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Effects of Dietary Conjugated Linoleic Acid on Lipid Peroxidation in Breast and Thigh Muscles of Broiler Chickens

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ABSTRACT

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The effect of dietary supplementation with conjugated linoleic acid (CLA) on lipid peroxidation in the breast muscles (*Pectoralis major* and *Pectoralis minor)* and thigh muscles of broiler chickens was investigated. A total of ninety-six 21-day-old Arbor Acres male broiler chickens were assigned to 2 dietary treatments (0 and 1.5% CLA) with 8 replicates per 6 chickens each. The left breast and thigh muscles of broiler chickens were excised on day 42. The broilers receiving the CLA diet exhibited lower malondialdehyde content and reactive oxygen species production ($P < 0.01$) in the breast and thigh muscles than the broilers receiving the control diet. After the dietary CLA supplementation, the glutathione (GSH) content in the breast and thigh muscles of the broiler chickens fed the CLA diet increased by 21.89 and 21.56%, respectively ($P < 0.05$) while the γ-glutamylcysteine synthetase（γ-GCS）activity increased by 28.57 and 25.80% ($P < 0.05$), respectively. Dietary CLA significantly increased the CLA content ($P < 0.01$) and saturated fatty acid content ($P < 0.05$), and decreased the monounsaturated fatty acid content ($P < 0.01$) in the breast and thigh muscles. These results showed that dietary CLA may decrease the lipid peroxidation level in the breast and thigh muscles of broiler chickens perhaps through increasing the γ-GCS activity to induce GSH synthesis and changing the fatty acid composition to increase oxidative stability.

Keywords: conjugated linoleic acid; diet effect; chicken meat; redox status; reactive oxygen species

Conjugated linoleic acid (CLA) refers to a class of spatial and geometric isomers of linoleic acid containing conjugated double bond, belonging to the polyunsaturated fatty acids (PUFA) family members. CLA has such physiological activities as immune modulation, anti-cancer effects, prevention of cardiovascular diseases and diabetes, and weight control.

In view of the above health-related effects of CLA, it is desirable to provide CLA-enriched products for human consumption. Ruminant products show relatively high concentrations of CLA (0.5–1.5% of total fatty acids), whereas meats from monogastric animals are poor sources of CLA (0.1–0.2% of total fatty acids) (Chin et al. 1992; Lin et al. 1995). Chicken meat, however, is an ideal candidate for CLA enrichment by feeding synthetic CLA, because CLA will not be further saturated before absorption and its deposition in tissues is relatively highly efficient. It has already

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been demonstrated that dietary CLA was readily incorporated in muscle tissue lipids of chickens (Szymczyk et al. 2001; Du and Ahn 2002).

While dietary CLA was incorporated in muscle tissue lipids of chickens, it decreased the lipid peroxidation level of muscle tissue. The increase of thiobarbituric acid reactive substances (TBARS) values, the lipid peroxidation maker for the minced breast muscle was inhibited by dietary CLA (Kawahara et al. 2009). TBARS values in the breast and thigh muscles of broiler chickens were significantly influenced by dietary CLA and storage time, and dietary CLA significantly decreased TBARS in breast and thigh meat after a 7-day storage at 4°C (Bolukbasi 2006). Moreover, Du et al. (2001) reported that after cooking and irradiation, cooked meat patties from hens fed CLA diet had lower malondialdehyde (MDA) content and produced less hexanal and pentanal than the control. Narciso-Gaytan et al. (2011) found that the lipid oxidation stability of vacuum-packed chicken meat cooked at 74°C was higher in the CLA treatment than in the menhaden fish oil and flaxseed oil treatments.

So far, little has been known about the mechanism by which dietary CLA decreases the lipid peroxidation level of chicken muscle. Because lipid peroxidation is the result of unsaturated fatty acids being attacked by free radicals, it was rational to postulate that dietary CLA may influence lipid peroxidation in the muscle of broiler chickens partly through affecting the reactive oxygen species (ROS) production. Although several studies revealed the antioxiadant properties of CLA (Palacios et al. 2003; Kim et al. 2005), the literature about the relationship between dietary CLA and ROS in poultry science is lacking.

The purpose of this study was to determine the effects of dietary CLA on the lipid peroxidation level, ROS, glutathione (GSH) content, antioxidant enzymes activities, and fatty acids composition of the breast and thigh muscles of broiler chickens.

MATERIAL AND METHODS

Experimental design. The animal management protocol for this research was approved by the Henan University of Animal Husbandry and Economy Animal Care and Use Committee. One-day-old Arbor Acres male broiler chickens were purchased from Lvye Poultry Husbandry Co., Ltd. (Guxing, China) and fed the same corn-soybean meal diet until the end of the third week. At 21 days of age (695 ± 35 g), ninety-six Arbor Acres male broiler chickens were randomly distributed into 2 dietary treatments, fed either the control or the 1.5% CLA-supplemented diet. Each treatment group had 8 replicates of 6 chickens each.

A corn-soybean meal diet was used, and energy was adjusted using soybean oil. In the CLA diet, 1.5% soybean oil was replaced by 1.5% CLA to keep both the CLA-supplemented diet and the control diet isoenergetic. The 1.5% CLA inclusion level was chosen as previous findings showed that with higher inclusion levels the CLA deposition efficiency decreased (Du and Ahn 2002; Suksombat et al. 2007; Narciso-Gaytan et al. 2011). The accumulation of CLA in meat increases linearly with the CLA level (0~1.5%) increasing in the diet. However, when dietary CLA exceeds 1.5%, the deposition efficiency of CLA begins to decrease. The diet compositions in weeks 1–3 and the control diet in weeks 4–6 are presented in Table 1. The experimental diets were formulated to meet or exceed the National Research Council (1994) requirements. The CLA was provided by the Qingdao Aohai Biology Technology Co., Ltd. (Qingdao, China) and contained 80.80% conjugated isomers (*c*9, *t*11 = 39.24; *t*10, *c*12 = 38.93; other CLA isomers = 2.63).

The present experiment was conducted at the experimental farm of the Henan University of Animal Husbandry and Economy. The broilers were housed in wire cages (1.4 m × 0.7 m × 0.4 m) and maintained on a 16 h light : 8 h darkness program. Temperature in the poultry house was set at 31~33°C for the first 5 days and was reduced by 2~3°C each week until 22°C. The broilers were vaccinated according to the routine immunization program for broilers, and had free access to feed and water throughout the experiment.

Average daily gain, average daily feed intake, and feed conversion ratio. At the end of the third week and the sixth week, the body weights and feed consumption of broiler chicken were recorded on a per replicate basis. Average daily gain (ADG), average daily feed intake (ADFI), and feed conversion ratio (FCR) were calculated for this period.

Breast and thigh muscles sampling. On day 42, body weights were recorded after a 12 h fasting, and one bird from each replicate with body weight close to the mean was selected. The slaughter-

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ing process was carried out in the experimental slaughterhouse of the Henan University of Animal Husbandry and Economy to avoid birds' suffering from stress due to long transport time, according to the specifications outlined by the China Poultry Breeding Committee. Birds were allowed bleeding for 2 min after stunning, and then were scalded in water at 60°C for 2 min before feather plucking by a machine, evisceration, and tissue sample collection. The whole left breast (*Pectoralis major* and *Pectoralis minor*) and thigh muscles were removed from each carcass at about 30 min postmortem, and were trimmed of visible skin, dissectible fat, and connective tissues. The muscle samples were separately ground for 20 s at 1500 rpm by a JYS-A900 meat grinder (Joyoung, China), and then vacuum-packed and stored in a BCD-572WDPM freezer (Qingdao Haier Group, China) at –30°C for 7 days until further analysis.

Preparation of muscles homogenate and measurement of antioxidant enzyme activity, MDA and GSH content, γ-glutamylcysteine synthetase activity, and ROS. After thawing for 2 h at room temperature, muscle samples were homogenized (10%, w/v) in a 0.88% sodium chloride water solution. A tissue homogenizer PT1200E (Shanghai Sigaole Biological Technology Co. Ltd., China) was set at 4000 rpm for 5 min. The homogenate was centrifuged at 3500 *g* for 20 min at 4°C, and the supernatant was collected, sub-packed in 0.5 ml tubes, and stored at –30°C until further analysis.

Total superoxide dismutase (TSOD) activity was assayed according to the methods of Spitz and Oberly (1989). Units of SOD activity were defined by the amount of enzyme required to inhibit the rate of formazan dye formation by 50% under defined conditions. The catalase (CAT) activity was measured according to the method of Aebi (1984). This method used the change in absorbance at 240 nm and 25°C of the solution of 10 mmol/l H_2O_2 in 50 mmol/l phosphate buffer, pH 7.0. The decrease in absorbance per unit time is a measure of the CAT activity. The glutathione peroxidase (GSH-Px) activity was assayed by the method of Lawrence and Burk (1976) using hydrogen peroxide as substrate. One unit of GSH-Px is expressed as the amount of GSH-Px needed to oxidize 1 mmole of nicotinamide adenine dinucleotide phosphate (NADPH) per min. The marker of lipid peroxidation, MDA, was quantified by measuring the TBARS with a spectrophotometer set at 535 nm. GSH was measured according to the two-two nitrobenzoic acid and thiol compound reaction. γ-Glutamylcysteine synthetase (γ-GCS) activity was assayed by catalyzing glutamic acid and cysteine to synthesize γ-glutamyl cysteine and monitoring changes in absorbance of NADPH at 340 nm. Commercial kits were purchased from

Table 1. Ingredients, chemical composition, and energy content of the control diet (in g/kg)

Ingredient	Weeks 1–3	Weeks 4–6	Nutrition levels	Weeks 1–3	Weeks 4–6
Maize	600.0	657.6	metabolizable energy (MJ/kg)	12.20	12.42
Soybean meal	342.1	285.0	crude protein	199.3	179.8
Soybean oil	20.0	20.0	calcium	9.2	9.0
Limestone	13.0	13.0	available P	4.2	4.1
$CaHPO_4$	16.0	16.0	lysine	10.5	9.2
DL-Methionine	2.0	2.0	methionine	5.0	4.7
Sodium chloride	3.5	3.5	methionine + cysteine	8.5	8.0
Choline chloride (50%)	1.0	1.0			
Multi-vitamin premix[1]	0.2	0.2			
Trace-minerals premix[2]	2.0	2.0			
BHT (33%)	0.2	0.2			
Total	1000.0	1000.0			

BHT = 2,6-ditert-butyl-4-methylphenol

[1]multi-vitamin premix provided per kg of diet: retinyl acetate 4.3 mg, cholecalciferol 0.0625 mg, α-tocopherol 18.75 mg, menadione sodium bisulphite 2.65 mg, thiamine-HCl 2.0 mg, pyridoxine-HCl 6.0 mg, cyanocobalamin 0.025 mg, niacin 50 mg, D-pantothenic acid 12 mg, folic acid 1.25 mg

[2]trace-minerals premix provided per kg of diet: Cu 8 mg, Fe 80 mg, Mn 100 mg, Zn 75 mg, Se 0.15 mg, I 0.35 mg

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Table 2. Effect of dietary CLA supplementation on the growth performance of broiler chickens over a 21-day experimental period

Items	ADG (g)	ADFI (g)	FCR	Significance
Control diet	61.1 ± 3.89	128.40 ± 6.85	2.10 ± 0.09	ns
CLA diet	60.8 ± 3.85	126.62 ± 3.92	2.08 ± 0.10	ns

CLA = conjugated linoleic acid, ADG = average daily gain, ADFI = average daily feed intake, FCR = feed conversion ratio, ns = non-significant

the Nanjing Jiancheng Biological Engineering Research Institute.

The ROS production in the breast and thigh muscles homogenate was detected according to the method of Cao et al. (2005). The 0.25 g of muscle was fully homogenated for 2 min in 1.5 ml phosphate buffer (0.32 mol/l sucrose, 10 mmol/l Hepes, 10 mmol/l α-phenyl-N-*tert*-butyl nitrone, 2 mmol/l 0.05% Tween 80, 5 mmol/l 2-mercaptoethanol, pH 7.4), and centrifuged at 13 200 *g* and 4°C for 20 min. The supernatant was incubated at 37°C for 1 h and then extracted by ethyl acetate. Electron spin resonance (ESR) spectra were measured at room temperature with an ER200D-SRC spectrometer (Bruker, USA). The mean height of three peaks in each signal was taken as the relative intensity of the ROS signal.

Intramuscular fat and fatty acid compositions of the breast and thigh muscles. The intramuscular fat contents in the breast and thigh muscles were measured using the Soxhlet petroleum-ether extraction method according to the Chinese National Standards GB/T 5009.6.2004, and the intramuscular fat content was determined as a weight percentage.

The fatty acids of the breast and thigh muscles were extracted and methylated according to a one-step method (Sukhija and Palmquist 1988), except that n-hexane replaced benzene for extraction. The fatty acid analysis was done on an Agilent 6890 Series EGC System gas chromatograph equipped with a HP Innowax (30 m × 0.32 mm inside diameter) cross-linked polyethylene glycol column and FID detector (Agilent Technologies, USA). The samples were analyzed under the following operating conditions: injector and detector temperature 250°C, programmed column temperature changed from the initial 210°C for 10 min, with a 2°C/min rate to the final temperature of 220°C, nitrogen gas flow 5 ml/min, hydrogen 40 ml/min, air 450 ml/min. Fatty acids were identified by comparison of their retention times with that of the internal standard (C17:0, Fluka 51633). The results were expressed as a percentage of total fatty acid methyl esters.

Peroxidability index. The peroxidability index (PI) was calculated according to the methods of Pirozhkov et al. (1992):

$$PI = A \times 0.025 + B \times 1 + C \times 2 + D \times 4 + E \times 6 + F \times 8$$

where:

A, B, C, D, E, F = percentage of fatty acids containing 1, 2, 3, 4, 5, 6 unsaturated bonds related to total fatty acids, respectively.

Statistical analysis. Data were expressed as the means ± standard deviation (SD). The means were compared by Student's *t*-test of SPSS software (Version 16.0, 2008). The level of $P < 0.05$ was set as the criterion of statistical significance.

RESULTS

As shown in Table 2, the ADG, ADFI, and FCR did not differ between the two groups ($P > 0.05$). No difference was observed in various antioxidant enzymes activities of the breast and thigh muscles between the two groups ($P > 0.05$) (Table 3).

Table 3. Effects of dietary CLA supplementation on antioxidant enzymes activities in the breast and thigh muscle of broiler chickens at the end of the sixth week

Treatment	TSOD (NU/mg)	CAT (U/mg)	GSH-Px (U/mg)
Breast muscle			
Control diet	3.67 ± 0.43	2.84 ± 0.36	25.74 ± 3.34
CLA diet	3.85 ± 0.47	2.90 ± 0.29	26.78 ± 4.34
Significance	ns	ns	ns
Leg muscle			
Control diet	2.87 ± 0.26	2.44 ± 0.19	37.68 ± 6.43
CLA diet	2.81 ± 0.35	2.40 ± 0.16	35.61 ± 6.26
Significance	ns	ns	ns

CLA = conjugated linoleic acid, TSOD = total superoxide dismutase, CAT = catalase, GSH-Px = glutathione peroxidase, ns = non-significant

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Table 4. Effect of dietary CLA supplementation on the MDA content, ROS production, GSH content, and γ-GCS activity in the breast and thigh muscle of 42-day-old broiler chickens

Treatment	MDA (nmol/g)	ROS (R)	GSH (mg/g)	γ-GCS (U/mg)
Breast muscle				
Control diet	2.29 ± 0.12	4.98 ± 0.46	20.24 ± 2.56	0.28 ± 0.05
CLA diet	1.58 ± 0.11	3.73 ± 0.35	24.67 ± 2.92	0.36 ± 0.06
Significance	**	**	*	*
Leg muscle				
Control diet	2.70 ± 0.21	4.78 ± 0.54	22.63 ± 3.09	0.31 ± 0.06
CLA diet	2.11 ± 0.17	3.65 ± 0.47	27.51 ± 3.48	0.39 ± 0.07
Significance	**	**	*	*

CLA = conjugated linoleic acid, MDA = malondialdehyde, ROS = reactive oxygen species, GSH = glutathione, γ-GCS = γ-glutamylcysteine synthetase, R = relative value

for the congener muscle, impact statistically significant at the level of $^{*}P < 0.05$, $^{**}P < 0.01$

As shown in Table 4, the broilers fed the CLA diet had lower MDA content in the breast and thigh muscles than the broilers fed control diet ($P < 0.01$). On the basis of the same amount of sample, the peak altitude represented the production of ROS. The peak altitude was lower in birds receiving the CLA diet ($P < 0.01$), indicating that dietary CLA supplementation decreased ROS pro-

Table 5. Effect of dietary CLA supplementation on IMF and the fatty acid profile (% of total FAME) of breast and thigh muscle of 42-day-old broiler chickens (%)

	Thigh muscle			Breast muscle		
	control diet	CLA diet	significance	control diet	CLA diet	significance
IMF	8.14 ± 1.65	6.73 ± 1.36	*	3.08 ± 0.59	2.47 ±0.52	*
Fatty acids (%)						
C14:0	0.31 ± 0.05	0.58 ± 0.06	**	1.79 ± 0.09	2.53 ± 0.12	**
C16:0	20.02 ± 4.02	24.81 ± 4.23	*	19.77 ± 3.95	22.65 ± 4.36	ns
C16:1 n-7	3.00 ± 0.61	2.08 ± 0.57	*	3.53 ± 0.73	3.10 ± 0.68	ns
C18:0	7.68 ± 1.24	10.09 ± 1.43	**	6.99 ± 1.15	9.97 ± 1.76	**
C18:1 n-9	32.98 ± 4.82	24.78 ± 4.37	**	30.45 ± 4.85	21.61 ± 3.26	**
C18:2 n-6	26.72 ± 3.26	26.38 ± 3.01	ns	26.18 ± 3.29	26.67 ± 3.54	ns
C18:3 n-3	1.04 ± 0.24	1.61 ± 0.27	*	1.93 ± 0.21	1.76 ± 0.23	ns
C20:4 n-6	4.68 ± 0.65	3.92 ± 0.61	*	4.43 ± 0.63	3.87 ± 0.51	*
C22:1 n-17	1.61 ± 0.26	1.75 ± 0.32	ns	2.00 ± 0.31	1.47 ± 0.30	*
C22:6 n-3	2.74 ± 0.16	2.30 ± 0.14	**	2.94 ± 0.18	2.34 ± 0.17	**
*c*9, *t*11-CLA	0.13 ± 0.02	2.56 ± 0.38	**	0.00 ± 0.00	2.46 ± 0.34	**
*t*10, *c*12-CLA	0.09 ± 0.03	1.13 ± 0.46	**	0.00 ± 0.00	1.56 ± 0.41	**
Total CLA	0.22 ± 0.04	3.69 ± 0.67	**	0.00 ± 0.00	4.02 ± 0.74	**
SFA	28.01 ± 4.16	33.48 ± 4.85	*	28.55 ± 4.09	35.15 ± 5.63	*
MUFA	36.59 ± 4.29	28.61 ± 4.15	**	35.98 ± 5.60	26.18 ± 4.92	**
PUFA	35.18 ± 5.62	34.11 ± 5.24	ns	35.48 ± 5.79	34.64 ± 5.17	ns
PI	70.38 ± 6.52	64.40 ± 6.08	*	72.18± 7.16	65.04 ± 6.62	*

CLA = conjugated linoleic acid, IMF = intramuscular fat, FAME = fatty acid methyl esters, ns = non-significant (for the congener muscle), SFA = saturated fatty acids, MUFA = monounsaturated fatty acids, PUFA = polyunsaturated fatty acids, PI = peroxidability index

for the congener muscle, impact statistically significant at the level of $^{*}P < 0.05$, $^{**}P < 0.01$

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duction in the breast and thigh muscles of broiler chickens. After dietary CLA supplementation, the GSH content in the breast and thigh muscles of broiler chickens increased by 21.89 and 21.56%, respectively ($P < 0.05$), while γ-GCS activity in the breast and thigh muscles increased by 28.57 and 25.80%, respectively ($P < 0.05$).

As shown in Table 5, dietary CLA supplementation remarkably decreased the intramuscular fat of the breast and thigh meat ($P < 0.05$). The broilers fed the CLA diet had higher total CLA contents of the breast and thigh muscles than the broilers fed the control diet ($P < 0.01$). Dietary CLA supplementation increased the saturated fatty acids (SFA) content ($P < 0.05$) and decreased the monounsaturated fatty acids (MUFA) content of the breast and thigh muscles ($P < 0.01$). The broilers on the CLA diet had lower PI value of the breast and thigh muscles than the broilers fed the control diet ($P < 0.05$), indicating that dietary CLA improved lipid stability of the breast and thigh muscles lipids.

DISCUSSION

The present experiment found that dietary CLA was effectively incorporated into the breast and thigh muscles of broiler chickens, which indicated that broiler meat is an ideal candidate for CLA enrichment. This result is basically in line with Du and Ahn (2002) who reported that CLA accumulated in the breast and leg muscles lipid and accounted for approximately 10 g/100 g fatty acids present in birds fed 15 g CLA/kg diet. Moreover, Suksombat et al. (2007) reported that feeding incremental levels of dietary CLA (0.0–1.5%) resulted in linear increases of CLA isomers concentrations in muscle tissue lipids of Arbor Acres broilers. It has already been demonstrated that CLA is readily incorporated into the egg of laying hen (Chamruspollert and Sell 1999).

The decreased MDA content in the breast and thigh muscles suggested that dietary CLA may improve the fresh chicken meat quality. The result is consistent with previous studies on the poultry animal model (Du et al. 2001; Bolukbasi 2006; Kawahara et al. 2009; Narciso-Gaytan et al. 2011). Moreover, in a study by Wiegand et al. (2002), growing-finishing barrows were fed a diet enriched with 0.75% CLA and the lipid oxidation level decrease of chops at days 1, 14, and 28 of retail storage was observed. Similarly, Corino et al. (2002) reported that the oxidative stability of muscle was increased after dietary CLA supplementation. Also, lower TBARS values have been reported in pig muscle samples from pigs receiving higher dietary inclusions of CLA (Corino et al. 2003). Chae et al. (2004) reported that adding CLA oil to raw and cooked ground beef patties reduced the development of lipid oxidation and extended the shelf-life of the meat. These findings, tighter with the results of this experiment, confirmed that dietary CLA supplementation was an effective measure to reduce lipid oxidation of meat of domestic animals. However, Kumari et al. (2014) reported that the CLA in meat increased substantially with the CLA feeding dose and concurrently resulted in the increase of TBARS values in chicken meat. In test tube studies, CLA was found not to act as an antioxidant but to accelerate lipid peroxidation instead (Igarashi and Miyazawa 2001).

Glutathione is the main cellular non-enzymatic antioxidant and free radical scavenger *in vivo* and critical for maintaining the redox status of the cell. The present experiment found that the GSH content of the breast and thigh muscles of broiler chickens receiving the CLA diet significantly increased while the ESR spectra clearly indicated that ROS production was lower in the muscles homogenates of these birds. Considered together, the results indicated that the increase of the GSH content quenched more ROS in the broiler chickens receiving the CLA diet compared to the broiler chickens receiving the control diet. Moreover, the lower ROS production in the broiler chickens receiving the CLA diet is in agreement with the lower lipid peroxidation level in these birds.

γ-Glutamylcysteine synthetase is the rate-limiting enzyme of cellular GSH biosynthesis. In the present experiment, the γ-GCS activity of the breast and thigh muscles was enhanced by dietary CLA supplementation and, as a result, induced more GSH synthesis. The precise mechanism, by which the dietary CLA influences the γ-GCS activity, is yet to be determined. The rate limiting enzyme in the synthesis of GSH is γ-GCS mainly regulated by peroxisome proliferator activated receptors (PPARs). As the ligands of PPARs, CLA, directly or indirectly, influenced the expression and activity of PPARs. This provided a possible mechanism for CLA to influence the γ-GCS activity and GSH synthesis.

No significant effects of dietary CLA on antioxidant enzyme activities were observed in the breast

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and thigh muscles in this experiment. The result was in line with Ko et al. (2004) who reported that 0.75% or 1% dietary CLA did not affect SOD, GSH-Px, and CAT activities in the intestinal mucosa and liver of Arbor Acres broilers. However, Zhang et al. (2008) found that 1% dietary CLA markedly elevated the liver CAT activity and increased total SOD activities in the liver, serum, and muscle of Arbor Acres broiler chickens. Dietary CLA caused a significant decrease in hepatic SOD activity while having no influence on hepatic CAT activity in male Sprague-Dawley rats (Kim et al. 2005). Vegetable oil rich in CLA effectively alleviated the oxidative stress and recovered antioxidant enzymes activities in the brains of the male albino mice fed sodium arsenite (Saha and Gosh 2011). It is not clear whether these differences between the present results and previous results were due to such factors as the CLA level, diet composition, feeding time, and animal species. To clarify this, further studies are necessary.

Lipid oxidation is the result of the unsaturated fatty acids being attacked by free radicals, so there is a direct relationship between the lipid oxidation degree and the fatty acids composition in tissues (Min and Ahn 2005). In the present experiment, effects of CLA on the fatty acid composition of the breast and thigh muscles were evident in increasing the SFA content and decreasing the MUFA content, with the latter being evident in the increase of C16:0 and C18:0 fatty acid content and the decrease of C16:1 and C18:1 content. This is in agreement with the studies of Aletor et al. (2003) and Sirri et al. (2003), and can potentially be ascribed to the fact that dietary CLA inhibits the activity of stearoyl-coenzyme A desaturase, which promotes the transformation of the saturated fatty acids to unsaturated fatty acids. The above-mentioned changes of the fatty acids composition of the breast and thigh muscles increased the lipid oxidation stability in agreement with the decrease of the oxidation trend index. The increase of lipid oxidation stability reduced the formation of lipid free radical after attacking the fatty acids and inhibited a subsequent oxidative reaction.

CONCLUSION

The results of this study indicated that dietary CLA reduced the lipid peroxidation of breast and thigh muscles of broiler chickens. Dietary CLA may play the role through: (*i*) increasing the CLA and the SFA contents, and decreasing the MUFA content so as to improve lipid stability and/or (*ii*) inducing GSH synthesis to quench more free radicals and relieve oxidative damage. However, the mechanism, by which CLA influences the γ-GCS activity and GSH synthesis, needs to be further studied.

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