Evaluation of White Striping prevalence and predisposing factors in broilers at slaughter

Elisa Russo,[*,1] Michele Drigo,[†] Corrado Longoni,[*] Raffaele Pezzotti,[*] Paolo Fasoli,[‡] and Camilla Recordati[§]

[*]*MSD Animal Health s.r.l., Via F.lli Cervi snc, Centro Direzionale Milano Due - Palazzo Canova, 20090, Segrate (MI), Italy;* [†]*Department of Animal Medicine, Production and Health, University of Padova, Viale dell'Università 16, 35020, Legnaro, Padova, Italy;* [‡]*Avicola Alimentare Monteverde s.r.l., via San Donato 31, Rovato (BS), Italy; and* [§]*Mouse & Animal Pathology Laboratory, Fondazione Filarete, Viale Ortles 22/4, 20139, Milano*

ABSTRACT White striping (**WS**) is an alteration of breast and thigh muscles of broiler chickens characterized by the presence of white striations parallel to the direction of muscle fibers. This study was performed to evaluate the prevalence and the predisposing factors to WS in commercial broilers of different weight reared in northern Italy. Fifty seven broiler flocks, including animals of medium- and heavy-weight, were grossly evaluated at slaughter for the presence of WS. For each flock, breeding data (mean BW at slaughter, ADG, sex, color of skin and fat, genetic line, age, antibiotic treatment, and prevalence of deep pectoral myopathy) were collected and statistically analyzed to assess their correlation with WS. Histology of breast fillets affected by different grades of WS was performed to evaluate potential differences between medium- and heavy-weight broilers. The overall prevalence of WS in medium- and heavy-weight broilers (mean BW 2.59 ± 0.13 kg and 3.64 ± 0.34 kg, respectively) was 70.2 ± 7.9% and 82.51 ± 8.5%, respectively, while the percentage of severe WS was 13.3 ± 7.1% and 25.7 ± 12.8%, respectively. A strong correlation was found between presence of WS, BW at slaughter, and ADG (Pearson correlation = 0.69, $P < 0.01$; Pearson correlation = 0.67, $P < 0.01$). WS also closely correlated with the prevalence of deep pectoral myopathy (Spearman's Rho slaughterhouse 1 = 0.74, Spearman's Rho slaughterhouse 2 = 0.51, $P < 0.01$). No correlation was found between genetics or sanitary status of the flock and WS. Histology confirmed that breasts with WS lesions were affected by a polyphasic degenerative and necrotizing myopathy, and that the lesions, as expected, were more severe in heavy-weight broilers. In conclusion, WS is a major concern in commercial meat poultry reared in Italy, affecting more severely heavier broilers, and it is mainly related to the BW and ADG of animals.

Key words: white striping, broiler chicken, breast fillet, daily gain, deep pectoral myopathy

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INTRODUCTION

In recent years, the market demand for a large amount of cheap poultry meat has pushed for an intensive genetic selection in this sector. Meat-type chickens are actually 2 times heavier at half the age than they were 50 years ago. This fast growth rate is associated to the onset of idiopathic and stress-induced myopathies such as deep pectoral myopathy, PSE-like disease, WS, and wooden breast (Petracci and Cavani, 2012).

White striping is described as an alteration of breast and thigh muscles characterized by the presence of white striations parallel to the direction of the muscle fibers. This condition is becoming increasingly important in meat-type chickens, and occurs mainly in heavier birds (Kuttappan et al., 2013a). Usually, WS is more evident on breast and thighs, but it can be observed also on tenders and drumsticks. Along a breast fillet it appears first in the cranial part, and then develops caudally (Kuttappan et al., 2013c). Kuttappan et al. (2013c) proposed to grossly classify the severity of WS into 3 categories (normal, moderate, and severe) based on the size and distribution of the white striations on the ventral surface of the superficial pectoral (*pectoralis major*) muscle. Histologically, WS is characterized by polyphasic lesions consistent with a degenerative myopathy with mild regeneration and replacement of damaged muscles by adipocytes and fibrosis (Kuttappan et al., 2013c).

According to Kuttappan et al. (2012c), the presence of WS does not impair the safety of the meat, but impairs its acceptance by the consumer as well as its quality. The consumer dislikes the visual appearance of raw breast fillets affected by WS and consider normal fillet more suitable for consumption. Breast fillets

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[1]Corresponding author: elisa.russo1982@gmail.com

affected by WS exhibit an increase in fat content and a decrease in protein content due to the replacement of necrotic muscle fibers by adipocytes, resulting in a more calorific meat. Also, the fatty acid profiles are different: normal breast fillets are richer in saturated fatty acid and polyunsaturated fatty acids, whereas breast fillets severely affected by WS are richer in monounsaturated fatty acid but poorer in eicosapentaenoic acid and docosahexaenoic acid, both of which are appreciated by the consumer (Kuttappan et al., 2012a, 2013b). Severe WS affects the quality parameters of poultry meat. The pH value is significantly higher, the Allo-Kramer-shear force is lower, the cook losses are increased, and the capacity for picking up marinade solutions is lower. All these abnormalities result in bad technological quality of the meat and in yield loss when it is employed for food secondary processing (Petracci et al., 2013).

The prevalence of WS has been evaluated in a few studies and results varied widely depending on the examined geographic region and weight of animals at slaughter: from 52 to 76% in the United States (Kuttappan et al., 2012a; Kuttappan et al., 2013a), and 12% in Italy (Petracci et al., 2013). The pathogenesis of WS is still unknown, but previous studies demonstrated that the prevalence of this defect is related to the growth performance of broilers and the BW at slaughter (Kuttappan et al., 2012a, 2013b). Also, the diet can influence the occurrence of WS since at the same age, animals fed with a low-energy diet presented less WS than animals fed with a high-energy diet (Kuttappan et al., 2012a); the dietary vitamin E level is not associated with WS in broilers (Kuttappan et al., 2012b). As regards the role of genetics, it is still unresolved whether the occurrence of WS is related to the genetic characteristics of the broilers, or to the different growing performance of distinct genetic lines (Kuttappan et al., 2013a).

The aims of this study were: 1) to evaluate the prevalence of WS in commercial broilers of different weights (medium and heavy) reared in Italy; 2) to identify potential factors correlated with the development of WS; and 3) to evaluate histologically the WS lesions in broilers of different weight.

MATERIALS AND METHODS

Sampling

This study was performed in 3 slaughterhouses located in northern Italy. Fifty seven broiler flocks were evaluated, including 19 flocks of female broilers slaughtered at medium weight (mean BW 2.6 kg, mean age 46 days), and 38 flocks of male broilers slaughtered at heavy-weight (mean BW 3.6 kg, mean age 55 days). To minimize seasonal effects, all sampling was performed during September 2013.

To evaluate the prevalence of WS, for each examined flock, 400 breasts were grossly scored for the presence of WS according to the grading system proposed by Kuttappan et al. (2013c): 0 = normal breast (no striations), 1 = moderate lesion (presence of striations <1 mm thick visible on breast surface), and 2 = severe lesion (presence of striations >1 mm thick visible on breast surface). A mean score and the percentage of the moderate (score 1) and severe (score 2) lesions were calculated for each flock.

To analyze the potential presence of correlations between WS and breeding parameters, the following data regarding each examined flock were collected: mean BW at slaughter, ADG, sex, color of skin and fat (dependent on the primary source of carbohydrates used in the diet: white for wheat, and yellow for corn), genetic line, age, antibiotic treatment (i.e., number of days during which the animals received antibiotics, excluding ionophores used as coccidiostat), and prevalence of deep pectoral myopathy (data on deep pectoral myopathy were collected in 2 of the 3 slaughterhouses, consisting 36 and 30 flocks).

Histology

After scoring for the gross degree of WS, muscle samples were collected for histological examination from the ventro-cranial portion of the superficial pectoral (*pectoralis major*) muscle and fixed in 10% neutral buffered formalin. Cross and longitudinal sections were obtained from each muscle sample and paraffin was embedded. Tissue sections were routinely stained with hematoxylin and eosin and evaluated in a blind fashion under a light microscope. Grading of muscular lesions (including separate evaluation of muscular degeneration/necrosis, fibrosis, adipose tissue infiltration, and perivascular inflammatory cell infiltrates) was performed as follows: grade 0, normal; grade 1, minimal; grade 2, mild; grade 3, moderate; and grade 4, severe.

Statistical Analysis

Normality of the distributions was tested with the Kolmogorov–Smirnov nonparametric test. Firstly, the Pearson correlation coefficient was calculated to evaluate the correlation between WS mean score and the main independent variables (BW and ADG), and between WS score 2 percentage and the main independent variables. Then, mean WS score and WS score 2 percentage were analyzed with the following linear models, including color of skin and fat, genetics, antibiotic treatment, slaughterhouse, prevalence of deep pectoral myopathy, and ADG as predictors (sex and age were not included in the analysis because they were highly correlated with ADG), as follows:

Model 1: $y_{ijk} = \mu + color_j + \beta_1 ADG_{xjk} + \beta_2 ADG^{*}color + \epsilon_{ijk}$, where y_{ijk} is WS mean score or WS score 2 percentage; μ is the intercept; $color_j$ is the fixed effect of the color; β_1 is the regression coefficient of y_{ijk} on animal ADG; β_2 is the regression coefficient of y_{ijk} on the interaction ADG*color; ϵ_{ijk} is the residual error.

Table 1. BW, ADG, percentage of WS score 0 (normal breast), 1 (moderate lesion), 2 (severe lesion) and mean WS score in medium size flocks and heavy flocks.

			Prevalence of WS (%)[1]				
Group	BW (kg)	ADG (g/day)	Score 0 (normal)	Score 1 (moderate)	Score 2 (severe)	Total prevalence	Mean WS[1] score
Medium	2.59 ± 0.13	56.78 ± 6.14	29.8 ± 7.9	56.9 ± 7.8	13.3 ± 7.1	70.2 ± 7.9	0.84 ± 0.13
Heavy	3.64 ± 0.34	65.94 ± 5.87	17.5 ± 8.5	56.8 ± 10.3	25.7 ± 12.8	82.51 ± 8.5	1.09 ± 0.19

[1]WS = White Striping.

Model 2: $y_{ijk} = \mu + genetic_j + \beta_1\ ADG_{xjk} + \beta_2\ ADG^{*}genetic + \epsilon_{ijk}$, where y_{ijk} is WS mean score or WS score 2 percentage; μ is the intercept; $genetic_j$ is the fixed effect of the genetic; β_1 is the regression coefficient of y_{ijk} on animal ADG; β_2 is the regression coefficient of y_{ijk} on the interaction ADG*genetic; ϵ_{ijk} is the residual error.

Model 3: $y_{ijk} = \mu + \beta_1$ antibiotic $treatment_j + \beta_2\ ADG_{xjk} + \beta_3\ ADG^{*}$antibiotic $treatment_j + \epsilon_{ijk}$, where y_{ijk} is WS mean score or WS score 2 percentage; μ is the intercept; β_1 is the regression coefficient of y_{ijk} on antibiotic treatment measured in days of treatment; β_2 is the regression coefficient of y_{ijk} on animal ADG; β_3 is the regression coefficient of y_{ijk} on the interaction ADG*antibiotic treatment; ϵ_{ijk} is the residual error.

Model 4: $y_{ijk} = \mu + slaughterhouse_j + \beta_1$ deep pectoral myopathy $percentage_{xjk} + \beta_2\ ADG + \beta_3$ deep pectoral myopathy percentage*slaughterhouse + β_4 deep pectoral myopathy percentage*ADG ϵ_{ijk}, where y_{ijk} is WS mean score or WS score 2 percentage; μ is the intercept; $slaughterhouse_j$ is the fixed effect of the slaughterhouse; β_1 is the regression coefficient of y_{ijk} on deep pectoral myopathy percentage; β_2 is the regression coefficient of y_{ijk} on animal ADG, β_3 is the regression coefficient of y_{ijk} on the interaction deep pectoral myopathy percentage*slaughterhouse, β_4 is the regression coefficient of y_{ijk} on the interaction deep pectoral myopathy percentage*ADG; ϵ_{ijk} is the residual error. The slaughterhouse was included in this model because each slaughterhouse followed a different internal protocol to evaluate the prevalence of deep pectoral myopathy.

Finally, Spearman's test was performed to evaluate the correlation between WS mean score and percentage of deep pectoral myopathy in the 2 slaughterhouses, and between WS score 2 and percentage of deep pectoral myopathy in the 2 slaughterhouses.

The Mann-Whitney U test was performed to compare the histological grading of medium- and heavy-weight breast with the same macroscopic score.

Statistical significance was set at $P < 0.05$.

RESULTS

The overall prevalence of WS was 78.4 ± 9.7%, and the mean score was 1.00 ± 0.21. The overall prevalence of WS in medium- and heavy-weight broilers was 70.2 ± 7.9% and 82.51 ± 8.5%, respectively. The prevalence of WS score 0, 1 and 2, mean BW and ADG for medium- and heavy-weight broilers are reported in Table 1.

Mean WS score and WS score 2 percentage resulted highly correlated with the mean BW (mean WS score: Pearson correlation 0.69, $P < 0.01$; WS score 2 percentage: Pearson correlation 0.62, $P < 0.01$), and with ADG (mean WS score: Pearson correlation 0.67, $P < 0.01$; WS score 2 percentage: Pearson correlation 0.67, $P < 0.01$). The ADG was the main factor associated with both mean WS score and WS score 2 percentage in all the models in which it was tested. The factors color of skin and fat, genetic line, and antibiotic treatment history were not associated with mean WS score or WS score 2 percentage (Table 2).

The factor deep pectoral myopathy percentage correlated with mean WS score (Spearman's Rho slaughterhouse 1 = 0.74, Spearman's Rho slaughterhouse 2 = 0.51, $P < 0.001$) and WS score 2 percentage (Spearman's Rho slaughterhouse 1 = 0.77, Spearman's Rho slaughterhouse 2 = 0.66, $P < 0.001$); in model 4 this effect was significant ($P = 0.011$) only for WS score 2 percentage; the interaction deep pectoral myopathy*slaughterhouse was also associated to both mean WS score and WS score 2 percentage (Table 3).

Histologically, breast samples with WS were affected by multifocal muscular degeneration and necrosis at various stages of development, associated with muscular regeneration, fibrosis, and adipose tissue infiltration (Figure 1). Occasionally, multifocal perivenular mononuclear cell (lymphofollicular) infiltrates were found in the perimysium (Figure 2). The degree of severity of the muscular lesions increased from normal breasts (score 0) to breasts affected by severe lesions (score 2). Overall, in heavy-weight broilers, lesions were more severe than in medium-weight broilers. All breasts classified as normal (score 0) were affected by at least minimal muscular degenerative changes and adipose tissue infiltration, occasionally associated with perivascular mononuclear cell infiltrates. Results of the histopathological examination are summarized in Table 4.

DISCUSSION

This study evaluated the presence of WS at slaughter in 2 broiler weight categories (medium and heavy). The overall prevalence found in this study (78%) is similar

Table 2. Analysis of Variance.

	Variable[1]	Effect	df	Mean Square	F	P^2
Model 1[3]	Mean WS score ($R^2 = 0.50$)	ADG	1	0.539	23.473	<0.001
		Color	1	0.015	0.670	NS
		ADG*color	1	0.011	0.479	NS
	Percentage of WS score 2 ($R^2 = 0.46$)	ADG	1	2395.183	27.278	<0.001
		Color	1	0.040	0.000	NS
		ADG*color	1	0.880	0.010	NS
Model 2[4]	Mean WS score ($R^2 = 0.56$)	ADG	1	0.981	48.433	<0.001
		Genetic	1	0.053	2.620	NS
		ADG*genetic	1	0.039	1.917	NS
	Percentage of WS score 2 ($R^2 = 0.53$)	ADG	1	3508.889	44.890	<0.001
		Genetic	1	121.805	1.558	NS
		ADG*genetic	1	89.457	1.144	NS
Model 3[5]	Mean WS score ($R^2 = 0.45$)	ADG	1	1.066	45.672	<0.001
		Antibiotic treatments	1	0.038	1.616	NS
		ADG*Antibiotic treatment	1	0.018	0.748	NS
	Percentage of WS score 2 ($R^2 = 0.46$)	ADG	1	3777.748	44.918	<0.001
		Antibiotic treatments	1	104.802	1.246	NS
		ADG*Antibiotic treatment	1	0.032	0.000	NS

[1]WS = White Striping.
[2]NS = Not Significant.
[3]The analysis included 55 records, 42 white broiler, 13 yellow broiler.
[4]The analysis included 40 records, 16 genetic 1, 24 genetic 2.
[5]The analysis included 55 records.

Table 3. Analysis of Variance.

	Variable[1]	Effect	df	Mean Square	F	P^2
Model 4[3]	Mean WS score ($R^2 = 0.61$)	Percentage of deep pectoral myopathy	1	0.057	3.136	NS
		Slaughterhouse	1	0.000	0.018	NS
		ADG	1	0.131	7.259	0.010
		Percentage of deep pectoral myopathy *Slaughterhouse	1	0.125	6.941	0.012
		Percentage of deep pectoral myopathy *ADG	1	0.028	1.527	NS
	Percentage of WS score 2 ($R^2 = 0.72$)	Percentage of deep pectoral myopathy	1	299.060	7.027	0.011
		Slaughterhouse	1	0.192	0.005	NS
		ADG	1	344.205	8.087	0.007
		Percentage of deep pectoral myopathy *Slaughterhouse	1	670.351	15.750	<0.001
		Percentage of deep pectoral myopathy *ADG	1	148.55	3.490	NS

[1]WS = White Striping.
[2]NS = Not Significant.
[3]The analysis included 50 records, 20 slaughterhouse 1, 30 slaughterhouse 2.

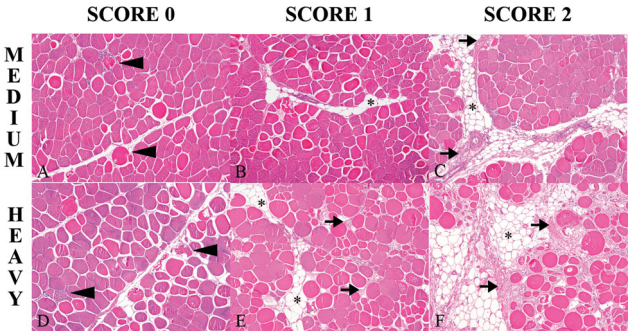

Figure 1. Histology of breast muscles of medium and heavy-weight broilers with White Striping score 0 (normal) (A, D), score 1 (moderate) (B, E), and score 2 (severe) (C, F) (hematoxylin and eosin staining, 100×). Grade of muscular degeneration (arrowheads), fibrosis (arrows), and adipose tissue infiltration (*) is more severe in heavy-weight broilers than medium-weight broilers, and increases from score 0 to score 2.

to the prevalence reported by Kuttappan et al. (2012a, 2013a) in the United States (54 to 76%), while it is considerably higher compared to the prevalence reported by Petracci et al. (2013) in Italy (12%).

In that study, the BW at slaughter was lower than the BW of the animals considered in the present study (Petracci et al. 2.75 kg, this study 3.29 kg), but this is likely not enough to explain such a large difference in the prevalence of WS between the 2 studies, suggesting that others factors might influence the prevalence of WS.

Body weight has previously been considered the most important predisposing factor for the development of WS (Kuttappan et al., 2012a, 2013b). The statistical analysis performed in this study showed clearly that in addition to the BW at slaughter, another important variable associated to WS is the ADG. This result

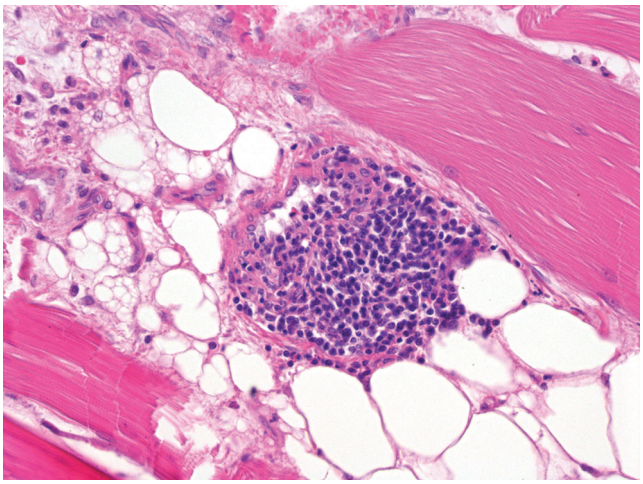

Figure 2. Histology of the breast muscle of a heavy-weight broiler with White Striping score 2 (hematoxylin and eosin stainig, 400×). In the perimysium, a perivenular mononuclear follicle-like infiltrate is present, infiltrating also the vein wall.

suggests that WS lesions could be reduced by modifying the diet and the growing curve, as reported by Kuttappan et al. (2012a). Since genetics was identified by Kuttappan et al. (2013a) as a potential predisposing factor, in the present study it was evaluated as a risk factor in a model taking ADG into account The result showed that genetics did not affect the prevalence of WS. However, genetic hybrids characterized by a high ADG have a higher WS prevalence than the ones characterized by lower ADG, which could explain the results obtained by Kuttappan et al. (2013a). The absence of a correlation between WS and antibiotic treatment suggests that there is no relationship between WS and the sanitary status of flocks. A strong correlation between WS and deep pectoral myopathy was found. Deep pectoral myopathy is a muscular lesion of deep pectoral muscle well described in both meat-type chickens and turkeys (Siller, 1985). The pathogenesis of WS is still unknown, but it seems to be completely different from the pathogenesis of deep pectoral myopathy. WS is a degenerative myopathy that can involve many muscles (breast, thighs, tenders, and drumsticks), while deep pectoral myopathy is characterized by acute necrosis restricted to the middle third of the *pectoralis minor* muscle. This necrosis is the result of ischemia produced by an increase of pressure in the portion between the tight fascia and the sternum when a heavy muscular effort is performed and the muscle increases in size (Siller, 1985). The predisposing factors of deep pectoral myopathy include genetics, high BW, and sudden intensive physical activity such as vigorous wing flapping when the birds are improperly handled (Bianchi et al., 2006, Petracci and Cavani, 2012). The results of our study confirm that the correlation between WS and deep pectoral myopathy is indeed due to the fact that these 2 conditions share ADG as a risk factor.

Histological examination confirmed that breasts with WS lesions were affected by a multifocal and polyphasic degenerative and necrotizing myopathy, as previously reported (Kuttappan et al., 2013c). As expected, degree of severity of lesions increased from moderately to severely affected breasts, and lesions were more severe in heavy-weight broilers than in medium-weight broilers, as indicated also by the results of the gross examination. Increased severity of WS lesions in heavier animals could be related either to their larger muscular masses, or to the later age of sacrifice. Despite the gross absence of lesions, normal breasts were always affected by mild muscular changes (muscular degeneration and fat infiltration), indicating that myopathic lesions are common in meat-type chickens. In examined muscle samples, perivascular (perivenular) lymphofollicular aggregates were also found. This finding has never been described in WS breasts, but it was recently reported in wooden breasts (Sihvo et al., 2014) suggesting a common pathogenesis for these conditions affecting meat-type chickens.

In conclusion, WS is widespread in commercial meat poultry in Italy, as confirmed by the high prevalence (78%) found in this study, it is more severe in heavier broilers, and mainly related with the BW and ADG of the flock.

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Table 4. Histological grading of muscular lesions in the breast muscles. Results are expressed as median score per group.

WS[1] score	Group	n	Muscular degeneration/necrosis	Fibrosis	Adipose tissue infiltration	Perivenular inflammatory infiltrates
0	Medium	9	1	0	1	0
	Heavy	12	1	0	1	0.5
1	Medium	9	1	0^{a}	2^{a}	0
	Heavy	11	2	1^{b}	2.5^{b}	1
2	Medium	10	3	0^{A}	2.5^{a}	2
	Heavy	10	3	2^{B}	3^{b}	2

[1]WS = White Striping.

[a,b]Median values within each column with different letters are significantly different ($P < 0.05$).

[A,B]Median values within each column with different letters are significantly different ($P < 0.01$).

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