Conjugated linoleic acids alter body composition differently according to physiological age in Moulard ducks

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ABSTRACT Conjugated linoleic acids (CLA) have been shown to have remarkable yet inconsistent metabolic effects in mice, rats, hamsters, chickens, cattle, and humans. In particular, effects on lipogenesis vary with tissue, physiological state, and species. In this study we tested the hypothesis that CLA would differentially affect ducks of the same genetic background but of differing age. Growing (7 wk) and maintenance (11 wk) Moulard ducks were grouped by age and fed a standard diet supplemented with 5% soybean oil (control) or 5% CLA isomer mixture. Birds were slaughtered after 3 or 6 wk for assessment of body composition including adipose, liver, viscera, and empty carcass weight. Serum nonesterified fatty acid (NEFA) and glucose concentrations were evaluated, and gene targets were cloned from the duck to use in quantifying mRNA abundance for genes involved in lipogenesis (fatty acid synthase, FAS; acetyl-CoA carboxylase, ACC) and lipid oxidation (carnitine palmitoyl transferase-1, CPT-1) in liver tissue from maintenance birds. After 3 wk, the growing CLA group exhibited a 24% decrease in dissectible adipose tissue ($P < 0.05$), whereas maintenance birds showed no significant diet effect. After 6 wk, the growing CLA group exhibited a 20% increase in liver mass compared with the control ($P < 0.05$), but no diet effect on adipose tissue. Maintenance birds receiving dietary CLA had a 42% decrease in adipose tissue mass after 6 wk; increased serum NEFA, ACC, and CPT-1 mRNA after 3 and 6 wk ($P < 0.05$); and increased FAS mRNA after 3 wk of treatment ($P < 0.05$). These data indicate that CLA have potent effects on lipid metabolism in ducks, but these effects differ depending on physiological age.

Key words: conjugated linoleic acid, body composition, adipose, duck

INTRODUCTION

Conjugated linoleic acid (CLA) isomers, a group of positional and geometric isomers of linoleic acid, have been studied extensively in a variety of experimental models due to their ability to modulate cancer, atherosclerosis, obesity, immune function, and diabetes (Brown and McIntosh, 2003). Conjugated linoleic acid is a natural food component found in the lipid fraction of dairy products and other ruminant fats and consists of more than 16 possible geometric isomers with cis-9, trans-11 CLA (9,11-CLA) and trans-10, cis-12 CLA (10,12-CLA) being the most abundant in commercial preparations (Bauman et al., 2000).

The mechanism of action for CLA isomers to decrease body fat accretion in chickens, pigs, and rodents (Park et al., 1999; Du and Ahn, 2002; Corl et al., 2008) or to inhibit milk fat production in ruminants (Peterson et al., 2004) is not fully understood. Studies in vitro and in vivo have led researchers to postulate that 10,12-CLA affects 2 key transcription factors in the peroxisome proliferator-activated receptor (PPAR) family ($\alpha$, $\gamma$) as well as sterol regulatory element binding protein 1c (SREBP-1c) to promote or inhibit the expression of downstream target genes responsible for $\beta$-oxidation and lipogenesis (Belury, 2002). Support for this concept has been documented in animal experiments where dietary CLA has been shown to increase mRNA abundance for oxidative enzymes responsive to PPAR-$\alpha$ when fed to growing mice (Moya-Camarena et al., 1999), rats (Rahman et al., 2001), and geese (Zhang et al., 2009). Similarly, other studies have documented decreased PPAR-$\gamma$ mRNA abundance in mice (Kang and Pariza, 2001) and SREBP-1c mRNA abundance in hamsters (Zabala et al., 2006) when CLA was fed as part of the diet. Alternatively, in vitro studies have reported that the addition of 10,12-CLA to the media of murine 3T3-L1 cells (Brodie et al., 1999), as well as porcine (Brandebour and Hu, 2005) and human preadipocytes (Brown et al., 2003) inhibited cell differentiation and decreased PPAR-$\gamma$ mRNA expression. Interestingly, a CLA mixture was also shown to increase lipid filling in the 3T3-L1 model, underscoring the complexity of the biological activity of CLA (Satory and Smith, 1999). The ability for 10,12-CLA to
attenuate lipogenesis in the mammary gland was characterized using the bovine MAC-T mammary epithelial cell line where 10,12-CLA decreased mRNA transcript abundance for key genes involved in lipid synthesis, an effect postulated to be secondary to decreased proteolytic activation of SREBP-1c (Peterson et al., 2004). Taken together, these findings present considerable evidence that adipogenesis can be controlled, at least in part, by CLA through SREBP-1c and PPAR-mediated pathways.

The application of CLA to decrease body fat accretion is of particular interest to food animal producers, as excess carcass fat decreases feed efficiency and profitability (Harris and Newman, 1994). Over the last 50 yr, food animal producers have implemented growth-promoting technologies to decrease production costs by improving feed efficiency and enhancing lean content to satisfy consumer demand (Avendaño-Reyes et al., 2006). Interestingly, growth-promoting technologies (anabolic steroids, β-adrenergic agonists) are of limited utility in poultry because meat birds are slaughtered before sexual maturity (Buttery and Dawson, 1990). Hence, poultry producers have relied on stringent genetic selection to propagate high-growth birds, which unfortunately has led to birds that are proportionally fatter than unimproved lines (Cartwright, 1991). The ability of CLA to decrease adipogenesis in vivo and in vitro may provide a promising method to decrease fat accretion and improve carcass quality in growing birds.

In the present study, we assessed for the first time the effects of CLA in the Moulard duck, a species not subjected to the intensive selection that broilers and turkeys have seen, and one that is often maintained beyond physical maturity under normal production practices. The broad objective of the study was to determine whether CLA affected body composition in growing or maintenance Moulard ducks. To help us understand potential mechanisms underlying the reduction we observed in adipose tissue mass in maintenance ducks, we cloned and sequenced portions of the duck fatty acid synthase (FAS), acetyl Co-A carboxylase (ACC), and carnitine palmitoyl transferase-1 (CPT) genes to assess their mRNA abundance in liver tissue from the maintenance group of birds by quantitative PCR, and measured serum glucose and nonesterified fatty acid (NEFA) concentrations.

MATERIALS AND METHODS

Birds and Diets

All animal use was approved by the California Polytechnic State University Animal Care and Use Committee. For this experiment, 7-wk-old (n = 60) and 11-wk-old (n = 60) male Moulard ducks were provided by Sonoma Foie Gras (Sonoma, CA). Birds were grouped by age and randomly assigned to 20 floor pens (pens numbered 1 to 20, 10 pens/age group, 6 birds/pen, 1.83 m²/bird). Diets were isonitrogenic and differed only by the inclusion of 5% CLA (CLA diet) or 5% soybean oil (soy oil diet) as the source of fat (Table 1). Soybean oil was chosen as the lipid source for birds in the control group based on a similar content of linoleic acid and its commercial availability, and the oils were assumed to be isonitrogenic. Diets were assigned based on pen number, where odd-numbered pens received the CLA diet and even-numbered pens received the soy oil diet (5 pens/diet per age group). Research from modeling growth characteristics in Moulard ducks has shown that growth potential is maximized from 5 to 10 wk of age with a plateau effect occurring at 11 wk of age (Marie-Etancelin et al., 2008). Thus, ducks starting dietary treatment at 7 wk of age were called growing, whereas ducks starting diets at 11 wk of age were called maintenance to signify that the birds were no longer growing and instead maintaining a BW.

The CLA isomer mixture was a fatty acid methyl ester preparation supplied by BASF (LUTA-CLA 60, Florham Park, NJ) as an amber-colored oil containing approximately 28% of the cis-9, 11-trans isomer, and 28% of the 10-trans, 12-cis isomer (BASF, Florham Park, NJ). Soy oil and CLA diets were formulated to meet or exceed all nutritional requirements of ducks (NRC, 1994; Table 1). A basal diet was formulated and mixed that included all ingredients other than the oils, and each experimental diet was prepared every 7 d by mixing either CLA or soy oil in a stainless-steel gear-driven commercial mixer (Hobart, Troy, OH). Diets were placed in sealed plastic containers and stored at 4°C to prevent oxidation of oil additives. Both diets were fed ad libitum as a mash.

![Table 1. Composition of experimental diets](image-url)
**Experimental Design**

At d 21 and 42 after commencing dietary treatment, 3 ducks per pen for each age group were killed in the morning by electrical stunning and exsanguination in accordance with industry standards. Feed was available at all times. Body weight was recorded by weighing the ducks in a box cart digital scale immediately before slaughter. At the time of slaughter, blood was collected and centrifuged at \( 825 \times g \) for 10 min at 4°C. Serum was collected and aliquots were frozen at \(-20^\circ C\) for serum glucose and NEFA analysis. Five-gram samples of right liver lobe were immediately snap frozen in liquid nitrogen and stored at \(-80^\circ C\) for subsequent mRNA analysis. The liver weight was recorded by weighing the dissected right and left liver lobes on a digital scale. All adipose tissue from crop to vent was dissected and weighed. The viscera weight included weights of heart, emptied intestine, lungs, kidneys, reproductive tract, and all other organs removed from the abdominal cavity. The weight of the emptied carcass including skin and feathers was designated as carcass weight. Feed intake was calculated by measuring the amount of feed required to refill feeders to a predetermined mark at the top of the feeder (measured 2 times/d per pen). Spillage could not be avoided and was assumed to be independent of dietary treatment.

**Serum Analysis**

Serum glucose concentration was determined by measuring glucose oxidase activity (Sigma Diagnostics, St. Louis, MO) and serum NEFA concentration was analyzed by a colorimetric method (Wako Pure Chemical Industries, Richmond, VA).

**Cloning and Sequencing of Genes**

Total RNA was isolated from frozen liver samples using a guanidinium thiocyanate-phenol-chloroform extraction (Chomczynski and Sacchi, 1987). Total RNA concentration was determined spectrophotometrically at 260 nm. First-strand cDNA synthesis was completed using a Reverse Transcriptase kit (Promega, Madison, WI) according to the manufacturer’s instructions with 1 µg of total RNA using oligo (dT) as the primer. Primers for FAS, ACC, CPT, β-actin, and 18S ribosomal RNA were designed according to published gene sequence information from the chicken and are reported in Table 2. The identity of the PCR products was confirmed by sequencing (Sequetech, Mountain View, CA), and the percent homology to chicken, bovine, porcine, and human sequence was compared using National Center for Biotechnology Information Basic Local Alignment Search Tool (BLAST).

**Quantitative Real-Time PCR**

Real-time PCR was completed with the Applied Biosystems Fast SYBR Green Master Mix kit according to the manufacturer’s instructions in a 25-µL reaction volume. Quantitative PCR primer pairs for FAS, ACC, CPT, β-actin, and 18S ribosomal RNA were designed from cloned duck cDNA sequence for each gene of interest using Beacon Designer (Premier Biosoft International, Palo Alto, CA; Table 3). β-Actin was used as the endogenous control and nontemplate controls were included on each 96-well plate. Quantitative PCR was performed with a 7500 Fast Real-Time PCR System (Applied Biosystems, Carlsbad, CA) on liver samples from 3 birds for each dietary treatment. Data were analyzed using the \( 2^{-\Delta\Delta CT} \) procedure (Livak and Schmittgen, 2001) following verification that amplification efficiencies were similar. The cycle threshold values were calculated as the cycle number at which fluorescence of the sample exceeded threshold, which was determined by multiplying the SD of the baseline by 10.

**Statistical Analysis**

For each age group, data were analyzed using the GLM in JMP (SAS Institute, Cary, NC) for the effects of pen location, diet, and treatment period duration as well as any interactions. In no case was there a significant effect that included pen location, and it was subsequently dropped from the final statistical model, leading to a 2-way ANOVA for the effects of diet, treatment period, and their interaction. When a main effect or interaction was significant in the 2-way ANOVA (\( P < 0.05 \)), individual unadjusted means comparisons
were made using Fisher’s least significant difference. All differences were considered significant at $P < 0.05$.

**RESULTS**

**Feed Intake**

Feed intake was not affected by diet or treatment period for the growing or maintenance age groups of ducks ($P > 0.05$; Tables 4 and 5).

**Body Composition: Growing Birds**

Dissectible adipose tissue mass was decreased after 3 wk of treatment for growing birds receiving dietary CLA compared with control birds receiving the soy oil diet ($P < 0.05$; Table 4). Although an additional 3 wk of treatment reduced the adipose deposition in birds receiving the CLA diet compared with birds receiving the soy oil diet, this was not statistically significant. The control group of birds fed the soy oil diet also showed a reduction in dissectible adipose mass between the 3- and 6-wk treatment periods, but this was not statistically significant ($P > 0.05$; Table 4). An additional 3 wk of CLA treatment led to a decrease in liver mass that was numerically equivalent to the decrease seen in the control. Treatment period significantly increased final carcass weight and BW in growing birds receiving either diet (Table 4).

**Body Composition: Maintenance Birds**

For the maintenance group of Moulard ducks, the effect of diet on dissectible adipose tissue was dependent on treatment period. Specifically, birds fed the soy oil diet gained adipose mass between the 3- and 6-wk treatment periods (Table 5), whereas dietary CLA attenuated this adipose gain, resulting in a significantly lower dissectible adipose mass after 6 wk of treatment compared with birds receiving the soy oil diet ($P < 0.05$; Table 5). Liver weight, BW, and carcass weight were not significantly different in either treatment group of adult Moulard ducks at any time point of the trial (Table 5). The CLA diet led to lower viscera mass compared with birds fed the soy diet ($P < 0.05$; Table 5).

**Serum Glucose and NEFA**

Serum glucose was not significantly different at 3- and 6-wk slaughter time points for the CLA and soy oil treatment groups for growing birds ($P > 0.05$; Table 4).

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**Table 3. Primer sequences for quantitative PCR analysis**

<table>
<thead>
<tr>
<th>Gene</th>
<th>Primer sequence (5′ → 3′)</th>
<th>Product size (bp)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FAS</td>
<td>Sense: ATGGGTCCTATTCTCTTGTGGCATG</td>
<td>185</td>
</tr>
<tr>
<td></td>
<td>Antisense: AGCAATTCACAGCAAGCCCATCA</td>
<td>143</td>
</tr>
<tr>
<td>ACC</td>
<td>Sense: TGAAGAGGGTGGTCTACACCTGAT</td>
<td>178</td>
</tr>
<tr>
<td></td>
<td>Antisense: TTACGAGATGGAGAGACCCATGGA</td>
<td>104</td>
</tr>
<tr>
<td>CPT</td>
<td>Sense: ACTCTATCCACCTTCAACGTCTCA</td>
<td>262</td>
</tr>
<tr>
<td></td>
<td>Antisense: TTCCATACCCGACTACAGAAAGT</td>
<td>185</td>
</tr>
<tr>
<td>18S</td>
<td>Sense: TCTTGGAGTGTGGTGACCGCTCTTCT</td>
<td>175</td>
</tr>
<tr>
<td></td>
<td>Antisense: GGAATGGTGAAGGATACACGACGAG</td>
<td>143</td>
</tr>
<tr>
<td>β-Actin</td>
<td>Sense: AAACCCATAATTGCAGTTAACCTTCC</td>
<td>104</td>
</tr>
</tbody>
</table>

1FAS, fatty acid synthase; ACC, acetyl Co-A carboxylase; CPT, carnitine palmitoyl transferase-1; 18S, 18S ribosomal RNA.

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**Table 4. Growing Moulard performance during the course of the study**

<table>
<thead>
<tr>
<th>Variable</th>
<th>3 wk on diet</th>
<th>6 wk on diet</th>
<th>SE</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW (kg)</td>
<td>Soy oil</td>
<td>CLA</td>
<td>Soy oil</td>
<td>CLA</td>
</tr>
<tr>
<td>3.70</td>
<td>3.54</td>
<td>4.36</td>
<td>4.23</td>
<td>0.06</td>
</tr>
<tr>
<td>Carcass (kg)</td>
<td>2.99</td>
<td>2.83</td>
<td>3.63</td>
<td>3.50</td>
</tr>
<tr>
<td>Adipose (g)</td>
<td>38.2</td>
<td>29.1</td>
<td>31.6</td>
<td>24.6</td>
</tr>
<tr>
<td>Liver (g)</td>
<td>80.2</td>
<td>88.6</td>
<td>63.4</td>
<td>75.8</td>
</tr>
<tr>
<td>Viscera (g)</td>
<td>264.0</td>
<td>280.3</td>
<td>241.0</td>
<td>253.1</td>
</tr>
<tr>
<td>Serum glucose (mg/dL)</td>
<td>196.8</td>
<td>205.4</td>
<td>206.3</td>
<td>198.2</td>
</tr>
<tr>
<td>Serum NEFA (µM)</td>
<td>525.3</td>
<td>564.0</td>
<td>579.4</td>
<td>632.6</td>
</tr>
<tr>
<td>Feed intake (g/d per bird)$^2$</td>
<td>51.3</td>
<td>51.9</td>
<td>43.4</td>
<td>49.7</td>
</tr>
</tbody>
</table>

$^1$Values for each variable are means ± SE (n = 15 birds/treatment). $P$-values are listed for the effects of diet (D), treatment period (T), or interactions (D $\times$ T). CLA = conjugated linoleic acid; NEFA = nonesterified fatty acids.

$^2$Values are mean feed intake (g/bird per treatment group per d). Feed intake per pen was the amount of feed needed to refill each pen’s feeder. These values were pooled weekly by pen and divided by the total number of birds in each pen and days to determine average gram intake/bird per treatment per group per day (n = 5).
4. Serum glucose was affected by treatment period in the maintenance group of birds. Specifically, after 6 wk of treatment, serum glucose was significantly increased for birds in the maintenance group receiving either diet ($P < 0.05$; Table 5).

The CLA treatment resulted in a numerical but non-significant elevation of serum NEFA in growing birds throughout the entire 6-wk trial compared with the control (Table 4). In the maintenance group of birds, dietary CLA significantly increased NEFA concentration at 3- and 6-wk treatment periods ($P < 0.05$; Table 5).

**mRNA Abundance**

The cloned segments of the duck ACC, FAS, CPT, β-actin, and 18S rRNA ranged from 68 to 84% homology to bovine, porcine, and human sequences, and between 94 and 100% homology to the chicken sequence. Quantitative PCR was performed with liver tissue samples from the maintenance group of ducks because these birds exhibited the greatest magnitude effect on adipose mass after 6 wk on the CLA diet. Conjugated linoleic acid led to an increase of FAS mRNA transcript abundance at 3 wk of treatment (Figure 1; $P < 0.05$). After being on feed for an additional 3 wk, FAS mRNA for CLA-treated birds had decreased to a level near that of the control. The ACC and CPT mRNA abundance were increased as a result of dietary CLA at the 3-wk slaughter time point compared with birds of the same age in the control group (Figures 2 and 3; $P < 0.05$). After 6 wk of CLA treatment, ACC and CPT mRNA levels remained elevated compared with birds of the same age receiving the soy oil diet, but these differences were not significant.

**DISCUSSION**

**Effect of Diet on Body Composition in Growing Ducks**

Growing birds receiving dietary soy oil or CLA had similar feed intake and increased body and carcass weights after being on the respective diets for 6 wk. We expected this result as both treatment groups are in a physiological state of growth and therefore should increase in size until physically mature. This is in agreement with studies performed in mice, hamsters, piglets, chickens, and humans where CLA modulated lipid metabolism without affecting BW (Park et al., 1997; Thom et al., 2001; Du and Ahn, 2002; Zabala et al., 2004; Corl et al., 2008). Viscera mass was significantly decreased in growing birds in the 6-wk treatment period group regardless of diet. Because both dietary groups were affected, it is assumed that this is a normal physiological change in Moulard ducks.

![Figure 1](image_url). Effects of 5% dietary conjugated linoleic acid (CLA) or soy oil for 3 and 6 wk on fatty acid synthase (FAS) mRNA abundance in liver tissue from birds maintaining a BW. Data were normalized to the abundance of β-actin and expressed on an arbitrary unit scale. Error bars represent SEM; bars with differing letters (a,b) were significantly different ($P < 0.05$; n = 3).
receiving the CLA diet in the first 3 wk of treatment. At the end of the 6-wk treatment, the difference in adipose mass persisted but was not significant. A similar effect was reported in pigs where CLA reduced backfat thickness in growing gilts with no effect on live BW (Ostrowska et al., 1999) and in mice where body fat was reduced by more than 50% in weanling mice receiving dietary CLA without affecting BW (Park et al., 1997).

Several studies have shown dietary CLA to increase liver weight in growing mice (Tsuboyama-Kasaoka et al., 2003; Degrace et al., 2004) and broiler chickens (Badinga et al., 2003). In our study, CLA-fed birds in the growing group had significantly larger liver mass than birds fed the soy diet after 6 wk of treatment without a difference in feed intake. This finding is consistent with studies performed in laying hens where CLA-fed birds had increased fat vacuolation and hepatic lipid infiltration, resulting in increased liver weight (Cherian and Goeger, 2004). Additionally, experiments using mice attributed increased liver mass in CLA-fed animals to be a result of increased liver lipid accumulation (DeLany and West, 2000), though such determination was not made in this experiment.

**Effect of Diet on Body Composition in Maintenance Birds**

The CLA did not affect feed intake, BW, or carcass weight in physically mature ducks. These findings are similar to long-term feeding trials conducted with 42-wk-old rats maintaining a BW where animals fed 1.5% dietary CLA for 36 wk did not display a significant difference in feed intake or BW (Scimeca, 1998).

Interestingly, in our trial there was a significant reduction in adipose mass in response to the CLA diet in the maintenance birds after 6 wk of treatment. There are limited data available involving the feeding of CLA to animals that are physically mature, and we are the first to report that CLA reduces adipose content in ducks in a maintenance state.

Unlike birds in the growing group, CLA treatment did not increase liver mass in birds maintaining a BW. This finding is interesting considering that ducks in the growing group slaughtered after 6 wk of CLA treatment had increased liver mass and were 1 wk younger than ducks in the maintenance group slaughtered after 3 wk. Hence, we expected ducks in the maintenance group to follow a similar trend and to have increased liver weight. Although the birds from both groups were not the exact same age at the time of slaughter, the effect of feeding duration can be considered. Perhaps the action of CLA to increase liver mass plateaus as birds enter physical maturity as observed in the maintenance group. This possibility warrants further investigation.

**Effect of CLA on Serum Glucose and Nonesterified Fatty Acids**

Circulating glucose and NEFA are precursors for the synthesis and oxidation of fat, respectively, and these metabolites can serve as indicators of energy balance in animals. Because glucose is the principal carbon source for lipogenesis in avian species, a bird in a positive energy balance will use glucose to replenish glycogen reserves and then convert excess glucose into fat. In contrast, a bird experiencing the initial stages of fasting will cleave fatty acids from lipid storage depots so they
can be released into the blood as NEFA and oxidized into a usable form of energy in muscle and liver tissues. Several researchers have postulated that CLA decreases adiposity by inhibiting glucose uptake and promoting lipolysis in adipocytes (Ostrowska et al., 1999; Mersmann, 2002) while concurrently increasing insulin sensitivity in muscle and liver tissues to accelerate glucose absorption and oxidation (Housenbech et al., 1998).

Here, we evaluated glucose and NEFA concentrations in CLA-fed birds to determine if this mechanism was plausible. In this study, glucose concentration was not significantly different for either treatment group at any time when measured in the growing group of birds. In the maintenance group of ducks, glucose concentrations were elevated after 6 wk of treatment for birds receiving both diets compared with values measured at 3 wk. Because both dietary groups were affected, it is assumed that this is an age-related physiological change. Nonetheless, all values were within normal ranges specified for avian species (210–550 mg/dL; Candeletta et al., 1993).

The CLA led to a numerical but nonsignificant increase in NEFA concentration in growing birds slaughtered after 6 wk of treatment. In contrast, CLA significantly increased NEFA concentrations at both time points for birds in the maintenance group. Increased NEFA concentrations indicate increased rates of lipolysis (Ostrowska et al., 1999; Azain et al., 2000) and may, in part, explain the lower adipose tissue mass in the maintenance group of birds after 6 wk of treatment. The fact that adipose mass increased in the control group during the second 3-wk period, the difference persisted but was not significant. These findings parallel those reported in a similar species where male goslings fed 2.5% CLA showed a decrease in the abundance of lipogenic enzymes FAS and ACC in liver tissue from the maintenance birds (Azain et al., 1993). It was very unexpected that CLA increased the transcript abundance of lipogenic genes FAS and ACC in liver tissue from the maintenance birds at the 3-wk treatment period compared with the control, because CLA-treated birds had less dissected adipose tissue indicating decreased lipogenesis. Although this difference was only significant at 3 wk, other researchers have reported similar findings in growing mice fed a 1% CLA mixture that exhibited increased rates of fatty acid synthesis in the presence of decreased fat deposition (DeLany and West, 2000).

The simultaneous increases in abundance of mRNA for genes involved in both lipid oxidation and lipogenesis indicates the complex nature of the effects of CLA isoforms on metabolism and warrants further investigation.

Based on our findings, it appears that CLA holds potential as a potent metabolic modifier in poultry species, yet further research to expand these findings is needed to determine the mechanism of action for CLA so improvements in feed efficiency and carcass quality can be optimized.

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