The Incidence of Muscle Abnormalities in Broiler Breast Meat – A Review

Xi Huang¹ and Dong Uk Ahn²,*

¹College of Food Science & Technology, Huazhong Agricultural University, Egg Processing Technology Local Joint National Engineering Research Center, National R&D Center for Egg Processing, Wuhan, Hubei 430070, China
²Department of Animal Science, Iowa State University, Ames, IA 50011, USA

Abstract  The dramatic improvements in the growth rate and breast muscle size and yield in broilers through the intensive genetic selection, and the improvement in nutrition and management over the past 50 years have introduced serious abnormalities that influenced the quality of breast meat. The abnormalities include pale-soft-exudative (PSE) conditions, deep pectoral muscle (DPM) myopathy, spaghetti meat (SM), white striping (WS), and woody breast (WB) that have serious negative implications to the broiler meat industry. The incidences of PSE and DPM have been known for several decades, and their prevalence, etiology and economic impact have been well discussed. However, other abnormalities such as SM, WS and WB conditions have been reported just for few years although these conditions have been known for some time. The newly emerging quality issues in broilers are mainly associated with the Pectoralis major muscles, and the incidences have been increased dramatically in some regions of the world in recent years. As high as 90% of the broilers are affected by the abnormalities, which are expected to cause from $200 million to $1 billion economic losses to the U.S. poultry industry per year. So, this review mainly discusses the histopathological characteristics and biochemical changes in the breast muscles with the emphasis on the newly emerging abnormalities (SM, WS, and WB) although other abnormalities are also discussed. The impacts of the anomalies on the nutritional, functional, mechanical and sensory quality of the meat and their implications to the poultry industry are discussed.

Keywords  broiler, muscle abnormalities, histopathological characteristics, biochemical changes, meat quality

Introduction

The consumption of chicken meat has been increased continuously over the past several decades thanks to the dramatic improvement in meat production through the intensive genetic selection for the growth rate and muscling, and the improvement in nutrition and management over the past 50 years (Aviagen, 2014). The improvements in the growth rate, breast muscle size, and yield in broilers, however, introduced serious abnormalities that influenced the quality of breast meat. Some of the abnormalities
such as pale-soft-exudative (PSE) conditions and deep pectoral muscle (DPM) myopathy were known for decades, while other abnormalities such as spaghetti meat (SM), white striping (WS) and woody breast (WB) have not been reported until recently even though these conditions also have been known for some time (Maiorano, 2017; Petracci and Cavani, 2012). All the old and new abnormalities are associated with the dramatic improvement in growth rate and muscle size, which affected the structure, metabolism and repair mechanisms in poultry muscles (MacRae et al., 2006; Velleman, 2015; Velleman et al., 2014).

Although the incidence levels of the abnormalities vary widely depending upon researchers, Kuttappan et al. (2016) reported that the percent of broilers exhibiting WS/WB was as high as 90% and caused the U.S. poultry industry from $200 million to $1 billion economic losses per year. This review discusses the etiology, histological, physiological and chemical aspects of the abnormalities. The impacts of the anomalies on the functional, mechanical and sensory quality of the meat and their implications to the poultry industry are also discussed.

**Pale-Soft-Exudative (PSE) Conditions**

The incidence of PSE is closely related to the increased susceptibility of the birds to stress while other abnormal conditions are more related to the myopathies. A genetic mutation in the ryanodine receptor of sarcoplasmic reticulum that regulates calcium uptake and release is generally believed to be associated with the stress-susceptibility and the development of PSE conditions in pigs (Barbut et al., 2008). In poultry, however, it is not clear if the genetic mutation is the primary cause of PSE (Strasburg and Chiang, 2009). The PSE conditions in chickens are mainly attributed to a rapid post-mortem decline in pH due to pre-slaughter stresses and environmental factors (heat stress) (Petracci et al., 2015). Sandercoc et al. (2009) reported that fast-growing birds exhibited a reduced thermoregulatory capacity, and thus those birds are more susceptible to heat stress during the pre-slaughter period. Heat stress causes various problems such as muscle damage, acid-base disturbances, and reduced meat quality. The broiler breast muscle is mainly composed of fast-twitching fibers that are mainly associated with the anaerobic glycolysis (Yost et al., 2002). Pre-slaughter stress conditions accelerate anaerobic glycolysis during the conversion of muscle to meat and lower muscle pH while the carcass temperature is still high. The low pH and high-temperature combination leads to protein denaturation and produces PSE conditions (Berri et al., 2005; Wilhelm et al., 2010). The PSE meat has an unattractive pale color, soft texture, and low water-holding capacity (WHC) and protein extractability (Barbut et al., 2008). Therefore, the processing ability of PSE meat is low, and the texture of the processed meat products is brittle and dry. The incidence level of PSE conditions is about 5% to 40%, and thus PSE conditions cause significant economic losses to the poultry industry (Owens et al., 2009).

**Deep Pectoralis Muscle (DPM) Myopathy**

DPM, also known as “green muscle disease,” was first described in turkeys and chicken breeders as “degenerative myopathy” (Dickinson et al., 1968). However, it has become common in all meat-type fast-growing birds (Bilgili and Hess, 2002; Grunder et al., 1984). DPM is defined as ischemic necrosis that develops in Pectoralis minor, and the genetics, physio-anatomical background and the management of birds are related to the incidence of DPM. The Pectoralis major and the Pectoralis minor are the two muscles involved in wing movements. When the birds are nervous, startled, flight and struggle because of feed or water outages, human activities, and excessive noises in and around chicken houses, they prone to have sudden movements of wings (Bilgili et al., 2000). In the turkeys and broilers, Pectoralis minor muscle that raises wings during the wing flapping expands its volume by about 20%-25% for the increased blood flow into the muscle. However,
**Pectoralis minor** muscle is sandwiched between the sternum and inelastic fascia, and thus cannot fully expand during the activities. This generates pressure to the **Pectoralis minor** muscle and occludes the blood vessels, and causes oxygen deficit and develops ischemia (Jordan and Pattison, 1998; Sille, 1985). In lighter birds, this pressure quickly returns to the normal state. In heavy birds, however, the pressure can be maintained long enough to lead to irreversible necrotic changes in the **Pectoralis minor** muscle (Bilgili and Hess, 2002). In the early stage, DPM muscles show acute inflammation, edema, and hemorrhages. In the later stage, however, the conditions change to degenerative necrosis with muscle fiber atrophy, follicular, segmental and granular decomposition, macrophage infiltrations, connective tissue proliferation, and eosinophile and phagocyte infiltrations (Fig. 1). In the most advanced lesions, normal muscle fibers are replaced by fibrous and adipose tissue and the color changes from pink to green (Bianchi et al., 2006).

The part with deep pectoral myopathy in breast muscle is trimmed off, which downgrades the breast and causes an economic loss for the industry (Kijowski et al., 2014). Under commercial rearing conditions, broilers and turkeys are relatively inactive, and the **Pectoralis minor** muscle is not used much. Therefore, the elasticity of the muscle compartment is reduced, and it is difficult for those birds to accommodate the swollen muscle after wing flapping. The reported incidence level of DPM varies widely depending upon the age of birds and genotype: Bianchi et al. (2006) reported that the incidence of carcasses affected by DPM was 0.84%, but the range of total DPM varied from 0% to 16.7% range. Considerable variations were also observed for early (range: 0% to 12.0%) and old (range: 0% to 5.6%) developing stages of DPM. Ross 308 exhibited a higher incidence of DPM than Cobb 500 (1.27% vs 0.35%). Lien et al. (2011) reported that the incidence of spontaneous DPM cases ranged from 3% to 17% and was greater in broiler chickens with a higher growth dynamic than those with normal growth rate. Badger (2011) identified 4.4% DPM cases in chickens at slaughter and cutting up the carcass. Kijowski et al. (2014) reported that the incidence of DPM cases in commercial slaughter chickens was between 0.02% and 1.9% in Poland.

**White Striping (WS)**

WS is characterized by white striations parallel to muscle fibers due to fiber degenerations with fat infiltration (lipidosis) and

![Fig. 1. Deep pectoralis muscle myopathy.](image-url) (A) Early stage of DPM showing muscle damage and hemorrhage, (B) Later stage of DPM showing muscle greening. DPM, deep pectoral muscle.
connective tissues (fibrosis) (Fig. 2). The intensity and the thickness of the white stripes can vary from bird to bird. Kuttappan et al. (2012a) developed a classification system for WS based on visual appearance: normal, moderate and severe. However, more extensive groupings can also be used when necessary. The major histopathological changes associated with WS include necrosis of fibers, degenerating and regenerating fibers of variable size, loss of cross striations, mineralization of nuclei, hyalinization, replacement of muscle fibers with fibrous connective tissues (fibrosis), accumulation of adipose tissues (lipidosis), and lymphocytes and macrophages infiltration (Ferreira et al., 2014; Russo et al., 2015; Sihvo et al., 2014). Kuttappan et al. (2013b) reported that the WS was not associated with any systemic infections, inflammatory, or stress conditions, but had increased levels of serum creatine kinase (CK), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH). When the damages are acute or continuous, and the attempts to repair the damages fail, satellite cells of the muscle tissues become fibroblasts or adipocytes and lead to fibrosis and lipidosis.

Kuttappan et al. (2009) and Kuttappan et al. (2013a) found that more than 50% (50.7%–55.8%) of broilers had WS conditions at 59–63 days of age. Trocino et al. (2015) found 74.5% of the fillets examined were WS at the gross examination, but 97% of the breast muscles examined showed histologically damaged muscle fibers. Many studies reported that the incidence of severe WS increased dramatically from 1.4%–8.7% (average 5%) in 2012 (Kuttappan et al., 2012a; Petracci et al., 2013) to 25.7%–32.3% (average 29%) in 2015 (Russo et al., 2015; Tijare et al., 2016), and to 75.5% in 2017 (Gratta et al., 2017), indicating very rapid increase of the incidence levels. Also, the occurrence of severe WS was higher in Ross 308 chickens than in Cobb 500 chickens (25.9% vs 7.41%), and up to 40% of medium and heavy broiler chickens raised under commercial conditions had WS conditions (Lorenzi et al., 2014).

Feeding a high-energy diet produced birds with higher body weight and accomplished lower feed conversion ratios but increased the incidence of WS breast fillets. Higher levels of lipids and lower amounts of protein in diets also increased the occurrence of WS in broilers (Kuttappan et al., 2009). Bauermeister et al. (2009) reported a higher incidence in broilers processed at an older age (8 wks old) than a younger age (6 wks old) because of the higher body weight at older ages. More than half of the broilers are processed at older ages currently (>8 wks), and the meats are deboned and used for portioning and further processing (Owens, 2018). So, WS in broiler breast fillets could affect the poultry meat market significantly and result in higher economic losses.

Fig. 2. White striping on a breast muscle (A) and white striping shown on a carcass (B).
The occurrence of WS in broilers was reduced by controlling the growth trajectory through feed reduction during the rearing period (Meloche et al., 2018b). Restricting feed from 13 to 21 d of age was effective in controlling muscle fiber degeneration, but too early feed restriction (first two-week post-hatching) had adverse effects on the growth of broilers (Kuttappan et al., 2012a). During the re-alimentation period, however, previously restricted birds showed a compensatory growth and the effects of feed restriction disappeared (Trocino et al., 2015). The broiler line with high ultimate pH had a higher incidence of moderate and severe WS than the low ultimate pH lines regardless of the sex (Alnahhas et al., 2016; Muldahl et al., 2015; Petracci et al., 2013).

WS meat exhibited a higher percentage of moisture, intramuscular fat and collagen, and lower contents of myofibrillar and sarcoplasmic proteins and ash (Kuttappan et al., 2013a; Petracci et al., 2013). Because of the decreased total protein content but increased collagen and fat contents in WS breast muscles, their WHC, protein solubility and marinade uptake are lower than the normal muscles (Sihvo et al., 2014). The digestibility and the quality of amino acids in collagen are inferior to myofibrillar proteins. Therefore, the nutritional quality of WS meat is lower than that of the normal meat (Mudalal et al., 2014). The solubility of myofibrillar proteins is highly correlated with the WHC (Warner et al., 1997) and the functionality of the meat (Bowker and Zhuang, 2013). Bowker and Zhuang (2016) reported that WS fillets had higher ultimate pH and Allo-Kramer (AK)-shear force but had lower a* - and b* - color values than the normal fillets (p<0.001). Lightness (L*) was generally higher in the WS than the normal breast fillets but was not always consistent. Some researchers reported that WS had a significant impact on the visual appearance of breast fillets and lowered the consumer acceptance of the meat (Kuttappan et al., 2012c; Mudalal et al., 2014). However, Soglia et al. (2018a) indicated that WS conditions did not have significant effects on meat quality.

There are a few other known causes of myodegeneration such as vitamin E and selenium deficiency, exertional or exercise-induced myopathy, toxins, and tissue hypoxia in poultry. Among those myopathies, nutritional muscular dystrophy (NMD) is characterized with white-colored bands on the breast and leg muscles in the direction of the muscle fibers, which is very similar to that of the WS muscles. The NMD is associated with the degeneration of muscle fascicules (Netke et al., 1969) and its microscopic characteristics are also similar to those of the WS (Kuttappan et al., 2009). The NMD is caused mainly by the vitamin E and Se deficiency and can be prevented by the dietary supplementation of vitamin E and Se (Walsh et al., 1993). However, dietary vitamin E level had little effect on the incidence of WS (Guetchom et al., 2012; Kuttappan et al., 2012b).

**Spaghetti Meat (SM)**

WS defect is also associated with another muscle abnormality termed SM (Sirri et al., 2016). While WS fillets exhibited necrosis and lysis of fibers, fibrosis, lipidosis, inflammation, loss of cross striation, and vacuolar and hyaline degeneration, SM is characterized by poor muscle cohesiveness due to the immature intramuscular connective tissues (Bowker and Zhuang, 2016; Radaelli et al., 2017; Sihvo et al., 2017). Collagen maintains the structural integrity of skeletal muscle (McCormick, 2009; Purslow, 2005): intramuscular connective tissues are composed of three layers called endomysium, perimysium, and epimysium, which surrounds individual muscle fibers, the bundles of muscle fibers, and the whole muscle, respectively. In SM, the density of connective tissues in endomysium and perimysium progressively decreases, and thus the muscle fiber bundles become easily disintegrated or mushy (look like spaghetti) (Puolanne and Vuotila, 2009).

The histopathological features of the SM abnormality share similar morphological changes to other myopathies (e.g., WS, WB): Swatland (1990) observed abnormal conditions similar to the SM in turkey breast muscles and attributed that to the
outgrowth of muscle fibers to their connective tissues. Ahn et al. (2010) observed that the perimysial septa of breast muscles in fast-growing birds were thinner than the slow-growing counterparts. Muscles with both WS/SM conditions showed the most severe histological lesions with increased adipose tissue infiltration in the endo- and perimysial spaces and high intra- and extra-myofibrillar water content (Radaelli et al., 2017). Using NMR, Baldi et al. (2018) found a movement of water from intra-myofibrillar to extra-myofibrillar area in SM. A higher proportion of extra-myofibrillar water in the superficial section of SM led to a reduction of WHC of the meat. Both WS and SM abnormalities influenced meat quality, but SM had more pronounced adverse effects on meat quality than the WS (Baldi et al., 2018).

**Wooden (or Woody) Breast (WB)**

WB is macroscopically characterized by bulged, hard and rigid muscles with a surface hemorrhage (Fig. 3) and the presence of a light-yellow viscous exudate on the muscle surface (Mazzoni et al., 2015; Mudalal et al., 2015). Wooden breast muscle has a larger cross-sectional area (Dalle Zotte et al., 2014), and higher intramuscular collagen and ultimate postmortem

![Fig. 3. WB conditions on a breast muscle and WB conditions shown carcass.](image1)

(A) WB: shows bulged areas (arrow) and hemorrhage, (B) WB with white striping: shows bulged area, white stripes, and hemorrhages, (C) Cross section of WB muscle: white lines (arrow) are signs of fibrosis and lipidosis, (D) WB conditions on a carcass shows bulged area, white stripes, hemorrhages and yellowish viscous fluids. WB, woody breast.
Breast Muscle Abnormalities

pH (Chatterjee et al., 2016; Clark and Velleman, 2016; Petracci et al., 2015; Soglia et al., 2016) than the normal muscle. Histopathologically, WB is characterized with increased degenerative and atrophic fibers, variability in fiber size, floccular/vacuolar degeneration and lysis of fibers, hyalinization, lipidosis, extensive fibrillar collagen deposition (fibrosis), and macrophage infiltration (Sihvo et al., 2014; Soglia et al., 2016; Velleman and Clark, 2015). The WB lesions begin focally and spread as a diffuse form as the age of the bird increased. Some birds showed markedly hardened breast muscle with no apparent fibrosis histologically, indicating that fibrosis is not the sole factor behind the hardness of the WB meat and the lesion is not restricted to the Pectoralis major muscles only.

The birds with high growth rate, feed efficiency, and breast muscle yield are more likely to develop myopathies including WB (Griffin et al., 2018) because the muscle tissues outgrow the supporting systems such as connective tissues and capillaries and change the structure and metabolism of the muscle (Petracci and Cavani, 2012; Sandercoc et al., 2009). Muscle fiber formation is virtually complete at hatch. Hyperplasia occurs during the embryonic development and hypertrophy after hatch. Hypertrophy can increase the muscle fiber size (cross-sectional area) 5–6 folds (Dransfield and Sosnicki, 1999). The post-hatch muscle growth, development, and maintenance are accomplished through the activities of satellite cells. Satellite cells are multipotential stem cells that are heterogeneous depending upon the fiber type of the muscle they are derived from and the expression of cell surface markers. They are located between the basement membrane and plasmalemma of muscle fibers (Mauro, 1961). The satellite cells proliferate, differentiate and fuse with adjacent fibers but remain quiescent after hatch (Moscatello et al., 1998). The muscle fiber enlargement is accomplished through the fusion of satellite cells into the existing muscle fibers that adds nuclei to the muscle fibers and increases protein synthesis (Allen et al., 1979; Velleman, 2015).

The extreme hypertrophy, however, results in reduced endomysial and perimysial connective tissue spacing (Velleman et al., 2014). Therefore, birds selected for hypertrophy have limited space for capillaries in the perimysial connective tissues area, which results in the decreased size and density of the capillary network (Sosnicki and Wilson, 1991). Both large muscle fibers and inadequate vascularization induce metabolic stress because the greater diffusion distances for oxygen, metabolites and waste products make it difficult to supply oxygen as well as remove the metabolic wastes and initiate muscle damages (Alnahhas et al., 2015; Alnahhas et al., 2016; MacRae et al., 2006). The low oxygen and nutrients from blood during the ischemic period lead to inflammation and oxidative stress rather than restoring the tissues to normal function. The re-supply of blood flow and oxygen to the ischemic cells lead to increased damage to cellular proteins and plasma membranes due to the production of superoxide by the xanthine-xanthine oxidase system and the deficiency of scavenger enzymes to remove the superoxide and other reactive oxygen species (ROS) (Halliwell and Gutteridge, 1990). An excess of ROS within the muscle tissue is involved in initiating the inflammatory mechanisms associated with the WS and WB muscle abnormalities (Mutryn et al., 2015).

The main inflammatory cells found around the degenerative fiber are macrophages (Sihvo et al., 2013). The activation of macrophages in stress conditions creates a source of additional free radicals. Macrophages use superoxide to destroy pathogens as their first line of defense. However, superoxide (O \( \cdot^- \)) is toxic to the cells and needs to be converted to a less toxic form quickly by superoxide dismutase. If not combated by the animals’ natural line of defense, however, the free radicals may lead to more cellular destruction due to lipid peroxidation of the biological membranes and compromise their structure and functions (Zhao et al., 1998). Trace minerals such as Se, Zn, Mn, and Cu are important for the activity of antioxidant enzymes, and play important roles in growth, immunity and gut health of animals (Cemin et al., 2018; York et al., 2016). Dietary supplementation of those minerals in combination with antioxidants enhanced the bird's natural defense system in the tissues, which are essential to combat free radical production (Kidd, 2004; Midorikawa et al., 2001).
Zambonelli et al. (2016) reported that WB muscle showed different genetic expression for glycolysis, oxidation, calcium signaling pathways, and proteoglycan and polysaccharide synthesis from normal muscles. The RNA-seq analysis and the microscopic and biochemical studies indicated that the localized hypoxia, increased muscle degradation, reduced glucose utilization, increased intracellular calcium and muscle fiber-type switching are the critical features of myopathic muscles (Abasht et al., 2016; Mutryn et al., 2015; Petracci et al., 2015). After the initial degeneration, the damages to the sarcoplasmic reticulum surrounding muscle fibers increase calcium influx and activate calcium-dependent protease (calpain) and initiate necrosis. WB exhibited a significantly higher amount of free calcium. The increase of sodium and calcium content changes the intracellular ion homeostasis, and the increase of glycolytic activity lowers the amount of glycogen reserve in the muscle, which leads to an increase of ultimate pH in abnormal muscles. Fiber-type switching, from type IIb fast-twitching fibers towards slow-twitching type I fibers, decreases the enzymes related to the glycolytic pathway and glycolysis (Soglia et al., 2016).

The instrumental texture of the cooked severe WB was harder than the normal breast fillets, and sensory analyses of the cooked WB showed more springiness and cohesiveness than the normal fillets (Chatterjee et al., 2016). However, the sensory texture attributes of WB were not uniform throughout the entire Pectoralis major muscle (Aguirre et al., 2018; Brambila et al., 2017). Tasoniero et al. (2017) reported that the hardness of WB is related to the extra water trapped in the myofibrillar matrix (myowater). Velleman and Clark (2015) suggested that the hardened area of wooden breast muscle is related to the very high levels of decorin expression and excessive collagen crosslinking. Decorin is a small leucine-rich proteoglycan that is involved in collagen crosslinking and other functions including cellular growth, collagen fibril structure, and extracellular matrix organization (Danielson et al., 1997). However, Radaelli et al. (2017) reported that the replacement of the degenerated muscle fibers with connective tissue (fibrosis) was not related to the muscle hardness. The activity of μ/m-calpain decreased significantly in WB during storage but the differences in the calpain activities during the postmortem period was not the primary factor for the increased hardness in WB (Soglia et al., 2018b). Marination decreased the toughness in WB fillets but did not eliminate tenderness differences between the normal and the WB fillets.

Compared with the normal breasts, WB meats showed higher drip and cooking losses and shear force for both raw and cooked meat (Aguirre et al., 2018; Chatterjee et al., 2016; Mudalal et al., 2014). Although, the objectionable texture characteristics of WB fillets can be overcome by grinding the meat, the compositional changes still can result in impaired functionality in further-processed products (Bowker and Zhuang, 2016; Mazzoni et al., 2015; Mudalal et al., 2015; Tijare et al., 2016). WB fillets had higher color L*, a*, and b*-values and pH than the normal breast fillets (Cai et al., 2018). Wold et al. (2017) developed a Near-infrared (NIR) spectroscopy for rapid and non-destructive detection and grading of WB in chicken breast fillets and found that the NIR was a practical and useful tool for detecting and grading WB syndrome in the processing line. They also used low-field NMR to measure WHC and found that WB muscles had a significantly higher share of loosely bound water due to muscle fiber degeneration.

WB conditions have been found in several countries around the world, including the United States, Finland, Italy, and Brazil. Although, the industry-wide incidence rates for the WB muscle are challenging to assess, approximately 5%–10% of commercially produced breast fillets display severe WB conditions. Owens (2014) reported that 30% to 50% of broilers at 56 days of age (BW>4.2 kg) showed severe WB conditions. Gratta et al. (2017) reported that 5.1% of commercial broilers were WB, while Cruz et al. (2017) estimated up to 85.9% at 35 d of age and up to 89.2% at 42 d of age. Tijare et al. (2016) observed up to 96.1% of WB incidence in broilers reared using commercial diets. WS often coexisted with WB. The first WS and WB cases were seen at 10–18 days of age, and the prevalence levels increased as the age of the birds increased (Sihvo et al., 2017; Wold et al., 2017). Myopathies are more directly related to the slope of the growth trajectory of birds and the
Breast Muscle Abnormalities

Reduced nutrient allocation at 90% density throughout the grow out period reduced the severity of WB. The quantitative control of nutrient intake, however, had a negative impact to the performance of the birds and processing characteristics (Meloche et al., 2018a; Meloche et al., 2018b; Trocino et al., 2015). Therefore, the concurrent manipulation of dietary amino acid and energy density is not a viable and practical solution for breast myopathies (Meloche et al., 2018a).

Woody Breast with White Striping

Mudalal et al. (2015) indicated that the measurements of height or compression force in the caudal part of fillets could be used as a tool to discriminate between WS and wooden breast abnormalities. Both WS and WB are observed in varying degrees of severity, and sometimes they appear together in one muscle. WS and WB alone or together negatively impacted several aspects of meat quality, including WHC and proximate composition (low protein coupled with high moisture, collagen, and fat contents) (Soglia et al., 2016; Tijare et al., 2016). The occurrence of both WS and WB is closely associated with up-regulation of protein metabolism and the regenerative process to repair the degenerative changes, and the down-regulation of carbohydrate metabolism (Kuttappan et al., 2017).

Both WS- and WB-lesioned areas showed myodegeneration with a variable amount of interstitial connective tissue accumulation (Sihvo et al., 2014), which changed the nutritional and technological properties of meat (Mazzoni et al., 2015; Mudalal et al., 2015), and negatively affected the consumer preference (Kuttappan et al., 2012c). Over 50% of consumers indicated that they would not buy WS and WB fillets (Kuttappan et al., 2012c).

The WB/WS myopathies have both macroscopic and microscopic similarities to other myopathic conditions like hereditary muscular dystrophy, nutritional myopathy, toxic myopathies, etc., but their etiologies are different (Kuttappan et al., 2016). Mutryn et al. (2015) identified a set of differentially expressed genes involved in myofiber reactions to oxidative stress in WS/WB muscles. Dridi and Kidd (2016) reported that WB meat is a result of a metabolic disorder that is associated with hypoxia and oxidative stress. Immunoglobulin superfamily was also overexpressed in abnormal muscles, indicating the presence of the tissue inflammation. The expression of interleukin-1-beta (IL1B-β), a cytokine protein, is vital for the angiogenic processes (Voronov et al., 2003). The low IL1B-β expression level is related to the lack of pain symptoms in chickens affected by WB/WS abnormalities because IL1B-β regulates the pain sensation during inflammation.

Both WS and WB abnormalities impair the appearance and the functional quality of breast meats, but WB had a more detrimental effect than WS, and the adverse effect was more severe when both WS and WB were present together (Mudalal et al., 2015; Tasoniero et al., 2016). There are no published studies on microbial shelf-life of WS and WB fillets, although both have higher ultimate pH than the normal meat (Mudalal et al., 2015; Petracci et al., 2013). The notable discolorations on the skin-side surface of severe WB fillets after cooking and the white striations on the ventral surface of raw WS fillets negatively impacted the purchasing decisions of consumers.

Although the selection for increased growth rate and breast yield played a role in the occurrence of the abnormalities in poultry breast muscle, the genetic basis for the incidence of these myopathies is still debatable: Lien et al. (2011) reported low heritabilities for DPM (0.02–0.1) and relatively low heritability for WS (0.19–0.34). Bailey et al. (2015) also reported that the development of myopathies has poor heritability and genetic correlations, and environmental factors such as nutrition and management contributed more than 90% and 65% for the incidence of WB and WS, respectively. However, Alnahhas et al. (2016) reported that WS had a high heritability (0.65) and also had medium to high correlations with body weight, breast meat yield, and intramuscular fat (0.33, 0.68, and 0.64, respectively). Significant genetic associations were also observed
between WS and the ultimate pH of breast and thigh meat (0.21 and 0.31, respectively), and breast cook loss (0.30). Chabault et al. (2012) found that WS was highly correlated with the intramuscular fat content of the *Pectoralis major* muscle (heritability 0.83) and suggested to use it as an indirect criterion for the selection against WS.

**Conclusions**

The incidences of the newly emerging myopathies (SM, WS, and WB) as well as the previously known abnormalities (PSE and DPM) are much higher in the heavy birds selected for the fast growth rate and high breast size and yield than the slow-growing birds. However, the heritability and correlations of the genetic parameters for growth rate, breast yield and breast size to the abnormalities are lower than those of the environmental factors that include nutritional density of the diet, management, and age of the birds. Although, all the abnormalities (myopathies) are different macroscopically they share many similarities in histopathological and biochemical characteristics. Also, all the abnormalities share the common initiating mechanisms – muscle fiber growth outpaces the development of connective tissues and vascularization. Many researchers have studied the initiating mechanisms and the histopathological and chemical changes in the abnormal meat. However, early detection and prevention methods for the abnormalities are not known yet. Developing non-invasive biomarkers based on mass spectroscopy for diagnostic purposes in live birds and a rapid, automatic detection and grading system in slaughtering and processing lines can be of help, but they cannot be the fundamental method to solve the issues related to the muscle abnormalities.

The abnormalities in breast meat raise concerns over the quality as well as safety and lower the consumer acceptance of the meat. In severe cases, meats with abnormalities are downgraded, and the defected meats are often used in processed products (e.g., sausages or nuggets) where the chemical composition can be modified during formulation (Qin, 2013). However, it only alleviates the quality problems (texture, color), not completely solve the problems. In 2017, USDA (2017) announced that the breast meat with the signs of inflammation (WS and WB abnormalities) should be trimmed off. The meats with inflammation are considered unwholesome and unfit for human consumption. Therefore, the economic loss due to the myopathies can be greater than the impact caused by the quality loss alone. So, more fundamental works to develop birds with no myopathic conditions or studies to prevent the initiating process through genetic selections or management for the high fibroblastic expression (collagen development) and angiogenesis may be necessary.

**Acknowledgment**

This study was supported by the National Natural Science Foundation of China 31471602, the National Key Research and Development Program of China (2018YFD0400302), and the Iowa Agriculture and Home Economics Experiment Station, Ames, Iowa. Project No. IOW03721 is sponsored by Hatch Act and State of Iowa funds.

**References**

Abasht B, Mutryn MF, Michalek RD, Lee WR. 2016. Oxidative stress and metabolic perturbations in wooden breast disorder in chickens. PLoS ONE 11:e0153750.

Aguirre ME, Owens CM, Miller RK, Alvarado CZ. 2018. Descriptive sensory and instrumental texture profile analysis of woody breast in marinated chicken. Poult Sci 97:1456-1461.

Ahn JY, Zheng JX, Li JY, Zeng D, Qu LJ, Xu GY, Yang N. 2010. Effect of myofiber characteristics and thickness of
Breast Muscle Abnormalities

Allen RE, Merkel RA, Young RB. 1979. Cellular aspects of muscle growth: Myogenic cell proliferation. J Anim Sci 49:115-127.

Alnahhas N, Berri C, Chabault M, Chartrin P, Boulay M, Bourin MC, Le Bihan-Duval E. 2016. Genetic parameters of white striping in relation to body weight, carcass composition, and meat quality traits in two broiler lines divergently selected for the ultimate pH of the pectoralis major muscle. BMC Genet 17:61.

Alnahhas N, Le Bihan-Duval E, Baëza E, Chabault M, Chartrin P, Bordeau T, Cailleau-Audouin E, Mèteau K, Berri C. 2015. Impact of divergent selection for ultimate pH of pectoralis major muscle on biochemical, histological and sensorial attributes of broiler meat. J Anim Sci 93:4524-4531.

Aviagen. 2014. Ross 308 broiler: Performance objectives. In-house publication, global. Aviagen Ltd., Newbridge, UK.

Badger C. 2011. Green muscle disease. American Pastured Poultry Producers Association Newslett 66:8-9.

Bailey RA, Watson KA, Bilgili SF, Avendano S. 2015. The genetic basis of Pectoralis major myopathies in modern broiler chicken lines. Poult Sci 94:2870-2878.

Baldi G, Soglia F, Mazzoni M, Sirri F, Canonico L, Babini E, Laghi L, Cavani C, Petracci M. 2018. Implications of white striping and spaghetti meat abnormalities on meat quality and histological features in broilers. Animal 12:164-173.

Barbut S, Sosnicki AA, Lonergan SM, Knapp T, Ciobanu DC, Gatcliffe LJ, Huff-Lonergan E, Wilson EW. 2008. Progress in reducing the pale, soft and exudative (PSE) problem in pork and poultry meat. Meat Sci 79:46-63.

Bauermeister LJ, Morey AU, Moran ET, Singh M, Owens CM, McKee SR. 2009. Occurrence of white striping in chicken breast fillets in relation to broiler size. Poultry Sci 88:33.

Bilgili SF, Hess JB. 2002. Green muscle disease in broilers increasing. World’s Poult 18:42-43.

Bilgili SF, Hess, JB, Lien, RJ, Downs, KM. 2000. Deep pectoral myopathy in broiler chickens. In Proceedings of the XXI World’s Poultry Congress, Montreal, Canada, World’s Poultry Science Association: Montreal, Canada. pp 20-24.

Bowker B, Zhuang H. 2016. Impact of white striping on functionality attributes of broiler breast meat. Poult Sci 95:1957-1965.

Bowker BC, Zhuang H. 2013. Relationship between muscle exudate protein composition and broiler breast meat quality. Poult Sci 92:1385-1392.

Brambila GS, Chatterjee D, Bowker B, Zhuang H. 2017. Descriptive texture analyses of cooked patties made of chicken breast with the woody breast condition. Poult Sci 96:3489-3494.

Cai K, Shao W, Chen X, Campbell YL, Nair MN, Suman SP, Beach CM, Guyton MC, Schilling MW. 2018. Meat quality traits and proteome profile of woody broiler breast (Pectoralis major) meat. Poult Sci 97:337-346.

Cemin HS, Vieira SL, Stefanello C, Kindlein L, Ferreira TZ, Fireman AK. 2018. Broiler responses to increasing selenium supplementation using Zn-L-selenomethionine with special attention to breast myopathy. Poult Sci 97:1832-1840.

Chabault M, Baëza E, Gigaud V, Chartrin P, Chapuis H, Boulay M, Arnould C, D’Abbadie F, Berri C, Le Bihan-Duval E. 2012. Analysis of slow-growing line reveals wide genetic variability of carcass and meat quality-related traits. BMC
Chatterjee D, Zhuang H, Bowker BC, Rincon AM, Sanchez-Brambila G. 2016. Instrumental texture characteristics of broiler Pectoralis major with the wooden breast condition. Poult Sci 95:2449-2454.

Clark DL, Velleman SG. 2016. Spatial influence on breast muscle morphological structure, myofiber size, and gene expression associated with the wooden breast myopathy in broilers. Poult Sci 95:2930-2945.

Cruz RFA, Vierra SL, Kindlein L, Kipper K, Cemin HS, Rauber SM. 2017. Occurrence of white striping and wooden breast in broilers fed grower and finisher diets with increasing lysine levels. Poultry Sci 96:501-510.

Dalle Zotte A, Cecchinato M, Quartesan A, Bradanovic J, Tasoniero G, Puolanne E. 2014. How does wooden breast myodegeneration affect poultry meat quality? 60th International Congress of Meat Science Technology, Punta Del Este, Uruguay. pp 476-479.

Danielson KG, Baribault H, Holmes DF, Graham H, Kadler KE, Iozzo RV. 1997. Targeted disruption of decorin leads to abnormal collagen fibril morphology and skin fragility. J Cell Biol 136:729-743.

Dickinson EM, Stevens JO, Helfer DHA. 1968. Degenerative myopathy in Turkeys. In Proceedings of 17th Western Poultry Disease Conference, Davis, CA, USA. p 6.

Dransfield E, Sosnicki AA. 1999. Relationship between muscle growth and poultry meat quality. Poult Sci 78:743-746.

Dridi S, Kidd MT. 2016. Molecular pathways involved in amino acid and phosphorus utilization. Ch. 8. Phytate Destruction. In Consequences for precision animal nutrition. C. Walk et al. ed. Wageningen Academic Publishers, Wageningen, The Netherlands. pp 119-128.

Ferreira TZ, Casagrande RA, Vieira SL, Driemerier D, Kindlein L. 2014. An investigation of a reported case of white striping in broilers. J Appl Poult Res 23:748-753.

Gratta F, Birolo M, Piccirillo A, Petracci M, Maertens L, Xiccato G, Trocino A. 2017. Effects of the feeding system on performance and myopathy occurrence in two broiler chicken genotypes. Ital J Anim Sci 16:48.

Griffin JR, Moraes L, Wick M, Lilburn MS. 2018. Onset of white striping and progression into wooden breast as defined by myopathic changes underlying Pectoralis major growth. Estimation of growth parameters as predictors for stage of myopathy progression. Avian Pathol 47:2-13.

Grunder AA, Hollands KG, Gavora JS, Chambers JR, Cave NA. 1984. Degenerative myopathy of the Musculus supracoracoideus and production traits in strains of meat-type chickens. Poultry Sci 63:781-785.

Guetchom B, Venne D, Chénier S, Chorfi Y. 2012. Effect of extra dietary vitamin E on preventing nutritional myopathy in broiler chickens. J Appl Poult Res 21:548-555.

Halliwell B, Gutteridge JM. 1990. Role of free radicals and catalytic metal ions in human disease: An overview. Methods Enzymol 186:1-85.

Jordan FTW, Pattison M. 1998. Deep pectoral myopathy of turkeys and chickens. In Poultry diseases. Saunders Elsevier, London, UK. pp 398-399.

Kidd MT. 2004. Nutritional modulation of immune function in broilers. Poult Sci 83:650-657.

Kijowski J, Kupińska E, Stangierski J, Tomaszewska-Gras J, Szablewski T. 2014. Paradigm of deep pectoral myopathy in broiler chickens. World’s Poultry Sci J 70:125-138.

Kuttappan VA, Bottje W, Ramnathan R, Hartson SD, Coon CN, Kong BW, Owens CM, Vazquez-Añon M, Hargis BM. 2017. Proteomic analysis reveals changes in carbohydrate and protein metabolism associated with broiler breast myopathy. Poult Sci 96:2992-2999.
Kuttappan VA, Brewer VB, Apple JK, Waldroup PW, Owens CM. 2012a. Influence of growth rate on the occurrence of white
striping in broiler breast fillets. Poult Sci 91:2677-2685.
Kuttappan VA, Brewer VB, Clark FD, McKee SR, Meullernet JF, Emmert JL, Owens CM. 2009. Effect of white striping on
the histological and meat quality characteristics of broiler fillets. Poult Sci 88:136-137.
Kuttappan VA, Goodgame SD, Bradley CD, Mauromoustakos A, Hargis BM. Waldroup PW, Owens CM. 2012b. Effect of
different levels of dietary vitamin E (DL-α-tocopherol acetate) on the occurrence of various degrees of white striping on
broiler breast fillets. Poult Sci 91:3230-3235.
Kuttappan VA, Hargis BM, Owens CM. 2016. White striping and woody breast myopathies in the modern poultry industry: A
review. Poult Sci 95:2724-2733.
Kuttappan VA, Huff GR, Huff WE, Hargis BM, Apple JK, Coon C, Owens CM. 2013a. Comparison of hematologic and
serologic profiles of broiler birds with normal and severe degrees of white striping in breast fillets. Poult Sci 92:339-345.
Kuttappan VA, Lee YS, Erf GF, Meullernet JF, McKee SR, Owens CM. 2012c. Consumer acceptance of visual appearance of
broiler breast meat with varying degrees of white striping. Poult Sci 91:1240-1247.
Kuttappan VA, Shivaprasad HL, Shaw DP, Valentine BA, Hargis BM, Clark FD, McKee SR, Owens CM. 2013b. Pathological
changes associated with white striping in broiler breast muscles. Poultry Sci 92:331-338.
Lien RJ, Bilgili SF, Hess JB, Joiner KS. 2011. Finding answers to ‘green muscle disease’. Watt Poultry USA 5:15-18.
Lorenzi M, Mudalal S, Cavani C, Petracci M. 2014. Incidence of white striping under commercial conditions in medium and
heavy broiler chickens in Italy. J Appl Poult Res 23:754-758.
MacRae VE, Mahon M, Gilpin S, Sandercock DA, Mitchell MA. 2006. Skeletal muscle fibre growth and growth associated
myopathy in the domestic chicken (Gallus domesticus). Br Poult Sci 47:264-272.
Maiorano G. 2017. Meat defects and emergent muscle myopathies in broiler chickens: Implications for the modern poultry
industry. Sci Ann Pol Soc Anim Prod 13:43-51.
Mauro A. 1961. Satellite cell of skeletal muscle fibers. J Biophys Biochem Cytol 9:493-495.
Mazzoni M, Petracci M, Meluzzi C, Cavani P, Clavenzani A, Sirri F. 2015. Relationship between Pectoralis major muscle
histology and quality traits of chicken meat. Poult Sci 94:123-130.
McCormick RJ. 2009. Collagen. In Applied muscle biology and meat science (Du M, McCormick RJ. Ed). CRC Press, Boca
Raton, FL, USA. pp 129-148.
Meloche KJ, Fancher BI, Emmerson DA, Bilgili SF, Dozier WA III. 2018a. Effects of reduced dietary energy and amino acid
density on Pectoralis major myopathies in broiler chickens at 36 and 49 days of age. Poult Sci 97:1794-1807.
Meloche KJ, Fancher BI, Emmerson DA, Bilgili SF, Dozier WA III. 2018b. Effects of quantitative nutrient allocation on
myopathies of the Pectoralis major muscles in broiler chickens at 32, 43, and 50 days of age. Poult Sci 97:1786-1793.
Midorikawa K, Murata M, Oikawa S, Hiraka Y, Kawanishi S. 2001. Protective effect of phytic acid on oxidative DNA
damage with reference to cancer chemoprevention. Biochem Biophys Res Commun 288:552-557.
Moscatello DK, Santra M, Mann DM, McQuillan DJ, Wong AJ, Iozzo RV. 1998. Decorin suppresses tumor cell growth by
activating the epidermal growth factor receptor. J Clin Invest 101:406-412.
Mudalal S, Babini E, Cavani, Petracci M. 2014. Quantity and functionality of protein fractions in chicken breast fillets
affected by white striping. Poult Sci 93:2108-2116.
Mudalal S, Lorenzi M, Soglia F, Cavani C, Petracci M. 2015. Implications of white striping and wooden breast abnormalities
on quality traits of raw and marinated chicken meat. Animal 9:728-734.
Mutryn MF, Brannick EM, Fu F, Lee WR, Abasht B. 2015. Characterization of a novel chicken muscle disorder through differential gene expression and pathway analysis using RNA-sequencing. BMC Genomics 16:399.

Netke SP, Velu JG, Norton HW, Scott HM. 1969. Muscular dystrophy in chicks fed crystalline amino acid diets. J Nutr 99:315-319.

Owens CM. 2014. Identifying quality defects in poultry processing. Watt Poultry USA, pp 42-50.

Owens CM. 2018. Woody breast in the poultry meat industry. Midwest Poultry Federation Convention.

Owens CM, Alvarado CZ, Sams AR. 2009. Research developments in pale, soft, and exudative turkey meat in North America. Poult Sci 88:1513-1517.

Petracci M, Cavani C. 2012. Muscle growth and poultry meat quality issues. Nutrients 4:1-12.

Petracci M, Mudalal S, Bonfiglio A, Cavani C. 2013. Occurrence of white striping under commercial conditions and its impact on breast meat quality in broiler chickens. Poult Sci 92:1670-1675.

Petracci M, Mudalal S, Soglia F, Cavani C. 2015. Meat quality in fast-growing broiler chickens. World’s Poultry Sci 71:363-374.

Puolanne E, Voutila L. 2009. The role of connective tissue in poultry meat quality. In Proceedings of the XVIII European Symposium on the Quality of Poultry Meat and XIII European Symposium Quality of Eggs and Egg Products, Turku, Finland. p 26.

Purslow PP. 2005. Intramuscular connective tissue and its role in meat quality. Meat Sci 70:435-447.

Qin N. 2013. The utilization of poultry breast muscle of different quality classes. M.S. thesis, University of Helsinki, Finland. http://hdl.handle.net/10138/41630.

Radaelli G, Piccirillo A, Birolo M, Bertotto D, Gratta F, Ballarin C, Vascellari M, Xiccato G, Trocino A. 2017. Effect of age on the occurrence of muscle fiber degeneration associated with myopathies in broiler chickens submitted to feed restriction. Poult Sci 96:309-319.

Russo E, Drigo M, Longoni C, Pezzoti R, Fasoli P, Recordati C. 2015. Evaluation of white striping prevalence and predisposing factors in broilers at slaughter. Poult Sci 94:1843-1848.

Sandercock DA, Barker ZE, Mitchell MA, Hocking PM. 2009. Changes in muscle cell cation regulation and meat quality traits are associated with genetic selection for high body weight and meat yield in broiler chickens. Genet Sel Evol 41:1-8.

Sihvo HK, Immonen K, Puolanne E. 2013. Myodegeneration with fibrosis and regeneration in the pectoralis major muscle of broilers. Vet Pathol 51:619-623.

Sihvo HK, Immonen K, Puolanne E. 2014. Myodegeneration with fibrosis and regeneration in the Pectoralis major muscle of broilers. Vet Pathol 51:619-623.

Sihvo HK, Lindén J, Airas N, Immonen K, Valaja J, Puolanne E. 2017. Wooden breast myodegeneration of Pectoralis major muscle over the growth period in broilers. Vet Pathol 54:119-128.

Sille WG. 1985. Deep pectoral myopathy: A penalty of successful selection for muscle growth. Poult Sci 64:1591-1595.

Sirri F, Maiorano G, Tavaniello S, Chen J, Petracci M, Meluzzi A. 2016. Effect of different levels of dietary zinc, manganese and copper from organic or inorganic sources on performance, bacterial chondronecrosis, intramuscular collagen characteristics and occurrence of meat quality defects of broiler chickens. Poult Sci 95:1813-1824.

Soglia F, Baldi G, Laghi L, Mudalal S, Cavani C, Petracci M. 2018a. Effect of white striping on turkey breast meat quality. Animal (in press). doi: 10.1017/S1751731117003469.

Soglia F, Mudalal S, Babini E, Di Nunzio M, Mazzoni M, Sirri F, Cavani C, Petracci M, 2016. Histology, composition, and
quality traits of chicken *Pectoralis major* muscle affected by wooden breast abnormality. Poult Sci 95:651-659.

Soglia F, Zeng Z, Gao J, Puolanne E, Cavani C, Petracchi M, Erthbjerg P. 2018b. Evolution of proteolytic indicators during storage of broiler wooden breast meat. Poult Sci 97:1448-1455.

Sosnicki AA, Wilson BW. 1991. Pathology of turkey skeletal muscle: Implications for the poultry industry. Food Struct 10:317-326.

Strasburg GM, Chiang W. 2009. Pale, soft, exudative turkey - The role of ryanodine receptor variation in meat quality. Poult Sci 88:1497-1505.

Swatland HJ 1990. A note on the growth of connective tissues binding turkey muscle fibers together. Can Inst Food Sci Technol J 23:239-241.

Tasoniero G, Bertram HC, Young JF, Dalle Zotte A, Puolanne E. 2017. Relationship between hardness and myowater properties in wooden breast affected chicken meat: A nuclear magnetic resonance study. LWT-Food Sci Technol 86:20-24.

Tasoniero G, Cullere M, Cecchinato M, Puolanne E, Dalle Zotte A. 2016. Technological quality, mineral profile, and sensory attributes of broiler chicken breast muscles affected by white striping and wooden breast myopathies. Poult Sci 95:2707-2714.

Tijare VV, Yang FL, Kuttappan VA, Alvarado CZ, Coon CN, Owens CM. 2016. Meat quality of broiler breast fillets with white striping and woody breast muscle myopathies. Poult Sci 95:2167-2173.

Trocino A, Piccirillo A, Birolo M, Radaelli G, Bertotto D, Filiou E, Petracchi M, Xiccato G. 2015. Effect of genotype, gender and feed restriction on growth, meat quality and the occurrence of white striping and wooden breast in broiler chickens. Poult Sci 94:2996-3004.

USDA-FSIS, 2017. Disposition instructions for “Woody breast” and “White Striping” poultry conditions. FSIS Notice 35-17, USDA, Washington, DC.

Velleman SG. 2015. Relationship of skeletal muscle development and growth to breast muscle myopathies: A review. Avian Dis 59:525-531.

Velleman SG, Clark DL. 2015. Histopathologic and myogenic gene expression changes associated with wooden breast in broiler breast muscles. Avian Dis 59:410-418.

Velleman SG, Coy CS, Emmerson DA. 2014. Effect of the timing of posthatch feed restrictions on the deposition of fat during broiler breast muscle development. Poult Sci 93:2622-2627.

Voronov E, Shouval DS, Krelin Y, Cagnano E, Benharroch D, Iwakura Y, Dinarello CA, Apte RN. 2003. IL-1 is required for tumor invasiveness and angiogenesis. Proc Natl Acad Sci USA. 100:2645-2650.

Walsh DM, Kennedy DG, Goodall EA, Kennedy S. 1993. Antioxidant enzyme activity in the muscle of calves depleted of vitamin E or selenium or both. Br J Nutr 70:621-630.

Warner RD, Kauffman RG, Greaser ML. 1997. Muscle protein changes post mortem quality traits relation to pork quality traits. Meat Sci 45:339-352.

Wilhelm AE, Maganini MB, Hernández-Blazquez FJ, Ida EI, Shimokomaki M. 2010. Protease activity and the ultrastructure of broiler chicken PSE (pale, soft, exudative) meat. Food Chem 119:1201-1204.

Wold JP, Veiseth-Kent E, Høst V, Løvland A. 2017. Rapid on-line detection and grading of wooden breast myopathy in chicken fillets by near-infrared spectroscopy. Plos One 12:e0173384.
Yost JK, Kenney PB, Slider SD, Russell RW, Killefer J. 2002. Influence of selection for breast muscle mass on myosin isoform composition and metabolism of deep pectoralis muscles of male and female turkeys. Poult Sci 81:911-917.

Zambonelli P, Zappaterra M, Soglia F, Petracci M, Sirri F, Cavani C, Davoli R. 2016. Detection of differentially expressed genes in broiler Pectoralis major muscle affected by white striping - Wooden breast myopathies. Poult Sci 95:2771-2785.

Zhao W, Han Y, Zhao B, Hirota S, Hou J, Xin W. 1998. Effect of carotenoids on the respiratory burst of rat peritoneal macrophage. Biochim Biophys Acta 1381:77-88.