

HOW DOES “WOODEN BREAST” MYODEGENERATION AFFECT POULTRY MEAT QUALITY?

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Abstract – The aim of the research was to study the effects of “Wooden Breast” (WB) myodegeneration on poultry meat quality. At a poultry meat cutting facility, 30 normal (N) and 30 WB (WB) fillets (*Pectoralis major* muscle) were selected to measure the weight, cross sectional area, pH and L*a*b* colour values at 48h *post mortem*, and pH, L*a*b* colour values, water-holding capacity and Warner-Bratzler shear force after 3 months of storage at -20 °C. Data on WB condition showed a negative effect on all of the variables, measured at the cranial end of fillets, where lesions were more visible.

I. INTRODUCTION

A great demand of poultry meat has put pressure on breeders, nutritionists and farmers to improve the growth rate, feed efficiency, and breast meat yield of the birds (1). However, challenging birds to reach high body weight within a short period of time has led to a dramatic increase of breast muscle defects within the last few years. Two of such have been described and named as the “White Striping” (WS, Kuttappan *et al.*; 2) and the “Wooden Breast” (WB, Sihvo *et al.*; 3). WS defect that has been rather intensively studied recently refers to macroscopic white striations running parallel to the direction of muscle fibres (2, 4, 5, 6, 7) and may show a tendency of separation of fibre bundles beneath the striated area (8, 9). Microscopically, WS-affected fillets exhibit chronic lesions characterized by fibrosis and lipodosis (5, 6, 7). WS has been suggested to be linked to increased growth rate, higher breast weight and yield (1, 2, 5, 6, 10, 11), accompanied by higher age at slaughter (1, 12) rather than gender and feeding regimen (7), dietary vitamin E level (7, 11), as well as genetics (10, 12). WS defect results in lowered technological quality of the fillets by increasing drip and cooking losses

(1), toughness (13), yellowness of colour (b* value (7), and pH (1, 9). Furthermore, WS defect significantly reduces consumer acceptance because of the fatty, marbled and abnormal appearance of the fillets (4), and that forces poultry processors to downgrade the affected fillets to processed products, which has economic consequences for the poultry industry (5).

While the aetiology and quality implications of WS have been investigated, little is yet known about the incidence of WB, its mechanisms of formation and/or the consequences on quality. Apart from the macroscopic and histopathological characteristics described by Sihvo *et al.* (3), the scientific community is investigating the causes of WB. Similarly to WS, the greatest suspicions seem to fall on the fast growth rate as well as the ever-increasing breast meat yield. As characterized by Sihvo *et al.* (3), an affected *Pectoralis major* muscle exhibits remarkable palpatory hardness that is diffuse or focally-extended. Furthermore, the hardened areas of a WB fillet are consistently out-bulging and pale, and the surface is often covered with clear or slightly turbid viscous fluid and/or petechiae or multifocally distributed small hemorrhages; also white striping is regularly seen. The aim of the study was to gain information on the potential effects of WB on the quality of chicken breast meat, and thus, help the industry to alleviate the financial burden caused by the condition.

II. MATERIALS AND METHODS

Broiler carcasses were deboned and breast muscles were collected forty-eight hours *post mortem* at a poultry cut-up plant. Altogether 60 breasts were selected, of which 30 were considered normal (N) and another 30 severely affected by the “Wooden Breast” (WB) condition.

The WB selection criteria included simultaneous presence of bulged as well as hard areas at palpation. The N vs WB condition was assessed by two experienced team members.

The right *Pectoralis major* muscle was weighed and subjected to analyses. pH was measured on cranial and caudal ends of the muscle with Mettler Toledo FE20, and the colour values of lightness, redness, yellowness, chroma and hue (L*a*b*, C* and H°, respectively) were measured with RM200QC colorimeter (X-Rite, Co, Neu-Isenburg, Germany). The muscle was cut transversely at the level of the thicker cranial portion, and muscle cross-sectional view was photographed and processed with digital image analysis software (Carl Zeiss, Model Axiovision 4.6.3.0) for muscle cross-sectional area (CSA). Breast samples were individually vacuum-packed, and kept frozen for 3 months at -20 °C. After thawing the samples were reweighed and thawing losses were calculated. Also pH and colour values were recorded again. For determination of cooking losses, fillets were again vacuum-packed and cooked in a water bath until core temperature of 74 °C.

Shear force (WBSF) measurements were performed on cylindrical samples (Ø1.25 cm) cut perpendicularly to the muscle fibre direction with a Warner-Bratzler cell (100-kg load cell, 2 mm/s crosshead speed) fitted on a TA-HDi Texture Analyzer (Stable Macro System, London, UK). WBSF values of the samples represent an average of 6 measurements.

Data were analysed using SAS 9.1 statistical analysis software for Windows (SAS, 2008) by considering the breast condition (N / WB) as an independent variable.

III. RESULTS AND DISCUSSION

The WB fillets differed significantly from the normal in breast weight, CSA, pH, colour values, and cooking loss.

WB fillets had a higher pH value at both 24 hours *post mortem* as well as after frozen storage, the difference being significant (P<0.01) however only for the cranial end (Tables 1 and 2). Previous studies on WS have showed a similar overall pH trend (1, 9).

Table 1. Breast (*Pectoralis major*) pH and L*a*b* colour values at 48 h *p.m.*

	N	WB	Significance	RSD ⁽¹⁾
pH Cranial	5.90	6.03	**	0.19
pH Caudal	5.87	5.92	ns	0.16
L* Cranial	50.9	54.6	***	3.4
L* Caudal	50.5	53.8	***	3.3
a* Cranial	-1.1	-0.3	**	1.4
a* Caudal	-1.2	-0.6	ns	1.4
b* Cranial	12.9	15.9	**	3.1
b* Caudal	12.4	13.3	ns	3.6
C* Cranial	13.0	16.0	**	3.2
C* Caudal	12.6	13.4	ns	3.6
H° Cranial	95.4	90.5	**	5.2
H° Caudal	96.8	94.3	ns	7.3

*, P<0.05; **, P<0.01; ***, P<0.001; ⁽¹⁾ Residual Standard Deviation

Table 2. Breast (*Pectoralis major*) pH and L*a*b* colour values after frozen storage.

	N	WB	Significance	RSD ⁽¹⁾
pH Cranial	5.91	6.03	***	0.12
pH Caudal	5.86	5.91	ns	0.13
L* Cranial	48.5	52.3	***	3.0
L* Caudal	48.9	51.3	***	2.4
a* Cranial	-0.4	-0.7	**	1.3
a* Caudal	-1.2	-1.2	ns	1.3
b* Cranial	16.7	19.8	**	3.7
b* Caudal	13.9	15.2	ns	3.7
C* Cranial	16.7	19.9	**	3.7
C* Caudal	14.1	15.4	ns	3.6
H° Cranial	92.1	88.8	**	4.6
H° Caudal	96.0	96.0	ns	6.23

*, P<0.05; **, P<0.01; ***, P<0.001; ⁽¹⁾ Residual Standard Deviation

WB samples were lighter in colour than the normal; an effect not observed with respect to WS (Kuttappan *et al.* (7); Petracci *et al.* (9)). The higher L* values were recorded both cranially and caudally, highlighting the significance of the characteristic in the WB condition.

Similarly to L*, also higher a* and b* values were recorded in WB yet the difference was significant only for the cranial end. The increased lightness and yellowness of the WB samples call for further investigation as those are not likely to be attributable to lipidosis that has been found to accompany the defect of WS [7]. Perhaps the colour changes within the WB fillets are more related to the marked fibrotic response. Also chroma (C*) and hue (H°) revealed the similar trend seen for redness (a*) value (Tables 1 and 2).

Comparable to WS (4, 5, 6, 7), also WB had a negative effect on water holding capacity (WHC) (Table 3). Total losses (thawing + cooking losses) were significantly higher in WB than N (31.4 vs 29.0%; $P < 0.05$), due to higher cooking losses (26.4 vs 23.5%; $P < 0.01$; Table 3).

Table 3. Breast (right *Pectoralis major*) weight, WHC, WBSF and cross sectional area (CSA)

	N	WB	Significance	RSD ⁽¹⁾
Weight, g	377	505	***	49.7
Thawing losses, %	5.4	5.0	ns	1.7
Cooking losses, %	23.5	26.4	**	3.7
Total losses, %	29.0	31.4	*	4.4
WBSF, N	15.5	16.9	ns	2.8
CSA, cm ²	25.1	30.3	***	4.5

*, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$; ⁽¹⁾ Residual Standard Deviation

The lower WHC of WB during storage and cooking may be caused by the degeneration of fibres since that is likely to result in marked reduction of myofibrillar proteins due to the replacement of myosin and actin, the proteins responsible for WHC, with connective tissue (1, 3). It was anticipated that the palpatory hardness of the fresh fillet would manifest itself as significant differences in the measurements of WBSF. However, the shear force of cooked meat was not significantly influenced by WB. With respect to the WS defect, the lower shear values in comparison to normal have been explained by extensive poor cohesion and the tendency of the fibre bundles to get separated (8; 9); phenomena that were also observed cranially in most of the WB muscles of our study. Nevertheless, even the cranial palpatory hardness still present after thawing was not reflected into the WBSF values. WB fillets were also significantly heavier (505 vs 377 g; $P < 0.001$) and had a higher CSA (30.3 vs 25.1 cm²; $P < 0.001$) than the normal. Similar findings have been reported also with respect to the WS condition (1, 2, 7, 12). Breasts of greater width and thickness have been associated to increased fibre diameter and length (14) as well as to a combination of hypertrophy and increased muscle fibre number (15). The histologic evaluation of Petracci *et al.* (1) on WS fillets from carcasses of relatively great weight revealed that large muscle fibre cross-sectional area had a

significant association with a greater incidence of abnormal fibres (with a so-called “internalization” of nuclei). The breast samples of our experiment are also currently under histological evaluation.

IV. CONCLUSION

This study showed that WB has, in general, negative effects on meat quality, resulting in colour that is lighter, less red and more yellow than normal, as well as in greater weight losses. Affected fillets are also characterized by higher weight and larger cross-sectional area, which suggests that birds' size is an issue. The triggers and mechanisms of the pathological course of events remain, however, yet to be established.

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