Relationship between pectoralis major muscle histology and quality traits of chicken meat

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ABSTRACT A trial was conducted to evaluate the influence of myodegeneration of pectoralis major muscle on quality traits and chemical composition of breast meat of heavy-size male broilers. For this purpose, a total of 72 pectoralis major muscles were randomly collected from broilers farmed under homogeneous conditions and graded into three categories (mild, n = 22; moderate, n = 33; and severe, n = 17) based on the presence of abnormal fibers (giant fibers, fibers with hyaline degeneration, and damaged and/or necrotic fibers) evaluated by histological and immunohistochemical analysis. Color, pH, drip loss, Allo–Kramer shear values, and chemical composition (moisture, proteins, total lipids, ashes, and collagen) were determined on nonmarinated breast meat. Purge loss and cook loss, total yield, and Allo–Kramer shear values were measured on vacuum-tumbled samples. Samples showing moderate myodegeneration had the highest mean cross-sectional area of the fibers, while samples with severe myodegeneration had myofibers of different diameter and without the characteristic polygonal shape, multifocal degeneration and necrosis, as well as infiltration of CD3-immunoreactive cells. Cooking losses of nonmarinated meat were lower in the mild group with respect to moderate and severe groups (21.4 vs. 24.7 and 24.7%; $P < 0.001$). Breast muscles with severe damage, in comparison with mild degenerated samples, showed higher moisture (75.4 vs. 74.4%; $P < 0.05$) and lower protein percentages (21.1 vs. 22.6%; $P < 0.001$). The lipid percentage of severely degenerated samples was higher than that from moderate group (2.94 vs. 2.36; $P < 0.05$), while collagen content was not modified by histological lesion levels. Marinated meat from the mild group had higher uptake and total marinade yield after cooking. In conclusion, almost all breast fillets of heavy broiler chickens produced under intensive farming systems had histological lesions, which reflected on the chemical composition of the meat and the impaired water holding/binding capacities of the meat.

Key words: histological evaluation, muscle damage, breast meat quality, marinated meat, chicken

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INTRODUCTION

In recent years, some studies indicate an increasing occurrence of new quality abnormalities in the breast muscles of broiler chickens. A white striping defect was first pointed out by Bauermeister et al. (2009) and has been described as the appearance of white striation parallel to muscle fiber on the surface of pectoralis major muscles. A recent survey estimated that the total incidence of white striped breast fillets in medium-size birds was around 12% (Petracci et al., 2013a), but observations on heavy birds indicate that the occurrence can reach also higher rates (Kuttappan et al., 2012a). Moreover, Sihvo et al. (2014) have signaled a novel myopathy, vernacularly referred to as "wooden breast," which shows pale expansive areas of substantial hardness accompanied with white striation. The specific etiologies of these muscle defects are not well-known, even if it is thought there is an involvement of the rapid growth rate of broilers achieved in the modern strains by genetics (Petracci and Cavani, 2012). On the other hand, histological observations of these quality abnormalities evidenced similar features. Kuttappan et al. (2012b) found that major histopathological changes associated with white striping consisted of loss of cross striations, variability in fiber size, floccular/vacuolar degeneration and lysis of fibers, mild mineralization, occasional regeneration (nuclear rowing and multinucleated cells), mononuclear cell infiltration, lipidosis, and interstitial inflammation and fibrosis; all are histopathological features attributable to a chronic inflammatory process. Sihvo et al. (2014) observed moderate to severe multifocal regenerative myodegeneration and necrosis, with a variable amount of interstitial connective tissue accumulation or fibrosis in fillets affected by a wooden consistency. The same features were also observed by

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Petracci et al. (2013b) when comparing breast muscle traits in 2 chicken commercial hybrids having different breast yields. Histological evaluations showed that strain with higher breast yield exhibited a greater incidence of abnormal fibers; however, birds belonging to standard breast yield hybrid also showed a not negligible incidence of myopathic abnormalities. Kuttappan et al. (2013b) observed that macroscopic white striping abnormalities were not confined to a singular hybrid, but all the 4 different commercial strains considered exhibited some degree of white striping.

Previous studies have evidenced that the white striping abnormality impaired not only the visual appearance, but also had an impact on chemical composition (Kuttappan et al., 2012a; Petracci et al., 2014) and reduced ability to retain and pick up liquids of the meat during processing (Petracci et al., 2013a). In addition, Petracci et al. (2013b) showed that myopathic changes may be associated with a detrimental effect on meat quality attributes by decreasing protein content and deeply reducing the ability of the meat to hold liquids during processing and storage.

This study aimed at evaluating the influence of myodegeneration (mild, moderate, and severe) of pectoralis major muscle on quality traits and chemical composition of breast meat in broilers.

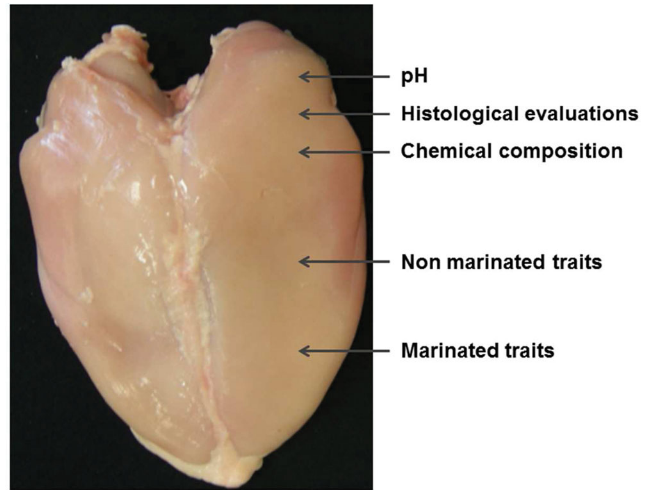

Figure 1. Positions for the determination of pH, histological traits, non-marinated technological properties (drip loss, cook loss, and Allo–Kramer shear force) and chemical composition (moisture, protein, lipid, ash, and collagen) as well as quality traits of marinated meat (marinade uptake, purge loss, total yield, and Allo–Kramer shear force).

MATERIALS AND METHODS

Bird Processing and Sample Collection

For this experiment, a total of 2,160 d old male chicks, from the 4 most representative commercial hybrids used worldwide, were supplied and hatched from the same commercial hatchery. The chicks were separately housed for genotype at a stocking density of 10 chicks/m^2 (60 birds/pen in 9 pens/group of about 6 m^2 each). Pens were equipped with pan feeders to assure at least 2 cm/bird of front space and an automatic drinking system (1 nipple/10 birds). Each pen was equipped with an individual bin as reservoir for the experimental feed, clearly labeled. On a daily basis, the feed was manually transferred from the bin to the feeder. Commercial feed was supplied to the chickens according to the following feeding program: starter (0 to 10 d), grower I (11 to 21 d), grower II (22 to 35 d), and finisher (36 d slaughtering). Feed and water were provided for ad libitum consumption.

Birds were slaughtered at the same body live weight of 3.6 kg. Two birds per each pen were randomly selected (18 birds/hybrid for a total of 72 birds), individually weighed, labeled, and processed under commercial conditions. The feed was withdrawn 4 h prior to catching, and birds were loaded in coops for transport to the plant. When the birds arrived at the processing plant, they were laired for 2 h, hung on a shackle line, and commercially processed. Birds were electrically stunned (120 V, 200 Hz) and after evisceration the carcasses were air-chilled (precooling at 5°C for 60 min, followed by chilling at 0°C for 90 min). At the exit of the chiller after 3 h postmortem, the carcasses were collected and approximately 1 cm^3 muscle sample of each pectoralis major breast muscle was removed, immediately frozen in liquid nitrogen-cooled in isopentane, and used to perform histology and immunohistochemistry analyses, as well as morphometrical and morphological evaluations. At 24 h postmortem, carcasses were deboned and cut up yields were determined according to the method described by the Working Group 5 of the World's Poultry Science Association (1984). Subsequently, color was measured on the bone-side surface of pectoralis major muscle, and pH and chemical composition (moisture, proteins, total lipids, ashes, and collagen) were determined on a sample obtained in the cranial part of each fillet according to the scheme reported in Figure 1. In addition, drip loss, cook loss, and Allo–Kramer shear values after cooking were determined on a sample removed from the middle part of each left breast fillet. Finally, a sample excised from the caudal part of each fillet were vacuum tumbled and marinade uptake, purge loss, cook loss, total yield, and Allo–Kramer shear values after cooking were determined on marinated samples.

Classification of Sample by Histology and Immunohistochemistry Evaluations

The specimens were oriented for the transverse fiber sectioning. For each pectoralis major muscle sample, 10 serial cross-sections (10 μm thick) were cut on a cryostat microtome at −20°C and mounted on poly-L-lysine coated glass slides (Sigma–Aldrich, St. Louis, MO) for histological (Masson's Trichrome) and immunohistochemistry. For immunohistochemistry, the avidin–biotin–peroxidase complex method was used. The sections were fixed for 10 min in 4% paraformaldehyde

in phosphate buffer saline (0.1 M, pH 7.2), rinsed in phosphate buffer saline, and incubated in 5% normal goat serum for 30 min at room temperature to reduce the nonspecific binding of the secondary antibodies. The sections were then incubated at 4°C in a humid chamber for 24 h with the polyclonal rabbit antiserum anti-CD3 1:1,000 (C7930, Sigma–Aldrich, St. Louis, MO). After washing, the sections were incubated at room temperature for 1 h with the biotin-conjugated goat antirabbit IgG secondary antibody, diluted 1:500 (Vector Laboratories, Burlingame, CA, USA), and then treated with avidin–biotin–peroxidase complex (Vector elite kit, Vector Laboratories, Burlingame, CA, USA). The immune reactions were visualized by applying a 3,3′-diaminobenzidine (DAB) chromogen solution (Vector DAB kit, Vector Laboratories, Burlingame, CA, USA). The sections were then counterstained with Orcein elastin stain, to detect if the inflammatory cells were located around skeletal muscle fibers and/or around the blood vessels.

In addition, for each pectoralis major muscle sample, the cross-sectional area of 100 fibers was estimated using KS 300 image analysis software (Kontron Elektronic, Munich, Germany) by outlining the fibers profiles on the monitor screen using a computer mouse. The fibers measured were selected at random, and the morphological and/or morphometric evaluation was performed in a blindly fashion. The cross-sectional area values were pooled for each experimental group and the mean was calculated.

For each section of muscle, the presence of abnormal fibers (giant fibers, fibers with hyaline degeneration, and damaged fibers with round profile) for 10 primary myofibers fascicle (PMF) was assessed and level of myodegeneration was graded as follows:

- Score F1, mild (n = 22): Abnormal fibers ranging from 2 to 4 for each PMF (Figure 2A);
- Score F2, moderate (n = 33): Abnormal fibers ranging from 5 to 10 for each PMF (Figure 2B);
- Score F3, severe (n = 17): Abnormal fibers represent the majority of the fibers for each primary myofibers fascicle (Figure 2C).

Meat Quality Analyses

Color (Commission Internationale de l'éclairage $L^* =$ lightness, $a^* =$ redness, and $b^* =$ yellowness) was measured in triplicate on the bone-side surface of each fillet using a Chroma Meter CR-400 (Minolta Corporation, Milan, Italy) and ultimate pH was determined by using a modification of the method initially described by Jeacocke (1977). Approximately 2.5 g meat samples were minced by hand, homogenized in 25 mL of 5 mM iodoacetate solution with 150 mM potassium chloride for 30 s, and the pH of the homogenate was determined using a pH meter calibrated at pH 4.0 and 7.0.

The samples weighing about 70 g obtained from the medial part of each fillet (8 × 4 × 3 cm) were placed in covered plastic boxes on sieved plastic racks in a 2 to 4°C cooler. After 48 h, samples were again weighed to determine the drip loss, and then vacuum-packaged and cooked in a water bath at 80°C for 45 min until the internal temperature reached 80°C (Petracci and Baéza, 2011). Meat samples were removed from the bags and reweighed to measure the cooking loss. After covered storage in a refrigerator for 2 h, a strip (approximately 2 × 4 × 1 cm) was excised from each cooked sample parallel to the fiber direction. Strips were sheared perpendicular to fiber direction using an TA.HDi Heavy Duty texture analyzer (Stable Micro Systems Ltd., Godalming, Surrey, U.K.) equipped with an Allo–Kramer shear cell using the procedure described by Sams et al. (1990). Shear values are reported as kilograms shear per gram of sample.

The samples (8 × 4 × 3 cm) weighing about 50 g excised from the caudal part of each fillet were individually tagged and marinated in a small-scale vacuum tumbler (model MGH-20, Vakona Qualitat, Lienen, Germany) with a 15% (wt/wt) water to meat ratio using a solution containing sodium tripolyphosphate (2.3%) and sodium chloride (7.6%), and the same tumbling conditions previously adopted by Petracci et al. (2013b). Meat samples were reweighed immediately after tumbling to measure marinade uptake and placed in covered plastic boxes on sieved plastic racks in a 2 to 4°C cooler. After 48 h, samples were again weighed to determine purge loss, and then vacuum packaged and cooked in an 80°C water bath for 24 min until the internal temperature reached 80°C. Meat samples were removed from the bags and reweighed to measure cook loss as well as total yield based on weight before marination. Finally, Allo–Kramer shear force was assessed as previously described.

As for proximate composition, moisture and ashes were determined in duplicate according to the Association of Official Analytical Chemists procedure (AOAC, 1990). Proteins were determined using the standard Kjeldahl copper catalyst method (AOAC, 1990). Total lipids were measured using a modification of the chloroform to methanol procedure described by Folch et al. (1957). Finally, collagen was determined on breast meat following the modified colorimetric method of Kolar (1990). About 4 g finely minced meat was hydrolyzed with sulfuric acid in an air oven at 105°C for 16 h. A reddish purple complex was formed by oxidizing with chloramine-T followed with 4-dimethylaminobenzaldehyde. The absorbance was measured at 558 nm and total collagen content was calculated by multiplying the amount of hydroxyproline by 7.5.

Statistical Analysis

The data were submitted to one-way ANOVA considering as main effect the myodegeneration level (F1 = mild, F2 = moderate, and F3 = severe) (SAS,

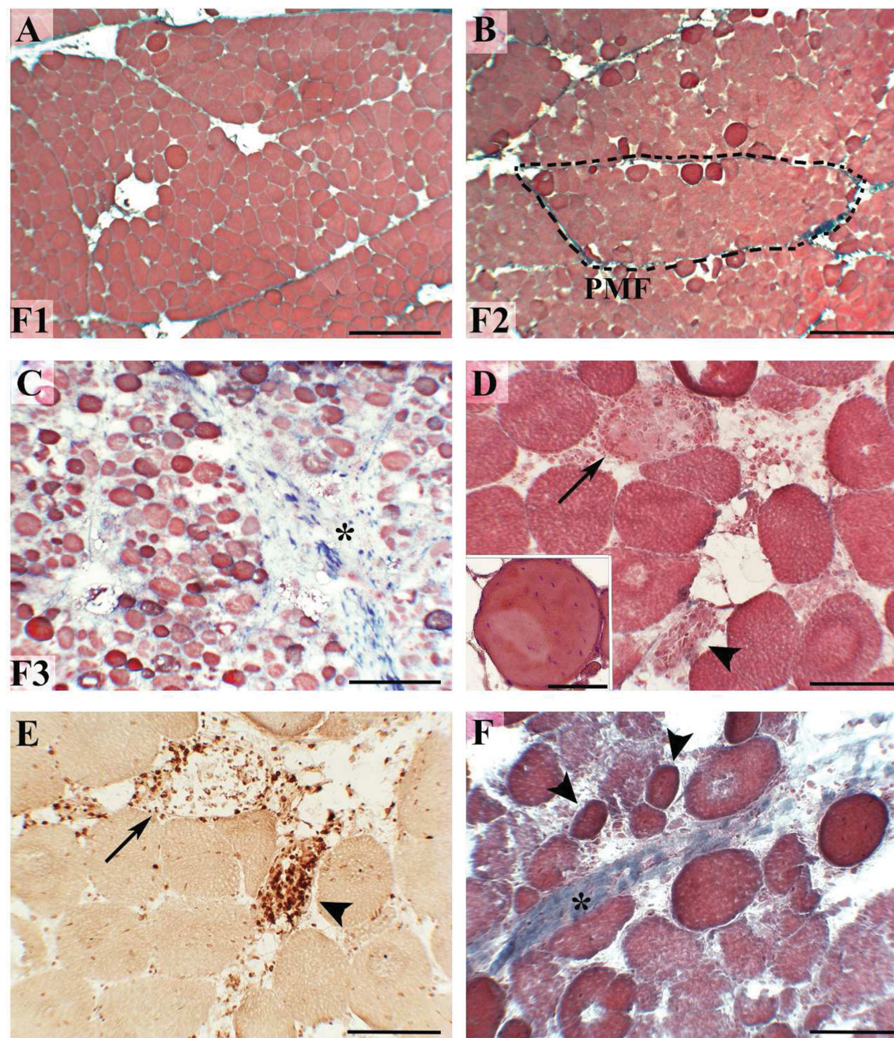

Figure 2. A = Muscle fibers in primary myofibers fascicle , illustrated in B, occur polygonal packed and homogenous in size with some abnormal fibers (stained with Masson's Trichrome, mild myodegenration, bar = 400 μm); B = number of abnormal fibers in the primary myofivers fascicle resulting in up to 10 (stained with Masson's Trichrome, moderate myodegenration, bar = 400 μm); C = number of muscle fibers is reduced; the muscle fibers, of variable size and rounded shape, are surrounded and separated or replaced by a loose or more organized connective tissue with the presence of inflammatory cell infiltration (asterisk), stained with Masson's Trichrome, severe myodegenration (bar = 400 μm); D = serial transverse section of breast muscle fibers (stained with Masson's Trichrome, bar = 100 μm); E = serial transverse section of breast muscle fibers (stained with CD3 antibody); arrows indicate a degenerate fiber, weakly stained, surrounded and infiltrated by mononuclear inflammatory cells (T lymphocytes) CD3 immunoreactive; furthermore, numerous T lymphocytes are clustered around a blood vessel (arrowheads), bar = 100 μm; inset, detail of an abnormal hyaline fiber with cytoplasm characterized by an amorphous-looking area and numerous central nuclei (bar = 20 μm); F = numerous tiny fibers (arrowheads) that surround greater-caliber fibers are immersed in a compromised perimysial connective tissue (asterisk); stained with Masson's Trichrome (bar = 100 μm).

1988). Means were separated using Bonferroni multiple range test with $P \leq 0.05$ considered as significant. Individual birds were considered as the experimental unit for the entire analysis.

RESULTS

The histological cross sections revealed myofibers of different diameter and devoid of their characteristic polygonal shape (Figure 2C), especially in samples with severe myodegeneration. In addition, diffuse thickening of the perimysial network with variable amounts of loose connective tissue, granulation tissue, or collagen-rich connective tissue (fibrosis) separating the muscle fibers were observed (Figure 2C). The morphological observations showed that most of the muscle fibers were characterized by multifocal degeneration and necrosis, amorphous-looking central area (central or eccentric) fibers, and infiltration of CD3-immunoreactive cells (T lymphocytes) within and around the degenerative fiber and/or blood vessels (Figure 2D, E). The degenerative lesions were often accompanied by thin fibers surrounded by larger-diameter fibers, in some cases weakly stained with Masson's Trichrome (Figure 2F), with several (up to 10) central nuclei. Samples showing moderate myodegeneration had the highest mean cross-sectional area of the fibers in respect to severe and mild groups (4,956 vs. 4,688 vs. 4,430 μm^2, $P < 0.001$) (Figure 3).

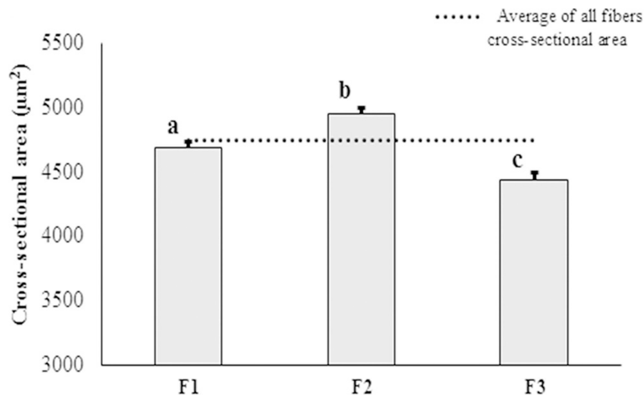

Figure 3. Effect of pectoralis major muscle degeneration on prevalence of abnormal fibers; F1 = 2 or 4 abnormal fibers; F2 = 5 or 10 abnormal fibers; F3 almost all abnormal fibers; bars with different superscript letters differ significantly ($P < 0.05$); values are expressed as mean ± SEM.

The carcass traits are presented in Table 1. Live weight at slaughter, carcass, and cut-up yields did not differ among groups. Quality traits of nonmarinated and marinated breast meat are reported in Tables 2 and 3, respectively. Among quality traits of nonmarinated meat, the only significant difference was found for cooking losses, which were lower in the mild group in respect to moderate and severe ones, which in turn did not differ from each other (21.4 vs. 24.7 and 24.7%; $P < 0.001$). Otherwise, pH, color coordinates ($L^*a^*b^*$), drip loss, and Allo–Kramer shear force were not affected by myodegeneration degree. On the other hand, chemical composition was noticeably modified. Breast muscles with severe damages showed higher moisture and lower protein percentages if compared with mildly degenerated samples, while moderate group exhibited intermediate values. The severe group had higher lipid content than moderate group as well as lower ash content if compared with both mild and moderate ones. Finally, collagen content was not modified by histological lesion levels. As for marinated meat, it was found that there was a higher percentage of marinade uptake in the mild group if compared with samples with severe myodegeneration, while the moderate group showed intermediate values. Mild samples also tended ($P = 0.057$) to exhibit lower cooking losses and overall had higher total marinade yield if compared with the other groups which did not differ from each other (95.5 vs. 93.2 and 92.5%; $P < 0.01$).

Table 1. Effect of pectoralis major muscle degeneration on slaughtering traits.

	Muscle degeneration degree				
Parameter	Mild (F1)	Moderate (F2)	Severe (F3)	SEM	Probability
n.	22	33	17		
Body live weight (g)	3,550	3,613	3,615	12.4	NS
Carcass weight (g)	2,588	2,618	2,645	10.7	NS
Carcass yield (%)	72.6	72.9	73.0	0.19	NS
Breast yield (%)[a]	28.1	29.1	28.5	0.27	NS
Thigh yield (%)[a]	15.2	14.9	14.9	0.12	NS
Drumstick (%)[a]	16.1	15.7	15.8	0.21	NS

[a]Based on carcass weight.

Table 2. Effect of pectoralis major muscle degeneration on quality traits of non-marinated breast meat.

	Muscle degeneration degree				
Parameter	Mild (F1)	Moderate (F2)	Severe (F3)	SEM	Probability
n.	22	33	17		
pH	5.96	5.95	6.01	0.01	NS
Color					
lightness (L^*)	54.7	55.1	54.7	0.29	NS
redness (a^*)	2.22	1.93	2.16	0.09	NS
yellowness (b^*)	2.77	2.97	3.07	0.10	NS
Drip loss (%)	1.31	1.48	1.44	0.05	NS
Cook loss (%)	21.4[b]	24.7[a]	24.7[a]	0.38	***
Allo–Kramer shear force (kg/g)	2.49	2.50	2.74	0.06	NS
Chemical composition					
moisture (%)	74.4[b]	74.8[a,b]	75.4[a]	0.13	*
protein (%)	22.6[a]	21.9[a,b]	21.1[b]	0.13	***
lipid (%)	2.57[a,b]	2.36[b]	2.94[a]	0.09	*
ash (%)	1.28[a]	1.26[a]	1.17[b]	0.02	**
collagen (%)	1.75	1.72	1.81	0.05	NS

[a,b]In scientific articles, differences among means are indicated by using different letters ($P \leq 0.05$).

* = $P \leq 0.05$.

** = $P < 0.01$.

*** = $P \leq 0.001$.

Table 3. Effect of pectoralis major muscle degeneration on quality traits of marinated breast meat.

Parameter	Muscle degeneration degree			SEM	Probability
	Mild (F1)	Moderate (F2)	Severe (F3)		
n.	22	33	17		
Marinade uptake (%)	13.1[a]	11.1[a,b]	10.4[b]	0.39	*
Purge loss (%)	2.17	1.87	2.09	0.07	NS
Cook loss (%)	13.7	14.5	14.5	0.16	0.057
Yield (%)	95.5[a]	93.2[b]	92.5[b]	0.38	**
Allo–Kramer shear force (kg/g)	2.80	2.69	2.78	0.07	NS

[a,b]In scientific articles, differences among means are indicated by using different letters ($P \leq 0.05$).

* = $P \leq 0.05$.

** = $P < 0.01$.

DISCUSSION

All considered samples were affected by mild to severe structural abnormalities of the pectoralis major muscle such as loss of the polygonal profile of the fibers, abnormal fibers increase, split fibers, compromised perimysial connective tissue, hyaline cytoplasm, nuclear internalization, inflammatory infiltration, and degeneration of fibers. Whereas many of the previous studies described breast muscle abnormalities in samples specially selected on the basis of macroscopic lesions (MacRae et al., 2006, 2007; Polak et al., 2009; Kuttappan et al., 2013b; Sihvo et al., 2014), in the present study fillets were randomly collected among heavy-sized broilers (live weight 3.6 kg) which are nevertheless recognized to be the market class that is more prone to develop higher incidence of myopathic lesions and meat abnormalities (Lorenzi et al., 2014; Kuttappan et al., 2013a).

Previously, Kuttappan et al. (2013b) observed a multiple rounded fibers and internalization of nuclei in severe white-striped breast muscles. Similarly, Sihvo et al. (2014) in chicken breast muscle showed myodegeneration accompanied by rounded fibers (reduced in number) and central nuclei. The presence of muscle fibers of small caliber shown in correspondence of the abnormal fibers is a further histological appearance that supports the hypothesis of muscular disorder. Abnormal fibers surrounded by fibers of small caliber (also referred to as regenerative fibers) have been described in chickens and turkeys with various myopathies including dystrophy (Pizzey and Barnard, 1983; Ashmore et al., 1988; Sósnicki et al., 1989; Polak et al., 2009).

In this study, many samples showed the presence of several muscle fibers with hyaline sarcoplasm and nuclei in the central position. These outcomes are in agreement with those previously observed in broiler (Nakada et al., 1998; MacRae et al., 2006; Polak et al., 2009) and turkey (Sósnicki et al., 1989; Mills et al., 1998) pectoralis muscles, where hyaline and necrotic fibers associated with central nuclei have been described. Moreover, we observed a diffuse thickening of the compromised endomysial and perimysial network with variable amounts of loose connective tissue, granulation tissue, or collagen-rich connective tissue (fibrosis) separating the muscle fibers. Hypercontraction of muscle fibers with prominent fibrosis has been also observed in turkey (Sósnicki et al., 1989, 1991) and chicken (Kuttappan et al., 2013b; Sihvo et al., 2014). In this regard, it has been hypothesized that in poultry selected for meat production, the growth of the connective tissue in muscle does not keep pace with muscle fiber radial growth and the fibers outgrow the supporting connective tissue, leading to muscle damage (Swatland, 1990; Kranen et al., 2000).

Necrotic fibers are among the most common abnormalities in broiler chickens (Soike and Bergmann, 1998; MacRae et al., 2006) and turkeys (Sósnicki et al., 1991). Our findings showed that the number of necrotic fibers was particularly noticeable in the breast muscle graded as moderate and severe, respectively. Generally, the damaged fibers appeared infiltrated by T lymphocytes with eventual lysis and phagocytosis of cell debris. The lymphocytic infiltration has also affected the endo- and perimysial connective tissue. In addition, perivascular inflammation, usually around veins, was observed in agreement with what described for white striping (Kuttappan et al., 2013b) and wooden breast abnormalities (Sihvo et al., 2014). The presence of T lymphocytes (confirmed by using of the specific chicken antibody) together with the aforementioned fibrosis and necrosis has confirmed that this is a chronic inflammatory process, which probably represents the undesired result of genetic pressure for increased growth rate of breast muscle.

Among the parameters used for evaluating the implication of muscle myodegeneration on meat quality, the greatest differences were observed in relation to chemical composition. Going from mild to severe level of myodegeneration, it was overall observed as a decrease of protein and ash content, and a concomitant increase of moisture and lipid. These modifications can be likely attributed to the occurrence of degeneration process for muscle fibers. Hence, increased presence of adipocytes at the perimysium level can explain the increase of intramuscular lipids, while loss of cellular liquids due to membrane damage following myodegeneration can justify the decrease of minerals. In addition, reduction in protein content coupled with moisture increase may also be an indirect effect of fiber degeneration and

atrophy. On the other hand, thickening connective tissue layers at perimysium level observed in samples with severe myodegeneration was not associated with a significant increase in total collagen content. However, if there was a strong decrease of total protein content in severely myodegenerated samples, we did not observe a proportional reduction in collagen content. In this regard, the percentages of collagen calculated on total protein content are 7.74, 7.85, and 8.58% in mild, moderate, and severe groups, respectively.

Similar features were previously observed when normal and severe white-striped fillets were compared (Kuttappan et al., 2012a; Petracci et al., 2014; Mudalal et al., 2014). Kuttappan et al. (2012a) found that severe white-striped fillets had higher fat content and lower protein content. The same differences were evidenced as well by Mudalal et al. (2014), who also observed a higher collagen content and a reduction of total amount of sarcoplasmic and myofibrillar proteins in severe white-striped fillets when compared to normal ones.

These modifications of the main components of the meat following myodegeneration also had some impact on its technological properties. In general, raw meat quality traits [such as ultimate pH, appearance (color), and ability to retain liquid during refrigerated storage (drip loss)] were not modified by muscle degeneration. Kuttappan et al. (2012a) and Petracci et al. (2013a), when comparing normal, moderate, and severe white-striped fillets, also found a small effect on neither pH, color, nor drip losses. On the contrary, in a previous study we observed increased drip losses in breast meat samples from a high-breast yield hybrid which overall presented a higher myodegeneration level (Petracci et al., 2013b). However, when abilities of the meat to retain its own liquids during cooking as well as to absorb marinade solution and retain it during cooking are considered, samples affected by severe histological lesions were noticeable impaired ($P < 0.05$). These modifications can be attributed to the set of microstructure observations previously described and to the strong decrease of proteins. Myofibrillar proteins are mainly responsible for the water holding capacity of the meat (Xiong, 2004), so it is not surprising that severe myodegeneration reduced the water holding ability. These results agree with our previous findings (Petracci et al., 2013a,b). In the first study, cook losses increased as the degree of white striping increased from normal to severe groups in both raw and marinated meat, even if marinade uptake was much lower (Petracci et al., 2013a). In the later study, breast meat from genotype with higher degree of histological lesions exhibited a decreased ability to retain liquid during cooking (Petracci et al., 2013b). It might be speculated that myodegeneration does not origin relevant modifications when the architecture of the muscle is maintained as intact, while if meat is submitted to a heat treatment or used for processing (i.e., marination) quality problems related to myodegeneration appear.

In conclusion, it has been demonstrated that currently almost all breast fillets of at least heavy broiler chickens produced under intensive farming systems had histological lesions such as fibers with hyaline degeneration, damaged fibers with a round profile, and necrotic fibers that can reach prevalence higher than 20% of severe cases. These lesions are most likely related to the growing occurrence of emerging breast meat abnormalities (white striping and wooden) and cover the main commercial hybrids currently used worldwide. As expected, it was also ascertained that the myodegeneration degree was reflected in an extensive modification of chemical composition and a noteworthy impairment of processing abilities of the meat.

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