Influence of thermally oxidized vegetable oils and animal fats on intestinal barrier function and immune variables in young pigs

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ABSTRACT: To evaluate the effect of feeding thermally oxidized lipids on metabolic oxidative status, gut barrier function, and immune response of young pigs, 108 barrows (6.67 ± 0.03 kg BW) were assigned to 12 dietary treatments in a 4 × 3 factorial arrangement in addition to a corn–soybean meal control diet. Main effects were 4 lipid sources (corn oil [CN], canola oil [CA], poultry fat [PF], and tallow [TL]) and 3 oxidation levels (original lipids [OL], slow oxidation [SO] of lipids heated for 72 h at 95°C, or rapid oxidation [RO] of lipids heated for 7 h at 185°C). Pigs were provided ad libitum access to diets for 28 d followed by controlled feed intake for 10 d. After a 24-h fast on d 38, serum was collected and analyzed for α-tocopherol (α-T), thiobarbituric acid reactive substances (TBARS), endotoxin, haptoglobin, IgA, and IgG. On the same day following serum collection, lactulose and mannitol were fed and subsequently measured in the urine to evaluate gut permeability. There was a source × peroxidation interaction for serum α-T concentration where pigs fed SO or RO had decreased (P < 0.05) serum α-T concentration compared with pigs fed OL in CA and CN diets but not in pigs fed PF and TL diets. There was no source × peroxidation interaction for serum TBARS, but among all lipid sources, pigs fed SO or RO lipids had increased (P < 0.05) serum TBARS compared with pigs fed OL. In addition, pigs fed CN or CA had greater (P < 0.05) serum TBARS compared with pigs fed PF or TL diets. There were no lipid source × peroxidation level interaction or lipid source or peroxidation level effects on serum endotoxin, haptoglobin, IgA, or IgG. Pigs fed lipid supplemented diets tended to have increased serum endotoxin (P = 0.06), IgA (P = 0.10), and IgG (P = 0.09) compared with pigs fed the control diet. There were no lipid source × peroxidation level interaction or lipid source or peroxidation level effects on urinary TBARS and lactulose to mannitol ratio. Compared with pigs fed the control diet, pigs fed diets containing lipids had a lower lactulose to mannitol ratio (P < 0.01). In conclusion, feeding weaning pigs diets containing 10% thermally oxidized lipids for 38 d, especially vegetable oils containing greater concentrations of PUFA, appeared to impair oxidative status but had little influence on gut barrier function or serum immunity parameters.

Key words: gut barrier function, immunity, oxidative stress, thermally oxidized lipids, young pigs

INTRODUCTION

Lipids produced from food processing facilities or restaurants have been thermally processed and are used commonly as economical sources of energy in animal feeds (Canakci, 2007). However, these lipids may be heated for a considerable length of time (Frankel et al., 1984) and may therefore contain various amounts of peroxidation products (Lin et al., 1989; Adam et al., 2008). Kimura et al. (1984) reported that feeding oxidized soybean oil impaired growth performance and caused diarrhea in rats. Alexander et al. (1987) and Behniwal et al. (1993) also reported that rats fed diets
containing oxidized corn or peanut oil impaired growth rate. Similarly, broilers fed oxidized poultry fat exhibited impaired growth rates (Cabel et al., 1988; Dibner et al., 1996), while DeRouchey et al. (2004) observed reduced growth rates in pigs consuming rancid choice white grease. The biological mechanisms to explain these observations are largely unknown.

Two lipid peroxidation products, malondialdehyde (MDA) and 4-hydroxynonenal (HNE), increase metabolic peroxidation in animals (Seppanen and Csallany, 2002; Uchida, 2003). Feeding oxidized sunflower oil increased markers of oxidative stress in the small intestine of pigs (Ringseis et al., 2007) and feeding oxidized poultry fat to broilers decreased intestinal villus length (Dibner et al., 1996). Feeding peroxidized lipids or treating cells with specific lipid peroxidation products decreased ex vivo primary antibody production to a bacterial pathogen (Takahashi and Akiba, 1999) and activated stress pathways (Biasi et al., 2006; Yun et al., 2009). However, little information has been reported regarding the effect of feeding peroxidized lipids on intestinal health or immune function in pigs. The following study was conducted to investigate the effect of feeding diets containing thermally oxidized lipids on metabolic oxidation status, gut barrier function, and immune response in young pigs.

MATERIALS AND METHODS

All animal use procedures were reviewed and approved by the University of Minnesota Institutional Animal Care and Use Committee.

Animals, Experimental Design, and Diets

General procedures regarding lipid peroxidation, diet formulation, and animal management have been described previously (Liu et al., 2014a,b). In brief, 2 or 3 pigs from the same dietary treatment were housed in a single pen with ad libitum access to feed and water for 28 d. From d 29 to 39, pigs were housed individually in metabolism crates and fed an amount of diet equivalent to 4% of their BW daily (2% at 0700 h and 2% at 1900 h), with ad libitum access to water.

After the morning feeding at 0700 h on d 37, all pigs were fasted for 24 h. A blood and urine sample was collected at 0700 h of d 38 to obtain serum and urine from fasted animals. Approximately 8 mL of blood was obtained by jugular venipuncture using a 10-mL serum tube (Becton Dickinson, Franklin Lakes, NJ). Blood samples were centrifuged (Heraeus Biofuge 22R Centrifuge; ThermoScientific, Hanau, Germany) at 2,500 × g for 15 min at 4°C and serum was harvested. Serum samples were frozen immediately and stored at −20°C for subsequent serum α-tocopherol (α-T) and thiobarbituric acid reactive substances (TBARS) analysis and endotoxin, haptoglobin, IgG, and IgA analysis. Immediately following bleeding, chlorhexidine (Bimeda, Inc., Oakbrook Terrace, IL) was placed into each plastic urine collection container to prevent microbial growth, after which the plastic containers were placed under each metabolism crate to collect approximately 200 mL of urine from each pig for the subsequent 4 h. After collection, all urine samples were stored in a freezer at −20°C for subsequent analysis. At 1200 h of d 38, after the net 29-h fast, each pig was fed 100 g of their assigned experimental diet, which contained 10 g of lactulose (Sigma, St. Louis, MO) and 2 g of mannitol (Sigma) as markers of intestinal permeability. Plastic containers containing 2 mL chlorhexidine (Bimeda, Inc.) were again placed under the funnel of each metabolism cage for a period of 6 h after the feeding for urine collection. After urine was collected, it was stored at −20°C for subsequent lactulose and mannitol analysis. Pigs were then fed their normal daily allotment before harvesting the next morning. At 0700 h of d 39, all pigs were euthanized with 1 mL pentobarbital sodium solution (Fatal-Plus Powder; Vortech Pharmaceuticals, Dearborn, MI) per 4.53 kg of BW by intracardiac injection. The spleen was excised and weighed to calculate its weight as a percentage of BW.

Serum and Urine Sample Analysis

Serum α-T concentration was analyzed (method 996.06; AOAC, 2001) at a commercial laboratory (Michigan State University Diagnostic Center for Population and Animal Health, Lansing, MI). Serum and urine TBARS concentrations were analyzed using Animal Models of Diabetic Complications Consortium protocols by Feldman (2004). Serum endotoxin concentration was measured using methodology previously used to evaluate serum endotoxin in growing pigs (Mani et al., 2013; PyroGene Recombinant Factor C Endotoxin Detection System; Lonza, Walkersville, MD). The concentration of serum haptoglobin, a major acute phase protein in pigs, was measured by a colorimetric assay (Phase Haptoglobin Assay; Tridelta Development Limited, Kildare, UK). Serum IgA and IgG were determined using commercial ELISA kits (E100-102 for IgA and E100-104 for IgG; Bethyl Laboratories Inc., Montgomery, TX) following the product instructions. Lactulose and mannitol concentrations in urine were determined by HPLC and the ratio of lactulose and mannitol was used as an in vivo indicator of small intestinal permeability according to the method described by Kansagra et al. (2003).
Thermally oxidized lipids in diets for pigs

**Statistical Analysis**

All data were analyzed using the MIXED procedure of SAS (version 9.2; SAS Inst. Inc., Cary, NC). A 2-way ANOVA was conducted to evaluate the main effects of lipid source (corn oil [CN], canola oil [CA], poultry fat [PF], and tallow [TL]), lipid peroxidation level (original lipids [OL], slow oxidation [SO], and rapid oxidation [RO]), and any 2-way interactions in a 4 × 3 factorial arrangement. The corresponding statistical model included the fixed effects of lipid source, peroxidation level, and lipid source × peroxidation level interactions. Group was included as a random effect. Individual pig was used as the experimental unit for all responses. All results are reported as least squares means. Mean comparisons were achieved by the PDIF option of SAS with the Tukey-Kramer adjustment. In addition, relationships between measures of lipid peroxidation and various variables were evaluated by simple linear correlation (Pearson correlation coefficients) analysis. Treatment effects were considered significant if \( P < 0.05 \), whereas values of \( 0.05 \leq P \leq 0.10 \) were considered statistical trends.

**RESULTS**

**Characteristics of Experimental Lipids**

The concentration of crude fat, moisture, insolubles, and unsaponifiables were similar among the 12 experimental lipids. As expected, CN and CA had greater concentrations of unsaturated fatty acids than TL, with PF being intermediate. Averaged among lipid sources, both SO and RO of lipids decreased the linoleic acid and linolenic acid concentrations compared with the OL, but changes in concentration of other major fatty acids were not observed. Lipid peroxidation tests indicated that all of the OL were relatively unoxidized, but SO and RO of lipids led to a marked increase in the production of primary and secondary peroxidation products, and the production of these peroxidation products caused by SO and RO in CN and CA was much greater than that in PF and TL.

**Metabolic Oxidative Status**

A lipid source × peroxidation level interaction (\( P < 0.01 \)) was observed for serum α-T concentration (Table 1), which was mainly caused by pigs fed CN and CA that were SO or RO, resulting in decreased serum α-T concentration, but not in pigs fed PF or TL that were SO or RO. In addition, pigs fed the control diet had a greater serum α-T concentration compared with pigs fed lipid diets (\( P < 0.05 \)). In contrast, no lipid source × peroxidation level interaction was observed for serum TBARS. Peroxidation level affected serum TBARS (\( P < 0.05 \)), where pigs fed SO or RO lipids had greater serum TBARS than pigs fed diets containing OL (\( P < 0.05 \)). Lipid source also influenced serum TBARS (\( P < 0.05 \)), and pigs fed either CN or CA supplemented diets had a greater serum TBARS than pigs fed PF or TL (\( P < 0.05 \)). There was no lipid source × peroxidation level interaction or lipid source or peroxidation level effects on urinary TBARS.

**Intestinal Barrier Function**

The ratio of urinary lactulose to mannitol was greater (\( P < 0.05 \)) in pigs fed the control diet compared with pigs fed the lipid supplemented diets (Table 1). Lipid source tended (\( P = 0.09 \)) to affect the ratio of lactulose to mannitol. The ratios of lactulose to mannitol for CN (0.07, 0.05, and 0.05) and CA (0.06, 0.04, and 0.07) were lower than that of PF (0.26, 0.05, and 0.13), with TL being intermediate (0.19, 0.09, and 0.05), for OL, SO, and RO, respectively. However, there were no statistically significant differences for lactulose to mannitol ratio between any 2 sources of lipids. There was no peroxidation level effect or lipid source × peroxidation level interaction for lactulose to mannitol ratio. Pigs fed diets supplemented with lipids tended to have greater (\( P = 0.06 \)) endotoxin concentrations than pigs fed the control diet. No lipid source × peroxidation level interaction or lipid source and peroxidation level effects were noted in the 24-h fasted serum endotoxin analysis.

**Immune Responses**

No lipid source × peroxidation level interaction, lipid source, or lipid peroxidation level effects were found in serum haptoglobin concentration (Table 1). In addition, no difference in serum haptoglobin was found between pigs fed lipid supplemented diets and pigs fed the control diet. No lipid source × peroxidation level interaction, lipid source, or lipid peroxidation level effects were found for serum IgA or IgG analysis. Pigs fed the lipid supplemented diets tended to have an increased serum IgA and Ig G compared with pigs fed the control diet (\( P = 0.10 \) and \( P = 0.09 \), respectively).

**Organ Weight**

No differences in spleen weights were observed between pigs fed the control and the lipids diets, and there was no oxidation level or lipid source × oxidation level interaction. A lipid source effect was observed where pigs fed TL diets had increased spleen weight compared with pigs fed CN diets (\( P = 0.02 \)) and also tended to have increased spleen weight compared with pigs fed PF (\( P = 0.07 \)).
Correlation Analysis
Lipids with various levels of peroxidation, measured using different peroxidation assays, affected serum α-T and serum TBARS concentration of pigs. As a result, correlation analysis between measures of lipid peroxidation and serum α-T and TBARS concentration was conducted to determine the association of various lipid peroxide measures in predicting these biological responses (Table 2).

Because of the limited number of animals used in the current experiment, correlations where the P-value was 0.2 or less were considered. For serum α-T concentration, negative correlations between serum α-T concentration and peroxide value (PV; P < 0.05), p-anisidine value (AnV; P < 0.01), TBARS (P < 0.01), hexanal (P < 0.01), 2,4-decadienal (DDE; P < 0.05), HNE (P < 0.05), and active oxygen method (AOM; P < 0.01) were observed. Oxidative stability index (OSI) value also had a positive correlation with serum α-T concentration (P = 0.15). All measures of lipid peroxidation (PV, AnV, TBARS, hexanal, DDE, HNE, AOM, and OSI) were correlated with the serum TBARS concentration.

DISCUSSION
The chemical composition and peroxidative status of the experimental lipids used in this study were as described by Liu et al. (2014b). All lipids were included in the diet at 10%, which was relatively greater than normally used in commercial diets, to help delineate differences among treatments if they existed. Because the original CN (400 IU/kg lipid) and CA (290 IU/kg lipid) contained a relative greater amount of total tocopherols compared with other 10 experimental lipids (<100 IU/kg), the calculated total tocopherol concentrations were greater in the diets containing original CN (75.1 IU/kg diet) than in the control (39.1 IU/kg diet) or in other lipid supplemented diets (35.1 IU/kg diet). However, the calculated daily consumption of total tocopherols based on the ADFI and the calculated dietary concentration of total tocopherols (64.1 IU/kg diet) were still greater than NRC (1998) recommended level for the young pigs weighing less than 20 kg.

α-T is the principal lipid-soluble antioxidant in tissues and blood (Rigotti, 2007). After absorption, α-T is transported in serum by lipoproteins where it initially functions to protect lipoproteins from degradation. The principal role of α-T is to neutralize reactive oxygen species (ROS) by a direct quenching mechanism (Halliwell and Gutteridge, 1989). The antioxidant vitamin E family and is the principal lipid-soluble antioxidant in tissues and blood (Rigotti, 2007). After absorption, α-T is transported in serum by lipoproteins where it initially functions to protect lipoproteins from degradation. The principal role of α-T is to neutralize reactive oxygen species (ROS) by a direct quenching mechanism (Halliwell and Gutteridge, 1989).
unsaturated fatty acids from free radical damage (Chung et al., 1992). In the current experiment, although all pigs had greater daily consumption of dietary α-T than NRC (1998) recommendations, pigs fed lipids that had been subjected to SO or RO exhibited lower serum α-T than pigs fed OL within the CN or CA treatment. Oxidative stress in animals fed peroxidized lipids has been well documented and can be explained by the enhanced turnover or catabolism rate of antioxidants caused by the oxidative stress (Benedetti et al., 1987; Liu and Huang, 1996; Eder, 1999). No decrease in serum α-T concentration was noted in SO or RO PF and TL. This finding is consistent with the relatively low concentrations of peroxidation products found in PF and TL compared with the concentrations found in CN and CA and is most likely due to differences in fatty acid composition among lipid sources. In addition, correlations were found between serum α-T and all measures of lipid peroxidation (PV, AnV, TBARS, hexanal, DDE, HNE, AOM, and OSI) and serum TBARS concentration. Thus, TBARS may be used to predict the oxidative status of pigs fed various levels of peroxidized lipids. Pigs fed CN or CA had greater TBARS compared with pigs fed PF or TL, which is consistent with their differences in lipid peroxidation products. The greater potential of metabolic oxidative stress caused by CN and CA is also consistent with the early findings in humans (Kleinvedel et al., 1993), rabbits (Hennigs et al., 1995), and rats (Csallany et al., 2000), in which excessive consumption of dietary PUFAs increased the risk of lipid peroxidation. The increased level of oxidative stress in pigs fed CN and CA may be due to the consumption of a greater amount of unsaturated fatty acids, which are particularly susceptible to autoxidation formation of fatty acid radicals (Sherwin, 1978).

Measurement of urinary secondary peroxidation products, such as MDA, can be biased as a marker of oxidative stress in vivo by ingestion of dietary lipid (Draper et al., 2000). Because of this, urine was collected after a 24-h fast to avoid the influence induced by the different dietary intake of peroxidized lipids. However, no lipid source or peroxidation level effects were found for urinary TBARS. One explanation might be due to the fact that thiobarbituric acid may react with the variety of compounds other than aldehydes in the urine, resulting in a lack of either sensitivity or specificity in urine TBARS analysis (Draper et al., 2000; Grotto et al., 2009; Campos et al., 2011). Serum α-T and TBARS results in the current

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### Table 2. The correlation coefficient between test of lipid oxidation and the analysis of serum α-tocopherol (α-T) and serum thiobarbituric acid reactive substances (TBARS)¹

<table>
<thead>
<tr>
<th>Item</th>
<th>PV</th>
<th>AnV</th>
<th>TBARS</th>
<th>Hex</th>
<th>DDE</th>
<th>HNE</th>
<th>AOM</th>
<th>OSI</th>
</tr>
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<tr>
<td>Serum α-T</td>
<td>-0.22</td>
<td>-0.37</td>
<td>-0.21</td>
<td>-0.30</td>
<td>-0.30</td>
<td>-0.35</td>
<td>-0.32</td>
<td>0.14</td>
</tr>
<tr>
<td></td>
<td>(0.03)</td>
<td>(&lt;0.01)</td>
<td>(0.03)</td>
<td>(&lt;0.01)</td>
<td>(&lt;0.01)</td>
<td>(&lt;0.01)</td>
<td>(&lt;0.01)</td>
<td>0.15</td>
</tr>
<tr>
<td>Serum TBARS</td>
<td>0.43</td>
<td>0.33</td>
<td>0.49</td>
<td>0.43</td>
<td>0.45</td>
<td>0.42</td>
<td>0.52</td>
<td>-0.32</td>
</tr>
<tr>
<td></td>
<td>(&lt;0.01)</td>
<td>(&lt;0.01)</td>
<td>(&lt;0.01)</td>
<td>(&lt;0.01)</td>
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<td>(&lt;0.01)</td>
<td>(&lt;0.01)</td>
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¹Top value represents correlation (r) and bottom value in the parentheses represents significance (P-value).

²PV = peroxide value; AnV = p-anisidine value; Hex = hexanal; DDE = 2,4-decadienal; HNE = 4-hydroxynonenal; AOM = active oxygen method; OSI = oxidative stability index.
study indicate that feeding weaned pigs diets containing 10% of thermally oxidized lipids, especially CN and CA that contain greater concentrations of PUFA, impairs their peroxidative status by increasing the production of lipid peroxidation products and depleting α-T in serum.

The gastrointestinal tract not only serves to digest and absorb nutrients from the intestinal lumen, but it also acts as the first protective barrier between the intestinal lumen and the body. Changes in gastrointestinal tract structure, such as gut barrier function, can be associated with changes in its nutrient absorption and pathogen resistance function. Poor gut barrier function may reduce the resistance of an animal to infectious agents such as endotoxins or pathogenic bacteria and may cause activation of the immune system. Therefore, the effect of thermally oxidized lipids, containing various levels of peroxidation products, on intestinal barrier function of young pigs was investigated.

One of the most popular methods used to evaluate gut barrier function is to measure intestinal permeability, which is generally dictated by paracellular permeability (Bjarnason et al., 1995). Paracellular permeability can be determined in vivo by urinary recovery of inert markers (Bjarnason et al., 1995; Wijtten et al., 2011a,b). The principle of the test is based on the fact that the orally administered lactulose can only be absorbed through the paracellular route when the barrier function is compromised, although the monosaccharide, such as mannitol, can be absorbed through both paracellular or transcellular routes and therefore provides an assessment of the absorptive surface. Therefore, lactulose and mannitol are used commonly together to minimize the influence of pre- and postintestinal factors on recovery of the paracellular markers because these 2 markers empty similarly from the stomach, are not metabolized in the small intestine, and are cleared in the same manner from the kidneys (Bjarnason et al., 1995). In the current experiment, lipid source, peroxidation level, and their interaction did not influence the paracellular permeability as measured by the lactulose to mannitol ratio. To date, few experiments have been conducted to evaluate the effect of thermally oxidized lipids on intestinal barrier function of pigs. Oxidized lipids containing secondary peroxidation products, such as MDA and HNE, can negatively influence cells directly by causing membrane perturbations, which contribute to poor membrane permeability. Previously, Dibner et al. (1996) reported that feeding oxidized PF to broilers resulted in intestinal structural injury as indicated by a decreased half-life of enterocytes. Assimakopoulos et al. (2004) suggested that intestinal oxidative stress was a key factor, resulting in intestinal physical injury as indicated by decreased villous density and total mucosal thickness. In addition, feeding thermally oxidized sunflower oil to growing pigs increased markers of oxidative stress in the small intestine (Ringseis et al., 2007). Therefore, consumption of thermally oxidized lipids may promote intestinal oxidative stress and, subsequently, cause intestinal injury and gut barrier dysfunction in pigs. In the current study, feeding 10% thermally oxidized lipids to nursery pigs for 38 d caused metabolic oxidative stress by depleting serum α-T and increasing serum TBARS. However, no impaired gut permeability was observed in pigs fed thermally oxidized lipids. The lack of an intestinal barrier function effect observed in the current study might be explained by the different levels of oxidative stress caused by the peroxidized lipids, duration of feeding period, or the animal species used.

In pigs, little information regarding feeding diets with and without lipids on gut permeability has been reported. In the current experiment, pigs fed lipid supplemented diets had tighter paracellular permeability compared with pigs fed the control diet as indicated by a lower lactulose to mannitol ratio. Fasting can be one of the major reasons, resulting in poor intestinal integrity and permeability due to malnutrition (Sundqvist et al., 1982; Fan et al., 2008). In the current study, all pigs had a 29-h fast before the gut permeability test to ensure that pigs consumed all gut permeability markers. As a result, the improved gut permeability of pigs fed lipid supplemented diets may have been associated with the added lipid that may help prevent malnutrition by delaying gastric digesta emptying and subsequently improving nutrient absorption (Hunt and Knox, 1968; Li and Sauer, 1994).

Endotoxins are combinations of lipid and polysaccharide side chains on the cell wall of gram-negative bacteria. Under normal conditions, no or minimal amounts of endotoxins appear in serum because of a tight intestinal barrier. Therefore, detection of serum endotoxins may indicate intestinal barrier malfunction (Lichtman, 2001). In the current experiment, no lipid source or peroxidation level effects or their interaction were observed in serum endotoxin concentrations, which are consistent with the results of the gut permeability measurement. However, the fact that pigs fed diets containing lipids had a tendency for a greater serum endotoxin concentration than pigs fed control diets is in contrast to the finding that pigs fed lipid supplemented diets had tighter paracellular permeability than pigs fed the control diet. This contrast between intestinal permeability marker and serum endotoxin concentration or bacterial translocation has also been reported previously (Wijtten et al., 2011b), where an increase in intestinal lactulose permeability of pigs was associated with a decrease in bacterial translocation. Because both intestinal permeability and intestinal lumen toxin or bacterial concentrations are important factors determining the serum endotoxin concentration or bacterial translocation, it is
speculated that the disassociation between gut permeability markers and serum endotoxin analyses is related to the different concentrations of intestinal lumen toxin or bacterial concentration (Wijtten et al., 2011b). As such, more research is needed to understand the effects of feeding lipid supplemented diets on intestinal toxin or bacterial concentrations of pigs.

Consumption of specific lipid peroxidation products may influence the immune response of animals because treating cells with HNE can increase activation of stress pathways (Biasi et al., 2006; Yun et al., 2009) and increase the expression of macrophage inflammatory mediators (Kumagai et al., 2004). Activation of stress pathways or overexpression of inflammatory mediators not only causes redistribution of nutrients away from growth processes in support of the immune system (Liu et al., 2010) but may also inhibit IGF-1 mRNA expression (Thissen and Verniers, 1997), both of which can contribute to poor growth performance. Haptoglobin, a representative acute phase protein produced by hepatocytes during inflammatory conditions (Kent, 1992), has been found in swine serum and has been shown to be increasingly produced during nonspecific bacterial infections and chemical stimulation (Shim et al., 1971; Hall et al., 1992). Therefore, serum haptoglobin can be considered a sensitive indicator in evaluating the activation of the immune system in pigs (Petersen et al., 2004). In the current experiment, no lipid source or peroxidation level effect or lipid source × peroxidation level interaction was found for serum haptoglobin concentration and no difference in serum haptoglobin was found between pigs fed lipid supplemented diets and pigs fed the control diet.

Immunoglobulin levels are determined for evaluation of the humoral immune status, with low levels of immunoglobulins being associated with humoral immunodeiciencies (Buckley, 1986) and greater immunoglobulin concentrations being associated with inflammatory and pathological conditions (Haye and Kornegay, 1979; Redman, 1979; Parreno et al., 1999). Previous studies conducted in broilers (Takahashi and Akiba, 1999) found that feeding oxidized fat decreased ex vivo primary antibody production in response to a bacterial pathogen. In the current experiment, no changes in serum IgA and IgG indicate that the different lipid peroxidation levels used in the current experiment did not influence the synthesis of antibodies. In agreement with serum endotoxin analysis, pigs fed the lipid supplemented diets tended to have increased serum IgA and IgG compared with pigs fed the control diet. This is supported by (Zou et al., 2010), who reported that increased synthesis of antibodies in pigs fed lipids diet helped to improve defense system for eliminating the endotoxin.

The spleen is an important organ for red blood cells and the immune system and is found in virtually all vertebrate animals. The spleen functions to remove old red blood cells and holds a reserve of blood in case of hemorrhagic shock while also recycling iron. The spleen also plays an important role in the synthesis antibodies and clearing antibody-coated bacteria along with antibody-coated blood cells by way of blood and lymph node circulation (Meibius and Kraal, 2005). The enlargement of the spleen, splenomegaly, is commonly associated with bacterial infections (Jackson et al., 2010). To our knowledge, this is the first report regarding the effect of lipid source and feeding thermally oxidized lipids on spleen weight in pigs. Pigs fed TL diets had greater spleen weights compared with pigs fed CN or PF. However, further investigations of mechanism responsible for the increased spleen weight in pigs fed TL are needed.

In conclusion, feeding weaned pigs diets containing 10% thermally oxidized lipids for 38 d, especially vegetable oils containing greater concentrations of PUFA, impaired metabolic oxidative status by depleting serum α-T and increasing serum TBARS. However, gut barrier function and immune response traits were not changed among pigs fed thermally oxidized lipids and unheated lipids, implying that pigs were relatively resilient to certain levels of lipid oxidation.

LITERATURE CITED


