Effects of dietary copper source and concentration on carcass characteristics and lipid and cholesterol metabolism in growing and finishing steers[1,2]

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ABSTRACT: We conducted an experiment to determine the effects of dietary copper (Cu) source and level on carcass characteristics, longissimus muscle fatty acid composition, and serum and muscle cholesterol concentrations in steers. Sixty Angus and Angus × Hereford steers were stratified by weight and initial liver Cu concentration within a breed and randomly assigned to treatments. Treatments consisted of: 1) control (no supplemental Cu); 2) 20 mg Cu/kg DM from Cu sulfate ($CuSO_4$); 3) 40 mg Cu/kg DM from $CuSO_4$; 4) 20 mg Cu/kg DM from Cu citrate; 5) 20 mg Cu/kg DM from Cu proteinate; and 6) 20 mg Cu/kg DM from tribasic Cu chloride. A corn silage-soybean meal-based diet was fed for 56 d. Steers were then switched to a high-concentrate diet. Equal numbers (n = 5) of steers per treatment were slaughtered after receiving the finishing diets for either 101 or 121 d. Serum cholesterol was not affected by treatment during the growing phase but was decreased ($P < .05$) in steers supplemented with Cu by d 84 of the finishing period and remained lower ($P < .05$) at subsequent sampling periods. Longissimus muscle cholesterol concentration tended to be reduced ($P < .11$) by Cu supplementation. Hot carcass weight and backfat were lower ($P < .05$) in animals receiving supplemental Cu. However, Cu-supplemented and control steers had similar marbling scores. Longissimus muscle polyunsaturated fatty acid concentrations (18:2 and 18:3) were increased ($P < .07$) and saturated fatty acid concentrations tended ($P < .11$) to be reduced by Cu supplementation. These results indicate that as little as 20 mg of supplemental Cu/kg diet can reduce backfat and serum cholesterol and increase muscle polyunsaturated fatty acids in steers fed high-concentrate diets.

Key Words: Copper, Steers, Cholesterol, Fatty Acids

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Introduction

Copper (Cu) is an essential element required by cattle and other animals for a number of biochemical functions (Davis and Mertz, 1987). Feeding pharmacological concentrations of Cu well above dietary requirements has altered lipid and cholesterol metabolism in nonruminants. The addition of 125 or 250 mg Cu/kg DM to a control diet adequate in Cu decreased plasma and breast muscle cholesterol concentrations in broilers (Pesti and Bakalli, 1996). Copper supplementation at high concentrations (250 mg Cu/kg DM) to diets adequate in Cu increased unsaturated and decreased saturated fatty acids in backfat of finishing pigs (Amer and Elliot, 1973).

Limited research suggests that dietary Cu at physiological concentrations may affect lipid metabolism in ruminants. Supplementing Cu (from Cu oxide needles) to Cu-deficient sheep led to increased adipose cell volume and increased in vitro lipolytic rates of adipose tissue (Sinnett-Smith and Woolliams, 1987). Recently, Ward and Spears (1997) reported that long-term Cu supplementation to diets marginal in Cu decreased backfat depth and tended to increase longissimus muscle area in steers. The present study was conducted to determine the effects of dietary Cu source and level on carcass characteristics and muscle cholesterol and fatty acid composition in steers.

Materials and Methods

Thirty-six Angus and 24 crossbred Hereford-Angus weaned steers (approximately 6 mo of age, 250 kg initial BW) were used in this experiment. Care, handling, and sampling of the animals herein were approved by

[1]Use of trade names in this publication does not imply endorsement by the North Carolina Agric. Res. Serv. or criticism of similar products not mentioned.

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the North Carolina State University Animal Care and Use Committee. Steers were purchased at feeder calf sales in North Carolina and transported 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), dewormed with Safe Guard (Hoechst-Roussel, Somerville, NJ), and confined in a grass pasture where they were supplemented with corn silage (2.0 kg DM·steer^{-1}·d^{-1}) for 25 d. Steers were then weighed on two consecutive days and allotted, by body weight and breed, to one of five 12-steer pens equipped with individual Calan gate feeders (American Calan, Northwood, NH). Steers were housed in covered, slotted-floor pens (10 × 5 m). All steers were fed for 25 d a corn silage-soybean meal-based diet (basal diet contained 10.2 mg of Cu/kg DM; 54.8 mg of Zn/kg DM) with 10 mg of Mo/kg DM from Na_2MoO_4 supplemented to the diet in order to reduce Cu status prior to initiation of the study (basal diet was the same as the growing diet, with the exception that 10 mg of Mo/kg DM from Na_2MoO_4 was added).

After adjusting to the Calan gate feeding system, steers were weighed on two consecutive days, implanted with Synovex-S (Fort Dodge Animal Health, Fort Dodge, IA), and bled via jugular venipuncture, and liver biopsies were obtained (Pearson and Craig, 1980). Two days later, following liver Cu analysis, steers were allotted to one of six groups based on liver Cu concentration, body weight, and breed. Groups were then randomly assigned to treatments. Treatments consisted of 1) control (no supplemental Cu), 2) 20 mg Cu/kg DM from Cu sulfate ($CuSO_4$), 3) 40 mg Cu/kg DM from $CuSO_4$ 4) 20 mg Cu/kg DM from Cu citrate ($C_6H_4Cu_2O_7$; Griffin Corp., Valdosta, GA), 5) 20 mg Cu/kg DM from Cu proteinate (Chelated Minerals Corp., Salt Lake City, UT), and 6) 20 mg Cu/kg DM from tribasic Cu chloride ($Cu_2(OH)_3Cl$; Micronutrients, Indianapolis, IN).

Steers were fed a corn silage-based growing diet for 56 d (Table 1; basal diet contained 10.2 mg of Cu/kg DM; 54.8 mg of Zn/kg DM). With the exception of Cu, diets were formulated to meet or exceed all nutrient requirements for growing steers (NRC, 1996). Diets were fed once daily in the morning in amounts adequate to allow ad libitum access to feed. Jugular blood samples were collected in nonheparinized Vacutainer tubes (Becton Dickenson, Franklin Lakes, NJ) on d 0, 28, and 56 for serum cholesterol determination.

At the end of the growing phase, steers were implanted with Synovex-Plus and switched over an 8-d period to a high-concentrate finishing diet (Table 1; basal diet contained 4.9 mg of Cu/kg DM and 50.0 mg of Zn/kg DM). Steers remained on the same dietary treatments that they received in the growing phase. Steers were bled on d 0, 28, 56, 84, and 96 and at the termination of the finishing phase. Equal numbers of steers per treatment (the heaviest half from each treatment) were slaughtered after receiving the finishing diets for either 101 or 121 d. Final weights were obtained on two consecutive days, and steers were then transported approximately 320 km to a commercial abattoir and slaughtered after an overnight period without feed. A final liver sample was obtained from each animal postmortem. Hot carcass weight was determined on the day of slaughter. Fat depth over the longissimus muscle (between the 12th and 13th ribs), estimated percentage of kidney, pelvic, and heart (**KPH**) fat, longissimus muscle area (**LMA**), bone maturity, marbling score, and USDA quality grade (**QG**) and yield grade (**YG**) were determined by a certified USDA grader 48 h after slaughter. After carcass grading, a longissimus muscle sample approximately 1.5 cm in depth encompassing the entire longissimus muscle surface area was sliced from the right side of the carcass medially from the area of back fat measurement to the spine of the carcass (approximate weight, 50 g). Samples were placed in plastic bags and immediately chilled on ice. Upon arrival at the laboratory, samples were frozen at –80°C until they were analyzed for total lipid, fatty acid composition, and cholesterol and Cu concentrations.

Table 1. Ingredient composition of basal diets

Ingredient	Growing	Finishing
	%[a]	
Corn silage	88.61	—
Soybean meal (48% CP)	9.00	6.00
Cottonseed hulls	—	5.00
Urea	1.00	.75
Calcium sulfate	.80	.80
Ground corn	.31	86.84
Salt	.20	.20
Calcium carbonate	.07	.40
Vitamin premix[b]	.01	.01
Mineral premix[c]	+	+
Monensin[d]	—	+

[a]Dry matter basis.
[b]Contained per kilogram of premix: 6,600,000 IU of vitamin A, 3,520,000 IU of vitamin D, and 6,600 IU of vitamin E.
[c]Provided per kilogram of diet: 30 mg of Zn as $ZnSO_4$, 20 mg of Mn as $MnSO_4$, .5 mg of I as $Ca(IO_3)_2(H_2O)$, .1 mg of Co as $CoCO_3$, and .1 mg of Se as Na_2SeO_3.
[d]Provided 33 mg of monensin/kg DM.

Analytical Procedures. Serum samples were analyzed for total cholesterol, high-density lipoprotein (**HDL**), and low-density lipoprotein (**LDL**) concentrations using the methods described by Sigma Chemical Co. (1995, 1997, and 1994, respectively). Triglyceride concentrations were determined enzymatically (Sigma Chemical Co., 1990), and NEFA concentrations were determined via an enzymatic, colorimetric method (Wako Chemicals, 1995).

Longissimus muscle (with visible external fat dissected) and liver tissue samples were thawed. The longissimus muscle and the final liver samples obtained at slaughter were diced into small pieces, mixed thoroughly, and then randomly sampled. Longissimus muscle and final liver subsamples and liver biopsy samples in their entirety were dried at 100°C for 48 h, weighed, and then prepared for Cu analysis using a microwave

digestion (Model MDS-81D, CEM, Matthews, NC) procedure described by Gengelbach et al. (1994). Copper concentration was determined using flame atomic absorption spectrophotometry (model 5000, Perkin-Elmer, Norwalk, CT). Triplicate 1-g subsamples of longissimus muscle and duplicate 1-g subsamples of final liver samples obtained at slaughter were taken from each tissue sample per animal and used for lipid extraction. Lipid extraction was accomplished via the chloroform:methanol procedure of Bligh and Dyer (1959). After extraction, the lipid-containing fraction was dried under N gas and placed in a 100°C drying oven for 3 h. The preweighed vial containing the dried lipid extract was then allowed to cool in a desiccator and weighed to determine percentage lipid. Total longissimus muscle cholesterol content was determined with the enzymatic method of Allain et al. (1974) as modified by Salé et al. (1984).

Fatty acid composition of longissimus muscle was determined with gas chromatography using a Hewlett Packard (Avondale, PA) Model 5890A Series II gas chromatograph fixed with a 6B90 series injector and flame ionization detector. The instrument was equipped with a 100-m × .25-mm (i.d.) fused silica capillary column (SP-2560, Supelco, Bellefonte, PA). Methyl ester derivatives of the fatty acids were prepared using a combination of $NaOCH_3$ followed by HCl/methanol as described by Kramer et al. (1997). Fatty acid methyl ester preparations were injected (1 μL) using the split mode. The carrier gas was helium, and the split ratio was 100:1 at 180°C. The oven temperature was programmed from an initial temperature of 140°C (0 min) to a final temperature of 225°C at the rate of 2.8°C/min. The final temperature was held for 18 min. Chromatograms were recorded with a computing integrator (Millennium chromatograph manager, Waters/Millipore, Bedford, MA). Standard fatty acid methyl ester mixtures were used to calibrate the gas chromatograph system using reference standards KEL-FIM-FAME-5 (Matreya, Pleasant Gap, PA). Identification of the fatty acids was made by comparing the relative retention times of fatty acid methyl ester peaks from samples with those of standards. These were calculated as normalized area percentages of fatty acids.

Statistical Analysis. Statistical analyses of data were performed using analyses of variance for a randomized complete block (breed) design and the GLM procedure of SAS (1988). The model for average daily gain, feed intake, feed efficiency, serum and tissue cholesterol concentrations, and tissue copper concentrations contained treatment, breed, and treatment × breed interactions. There were no breed effects or treatment × breed interactions. Carcass and fatty acid composition data were analyzed as a split plot design. The model included treatment, time, breed, and all possible interactions. There were no time or breed effects or treatment × time, breed × time, treatment × breed, or treatment × time × breed interactions. When treatment was significant ($P < .10$), differences among means were determined using single degree of freedom orthogonal contrasts. Comparisons made were 1) control vs Cu, 2) 20 mg Cu from $CuSO_4$ vs 40 mg Cu/kg DM from $CuSO_4$, 3) 20 mg Cu/kg DM from $CuSO_4$ vs 20 mg Cu/kg DM from Cu citrate, 4) 20 mg Cu/kg DM from $CuSO_4$ vs 20 mg Cu/kg DM from Cu proteinate, and 5) 20 mg Cu/kg diet DM from $CuSO_4$ vs 20 mg Cu/kg DM from tribasic Cu chloride.

Results and Discussion

One animal from the Cu proteinate treatment broke its leg and was removed from the study. All data collected from this animal were omitted from the statistical analyses. Steer performance and liver plasma Cu concentrations are reported elsewhere (Engle et al., 1998). Briefly, steer performance was not affected by treatment during the 56-d growing period. Supplemental Cu during the finishing phase, regardless of the level or source, reduced ($P < .05$) gain by 19%, feed intake by 6.3%, and gain:feed by 14%. As expected, liver Cu concentrations were higher in the steers receiving 20 or 40 mg Cu/kg DM. However, liver and plasma Cu concentrations in control steers (63.2 ± 4.1 mg of Cu/kg DM and .80 ± .03 mg of Cu/L, respectively) remained above concentrations considered to be indicative of Cu deficiency (liver < 20 mg of Cu/kg DM and plasma < .6 mg of Cu/L; Mills, 1987).

Serum cholesterol concentrations were unaffected by dietary treatment during the growing phase (Table 2). During the finishing phase, serum cholesterol concentrations were unaffected through d 56 but were lower ($P < .05$) on d 84, 96, and 116 in steers supplemented with Cu than in the controls (Table 2). These results are consistent with findings in broilers, in which the addition of much higher concentrations of Cu (125 to 250 mg Cu/kg DM) reduced plasma cholesterol (Pesti and Bakalli, 1996). On d 84 and 116, steers supplemented with 20 mg Cu/kg DM from tribasic Cu chloride had higher serum cholesterol concentrations than steers supplemented with iso-amounts of $CuSO_4$. Serum cholesterol was not further reduced when supplemented Cu from $CuSO_4$ was increased from 20 to 40 mg/kg.

On d 96 of the finishing phase, LDL and HDL cholesterol concentrations were determined (Table 3). High density lipoprotein and LDL cholesterol concentrations tended to be reduced ($P < .11$) in animals receiving supplemental Cu. Serum triglycerides and NEFA were not affected by treatment (Table 3). Plasma triglyceride concentrations were decreased in broilers fed 250 mg Cu/kg (Bakalli et al., 1995).

Longissimus muscle cholesterol concentration tended to be reduced ($P < .11$) by Cu supplementation (Table 4). Copper concentrations in muscle and percentage lipid in muscle and liver were not affected by treatment (Table 4). Compared with controls, liver Cu concentrations were over threefold higher ($P < .05$) at the end of the study in steers that had received 20 mg of supplemental Cu/kg of diet (Table 4). Steers receiving 40 mg

Table 2. Effects of copper concentration and source on serum total cholesterol concentrations (mg/dL) in steers[a]

	Added Cu, mg Cu/kg DM						
Item	Control, 0	$CuSO_4$, 20	$CuSO_4$, 40	Cu citrate, 20	Cu proteinate, 20	Cu chloride, 20	SEM
Growing phase							
d 0	67.6	61.7	67.4	58.6	56.5	72.3	6.3
d 28	83.6	84.8	87.4	90.7	82.2	86.5	7.1
d 56	116.1	120.3	107.3	112.5	118.4	120.4	6.4
Finishing phase							
d 0	116.1	120.3	107.3	112.5	118.4	120.4	6.4
d 28	79.4	89.0	87.0	82.4	95.0	94.9	6.3
d 56	84.0	85.4	80.6	83.8	78.4	90.1	7.5
d 84[bc]	106.7	81.0	74.0	74.4	77.8	100.8	7.9
d 96[b]	104.7	74.2	73.9	64.4	73.7	75.6	8.1
d 116[bc]	109.3	76.3	89.5	94.3	87.7	100.5	8.5

[a]Prior to slaughter of the first group of steers on d 101 means are based on n = 10 animals/treatment, except for Cu proteinate (n = 9).
[b]Control vs copper ($P < .05$).
[c]20 mg $CuSO_4$ vs 20 mg Cu chloride ($P < .05$).

Cu/kg had higher ($P < .05$) liver Cu concentrations than those receiving 20 mg Cu/kg diet from $CuSO_4$ (Table 4).

Different chemical forms of Cu have been shown to be more bioavailable than $CuSO_4$. Dove (1998) reported that Cu citrate stimulated growth in weanling pigs at concentrations half those required for maximal growth with $CuSO_4$. In steers fed diets high in Mo and S, tribasic Cu chloride was more bioavailable than $CuSO_4$ (Spears et al., 1997). However, in the absence of high levels of antagonists both sources had similar bioavailabilities. Ward et al. (1996) have shown Cu proteinate to be more available than iso-amounts of $CuSO_4$ in heifers fed diets high in Cu antagonists (sulfur and molybdenum). However, few Cu source effects were detected in the present study.

The elevated liver Cu concentrations in Cu-supplemented steers may in part explain the decreased serum cholesterol and trend for decreased muscle cholesterol observed. Kim et al. (1992) have shown, in rats, that Cu deficiency causes hypercholesterolemia by increasing hepatic reduced glutathione (**GSH**) concentrations, which increase the activity of 3-hydroxy-3-methyl-glutaryl coenzyme A (**HMG-CoA**) reductase, the rate-limiting enzyme in cholesterol synthesis. It has been hypothesized that high concentrations of liver Cu regulate cholesterol biosynthesis indirectly by decreasing the reduced form of glutathione (GSH) and increasing the oxidized form of glutathione (**GSSG**; Kim et al., 1992; Bakalli et al., 1995). The decrease in cellular GSH is thought to protect hepatic cells from the harmful free radical-forming toxic effects of high Cu. As Cu enters the liver cells, it is rapidly complexed by GSH, and then it is transferred to metallothionein, the form in which it is stored (Freedman et al., 1989). Increasing cellular GSSG concentrations has been shown to decrease the activity of HMG-CoA reductase (Gilbert and Stewart, 1981; Roitelman and Schechter, 1984; Ziegler, 1985; Gilbert, 1990). With a decrease in HMG-CoA reductase activity, carbon flux through the mevalonate pathway would be reduced, thus decreasing cholesterol synthesis. In most mammals, the primary site of endogenous cholesterol synthesis is the liver (Siperstein, 1970). However, in ruminants the primary site of cholesterol synthesis is the small intestine and adipose tissue, and the liver produces a small proportion of the total endogenous cholesterol (Liepa et al., 1978). The absorption of Cu from the intestinal lumen into the enterocyte is thought to require mechanisms similar to those in the

Table 3. Effects of copper concentration and source on serum lipoprotein, triglycerides, and nonesterified fatty acids

	Added Cu, mg Cu/kg DM						
Serum measurement[a]	Control, 0	$CuSO_4$, 20	$CuSO_4$, 40	Cu citrate, 20	Cu proteinate, 20	Cu chloride, 20	SEM
HDL cholesterol, mg/dL[b]	86.0	62.2	64.4	55.4	64.2	63.2	11.1
LDL cholesterol, mg/dL[b]	8.0	7.2	7.3	7.0	7.0	7.2	.4
Triglycerides, mg/dL	22.2	21.0	23.0	23.9	21.8	21.2	2.4
NEFA, m*M*	.28	.24	.34	.36	.39	.30	.03

[a]Day 96 of finishing phase.
[b]Control vs copper ($P < .11$).

Table 4. Effects of copper concentration and source on tissue cholesterol, lipid, and copper concentration

	Added Cu, mg Cu/kg DM						
Item	Control, 0	$CuSO_4$, 20	$CuSO_4$, 40	Cu citrate, 20	Cu proteinate, 20	Cu chloride, 20	SEM
Longissimus muscle							
% DM	30.5	27.4	30.3	30.9	29.7	29.1	1.3
Cholesterol, mg/100 g wet wt[a]	74.5	70.6	65.4	66.6	64.2	68.0	6.4
Lipid, % wet wt	4.5	4.0	4.6	5.0	4.1	4.2	.6
Copper, mg/kg DM	3.5	4.2	3.0	5.5	3.7	4.4	.8
Liver							
% DM	28.2	27.9	26.8	27.3	27.6	29.0	.6
Lipid, % wet wt	3.1	3.3	2.8	2.8	2.5	2.5	.4
Copper, mg/kg DM							
Initial	75.0	80.0	72.2	78.8	86.4	77.7	24.3
Final[bc]	63.2	290.3	379.6	253.6	339.1	246.6	22.3

[a]Control vs copper ($P < .11$).
[b]Control vs copper ($P < .05$).
[c]20 mg $CuSO_4$ vs 40 $CuSO_4$ ($P < .05$).

liver. Therefore, Cu may have reduced cholesterol synthesis in both the liver and intestine (via similar mechanisms), resulting in an overall reduction in serum cholesterol.

Dressing percentage, KPH fat, LMA, QG, and YG were unaffected by dietary treatment (Table 5). Hot carcass weight and backfat were lower ($P < .05$) in animals receiving supplemental Cu. However, Cu-supplemented steers and control steers had similar marbling scores (Table 5). This is consistent with the similar total lipid content of muscle samples from control and Cu-supplemented steers (Table 4). The decrease in backfat depth observed in steers receiving supplemental Cu is similar to that observed by Ward and Spears (1997). Konjufca et al. (1997) observed a decrease in fatty acid synthetase in poultry fed elevated levels of Cu. A decrease in fatty acid synthesis could explain the decreased backfat observed. The observed reduction in backfat in Cu-supplemented steers could also have been caused by the reduced performance observed in the animals receiving supplemental Cu (Engle et al., 1998). However, animals with a reduced backfat depth usually have less marbling than animals with greater backfat depth of the same contemporary group. Steers supplemented with 20 mg Cu/kg DM from $CuSO_4$ had higher ($P < .05$) marbling scores than steers supplemented with 20 mg of Cu/kg DM from Cu citrate and Cu proteinate (Table 5).

Longissimus muscle polyunsaturated fatty acids, 18:2, and 18:3 were increased ($P < .07$) and saturated fatty acids tended to be decreased ($P < .11$) in steers supplemented with Cu (Table 6). Steers supplemented with 20 mg of Cu from Cu proteinate had a higher ($P < .05$) fatty acid percentage of 22:1 in longissimus muscle than steers supplemented with 20 mg of Cu from $CuSO_4$. Steers supplemented with Cu had a lower percentage of 18:1 *trans* than control steers (Table 6). Desaturase activity was not measured in the present study. However, feeding pharmacological concentrations of Cu has increased estimated desaturase activity in pigs

Table 5. Effects of copper concentration and source on carcass characteristics of finished steers

	Added Cu, mg Cu/kg DM						
Item	Control, 0	$CuSO_4$, 20	$CuSO_4$, 40	Cu citrate, 20	Cu proteinate, 20	Cu chloride, 20	SEM
Marbling[afg]	4.9	5.5	5.2	4.8	4.8	5.2	.18
Dressing percentage	57.3	58.6	59.7	58.4	57.5	58.7	.88
Hot carcass wt, kg[e]	324.5	307.1	306.0	311.6	302.3	312.8	6.6
12th rib backfat, cm[e]	1.3	1.1	1.1	.9	.9	1.1	.09
KPH, %[b]	1.8	1.9	2.0	1.7	1.8	1.5	.13
USDA yield grade	2.0	2.0	2.5	1.8	2.0	2.2	.22
LMA, cm^2 [c]	80.6	80.0	75.9	82.9	80.0	82.2	2.9
USDA quality grade[d]	16.3	17.1	16.8	15.7	16.3	17.3	.58

[a]4 = slight; 5 = small; 6 = modest.
[b]Kidney, pelvic, and heart fat.
[c]Longissimus muscle area.
[d]$Select^+$ = 16; $Choice^-$ = 17; Choice = 18.
[e]Control vs copper ($P < .05$).
[f]20 mg $CuSO_4$ vs 20 mg Cu citrate ($P < .05$).
[g]20 mg $CuSO_4$ vs 20 mg Cu proteinate ($P < .05$).

Table 6. Effects of copper concentration and source on fatty acid composition of longissimus muscle of finished steers

	Added Cu, mg Cu/kg DM						
Fatty acids, % by weight	Control, 0	$CuSO_4$, 20	$CuSO_4$, 40	Cu citrate, 20	Cu proteinate, 20	Cu chloride, 20	SEM
	%						
14:0	3.44	3.56	3.91	3.34	3.46	3.56	.26
14:1	.58	.64	.84	.62	.69	.65	.11
15:0[a]	.63	.55	.74	.67	.52	.59	.07
16:0	27.68	27.65	26.96	27.20	26.20	27.14	1.34
16:1 *cis*	3.48	3.80	4.26	4.41	3.80	3.64	.28
17:0	1.89	1.63	2.02	1.71	1.61	1.83	.21
18:0	13.62	11.71	11.32	11.78	12.45	12.04	1.02
18:1 *trans*[b]	7.54	6.25	6.32	5.97	6.30	6.59	.42
18:1 *cis*	32.42	35.73	35.05	33.73	33.14	34.91	1.54
18:2[c]	2.35	3.31	3.21	3.49	3.40	3.39	.38
18:3[c]	.07	.20	.15	.16	.19	.12	.04
20:0	4.98	4.52	4.61	5.14	5.31	5.01	.74
22:1[d]	1.30	.41	.63	1.79	2.92	.53	.95
Unsaturated	47.73	50.35	50.47	50.18	50.44	49.83	1.4
Saturated[e]	52.23	49.63	49.56	49.84	49.55	50.15	1.1
Unsaturated:saturated[e]	.91	1.02	1.02	1.01	1.02	.99	.06
Monounsaturated	45.32	46.84	47.11	46.84	46.84	46.31	1.3
Polyunsaturated[f]	2.41	3.51	3.36	3.66	3.59	3.51	.56

[a]20 mg $CuSO_4$ vs 40 mg $CuSO_4$ ($P < .09$).
[b]Control vs copper ($P < .05$).
[c]Control vs copper ($P < .07$).
[d]20 mg $CuSO_4$ vs 20 mg Cu proteinate ($P < .05$).
[e]Control vs copper ($P < .11$).
[f]Control vs copper ($P < .10$).

(Thompson et al., 1973). In calves fed 1,000 mg of Cu/L of milk replacer, apparent Δ-9-desaturase enzyme activity in liver and heart was greater than that in control calves fed 10 mg of Cu/L of milk replacer (Jenkins and Kramer, 1989). Findings by Jenkins and Kramer (1989) are consistent with reports of high dietary Cu promoting Δ-9-desaturase activity in rat and swine liver (Cunnane, 1982).

Copper supplementation may have altered microbial biohydrogenation of fatty acids in the rumen, thus allowing a greater amount of unsaturated fatty acids to be absorbed by the lower gut and less 18:1 *trans* produced in the biohydrogenation process (Christie, 1981). Essig et al. (1972) found that supplementing Angus steers fed a 75% concentrate diet (adequate in Cu) with 4.4 g of $CuSO_4$ per 100 kg BW (via bolus) decreased ($P < .05$) acetic, propionic, butyric, and total VFA concentrations 2 h after feeding. Although short-chain fatty acids were not measured in this study, it is evident that Cu can alter rumen microbial fermentation.

The present study substantiates earlier research (Ward and Spears, 1997) indicating that dietary Cu affects backfat in finishing cattle. The 15 to 31% decrease in backfat observed in the present study would result in less trim wastage during processing. To our knowledge, this is the first research indicating that dietary Cu at concentrations probably within the physiological range (especially 20 mg/kg treatments) may increase polyunsaturated fatty acids and reduce cholesterol in beef produced for human consumption.

Implications

Copper supplementation at levels as low as 20 mg of supplemental copper/kg of diet can alter carcass backfat and cholesterol and lipid metabolism in steers fed high-concentrate diets. The potential for reducing cholesterol and altering fatty acid composition (saturated:unsaturated ratio) in beef produced for human consumption has many health benefits. A reduction in backfat would decrease the amount of trim required at processing and ultimately reduce wastage per carcass. Further research is needed to determine the role of copper on growth and lipid and cholesterol metabolism in beef cattle.

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