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Response Time of Broiler Chickens to Cimaterol: Meat Tenderness, Muscle Composition, Fiber Size, and Carcass Characteristic

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Response Time of Broiler Chickens to Cimaterol: Meat Tenderness, Muscle Composition, Fiber Size, and Carcass Characteristics[1,2]

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ABSTRACT: The response time to cimaterol (CIM), a β-adrenergic agonist, by broiler chickens for carcass characteristics, muscle composition, muscle fiber size, catheptic enzyme activity, and tenderness was determined. Two trials were conducted in which chickens were fed a control diet (CON) containing 0 ppm of CIM or a diet containing 1 ppm of CIM. Trial 1 consisted of 55, 31-d-old broiler chickens individually fed for up to 48 h. At 0, 6, 12, 18, 24, and 48 h, five CON and five CIM-fed chickens were killed. Trial 2 consisted of 160, 33-d-old broiler chickens group-fed for up to 14 d. At 2, 4, 6, 8, 10, 12, and 14 d, 10 CON and 10 CIM-fed chickens were killed. The breast muscle (BM) and leg muscle (LM) weight, cathepsin B and L activities, DNA, RNA, and protein concentration, and BM shear force value (SFV) were measured in both trials. Thigh muscle (TM) SFV were measured in Trial 2 only. Fiber size of BM was measured (five birds per treatment) at d 2, 6, 10, and 14. In Trial 1, BM weight and SFV were lower in CIM-fed birds at 6 h ($P < .05$). In Trial 2 BM SFV were higher at d 8 ($P = .06$) and d 10 ($P < .05$) in CIM-fed chickens. The SFV of CIM-fed chickens were higher at d 4, 8, 10, 12, and 14 ($P < .05$). The BM of CIM-fed chickens had a higher protein:DNA ratio ($P < .05$) at d 6 through 14, whereas LM of CIM-fed chickens had a higher protein:DNA ratio at d 8, 10, and 14. Fiber size of the BM in CIM-fed chickens tended to be larger at d 10 ($P = .13$) and at d 14 ($P = .17$). Total BM weight and BM as a percentage of final body weight (FBW) was higher at d 10 and 14 ($P < .01$) in CIM-fed chickens. Total LM weight and LM as a percentage of FBW was higher at d 14 ($P < .01$) in CIM-fed chickens. In the BM of CIM-fed chickens, protein:DNA ratio increased by d 6, SFV by d 8, muscle fiber size by d 10, and BM weight and BM as a percentage of FBW by d 10. The TM or LM of CIM-fed chickens showed increases in SFV by d 4, protein:DNA ratio by d 8, and LM weight and LM as a percentage of FBW by d 14. Response times to treatment with CIM differed for the various traits measured (i.e., meat tenderness, muscle composition, fiber size, or carcass characteristics).

Key Words: Cimaterol, Cathepsins, DNA, RNA, Protein, Meat Characteristics

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Introduction

The use of β-adrenergic agonists provides an excellent model for studying changes in muscle growth. Recent studies have dealt with the effect of cimaterol (**CIM**), a β-adrenergic agonist, on various carcass traits of meat animals. It is established that CIM decreases fat accumulation and increases protein accretion in the muscle of many animal species, including sheep (Baker et al., 1984), poultry (Dalrymple et al., 1984), and swine (Jones et al., 1985). It also decreases tenderness in the meat of various species (Jones et al., 1985; Allen et al., 1986; Hamby et al., 1986; Morgan et al., 1989).

The mechanisms that are involved in making meat of CIM-treated animals less tender are unknown. Some researchers have investigated catheptic enzyme activities in CIM-treated animals with the hypothesis that enzyme activities are lowered by CIM and this reduces tenderness (Forsberg et al., 1987; Kretchmar et al., 1988). This would support the idea that CIM acts to increase muscle accretion via a reduction in muscle degra-

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dation (Young et al., 1990). Morgan et al., (1989) found that cathepsin B and L activities were lower in CIM-treated chickens and were negatively correlated with shear force value (**SFV**).

Muscle hypertrophy in CIM-treated animals may be involved with the observed tenderness reduction. Maltin et al. (1986) observed an increase in the area of type II fiber of rats treated with clenbuterol. Kim et al. (1987) and Beermann et al. (1987) found that CIM-treated sheep had increased cross-sectional areas in type II fibers. Both studies demonstrated that DNA concentration was lower in CIM-treated lambs. This suggests that muscle fibers may become more densely packed due to muscle fiber hypertrophy, which may cause a toughening effect.

In most studies CIM has been fed for a period of 6 to 12 wk before muscle sampling. The objective of this study was to investigate the response time of carcass variables to CIM by broiler chickens during hours (0 to 48 h) or days (0 to 14 d) after initiation of treatment.

Experimental Procedure

In Trial 1, 55 broiler chickens (31 d old) were randomly assigned to individual pens and given ad libitum access to either a control diet (**CON**) containing 0 ppm of CIM or a diet containing 1 ppm of CIM. In Trial 2, 160 broiler chickens (33 d old) were divided equally into two electrically heated battery brooders (Petersime Incubator, Gettysburg, OH). Within each battery, chickens received the CON diet (n = 80) or the CIM diet (n = 80). Eight chickens were housed in each pen with 10 pens per battery. All chickens had free access to tap water and had ad libitum access to a 20% CP growing diet (90% DM, 3,131 kcal of ME/kg) consisting of 64.3% grain sorghum, 25.3% soybean meal (47.5% CP), 5.0% animal fat, and a vitamin and mineral supplement. All chickens were withheld from feed for 3 h before starting the trial to ensure immediate ingestion of feed.

In Trial 1 five chickens were killed at 0 h (before feeding) to obtain baseline values for carcass variables, and additional chickens (five CON and five CIM) were killed at 6, 12, 18, 24, and 48 h. In Trial 2 10 CON and 10 CIM-fed chickens were killed at time 0 for use as baseline values, and additional chickens (10 CON and 10 CIM) were killed at d 2, 4, 6, 8, 10, 12, and 14.

Breast muscle weight (**BM**) and leg muscle weight (**LM**) were recorded at each kill period. The BM consisted of the pectoralis major; LM consisted of the gastrocnemius and peroneus longus. In Trial 2 the thigh muscle (**TM**; consisting of the biceps femoris, semitendinosus, semimembranosus, and tensor facia latae) were removed for SFV analysis.

Cathepsin B and L activities were measured in BM and LM samples. Total cathepsin B and L activities were determined in samples prepared using the procedures of Moeller et al. (1976) with slight modifications. One BM and both LM were removed immediately postmortem, frozen in liquid nitrogen, and stored at −70°C until further analysis for each trial. Ten grams of muscle (BM or LM) was homogenized in 50 mL of ice-cold .25 *M* sucrose containing .02 *M* KCl. The homogenate was filtered through two layers of cheesecloth and adjusted to a pH of 7.3 with 1 *N* KOH. The filtrate was centrifuged at 6,000 × *g* for 6 min, and the supernatant was decanted and saved. The pellet was resuspended in 25 mL of the sucrose-KCl solution and recentrifuged. Supernatants were combined and measured for protein content using the biuret method (Gornall et al., 1949). Samples were diluted to attain similar protein levels (.3 mg/mL) for the cathepsin assay. Total and specific cathepsin B and L activities were measured by the techniques of Barrett (1980) with modifications. The substrate N-carbobenzoxy(CBZ)-L-phenylalanine-L-arginine-4-methyl-7-amino hydrochloride was used at an assay concentration of 1 m*M*. The buffer system was a .704 *M* potassium phosphate, .096 *M* sodium phosphate at pH 5.8. This substrate is hydrolyzed by both cathepsins B and L (Barrett and Kirschke, 1981).

Muscle protein, DNA, and RNA contents were determined in the BM and LM of chickens in Trial 2. Protein content of whole muscles was determined using the biuret method of Gornall et al. (1949). DNA was analyzed using the method of Labraca and Paigen (1980). RNA was separated using the method of Shiboko et al. (1967) and quantified using the procedure of Lin and Schjeide (1969).

Muscle fiber size of the BM was determined in five chickens per treatment at d 2, 6, 10, and 14. Frozen muscle samples were cryosectioned and fixed on a glass slide. Slices of muscle were approximately 8 μm in thickness. Sample preparation and staining was accomplished using the procedures of Guth and Samaha (1970). Area of the muscle was determined using the Bio Quant software program (R & M Biometrics, Nashville, TN).

Shear force values were determined on cooked BM (both trials) and on TM in Trial 2 that had remained on the carcass at room temperature for 2 h postmortem. The BM and TM were excised and placed in a 4°C cooler for 24 h, to allow any postmortem enzymatic tenderization to occur, frozen, and stored at −70°C. Samples were removed from the freezer, allowed to temper at 4°C

Table 1. The effect of cimaterol on breast and leg muscle weights, feed consumption, cathepsin B and L activity, and shear force values during 48 hours (Trial 1)

Time on feed, h	Breast wt, g[b]		Leg wt, g[b]		Feed consumed, g		Cathepsin B and L activity[a]				Shear value of breast, kg[c]	
							Breast		Leg			
	CON[d]	CIM[e]	CON	CIM	CON	CIM	CON	CIM	CON	CIM	CON	CIM
0	25.85	N/A[f]	20.04	N/A	N/A	N/A	760.4	N/A	279	N/A	1.30	N/A
6	28.14*	23.59	18.70	19.27	47.02	40.76	970.85	897.3	375.88	283.02	2.28*	1.05
12	27.05	25.41	20.33	18.91	62.34	57.42	827.97	793.12	275.08	272.41	.82	.76
18	25.98	26.41	19.01	19.63	86.64	76.52	746.72	849.12	304.39	171.53	1.18	.61
24	24.61	24.98	20.05	18.15	107.28	95.36	777.26	702.04	197.65	217.74	.95	1.14
48	28.81	31.53	22.55	21.74	181.14	174.26	711.74	918.31	308.17	388.29	1.87	1.18
SEM[g]	.51	.79	.38	.50	9.87	.79	43.79	51.96	28.64	26.23	.15	.09

[a]Total activity, nanomoles·milligram^{-1}·minute^{-1} of product released.
[b]Breast muscle consisted of the pectoralis major and leg muscle consisted of the gastrocnemius and peroneous longus.
[c]Shear values are presented as kilograms of peak force to shear a .5-cm × 1-cm × 3-cm sample of breast muscle.
[d]CON = birds fed 0 ppm of cimaterol in diet (n = 5).
[e]CIM = birds fed 1 ppm of cimaterol in diet (n = 5).
[f]Data not available.
[g]Pooled SEM for each feeding treatment.
*Significant difference ($P < .05$) within time on feed for specific variable.

for approximately 14 h, double-wrapped in aluminum foil, and heated to an internal temperature of 82°C in a 177°C electric oven. Temperature was monitored using small-diameter, copper-constantan thermocouples attached to a digital thermometer (Omega Engineering, Stamford, CT). Samples were allowed to cool at room temperature for 2 h before testing. In Trial 1, a 3-cm × 3-cm × .5-cm sample was removed from the thick, anterior portion of the BM. Three .5-cm × 1-cm × 3-cm slices were obtained from this sample with fiber direction parallel to the 3-cm length. Shear force values were determined using an Instron Universal Testing Machine (Instron, Canton, MA) with a Warner-Bratzler shear attachment. In Trial 2, a 3-cm × 3-cm × 1-cm sample was removed from the BM and TM. Three 1-cm × 1-cm × 3-cm slices were obtained from this with fiber direction parallel to the 3-cm length. Peak SFV are presented as the peak load (in kilograms) required to shear the muscle sample. Shear values were obtained using the Instron machine and a 500-kg load cell with a full scale load of 1, a preset crosshead speed of 250 mm/min, and a proportional chart speed ratio of 2:1 (millimeters/minute).

Statistical analyses of data collected in Trial 1 involved in an analysis of variance for a completely randomized design with individual chickens as the sampling unit (Steel and Torrie, 1980). Trial 2 consisted of a 2 × 8 factorial arrangement of treatments with battery as replication. Treatment means, standard deviations, and analysis of variance were calculated using SAS (1985). A series of orthogonal contrasts was used to determine treatment differences within kill periods for each variable.

Results and Discussion

No differences ($P > .05$) were observed in Trial 1 for any of the variables measured (Table 1), with the exception that SFV and BM weight at 6 h were lower in CIM-fed chickens than in CON chickens ($P < .05$). Chickens were withheld from feed for 3 h before starting the feeding trials, which possibly put them in a catabolic state. Feed consumption of CIM-treated chickens is lower than that of controls for up to 2 d (Gwartney et al., 1991).

In Trial 2, BM SFV were higher in CIM-fed chickens on d 8 ($P = .06$) and on d 10 ($P < .05$) (Table 2). Thigh muscle SFV were higher ($P < .05$) in CIM-fed chickens at d 4, 8, 10, 12, and 14 (Table 2). Morgan et al. (1989) and Gwartney et al. (1991) observed higher SFV in BM of CIM-fed chickens after 21 d of feeding CIM. Decreases in meat tenderness have been observed in pork (Jones et al., 1985), lambs (Hamby et al., 1986), and cattle (Allen et al., 1986) after feeding a β-adrenergic agonist. The unique finding in this study was that tenderness differences were observed after as little as 4 d of treatment. Early changes in tenderness may occur in other species, but researchers have not identified them. Perhaps tenderness differences occur in broiler chickens earlier because chickens respond faster than other animals due to their rapid growth rate.

Thigh muscle was used for SFV in this study to relate CIM-induced tenderness differences in muscle to different populations of type I and type II fibers. Differences in SFV were observed earlier in LM than in BM, which may be due to response time of muscle fiber types to CIM. Watson-Wright and Wilkinson (1986) reported that skeletal muscle

Table 2. The effects of cimaterol on breast and thigh muscle shear values (SFV) and breast and leg cathepsin B and L activities during 14 days (Trial 2)

Time on feed, d	Breast[a] SFV		Thigh[a] SFV		Breast cathepsin B and L activity		Leg cathepsin B and L activity	
	CON[b]	CIM[c]	CON	CIM	CON	CIM	CON	CIM
0	1.41[d]	1.83	1.49	1.33	1,237[e]	1,099	296	325
2	2.10	2.37	1.37	1.17	1,621	1,885	330	364
4	3.28	3.54	1.61*	2.10	1,724	1,472	352	390
6	3.03	3.62	1.59	1.77	1,476	1,207	315	371
8	2.19*	3.61	1.88*	2.11	1,172	1,026	418	438
10	2.64*	5.14	1.82*	2.00	1,185	925	404	453
12	1.70	2.71	1.57*	1.98	1,388	1,346	394	439
14	2.29	3.43	1.70*	1.99	1,347	1,251	329	387
SEM[f]	.13	.16	.04	.08	60	58	13	14

[a]Breast consisted of the pectoralis major and leg consisted of the combined gastrocnemius and peroneous longus.
[b]CON = birds fed 0 ppm of cimaterol in diet.
[c]CIM = birds fed 1 ppm of cimaterol in diet.
[d]Shear values are presented as kilograms of peak force to shear a 1-cm × 1-cm × 3-cm sample of breast muscle.
[e]Total activity, nanomoles · milligram^{-1} · minute^{-1} of product released.
[f]Pooled SEM for each feeding treatment.
*Significant difference ($P < .05$) within time on feed for specific variable.

possesses receptors of the β-2 subclass, but that receptor density is a function of muscle type. Type II fibers seem to have a low β-receptor density, whereas type I fibers contain a high β receptor density; therefore the slower response in BM to CIM treatment may be due to the lower proportion of β-receptors present.

Differences among SFV between muscles were not analyzed; however, TM tended to be lower than BM (Table 2). Muscles in the leg of chickens have more connective tissue because of the perimysium that surrounds the individual muscles. However, increased fat levels associated with LM compared to BM (Morgan et al. 1989; Gwartney et al., 1991) may reduce SFV.

No differences between treatments ($P > .05$) were found in total cathepsin B and L activities for either BM or LM in Trial 2 (Table 2). However, mean activity for BM was lower at all kill periods in CIM-fed chickens except for d 2, which may have been higher because (as mentioned previously for Trial 1) chickens may have been in a catabolic state due to reduced feed consumption. Cimaterol-fed chickens began to show the effects

Table 3. The effects of cimaterol on breast and leg muscle weights and as a percentage of final body weight during 14 days (Trial 2).

Time on feed, d	Breast[a] wt, g		Leg[a] wt, g		Breast, %		Leg, %	
	CON[b]	CIM[c]	CON	CIM	CON	CIM	CON	CIM
0	64.1	71.4	53.6	59.9	6.57	6.85	5.53	5.79
2	78.6	78.0	61.7	63.6	6.80	6.61	5.32	5.37
4	88.6	95.4	67.0	67.6	7.12	7.42	5.41	5.28
6	101.0	101.9	81.2	89.9	7.09	7.35	5.67	5.90
8	106.4	109.7	82.1	89.9	7.31	7.30	5.67	5.90
10	119.6**	135.0	98.3	103.5	6.99**	8.12	5.75	6.16
12	146.8	149.4	112.5	117.0	7.95	8.10	6.09	6.35
14	139.8**	163.9	108.9**	132.4	7.36**	8.07	5.75**	6.53
SEM[d]	3.68	4.16	2.79	3.22	.068	.088	9.04	8.06

[a]Breast consisted of the pectoralis major and leg consisted of the combined gastrocnemius and peroneous longus.
[b]CON = birds fed 0 ppm of cimaterol in diet.
[c]CIM = birds fed 1 ppm of cimaterol in diet.
[d]Pooled SEM for each feeding treatment.
*Significant difference ($P < .05$) within time on feed for specific variable.
**Significant difference ($P < .01$) within time on feed for specific variable.

Table 4. The effect of cimaterol on breast[a] muscle protein, RNA, and DNA (Trial 2)

Time on feed, d	Protein, mg/g		RNA, mg/g		DNA, mg/g		Protein:RNA		Protein:DNA		RNA:DNA	
	CON[b]	CIM[c]	CON	CIM	CON	CIM	CON	CIM	CON	CIM	CON	CIM
0	239.5	240.6	.631	.638	.367*	.413	389	387	659*	583	1.73	1.54
2	231.4	240.9	.683	.709	.392	.385	361	345	596	641	1.79	1.86
4	248.6	239.5	.657	.712	.343	.334	401	346	725	720	1.91	2.14
6	228.0	239.9	.780	.804	.354*	.299	318	307	648*	804	2.20	2.64
8	257.1	248.7	.603*	.854	.346*	.297	437*	300	755*	841	1.79*	2.89
10	245.2	246.3	.914	.911	.361*	.285	280	277	682*	869	2.53*	3.19
12	240.1	249.6	.694	.626	.344*	.281	359	413	709*	898	2.06	2.27
14	236.0	234.4	.790	.693	.314*	.269	320	349	759*	878	2.54	2.62
SEM[d]	1.81	1.87	.02	.02	.004	.007	11.1	8.71	10.98	16.81	.07	.08

[a]Breast muscle consisted of the pectoralis major.
[b]CON = birds fed 0 ppm of cimaterol in diet.
[c]CIM = birds fed 1 ppm of cimaterol in diet.
[d]Pooled SEM for each feeding treatment.
*Significant difference ($P < .05$) within time on feed for specific variable.

of the β-adrenergic agonist after they recovered from the catabolic period, which may have delayed other CIM-related changes.

Total BM weight and BM as a percentage of final BW were elevated at d 10 and 14 ($P < .01$) in CIM-fed chickens (Table 3). This finding is in agreement with the results of Morgan et al. (1989), who found higher BM percentage after only 17 d of feeding CIM. Leg muscle weight and LM percentages were higher at d 14 ($P < .01$) in CIM-fed chickens. The increase in percentage of LM and BM indicates that there was an improvement in leanness of CIM-fed chickens.

In Trial 2 the protein:RNA ratio was lower ($P < .05$) in the BM only at d 8 and in the LM at d 2 to 8 of CIM-fed chickens (Tables 4 and 5), indicating that RNA increased in relation to protein. The RNA:DNA ratio was higher ($P < .05$) in the BM at d 8 and 10, and at d 4 and 8 in the LM of CIM-fed chickens. The increase in RNA:DNA ratio indicates an increase in protein synthetic capacity per unit DNA in CIM-fed chickens. Neither protein:RNA nor RNA:DNA ratio differences were maintained throughout the study. Beermann et al. (1987) reported that RNA:DNA ratio was higher in CIM-fed lambs at 7 wk of feeding but was similar at 12 wk. These data reveal that CIM may alter the protein synthetic capacity of the muscle early in treatment, but the differences are attenuated with continued treatment. McElligott et al. (1989) observed that the growth response to clenbuterol in rats is attenuated after 2 wk of continuous treatment.

The protein:DNA ratio was higher ($P < .05$) and DNA per unit amount was lower ($P < .05$) in the BM of CIM-fed chickens from d 6 to d 14 and in LM on d 8, 10, and 14 (Tables 4 and 5). Kim et al. (1987) and Beermann et al. (1987) identified similar trends. This indicates that β-agonists increase muscle size by hypertrophy, which may occur by

Table 5. The effect of cimaterol on leg[a] muscle protein, RNA, and DNA (Trial 2)

Time on feed, d	Protein, mg/g		RNA, mg/g		DNA, mg/g		Protein:RNA		Protein:DNA		RNA:DNA	
	CON[b]	CIM[c]	CON	CIM	CON	CIM	CON	CIM	CON	CIM	CON	CIM
0	242.4	243.0	.624	.709	.558	.579	411	361	438	424	1.13	1.24
2	249.9	255.4	.688*	.558	.557	.526	392*	482	455	494	1.25	1.09
4	233.6	247.5	.547*	.766	.480	.501	434*	339	489	502	1.14*	1.54
6	242.0	234.6	.645	.727	.479	.481	414*	336	508	496	1.34	1.54
8	254.2	259.3	.608	.758	.545*	.422	420*	342	472*	633	1.12*	1.86
10	247.3	269.1	.581	.588	.498*	.399	435	468	500*	685	1.16	1.48
12	239.2	227.4	.690	.751	.515	.462	366	318	471	494	1.37	1.65
14	241.7	240.6	.606	.532	.550*	.449	417	462	463*	554	1.13	1.22
SEM[d]	2.77	3.05	.02	.02	.01	.01	11.96	12.11	10.2	13.54	.03	.04

[a]Leg muscles consisted of the gastrocnemius and peroneous longus.
[b]CON = birds fed 0 ppm of cimaterol in the diet.
[c]CIM = birds fed 1 ppm of cimaterol in the diet.
[d]Pooled SEM for each feeding treatment.
*Significant difference ($P < .05$) within time on feed for specific variable.

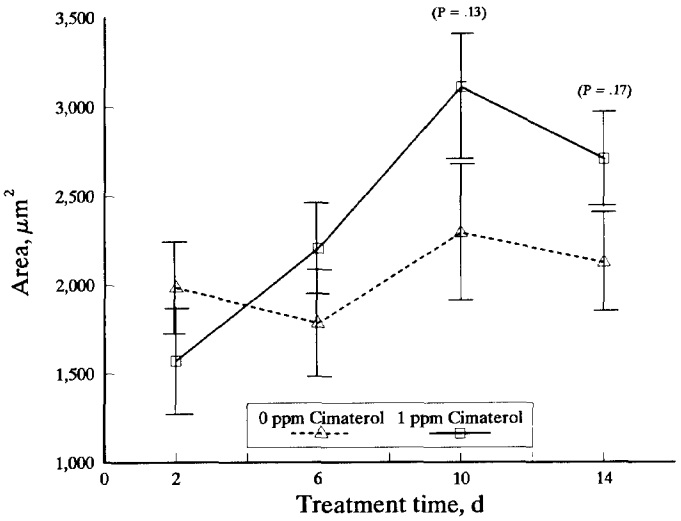

Figure 1. Fiber area of the pectoralis major muscle of cimaterol-fed and control broiler chickens (n = 5).

decreasing the rate of the muscle protein degradation. Changes in degradative activity in the muscle may cause decreased tenderness postmortem, as was observed in this study. However, differences in the cathepsin B and L activities were not demonstrated, which may indicate that other degradative systems, such as the calpain enzymes, may be responsible.

Muscle fiber size (Figure 1) in the BM tended to be larger in CIM-fed chickens than in CON chickens at d 10 (P = .13) and d 14 (P = .17). These differences were not significant, possibly because of the limited number (n = 5) of samples. These data were similar to those reported in rats (Maltin et al., 1986) and lambs (Beermann et al., 1987; Kim et al., 1987). Differences in both of the studies with lambs were primarily in type II fibers. The BM of chickens is primarily type II fibers, so it would be expected to be affected by CIM. The changes in fiber size tend to support the protein:DNA ratio data that muscle hypertrophy causes a change in muscle mass. Because tenderness differences were observed at an earlier date, it can be concluded that tenderness differences are not the manifestation of large fibers.

Implications

Chickens fed cimaterol begin to show tenderness differences as early as 4 d in thigh muscle and 8 d in breast muscle. Muscle fiber type may influence the ability of cimaterol to alter their compositional makeup. The protein:DNA ratio increases, at which time muscle weight begins to increase, suggesting that cimaterol has the ability to allow more protein per given DNA unit. This may be the result of decreased degradation rates of skeletal protein when cimaterol is fed.

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