Focal Myonecrosis Effects in Turkey Muscle Tissue

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Introduction

Modern turkeys, like swine, have been subjected to intense genetic selection for rapid lean muscle growth. Because selection has been based on only a few traits that are primarily of economic importance, little attention has been paid to the functional muscle physiology. Selection to achieve maximal growth performance may partially be responsible for increased incidence of such conditions as leg weakness and edema, deep pactoral myopathy (DPM) and focal myopathy (FM) (Cherel et al., 1992; Siller, 1985; Sosnicki et al., 1989; Sosnicki and Wilson, 1991; Swatland; 1990).

There is also evidence that the occurrence of pale, soft, exudative or PSE-like breast (Pectoralis major) muscle and alterations in the texture, cohesiveness and juiciness of processed turkey breast meat are related to size of the birds, stressful pre-slaughter handling conditions, and onset of rigor morris (Addis, 1986; Gray, 1989; Froning et al., 1978; Ma and Addis. 1973; Sante et al., 1991; Swatland. 1990; Van Hoof, 1979; Van Hoof and Dezeure-Wallays, 1980). PSE-like turkey breast meat was not considered a problem, even though many years ago it was noticed that pH decline in the pectoralis muscle is faster in most turkeys than in the most severely PSE porcine muscle (Ma and Addis, 1973). Only recently, when processed turkey meat became one of the fastest growing products of the food industry, has PSE-like turkey breast meat become a concern.

This paper focuses on abnormalities of skeletal muscle in domestic turkeys and their relationship to meat quality.

Poultry Skeletal Muscle

Problems in meat quality are ultimately caused by changes in the biochemistry and morphology of the muscles themselves, as well as by post-mortem events. Simplistically, there are three major fiber types, designated "white," "red" and "intermediate" in vertebrate skeletal muscle. The fibers differ in their amounts of sarcoplasmic reticulum (SR), mitochondria, oxidative and glycolytic enzymes and substrates. They also differ in the isoforms of contractile proteins such as myosin light (MLC) and myosin heavy (MHC) chains, regulatory proteins, speed of contraction and other mechanical properties, and in their innervation pattern.

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Avian muscle fiber types are classified based on myosin Ca^{+2}-ATPase activity, MLC and MHC isoforms and metabolic enzyme levels: fast-contracting and glycolytic, (IIW or FG; white). fast-contracting, oxidative and glycolytic, (IIR or FOG; intermediate), and slow-contracting oxidative, (IRA and IRB or SO; red) (Carpenter et al., 1984; Khan, 1979; Sosnicki and Cassens. 1987). FG (IIW) and FOG (IIR) fiber types are focally innervated (i.e., a single fiber has one neuromuscular junction) whereas SO (IRB) fibers are multiply innervated ("tonic" fibers that do not conduct action potential).

Most skeletal muscles contain a "continuum" of different fiber types, typically forming a mosaic pattern (Gauthier. 1987). The pectoralis superficialis, major breast muscle of chicken and turkey, is an exception — it contains only "fast" forms of MLC and MHC and has predominantly a glycolytic energy metabolism (Bandman, 1985; Maruyama and Kanemaki, 1991).

Poultry and mammalian skeletal muscle present similar post-mortem biochemical events affecting such key meat quality attributes as protein functionality, water-holding capacity, toughness/tenderness, or processing yield (Dutson and Carter, 1985). However, because the onset of rigor mortis in poultry muscles (especially in the Pectoralis), is accelerated, differences in quality between red and white muscles are more pronounced than in red-meat animals. For instance, myofibrillar proteins isolated from turkey "white" breast meat form gels having greater rigidity than the proteins isolated from "red" thigh and leg muscles (Daum-Thunberg et al., 1992). Thigh meat gels also exhibit higher cooking yield, water-holding capacity, and shear strain at failure than breast meat gels.

Capture Myopathy (CM)

CM is a syndrome associated with the trapping, handling and transporting of wild mammalian and avian species, including the turkey (Meleagris gallopavo) (Chalmers and Barrett, 1982; Spraker et al., 1987).

The lesion in wild turkey muscle is characterized by multifocal areas of muscle fibers containing basophilic sarcoplasm, rhabdomyolysis with subsequent phagocytosis by macrophages or loss of striation with marked disruption and fragmentation of myofibrils (Spraker et al., 1987). Although clinical signs of capture myopathy in wild turkeys are not often found, histopathological subclinical lesions are present in the pectoralis, wing and thigh muscles (Spraker et al., 1987).

These findings indicate that wild turkeys are sensitive to stressful conditions, and care should be taken during trapping, handling and transporting the birds. It may be assumed, therefore, that domestic turkeys are also predisposed to stress (i.e. preslaughter handling) and prone to muscle damage (Froning et al., 1978; Mills and Nicoli, 1990; Van Hoof, 1979).

Leg Edema Syndrome (Transport Myopathy)

A phenomenon possibly related to capture myopathy of wild turkeys is leg edema syndrome. Acute necrosis of muscle fibers, shrunken and pyknotic nuclei, infiltration of the walls of blood vessels by mononuclear cells, fiber hypercontraction or proliferation of endomysial and perimysial connective tissue are characteristic of this syndrome (Sosnicki et al., 1988a; The Merck Veterinary Manual. 1986). The edematous subcutis which predominantly occurs in the medial thigh is often several millimeters thick, usually amber or green and red in color. Acute multifocal muscle necrosis is found primarily in the adductor muscle.

Leg edema may affect a high percentage of birds from a given lot with severe economic consequences in terms of condemned parts. It primarily affects turkey toms and is present in about 5% of flocks in the upper Midwest, USA, with a morbidity of 2% to 70% (The Merck Veterinary Manual, 1986). Although the cause of the syndrome is unknown, it appears to be associated with increased body weight and size, increased transport time and confinement rearing "transport myopathy" of turkeys (Sosnicki et al., 1988a; The Merck Veterinary Manual, 1986). Signs of the syndrome are rarely seen on the farm and while the pathogenesis is unknown, it is presumed to be due to impaired circulation (The Merck Veterinary Manual, 1986).

Deep Pectoral Myopathy (DPM)

DPM is a polygenic abnormality of the supracoracoideus (deep pectoralis) muscle. The affected necrotic muscles usually have a dry stringy texture, a discoloration ranging from light yellow to green to blue, a dehydrated wood-like texture and a gross edematous appearance (Siller and Wight, 1978; Siller et al., 1979).

Anatomical and histopathological studies of the circulation, occlusion experiments, electrical stimulation and exercise studies established that the myopathy is due to an ischemia brought about by swelling of the muscle during exercise (Harper. 1983; Hollands et al.,1971; Orr and Riddell. 1977; Siller and Wight, 1978; Siller et al., 1979). The combination of an inelastic muscle fascia and a rigid sternum causes swelling of the supracoracoideus muscle during exercise. The swelling creates an occlusion of cranial and caudal pectoral arteries causing a loss of blood circulation to the muscle midregion, bringing about its degeneration (Siller, 1985). Selection against the trait, and marketing of birds at ages before the problem appears have dramatically reduced the incidence of DPM.

Focal Myopathy (FM) and PSE-Like Breast Meat Condition

FM of turkey skeletal muscle was detected in 1968 as a disorder characterized by segmental necrosis and hyaline degeneration (hypercontraction or "giant" fibers) of the FG and SO muscles of the pectoral and cervical regions, with proportionally more SO muscles being involved (Dickinson et al., 1968; Maronopot et al., 1968). Although the incidence of FM was related to a peculiar lameness and generally poor bird conditions, healthy birds showed similar degenerative symptoms (Maronopot et al., 1968).

Degenerative changes observed recently in the pectoralis major and iliofibularis (biceps femoris) muscle included granular necrosis with phagocytosis by mononuclear cells of several adjacent muscle fibers or multi-fiber areas, hypercontraction of muscle fibers, infiltration of the endomysial connective tissue with mononuclear cells and fatty tissue replacement in the necrotic areas (Sosnicki et al., 1988b, 1989,1991a,b; Sosnicki and Wilson, 1991; Wilson, 1990; Wilson et al., 1990). Necrotic areas usually presented a uniform low activity of Ca^{+2}-ATPase, diffuse activity of succinic dehydrogenase (SDH) in muscle fibers and alkaline phosphatase in capillaries. There was also a positive reaction for acid phosphatase in muscle fibers undergoing necrosis or hypercontraction. Electron microscopy showed dilation of the SR, intense Z-line streaming and the presence of several myeloid and lysosomal dark-bodies in the necrotic areas (Sosnicki et al., 1991a,b).

From a study of the endomysial and perimysial septa in the pectoralis muscle, Swatland (1990) postulated that in modern turkeys selected for rapid growth, relatively faster growth of the muscle fibers than connective tissue may lead to fiber necrosis and/or loss of connective tissue integrity (Swatland, 1990). It was also found that the outgrowth of breast muscle fibers over supportive connective tissue may predispose products to fragmentation and poor cohesion, and "the fragmentation of turkey rolls may have some relationship to the size of turkeys" (Swatland, 1990).

The incidence of FM and the activity of serum creatine kinase (an indicator of muscle damage) was correlated with age and growth rate of primitive and growth-selected turkey strains (Wilson 1990; and Wilson at al., 1990). Progressively more damage was observed in the muscles of faster growing (selected) and older birds. These birds also had higher serum enzyme levels. Wilson and co-workers (1990) suggested that selection for rapid growth led to muscles that "outgrow their life-support systems," and "bring about muscle damage when coupled with the conditions used to grow turkeys" (Wilson, 1990).

The occurrence of FM symptoms in the breast muscle of 23 week-old birds compared to 18 week-old turkeys was more severe in the older birds (Sosnicki and Wilson, 1992). This was paralleled by lower breast muscle pH of 23 week-old than 18 week-old birds (Sosnicki and Wilson, 1992). Similarly, it has been reported that the early post-mortem rate of pH decline was 1.4 fold faster in a high-performance turkey breed compared to a primitive, slow-growing turkey strain (Sante et al., 1991).

One possible cause of FM in turkey breast muscle is localized muscle microischemia due to a low capillary-to-fiber ratio, and low physical activity of the birds under sedentary growing conditions. Thus, lower values of capillary density and capillary-to-fiber ratio, and greater intercapillary distance (which is the most important limiting parameter for aerobic capacity of the skeletal muscle) were found in the necrotic regions of the pectoralis major and biceps femoris turkey muscle (Sosnicki at al., 1991 a,b). Interestingly, the breast muscle of modern domestic turkeys contains bigger and less aerobic fibers (lower mitochondria volume) than wild birds (Addis, 1986; Swatland, 1985).

As a consequence of structural/functional alterations lead-

ing to microischemia, there may be a high rate of lactic acid production promoting localized breast muscle acidosis. Simultaneous reduction in the rate of energy production may enhance formation of free radicals via reduced mitochondrial respiratory control, typically resulting in degradation of muscle cell membranes and irreversible cell injury. Intracellular proteins may be degraded even in the absence of metabolic energy, and the calpains may play a major role in the enhanced proteolysis in skeletal muscle depleted of ATP (Fagan et al., 1992).

Ante-Mortem Causative Factors of PSE-Like Breast Meat

Exposure to heat or cold stress, or loading turkeys into trucks and confining them in cages during transportation to the processing plant may enhance localized ischemia in breast muscle. This, in turn, may elevate glycolytic metabolism and cause accumulation of lactic acid, resulting in a dramatic decrease of pH in muscle fibers and blood (Froning et al., 1978; Mills and Nicoli, 1990). These conditions may also promote severe FM damage to the breast and other muscles (Sosnicki and Wilson, 1991; Sosnicki, 1993).

We have recently reported that significantly higher pH and ATP levels and lower lactic acid content may be found in the breast muscle of turkeys slaughtered at the farm, as compared to birds receiving 12 hours of preslaughter transportation (Sosnicki and Wilson, 1992). High blood plasma lactic acid concentrations were also negatively correlated with breast meat pH (measured 25 minutes post-slaughter) and water-holding capacity (R^2=0.5; $P<0.05$). Although this relationship explained only approximately 50% of the variation observed, it implies a direct link between accelerated lactic acid production ante-mortem, and poor breast meat quality (Sosnicki, 1993).

Thus, upon transportation to the processing plant (especially during hot and humid weather conditions), the domestic turkey is likely to develop localized and/or systemic acidosis due to unknown, probably stress-related, genetic factors. This, in turn, may cause accelerated glycolytic metabolism and accumulation of lactic acid in the breast muscle. Simultaneous decrease of pH in muscle fibers and blood before slaughtering may result in accelerated rigor mortis process (Froning et al. 1978; Mills and Nicoli, 1990; Vanderstoep and Richards. 1974; Sosnicki and Wilson, 1991; 1992; Van Hoof, 1979; Van Hoof and Dezeure-Wallays, 1980).

Post-Mortem Causative Factors of PSE-Like Breast Meat

Electrical stunning and occasional struggling of turkeys on the processing line (either before or after exsanguination) cause intense tetanic muscle contractions which further accelerate ATP depletion, accumulation of lactate and pH decline. Under such conditions, the pH of breast muscle may be as low as 5.7 when measured 25 minutes post-mortem (Sosnicki and Wilson, 1992). Linear regression analysis of the relationship between post-mortem pH and ATP indicated that the decline of one pH unit is associated with the depletion of 3.86 um/g of ATP (R^2=0.72; significant at $P<0.01$). Thus, the onset of rigor mortis in turkey breast muscle (ATP<1.0 um/g) may occur at pH of about 5.7 and between 0.5 and 1 hour post-mortem. Similarly, Sante et al., (1991) reported that the initial rate of pH decline in turkey breast muscle is 2.2 units/hour; their turkey muscle ultimate pH was reached 35 minutes post-mortem.

The combination of rigor status, low pH (<5.8) and high breast muscle temperature (i.e. above 35°C) may cause alterations in breast muscle proteins leading to very pale, exudative, soft and non-cohesive PSE-like meat with reduced protein functionarty (Sosnicki and Wilson, 1992). In fact, our recent studies have indicated that light-colored breast meat typically exhibits lower initial and ultimate pH, and lower water-holding capacity compared to normal-colored meat (Sosnicki, unpublished data). Value-added breast muscle products made from the light-colored meat usually had higher package purge, poorer meat binding, softer texture and lower flavor intensity compared to the product made from normal-colored meat. Barbut (1993) also recently reported that lighter breast meat exhibits lower pH and gel strength, and higher cooking loss compared to darker-colored meat.

PSE Syndromes In Swine vs. Domestic Turkeys

In swine, rapid glycolysis while the muscle temperature is still high results in PSE muscle with poor water-holding capacity (for review see Greaser, 1986). This condition is typically triggered by stressful preslaughter handling or excessive ambient temperatures, and has been termed Porcine Stress Syndrome (PSS) (Lister, 1987). Live animals susceptible to PSS (and which will develop the PSE condition post-mortem) can be detected by challenging them with the anesthetic halothane. A similar condition, termed malignant hyperthermia (MH), occurs in some humans where anesthetics trigger muscle contraction and an often fatal rise in body temperature.

The ultimate cause of these syndromes (PSS, MH, PSE) has been recently traced to a genetic defect in the Ca^{+2}-release channel (also termed the ryanodine receptor because of its affinity for the plant alkaloid, ryanodine). The ryanodine receptor is a homotetrameric protein that bridges the transverse tubule and SR terminal cistemae membranes, and is believed to play a key role in excitation-contraction coupling (it acts as the major pathway for calcium release from the terminal cisternae to initiate muscle contraction) (MacLennan et al., 1990; Otsu et al., 1991). Linkage studies have traced the disease to chromosome 6, and molecular probes for the gene that codes for the ryanodine receptor have been shown to co-segregate with the halothane gene (MacLennan et al., 1990; Otsu et al., 1991). More recently, a point mutation (**ryr1**) in the ryanodine receptor gene has been identified as being associated with MH in six lean, heavily-muscled breeds of swine (Fujii et al. 1991). Arginine 615 in the normal ryanodine receptor is converted to Cys 615 in **ryr1**. Sarcoplasmic reticulum fragments prepared from pigs displaying the halothane sensitive condition have altered calcium release properties and altered ryanodine binding (Mickelson et al., 1988; Ervasti et al., 1991). Thus. it has been postulated that the wide-spread selection for leanness and muscularity has also spread the

ryr1 gene in the breeding stock (MacLeman et al., 1990).

Our previous studies have indicated that several similarities exist between the development of PSE meat in swine and turkeys subjected to intense genetic selection for rapid muscle growth (Sosnicki and Wilson, 1990, 1992; Sosnicki, 1993). For example, necrosis and hypercontraction of muscle fibers are commonly observed in PSE prone pigs and FM is seen in turkey muscle (Bergman, 1972; Bickhard et al, 1972; Dutson et al., 1974; Greaser, 1986; Schulman, 1980; Sosnicki, 1987; Sosnicki et al., 1988b, 1989,1991a,b).

Several alterations in turkey breast muscle and PSE swine are consistent with calcium-induced cell injury. In ischemic muscle, a close relation between lactic acid accumulation, decrease in the resting membrane potential and extracellular pH and increase of extracellular K^+ concentration was reported (Hagberg et al., 1985). This indicates that a high lactic acid level induces ion disturbances; i.e., a disturbed calcium regulation is a key event in the pathogenesis of ischemic muscle damage, and the development of irreversible cell injury (Hagberg et al. 1985).

There is also evidence that following exercise, heavy turkeys develop hyperthermia and severe lactic acidosis due to anaerobic muscle functioning (Boulianne and Hunter, 1989; 1992). These authors also concluded that exercise of the domestic heavy turkeys may create hemodynamic instability leading to sudden death. Hyperthermia and respiratory and metabolic acidosis associated with high plasma lactic acid levels are also characteristics of PSS/PSE swine (Heffron, 1987; Lister, 1987).

As a consequence of increased calcium permeability of the SR and mitochondria membranes, muscle cell destruction and activation of proteases and phospholipases may occur (Hagberg et al. 1985; Heffron, 1987). The low-Ca^{+2} requiring form of calcium activated factor (um CAF), and activities of calpain I or cathepsins B and L may play a major role in muscle cell degradation (Goll, 1991;Ouali, 1990).

Whether a genetic defect in the Ca^{+2}-release channel similar to PSS/PSE occurs in modern turkeys is yet to be determined.

Protein Functionality in PSE Swine and Turkey Breast Muscle

Bendall and Wismer-Pedersen, (1962) first demonstrated low solubility of sarcoplasmic and poor extractibility of myofibrillar proteins in PSE pork. This led them to suggest that sarcoplasmic proteins were being denatured during rapid postmortem glycolysis and deposited on the filaments in the myofibrils, forming a sarcoplasmic protein "coating."

Recent gel electrophoresis and immunochemical staining of myofibrils showed that only two enzymes, creatine kinase and phosphorylase, weakly co-precipitate with the myofibril fraction of PSE muscle (Greaser, personal communication). Therefore, it has been suggested that the contractile filaments (myosin and actin) in the myofibrils would not be fully covered by these proteins, and partial myosin denaturation rather than sarcoplasmic proteins coating appears to be responsible for the poor extractibility of the myofibrillar proteins in PSE meat (Greaser, personal communication). This idea is consistent with the suggestions of Offer (1991) that myosin is altered in PSE muscle.

Only a limited number of somewhat related studies have been conducted concerning turkey proteins. Daum et al., (1992) stated that there was no evidence of irreversible loss of functional properties of the breast muscle proteins. Their work involved the effect of pH on properties of post-rigor, comminuted turkey breast and thigh meat, not the effect of low initial breast muscle pH/high temperature on protein functionality. Artega and Naki (1992) reported that turkey breast myosin is more temperature sensitive than other (mammalian species) myosins, based on the evaluation of circular dichroism of thermally denatured purified myosin. However, the thermal denaturation was fully reversible if the protein was incubated for 24 hours at 4°C (Artega and Naki, 1992).

Conclusion

The turkey industry can be justifiably proud of the achievements in intensive genetic selection, nutrition research and veterinary medicine. This has resulted in continuing progress in producing heavier turkeys and improving feed efficiency at a given age.

However, there is little information regarding whether turkey muscles, especially the pectoralis, have achieved the phylogenetic growth in size by increasing the number of muscle cells (in the embryonic stage and/or via satellite cell proliferation), the diameter of fibers or their length. Furthermore, there is little information on the relationship among growth, metabolism and quality of the meat of this economically important species. It is doubtful, however, that growth can be pushed without limits. Siller (1985). described DPM as a "penalty of successful selection," a "man-made" disease. Although it remains to be seen whether FM, leg edema or PSE-like turkey breast muscle syndromes may also be inadvertently linked to genetic selection, it appears that the traditional approach of breeding and growing turkeys to maximize their growth performance may have or may soon reach a point of diminishing returns.

Future research programs should be focused on the complex molecular, biochemical and morphological alterations occurring in developing turkey muscle as well as on postmortem processes. Because the rate of rigor mortis is a key event in post-mortem conversion of muscle to meat and meat quality, development of chilling methods preventing PSE-like breast meat should be explored.

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