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Pathological Changes in the Microstructure of Pale, Soft, Exudative (PSE) and Normal Turkey Breast Muscle

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The aim of the study was to determine the type and extent of histopathological changes in PSE and normal breast muscle of turkey. Changes in fibre size (atrophy, hypertrophy – giant fibres), changes in fibre shape (angular fibres), degenerative lesions (necrosis with phagocytosis) and connective tissue hypertrophy were evaluated. The present study showed that significantly fewer normal fibres were found in the muscles of PSE group compared to the normal group. Moreover, the histopathological changes observed in all birds from the PSE and normal groups included muscle fibre atrophy and change in fibre shape (angular fibres), with both the percentage of female turkeys showing this type of pathology and the frequency of atrophic and angular fibres being significantly higher in birds from the PSE group. Also in the case of giant fibres, the muscles of PSE group were characterized by a greater number of animals with giant fibres and a significantly higher proportion of giant fibres compared to normal group. In the case of connective tissue hypertrophy and necrosis with phagocytosis, the changes observed in this study were not extensive in either groups of birds. In summary, the PSE syndrome contributes to a significant increase in the frequency of histopathological changes in muscle tissue, and this may directly translate into meat quality.

Key words: PSE, histopathological changes, breast muscle, turkey.

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Improvement of meatiness is a major task for breeders. Today it is known that meat quality is an outcome of many factors associated with animal nutrition, rearing and slaughter, as well as depending on breeding stock (genotype) (KŁOSOWSKA *et al*. 1998, 2004; ELMINOWSKA *et al.* 2004; MŁYNEK *et al.* 2006). On the other hand, selection of turkeys for increased growth rate and breast muscle size causes undesirable metabolic and physiological disorders in the muscle tissue, which together with other stress factors lead to the occurrence of many meat quality defects (KŁOSOWSKA *et al*. 2000; ELMINOWSKA *et al.* 2005; ODA *et al*. 2009; STRASBURG & CHIANG 2009). The most frequent muscle tissue defects include atrophy (caused by injury to muscle innervation or muscle inactivation) and necrosis. Another pathology is muscle fibre hypertrophy and giant fibre development, as well as connective tissue hypertrophy. A considerable proportion of pathology is made up by changes in fibre shape (angular fibres). Research to date shows that the type and extent of histopathological changes may have a genetic background (WIMMERS *et al.* 2006), and depend on the animal's breed (KŁOSOWSKA *et al.* 1995; WALASIK *et al*. 2000; FIEDLER *et al.* 2001; BOGUCKA *et al.* 2006; SCHUBERT-SCHOPPMEYER *et al*. 2008; SOBCZAK *et al.* 2009), age or body weight (WOJTYSIAK *et al*. 2012). Much evidence also shows that histopathological changes in muscle tissue may be related to the susceptibility of animals to stressors and the incidence of PSE (SOŚNICKI 1987; KŁOSOWSKA 1995; FIEDLER *et al.* 1999). In the poultry industry PSE meat (pale, soft, exudative), characterized by light colour, soft texture and reduced water binding and holding capacity (OWENS *et al.* 2000; LI *et al.* 2015), is a major concern as it causes substantial economic losses (OWENS *et al.* 2009), reduces performance, and adversely affects the quality of meat raw material (CARVALHO *et al.* 2014).

Therefore, an understanding of muscle tissue microstructure, in particular the identification of the type and extent of pathological changes, can be an important factor in making a proper and objective evaluation of meat quality or a way of verifying the existing evaluation methods.

The aim of the study was to determine the type and extent of histopathological changes in normal and PSE breast muscle of female turkeys.

Material and Methods

Among 258 female turkey of the BUT-9 line (from one flock), 30 normal and 24 PSE boneless, skinless whole turkey breast meats were collected from the deboning lines of a local commercial processing plant. The birds were 16 weeks old and live weight was 9.5 kg. Breast muscles were classified into normal and PSE based on L* colour parameter, measured with a colorimeter on the cranial, medial surface (bone side) in an area free of obvious colour defects on a total of 258 breast meats on the deboning line (30 minutes *postmortem*), following the method of OWENS *et al.* (2000). To select the samples, a portable pH meter (Matthäus, Germany) with a glass electrode standardised for pH 4.0 and 7.0 according to Polish Standard PN-77/a-82058 with automatic correction for muscle temperature was used to calculate pH at 30 minutes *postmortem*. This served as a basis for classifying breast muscles into normal ($46<L^*<53$; $5.9 \leq pH < 6.2$) and PSE ($L^*>53$; $pH<5.9$).

For histological examination muscle samples were taken 30 minutes *post mortem* from the right carcass side deep within the breast meat. Tissue samples were collected from breast muscles prior to excision from the carcass. Thus, breast muscles were cut along muscle fibres, pinned to a cork plate and then the muscle fragment was cut across muscle fibres. The prepared muscle section was frozen in isopentane that was cooled using liquid nitrogen and stored at -80°C until analysis. Serial transverse sections of 10 μm were cut at -20°C in a cryostat (Slee MEV, Germany) and stained by hematoxylin and eosin. The histopathological changes were assessed by analysing 10 pictures of muscle samples of 1000 μm^2 area each using a Nikon E600 (Japan) light microscope. According to DUBOWITZ & SEWERY (2007), the following histopathological changes were examined: changes in fibre size (atrophy, hypertrophy – giant fibres) and shape (angular fibres), degenerations (necrosis with phagocytosis) and overgrowth of connective tissue (fibrosis). A minimum of 300 muscle fibres per sample were counted except connective tissue, which was estimated subjectively according to the following scale: '0' – no change, '+' – minor change and '++' – major change, as described previously by WALASIK *et al*. (2000).

Statistical analysis software (SAS Institute, Cary, NC, USA) was used to perform analysis of variance (General Linear Models procedure). The model included the turkey group as the main effect. The results are presented as least squares means (LSM) ± standard error (SE). Differences were considered to be significant at $P<0.05$.

Results and Discussion

The percentages of birds from the PSE and normal groups that showed histopathological changes of varying extent are presented in Tables 1 and 2.

The histological analysis of breast muscles from female turkeys showed that PSE muscles had a significantly lower proportion of structurally normal fibres compared to the normal group (Table 1).

Table 1

Percentage of turkey showing histopathological changes in PSE and normal breast muscle

Traits	Normal	PSE
Giant fibres (%)	46.7	100
Angular fibres (%)	100	100
Atrophic fibres (%)	100	100
Necrosis with phagocytosis (%)	26.7	37.5
Fibrosis (%)	13.3	16.7

Table 2

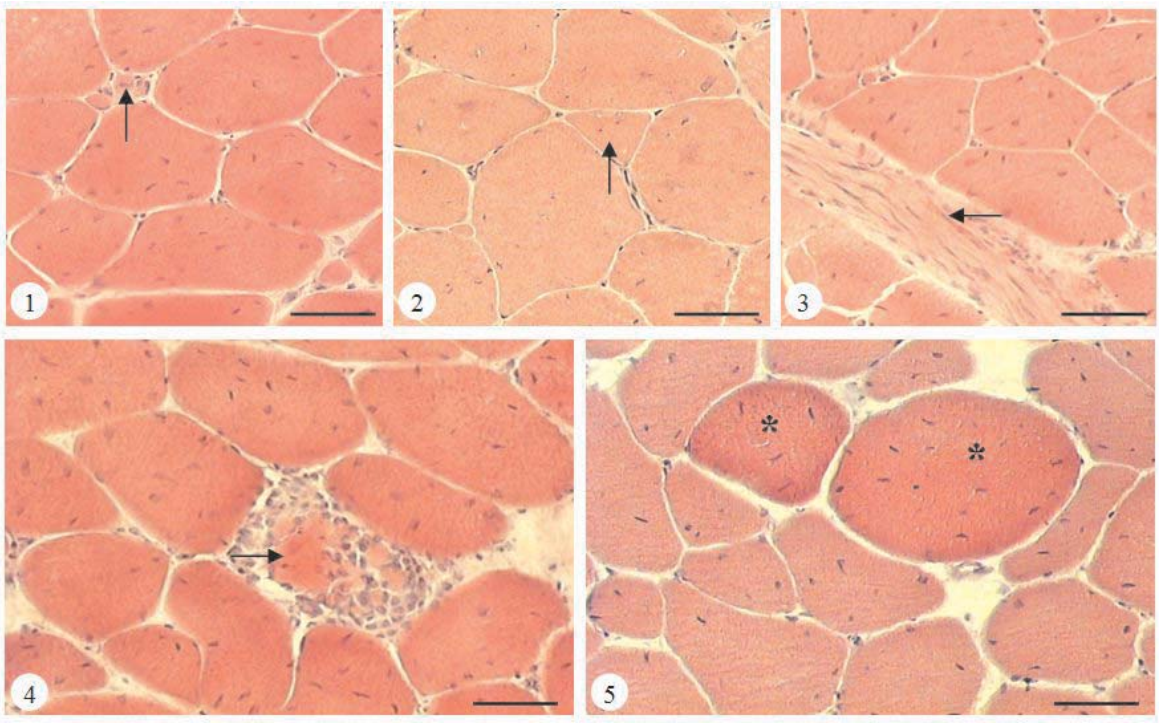
Least squares means and standard errors (SE) for incidence of histopathological changes in PSE and normal breast muscle

Traits	Normal	PSE
Normal fibres (%)	97.32 ± 1.84^a	93.21 ± 1.63^b
Giant fibres (%)	0.63 ± 0.09^a	2.68 ± 0.34^b
Angular fibres (%)	0.48 ± 0.12^a	0.92 ± 0.13^b
Atrophic fibres (%)	1.19 ± 0.27^a	2.74 ± 0.32^b
Necrosis with phagocytosis (%)	0.38 ± 0.06^a	0.45 ± 0.08^a
Fibrosis (%)	+	+

a, b, c – LSMs marked with different superscripts differ significantly at $P<0.05$.

Among the pathological changes of muscle fibres, the microscopic observations showed the presence of changes in muscle fibre size (atrophic and giant fibres), changes in fibre shape (triangular, elongated, trapezoid), degenerative lesions (necrosis with phagocytosis) and overgrowth of connective tissue (Figs 1-5).

The results suggest that the PSE syndrome has a significant effect on both the type and extent of pathological changes in turkey breast muscle. The current study demonstrated for the first time that the most extensive histopathological changes in breast muscles from turkey were muscle fibre atrophy and change in fibre shape (Figs 1 and 2). Muscle fibre atrophy and changes in fibre shape were observed in the present study in all birds from both PSE and normal groups, but a significantly higher frequency of atrophic and angular fibres was found in the muscles of PSE group turkeys. These results are consistent with an earlier study on pigs (WALASIK *et al*. 2000; BOGUCKA *et al*. 2006; WOJTYSIAK *et al.* 2012), which showed that muscle fibre atrophy and change in fibre shape are the most frequent pathological changes in skeletal muscles. Muscle fibre atrophy involves a gradual decrease in fibre volume, which may result from undernutrition, inactivation, aging or wasting of the body, and also injury to muscle innervation. Because the analysed animals in the current study were of the same age and were kept under the same conditions, it appears that the observed muscle fibre atrophy was due to atrophy caused by injury to muscle innervation. According to PACIELLO & PAPPARELLA (2009) and THUILLIEZ *et al.* (2009), both myofibrils and sarcoplasm disappear in the atretic process induced by loss of muscle fibre innervation. The fibres gradually decrease their volume, they change in cross-sectional shape from circular to polygonal, and their nuclei are positioned closer to one another. Another type of histopathological change observed in the present study in turkey breast muscles were degenerative lesions leading to breakdown of muscle fibres, namely necrosis with phagocytosis (Fig. 4) as well as overgrowth of connective tissue (Fig. 3). Earlier studies suggested that necrotic fibres are among the most common abnormalities in broiler chickens (SOIKE & BERGMANN 1998; MACRAE *et al*. 2006; MAZZONI *et a*l. 2015) and turkey (SOŚNICKI *et al*. 1991). Nevertheless, in the present study, these changes were less intense for both necrosis with phagocytosis and overgrowth of connective tissue. It is worth noting that in both cases the PSE syndrome had no significant effect on the extent of these pathological changes. Generally, the necrotic fibres appeared infiltrated by histiocytes, macrophages and lymphocytes with eventual lysis



Figs 1-5. Exemplary cross-section of turkey breast muscle stained for hematoxylin-eosin: 1 – atrophy (arrow); 2 – angular fibres (arrow); 3 – connective tissue hypertrophy (arrow); 4 – fibre necrosis with phagocytosis (arrow); 5 – giant fibres (*). Bar = 50 μm.

and phagocytosis of cell debris (HAUSMANOWA-PETRUSEWICZ 1993). Characteristically, in this type of fibre the protoplasm also contains disintegrated residual fibres. SOŚNICKI *et al.* (1991), in ultrastructural studies of the pathology of muscles in rapidly growing turkeys, showed that fibres affected by degenerative lesions (necrosis with phagocytosis) were characterized by dilatation of the sarcoplasmic reticulum, intense Z-line streaming, and the presence of several myeloid and lysosomal dark-bodies in the necrotic areas.

One of the major pathological changes of muscle tissue, which directly translates into meat quality, is muscle fibre hypertrophy, or giant fibres (Fig. 5). Many earlier studies have shown that frequency and size of giant fibres are significantly related to acidity and texture parameters of meat (FAZARINC *et al*. 2002; SCHUBERT-SCHOPPMEYER *et al*. 2008; SOBCZAK *et al.* 2009). Fibres of this type have been observed on histologically stained muscle sections of different animal species such as cattle (SINK *et al.* 1986), pig (FAZARINC *et al*. 2002; SCHUBERT-SCHOPPMEYER *et al.* 2008; WOJTYSIAK 2012; WOJTYSIAK *et al.* 2012), rabbit (DALLE ZOTTE *et al*. 2010), sheep (DEMIRTAS 2016), chicken (MIRAGLIA *et al*. 2006; BRANCIARI *et al*. 2014) and turkey (REMIGNON *et al*. 2000).

In the literature the giant fibres generally represent less than 2% of the total muscle fibres. However, in the present study the giant fibres tended to be numerous especially in the muscles from PSE group. Importantly, the giant fibres occurred in all birds from the PSE group. Similar results were obtained for the frequency of giant fibres, the percentage of which was significantly higher in the muscles of PSE turkeys compared to the normal group. Moreover, in the present study, microscopy revealed that the giant fibres are round or oval in shape, and have a homogeneous cytoplasm. Typically giant fibres are often separated from the other muscle fibres that form muscle bundles (BOGUCKA *et al.* 2006; WOJTYSIAK 2012). The fibres of this type also have a specific location. In the present study, the giant fibres, from both PSE and normal groups, were generally distributed separately, sometimes in groups near the periphery of the muscle bundles, but they were not observed in every muscle bundle. Similarly to previous research by KULIŠEK *et al.* (2009), the presence of giant fibres was also found within the muscle bundles. The size of giant fibres largely depends on the size of normal muscle fibres. FAZARINC *et al.* (2002) noted that giant fibres can be three times as large as normal myofibres. The etiology of giant fibre formation has not been conclusively established. Among the many hypotheses that explain this process, most authors believe that giant fibres are formed as a result of nervous stimulation. KŁOSOWSKA *et al*. (1995) found that giant fibres may be the initial stage of hyaline degeneration, which begins with fibre contracture. SCHUBERT-SCHOPPMEYER *et al.* (2008) suggested that giant fibres arise from hypercontraction of individual fibres with signs of structural disintegration. In turn, SINK *et al*. (1986) reported that giant fibre formation could be related to environmental, genetic and metabolic factors. FAZARINC *et al*. (2002) in porcine *longissimus* muscle and BRANCIARI *et al.* (2014) in chicken *pectoralis major* muscle showed that the occurrence of giant fibres is correlated with the pH value and the glycogen content. In turn, REMIGNON *et al*. (2000) suggest that giant fibres occur independently of the birds' genetic background and muscle type, but depend on the biochemical processes taking place during *rigor mortis*. Furthermore, it is worth noting that most of the research to date suggests that giant fibres are the only pathological change in muscle tissue to occur only *post mortem* and never *in vivo* (FIEDLER *et al*. 1999; SCHUBERT-SCHOPPMEYER *et al*. 2008). On the other hand, a recent study by BRANCIARI *et al.* (2014) suggested that giant fibres in chicken muscle may develop in *pre rigor* muscles independently of muscle type and/or genetic line. Likewise, DEMIRTAS (2016) demonstrated that giant fibres may appear in sheep muscles before *rigor mortis.*

The main reason for the increased proportion of giant fibres in the muscle structure may be the susceptibility of animals to stress as well as the PSE syndrome. These suppositions, observed also in the present study, were confirmed by a previous study on pigs (KŁOSOWSKA & FIEDLER 2003), which showed that the frequency of giant fibres is determined by the presence of PSE defects. The fact that the presence or absence of giant fibres in muscles may be associated with the susceptibility of animals to stress is also confirmed by FIEDLER *et al*. (1999). In turn, in a study with pigs, FAZARINC *et al*. (2002) concluded that the giant fibre syndrome is not related to g.1843C>T mutation of the ryanodine receptor (RYR1), although in recessive homozygous pigs (RYR1 nn) the authors observed a higher proportion of giant fibres compared to heterozygous (Nn) and dominant homozygous pigs (NN).

It is concluded from the results obtained that the PSE syndrome contributes to the incidence of pathological changes in turkey breast meat, and in particular increases the frequency of giant, atrophic and angular fibres, which may decrease turkey meat quality.

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