

MYOPATHY AS A DESTABILIZING FACTOR OF MEAT QUALITY FORMATION

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Key words: *meat quality, pork, poultry meat, muscle tissue, myopathy, muscle fiber lesions, stress, nutritional myopathy, selenium, vitamin E*

Abstract

This review paper is devoted to myopathy of slaughter animals and poultry, and examines a relationship between fast growth of muscle tissue in hybrid pigs, broiler chickens and turkey, and high frequency of detection of spontaneous or idiopathic myopathies. The development of myopathy reduces consumer and technological properties of meat, and leads to emergence of different pathological conditions (PSE or RSE meat, «destructured meat», «white» or «green» meat, punctate hemorrhage, «wooden breast» and others). Two types of myopathic conditions are examined: myopathies caused by stress in animals and nutritional myopathies, which contribution to meat quality deterioration seems to be determinative. It is shown that the basis of the mechanism of the myopathy development is the mechanism of the successive changes in muscle tissue: damage of cell membranes and release of mitochondrial calcium, which causes hypercontraction, dystrophic changes, atrophy and necrosis of muscle fibers. To alleviate the damaging effect of two types of myopathies, different substances-adaptogens (selenium, vitamin E, flavonoids and others) can be used. It is stated that the requirements of animals in adaptogens change with an increase in the indicators of their productivity.

Introduction

Over the last decades, the agriculture has achieved an exceptional success with regard to the growth of the efficiency indicators in beef husbandry and poultry production, including an increase in the rates of muscle tissue gain in feeding as well as the rise in the mass fraction of muscle tissue in the body of slaughter animals and poultry. For example, over the last 50 years, the size and live weight of chickens and turkey changed by two times mainly due to the content of pectoral muscles; while in pork carcasses, the content of muscle tissue increased from 44–49% to 58–62% during the same period [1,2].

However, this success has some negative consequences. The consumer and technological properties of meat quality have changed. The problem of a decrease in meat quality and prevalence of specific defects in meat and meat products associated with myopathy has been discussed more and more often in foreign and domestic practice. After multiple studies, the scientists formed an opinion that the genetic progress enhanced a burden on fast-growing slaughter animals and poultry, and led to the morphological and biochemical modifications of muscle tissue that deteriorated meat consumer properties. The performed studies show that fast-growing hybrids demonstrate high frequency of detection of spontaneous or idiopathic myopathies, including those that are caused by stress, which are of great importance for meat quality and result in emergence of different pathological conditions (for example, PSE meat). Simultaneously, economic importance of meat quality questions both for consumers and meat processors is more and more actively recognized worldwide [1].

Muscle tissue, which is the major part of the animal body, is considered the main component taking part in meat quality formation [2]. In this connection, the study of myopathic conditions caused by feeding and stress in animals is of special interest in the context of lifetime formation of meat quality. This paper presents the review information on the results of the foreign research in the field of myopathy investigation and their effect on meat quality with the aim of generalization and analysis of modern scientific knowledge on this question, and determination of the main myopathy types as the most destabilizing factors of lifetime meat quality formation in fast-growing animals and poultry.

Main part

Muscle tissue damage caused by myopathy

Myopathy (Greek: mys, my[os] muscle + pathos: suffering, disease) is a progressive degenerating neuromuscular disease, in which the metabolism disorder occurs leading to a reduction of the muscle tonus, damage of muscle fibers with their following atrophy. Forms of myopathy and its types can be different. Among them are genetically conditioned (inherited and innate) myopathies, in which a gene defect that caused a disease is known; myopathies of the metabolic character (as a rule, they are also inherited), toxic myopathies; infectious myopathies; nutritional myopathies and other myopathies, including myopathies of unknown etiology [3,4].

All species of mammals including humans are subjected to myopathies [5]. Due to similarity of etiology, myopathies in pigs and birds are called an animal model of the human disease [4,6]. Study of the myopathy development

in animals can be a key to understanding this disease in humans [6,7,8].

Pig husbandry and poultry production have the highest economic losses associated with myopathy. For example, it was estimated that losses due to myopathy in broiler chickens in Poland were 1.2 euro per affected carcass (in prices of 2009). In poultry production enterprises, up to 70% of poultry stock can be affected by myopathy manifested in pectoral muscles [9].

Lifetime characteristics of animal skeletal muscles are of fundamental importance for meat quality formation. Their diversity is enormous taking into consideration the fact that skeletal musculature of an animal includes about 570 muscles with different forms, sizes, location and functions. With that, in general, muscle tissue directly participates in formation of meat tenderness, juiciness and color, and also determines its moisture holding capacity in the raw and finished (processed) form. Its characteristics contribute to a significant variability of meat quality parameters [2,10].

It is known that muscles consist of muscle fibers with different sizes (length and diameter). A fiber diameter varies within a muscle and between muscles, depends on age, physical load, nutrition state and animal species. Muscle fibers in external muscles always have a low diameter (10–30 μm); on the contrary, a diameter of the main muscles of limbs can achieve significant sizes (40–65 μm). A fiber size increases with age until sexual maturation. A fiber diameter is slightly larger in males than in females. It can depend on feeding: according to data [2], the positive genetic and phenotypic correlation between the intramuscular fat content and a muscle fiber cross-sectional area is observed in pigs. In the old age, a fiber diameter slowly decreases. However, as a result of diseases, pregnancy and/or nutrition state, a fiber diameter can change [3]. Along with average muscle fiber sizes, a ratio of muscle fiber types plays an important role in meat quality formation.

Type II fibers (white, fast, glycolytic) are more sensitive to the development of pathological conditions compared to type I fibers (red, slow, oxidative). Therefore, the more such fibers in a muscle, the higher the probability of the development of myopathy. Selection studies aimed at increasing the content of type I muscle fibers show the possibility of meat quality improvement [10,11]. The highest content of type II fibers is typical for muscles *m. Longissimus*, *m. Psoas* and *m. Semitendinosus*. In this connection, it is recommended to send them to histological investigations to diagnose myopathic conditions [3,12,13].

Muscle fibers are dynamic structures, which upon certain conditions can turn from one type to another according to the transition scheme: type I \leftrightarrow type IIA \leftrightarrow type IIX \leftrightarrow type IIB* [2]. The predominant type of muscle fibers depends on many factors — muscle function, animal species, breed, age, physical activity, ambient temperature and nutritional behavior. For example, pork skeletal muscles show

an inclination to an increased content of type IIB muscle fibers [14]. Pork *m. Longissimus dorsi* contains about 10% of type I fibers, 10% of type IIA, 25% of type IIX and 55% of type IIB [2]. With that, the content of type 2B muscle fibers in *m. Longissimus dorsi* can reach more than 90%, which is a result of selection [15].

The total fat content in muscles positively correlates with the content of oxidative fibers and negatively correlates with the content of glycolytic fibers. For example, in pigs, the mass fraction of fat can be up to three times higher in the white part of *m. Semitendinosus* than in the red part of this muscle [16]. At the same time, other studies suggest an absence of strict relationship between the total content of intramuscular fat and a ratio of fiber types in muscles [17].

Fibers of the glycolytic type are characterized by larger diameter compared to oxidative and intermediate fibers irrespective of animal age.

A local increase in a muscle fiber diameter characteristic of glycolytic fibers is revealed in muscle tissue after slaughter as hypertonic giant fibers. This defect is typical for PSE meat and especially often is seen in poultry and pork. It is directly linked with poor quality meat. Muscle tissue predisposition for the post mortem development of giant fibers («nodes of hypercontraction») is formed lifetime and can also be diagnosed lifetime by skeletal muscle biopsy with the following sample incubation at 37 °C for 60 min [11,18].

Skeletal muscles are prone to atrophy and many other degenerative lesions beginning from focal ruptures of sarcolemma and ending with necrosis. Necrosis can vary by the severity degree from several necrotic fibers to gross lesions of muscles. The unique peculiarity of necrosis in fibers of skeletal muscles is the fact that it can be segmental affecting only part of a fiber, which is a consequence of multinuclear nature of muscle cells. In the necrotic segments of muscle fibers, myofibrils and other cytoplasmic structures and sarcolemma are subjected to lysis; however, the basal plate and endomysium are often undamaged [3].

The main cause of atrophy is undernutrition and/or insufficient intake of certain nutrients with feed. When nutrients are deficient, muscle proteins can be mobilized as their source for the whole body. Muscle tissue atrophy is the most prevalent damage of skeletal muscles and can be observed in different types of myopathy. With that, an outcome of atrophy can be degenerative changes in muscle tissue up to coagulative necrosis [3].

Pathogenesis of different myopathies, including nutritional, is characterized by a specific sequence. As muscle necrosis leads to damage and finally destruction of sarcolemma of fibers, normal ionic gradients cannot be maintained. Calcium ions, which are usually in lower concentration in cytoplasm than in the extracellular fluid, diffuse into a cell. Entry of calcium ions leads to activation of calcium-dependent proteases, such as calpains, and the following destruction of myofibrils and other structural elements of a muscle fiber. Hence, in muscle tissue, like in other tissues, there is a successive common destructive pathway of overloading of mitochondrial

* In addition to two main types of muscle fibers, there are five intermediate types.

calcium, which begins as a result of membrane or energy deficiency and ends with coagulation and dissolution of contractile proteins. Histologically, an increase in the content of calcium ions taken from blood is observed in muscle fiber sarcoplasm; with that, blood supply of a damaged muscle is not disturbed. An increase in the concentration of calcium ions in sarcoplasm determines formation of areas of myofibril supercontraction in the structure of muscle fibers. A change in homeostasis results in destruction of cytoskeletal proteins, damage of the structure of cell organelles and their necrosis in addition to the development of contractures of myofibrillar structures. Due to color changes in damaged areas of a muscle, myopathies with the severe form of necrosis are often called «white muscle disease»**. It is also worth noting, that less severe forms of muscle necrosis, in which smaller part of fibers is affected, is often difficult to detect upon general visual examination. However, they undoubtedly play a role in meat quality formation [3].

Myopathy is diagnosed most easily by the method of macroscopic examination in poultry after slaughter. The disease is manifested above all by changes in color (from pink to red or green depending on the degree of the development) and texture (increased hardness and fibrousness) of affected muscles [9].

Microscopic investigation of animal muscle tissue can detect damaged and even necrotic fibers. Changes in muscle fibers can be widely distributed and characterized as multifocal monophasic or even polyphasic lesions. This can point to recent nonfatal episodes of myopathy or to chronic pathology [3].

At the end of the 1990s, observations and descriptions of the defect of pork muscle tissue, which was caused by myopathy and called «destructured meat», appeared. The defect was manifested only in pork hams. The color of affected muscles was strongly altered: meat looked very pale and greyish. Colorimetric investigations showed that the values L (lightness), a (redness) and b (yellowness) increased in muscles depending on how much they were affected by this defect. Highly destructured muscles lost their organized structure. They had higher glycolytic potential compared to unaffected muscles. Histological analysis revealed similarity of muscle destruction with the PSE defect, which was manifested as an increase in the interfibrillar space and supercontraction of muscle fibers [19].

Emergence of «destructured meat» led to significant losses in production of cooked hams (grey color untypical for meat products produced with sodium nitrite, the unbound and fragmented structure of the finished product, the presence of holes, bad consistency and slicing) [19].

To classify a level of ham muscle damage by this defect, the following gradation of meat after boning and trimming was proposed: 1 — ham muscles do not have the «destructured muscles» defect; 2 — the defect is seen, superficial,

located only in the semimembranosus muscle; 3 — the semimembranosus muscle is strongly altered and the defect partly affects adjacent muscles; 4 — ham muscles are destructured (destroyed) [19].

The large-scale investigations carried out in five commercial slaughterhouses in France (more than six thousand pork carcasses), allowed assessing the prevalence of «destructured meat» — the defect seriously or fully affected ham muscles in up to 17% of pork carcasses. With that, more than 80% of hams with this defect had the pH value of aged meat lower than 5.60. Later on, the low pH value was recognized as the main risk factor for emergence of this defect. Also, the backfat depth, leanness and carcass weight of an animal were assigned to the risk factors [19,20]. At the same time, «destructured meat» cannot be regarded as the PSE defect. Maximum quantity of carcasses with destructured ham muscles was revealed at pH 5.5–5.6 (44.9%). At pH lower than 5.5, the proportion of carcasses with the defect reduced (up to 32.5%). In addition, an effect of gender, stress, genetics, maturity of collagen in muscle tissue was evaluated; however, causes leading to this defect still are not fully revealed. Finally, the defect was called a «disease of thin piglets» as this defect appeared in large quantities in enterprises that turned to raising pigs with backfat depth less than 14 mm [19,20, 21]. In more recent studies of pork muscles, an association was found between emergence of defects of the «destructured meat» type and the prevalence of type 2B fibers [12,13].

Myopathy in chickens is known under the names: deep pectoral myopathy, green muscle disease, Oregon disease or degenerative myopathy [4,6]. It is ischemic, spontaneous necrosis, which in chickens affects mainly the pectoralis minor muscle (m. Pectoralis minor) and leads to changes in color and texture of muscle tissue. Two stages are recognized in poultry myopathy progression — early and late. At the early stage, a muscle has characteristic reddish or pink hemorrhages; later on, muscle tissue becomes green or pale grey and contracts; at the micro level, multiple nodes of contraction are formed [6,9]. By symptom classification, three phases are recognized in poultry myopathy: the first phase — an acute inflammatory lesion with multiple hemorrhages, the second phase — a muscle becomes pale and resembles «fish flesh»; the third phase — the progressive degeneration with green necrotic areas [23].

The etiology of the disease in poultry (chickens and turkey) as well as in pigs is not fully revealed. It is believed that the main reason is the fact that selection in meat breeds of poultry was focused on growth rate, musculature and feed conversion (pectoral muscle degeneration is not observed in wild birds). An increase in the muscle fiber diameter leads to a decrease in the free space for connective tissue, reduction of blood supply and disorder of metabolism in pectoral muscles. These changes constrain the mechanisms of muscle recovery, which, apparently, provoke appearance of myopathy. Moreover, the development of myopathy in the pectoralis minor muscle is influenced by its characteristic anatomical location, which prevents hypertrophy of muscle mass upon

** Nutritional myopathy is also described in scientific literature under the names «nutritional myodegeneration», «nutritional muscle dystrophy», «lamb disease» and others.

physical load, causing occlusion of blood vessels and inducing tissue necrosis. Along with the pectoralis minor muscle, the pectoralis major muscle can also be affected [9, 23, 24].

In fast-growing broiler chickens, myopathy was described as a defect, which was given a name «wooden breast» [25]. Macroscopically, the affected pectoralis major muscle is hard, pale, with the

presence of bulges, sometimes is superficially covered with small hemorrhages, exudate and occasionally has characteristic white striping; affected parts also show extended areas with poor binding of muscle bundles [26]. Histologically, the condition was determined as moderate or severe polyphasic myodegeneration with regeneration of muscle tissue. With this defect, inflammation and necrosis are accompanied by accumulation of interstitial connective tissue (fibrosis), which explains an increase in hardness of the affected tissue as a result of an increase in the content of intramuscular collagen [27]. Moreover, upon muscle damage, there is an increase in an amount of extramyofibrillar water, which is significantly lost over time leading to an increase in meat hardness. With that, affected muscles can show heterogeneity of structural-mechanical properties in different layers [28].

Therefore, the study of myopathic conditions demonstrates that primarily animals that can gain muscle mass quickly are prone to the emergence and development of the disease. With that, microstructural changes in muscle tissue inevitably lead to meat quality deterioration and loss of meat functional-technological and consumer properties.

Myopathies caused by stress in animals

Formation of meat quality takes place as a result of post mortem evolution of animal muscles into meat and depends not only on biological characteristics of muscles, but also on the stress level in animals before slaughter [2].

Stress can cause muscle dysfunctions and dystrophic changes not only in humans and animals, but even in insects. The studies carried out on *Drosophila* showed that due to stress damaged muscles were less adaptive, more sensitive to energetic stress and to changes in the ambient temperature [29,30].

Stress in farm animals is the main cause of myopathies and meat quality deterioration. With that, the problem still has such a scope that there is a real danger that consumers can begin associate low meat quality with the problems of meat product safety in general [31]. It is known that stress leads to emergence of meat with PSE (pale, soft, exudative) and DFD (dark, firm and dry) defects [32]. Over the last decades, other degrees of meat quality deterioration have been distinguished such as RSE (red, soft and exudative) and RFN (red, firm and non-exudative). For example, RSE pork is characterized by red color as in «normal» pork (RNF), but shows properties of exudative PSE pork [33].

In pork production, the proportion of meat with reduced quality resulting from stress before slaughter ranges from 10% to 30%, and in several countries up to 60%. As extreme clinical signs, porcine stress syndrome (PSS) is manifested

as paralysis of the heart and necrosis of *m. longissimus dorsi*. As a result of movements, overheating, overstocking and other factors, the content of myoplasmic calcium increases in porcine blood and tissues. In the USA, up to 8% of pork carcasses are rejected due to myopathy caused by PSS [31,34].

It is believed that a cause of wide distribution of PSS was parents heterozygous by mutation in *ryr 1* gene. This gene is responsible for synthesis of ryanodine receptor protein, which is found in the sarcoplasmic reticulum of a muscle fiber. The main function of this protein is regulation of the calcium ion concentration in cytoplasm. Even insignificant stress impact on an animal — carrier of this mutation leads to a sharp increase in the content of calcium ions in sarcoplasm, as a result of which muscle fibers begin to work in the regime of the extremely enhanced muscular load. A rise in myoplasmic calcium (Ca²⁺) is accompanied by an increase in heat production due to activation of phosphorylase and breakdown of ATP, glycogenolysis is enhanced with production of lactic acid, CO₂ and excess of heat. An ATP deficiency is created, which leads to a damage of the actin-myosin complex, emergence of supercontractions of myofibrillar structures of muscle fibers and fast muscle rigidity. Blood pH drops and metabolic acidosis develops. Upon the severe form of PSS, an animal, as a rule, dies. Mild forms of the disease are latent and are detected only during carcass cutting, when the so-called «white muscles» (visually revealed areas or whole muscles that are distinguished by paleness and wateriness) are observed. In these muscles, macroscopically visible areas with bloody color («punctate hemorrhages») are often found. Histologically, areas with hyaline degeneration and necrosis of muscle fibers are revealed [34].

The first studies on an effect of the halothane sensitivity gene — HAL locus with two alleles N (normal) or n (sensitive) — on zootechnical indicators of pigs go back to the beginning of the 1970s. An interest to n allele was quickly recognized in terms of its influence on meat quality deterioration (a risk of the PSE development). Since 1993, the use of the molecular test has allowed recognizing animals with Nn and NN genotypes as halothane-insensitive pigs and assessing an impact of n allele on productivity of animals and meat quality [21,35,36,37]. This led to the fact that animals — carriers of *ryr1* gene with nn alleles were practically excluded from meat production. However, recent studies showed that the work only on one genetic factor *ryr 1* did not enable achieving a significant increase in quality of produced pork and reduction of losses. Despite the genetic selection and use of stress-resistant (halothane-negative) animals, the problem remains to be topical. In the practice of pig slaughter and processing, the result of lifetime stress and the development of myopathy in different forms continues to be found in meat quality assessment [34].

Therefore, the development of pig husbandry shows that genetic knowledge is important but it does not make it possible to fully solve the problem of PSS. It was estimated that only 4% of low quality meat was conditioned by genetics

[31]. Pork quality and yield are still influenced by multiple *ante mortem* and *post mortem* factors including transportation and pre-slaughter holding, methods of slaughter and carcass chilling. All this indicate an increased necessity to pay close attention to stability of pork quality and the ways of its improvement [33].

It should be remembered that the age, at which animals are slaughtered and which has been steadily reduced, also makes a contribution in addition to genetics. For example, it was found that the frequency of myopathy detection was influenced by poultry age and weight (positive linear correlation) [9]. Moreover, at present, there is still no verification of the role of genetic mutations in the development of myopathy in chickens and turkey. There are several explanations of the myopathy development that are not connected with genetics, namely:

- Excessive hypertrophy of muscle fibers of the glycolytic type, immaturity of collagen and/or inadequate development of intramuscular connective tissue, disorders of vessels and blood supply of muscles;
- Heat stress — high muscle temperatures due to flapping, struggle, stress, and high metabolic rate in muscles before slaughter (spontaneous or idiopathic, stress-induced myopathy caused by oxidative damage of proteins [38]);
- Large muscle mass of different carcass parts, which is difficult to rapidly chill after slaughter [1], or incubation of muscles at 35 °C [39].

Nevertheless, to prevent PSS, it is necessary to continue creating favorable conditions for animal holding, adhering to the rules of their transportation and the norms of humane slaughter.

Nutritional myopathies

Recently, the term nutritional myopathy appeared in the literature. The majority of researchers link it with deficiency of selenium and vitamin E, as well as with an increased level of oxidative stress emerging on the background of the high animal growth rate.

Nutritional myopathies are largely known as diseases of calves, lambs, pigs and foals. The first clinico-pathological descriptions of nutritional myopathies (by the example of beef cattle) date back to the 1890s; however, this disease was already well known at that time in Germany, France, Switzerland and Scandinavia. Nutritional myopathies are seldom found in predators (this fact clearly indicates once more the benefit of the meat diet) [3].

The most common deficiency in nutrition that leads to nutritional myopathy in the majority of animals is selenium deficiency. Nutritional myopathy caused by deficiency of vitamin E in the absence of selenium deficiency is seldom observed in mammals; however, it can be common in birds and reptiles. On the contrary, deficiency of vitamin E in combination with lowered selenium status, can lead to nutritional myopathy in different species. In this connection, selenium was recognized as an important nutrient and was

involved in explanation of causes of nutritional myopathy as far back as the end of the 1950s [3,40].

Nutritional myopathy, like other types of myopathies, can be accompanied with necrosis of muscle fibers. As a rule, it has selective segmentary multi-focal and polyphasic character, leaving the basal plate and satellite cells unaffected, and consequently, ensuring quick and effective regenerative rehabilitation. In extreme cases, nutritional myopathy can be accompanied with myoglobinuria, myocardial damage and rhabdomyolysis. Massive myoglobinuria damaging renal tubules can lead to acute renal failure [3].

Animals with high feed conversion efficiency and significant muscle mass show the highest sensitivity to myopathy and cardiomyopathy caused by selenium deficiency. The problem of such myopathies emerges in different parts of the world not only in animals but also in humans. Selenium deficiency associated human cardiomyopathy named Keshan disease was described for the first time in China as far back as the 1930s [41].

The development of nutritional myopathy is closely correlated with the regions with low (<0.005% of dry substances) concentrations of selenium in plants. Historically, nutritional myopathy is considered a disease of young animals, especially very young. Fast postnatal growth, apparently, predisposes to the problems of nutrient deficiency, although nutritional muscle degeneration is found also in adult animals. At the same time, spontaneous nutritional myopathy can arise prenatally and muscle damages can be observed already at birth. With that, the symptoms of nutritional myopathy can be unobservable in parents (usually, cows or sheep) before or during parturition [3].

One of the most complicated aspects of nutritional myopathies is irregularity and unpredictability of their emergence, especially, in the grazing system. With the same content of alpha-tocopherol, polyunsaturated fats and selenium in pig diets, accumulation of vitamin E and selenium in their organs and muscle tissue can be different. This, in turn, can be associated with the health status of animals, including heart diseases [42].

Therefore, it is agreed that in general the cause of nutritional myopathy is deficiency of one or two substances — selenium and vitamin E. In case of low selenium content in a diet, an increased intake of vitamin E can retard the development of myopathy associated with selenium deficiency [3].

Metabolism of selenium and vitamin E is not fully studied. However, the understanding of factors influencing the integrity of cell membranes and their changes in myopathies led to the understanding of the role of these substances. Enzymes containing vitamin E and selenium are necessary as physiological antagonists of the group of chemically diverse substances known as free radicals. Some of free radicals are endogenous products of normal cell function, others are external and undesirable factors that take part in cell metabolism. They can be produced both in cells and outside cells as products of tissue irradiation, reactions on

pharmaceutical drugs and in inflammations. One of the main sources of free radicals is a process of cell detoxification, which makes substances that enter a cell less harmful transforming them into epoxides. Free radicals can initiate cell damage causing peroxidation of membrane lipids and physico-chemical damage of protein molecules including mitochondria, endoplasmic reticulum and cytosol. Protection against an impact of free radicals is provided by the constant presence of small molecules-scavengers, such as tocopherols, ascorbate, beta-carotene as well as by the presence of selenium-containing enzymes of the system glutathione peroxidase / glutathione reductase. Thus, modern concepts about the development of nutritional myopathy imply that in case of absence of sufficient protection by selenium and/or vitamin E, cell membranes are modified by free radicals. The ability of such membranes to support differential ionic gradients is reduced or lost. This initiates a sequence of events when calcium entry leads to hypercontraction of myofibrils and their necrosis [3,42,43].

In addition to drawbacks of a diet associated with deficiency of selenium and vitamin E, there are several factors that can also lead to the development of nutritional myopathy. Among them are feeding with rancid or oxidized fats; feeding with recently harvested grain; feeding of piglets with pea; the presence of several metals in mineral mixes (supplementary feed) as contaminants (including silver, copper, cadmium, cobalt, vanadium, tellurium, zinc and possibly other metals); copper deficiency, unusual physical load; pharmaceutical drugs; toxic substances in feed; disorders in the thyroid gland function and others [3].

Nutritional myopathy of pigs was observed as a spontaneous disease in all countries with intensive breeding of these animals. Until recently, it was considered that classical lesions of skeletal muscles in pigs upon deficiency of selenium and/or vitamin E occur more rarely than changes in the heart muscle and hepatosis. However, systemic microscopic investigations carried out abroad revealed significantly higher frequency of muscle lesions in pigs than it was thought earlier [3].

In outbreaks of nutritional myopathy, both growing and adult pigs can be affected; with that, nutritional myopathy in the latter can occur without any clinical signs or cause slowness of movements and apathy. Although nutritional myopathy is not considered an innate disease, nevertheless, piglets at the age of 1 day can have gross lesions causing muscle weakness or paresis [3].

In nutritional myopathy in pigs, mineralization of damaged muscle fibers is often not abundant and, even when it occurs and visible, it is difficult to find it in pale porcine muscles. This explains the fact that in nutritional myopathy of pigs, lesions in the heart and liver are found much more frequently; with that, there is relatively a low number of reports about lesions of skeletal muscles. Nevertheless, lesions ranged from microscopic to pronounced macroscopic can be observed in muscles in experimental investigations of nutritional myopathy in pigs. In the severe form of the

disease microscopic lesions of porcine muscles consist in multifocal polyphasic necrosis [3,40].

To describe a degree of histopathological changes in skeletal muscles, a semi-quantitative scale of severity of lesions was proposed: 0–normal muscles, 1–mild changes, 2–moderate changes, 3–severe changes. Mild changes were defined as the presence of individual, separated muscle fibers with increased volume and loss of striation or a very low number of degenerating muscle fibers, sometimes with mild infiltration of macrophages. Moderate changes were defined as multifocal degenerating or necrotic muscle fibers with or without infiltration of macrophages, while severe changes were defined as multifocal, relatively widespread degenerating or necrotic muscle fibers with or without infiltration of macrophages [40].

Therefore, selenium and vitamin E play an important role in prophylaxis of nutritional myopathies. With that, selenium being a constituent of selenoproteins is a key factor as it affects endocrine, immune, inflammatory and reproductive processes. Glutathione peroxidases belonging to the family of selenium-containing proteins inactivate peroxides and thereby support physiological functions of muscle tissue. Recently, it has been found that levels of selenium and vitamin E intake that earlier were considered sufficient, do not exclude the development of nutritional myopathy. In this connection, at present, the question of correspondence of existing strategies of feeding highly productive pigs in the context of assurance of their health and, consequently, quality of produced pork has been raised [40].

Conclusions

An increasing number of scientific publications show that the topicality of the problem of myopathy of slaughter animals (especially pigs) and poultry (chickens and turkey) is not diminished. Taking into consideration not only the prevalence of this disease, but also the fact that myopathy affects the most valuable parts of carcasses (*m. longissimus dorsi* and ham muscles in pigs; pectoral muscles in poultry), it leads to considerable economic losses. In this connection, a deep study of this disease is very important to reveal factors influencing its frequency and degree (category) of the development.

At present, scientific investigations are carried out in several directions with the aim of the development of prophylactic measures. For example, in the area of prevention of myopathies caused by stress, there are several studies, which were aimed at inhibition of key enzymes of muscle glycolysis after animal slaughter by peroral administration of different substances shortly before slaughter. Up to now, the results of these studies were not considered successful, despite the fact that several substances can inhibit tissue enzymes (for example, citrates and acetates –phosphofructokinase) [44]. To prevent the emergence and development of nutritional myopathies, investigations have been continued in the field of studying an effect of different doses of selenium and vitamin E with regard to sources of these substances, dura-

tion of feeding period, feed composition and other factors [1,12,40,43,45,46].

Recently, a new direction in studying myopathic conditions has emerged — nutrigenomics, which is aimed at investigation of an influence of food nutrients on gene expression and meat quality. As a measure for reducing risks of myopathy development, an addition of different adaptogens to an everyday diet is considered. With that, a choice of a diet is one of the potential tools of meat quality management influencing even muscle fiber characteristics [43,47].

Therefore, a search for nutrients-adaptogens and regulators of the targeted development of muscle tissue, that ensure its stability to unfavorable environmental factors, above all, stress factors, which have a destructive effect on muscle tissue microstructure, can be the main way of meat quality assurance with the following intensification of its production.

Acknowledgment

The work was supported by the grant No. 19-16-00068 of the Russian Science Foundation.

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The authors declare no conflict of interest

Received 20.08.2019 Accepted in revised 12.09.2019 Accepted for publication 25.09.2019