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Review Article

Quality defects of poultry meat – An overview

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Abstract

Rapid genetic selection for accelerated growth, enhanced feed conversion efficiency, and disproportionately large breast muscle yield in modern broilers has unintentionally led to an increased prevalence of breast muscle quality defects collectively termed growth-related myopathies. These include Pale Soft Exudative (PSE)-like condition, Deep Pectoral Myopathy (DPM), White Striping (WS), Spaghetti Meat (SM), Woody Breast (WB), and their combined manifestations (WS+WB, WS+SM). These abnormalities compromise structural integrity, technological properties, water-holding capacity, sensory quality, and consumer acceptability, resulting in significant economic losses across the poultry value chain. Emerging evidence associates these defects with impaired vascularization, hypoxia, dysregulated muscle fibre regeneration, oxidative stress, and fibrosis induced by extreme hypertrophic muscle development. Recent advancements in non-invasive detection technologies, including hyperspectral imaging, near-infrared spectroscopy (NIRS), elastography, magnetic resonance imaging (MRI), and machine learning–assisted phenotyping, are transforming industrial defect-screening capabilities. Mitigation strategies must integrate genetic selection for balanced growth, nutritional modulation (antioxidants, arginine, omega-3 PUFA), and optimized management to reduce growth–vascular mismatch. Future opportunities lie in precision phenotyping, integrative omics, and AI-driven prediction models that support sustainable production without compromising meat quality. This article synthesizes current scientific understanding of the etiology, phenotypic characterization, industrial impact, detection methodologies, and mitigation strategies of breast muscle myopathies, offering a forward-looking perspective for the poultry industry.

Keywords: Broiler myopathies, Pale soft exudative, Spaghetti meat, White striping, Woody breast

Highlights

- Poultry meat quality defects such as PSE, WS, DPM, SM impair quality and consumer acceptance.
- Rapid growth and stress are key drivers of muscle myopathies in intensively raised broilers.
- Meat quality defects cause economic loss through poor yield, texture and processing quality.
- Most defects do not pose food safety risk but reduce market value and consumer trust.
- Genetics, nutrition, management and advanced detection methods may help minimize these defects.

INTRODUCTION

The quality of poultry meat plays a pivotal role in determining consumer acceptance, product value, and long-term viability of the poultry sector. Meat quality is primarily evaluated through sensory and functional traits such as colour, texture, juiciness, tenderness, flavour profile, and nutritional composition. Any deviation from these expected characteristics results in downgrading of meat, economic losses to processors and growers, and reduced consumer confidence (Borpuzari et al., 2025).

Quality deterioration in broiler meat arises from a combination of pre-slaughter factors-including genetic background, feeding strategy, management, bird handling, transport, and physiological stress and post-slaughter variables, such as chilling efficiency, carcass handling, storage temperature, and packaging atmosphere. In recent years, an array of emerging myopathies and quality defects has been documented, notably pale-soft-exudative (PSE) meat, deep pectoral muscle (DPM) myopathy, white striping (WS), spaghetti meat (SM), and woody breast (WB) (Maiorano, 2017). These abnormalities compromise structural integrity, water holding capacity, processing performance, and overall sensory attributes.

With consumers becoming increasingly aware of meat appearance and texture, and regulatory agencies enforcing tighter quality control, managing such defects has become a core objective in modern poultry production. Addressing these abnormalities is essential not only to improve product uniformity and consumer satisfaction but also to reduce wastage and protect the economic sustainability of the poultry value chain.

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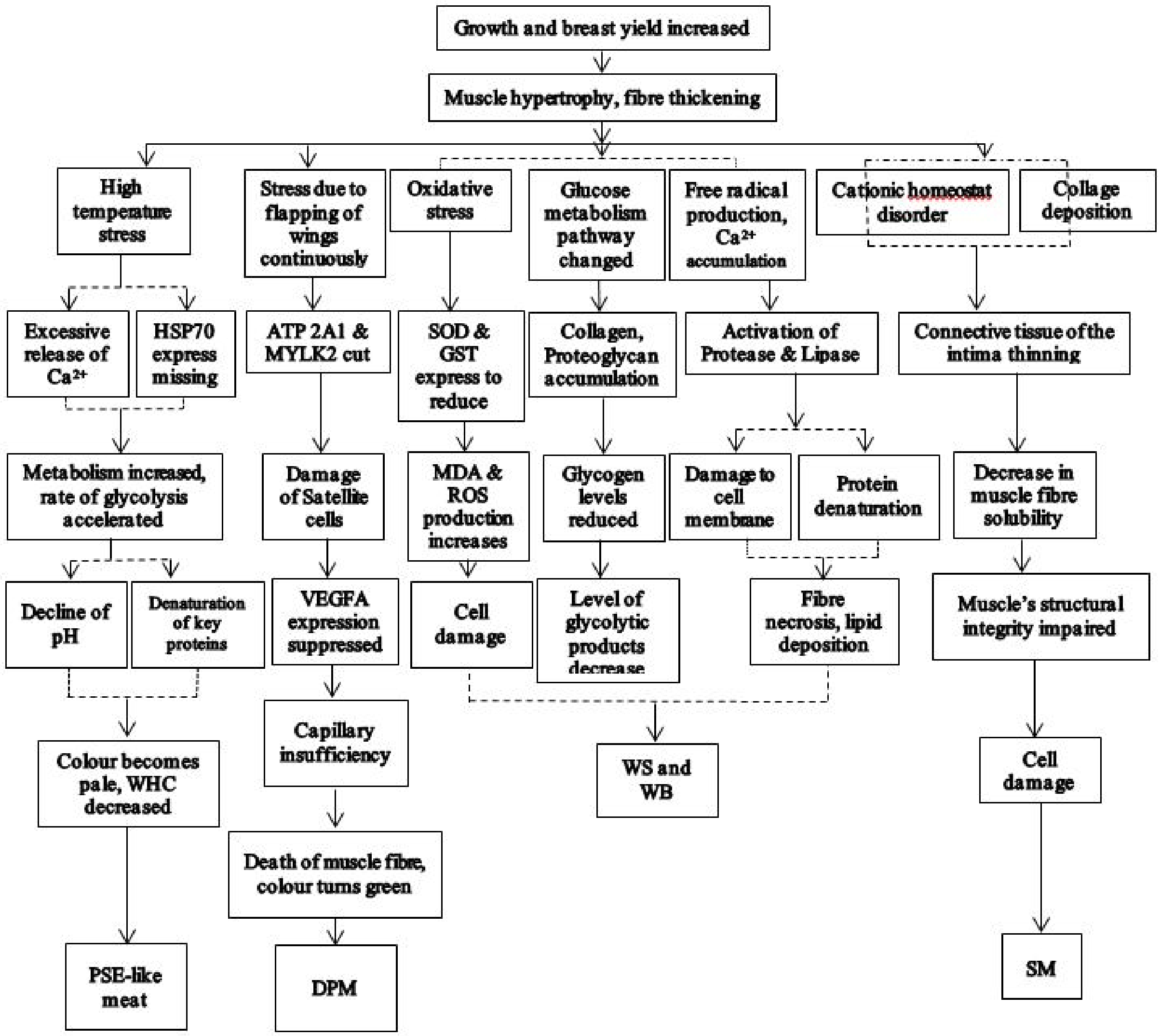

Fig. 1. Mechanisms of formation of abnormal meat (Wang et al., 2023)

Pale soft exudative meat: PSE syndrome represents one of the most prevalent quality defects in commercial broiler meat and is largely attributed to accelerated post-mortem glycolysis triggered by acute pre-slaughter stress. The accelerated growth rate and pronounced muscular hyperplasia characteristic of modern broiler genotypes substantially heighten the susceptibility of pectoral muscle tissue to various stressors, thereby predisposing birds to the development of spontaneous or idiopathic breast muscle myopathies. The mechanistic pathways underlying the emergence of PSE-like meat and other breast meat abnormalities are illustrated in Fig. 1. Among the precipitating factors, acute stress particularly thermal stress and intense physical stimulation is widely recognized as the predominant trigger for PSE-like conditions in broiler meat. Such stressors activate the neuroendocrine axis, modulate immune responses, and perturb normal physiological and metabolic homeostasis, ultimately accelerating postmortem glycolysis and impairing muscle integrity.

The abnormality occurs when birds experience intense physiological stress immediately prior to slaughter - during catching, loading, transport, shackling, electrical stunning, or scalding causing a rapid depletion of intramuscular glycogen and a concomitant surge in anaerobic glycolytic activity (Sandercock et al., 2001; Owens et al., 2009). As a result, lactic acid accumulates excessively while carcass temperature remains elevated, leading to a precipitous decline in muscle pH within the initial hours after slaughter (Barbut et al., 2005; Bianchi et al., 2006). This atypical biochemical trajectory denatures myofibrillar and sarcoplasmic proteins, severely compromising their functionality. PSE meat develops in the breast rather than in the leg meat because significant protein denaturation occurs when the muscle reaches an acidic pH while the carcass temperature is still high. This happens mainly in white muscles, like the breast, which are more glycolytic than the red muscles of the legs. As a result, pm glycolysis proceeds much faster in breast tissue (Feiner, 2006).

The L* (lightness) colour value is of less consistent in poultry meat, especially when PSE meat is identified based on low early post-mortem pH values. Pietrzak et al. (1997) reported higher L* values in PSE turkey meat, whereas other studies (Rathgeber et al., 1999) found no significant differences in L* values between muscles with fast and normal glycolysis rates. On the other hand, research by Zhang and Barbut (2005) in chickens, and Barbut (2007) in turkeys, advocated the use of high L* values (>51-53) as indicators of PSE-like meat, which was characterized by paler colour, reduced WHC, and altered texture. Fraqueza et al. (2006) did not observe a direct relationship between pH at 15 min (pH_{15}) pm and L* values; they, however, did confirm an association between excessively high L* values ($\geq$50) and low ultimate pH values (pH_{24}< 5.8) in PSE-like turkey breast meat.

PSE fillets are typified by pale colouration due to reduced light absorption and increased surface reflectance; soft and flaccid muscle structure with reduced myofibrillar integrity, and markedly low water-holding capacity (WHC), resulting in exudation of fluid during storage or processing (Owens et al., 2000; Barbut et al., 2005; Zhang & Barbut, 2005).

The pathological combination of low ultimate pH ($\leq$5.8) and high protein denaturation facilitates water migration from the intracellular to extracellular spaces, manifesting as drip loss and impaired juiciness (Woelfel et al., 2002; Zhang & Barbut, 2005). These changes adversely affect further-processing performance, particularly in marination, tumbling, emulsification, and cooking leading to yield losses, poor binding, and weakened textural attributes (Woelfel et al., 2002; Barbut, 2002).

Although genetics have been implicated, evidence indicates that PSE in broilers is primarily a functional stress-induced metabolic disorder, unlike the heritable RYR1 mutation-associated PSE found in pork (Woelfel et al., 2002; Huff-Lonergan & Lonergan, 2005). Broiler strains selected for rapid growth demonstrate elevated basal metabolic rates and increased stress susceptibility, thereby predisposing them to acute pre-slaughter stress and subsequent PSE manifestation (Sandercock et al., 2001).

Key physiological basis of PSE development are: (i) Pre-slaughter stress → catecholamine surge → rapid ATP depletion. (ii) Rapid anaerobic glycolysis → excess lactic acid formation. (iii) Early pH drop at high carcass temperature. (iv) Protein denaturation → poor WHC → pale appearance + exudation

The occurrence of PSE results in downgraded carcasses, reduced consumer acceptance due to poor appearance and drip loss,lower processing yields, especially in water-enhanced or further-processed products. Thus, effective control of pre-slaughter stressors and temperature-pH interactions during early post-

Table 1. Processing techniques to improve process ability of PSE meat (Wang et al., 2023)

Processing Technique	Effect	Reference
Adding non-meat ingredients (starch, soy protein isolates, carrageenan, transglutaminase, collagen)	Reduce cooking loss, improve protein binding, and improve gel properties	Lee and Chin (2020); Daigle et al. (2005); Lesiow et al. (2017).
Marination and tumbling	The pale colour and shelf-life of PSE-like meat improve; yield loss reduced	Haddad et al. (2017); Paula et al. (2019).
High-pressure processing (HPP)	The surface hydrophobicity of the protein increased; WHC improved	Chan et al. (2011).
Pulsed electric field (PEF) ·	The rheological property of PSE-like meat protein changed; protein solubility, surface hydrophobicity and active sulfhydryl group increased	Dong et al. (2020).
● pH shifting and low-speed shearing· ● Glycosylation· ●Isoelectric solubilization/ precipitation (ISP) ●Glycosylation	Partially deteriorating protein conformations and dispensability recovered	Yang et al. (2020).

mortem handling is essential to mitigate PSE incidence.

To mitigate the quality deterioration associated with PSE-like broiler meat, a range of technological interventions has been explored to modify its undesirable physicochemical attributes. These approaches are summarized in Table 1 which includes the incorporation of functional auxiliary ingredients such as starches, edible gums, soy protein isolate, carrageenan, and transglutaminase as well as the application of innovative processing technologies and targeted chemical modifications. Current ameliorative strategies encompass non-meat ingredient supplementation, marination and mechanical tumbling, high-pressure processing, high-intensity ultrasound, protein glycosylation, pulsed electric field treatment, isoelectric solubilization–precipitation, pH-shifting techniques, and controlled low-speed shearing.

Deep Pectoral Myopathy (DPM): Also referred to as green muscle disease, DPM is a degenerative ischemic necrosis affecting the *Pectoralis minor* muscles of broilers and turkeys. The pathology originates from the unique anatomical configuration of the *Pectoralis minor*, which is enclosed by rigid fascial sheaths and the sternum, forming a compartment-like space with limited ability to expand during episodes of increased blood flow (Richardson & McCracken, 1979; Bilgili & Hess, 2002).

During vigorous wing flapping induced by stress, handling, transport, or struggling during shackling, there is a sudden increase in oxygen demand and muscle perfusion. However, the surrounding fascia constrains expansion, resulting in intra-compartmental pressure exceeding capillary perfusion pressure. This leads to vascular compression, oxygen deprivation, and ultimately ischemic necrosis (Bilgili et al., 2000; Bilgili & Hess, 2002; Maiorano, 2017). The lesion is characterized macroscopically by a discoloured greenish or yellowish necrotic area, degeneration and fragmentation of muscle fibres, and fibrosis and calcification in advanced cases (Richardson & McCracken, 1979). In lighter birds, this condition often resolves rapidly. In contrast, heavier birds are more prone to prolonged pressure episodes, which can lead to irreversible muscle damage and necrosis (Bilgili & Hess, 2002). Early manifestations of DPM include acute inflammation, characterized by oedema and haemorrhage. As the pathology advances, degenerative changes occur, such as muscle fibre atrophy, segmental and granular degeneration, infiltration by macrophages, connective tissue proliferation, and the presence of eosinophils and phagocytes (Fig. 2). In severe cases, normal muscle fibres are replaced by fibrous and adipose tissue, accompanied by a colour change from pink to green (Bianchi et al., 2006).

Histopathologically, DPM is marked by coagulative necrosis, infiltration of heterophils and macrophages, and regenerative attempts in peripheral zones, while necrotic cores undergo liquefaction or fibrotic replacement (Sihvo et al., 2014).

Predisposing factors include high-yield genetic strains selected for rapid breast muscle accretion (Bilgili & Hess, 2002), excessive bird activity prior to slaughter - particularly in heavy broilers and breeders, wing flapping during manual catching and shackling, stress from transport, noise, lighting intensity, or rough handling. Unlike other quality defects such as PSE, DPM is not associated with post-mortem biochemical shifts but with ante-mortem vascular obstruction and ischemia.

Although DPM seldom affects the *Pectoralis major* (breast fillet), it leads to partial or complete trimming of the affected minor muscle, condemnation of severely affected carcasses, and yield and revenue loss, particularly in premium whole-muscle and deboned products (Kijowski & Konstañczak, 2009; Maiorano, 2017). In high-volume processing plants, the condition may remain undetected until deboning, where the characteristic discolouration prompts downgrading.

The occurrence of DPM can vary significantly based on age and breed of the bird. For example, Bianchi et al. (2006) found that 0.84% of carcasses were affected by DPM, but total cases could range from 0 to 16.7%. Early-stage DPM varied between 0 and 12.0%, while late-stage cases ranged from 0 to 5.6%. According to Lien et al. (2011), spontaneous DPM cases ranged from 3 to 17% and were more common in birds that grow faster than average. The DPM incidence in commercially slaughtered chickens in Poland ranged between 0.02 and 1.9% (Kijowski et al., 2014).

Mitigation strategies

Reduction in DPM occurrence relies on minimizing pre-slaughter excitement and excessive wing activity through optimized lighting and environmental control during catching and transport, training of catching crews, controlled handling and shackling techniques, use of automated harvesting systems to reduce wing flapping.

Pink Colour: The pink colour defect refers to the persistent or reappearing pinkish hue observed in fully cooked poultry products, leading consumers to falsely perceive the product as undercooked or unsafe (Fig. 3). Although colour change during cooking is fundamentally associated with myoglobin denaturation, this defect is multifactorial and arises due to complex biochemical interactions involving muscle pigments, processing conditions, and storage variables (Hunt et al., 1999; Claus & Jeong, 2018). During thermal processing, myoglobin typically undergoes stepwise structural denaturation, transitioning from its native state to globin-haemochrome forms, which then convert to brownish metmyoglobin derivatives at high internal temperatures (>74°C). In defective cases, however, the pigments either do not fully denature or re-associate with nitric oxide- or carbon monoxide–reactive compounds, generating heat-stable porphyrin complexes that impart a pink colouration even after reaching microbiologically safe temperatures (Ahn & Maurer, 1987; Barbut, 2015).

Several intrinsic and extrinsic determinants contribute to this anomaly

Intrinsic factors- Low myoglobin concentration in poultry muscle makes pigment transformations highly sensitive to environmental and processing conditions (Cornforth & Jayasingh, 2004). Variations in ultimate pH, particularly elevated pH (>6.0), increase myofibrillar water retention, reduce light scattering, and stabilize pink hues (Jeong & Claus, 2010). Antioxidants present either naturally or *via* dietary supplementation may inhibit pigment oxidation, maintaining residual pink colour (Ahn & Maurer, 1987).

Extrinsic and technological factors- Carbon monoxide exposure, even in trace amounts during gas-assisted packaging or incomplete combustion, forms CO-myoglobin complexes resistant to heat denaturation (Cornforth, 1994). Addition of ingredients such as phosphates, reducing sugars, or reducing agents (e.g., ascorbates) may enhance the development of heat-resistant porphyrins (Trout, 1989; Suman et al., 2010). Rapid chilling post-cooking can trap partially denatured pigments before full thermal conversion.

Furthermore, pink discolouration is distinct from food safety concerns. Numerous studies confirm that fully cooked meat exhibiting pink colouration nonetheless meets microbiological safety standards when internal temperatures reach recommended thresholds (Hunt et al., 1999; Claus & Jeong, 2018). The issue, therefore, is perception-driven, impacting consumer confidence and leading to product rejection or returns. Industrial implications include consumer misinterpretation of pinkness leads to complaints and reduced market acceptance, and manufacturers may resort to overcooking to eliminate visible pink colour, diminishing product juiciness and tenderness and reducing cooking yields (Claus & Jeong, 2018).

Mitigation strategies

Optimization of thermal processing schedules to ensure uniform heat penetration, pH control through ingredient formulation, avoidance of CO/NO exposure and careful monitoring of smoking or grill systems and controlled chilling to prevent stabilization of partially denatured pigments may significantly reduce the occurrence of pink discolouration of poultry meat.

White Striping (WS): WS is a myopathic abnormality of the *Pectoralis major* muscle, visually characterized by white, parallel striations oriented along the longitudinal axis of the breast fillet (Fig. 4). These striations represent intermuscular and perimysial lipid and connective tissue deposition, replacing normal contractile fibres (Kuttappan et al., 2012a; Velleman & Clark, 2015; Petracci et al., 2019).

Etiopathogenesis: The disorder is strongly linked to intense genetic selection for accelerated growth rate, hypertrophic breast muscle development, and improved feed efficiency in modern broiler strains (Petracci & Cavani, 2012). Rapid muscle accretion outpaces the capacity of the vascular system to supply adequate oxygen, resulting in localized hypoxia, oxidative stress, and metabolic dysregulation (Sihvo et al., 2014). This induces degeneration and lysis of muscle fibres, infiltration of inflammatory cells, fibroblast proliferation, and progressive fibrosis and adipogenesis (Kuttappan et al., 2013a; Alnahhas et al., 2016).The severity of WS is commonly categorized as normal (score 0), moderate (score 1), and severe (score 2 or 3) based on thickness and distribution of white striations (Kuttappan et al., 2012a). Severe WS is frequently co-associated with other emerging myopathies such as woody breast (WB) and spaghetti meat (SM) (Petracci et al., 2019).

Physiological implications: Histological analyses consistently show loss of myofibre architecture, perivascular and perimysial lipid accumulation, fibrosis, and regeneration attempts with small diameter myofibres (Sihvo et al., 2014; Clark & Velleman, 2016). These alterations are indicative of chronic myodegenerative pathology, distinguishing WS from acute stress-related anomalies such as PSE.

Table 2. White Striping and its impact on meat quality

Attribute Affected	Consequences
Water-holding capacity (WHC)	Reduced WHC → increased drip and cook loss (Mudalal et al., 2014)
Texture and mouthfeel	Perceived as tougher, less cohesive (Petracci et al., 2019)
Nutritional profile	Increased fat and decreased protein content (Petracci & Cavani, 2012)
Consumer acceptance	Lower visual appeal; product rejection (Kuttappan et al., 2012b)

Table 3. Woody Breast and its impact on meat quality

Quality Aspect	WB Effect
Texture & Palatability	Fillet becomes rigid, chewy, and rubbery (Petracci et al., 2019)
Water-Holding Capacity	Reduced WHC → high drip and cook loss (Mudalal et al., 2015)
Functional Traits	Poor marinade uptake, reduced binding, impaired slicing and portioning

Impact on meat quality: Although WS has limited effect on overall safety, it negatively influences technological, sensory, and nutritional attributes (Table 2).

Consumers frequently associate WS with inferior meat quality due to visible abnormal striations, resulting in reduced purchase intent (Kuttappan et al., 2012b).Rapid growth rate and genetic strain predisposition, greater market age and heavier final body weight, elevated breast muscle yield percentage and imbalance between muscle hypertrophy and microvascular development are the risk factors associated with WS.WS leads to downgrading of fillets,decreased yield in marinated and further-processed products, andincreased trimming losses.Given its growing prevalence in modern broiler production, WS represents a major quality and economic challenge for primary processing plants and downstream industries.

Woody Breast (WB)

WB is a chronic degenerative myopathy affecting the *Pectoralis major* of fast-growing broilers. The abnormality is characterized by marked firmness or hardness of the cranial region of the breast fillet, loss of elasticity, and a distinctive bulging appearance (Fig. 5). WB is frequently palpable at slaughter and persists post-processing, indicating profound structural alterations of the muscle tissue (Kuttappan et al., 2016; Velleman & Clark, 2015; Petracci et al., 2019).

Pathophysiological basis: WB is strongly associated with extreme selection pressure for rapid breast muscle hypertrophy and feed efficiency, which creates a mismatch between muscle growth and supportive tissue development (Petracci & Cavani, 2012; Sihvo et al., 2014). As muscle mass rapidly increases, capillary density becomes insufficient to meet oxygen demand, tissue oxygenation decreases, and localized hypoxia and oxidative stress develop (Sihvo et al., 2014; Avci-Kupeli, 2025). The resulting metabolic disturbance triggers myofibre degeneration and necrosis, fibroblast proliferation, and replacement of muscle tissue by dense connective tissue (fibrosis) and adipose infiltration (Velleman & Clark, 2015; Soglia et al., 2021). In severe WB fillets, palpable hard knots or ridges form along the muscle surface, and haemorrhages or serous exudates (gel-like secretion) may appear on cut surfaces (Kuttappan et al., 2016).

Microscopic lesions include myofibre fragmentation and hyalinization, lipidosis and localized adipocyte accumulation, increased collagen deposition and excessive connective tissue cross-linking, fibrosis replacing functional muscle fibres (Soglia et al., 2017; Clark & Velleman, 2016). WB therefore represents a fibrotic, collagen-rich muscle phenotype, distinct from WS or SM.

Implications on meat quality: WB severely compromises the technological and sensory properties of breast meat (Table 3).

In severe cases, fillets lose flexibility and crack when folded - a hallmark diagnostic attribute. WB is seen primarily in market-ready broilers of high breast yield phenotypes, heavier body weight and older slaughter age, and rapid early growth curve with excessive protein accretion (Kuttappan et al., 2016; Petracci et al., 2019).

Economic consequences: WB is one of the most costly muscle defects in modern poultry production. Processing plants face downgrading of fillets, increased trimming and yield loss, and redirection to low-value comminuted products due to poor whole-muscle texture. Industry estimates indicate substantial economic losses globally due to WB prevalence in heavy broiler lines.

Table 4. Effects on technological, sensory, and nutritional properties on Woody Breast and White Striping

Attribute	WB + WS Combined Effect
Texture	Very tough, rubbery, inflexible surface
Water-holding capacity	Lowest WHC among myopathies, high drip and cook loss
Appearance	Highly visible white striations+surface bulging → strong consumer rejection
Nutritional composition	Increased fat, reduced protein, lower nutritional value (Petracci & Cavani, 2012)

Table 5. Processing techniques to improve process ability of WS and WB meat (Wang et al., 2023)

Processing Technique	Effect	Reference
Blade tenderization	The cooked meat texture of WB meat improved	Tasoniero et al. (2019)
Marination and tumbling	The hardness and chewability of WB meat reduced; the WHC of WS meat improved	Maxwell et al. (2018); U-Chupaj et al. (2021)
Ultrasound	Economic losses of WB meat reduced	Li et al. (2021)

Woody Breast and WS frequently occur concurrently in modern broilers and are now recognized as different pathological expressions of a shared degenerative myopathy linked to accelerated breast muscle hypertrophy (Sihvo et al., 2014; Petracci et al., 2019). While each condition presents distinct morphological features -firmness and fibrosis in WB, and lipid and connective tissue infiltration in WS, their simultaneous manifestation indicates a more advanced, chronic muscular degeneration.

Pathophysiological relationship: Rapid growth and intense selection for high breast yield create a physiological imbalance where muscle accretion outpaces vascular development, oxygen delivery capacity, and ECM organization (Petracci & Cavani, 2012; Kuttappan et al., 2016). As a result localized ischemia and oxidative stress induce myofibre necrosis (Sihvo et al., 2014), regenerative attempts are inadequate, and muscle tissue is gradually replaced by fibrous and adipose tissues (Velleman & Clark, 2015).

When WS and WB co-exist, the muscle exhibits both fibrosis, stiffness, rigid texture (WB), and white lipid–rich striations (WS). This dual presentation signifies severe phenotypic expression of breast muscle myopathy. The combined defect is more prevalent in heavier broilers (>3.0 kg), older slaughter ages, high-breast-yield genetic lines (Kuttappan et al., 2016; Petracci et al., 2019). Severity grading often shows: Mild WS → early ECM remodeling, and Severe WS + WB → advanced fibrosis and collagen deposition (Soglia et al., 2021).

Thus, WS can be viewed as a precursor to WB in many birds, although progression is not strictly linear. Effects on technological, sensory, and nutritional properties are presented in Table 4.

The co-occurrence of WB and WS significantly worsens marinade uptake, binding in processed products,slicing/portioning performance, and cooking yield (Mudalal et al., 2015).

Industrial and economic impact: Products exhibiting both WB and WS are frequently downgraded or diverted to comminuted or further-processed channels due to unacceptable visual defects, poor texture, and impaired functionality. This increases trimming losses, processing costs, and raw material inefficiencies in high-value product streams. The combined occurrence is currently regarded as one of the most economically damaging meat quality challenges in the poultry industry.

In contrast to PSE-like meat, the range of technological interventions available to enhance the quality of WS- and WB-affected meat remains limited. As presented in Table 5, only three techniques, namely blade tenderization, marination with or without tumbling, and ultrasound treatment have been systematically evaluated for their potential to improve the characteristics of WB meat.

Spaghetti Meat (SM)

SM is an emerging myopathic disorder of broiler *Pectoralis major* muscle characterized by a loss of structural cohesiveness and a stringy, loosely fragmented muscle appearance resembling "strands of spaghetti" (Fig. 6).The condition primarily involves severe degradation of the perimysial and endomysial connective tissue network, resulting in reduced structural integrity and easy separation of muscle fibre bundles (Petracci et al., 2019; Baldi et al., 2021).

Etiopathogenesis and histopathology: Current evidence suggests that SM arises from a dysregulated balance between muscle fibre hypertrophy and extracellular matrix (ECM) organization. Modern

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Fig. 2. DPM myopathy in broiler breast (Adapted from Huang and Ahn, 2018)

a. Early stage of DPM showing muscle damage and haemmorhage

b. Late stage of DPM showing muscle greening of deep pectoral muscle

Fig. 3. Pink colour of poultry meat

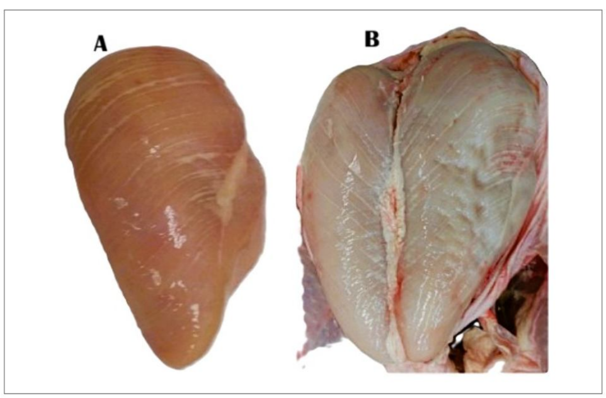

Fig. 4. WS on a breast muscle (A) and on the whole carcass (B) (Adapted from Huang and Ahn, 2018)

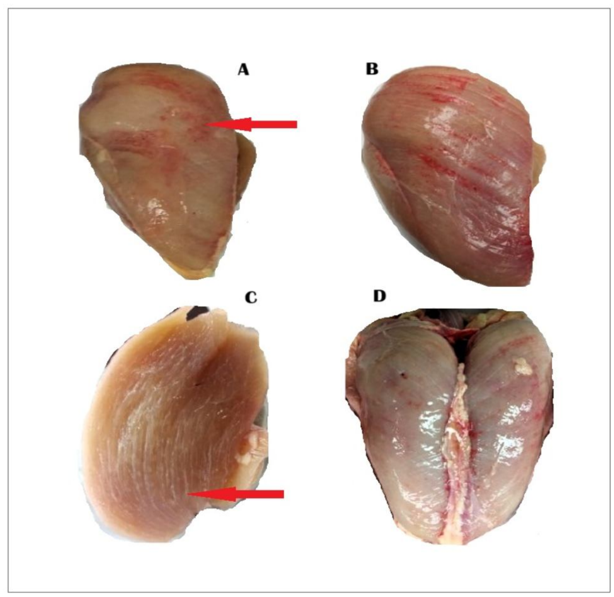

Fig. 5. Wood Breast conditions on a breast muscle and whole carcass (Adapted from Huang and Ahn, 2018)

a. WB conditions on a carcass showing white stripes, haemorrhage

b. WB with WS: showing bulged area, white stripes and haemorrhage

c. Cross section of WB muscle: white lines (arrow) are signs of firbrosis and lipidosis haemorrhage

d. WB showing bulged area (arrow) and haemorrhage

Fig. 6. Spaghetti meat in broiler (Adapted from Maiorano, 2017**)**

broiler genotypes, selected for rapid breast muscle growth, exhibit increased myofibre diameter without proportional development of capillary density and supporting connective tissue. This creates localizedhypoxia and metabolic strain, predisposing the muscle to myodegeneration (Sihvo et al., 2014; Clark & Velleman, 2016; Soglia et al., 2021).

Histological features of SM includemarked disruption and thinning of perimysial collagen fibres,loosening and separation of muscle fibre bundles (Petracci et al., 2019),edema and infiltration of inflammatory cells,reduced collagen crosslinking and decreased structural integrity of the ECM (Soglia et al., 2020). Unlike PSE or pink colour defect, SM represents a chronic degenerative pathology rather than an acute stress-related metabolic shift.

SM frequently co-occurs with white striping (WS), and the two conditions represent different manifestations of the same underlying myodegenerative process.WS is dominated by lipid infiltration and fibrosis while SM is characterized by ECM degradation and muscle fibre separation (Petracci et al., 2019; Soglia et al., 2021). Both disorders originate from rapid muscle hypertrophy, insufficient vascularization, oxidative stress and myofibre degeneration (Petracci & Cavani, 2012; Kuttappan et al., 2013b). Thus, WS and SM are not independent conditions; they represent concurrent pathological outcomes of accelerated breast muscle accretion.

Effects on technological and sensory quality: SM imposes significant challenges to meat processing due to markedly reducedwater-holding capacity,poor binding ability and loss of cohesiveness during marination or tumbling, reduced cooking yields and texture defects (Soglia et al., 2020).From a consumer standpoint, the stringy and irregular appearance leads to strong negative perception and rejection at point of purchase (Mudalal et al., 2014).

Increased downgrading and trimming losses, reduced suitability for whole-muscle retail cuts and forced diversion toward low-value processed products are some of the economic implications of SM. Due to its impact on structure and texture, SM is considered more detrimental to processing yields than WS alone.

Breast muscle myopathies not only alter surface colour and texture but also compromise the functional properties of myofibrillar proteins. The quality effects associated with each abnormality are detailed in Table 6, with all defects producing some degree of impairment to meat functionality. Given their significant economic ramifications, these myopathies

Table 6. Effect of broiler muscle myopathies/ abnormalities on sensory and functional properties of muscle and proteins (Wang et al., 2023)

Item	Appearance	Quality Characteristics	Meat Gel Quality	Reference
Deep Pectoral Muscle	Muscle colour turns green	Z-line disintegration; action filaments decomposition; nuclear and organelle destruction	Not investigated	Barbut et al., 2019
Pale-soft-exudatives like broiler meat and	Pale, soft, exudative	Proteins solubility declines; myosin thermal stability and pH decline	Gel structure and water holding capacity of salt soluble protein decrease	Chen et al. (2018); Dong et al. (2020)
Wooden Breast	Texture hardness, the caudal area has a ridge protrusion	Protein content and water holding capacity (WHC) declines; hardness, pH, water content and crude fat content increased	Gel microstructure loose; have a higher á-helix content, lower â sheet and â-turn content	Baldi et al. (2019); Soglia et al. (2020); Liu et al. (2011)
White striping	White fat deposits	Same as Woody Breast	Same as Woody Breast	Kuttappan et al. (2016)
Spaghetti meat	Impaired muscular integrity, connective tissue sparse	Muscle pH, WHC, and protein solubility declined; emulsion stability impaired	Not investigated	Baldi et al. (2018); Tasoniero et al. (2020)

are highly relevant to the poultry industry. Numerous studies have aimed to elucidate the etiological mechanisms of broiler breast muscle abnormalities, with current evidence suggesting that some of these conditions may originate from underlying, potentially heritable metabolic disorders.

Detection and diagnostic approaches: Early and accurate identification of PSE, DPM, WS, SM, and WB remains critical because most abnormalities are not externally visible until deboning. Conventional assessments like visual inspection and palpationare subjective and operator dependent. Thus, the poultry sector is transitioning toward objective, quantitative, and non-destructive diagnostic systems (Mudalal et al., 2014; Soglia et al., 2021).

Instrumental and imaging-based detection: (i) Colourimetry and reflectance spectroscopy quantify surface colour, lightness (L*), and pH-related optical changes, enabling accurate detection of PSE meat (Barbut, 2015; Suman et al., 2016). (ii) Near-infrared (NIR) spectroscopy and hyperspectral imaging (HSI) provide spatially resolved information on muscle composition (water, protein, and lipid distribution), enabling classification of WS, WB, and SM with high precision (Xiong et al., 2017; Xing et al., 2020). (iii) Magnetic resonance imaging (MRI) and ultrasound-based elastography quantify structural stiffness and collagen deposition, facilitating non-invasive identification of woody tissue (Wold et al., 2017; Yalcin et al., 2019).

Machine learning and Artificial Intelligence (AI): Integration of imaging with AI classifiers has yielded automated defect recognition systems capable of predicting WS/WB severity on fast-moving processing lines (Wold et al., 2017; Soglia et al., 2021). Deep learning models are increasingly used tograde severity (mild/moderate/severe),map affected areas, andsupport carcass sorting during high-throughput deboning. AI-enabled detection is emerging as a future industry standard for quality screening.

Mitigation and management strategies

Since WS, SM, and WB are tightly linked toexcessive breast muscle hypertrophy and impaired vascularization, mitigation measures must addressgenetic, nutritional, and management factors (Petracci et al., 2019).

Genetic and breeding interventions: Incorporation of breast myopathy scoringinto genetic selection programmes, prioritizing balanced growth traits instead of extreme breast yield (Petracci & Cavani, 2012) and selection for vascular development markers, oxidative capacity, and improved microcirculation are the genetic approaches in mitigation of the breast muscle myopathy in broilers. Genetic strategies reduce population-level susceptibility but require multiple selection cycles.

Nutritional strategies: Supplementation with antioxidants (e.g., vitamin E, selenium) educes oxidative stress and improves muscle integrity (Abasht et al., 2016). Use of arginine, omega-3 fatty acids, and phytogenics enhances vasodilation and capillary perfusion.Controlled energy-protein balance during early growth reduces the hypertrophy-vascularization mismatch.

Management strategies: Moderation of early growth curves (e.g., phase feeding, feed restriction, lighting programmes), optimal stocking density, ventilation, and stress reduction prior to harvest to limit PSE and DPM occurrence are useful management strategies to reduce occurrence of these myopathies.

Processing interventions: Electrical stimulation and rapid chilling reduce PSE by balancing pH-temperature decline (Barbut, 2002). Optimized deboning schedules improve product appearance and reduce mechanical damage. Since no single intervention eliminates defects, a multifactorial control strategy is required.

Future perspectives: The increasing global demand for lean meat, combined with decades of selection for growth rate and breast yield, has unintentionally intensified muscle myopathies. Future sustainability of poultry production will require system-level innovation, emphasizing: (i) Precision phenotyping integrated with genomics, metabolomics, and transcriptomics to identify early predictors of myopathy (Abasht et al., 2016). (ii) Real-time defect prediction *via* AI-enabled online sorting systems. (iii) Development of rapid point-of-processing screening tools (portable NIR or handheld ultrasound). (iv) Refinement of breeding goals toward functional muscle quality instead of only yield.

Conclusion

Meat quality defects such as PSE, DPM, pink colour, WS, SM, and WB represent major challenges for the poultry industry, affecting not only the functional and sensory properties of meat but also its economic value and consumer acceptance. These conditions arise from a complex interplay of genetic, physiological, nutritional, and environmental factors, with fast growth rates and intensive production systems being important contributors. While most of these defects do not pose direct food safety risks, they compromise technological properties, reduce processing yields, and diminish consumer confidence in poultry products.

Addressing these challenges requires a holistic approach that combines genetic selection, optimized nutrition, effective management practices, stress reduction, and the adoption of innovative processing and detection technologies. Moreover, ongoing research into the molecular, metabolic, and structural mechanisms underlying these defects will be crucial in developing sustainable prevention and mitigation strategies. By minimizing the occurrence of such abnormalities, the poultry sector can improve product quality, enhance consumer satisfaction, reduce economic losses, and strengthen the long-term sustainability of the global poultry meat industry. A 'One Health' perspective linking poultry welfare, economic viability, and consumer expectations will be central to future poultry production and marketing systems. Emerging abnormalities such as WS, SM, and WB highlight the complex trade-off between growth efficiency and muscle quality. The integration of non-destructive diagnostics, precision breeding, and optimized nutrition/management strategies offers a feasible path toward controlling quality defects without compromising production efficiency.

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