDGS = sow gestation and lactation diets containing 40 and 20% DDGS, respectively.

[3]CON = corn-soybean meal nursery diets; Ox-D = nursery diets containing 30% peroxidized DDGS; Ox-D+5VE = nursery diets containing 30% peroxidized DDGS and 5 times the recommended (NRC, 1998) level of vitamin E as dl-**α**-tocopheryl acetate.

[4]Pooled standard error of the mean.

[5]Time effect ($P < 0.05$).

[6]MD × ND × day ($P = 0.08$).

[7]Interactive mean of MD × ND.

[8]C-SBM > DDGS on d 19 ($P < 0.05$).

[9]Ox-D + 5xE > Ox-D and CON on d 47 and d 68 ($P < 0.05$). Ox-D > CON on d 68 ($P < 0.1$).

[a–c]Within a variable, means for a sample day without a common superscript differ ($P < 0.05$).

[d–f]Within a row, means without a common superscript differ ($P < 0.05$).

contribute to the postweaning decline in vitamin E status. Recently, Zhu et al. (2012) reported greater MDA content of serum from pigs 2 wk after weaning compared with pigs that were not weaned, but vitamin E concentrations of serum were not reported. Therefore, a combination of factors (i.e., reduced bioavailability and metabolic and environmental changes) may contribute to declining concentrations of vitamin E in pig serum postweaning.

Dietary peroxidized lipids have been shown to reduce serum vitamin E (Eder, 1999; Boler et al., 2012; Liu et al., 2012a) and increase TBARS content (Yuan et al., 2007; Boler et al., 2012; Liu et al., 2012a) compared with feeding diets containing unperoxidized lipids. At weaning, pigs from sows fed DDGS had reduced ($P < 0.05$) α-tocopherol concentrations in serum compared to those from sows fed C-SBM (5.6 vs. 6.7 ± 0.12 μg/mL; Table 5), but not when pigs were fed Ox-D (maternal diet × nursery diet × day; $P < 0.08$). The effect of sow diet on the α-tocopherol status of the pig at weaning is likely related to its concentration in milk. At d 47 and d 68 of age, pigs fed Ox-D+5VE had greater ($P < 0.05$) α-tocopherol concentration in serum than those fed other nursery diets (Table 5). Furthermore, at d 68 of age, pigs fed Ox-D tended to have greater ($P = 0.08$) α-tocopherol concentration in serum compared than those fed CON. Regardless of sampling day, concentrations of α-tocopherol in serum differed ($P < 0.05$) for each nursery diet (2.5, 2.8, and 3.4 ± 0.09 μg/mL for CON, Ox-D, and Ox-D+5VE, respectively; Table 5). Our results agree with those of others who have reported increased serum α-tocopherol with increased dietary vitamin E content (Chung et al., 1992).

Among pigs from sows fed DDGS, pigs fed the Ox-D+5VE nursery diets had reduced ($P < 0.05$) serum Se concentrations compared with those fed Ox-D (maternal diet × nursery diet; $P = 0.09$; Table 5). Regardless of sow diet, pigs fed Ox-D+5VE had reduced ($P < 0.05$) Se concentrations in serum compared with those fed other nursery diets (Table 5). Inclusion of DDGS in sow diets increased ($P < 0.05$) the concentration of serum TBARS in pigs fed CON nursery diets compared with pigs from sows fed C-SBM (maternal diet × nursery diet interaction; $P = 0.01$; Table 5).

Table 6. Effect of maternal diet (MD) and nursery diet (ND) on the concentration of α-tocopherol, Se, and glutathione (GSH) in liver, and the concentration of amino acids in serum from nursery pigs[1]

	Maternal Diet[2]		Nursery Diet[3]				*P*-value		
Item	C-SBM	DDGS	CON	Ox-D	Ox-D+5VE	PSEM[4]	MD	ND	MD × ND
Serum amino acids, umol/L[5]									
Cystathione	5.4	5.8	5.2[a]	5.6[a,b]	6.2[b]	0.4	0.23	0.03	0.31
Cystine	2.5	2.9	2.4	2.8	2.8	0.9	0.13	0.10	0.08
Met	58.8	54.6	48.3[x]	59.1[x,y]	62.6[y]	5.5	0.49	0.09	0.27
Tau	234.4	278.6	198.6[a]	256.2[a,b]	314.6[b]	57.0	0.23	< 0.01	0.10
Total sulfur amino acids[6]	315.3	343.0	247.6[a]	343.3[b]	396.6[b]	49.6	0.50	< 0.01	0.02
Liver[5,7]									
α-Tocopherol, ug/g	3.5	3.2	2.1[a]	2.6[a]	5.2[b]	0.2	0.27	< 0.01	0.53
Se, ug/g	0.6	0.6	0.64[a]	0.50[b]	0.58[a,b]	0.05	0.84	0.02	0.20
Total GSH, nmol/g	97.8	99.6	94.4[x,y]	87.7[x]	114.1[y]	8.2	0.86	0.09	0.50

[1]Values represent the least squares means of 27 pens/maternal dietary treatment and 18 pens/nursery dietary treatment.

[2]C-SBM = corn-soybean meal sow diets; DDGS = sow gestation and lactation diets containing 40 and 20% distillers dried grains with solubles, respectively.

[3]CON = corn-soybean meal nursery diets; Ox-D = nursery diets containing 30% peroxidized DDGS; Ox-D+5VE = nursery diets containing 30% peroxidized DDGS and 5 times the recommended (NRC, 1998) level of vitamin E as dl-**α**-tocopheryl acetate.

[4]Pooled standard error of the mean.

[5]Final sample collected at 68 d of age.

[6]Sum of Cystathione, Cystine, Met, and Tau.

[7]Liver data are on "as-is" basis.

[a,b]Within a row, means within an effect without a common superscript differ ($P < 0.05$).

[x,y]Within a row, means within an effect without a common superscript differ ($P \leq 0.1$).

Therefore, feeding diets with Ox-D did not induce metabolic oxidative stress in nursery pigs in the current study. Other researchers have reported that feeding diets containing DDGS did not increase circulating concentrations of TBARS in pigs (Weber and Kerr, 2011; Song et al., 2013) or broilers (Heincinger et al., 2011), which is in agreement with results of the current study. Our results agree with those reported by Song et al. (2013), who reported that feeding a DDGS source with high concentrations of peroxidized lipids and S increased vitamin E status of pigs. They concluded that sulfur-containing compounds present in DDGS may have provided antioxidant benefits to overcome the potential negative effects contributed from peroxidized lipids.

Sulfur-containing AA have metabolic antioxidant properties. Methionine is a precursor of Cys, which can be used to synthesize GSH or Tau. Sulfur-containing amino acids (**SAA**) counteract oxidative stress by acting as reducing agents (Atmaca, 2004). Pigs fed Ox-D or Ox-D+5VE had increased ($P < 0.05$) concentrations of sulfur-containing amino acids (sum of cystathione, Cys, Met, and Tau) in serum compared with those fed CON (Table 6). Pigs fed diets containing Ox-D or Ox-D+5VE had 30% greater ($P < 0.05$) intake of Met+Cys (calculated using ADFI × mean dietary concentration of Met+Cys across phases) compared with those fed CON (4.4, 5.6, 5.6 ± 0.6 g/d for C, Ox-D, and Ox-D+5VE, respectively). In addition to SAA, corn contains relatively high concentrations of phenolic compounds (e.g., ferulic acid) that have significant antioxidant properties (Adom and Liu, 2002) and are likely concentrated by threefold in DDGS. Furthermore, the Ox-D nursery diet in the current study contained 22% higher concentrations of vitamin E than CON (mean weighted by duration of phase), allowing for increased vitamin E intake. Consequently, we suggest a cumulative effect of greater intake of SAA, vitamin E, and other antioxidant compounds present in DDGS counteracted the negative impacts of peroxidized lipids in DDGS.

Sow diet did not affect α-tocopherol, Se, or total GSH content of pig liver (Table 6). Pigs fed Ox-D+5VE had greater ($P < 0.05$) α-tocopherol concentration in liver than those fed other nursery diets. Other researchers have shown that dietary vitamin E concentrations correspond with α-tocopherol content of liver (Chung et al., 1992). The concentration of Se in liver was reduced ($P < 0.05$) by 22% in pigs fed Ox-D compared to CON, but this effect was mitigated by feeding Ox-D+5VE. However, this effect may not be entirely explained by the dietary Se content, which was only 12% less (mean weighted by duration of phase) for the Ox-D diets compared to CON. The unequal magnitude of differences suggests that some nutrients in Ox-D may have impaired Se availability. Total S concentration of diets containing Ox-D was twice that of CON. Sulfuric acid is used during the ethanol and DDGS production process and contributes to the total S content in DDGS

(Rosentrater et al., 2012). Dietary sodium and potassium sulfate can partially alleviate depressed growth associated with feeding high (≥ 5 ppm) concentrations of Se to rats, suggesting that excess S can reduce Se absorption and utilization (Halverson and Monty, 1960; Ganther and Baumann, 1962; Ardüser et al., 1985). Some researchers have suggested that this antagonistic relationship exists only with selenate (Halverson et al., 1962; Ardüser et al., 1985), but others have also shown it to occur with selenite (Halverson and Monty, 1960; Ganther and Baumann, 1962). Sodium selenite was added at the same concentration (0.3 ppm) to all experimental diets in the current study, and it is unknown if antagonism exists between S and Se in swine when Se is supplemented at this concentration.

In summary, the antioxidant status of pigs during the postweaning period was not reduced with the inclusion of DDGS in diets fed to nursery pigs or sows. In our experiment, feeding moderately peroxidized DDGS to sows and highly peroxidized, high-S DDGS to nursery pigs did not contribute to MHD. These findings imply that sulfur-containing AA or other inherent antioxidant compounds present in DDGS masked any negative impacts of peroxidation products and eliminated the need for additional supplemental vitamin E in nursery diets.

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