Effects of dietary copper on the expression of lipogenic genes and metabolic hormones in steers

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ABSTRACT: An experiment was conducted to determine the effects of Cu supplementation on performance, subcutaneous adipose tissue mRNA expression of acetyl CoA carboxylase (ACC), stearoyl CoA desaturase (SCD), uncoupling protein 2 (UCP2), and leptin in growing and finishing steers. Forty-eight purebred Angus steers were allotted to one of five treatments: 1) control (no supplemental Cu); 2) 10 mg Cu/kg DM from CuSO4; 3) 10 mg Cu/kg DM from a Cu amino acid complex (Availa Cu); 4) 20 mg Cu/kg DM from CuSO4; 5) 20 mg Cu/kg DM from Availa Cu. Steers were fed an alfalfa hay corn-based diet for 56 d (basal diet contained 7.1 mg Cu/kg DM) and switched to a high-concentrate diet for 144 d (basal diet contained 6.1 mg Cu/kg DM). Blood samples were obtained every 28 d throughout the entire experiment. On d 112 of the finishing period, subcutaneous adipose tissue biopsies were obtained from the tailhead of three animals per treatment and analyzed for ACC, SCD, UCP2, and leptin mRNA expression. Animal performance was not affected by Cu supplementation during the growing phase. Steers receiving 10 mg Cu/kg DM from Availa Cu had higher (P < 0.05) ending body weights and tended (P < 0.10) to have higher ADG than steers receiving 10 mg Cu/kg DM from CuSO4 during the finishing phase. Serum concentrations of nonesterified fatty acid and insulin were not affected by Cu supplementation. Steers receiving supplemental Cu tended (P < 0.11) to have less backfat relative to controls. However, dietary Cu did not influence the level of subcutaneous adipose tissue ACC and SCD mRNA. Neither UCP2 nor leptin gene expression was affected by Cu supplementation. These results indicate that dietary Cu supplementation (10 to 20 mg Cu/kg DM diet) may alter lipid metabolism of subcutaneous adipose tissue; however, it does not seem to affect expression of certain lipogenic genes.

Key Words: Adipose Tissue, Copper, Lipid Metabolism, Performance, Steers

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Introduction

Copper is believed to regulate lipid metabolism in mammals. Dietary Cu supplementation decreased backfat depth and tended to decrease cholesterol concentrations in serum and longissimus muscle (Ward and Spears, 1997; Engle et al., 2000a). In addition, dietary Cu supplementation has been shown to modulate fatty acid composition by increasing PUFA in longissimus muscle (Engle et al., 2000a,c). The changes of lipid metabolism by dietary Cu might be related to modulation of expression of genes involved with adipose tissue accumulation, composition, and metabolism. Thus, the current study was designed to examine the influence of dietary Cu on the expression of lipogenic enzymes and metabolic hormones.

Acetyl CoA carboxylase (ACC) is the rate-limiting enzyme of fatty acid synthesis, which is allosterically controlled and responds to both hormonal and metabolite stimuli in all species (Munday et al., 1991). The rate-limiting step in the biosynthesis of MUFA is the insertion of a cis-double bond in the δ9 position of fatty acyl-CoA substrate. This oxidative reaction is catalyzed by stearoyl-CoA desaturase (SCD). The regulation of SCD is of considerable physiological importance because it influences the composition of fatty acids in muscle and adipose tissue (Kim and Ntambi, 1999). Uncoupling proteins (UCP) uncouple mitochondrial respiration from ATP phosphorylation and increase thermogenesis in mammals (Flier and Lowell, 1997). Of the three UCP subtypes, UCP2 is found in white adipose tissue, is linked to obesity, and is induced by fat feeding (Fleury et al., 1997). The product of the ob gene, leptin, is proportional to the level of adiposity, suppresses food intake, and increases energy expenditure (Hossner, 1998).

We have developed a nonisotopic ribonuclease protection assay (Turnbow and Garner, 1993) to examine the
expression of ACC, UCP2, SCD, and leptin transcripts in subcutaneous adipose tissue in response to dietary Cu supplementation in cattle.

Materials and Methods

Animals, Diets, and Experimental Procedure

Forty-eight purebred Angus weaned steers (250 kg initial BW) were utilized in this experiment. Care, handling, and sampling of the animals herein were approved by the Colorado State University Animal Care and Use Committee. Steers were transported approximately 250 km from our experiment station in Saratoga, WY, to our beef cattle feedlot facility. Upon arrival, calves were weighed, vaccinated with Cattle Master 4 (Pfizer Animal Health, Exton, PA) and Vision 7 (Bayer, Shawnee Mission, KS), and wormed with Safe Guard (Hoechst-Roussel, Somerville, NJ). Steers were then weighed on two consecutive days and allotted, by body weight, to 1 of 48 individual pens (2.2 m × 20 m) equipped with automatic waterers (water contained 0.01 mg Fe/L, 0.12 mg Zn/L, 0.01 mg Cu/L, and 0.03% S). All steers were fed a grass hay diet for 14 d.

Growing Phase. After adjusting to the individual pen, steers were weighed on two consecutive days, implanted with Synovex-S (Fort Dodge Animal Health, Fort Dodge, IA), and bled via jugular venipuncture. Steers were then allotted to one of five groups based on body weight. Groups were then randomly assigned to treatments. Treatments consisted of 1) control (no supplemental Cu), 2) 10 mg Cu/kg DM from CuSO4, 3) 10 mg Cu/kg DM from a Cu amino acid complex (Availa Cu) (Zinpro Corp., Eden Prairie, MN), 4) 20 mg Cu/kg DM from CuSO4, and 5) 20 mg Cu/kg DM from Availa Cu.

Steers were fed an alfalfa hay corn-based growing diet for 56 d (Table 1: basal diet contained 7.1 mg Cu/kg DM, 32.8 mg Zn/kg DM, 68.1 mg Fe/kg DM, 0.13% S, and 0.59 mg Mo/kg DM). Diets were formulated to meet or exceed all nutrient requirements for growing steers with the exception of Cu (NRC, 1996). Diets were fed once daily in the morning in amounts adequate to allow ad libitum access to feed. Daily feed offerings were recorded and feed refusals were measured every 28 d. Steers were weighed and bled on 0, 28, and 56 d of the growing phase. Jugular blood samples were collected prior to the morning feeding in heparinized trace mineral-free Vacutainer tubes (Becton Dickenson Co., Franklin Lakes, NJ).

Finishing Phase. Steers remained on the same dietary treatments as in the growing phase, but were gradually switched (over a 14-d period) to a high concentrate finishing diet shown in Table 1 (basal diets contained 6.1 mg Cu/kg DM, 42.3 mg Zn/kg DM, 49.7 mg Fe/kg DM, 0.19% S, and 0.42 mg Mo/kg DM). Diets were formulated to meet or exceed all nutrient requirements for growing steers with the exception of Cu (NRC, 1996). Diets were fed once daily in the morning in amounts adequate to allow ad libitum access to feed. Daily feed offerings were recorded and feed refusals were measured every 28 d. Steers were weighed and bled on 0, 28, and 56 d of the growing phase. Jugular blood samples were collected prior to the morning feeding in heparinized trace mineral-free Vacutainer tubes (Becton Dickenson Co., Franklin Lakes, NJ).

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hexamer, 500 μM dNTPs, 10 mM DTT, 20 U of RNase inhibitor and 200 U of MMLV reverse transcriptase (Promega, Madison, WI) at 37°C for 60 min. cDNA was amplified with each sense and a T7 promoter-containing antisense primer (Table 2). The antisense primer was designed to contain a 27-bp bacteriophage T7 promoter at the 5′ end of antisense primer. The reactions of PCR were carried out in a 50 μL reaction volume containing 5 μL of the RT product, 2.5 mM MgCl₂, 200 μM dNTPs, 400 nM each of sense and antisense primers, and 2.5 U of Taq DNA polymerase. The thermal cycling parameters of PCR were as follows: 1 cycle at 94°C for 3 min, followed by 35 cycles at 94°C for 60 s, 58°C (ACC, leptin, SCD), and 63°C (UCP2) for 60 s, and 72°C for 60 s with a final extension at 72°C for 5 min. Riboprobes were synthesized by in vitro transcription carried out in a 20 μL reaction volume containing 5 μL of PCR products; 20 U of RNase-inhibitor; 20 U of T7 RNA polymerase (Ambion, Austin, TX); 0.5 mM each of ATP, GTP, and UTP with 0.3 mM CTP; and 0.2 mM biotin-labeled-14-CTP (Gibco BRL, Grand Island, NY). The reaction was performed at 37°C for 2 h. Template DNA was digested using 10 U of RNase-free Dnase I for 15 min at 37°C. Transcripts were denatured by heating at 95°C for 3 min and separated on 5% acrylamide/8 M urea denaturing polyacrylamide gels. After staining with 2.0 μg/mL of acridine orange for 15 min, the full-length riboprobe was eluted from the gel overnight at 37°C, precipitated, and quantified at 260/280 nm.

Adipose tissue total RNA was hybridized with 40 pg of riboprobe per microgram of total RNA in 30 μL of hybridization buffer (80% formamide, 0.4 M NaCl, 1 mM EDTA, 40 mM PIPES, pH 6.8) overnight at 45°C. Single-stranded RNA was then digested with a mixture of 2.5 U/mL RNase A and 100 U/mL RNase T1 for 30 min at 37°C. After inactivation of RNase with 20 μL of 10% SDS and 0.5 μL of 20 mg/mL proteinase K for 20 min at 37°C, protected RNA was extracted with 400 μL of phenol/chloroform/isoamylalcohol (25:24:1) and separated on a 5% acrylamide/8 M urea denaturing polyacrylamide gel.

Table 2. The sequences of sense (S) and antisense (AS) primers used for synthesis of acetyl CoA carboxylase (ACC), stearoyl CoA desaturase (SCD), uncoupling protein 2 (UCP2), and leptin riboprobes

<table>
<thead>
<tr>
<th>Gene</th>
<th>Primer sequences</th>
<th>Length (bp) of riboprobes</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACC</td>
<td>S 5′-GATGGGCGGGATGGTCTCTTTTC-3′ AS 5′-CCAAGGTTCTTCAATAGC-TACTATAGGTAGGCGAGGTCCAGTGACTGATA-3′</td>
<td>436</td>
</tr>
<tr>
<td>SCD</td>
<td>S 5′-TTCCGGAGCTGGCTTTTTCTTCT-3′ AS 5′-CCAAGGTTCTTCAATAGC-TACTATAGGTAGGCGAGGTCCAGTGACTGATA-3′</td>
<td>337</td>
</tr>
<tr>
<td>UCP2</td>
<td>S 5′-CCATGCGGGCGGCTTCTTGT-3′ AS 5′-CCAAGGTTCTTCAATAGC-TACTATAGGTAGGCGAGGTCCAGTGACTGATA-3′</td>
<td>531</td>
</tr>
<tr>
<td>Leptin</td>
<td>S 5′-TCCAGGAGTACACCAAACCC-3′ AS 5′-GGATCCTAATACGACTCACTATAGGGAGCACCCGGGACTGAG-3′</td>
<td>423</td>
</tr>
</tbody>
</table>

Table 3. Effects of supplemental copper source and concentration on performance of growing and finishing steers

<table>
<thead>
<tr>
<th>Item</th>
<th>Control</th>
<th>10 mg CuSO₄</th>
<th>10 mg Availa</th>
<th>20 mg CuSO₄</th>
<th>20 mg Availa</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of animals</td>
<td>10</td>
<td>9</td>
<td>10</td>
<td>9</td>
<td>9</td>
<td>—</td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>259.1</td>
<td>260.2</td>
<td>261.1</td>
<td>258.7</td>
<td>258.4</td>
<td>11.9</td>
</tr>
<tr>
<td>Initial</td>
<td>318.0</td>
<td>320.1</td>
<td>322.4</td>
<td>321.8</td>
<td>323.7</td>
<td>8.9</td>
</tr>
<tr>
<td>End of growing</td>
<td>554.7</td>
<td>550.0</td>
<td>561.0</td>
<td>558.5</td>
<td>558.8</td>
<td>5.0</td>
</tr>
<tr>
<td>ADG, kg</td>
<td>1.05</td>
<td>1.07</td>
<td>1.09</td>
<td>1.12</td>
<td>1.16</td>
<td>0.09</td>
</tr>
<tr>
<td>End of growing</td>
<td>1.64</td>
<td>1.60</td>
<td>1.66</td>
<td>1.64</td>
<td>1.64</td>
<td>0.05</td>
</tr>
<tr>
<td>Feed intake, kg DM/d</td>
<td>8.7</td>
<td>7.8</td>
<td>8.3</td>
<td>9.0</td>
<td>8.2</td>
<td>0.80</td>
</tr>
<tr>
<td>End of finishing</td>
<td>11.2</td>
<td>11.4</td>
<td>9.9</td>
<td>10.9</td>
<td>11.1</td>
<td>0.93</td>
</tr>
<tr>
<td>Feed efficiency (gain:feed)</td>
<td>0.12</td>
<td>0.14</td>
<td>0.13</td>
<td>0.12</td>
<td>0.14</td>
<td>0.02</td>
</tr>
<tr>
<td>End of finishing</td>
<td>0.15</td>
<td>0.14</td>
<td>0.17</td>
<td>0.15</td>
<td>0.15</td>
<td>0.02</td>
</tr>
</tbody>
</table>

*10 mg CuSO₄ vs 10 mg Availa (P < 0.05).
**10 mg CuSO₄ vs 10 mg Availa (P < 0.10).
Table 4. Effects of supplemental copper source and concentration on serum nonesterified fatty acid (NEFA) concentrations (μEq/L) of growing (G) and finishing (F) steers

<table>
<thead>
<tr>
<th>Day of trial</th>
<th>Control</th>
<th>10 mg CuSO₄</th>
<th>10 mg Availa Cu</th>
<th>20 mg CuSO₄</th>
<th>20 mg Availa Cu</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>G 0</td>
<td>172.3</td>
<td>187.7</td>
<td>186.2</td>
<td>184.3</td>
<td>147.7</td>
<td>16.8</td>
</tr>
<tr>
<td>G 28</td>
<td>242.2</td>
<td>197.7</td>
<td>198.9</td>
<td>179.1</td>
<td>155.8</td>
<td>31.8</td>
</tr>
<tr>
<td>G 56</td>
<td>216.1</td>
<td>240.9</td>
<td>199.5</td>
<td>179.8</td>
<td>187.6</td>
<td>24.4</td>
</tr>
<tr>
<td>F 28</td>
<td>168.1</td>
<td>186.9</td>
<td>129.8</td>
<td>163.1</td>
<td>156.6</td>
<td>20.7</td>
</tr>
<tr>
<td>F 56</td>
<td>158.4</td>
<td>190.6</td>
<td>186.1</td>
<td>152.8</td>
<td>164.4</td>
<td>16.9</td>
</tr>
<tr>
<td>F 84</td>
<td>184.3</td>
<td>216.7</td>
<td>236.0</td>
<td>214.5</td>
<td>196.6</td>
<td>19.9</td>
</tr>
<tr>
<td>F 112</td>
<td>211.5</td>
<td>218.9</td>
<td>266.8</td>
<td>154.2</td>
<td>200.5</td>
<td>25.6</td>
</tr>
<tr>
<td>F 144</td>
<td>237.5</td>
<td>199.7</td>
<td>202.9</td>
<td>237.5</td>
<td>168.9</td>
<td>23.2</td>
</tr>
</tbody>
</table>

aNumber of animals = 10 (control), 9 (10 mg CuSO₄), 10 (10 mg Availa Cu), 9 (20 mg CuSO₄), 9 (20 mg Availa Cu).
bG: growing phase, F: finishing phase.

The protected RNA was electrophoretically transferred (Transblot, BioRad, Hercules, CA) onto a positively charged nylon membrane (Schleicher & Schuell Inc., Dassel, Germany) with 0.5×TBE at 400 mA for 1 h. After UV-crosslinking, the membrane was incubated with alkaline phosphatase-conjugated strepavidin (Promega) for 1 hr and incubated with CDP-star (Roche Diagnostics Corp., Indianapolis, IN) for 10 min at room temperature and exposed to X-ray film (Kodak, BioMax, Rochester, NY) for 1 h at room temperature. Autoradiograph was scanned (ScanJet 6100C, Hewlett Packard, Palo Alto, CA) and the image was analyzed using Scion Image software (Scion Corp., Frederick, MD). A ratio was calculated for the intensity of target band vs 18S internal standard band on each lane of the gels.

Statistical Analysis

Statistical analyses of data were performed using analyses of variance for a completely randomized design using the GLM procedure of SAS (SAS Inst. Inc., Cary NC). When treatment was significant \( P < 0.05 \), differences among means were determined using single degree-of-freedom contrasts. Comparisons made were 1) Control vs Cu, 2) 10 mg Cu from CuSO₄ vs 10 mg Cu from Availa Cu, 3) 20 mg from CuSO₄ vs 20 mg from Availa Cu, and 4) 10 mg Cu vs 20 mg Cu.

Results and Discussion

One steer in the 10-mg CuSO₄ treatment died. All data collected from that animal were removed from statistical analysis. Performance was unaffected by dietary Cu treatment during the growing phase (Table 3). During the finishing phase, steers receiving 10 mg Cu/kg DM from Availa Cu had higher \( P < 0.05 \) ending body weights and tended \( P < 0.10 \) to have higher average daily gains than steers receiving 10 mg Cu/kg DM from CuSO₄ (Table 3). Previous research regarding Cu addition to growing and finishing diets fed to steers has shown inconsistent results. In agreement with the present study, Ward et al. (1993) and Engle and Spears (2000a) reported that Cu supplementation at concentrations ranging 5 to 40 mg Cu/kg DM to Angus steers fed a growing diet did not affect gain or feed intake. However, Cu addition at 5.0 mg Cu/kg DM to a corn silage-based diet containing 5.2 mg Cu/kg DM increased average feed intake in Angus steers (Ward and Spears, 1997). Furthermore, Engle and

Table 5. Effects of supplemental copper source and concentration on serum insulin concentrations (μIU/mL) of growing (G) and finishing (F) steers

<table>
<thead>
<tr>
<th>Day of trial</th>
<th>Control</th>
<th>10 mg CuSO₄</th>
<th>10 mg Availa Cu</th>
<th>20 mg CuSO₄</th>
<th>20 mg Availa Cu</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>G 0</td>
<td>16.36</td>
<td>18.83</td>
<td>13.68</td>
<td>13.94</td>
<td>15.86</td>
<td>2.09</td>
</tr>
<tr>
<td>G 56</td>
<td>17.07</td>
<td>19.55</td>
<td>15.38</td>
<td>12.18</td>
<td>16.22</td>
<td>2.68</td>
</tr>
<tr>
<td>F 28</td>
<td>17.32</td>
<td>20.90</td>
<td>22.91</td>
<td>16.27</td>
<td>16.47</td>
<td>2.97</td>
</tr>
<tr>
<td>F 84</td>
<td>15.43</td>
<td>16.41</td>
<td>17.92</td>
<td>16.87</td>
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<td>1.05</td>
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<tr>
<td>F 144</td>
<td>14.93</td>
<td>16.06</td>
<td>19.43</td>
<td>18.91</td>
<td>18.33</td>
<td>1.94</td>
</tr>
</tbody>
</table>

aNumber of animals = 10 (control), 9 (10 mg CuSO₄), 10 (10 mg Availa Cu), 9 (20 mg CuSO₄), 9 (20 mg Availa Cu).
bG: growing phase, F: finishing phase.
Spears (2000b) reported that Cu supplementation to finishing steers at 10 or 20 mg Cu/kg DM had no effect on performance. In another experiment, however, Cu supplementation at 20 or 40 mg Cu/kg DM to finishing steers decreased performance relative to the unsupplemented controls (Engle and Spears, 2000a). Ward and Spears (1997) reported higher average daily gains and feed efficiencies (gain:feed) in finishing steers supplemented with 5 mg Cu/kg DM (basal diet contained 3 mg Cu/kg DM) relative to unsupplemented steers. It is evident that conflicting performance results exist in cattle consuming high concentrate diets supplemented with Cu. The reasons for the discrepancy between results are not clear. There are many factors that could potentially affect an animal’s response to Cu supplementation, such as the Cu concentration of the basal diet, the duration and concentration of Cu supplementation, the absence or presence of dietary Cu antagonists (S, Mo, Zn, and Fe), environmental and health factors, and breed differences in Cu metabolism.

During the 56-d growing phase and 144-d finishing phase, the concentration of blood NEFA was not affected by dietary Cu addition (Table 4). These results are consistent with findings in cattle, in which the addition of 20 mg Cu/kg DM from various Cu sources had no effect on serum triglyceride and NEFA concentrations (Engle et al., 2000a). Serum insulin concentrations from Cu-supplemented steers were not different from those of control (Table 5). At the end of the study, subcutaneous adipose tissue (backfat) ultrasonic measurements were taken over the longissimus muscle between the 11th and 12th rib. Copper-supplemented steers tended (P < 0.11) to have less backfat than controls (1.32 vs. 1.43 cm, SEM = 0.10, respectively). This is in agreement with previous findings.
Figure 2. Densitometric quantification of acetyl CoA carboxylase (ACC), stearoyl CoA desaturase (SCD), uncoupling protein 2 (UCP2), and leptin mRNA from subcutaneous adipose tissue of steers supplemented with Cu. On d 112 of finishing phase, adipose tissue was obtained from three steers of each treatment. Data are expressed as mean ± SD of three animals in each group.

in finishing cattle (Ward and Spears, 1997; Engle et al., 2000a), indicating that Cu addition to finishing diets low in Cu may alter lipid metabolism.

This study is the first to investigate the effects of dietary Cu on gene expression of bovine subcutaneous adipose tissue. The ribonuclease protectin assay of total RNA in bovine subcutaneous adipose tissue revealed the presence of a protected double-stranded RNA at 436, 337, 531, and 423 bp, for ACC, SCD, UCP2, and leptin, respectively (Figure 1). These lengths coincided with those expected. The ACC mRNA levels were not affected by Cu supplementation (Figures 1B and 2). Because this study is the first to examine the effects of Cu on ACC gene expression in ruminants, no literature exists for comparison. This gene is highly regulated by diet, hormones, and other physiological factors. Food intake induces, whereas starvation reduces, the expression of the ACC gene in nonruminants (Abu-Elheiga, et al., 2001), and the products of ACC provide substrates for fatty acid synthesis via fatty acid synthase (FAS). In chicks, high levels of dietary Cu (180 mg/kg DM diet) decrease FAS activity (Konjuha et al., 1997), and, in rats, Cu deficiency increases hepatic FAS mRNA (Wilson et al., 1997). In relation to the physiological mechanism of Cu, Tang et al. (2000) found that Cu deficiency in rats increased hepatic expression of SREBP-1, a strong enhancer of transcription in the FAS promoter region. By contrast, Fields and Lewis (1997) reported that the increase in hepatic lipid synthesis induced by Cu deficiency might be due to iron accumulation, and not due to Cu deficiency itself. Rats consuming diets low in iron had lower hepatic cholesterol and lipogenic enzyme activities (Stang and Kirchgesnner, 1998).

In the current study, dietary Cu concentration did not influence SCD expression in subcutaneous adipose tissue (Figures 1C and 2), supporting previous findings that Cu addition at concentrations ranging from 10 to 40 mg Cu/kg DM to finishing diets low in Cu (basal diet 3 to 5 mg of Cu/kg DM) did not alter the ratio of MUFA/SFA of backfat in steers (Engle et al., 2000c; Engle and Spears, 2000b). Interestingly, Engle et al. (2000b,c) and Engle and Spears (2000b) reported increased proportions of PUFA in total fat in longissimus muscle (but not in subcutaneous adipose tissue) of cattle supplemented with Cu. This may be due to tissue-specific Cu distribution, metabolism, and turnover, which are largely organ specific. The primary sites of Cu accumulation and turnover are liver, brain, and muscle in rats (Levenson, 1998; Linder et al., 1998). Thus, different Cu partitioning, as well as tissue-specific gene expression, may be responsible for the tissue-specific pattern of PUFA between skeletal muscle and adipose tissue. Moreover, pharmacological concentrations of Cu have been shown to alter to the apparent Δ6-desaturase enzyme activity in heart, liver, and muscle in calves (Jenkins and Kramer, 1989). Although Δ6-desaturase enzyme activity and gene expression were not measured in subcutaneous adipose tissue and muscle in the present experiment, further investigation examining the effects of Cu on the activity and expression of Δ6-desaturase enzyme is warranted.
Neither UCP2 nor leptin gene expression were affected by Cu supplementation (Figures 1B,C and 2). As both are involved in maintaining energy balance, this suggests that Cu supplementation did not affect energy metabolism in bovine adipose tissue. Although the effect of Cu on lipolytic activity in adipose tissue was not examined in the current study, others have reported that Cu increases basal and norepinephrine-stimulated lipolysis in sheep (Sinnett-Smith and Woolliams, 1987). However, it seems uncertain whether Cu can modulate lipolysis in subcutaneous adipose tissue because we observed no effects on serum NEFA concentrations. Thus, further study is needed to explain why the decrease in backfat thickness observed in this study as well as previous studies was not accompanied by an increase in serum NEFA concentrations.

Implications

Copper supplementation at levels as low as 10 to 20 mg/kg DM diet has no influence on steer performance and lipid metabolism in subcutaneous adipose tissue, although the amount of supplemental Cu may be too low to induce expression of the genes involved in lipid metabolism. Further research is needed to investigate the effect of Cu on ruminant lipid metabolism in liver, muscle, and different adipose tissue depots.

Literature Cited


