Impact of coccidiosis control program and feeding plan on white striping prevalence and severity degree on broiler breast fillets evaluated at three growing ages

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ABSTRACT This study investigated the impact of 2 coccidiosis control systems (vaccine vs anticoccidial) and 2 feeding plans (standard energy vs low energy content, the latter supplemented with threonine and enzymes in the second half of the production cycle) on white striping (WS) prevalence and severity in chicken broiler breasts at commercial slaughter age (51 d). The age of lesion onset was also investigated with the sacrifice of 80 chicks at 12, and 80 chicks at 25 d of age. Seven hundred and twenty ROSS 708 strain male chicks were divided into 4 groups: a non-vaccinated group fed with standard diet (CONTROL); two groups vaccinated against coccidiosis but fed either a standard diet (VACC) or a low-energy diet supplemented with threonine and enzymes (VACC–LE plus); and a fourth group fed a standard diet containing anticoccidial additive except during the finishing period (COX). After live performance, yields, and fillet pH were measured, the breasts were weighed and scored as level 0 (no WS), level 1 (moderate WS), and level 2 (severe WS) at each of the 3 ages; data were covariate for slaughter weight. The results suggest an ameliorative effect of coccidiosis control systems when compared to the control group in terms of live weight, breast yield, and whole breast weight, with heavier fillets characterized by higher pH values. WS appeared at 25 d of age with an average prevalence of 11.5% and with lesions of moderate severity. There were no statistically significant differences due to the experimental treatment at this age. At commercial slaughter age, total average prevalence was 96%, with COX birds showing higher level 2 prevalence (77.6%). This could be related to the higher slaughter weight reached by the COX group ($P < 0.001$) and the treatment effect ($P < 0.01$) that probably adds to the effect of live weight. Diet had no effect on overall live performance of VACC-LE plus chickens, which were similar to those of the VACC group.

Key words: chicken, white striping, coccidiosis control, feeding plan

2015 Poultry Science 94:2114–2123
http://dx.doi.org/10.3382/ps/pev205

INTRODUCTION

In recent decades, increased consumer demand has required producers to intensify production output, mainly through high-energy diets (Kuttappan et al., 2012a) and an intensive selection of strains exhibiting faster growth with higher breast yields; an increase in the incidence of *Pectoralis major* myopathies has however been observed over this same period (Petracci et al., 2013a). White Striping (**WS**), an emerging meat quality issue, is a muscle defect described as the appearance of macroscopic white striations running parallel to the direction of muscle fibers in broiler breast fillets first pointed out by Bauermeister et al. (2009) and Kuttappan et al. (2009). More recently, Kuttappan et al. (2013a) noticed that these lesions could exhibit a variable degree of severity and suggested a score based on the size and distribution of white striations on the breast surface. Fillets with apparently no lesions were classified as normal; those with easily observed striations generally having a thickness less than 1 mm were scored as moderate; and breasts with white striations thicker than 1 mm covering a greater surface area were scored as severe. Macroscopic WS prevalence ranges from 12 to over 50% (Kuttappan et al., 2009, 2012a; Petracci et al., 2013b). Poultry processors are obliged to downgrade WS breasts and process them further instead of being able to offer them for fresh retailing, at great economic loss (Petracci et al., 2013b). Indeed, visual appearance is the most important attribute available for consumers to assess the quality of a meat product at purchase, and WS may decrease breast

Received March 6, 2015.
Accepted May 15, 2015.
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fillet acceptability (Kuttappan et al., 2012b). Histologically, a widespread degenerative myopathy associated with chronic lesions characterizes severe degrees of WS. Hypertrophy, cell changes (appearance of giant fibers, small size, rounded fibers, internalization of nuclei, loss of cross striations), and tissue damage with degeneration and necrosis of fibers (appearance of inflammatory cell infiltration, fibrosis, lipidosis) have all been observed (Kuttappan et al., 2013a; Petracci et al., 2013a). Several authors associate WS occurrence with higher live weight at older ages (Bauermeister et al., 2009) or the higher growth rate of birds with heavier breasts (Kuttappan et al., 2009, 2013a). Genotype seems to play a key role, given that high breast yield strains generated thicker fillets with higher prevalence of both moderate and severe WS than standard breast strains (Kuttappan et al., 2012a). The implementation of diets with higher energy levels might also play a role, since growth rate and breast meat yield are both higher in birds fed this type of diet than those fed reduced energy diets (Kuttappan et al., 2012a). Breast muscle portions are not affected with the same intensity. The severity of WS is greater toward the cranial end of the ventral surface, where the fillet exhibits maximum thickness, than the caudal portion (Kuttappan et al., 2013a), probably because convexity or stretching is greater in this region. Moreover, Brewer et al. (2012) found a high correlation (r = 0.81) between fillet weight and cranial thickness. This finding suggests greater WS occurs in conjunction with heavier or thicker fillets (Kuttappan et al., 2013c). White and red muscles are not subject to the same WS occurrence; Kuttappan et al. (2013c) found that *Pectoralis major* and *Iliotibialis* presented a higher incidence than *Gastrocnemius*, suggesting the greater susceptibility of white glycolytic fibers. Hoving-Bolink et al. (2000) suggested that genetic selection for chickens with higher breast yield might have adversely affected blood supply; indeed, the lower capillary density might have resulted in a reduced supply of nutrients, oxygen, and the removal of catabolites, leading to tissue damage. Moreover, Kuttappan et al. (2013a) suggested that severe degrees of WS (associated with faster bird growth rate) make muscles prone to developing overstretching, or ischemia, resulting in tissue damage and attempted reparative response. Not all potential causes of WS lesion have been fully investigated. To date, no studies have explored the link between anticoccidials and WS, even though the toxicity of coccidiostats in skeletal muscle, visible as focal degeneration and necrosis, has already been reported in broilers (Sandercock and Mitchell, 2003; Chapman et al., 2010), turkeys (Markiewicz et al., 2014), and other species (Novilla, 1992).

Our study was conducted to investigate certain aspects not yet considered, such as the impact of 2 coccidiosis control programs (vaccine vs anticoccidial additive) and feeding plan on WS occurrence. This also implied considering the relationship between anticoccidial drug administration and anticoccidial vaccination of chickens fed diets with different energy density and bioavailability of nutrients and WS prevalence. Moreover, the observation of the breasts of chickens at different ages (12, 25 and 51 d) was intended to identify the age of onset of WS. This study will hopefully make a significant contribution to the identification or exclusion of factors leading to WS.

MATERIALS AND METHODS

Experimental Design and Management

The experiment was performed in a broiler unit consisting of a central corridor and 12 pens on each side. In this study, 720 male 1-day-old chicks of ROSS 708 hybrid were individually tagged at wing level, randomly divided into 4 experimental groups (**CONTROL**, **VACC**, **VACC-LE plus**, **COX**) of 180 birds each, and housed in floor pens until 51 d of age. They were vaccinated for Newcastle disease, infectious bronchitis, and Marek's disease at the hatchery.

On the fourth day of life, the vaccine against coccidiosis Paracox-5 (MSD Animal Health S.r.l., Segrate, Milano, Italy) was administered in drinking water to chicks in groups VACC and VACC-LE plus, following the manufacturer's instructions.

The rearing period was divided into 4 feeding phases: first period (0 to 12 d of age), second period (12 to 25 d of age), third period (25 to 42 d of age), and fourth period (42 to 51 d of age).

Animals received different feeding plans: the non–vaccinated groups (CONTROL and COX) and one vaccinated group (VACC) were fed standard diets throughout the entire rearing period, whereas group VACC–LE plus was fed low energy diets (C3 and C4) supplemented with threonine (0.6 and 0.4 g/kg, respectively), xylanase (2 g/kg) and Quantum^{TM} phytase 2500 D (6-phytase) (EC 3.1.3.26) (0.3 g/kg) enzymes in the last 2 periods. The COX diet contained anticoccidial additive except during the finishing period.

Feed and water were provided for ad libitum consumption and feed was provided daily in the feeders. During the first and the second periods, birds were reared in 3 m^2 pens at a stocking density of 20 birds/m^2 (60 birds/pen) and divided into 12 pens (3 replicates/treatment).

At the first and the second feeding changes (12 and 25 d of age), 2 intermediate slaughterings were performed with the sacrifice of 80 animals (20 per treatment) each (160 slaughtered overall) by cervical vertebrae dislocation as specified by Reg. (CE) No. 1099/2009.

At 26 d of age, the remaining chickens in each pen were equally allotted into two 3 m^2 pens, in this way doubling the number of pens per treatment (6 replicates/treatment) with a stocking density at 51 d of 26.4 kg/m^2 (an average number of 22 birds/pen, 7.3 birds/m^2). All pen floors were covered with beech shavings and equipped with a 120 cm circumference feeder (to ensure 4.4 cm of front space) and a 40 cm

diameter automatic bell drinker. Photoperiod length was set to Council Directive 2007/43/EC recommendations. Environmental conditions were recorded daily by measuring the minimum and maximum temperatures inside pens at ground level and in the poultry house central hallway, where relative humidity was also measured. Light intensity was monitored at bird head height using a digital illuminometer (4 in 1 Multi-Function Environment Meter Lafayette; Product code: DT-8820). Animals were monitored twice daily throughout the study to assess availability of feed and water, mortality, and any potential conditions of morbidity.

At every feeding change (12, 25, and 42 d of age) and at the end of the rearing period, all birds were individually weighed. Feed intake (**FI**) was recorded by weighing the feed provided and subtracting the residuals. Average daily gain (**ADG**), FI, feed conversion ratio (**FCR** = kg of feed consumed/kg of weight gain), and mortality were determined for each feeding phase for the entire rearing period with the pooling of the first 2 (0 to 25 d) and the last 2 (25 to 51 d) periods.

The study was approved by the Ethical Committee for Animal Experimentation of the University of Padova, Italy (Project number 17/2014 approved in May 2014, Prot. No. 71360). Birds were handled according to the principles stated in EC Directive 86/609/EEC regarding the protection of animals used for experimental and other scientific purposes.

Feeding Plan

Nutega (Nuevas Tecnologías de Gestión Alimentaria S.L., Madrid, Spain) formulated the diets according to Ross 708 nutrient specifications. During the first 2 feeding phases (0 to 12 and 12 to 25 d of age), birds in CONTROL, VACC and VACC-LE plus groups received the same standard diet indicated as B1 (Starter) and B2 (Grower), respectively. Birds from COX group received A1 (Starter) and A2 (Grower) diets with the same chemical composition as the B1 and B2 diets containing the anticoccidial Maxiban G 160 premix in a 0.5 kg/ton dose (Elanco Animal Health division of Eli Lilly & Co., Indianapolis, IN). During the third feeding phase (25 to 42 d of age), CONTROL and VACC groups received diet B3, whereas COX group received diet A3 enriched with anticoccidial Elancoban 200 premix in a 0.5 kg/ton dose (Elanco Animal Health division of Eli Lilly & Co., Indianapolis, IN). VACC-LE plus group received a finishing diet C3 characterized by lower energy content (4,560 kcal gross energy (**GE**)/kg) than the A3 and B3 diets (4,638 and 4,659 kcal GE/kg, respectively). During the finishing period (42 to 51 d of age), CONTROL, COX, and VACC groups received a standard Finisher diet (4,639 kcal GE/kg), whereas VACC-LE plus birds continued receiving a low-energy diet (C4; 4,551 kcal GE/kg) (Tables 1 and 2). As above stated, in the periods 3 and 4 the diets C3 and C4 were also supplemented with threonine, xylanase, and phytase, with the purpose of maximizing nutrient bioavailability to birds, and to verify if the combination with a low energy diet could be beneficial for WS prevalence reduction.

Table 1. Feeding plan.

	Treatments (T)			
	CONTROL	COX	VACC	VACC-LE plus
Period 1 (0 to 12 d)	B1	A1	B1	B1
Period 2 (12 to 25 d)	B2	A2	B2	B2
Period 3 (25 to 42 d)	B3	A3	B3	C3
Period 4 (42 to 51 d)	Finisher	Finisher	Finisher	C4

Chemical Analysis of Feed Samples

Experimental diets were sampled, finely ground, and analyzed using AOAC (2002) methods to determine the concentrations of dry matter (**DM**; Method no. 934.01), ash (Method no. 967.05), crude protein (**CP**; Method no. 2001.11), ether extract (extraction with diethyl ether reagent, Method no. 920.39), crude fiber (**CF**; Method no. 978.10), and starch (amyloglucosidase-alpha-amylase, Method no. 996.11). Neutral detergent fiber (**NDF**) content was analyzed according to Mertens (2002), and minerals (Ca, P, K, and Na) were quantified by ICP-OES (Spectro Ciros Vision EOP) after microwave digestion (AOAC 2000, Method no. 999.10). Data were reported as g/kg as fed. GE and Metabolizable energy (**ME**) values were calculated on the basis of equations in the European Tables (Janssen, 1989).

Slaughter and Processing of Birds

In order to detect the onset of WS in the breast muscle (*Pectoralis major*) and to study its evolution, randomly selected chickens were weighed and sacrificed as described above at 12 and 25 d of age, and their whole breasts (*Pectoralis major* and *Pectoralis minor*) were dissected and weighed. *Pectoralis major* muscle was macroscopically observed to score as level 0 (no WS), level 1 (moderate WS), and level 2 (severe WS) according to Kuttappan et al. (2013a), whose ranking system is based on the size and distribution of white stripes on the fillet surface: level 0 when white stripes were visually absent; level 1 when white stripes were $\leq$1 mm thick, and level 2 when white stripes were $\geq$1 mm thick and covered a large surface of the fillet. Ultimate pH (**pHu**) was determined in the cranial and caudal portions of the fillet using a FG2-FiveGo portable pH-meter (Mettler Toledo, Greifensee, Switzerland).

At 51 d of age, the remaining birds were individually weighed, subjected to a 12 h total feed withdrawal, and processed in an authorized commercial slaughterhouse using electrical stunning. Carcasses (obtained by removing head, neck, shanks, and abdominal fat

Table 2. Ingredients and nutrient composition of experimental diets.

	Starter	Grower			Finisher	
	A1-B1	A2-B2	A3-B3	C3	C4	Finisher
Ingredients (g/kg as fed)						
Corn 7.7%	536	627	653			680
Corn 7.9%				682	705	
Soybean meal solv extr 47.5%	361	303	270			244
Soybean meal solv extr 47%				246	226	
Soybean/Sunflower oil	30.0	35.8	47.4			47.4
Soybean oil				36.0	35.0	
Gluten 56	30.0					
Calcium carbonate	14.3	11.2	10.4	10.1	10.2	9.9
Monocalcium phosphate 22.5/16	13.6	9.50	8.30	10.3	8.90	8.00
Sodium bicarbonate	1.10	1.40	0.70	0.70	0.70	0.80
Salt	2.10	1.90	2.40	2.40	2.40	2.30
DL-Methionine 99%	3.50	3.20	2.20	2.10	1.90	2.00
L-Lysine HCL 98%	2.80	2.00	0.70	2.10	1.80	0.90
L-Threonine 98%	0.60	0.50		0.60	0.40	
Vitamin[1] and mineral[2] premix	5.00	5.00	5.00	5.00	5.00	5.00
Xylanase				2.00	2.00	
Phytase-2500				0.30	0.30	
Monteban 100 premix[3] (A1 and A2 only)	0.0005	0.0005				
Elancoban 200 premix[4] (A3 only)			0.0005			
Analysed nutrient composition (g/kg)						
Dry matter (DM)	897	897	898	898	896	912
Ash	52.5	43.5	41.5	43.0	39.0	40.0
Organic matter[5] (OM)	844	854	856	855	857	872
Crude protein (N × 6.25) (CP)	240	197	179	172	164	168
Ether extract (EE)	48.5	54.0	69.0	58.0	56.0	70.0
Crude fibre (CF)	20.0	21.0	21.5	16.0	14.0	22.0
NDF	108	107	112	108	126	110
Starch	349	403	424	425	446	436
Non-nitrogenous extracts[6]	536	582	587	608	624	613
NNCC[7]	496	551	566	575	568	594
Ca	10.8	8.55	7.40	7.80	7.90	7.30
P	6.95	5.85	5.05	5.60	5.50	4.70
Ca/P	1.55	1.46	1.47	1.40	1.43	1.55
K	9.55	8.90	7.85	7.80	7.60	7.30
Na	1.40	1.30	1.35	1.40	1.30	1.40
GE (kcal/kg)[8]	4,581	4,582	4,649	4,560	4,551	4,639
ME (kcal/kg)[8]	3,055	3,156	3,258	3,130	3,148	3,283

[1]Provided the following per kilogram of diet: E672 vitamin A, 12,500 I.U.; E671 vitamin D3, 5,000 I.U.; 3a700 vitamin E, 30 mg (alfa – tocopheryl acetate); vitamin K, 5 mg; vitamin B1, 3 mg; vitamin B2, 6 mg; vitamin B6, 3 mg; vitamin B12, 0.03 mg; vitamin PP, 40 mg; pantothenic acid/calcium D - pantothenate, 20 mg; biotin, 0.2 mg; folic acid, 0.75 mg.

[2]Provided the following per kilogram of diet: Fe, 78 mg; I, 0.925 mg; Cu, 20 mg; Mn, 71.15 mg; Zn, 75 mg; Se, 0.27 mg (as E1 ferrous sulphate monohydrate, E2 potassium iodide, E4 cupric sulphate pentahydrate, E5 manganese oxide, E6 zinc sulphate monohydrate, E8 sodium selenite).

[3]Anticoccidial additive ingredients (%): Narasin sodium, 10; diluent (may include rice hulls, corn grits, or similar), 85 – 90; anti-dusting oil, 0–5. Elanco Animal Health division of Eli Lilly & Co., Indianapolis, IN.

[4]Anticoccidial additive ingredients (g/kg of additive): Monensin granulated, 930 (equiv. Monensin sodium: 200); antidusting oil, 20; rice hulls or limestone granular, 50. Elanco Animal Health division of Eli Lilly & Co., Indianapolis, IN.

[5]Organic matter content = DM – Ash.

[6]Non-nitrogenous extracts content = 100 – (Ash + CP + EE + CF).

[7]NNCC: non-nitrogenous cellular content = OM – (CP + NDF).

[8]Estimated according to Janssen (1989).

from bled, plucked, and eviscerated birds) were chilled, stored at 4°C, and transported to a commercial processing plant where the chilled carcasses and whole breasts were weighed to compute carcass and breast yields, pHu was measured on the cranial and caudal portions of the fillets, and fillets were subjected to visual WS scoring as above.

Statistical Analysis

A SAS (2004 -version 9.3) statistical software package was used. Individual live weight (**LW**) and ADG data were evaluated by using ANOVA and processed by choosing a mixed model that considered the pen as a random effect and treatment as a fixed effect

(PROC MIXED). Slaughter yields, breast yields, and pHu were also analyzed using the same mixed mode, with slaughter weight as covariate (COV). FI and FCR data, calculated at pen level, were processed through a one-way ANOVA with treatment as a fixed effect (PROC GLM). WS prevalence percentages were processed through ANCOVA analysis to detect possible treatment and live weight effects. WS scores were analyzed through the non-parametric Kruskal-Wallis test (PROC NPAR1WAY). Post hoc pairwise contrasts were evaluated by Bonferroni adjustments and three significance levels were assigned: *: $P < 0.05$; **: $P < 0.01$; and ***: $P < 0.001$. The minimum number of birds required for this study was defined as 136 animals per group, using the formula:

$$(1.96^2(\text{Patt}(1 - \text{Patt})))/\text{D}^2$$

(where P_{att} is the expected prevalence and D is the absolute precision) and assuming an estimated prevalence of about 80%, an absolute precision of 7%, and a confidence level of 95%. Considering an average mortality of around 5%, the decision to rear 180 chicks per group (a total number of 720) was made. The purpose of the first 2 slaughters (at 12 and 25 d of age) was to detect WS onset. For this reason, the necessary sample size was assessed as 18 animals per group when assuming a prevalence ≥15% and a confidence level of 95% (Cannon and Roe, 1982).

RESULTS AND DISCUSSION

Diet Formulation

Dietary ingredients and formulation are reported in Table 2. Diets administered to the 4 experimental groups during period 1 and period 2 (A1 and B1; A2 and B2, respectively) were characterized by a similar formulation. The formulation of diets A3, B3 (of period 3), and Finisher (of period 4) differed from that of diets C3 and C4 (of period 3 and 4, respectively), which were low-energy diets. Diets with different GE levels differed in corn and soybean with different protein content, oil source, and oil content.

Diet nutrient content is presented in Table 2. The differences in the ingredients of diets fed at period 3 and period 4 regarded nutrient content in terms of CP, EE, CF, and ME. As intended, the GE of diets A3 and B3 was higher than that of C3, whereas ME estimation provided a similar difference (3,258 vs 3,130 kcal/kg, respectively). Finisher diet differed from C4 in DM, organic matter, CP, EE, and CF, and therefore led to higher GE and estimated ME (3,283 vs 3,148 kcal/kg, respectively). Consequently, the differing ME content between standard and low-energy diets was expected to produce differences in bird growth rates.

Live Performance

At 12 d of age, CONTROL group broiler live weight was lower than that of animals in the other experimental groups ($P < 0.001$), whereas at 25 d of age, CONTROL and COX group birds were heavier than the vaccinated groups ($P < 0.05$; Table 3). At 42 d of age, CONTROL group chickens again had the lowest live weight, whereas COX and VACC-LE plus groups had the highest ($P < 0.001$). At final slaughter (51 d of age), COX and VACC-LE plus birds were still heavier (3,667 and 3,672 g, respectively) than CONTROL group birds (3,489 g; $P < 0.01$).

In period 1 (0 to 12 d of age), CONTROL group birds exhibited an unfavorable FCR (1.21) compared with those in the other experimental treatments (1.11, 1.13, 1.10 for COX, VACC, and VACC-LE plus, respectively; $P < 0.01$) due to their significantly ($P < 0.001$) lower ADG. During period 2 (12 to 25 d of age), both CONTROL and COX groups showed higher ADG than VACC and VACC-LE plus groups ($P < 0.001$), together with a favorable FCR ($P < 0.001$). In period 3 (25 to 42 d of age), although no difference in terms of FI emerged among groups, COX and VACC-LE plus birds had higher ($P < 0.05$) ADG (102 and 105 g/d, respectively) than those of CONTROL and VACC groups (80.3 and 95.2 g/d, respectively).

During the finishing period (42 to 51 d of age), CONTROL group chickens provided the best live performance, partially recovering the worst performance recorded in previous periods. In this fourth period, vaccinated groups had improved live performance and ranked in between the other 2 groups, whereas the ADG of the COX birds declined dramatically.

Overall, during the first 2 feeding periods, non-vaccinated groups showed higher ADG (49.4 and 50.7 g/d for CONTROL and COX, respectively) and favorable FCR (1.42 for both treatments) than VACC and VACC-LE plus groups (46.4 and 47.2 g/d; 1.47 and 1.48, respectively; $P < 0.001$). This evidence supports the depressive effect of anticoccidial vaccine on both FI and live performance in the early growing phase. Only VACC birds experienced FI depression however, whereas VACC-LE plus birds appeared to have been less affected (64.4 vs 65.6 g/d; $P < 0.05$). During the last 2 feeding periods (25 to 51 d of age), coccidiosis control systems proved capable of improving live performance, with the highest ADG and the most favorable FCR exhibited by VACC-LE plus birds.

Considering the entire rearing period (0 to 51 days), there were no differences for FI and FCR among groups, but higher ADG was observed in COX and VACC-LE plus groups (71.1 and 71.2 g/d, respectively) than in the CONTROL group (67.6 g/d; $P < 0.01$). Treatments did not affect animal health, and the average mortality recorded during the trial was 5% (Table 3).

Under comprehensive analysis, the data obtained suggest an overall ameliorative effect of coccidiosis control systems on live performance. The favorable effect

Table 3. Live performance.

	Treatments (T)					
Periods	CONTROL	COX	VACC	VACC – LE plus	SE	P
Live weight, LW (g)[1]						
12 d	370^{B}	409^{A}	402^{A}	417^{A}	5	***
25 d	$1277^{A,B,a}$	1308^{A}	1199^{C}	$1222^{B,C,b}$	11	***
42 d	2635^{C}	3033^{A}	2826^{B}	2992^{A}	26	***
51 d	3489^{b}	3667^{a}	$3593^{a,b}$	3672^{a}	36	**
Average daily gain, ADG (g/d)[1]						
0 - 12 d	27.4^{B}	30.6^{A}	30.0^{A}	31.2^{A}	0.4	***
12 - 25 d	69.5^{A}	69.2^{A}	61.4^{B}	62.0^{B}	0.7	***
25 - 42 d	80.3^{C}	$102^{A,B,a}$	$95.2^{B,b}$	105^{A}	1.3	***
42 - 51 d	$94.9^{A,a}$	66.7^{C}	$85.4^{A,B,b}$	$75.1^{B,C,c}$	2.1	***
0 - 25 d	49.4^{A}	50.7^{A}	46.4^{B}	47.2^{B}	0.4	***
25 -51 d	$85.1^{B,b}$	$90.2^{A,B,a}$	91.5^{A}	94.7^{A}	1.2	***
0–51 d	67.6^{b}	71.1^{a}	$69.6^{a,b}$	71.2^{a}	0.7	**
Feed intake, FI (g/d)						
0 - 12 d[2]	32.8	33.7	34.2	34.5	0.4	NS
12 - 25 d[2]	$100^{A,B}$	$103^{A,a}$	96.7^{B}	$98.5^{B,b}$	0.8	**
25 - 42 d[3]	204	212	207	203	4.8	NS
42 - 51 d[3]	196^{a}	168^{b}	$181^{a,b}$	$178^{a,b}$	6.0	*
0 - 25 d[2]	65.5^{a}	67.2^{a}	64.4^{b}	65.6^{a}	0.5	*
25 - 51 d[3]	202	200	199	195	4.1	NS
0 - 51 d[3]	127	126	126	124	2.0	NS
Feed conversion ratio, FCR						
0 - 12 d[2]	$1.21^{A,a}$	1.11^{B}	$1.13^{B,b}$	1.10^{B}	0.01	**
12 - 25 d[2]	1.44^{B}	1.48^{B}	1.55^{A}	1.58^{A}	0.01	***
25 - 42 d[3]	2.51^{A}	2.08^{B}	2.14^{B}	1.92^{B}	0.07	***
42 - 51 d[3]	$1.97^{B,c}$	$2.52^{A,a}$	$2.12^{A,B,b,c}$	$2.39^{A,B,a,b}$	0.09	**
0 - 25 d[2]	1.42^{B}	1.42^{B}	1.47^{A}	1.48^{A}	0.01	***
25 - 51 d[3]	2.37^{A}	$2.20^{A,B}$	$2.17^{A,B}$	2.06^{B}	0.05	***
0 - 51 d[3]	1.97	1.92	1.90	1.84	0.04	NS
Mortality (%)[4]						
0 - 12 d[5]	1.09	1.11	0.0	0.56	1.19	NS
12 - 25 d[5]	0.64	0.60	1.26	0.62	1.62	NS
25 - 42 d[6]	1.49	2.17	4.35	2.08	4.94	NS
42 - 51 d[6]	2.11	4.49	0.69	1.48	3.11	NS
0 - 25 d[5]	1.65	1.65	1.11	1.11	1.76	NS
25 - 51 d[6]	3.60	6.52	5.04	3.57	5.78	NS
0 - 51 d[6]	4.39	6.65	5.08	3.89	4.91	NS

[a–c]Means within the same row followed by different lowercase superscript letters differ $P \leq 0.05$ (*).

[A–C]Means within the same row followed by different uppercase superscript letters differ $P \leq 0.01$ (**); $P \leq 0.001$ (***).

[1]Degrees of freedom: 3 for treatments at numerator; 20 per treatment for least square means at denominator.

[2]Degrees of freedom: Model = 3; Error = 8; Total correct: 11.

[3]Degrees of freedom: Model = 3; Error = 20; Total correct: 23.

[4]Data processed through Kruskal-Wallis analysis.

[5]Degrees of freedom: Model = 3; Error = 8; Total correct: 11.

[6]Degrees of freedom: Model = 3; Error = 20; Total correct: 23.

the carboxylic ionophores (Narasin and Monensin) have on growth was confirmed. Indeed, during their period of use as additives, these polyether antibiotics ensured an intestinal integrity that contributed to improved ADG and FCR. During period 4, COX group underwent anticoccidial withdrawal and, as expected, exhibited lower ADG and FCR. Despite the depressive effect on feed consumption observed in VACC and VACC–LE plus birds during period 2 which also negatively affected ADG and FCR, the 2 coccidiosis control programs (VACC and COX) exhibited similar live performance when the rearing period was considered in its entirety.

These results support previous studies (Govoni et al., 1987; Shirley and Long, 1990 and Shirley, 1993; Williams and Gobbi, 2002) in which no statistically significant differences in terms of commercially important performance criteria were found between vaccinated birds and those given anticoccidial treatment. Chickens fed low energy diets (VACC-LE plus) during

Table 4. Slaughter yields, breast yields, and *Pectoralis major* muscle pH at 12, 25, and 51 d of age.

	Treatments (T)					Probability	
Attributes	CONTROL	COX	VACC	VACC – LE plus	SE	T	COV slaughter weight
Slaughter weight, SW (g)							
12 d[1,3]	353^{b}	405^{a,b}	393^{a,b}	423^{a}	12	*	-
25 d[1,3]	1335^{a}	1298^{a}	1157^{b}	1262^{a,b}	28	*	-
51d[2,4]	3489^{b}	3667^{a}	3593^{a,b}	3672^{a}	36	**	-
Whole breast weight (g)							
12 d[1,3]	58.8^{A}	56.2^{A}	55.2^{A,B}	51.2^{B}	1.0	**	***
25 d[1,3]	224^{B}	249^{A}	236^{A,B}	237^{A,B}	3.5	**	**
51 d[2,4]	811^{B}	837^{A}	840^{A}	845^{A}	4.8	***	***
Breast yield (% SW)							
12 d[1,3]	15.0^{a}	14.3^{a}	14.0^{a,b}	13.0^{b}	0.3	**	**
25 d[1,3]	17.8^{B}	19.6^{A}	18.6^{A,B}	18.7^{A,B}	0.3	**	*
51 d[2,4]	22.5^{B}	23.2^{A}	23.3^{A}	23.4^{A}	0.1	***	***
Cranial pHu							
12 d[1,3]	6.15	6.29	6.34	6.25	0.05	NS	NS
25 d[1,3]	6.16^{A,B,b}	6.15^{B}	6.14^{B}	6.29^{A,a}	0.03	*	NS
51 d[2,5]	5.96^{B}	6.06^{A,B}	6.14^{A}	6.07^{A,B}	0.02	**	NS
Caudal pHu							
12 d[1,3]	6.18	6.17	6.34	6.19	0.04	NS	NS
25 d[1,3]	6.16	6.20	6.14	6.22	0.03	NS	NS
51 d[2,5]	5.83^{b}	5.91^{a,b}	5.92^{a,b}	5.96^{a}	0.03	**	***

^{a,b}Means within the same row followed by different lowercase superscript letters differ $P \leq 0.05$ (*).

^{A,B}Means within the same row followed by different uppercase superscript letters differ $P \leq 0.01$ (**); $P \leq 0.001$ (***).

[1]20 animals per Treatment.

[2]133 (CONTROL), 132 (COX), 129 (VACC) and 134 (VACC-LE plus) animals.

[3]Degrees of freedom: 4 (treatment effect = 3, LW effect = 1) at numerator; 8 (treatment effect), 67 (LW effect) at denominator.

[4]Degrees of freedom: Model = 4 (treatment effect = 3, LW effect = 1); error = 523 (20 for treatment at denominator, 503 for LW effect at denominator); total correct = 527.

[5]Degrees of freedom: Model = 4 (treatment effect = 3, LW effect = 1); error = 205 (20 for treatment at denominator, 185 for LW effect at denominator); total correct = 209.

periods 3 and 4 did not fill the energy gap by higher FI. Nevertheless, birds in this experimental group expressed live weight, ADG, and FCR similar to those fed higher energy diets (COX and VACC). VACC-LE plus birds might have benefited from the dietary inclusion of enzymes (phytase and xylanase), more abundant lysine supplementation, and the inclusion of threonine in the last 2 rearing periods, thereby increasing the digestive utilization of nutrients and promoting protein synthesis. It is therefore likely that the ME of C3 and C4 was effectively higher than estimated.

Slaughter Yields, Breast Yields, and pHu Values

At first slaughter at 12 day of age, CONTROL group birds had higher breast yield than VACC-LE plus birds (15.0 vs 13.0% slaughter weight –**SW**-, respectively; $P < 0.01$) despite their lower SW (Table 4). At second slaughter at 25 day of age, COX group chickens showed significantly higher breast yield than CONTROL group birds (19.6 vs 17.8% SW, respectively; $P < 0.01$) due to the higher breast weight of the former (249 vs. 224 g; $P < 0.01$). Although showing lower SW than CONTROL and COX ($P < 0.05$) birds, VACC group birds had intermediate breast weight and breast yield (18.6% SW) compared to the other 3 treatments. At commercial slaughter, breast yield was higher in animals treated with the coccidiosis control program (average 23.4%) than those in the CONTROL group (22.5% SW; $P < 0.001$).

The values of cranial and caudal pHu (Table 4) measured on the breasts of birds slaughtered at 12 d of age were not affected by the experimental treatments. At 25 d of age, cranial pHu of VACC-LE plus birds was higher than in CONTROL birds (6.29 vs 6.16; $P < 0.05$), also COX and VACC birds (6.15 and 6.14, respectively; $P < 0.01$). At 51 d of age, statistical differences among groups emerged in both cranial and caudal regions. The pHu was significantly ($P < 0.01$) lower in breasts of CONTROL group birds than VACC birds measured at cranial level and those in VACC-LE plus group at caudal level.

The significance of the covariate indicates a strong SW effect on breast weight and breast yield (%SW),

Table 5. Slaughter and breast yields at 51 d of age.

Attributes	Treatments (T)					Probability	
	CONTROL	COX	VACC	VACC-LE plus	SE	T	COV slaughter weight
Carcass weight, CW (g)[1, 2]	2700^{C}	2770^{A}	2737^{B}	2753^{A,B}	6.7	***	***
Carcass yield (% SW)[1, 2]	75.0^{C}	77.0^{A}	76.0^{B}	76.5^{A,B}	0.2	***	NS
Breast yield (% CW)[1, 2]	30.0^{b}	30.2^{a,b}	30.6^{a}	30.6^{a}	0.2	*	***

^{a,b}Means within the same row followed by different lowercase superscript letters differ $P \leq 0.05$ (*).

$^{A–C}$Means within the same row followed by different uppercase superscript letters differ $P \leq 0.001$ (***).

[1]133 (CONTROL), 132 (COX), 129 (VACC) and 134 (VACC-LE plus) animals.

[2]Degrees of freedom: Model = 4 (treatment effect = 3, LW effect = 1); error = 523 (20 for treatment at denominator, 503 for LW effect at denominator); total correct = 527.

and also on breast pHu measured in the caudal regions of birds sacrificed at 51 d of age. Analogously, breast yield (% carcass weight - **CW**-) was also influenced by SW (P COV < 0.001) and was also strongly affected by the treatments (Table 5). As regards the latter, CONTROL chickens had the lowest carcass and breast yields (75.0 and 30.0%, respectively) at commercial slaughter age. The carcass yield was in favor of birds treated with coccidiosis control programs (77.0 and 76.5% for COX and VACC-LE plus, respectively), whereas breast yield referred to CW was higher in VACC and VACC-LE groups than in the CONTROL group ($P < 0.05$) (Table 5).

As observed by Kuttappan et al. (2013b) and Petracci et al. (2013a), it can be confirmed that heavier birds exhibit favorable breast yields. Animals treated with coccidiosis control programs showed better breast yields, even after covariate adjustment, thus suggesting a positive effect of the treatment regardless of SW.

The feeding plan (VACC vs VACC-LE plus) did not reveal any remarkable or statistically significant differences in the yields considered, indicating that C3 and C4 diets fulfilled energy and nutrient requirements, even if with lower GE content.

Lighter breasts were characterized by lower pH values; these findings are similar to those of previous studies by Petracci et al. (2013a,b), who observed lower pH values in standard yield hybrids compared to higher breast yield hybrids. Selection for increased breast muscle mass associated with hypertrophy can lower both glycolytic potential and activity by reducing glycolytic storage, thus resulting in heavier breasts with higher pHu (Berri et al., 2004; Le Bihan-Duval et al., 2008).

White Striping Prevalence

Breast lesions attributable to WS were not observed in chickens at 12 d of age in any of the 4 experimental groups. Lesions were detected at 25 d of age in all 4 experimental groups with a prevalence ranging from 5.4% (CONTROL and COX) to 29.4% (VACC) but the difference was not statistically significant (Table 6). When considering WS mean scores (Table 7) of the 4 treatments obtained at 12 and 25 d of age, the previous results were confirmed. At 51 d of age, the total WS prevalence was on average very high (95.1%) but showed no difference among treatments.

As reported above, WS prevalence was macroscopically scored as level 0, level 1, and level 2. At 25 d of age, only level 1 was detected, with no differences among treatments, whereas at 51 d of age, WS severity was distributed according to 4% to level 0 (treatment effect: NS), 29.4% to level 1 (treatment effect: $P < 0.05$), and 66.6% to level 2 (treatment effect: $P < 0.01$) (Table 6). At commercial slaughter age, the prevalence of moderate WS (level 1) was higher in the breasts of CONTROL and VACC than COX group birds (36.8 and 33.5% vs 19.1%; $P < 0.05$). On the contrary, the prevalence of severe WS (level 2) was higher in COX than CONTROL and VACC groups (77.6 vs 62.6 and 61.6%, respectively; $P < 0.01$), and also higher than in VACC-LE plus birds (64.4%; $P < 0.05$).

The significance of the covariate highlights a strong SW effect on WS prevalence at 51 d of age, but not earlier. Despite being covariated with SW, WS prevalence was influenced by the treatments at the commercial slaughter age (Table 6).

The mean WS scores in Table 7 highlight the effect of treatment at 51 d of age, showing a greater lesion severity for COX than CONTROL and VACC groups (1.79 vs 1.50 and 1.58, respectively; $P < 0.001$), whereas group VACC-LE plus birds exhibited an intermediate degree of severity (1.63).

The results obtained in this study differ from those in literature. In our study, total WS prevalence was on average very high (95.1%), and with a higher contribution from WS level 2 (66.6%) than WS level 1 (29.4%). Total WS prevalence was much higher than that observed by Lorenzi et al. (2014) on broilers in similar weight range (3.0 to-3.8 kg): with 45.2% at level 1 and 7.5% at level 2.

Kuttappan et al. (2012a), Petracci et al. (2013a), Lorenzi et al. (2014) found that higher breast yield genotypes are more prone to develop WS lesions. Moreover, heavier fillets tend to exhibit a greater degree of severity. The significance of the covariate can confirm this relationship. Genetic selection for more favorable commercial traits has implications for both product quality and animal welfare, given that skeletal muscle development and metabolism are both altered and this leads to an elevated incidence of spontaneous and

Table 6. White Striping prevalence observed on *Pectoralis major* muscle at 12, 25, and 51 d of age.

	Treatments (T)					Probability	
Prevalence	CONTROL	COX	VACC	VACC – LE plus	SE	T	COV slaughter weight
Total prevalence							
12 d[1,3]	0	0	0	0	-	-	-
25 d[1,3]	5.4	5.4	29.4	5.8	8.2	NS	NS
51d[2,4]	98.4	96.7	96.1	92.7	1.6	NS	*
Level 0 prevalence (absence)							
12 d[1,3]	100	100	100	100	-	-	-
25 d[1,3]	94.6	94.6	70.6	94.2	8.2	NS	NS
51d[2,4]	1.6	3.3	3.9	7.3	1.6	NS	*
Level 1 prevalence							
12 d[1,3]	0	0	0	0	-	-	-
25 d[1,3]	5.4	5.4	29.4	5.8	8.2	NS	NS
51d[2,4]	36.8^{a}	19.1^{b}	33.5^{a}	$28.3^{a,b}$	3.4	*	*
Level 2 prevalence							
12 d[1,3]	0	0	0	0	-	-	-
25 d[1,3]	0	0	0	0	-	-	-
51 d[2,4]	61.6^{B}	$77.6^{A,a}$	62.6^{B}	$64.4^{A,B,b}$	2.8	**	***

[a,b]Means within the same row followed by different lowercase superscript letters differ $P \leq 0.05$ (*).

[A,B]Means within the same row followed by different uppercase superscript letters differ $P \leq 0.01$ (**); $P \leq 0.001$ (***).

[1]20 animals per Treatment.

[2]133 (CONTROL), 132 (COX), 129 (VACC) and 134 (VACC-LE plus) animals.

[3]Degrees of freedom: Model = 4 (Treatment effect = 3, LW effect = 1); Error = 7; Total correct: 11.

[4]Degrees of freedom: Model = 4 (Treatment effect = 3, LW effect = 1); Error = 19; Total correct: 23.

Table 7. Non-parametric Kruskal - Wallis test on the mean White Striping scores on *Pectoralis major* muscle at 12, 25, and 51 d of age.

	Treatments (T)				
Mean score[1]	CONTROL	COX	VACC	VACC-LE plus	P
12 d[2]	0	0	0	0	NS
25 d[2]	0.15 ± 0.37	0.10 ± 0.31	0.15 ± 0.37	0.05 ± 0.22	NS
51d[3]	1.50 ± 0.57^{B}	1.79 ± 0.46^{A}	1.58 ± 0.57^{B}	$1.63 \pm 0.60^{A,B}$	***

[a,b]Means within the same row followed by different lowercase superscript letters differ $P \leq 0.05$ (*).

[A,B]Means within the same row followed by different uppercase superscript letters differ $P \leq 0.001$ (***).

[1]Degrees of freedom: 3.

[2]20 animals per Treatment.

[3]133 (CONTROL), 132 (COX), 129 (VACC) and 134 (VACC-LE plus) animals.

stress-induced myopathies. Higher muscle hypertrophy may not be supported by adequate capillary density and may lead to diminished oxygen and nutrient supply to the muscle and to lower catabolite removal, with undesirable consequences on meat quality, especially when animals are exposed to metabolic loads (Hoving-Bolink et al., 2000).

Regardless of SW or breast weight, 51-day-old COX group birds showed higher level 2 WS prevalence than VACC groups, assuming the use of ionophores against coccidiosis as a predisposing WS factor. This could be partially due to the protective effect of anticoccidials on intestinal integrity that leads birds to exhibit higher live performance in the first growing period but mainly to their proven toxicity on muscle fiber cells (Novilla, 1992; Chapman et al., 2010; Markiewicz et al., 2014).

Moreover, Sandercock and Mitchell (2003) suggested that treatment with monensin could increase Na^{+} and Ca^{2+} intracellular concentration, resulting in increased Ca^{2+} accumulation (from outside and due to altered compartmentalization) and concomitant high CK loss. This is signal of tissue damage associated with degenerative myopathy, and its concentration, together with other enzymes, and is elevated in severe WS conditions. WS seems unrelated to systemic infectious or inflammatory conditions because birds with normal and seriously affected breasts did not show any difference in hematological parameters (Kuttappan et al., 2013b).

The fact that VACC birds had the same WS prevalence level 2 and mean WS scores as CONTROL birds proves the absence of any vaccination effect on WS appearance. VACC-LE plus diets (C3 and C4)

were tested with the intention of slowing down bird growth for the purpose of reducing the WS prevalence caused by rapid growth, as was recently observed by Kuttappan et al. (2012a), who also fed birds with high and low energy diets. As the chickens in our study utilized nutrients with the diets C3 and C4 more efficiently, due to higher amino acids and enzymes supplementation, final slaughter weight was comparable to that of the VACC group. Unfortunately, the likely higher nutrient bioavailability at muscle tissue level was not able to restrain WS prevalence.

In conclusion, this study has demonstrated that both of the coccidiosis control programs adopted were capable of increasing growth and carcass yields over those of untreated chickens. It has also shown that the early (25 d of age) macroscopic appearance of WS was not affected by the experimental treatments. At commercial slaughter age, total average WS prevalence was 96%, with a level 2 prevalence that was significantly higher for COX birds, even though SW had been included as covariate. This study suggests that vaccination against coccidiosis has no effect on WS occurrence.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the financial support from MSD Animal Health. Authors are grateful to Gabriele Baldan, Francesca Pengo, Roberto Lazzaretti and Barbara Contiero, for their technical support.

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