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Breast Muscle Abnormalities in Broiler Chickens

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Thanks to a strong consumer demand, broiler chicken meat production and consumption has reached record levels in the US and it is soon projected to surpass pork and become the most consumed animal protein globally[1]. Broiler chickens have been successfully bred for overall health and disease resistance, growth efficiency, and lean muscle deposition, providing the producers with the "genetic potential" to optimize market driven traits, such as meat yield, safety, quality[2].

The preference for white meat (breast muscle) by the consumers and economic forces have led to a steady increase in market weights of broiler chickens[3], although even at higher weights broiler chicken is developmentally considered a juvenile animal. This is the phase of growth where high rate of cellular protein synthesis and accretion leads to a most efficient growth of an animal, including the muscle tissue. Muscle growth is a complex and highly regulated process that naturally involves both protein synthesis and degradation, as rapidly growing muscle tissue undergoes degeneration and regeneration. It should be pointed that protein accretion and growth of muscle tissue usually is not a metabolic priority for an animal, unless all other physiological needs are met. Under optimal health and environmental conditions, the muscle growth is maximal, as complex regulatory control assures protein synthesis and tissue regeneration exceeds that of degradation and degeneration.

The efficiency and rate of growth and muscle accretion, at times, can overwhelm the regulatory and homeostatic mechanisms, cause cellular dysfunction and lead to spontaneous or idiopathic abnormalities (myopathies)[4]. Breast myopathies in broiler chickens are usually manifested following slaughter and carcass portioning, as no growth problems and/or health issues are detectable ante-mortem. Affected breast muscle(s) can exhibit localized or diffuse changes in organoleptic quality (i.e., color, texture, and composition), but never in product safety. No infectious and/or pathogenic agents have been associated with myopathies. Examples of well-defined degenerative myopathies of chickens include deep pectoral myopathy (DPM), white striping (WS), and hard or woody breast (WB).

Deep Pectoral Myopathy: Also referred as the Oregon Disease or Green Muscle Disease, was first described turkeys[5], later appearing in meat-type chickens[6]. DPM is an ischemic necrosis of the deep pectoral muscle (tenders)[7]. This muscle is enveloped by an inelastic fascia and located in a tight compartment (surrounded by the superficial pectoral muscle and the sternum), which limits the expansion of the muscle upon exertion. As a consequence, pressure builds up within the muscle and occludes the vascular supply, resulting in self-strangulation and muscle death[8]. The incidence and severity

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of DPM increases with high slaughter weights, especially in higher-yielding strains and in males. Increased bird activity (nervousness, flightiness, struggle, and wing flapping) is the primary trigger during rearing[9].

White Striping: The white striping condition is characterized by white striations with varying severity parallel to the direction of muscle fibers in broiler superficial pectoral muscles (fillets)[10-14]. In fact, the occurrence of pale streaks is a manifestation of infiltration of fat as a sequel to myofiber necrosis. The lesions occur more toward the cranio-ventral surface of the fillet compared with the other regions. The severity of these lesions is associated with changes in the proximate composition of the breast meat, especially a decrease in proportions of protein and an increase in fat[15-16]. Furthermore, WS affects not only the nutritional value[17], but also the muscle protein functionality and sensory characteristics of breast fillets[18-19].

Woody Breast: The WB lesions are aseptic and characterized as severe degenerative pectoral myopathy. The breast fillets affected by this myopathy are diffusely hardened (dense) to the touch or exhibit hardened palpable ridges[20]. WB is not uniformly distributed and is worst at the anterior portions of the fillets[21]. Affected fillets can also exhibit white striping and thickened loose connective tissue. In severe lesions, there may be petechial hemorrhages and yellow sterile viscous fluid (transudate) may be observed on the thickest portion of the muscle[22]. Breast fillets affected by WB, both with or without WS, exhibit poorer nutritional value, harder texture, and impaired water holding capacity as compared to normal fillets[19, 23-24].

Breast myopathies can be observed in all breeds/strains of chickens as early as 2 weeks of age with varying prevalence under a wide-range of slaughter weights, management, feeding and rearing systems[25]. Affected muscles histologically show changes typical of focal or diffuse ischemia, including varying severities of muscle fiber fragmentation, swelling, and degeneration, as well as connective tissue, fat and inflammatory cell infiltration[8, 19].

The exact cause(s) of these myopathies are yet to be identified, however, an acute ischemic insult as a result of expansion of the muscle (i.e., sudden exertion, overstretching and/or compression) and limitations in the rate of blood supply (oxygen and nutrients) to and metabolic waste-product removal (carbon dioxide and lactic acid) from the muscle fibers is likely to be involved. Breast muscles of broiler chickens are made up of predominantly white (glycolytic and anaerobic) muscle fibers that are extremely susceptible to ischemic stress. Loss of cellular homeostasis[26] and breakdown muscle fiber membrane integrity triggers a rapid inflammatory and regenerative response in the muscle. Myopathies manifest themselves at different rates and stages when the extent of muscle damage exceeds the capacity of the repair systems to cope[27-29]. Recent gene expression analysis confirmed that localized hypoxia, oxidative stress, increased intracellular calcium, and fiber-type switching are the significant factors in the pathogenesis of these myopathies[30].

Although the genetic basis (i.e., heritability) of broiler chicken myopathies is very low[31], breeding efforts have been put in place for several years to reduce their prevalence. Extensive research is also underway to determine the non-genetic factors in the etiology of woody breast (i.e., flock management,

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environment and nutritional programs)[32]. In the meantime, the processors are aggressively grading out affected fillets following deboning to maintain finished product quality specifications.

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