

Review

Broiler White Striping: A Review of Its Etiology, Effects on Production, and Mitigation Efforts

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Abstract: With an increase in the demand and production of chicken products, broilers have been bred to grow larger at a faster rate. This has led to several myopathies, one of which being white striping, which is now common to almost all broilers and is characterized as the deposition of fat within the broiler breast muscles. The purposes of this review are to examine the change in chicken production over the years, and to consider normal muscle growth physiology and the suspected pathological process and causes of white striping, as well as white striping visualization and detection in fast-growing broilers. With the increase in growth rate and size, multiple biochemical processes are affected in broilers, leading to hypoxia and inflammation, subsequent formation of foam cells from macrophages, and the deposition of fat in the form of white stripes along the muscle tissues. The negative effects of white striping include increased fat content and calories and decreased protein content, as well as reductions in palatability and drip loss. Thus, this review also explores the effects of white striping on meat quality, and finally evaluates several strategies attempted to mitigate white striping.

Keywords: poultry; broiler production; muscle growth; white striping; meat quality



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1. Introduction

The annual broiler production has increased in the United States by approximately six-fold over the last 50 years. Approximately 60 billion pounds of live weight broilers were produced in the year 2021 [1]. The increased production is due to the increased demand and consumption of poultry products—up from 15.5 kg per capita in 1960 to 51.4 kg per capita in 2021 in the United States [2]. In order to fulfill this increased need for poultry products, broilers were bred to grow faster with greater yields using genetic selection. In 1925, broilers reached a market weight of 1.13 kg in 112 days; in 2021, broilers reached a market weight of 2.93 kg in 47 days [3]. Although this fast growth was meant to fulfill market expectations, it eventually led to unwanted pathological changes, such as white striping in meat. White striping (WS) is the deposition of fat along the muscle fibers developing during a bird's growth, mainly cranially to caudally, in the direction of the myofibers of the pectoralis major muscles. Other broiler breast myopathies include wooden breast, spaghetti meat, deep pectoral myopathy, and pale, soft, and exudative meat, and they are also associated with fast-growing broilers. Kuttappan et al. [4] have shown that white striping has an incidence of 90% in all chicken breast products. White striping studies have included the investigation of phenotypes at the histological, pathological, and biochemical and biomolecular levels. Researchers identified cells, gene markers, and other molecular species that contributed to the presence of white striping, even though no treatment has been discovered yet [5]. In this literature review, we will describe (1) how chicken production has changed over the years to ultimately generate larger birds more quickly; (2) muscle growth physiology; (3) the suspected pathological process of white striping; (4) the causes of white striping; (5) how white striping is visualized, detected, and measured; (6) the effects of white striping on meat quality and production; and (7) strategies attempted to mitigate white striping and outcomes from their applications.

2. Recent History of Chicken Production

Before the establishment of the industry in the twentieth century, poultry production was based upon individual households managing their own flocks of chickens. Moreover, production was limited due to a lack of knowledge about those factors that have the greatest impacts upon efficiency (e.g., housing, feed, sunlight and vitamin D, etc.). Thus, chickens were raised primarily for egg production as a source of income. Chicken meat was only a byproduct of egg production and was considered a delicacy that would only be consumed on special occasions [6].

Early efforts at broiler line development began in the 1920s in the United States. Confined housing and the caging system for birds were developed, which improved production and decreased mortality rates [7]. Then, broiler production became more prevalent in areas such as New England, Arkansas, and Georgia. In the 1940s, broiler production was bolstered by an increased demand to feed troops in World War II. People were also encouraged to raise their own chickens, as they were easy to raise and had a high production value. At that point in time, hatcheries, farms, feed mills, and processors were all separate entities; however, as businesses began working together, an integrated industry grew. This made the production of chicken products more efficient and profitable. Moreover, chicken sales went from whole carcasses to offering specific cuts that were fabricated and packaged as ready-to-cook. Nevertheless, broilers were still not included as a separate statistic for chicken data in the 1949 USDA reports [7].

In the 1950s, poultry meat was mainly fabricated from broilers—no longer as a delicacy. Moreover, the USDA recognized the importance of the broiler industry and introduced a program in which the quality of broiler products was monitored. At this time, the National Broiler Council was organized. Because of efficient production with minimal labor, 90% of chicken products came from the integrated operations. In the 1970s, advancements in genetics, nutrition, medicine, and mechanization further advanced broiler production in the United States. Chicken surpassed beef and pork as the top selling meat in 1992. The United States also started exporting chicken products to other countries with billions of dollars of revenue, and the USDA initiated more programs and protocols to monitor quality control and reduce production hazards [6,8].

Advancements in chicken nutrition also contributed to improvements in broiler growth. Prior to the twentieth century, chickens were fed household scraps, such as vegetables, and were expected to find insects [9]. Books published in the early 1910s provided guidance into feeding chickens with the introduction of the concept of feeding grains [10]. Today, chickens eat a mix of starch-rich grains, such as corn and protein-rich legumes, such as soybeans, as well as oilseeds, vitamins, and minerals [11]. Starter, grower, and finisher feeds have been adapted to optimize bird nutrition at different stages of life. The feed ingredients for broilers and layers are similar; however, as meat birds, broilers require more protein and energy to bolster their growth rates and yields. Feed efficiency also increased as a result of these dietary changes. In 1985, 3.22 kg of feed was needed to produce a 1.40-kg broiler. In 2010, 3.66 kg of feed was needed to grow a 2.44-kg broiler [12]. In 2001, the breast muscle weights for Ross 308 were 8.4, 9.9, 10.3, and 9.8% higher than those in 1957 [13].

Genetic selection is another reason that today's broilers grow larger at a faster rate. Producers select for broilers that grow faster, with better immunity and feed efficiency. The United States produces approximately 8.6 billion broilers every year, which has made it easy for the industry to select from a large pool of chickens for breeding. Advances in reproduction and DNA technology have also made the breeding process significantly faster [14]. Because of the short life of broilers, they grow expeditiously. Moreover, Kuttappan et al. [15] investigated the relationship between the growth rate of broilers and their degree of white striping. They found that broilers who were fed a high-energy diet for growth demonstrated more severe degrees of white striping, suggesting a positive correlation between a high growth rate and the appearance of white striping. Furthermore, according to Alnahhas

et al. [16], white striping is also highly heritable, with h^2 of 0.65 ± 0.08 . Thus, like with advances in nutrition, selection for rapid growth has heightened the risk for white striping.

3. Muscle Growth Physiology

Muscle growth is defined by both hypertrophy and hyperplasia, where hyperplasia happens in ovo, thus the number of muscle fibers a chick can have is established before hatching. During development, muscular tissue is formed in myogenesis, in which, after cell proliferation and differentiation, the myoblasts fuse to become myofibers [17]. Post-hatching muscle growth occurs via increases in muscle fiber size that occur through the active satellite cells fusing to the muscle fibers [18].

After the somite specification and the determination of the sclerotome, different regions of the somite will then develop into their own respective derivatives. The ventral-medial portion of the somite gives rise to bones and cartilage, the dermatome gives rise to the skin and connective tissues, and the myotome gives rise to the skeletal muscle. Through different series of signaling and inhibition, myogenesis takes place [19]. Some somites diverge into myotome cells; then, myogenic bHLH (basic helix-loop-helix) proteins, such as transcription factors MyoD and MRF5, bind to muscle-specific genes to promote muscle differentiation. Thus, myotome cells start to produce the myogenic bHLH proteins and become committed muscle precursor myoblasts. The myogenic bHLH proteins also help with myoblast proliferation, specifically the fibroblast growth factors. After a few rounds of proliferation, the myoblasts undergo differentiation. The differentiation of some myoblasts can also signal other myoblasts to differentiate. Muscle cells begin to fuse to form fibers; fused cells must leave the cell cycle. During fusion, the cells align into chains by force generated by actin-based structures, eventually forming the multinucleated myotubules of the muscle [20]. The fusion of myoblasts activates myogenin—another bHLH protein—which then mediates the differentiation of other muscle cells. The myoblasts and muscle fibers are held together by fibroblasts, which can help arrange and orientate the muscle cells. Myoblasts then fuse through signaling pathways, such as the β 1D-integrin-FAK pathway, the MEK5-ERK5-SP1-Klf2/4-Npnt pathway, and the NFATc2-IL-4 pathway [21]. After fusing, the skeletal muscle fiber can then develop, mature, and form different isoforms, such as type I and type II muscle fibers [22,23]. The formation of muscle fibers is completed in ovo. Then, muscle fiber growth after hatching is caused by hypertrophy from satellite cell nuclei recruitment, which causes the enlargement of muscle fibers [24].

Muscles have vasculature and innervation. The vasculature maintains homeostasis for the muscles by providing the key nutrients and removing waste products, and it is also crucial in activating muscle regeneration [25]. In recent studies, it was shown that endothelial cells of the blood vessel can regulate myogenesis and myogenic cell expansion [26]. Nerves are involved in muscle movement and closely correlate to muscle growth, as well. Thus, nerve damage can lead to muscle atrophy, which is also known as neurogenic atrophy [27]. Muscles that are no longer innervated have functional deficits, no longer contract anymore, and can degenerate [28].

4. The Suspected Pathological Process of White Striping

White striping can be seen grossly as white lines extending down along with the muscle fibers. Samples with severe white striping demonstrated signs of tissue degeneration. White striping manifests from the progressive deterioration of the muscle structure as dysfunction occurs in physiological processes of muscle metabolism, growth, and vascular homeostasis (Figure 1).

White striping is associated with vascular inflammation and macrophage infiltration as the fast growth of the pectoralis major muscle disrupts its metabolism and homeostasis [29]. Hypoxia is also involved in the formation of the white stripes. In hypoxic conditions, there is a reduction in the production of ATP in the cells, as there is no oxygen input for cellular respiration, which also affects the cells' abilities to control the influx and efflux of ions such

as sodium and potassium, as there is an inhibition of the energy-dependent sodium and potassium pumps, and ion gradients are disturbed. Excess sodium can result in vacuole formation, which can be observed histopathologically [30]. Dysregulation in ion gradient homeostasis leads to cell swelling, cell rupture, and necrosis [31]. Muscle satellite cells, which play an important role in muscle regeneration and repair, cannot proliferate, as the tissues are growing too fast, leaving no room for vascularization, which leads to necrotic lesions [32,33]. Satellite cells can produce collagen, which can lead to the fibrosis seen in the affected tissues [34]. The inflammation of the tissues is also related to acetyl-CoA accumulation, where it can cause β lipid oxidation impairment, leading to increased fat accumulation [35,36]. With inflammation, as well as lipid accumulation, the white blood cells will migrate to the tissue of interest from the basement membrane, and through the blood vessel walls after inside-out signaling and integrin activation [37]. The macrophages will phagocytose the dead cells [38] and can also ingest the lipids and become lipid-laden foam cells, which contribute to the visible white striping [39]. Foam cells can also release cytokines, which will recruit more immune cells, exacerbating the foam cell formation [40].

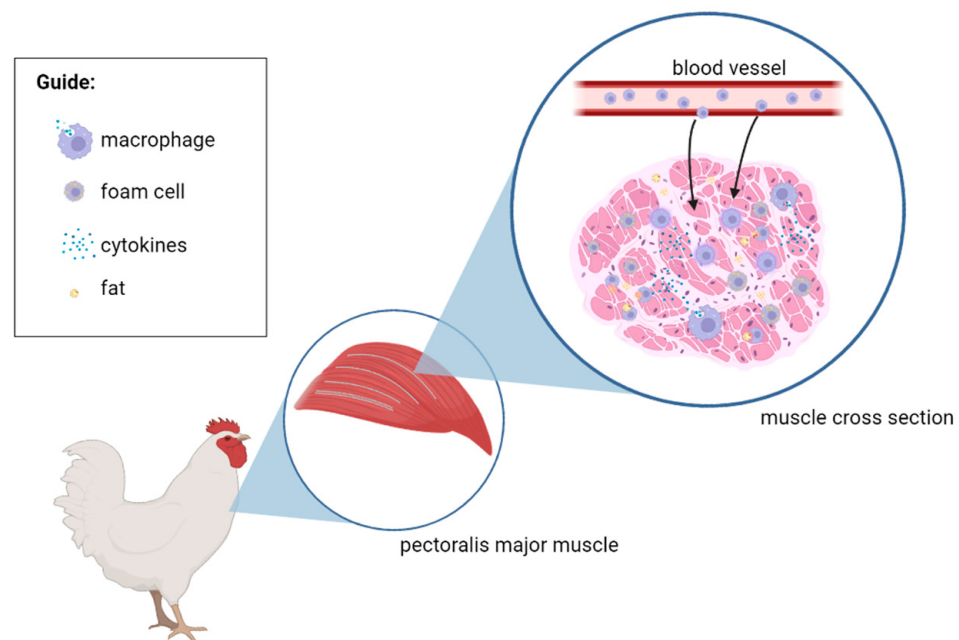


Figure 1. A schematic diagram of the suspected mechanism causing white striping. Created with BioRender.com. Accessed on 29 March 2023.

5. The Causes of White Striping

It is believed that hypoxia underlies the etiology of white striping, which is associated with metabolic disturbances, as well as heart and vascular diseases in rapid-growing broilers [41]. With an increased incidence of hypoxia, a broiler's metabolism can be described as becoming increasingly anaerobic. The pectoralis major muscles are mostly made up of type IIB fibers with low numbers of mitochondria, which makes the broilers more susceptible to damage and explains the localization of the white striping compared to the rest of the body [42]. With less ATP produced from fewer mitochondria, reduced apoptosis can occur where there could be increased inflammatory responses [43], leading to inflammatory diseases in the body, such as atherosclerosis, which has a similar disease mechanism as white striping in the muscles [44–46]. According to a study performed by Boerboom et al. [47], several metabolic pathways were altered in the broilers that had white striping, such as the TCA cycle, β -oxidation, and taurine metabolism. Due to hypoxia, the taurine metabolism pathway is negatively affected. Boerboom found that there are increased taurine levels. Although taurine plays a role in protecting the tissue from damage caused by hypoxia, it is also involved in regulating calcium homeostasis of the tissue;

increased taurine levels led to increased calcium levels, as well as osmosis in the tissues, causing swelling. This swelling will further decrease the amount of oxygen reaching the tissues [47].

Boerboom's group suggested faulty β -oxidation because they saw increased levels of fatty acids with decreases in acylcarnitine esters. In a process characterized as lipotoxicity, several pathogenic processes are associated with fatty acid accumulation that substantially affect the tissue [48]. Fatty acid intermediates, such as ceramides, act as lipid second messengers to initiate cell apoptosis. Free fatty acids are precursors to phospholipids that can affect membranes such as the inner mitochondrial membrane and result in reactive oxygen species (ROS). The subsequent accumulation of ROS results in damage to biomolecules that form cellular structures, organelles, proteins, nucleic acids, and lipids; if antioxidants in the cells are spent from the ROS, then oxidative stress processes contribute further to pathogenesis. The fatty acids are also able to increase the intracellular calcium content, which can lead to cytotoxicity, eventually leading to cell death, which the intermediates in β -oxidation are also able to induce. Some intermediates of the TCA cycle increased, while others were reduced back to the previous intermediate. For example, where oxaloacetate was originally converted to citrate, it was reduced back to malate in broilers with white striping. This backwards conversion is due to the electron transport chain not being able to oxidize the reducing equivalents under low oxygen circumstances [47].

Besides examining pathophysiology within the muscle, one could also consider etiologies of WS from a more basic perspective. Recently developed diets provide more energy with greater amounts of corn and fat than diets from over sixty years ago [49]. Commercial growers today can capitalize on dietary advancements because of decades of genetic selection for growth and yield. Thus, it is important to note that selection and, thus, genetics have been a significant cause of WS. In examining one line each of moderate- and high-yield broilers in the United Kingdom, Bailey et al. [50] noted greater levels of white striping in the high-yield broilers. They characterized the heritability of traits such as body weight and body yield at $h^2 = 0.271 - 0.418$ with WS at $h^2 = 0.338$. In Brazil, Panisson et al. [51] compared three lines—high yield Cobb 500[®] and Ross 308[®] with moderate yield Embrapa 021[®]—with diets of varying nutrient densities and found that, regardless of diet, the higher yield lines had a greater incidence of WS, with the higher yield lines being 24.4–28.0 and 11.0–25.1 times, respectively, more likely to have higher WS scores by days 42 and 49. When describing the history of broiler production, comparisons were made between broilers from the 1950s and 1960s to today's broilers. A striking contrast was noted between the genetics of broilers from these two eras by Havenstein et al. [49,52]: in examining the growth and carcass composition of broiler lines from these two eras (2001 vs. 1957) when both were fed diets from the two respective eras, they observed that the 2001 broiler line had better food conversion, greater body weights, and greater carcass yields, but greater mortality rates, regardless of diet, relative to the 1957 line. Performance results like those of the 2001 line support the significant contribution that genetic selection has played on positive traits, such as yield, and negative traits, such as the incidence of WS. One genome-wide analysis study has implicated three putative quantitative trait loci (QTL) regions on chromosomes 1, 17, and 18 within the chicken genome to be associated with WS when comparing one of each pHu+ and pHu- line [13]; more recently, 18 SNPs have been associated with WS in Cobb 500[®] broilers with six QTLs on chromosome 5 with possible candidate genes involved in insulin secretion, cardiac electrical activity, and inflammation [53]. Genetic selection has contributed significantly to the incidence of WS. Research is ongoing to link growth traits, meat quality, WS, and other myopathies that have arisen with high yield fast-growing broilers.

6. Visualization, Detection, and Measurement of White Striping

According to USDA poultry inspection regulations, while birds are checked for signs of disease before slaughter to remove the unfit cuts, white striping is not often considered with trimming, as it is only considered an issue regarding meat quality and not necessarily

meat safety [48,54]. Furthermore, antemortem detection for white striping has not been considered a priority; however, there are studies that provide useful information for the prediction of white striping. According to Alnahhas et al. [16], a positive correlation was found between the weight of the bird at the time of slaughter and the severity of white striping, which complements the findings of Kuttappan's research, in which high-energy diets were positively correlated with WS incidence. Additionally, Silva et al. [55] utilized real time ultrasound to measure the breast volume, area, and thickness for broiler chickens. Computer imaging and machine learning systems can also be used to score white striping with models, which can be faster and more accurate than human scoring [56]. Imaging and machine learning could help detect white striping antemortem. Additionally, some researchers are considering the detection of white striping of a carcass with a spectrophotometry-based sensor with the skin still attached [57].

Upon slaughtering broilers, white striping can be detected visually as white striations present along the muscle fibers. Striping scores have been created to describe the severity of white striping in the pectoralis major muscle [58]. White striping scores range from 0 to 3 depending on the amount of striping seen on the breast muscle—0 denotes normal, or no white striping; a score of 1 represents mild white striping, characterized as the stripes being under 1mm thick; a score of 2 is for moderate white striping, where the white stripes are between 1 and 2 mm thick; and a score of 3 represents severe white striping, where the stripes are over 2 mm thick and cover most of the breast muscle (Figure 2) [58]. In a study performed by Trocino et al. [59], more male broilers showed severe white striping, whereas more females showed moderate white striping. However, more females exhibited white striping overall. Broilers that were fed a restricted diet presented more white striping cases, as well as more severe cases.

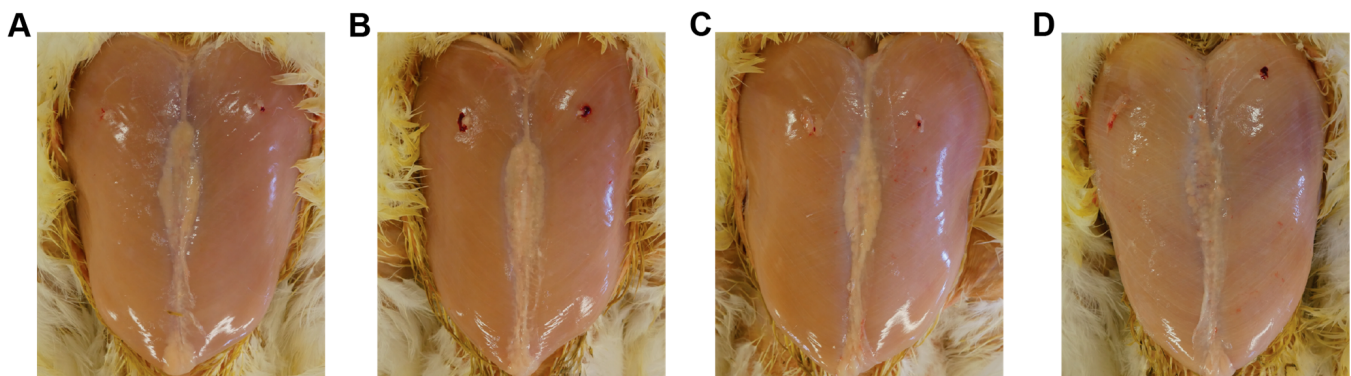


Figure 2. White striping scores for pectoralis major muscles. White striping in broiler breast can range from almost no striping in a normal breast to severe levels of striping. These photographs of the pectoralis major muscles of 42-day old Cobb 500 broilers at market weight demonstrate the range in the scoring system from Kuttappan et al. [58]. Breasts could be: (A) normal with little to no striping, or could have (B) mild striping, (C) moderate striping, or (D) severe striping.

Sections of grossly necropsied samples can be evaluated histopathologically. There is apparent fat infiltration around the muscle fibers affected by white striping, accompanied by different levels of necrosis, atrophy, vacuolar degeneration, fibrosis, and macrophage infiltration [60]. Necrosis can be visualized microscopically around the muscle fibers if there is a presence of a bundle of dead cells, which is a result of increased β -oxidation from the increased amount of long-chain fatty acids [47] with macrophage infiltration present to clear away the dead cells [61]. Vacuolar degeneration is the swelling of cells observed due to cell injuries, which might also derive from the increased β -oxidation, where the cell is trying to repair itself [62]. Atrophy can be seen when comparing normal tissues with tissues exhibiting white striping, where there is a reduction in the size of the muscle fibers in tissues with white striping. When examining the tissues, fibroblasts can be seen initiating scar-like fibrous connective tissue [63]. Further analyses can be performed with

immunohistochemistry. Staining for biomarkers such as MYH15 and NCAM can also delineate the presence of white striping, as studies have shown increases in their levels were proportional to the severity of white striping [5].

7. The Effects of White Striping on Meat Quality and Production

Chicken breast products with white striping do not appeal to customers because of the decreased meat quality, and some scientists point to white striping as a symptom of other metabolic issues [64]. In the studies of Petracci et al. [65], normal chicken breast muscles have a fat content of $0.78 \pm 0.09\%$, while breast muscles with severe white striping have a fat content of $2.53 \pm 0.30\%$. This increase in intramuscular fat can have a negative effect on palatability, as cooked breast muscle with white striping can taste tougher [66]. This increase in fat content is also related to a decrease in protein content. Compared to normal broilers, broilers with severe white striping had 2% lower protein content. Broilers with severe white striping also had 0.13% greater collagen content [65], representing a decrease in the protein quality. The calories are also higher as a result of the increased fat content—broilers with severe WS had 13.56 kcal/100 g more than normal broilers [15]. All of these aspects contribute to the lesser relative nutritional value of the breast muscles of broilers affected by white striping. White striping also led to a 12% of loss in production [66], therefore, it is important for researchers to solve this problem.

In a study by Mudalal et al. [67], it was discovered that breast muscle with white striping had a higher pH level, which is explained by the lowered glycolytic potential. The breast muscle also had a lower drip loss because the high pH can allow the muscles to retain liquid better. In that particular study, a breast with white striping had greater cooking loss values. Breast muscles with white striping had higher compression values, suggesting that the meat is less tender than the breasts without white striping. The breasts with white striping are also less juicy than the normal chicken breasts [68] and can be chewier [69], as they have higher toughness values, which may be due to the increase in the collagen content. Kong et al. [70] discovered that in breast fillets with white striping, there is a decrease of phospholipids, which can be assumed to lead to a lower sensory aroma profile, since phospholipids have shown to increase the aroma in beef. There was also a decrease in the histidine levels, which can be assumed to decrease the palatability of breast fillets with white stripes, as histidine contributes to the taste activity value, which measures how a component contributes to the overall taste [71].

All of these findings suggest an overall decreased meat quality in the chicken breast muscles exhibiting white striping. White striped broiler breast meat features are listed in Table 1.

Table 1. Literature describing the effects of white striping on meat quality and production.

Effect of White Striping on Meat Quality and Production	Study
Decreased production	Marchesi et al., 2019 [66]
Increased fat content	Petracci et al., 2014 [65]
Decreased palatability	Kuttappan et al., 2012 [15]
Decreased protein content	Petracci et al., 2014 [65]
Increased calorie	Kuttappan et al., 2012 [15]
Increased pH level	Mudalal et al., 2015 [69]
Decreased drip loss	Mudalal et al., 2015 [69]
Indicator of hypoxia	Boerboom et al., 2018 [47]
Indicator of decreased β oxidation activity	Boerboom et al., 2018 [47]
Increased collagen content	Petracci et al., 2014 [65]
Decreased juiciness	Lee et al., 2021 [68]
Increased chewiness	Brambila et al., 2016 [69]

It should be noted that while there do not seem to be animal health concerns specifically related to white striping, there are health and welfare issues associated with the management of fast-growing broilers. These issues include skeletal abnormalities, lameness, skin lesions, and high mortality [72,73]. According to Dixon [73], a slower-growing broiler had better all-round welfare compared to the faster-growing birds. The slower-growing birds were more active and had lower gait scores, breast cleanliness scores, feather scores, and hock burn incidence. They also had less lameness, leading to less culls, contributing to a lower mortality score compared to the faster-growing birds.

8. Strategies Attempted to Reduce White Striping

Attempts have been made to mitigate white striping in broilers while avoiding negative effects to broiler growth performance (Table 2). One mitigation strategy is dietary supplementation of ingredients theorized to reduce white striping. For example, Bodle et al. [74] evaluated increasing digestible arginine, supplementing vitamin C, increasing the vitamin premix supplementation, reducing the dietary amino acid density in the grower phase, and combinations of these four strategies. Because hypoxia is associated with white striping, it was thought that arginine supplementation could help with vasodilation [75], thus supplying the muscles with better oxygen resources. Furthermore, they theorized that vitamin C could work against free radicals to prevent cell damage [76]. Moreover, they thought reductions in amino acid supplementation could slow muscle growth, thus allowing more blood flow into the muscle and possibly reduce white striping incidence. However, Bodle et al. [74] concluded that the overall treatment effects for each strategy were not significant. Others have tried to reduce the lysine levels in the diet to lessen WS incidence; lysine bolsters proteins synthesis, thus reductions might slow the growth [77]. Broilers fed an 85% dietary lysine diet had a lower white striping incidence and less tissue damage [78]. Similar results were seen in another study as well, where the reduction of dietary lysine (75% and 85%) at different growth time points (18 to 26d and 28 to 40d) also decreased the severity of WS at 48 and 61 days of age [79]. Alternative methionine supplementation with roasted cowpeas and sunflower seed meal also showed a reduction in WS incidence through decreased *PPARG*, *PTGS2*, and *CD36* expression in the treatment groups [80]. There does not appear to be a single solution to the resolution of WS; there could be multiple underlying factors leading to the disease. Nevertheless, the ultimate goal is for producers to raise their chickens with less white striping, reduced oxidative stress, and with normal muscle growth.

Table 2. Dietary intervention studies to reduce white striping.

Strategy	Mechanism	Outcome	Study
Increase digestible arginine	Arginine aids vasodilation; improved muscle oxygenation	Effects not significant	Bodle et al., 2018 [74]
Vitamin C supplementation	Free radicals scavenging; cytoprotection	Effects not significant	
Increase vitamin premix supplementation	Antioxidants; cytoprotection	Effects not significant	
Reducing dietary amino acid density in the grower phase	Muscle growth decrease; improved hemodynamics/circulation	Effects not significant	Ahsan et al., 2022 [78] Meloche et al., 2018 [79]
Decrease lysine supplementation	Lessens proteins synthesis; reduces growth, vasoconstriction, and hypoxia	Lower white striping incidence and less tissue damage	
Alternative methionine source	Legume antioxidant properties	Lower white striping incidence	
Vitamin E supplementation	Antioxidants; cytoprotection	WS incidence decreased	Kuttapan et al., 2012 [81]
Decrease feed intake	Less energy for muscle growth	WS incidence decreased	Meloche et al., 2018 [79]
Feed restriction	Less energy for muscle growth	WS incidence increased	Trocino et al., 2015 [59]

Feeding the birds with antioxidants, such as vitamin E, was thought to be helpful in mitigating the cell damage that leads to adipose tissue replacing muscle tissue growth. However, there were not any significant effects associated with it [81]. Feed restriction studies have also been performed to discern their effects on white striping. Meloche et al. [82] found that a decrease in feed intake was associated with a decreased WS severity. However, a second study is conflicting: Trocino et al. [59] found that feed restrictions led to the increase of white striping in breasts.

Researchers have also attempted to use genetic selection to reduce the incidence and severity of WS. They discovered that CTSD, LSP1, troponin I2 fast skeletal type (TNNT2), synaptotagmin 8 (SYT8), and MOB kinase activator 2 (MOB2) could be responsible for the appearance and inheritance of WS, as they are related to pancreatic β cell and insulin secretion [53]. Altering these genes and selectively breeding the birds may help with decreasing WS incidence.

Variability exists between different mitigation techniques. Further studies need to be conducted to better understand how to mitigate white striping while preserving favorable traits.

9. Conclusions

Improvements in genetics, nutrition, and management have led to a significant increase in broiler production. With advances in production came an increased demand for poultry meat. However, these advances have led to some trade-offs in meat quality, as is the case with myopathies that have arisen in broiler muscles. White striping is one example of a myopathy that affects the broiler industry, particularly in terms of consumer satisfaction, but also through some financial losses to the industry. Much has been determined about white striping. Hypoxia, inflamed muscle tissues, accumulation of lipids, and the production of cytokines further recruit macrophages into the tissue, likely leading to foam cells, much like an atherosclerotic process with the accumulation of fat and fibrosis leading to the white stripes. Nutritional interventions have been attempted because dietary changes are easily implemented; only some of these interventions have proven effective. More investigation is ongoing to better understand the pathophysiology of white striping. Other researchers are examining myopathies such as wooden breast and spaghetti meat—two other serious broiler conditions—with some theorizing that they all share similar metabolic dysfunctions. All of these conditions are likely the product of the efficacy of fast-growing broilers. There is much still to attempt in further examining mitigation strategies—such as diet trials—to decrease the incidence of these myopathies in broiler chickens. Nutritional strategies should likely include nutrients and substances that aid in decreasing inflammation, reducing fat accumulation in the breast, or providing antioxidant properties. Additionally, studies in which fast-growing broilers are grown more slowly could be informative; however, reducing efficiency could lead to decreases in overall production and increased management costs per bird. Additionally, genetic selection for broilers without myopathies or introduction of new genetic stock could lead to reductions in white striping. Ultimately, a reexamination of the nutrition and genetics of fast-growing broilers could lead to reductions in myopathies, such as white striping.

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