

Ontogenesis of Muscle Tissue

Bon E.I., Maksimovich N.Ye., Kostyukevich D.S., Kalko M.D., Misik V.A., Kipen K.M

Grodno State Medical University, department of pathological physiology

***Corresponding Author:** Bon E.I, Grodno State Medical University, department of pathological physiology.

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Abstract

Muscles are remarkable biological structures responsible for the movement, stability, and complex coordination of the human body. The growth and development of muscle tissue during postnatal ontogenesis occurs through a series of sequential differentiations that lead to changes in the morpho functional and metabolic organization of muscle fibers. The structural organization and functioning of muscle tissue are based on a complex network of specialized proteins that facilitate the processes of contraction, relaxation, cytoskeletal maintenance, and signal transduction. Muscle fibers of all types at different stages of postnatal ontogenesis are characterized by high energy metabolism lability. The protein composition of skeletal muscles is not a static formation, set once and for all. In this article, we will consider the features of muscle tissue in the process of ontogenesis.

Key words: ontogenesis; muscle; protein; development

Introduction

The ability to move is a natural human trait. This innate ability to move is known as motor function or locomotor function, and it is described by motor capabilities, which are understood as a combination of morphological and functional features of the body, physical qualities, motor skills, abilities, and health. Motor function develops naturally from birth to around the age of 18-20, and it can be stimulated through physical exercise.

After the age of 30-35, there is a decline in motor capabilities, which is particularly noticeable after the age of 18. Although much work has been done on locomotor function after the age of 65, researchers have faced significant challenges. This is because motor function is affected by its natural decline with age, as well as sedentary lifestyle and medical conditions. [1,3]

The development of skeletal muscles is a multi-step process, and understanding it is central to a wide range of fields and applications, from potential medical value for human society to economic value associated with improving farm animals.[16]

Skeletal muscles are a complex and heterogeneous tissue, and characterizing their cellular heterogeneity, as well as their transcriptional and epigenetic features, is essential for understanding the details of their ontogenesis. The formation of skeletal muscles is a complex process that requires precise spatio-temporal control of key myogenic factors. Emerging evidence suggests that RNA processing plays a crucial role in regulating these factors, and that multiple post-transcriptional regulatory pathways work in a dependent and independent manner, ensuring precise control of transcripts throughout muscle development and regeneration.

In addition to the general roles of myogenic regulatory factors, it is becoming increasingly clear that processes related to non-coding RNAs (ncRNAs) are crucial for the formation, growth, and repair of skeletal muscles, indicating a key role for processes such as microRNAs (miRNAs), long non-coding RNAs (lncRNAs), and circular RNAs (circRNAs) in myogenesis.

Myogenic stem cells (MuSC) in embryos and adult skeletal muscles are regulated by the transcription factors Pax3 and Pax7, which determine their stem characteristics, while their lineage determination and terminal differentiation are determined by myogenic regulatory factors (MRF). Regulation of myogenic stem cell activity involves multiple transcription factors, such as myoblast differentiation protein 1 (MyoD1), myogenin (MyoG), myogenic factor 5 (Myf5), and myogenic factor 6 (Myf6), as well as other myogenesis-related regulatory transcription factors, including the paired box 3 (PAX3), paired box 7 (PAX7), and myocyte enhancer factor 2 (MEF2) families.[11,12,13]

Individual development of the body, both during development and in old age, is associated with age-related changes in skeletal muscles. The growth and development of muscle tissue in postnatal ontogenesis occur through a sequential series of differentiations, during which changes in the morphofunctional and metabolic organization of muscle fibers occur. As is known, the processes of differentiation are based on the activation of a new synthetic apparatus, which involves the involvement and increased proliferation of satellite cell nuclei and the appearance of new RNA, while the growth of muscle cells relies on the existing capabilities of cellular synthesis in the ergastoplasmic zones on already prepared matrices.[2]

The formation of muscle fibers occurs primarily during the embryonic stage. Therefore, the mother's lifestyle, particularly nutrition and exercise during pregnancy, has a critical impact on the development of fetal skeletal muscles and the subsequent metabolic health of the offspring. The formation of fetal skeletal muscles is divided into primary and secondary stages. Primary myogenesis begins in the first trimester of pregnancy, leading to the development of primary muscle fibers. In contrast, secondary myogenesis occurs in the last trimester and results in the formation of secondary muscle fibers, which make up the majority of the muscle tissue. Physical exercise during pregnancy contributes to the development of fetal muscles and improves the metabolic health of offspring. Nutrients from food have a key impact on the composition and function of the intestinal microbiota, and this is also true during pregnancy. For example, a high-fiber diet can mitigate the microbial imbalance caused by obesity, thereby reducing cognitive and social impairments in offspring through the production of short-chain fatty acids (SCFAs).[20]

CREG1 (cellular repressor of E1A-stimulated genes 1) is a protein involved in cellular differentiation and homeostasis regulation. However, its role in skeletal muscle satellite cells differentiation and muscle regeneration is poorly understood.[21]

As is known, the basis of differentiation processes is the activation of a new synthetic apparatus associated with the involvement and increased proliferation of satellite cell nuclei and the appearance of new RNA, while the growth of muscle cells relies on the existing capabilities of cellular synthesis in the ergastoplasmic zones on already prepared matrices. The signal for the next re-differentiation can be either neurogenic, from synaptic endings, or humoral. The organization of motor control is very complex, and its development in mammals takes a long time during postnatal ontogenesis. During this time, the muscle tissue matures.[2]

Circular RNA (circRNA) is a new type of endogenous non-coding RNA that is formed by reverse splicing of an mRNA precursor.

The biogenesis, degradation, transport from the nucleus to the cytoplasm, localization, and even translation of circRNA is controlled by RNA-binding proteins (RBPs).

They are widely distributed in plants and animals and are characterized by a stable structure, high conservation in cells or tissues, and specific expression patterns at different stages of development in different tissues. CircRNAs are involved in the regulation of fat and muscle cells in cattle and are differentially expressed in tissues composed of these cells.[14,15]

Of particular interest are the data from embryogenetic studies of neuromuscular connections in the muscles of different parts of the fetal body, which have revealed the non-synchronous maturation of nerve elements in functionally distinct muscles. The differences between the spindle fibers of these two muscle groups lie in the fact that throughout their intrauterine development, their nerve apparatus retains a structure specific to each muscle group. This includes the branching pattern of the main nerve fibers, as well as the number and length of primary, secondary, and tertiary branches, and their positioning relative to the intrafusal muscle fibers.[4]

In the early stages of embryogenesis, maternal factors are the proteins. They are synthesized in the egg cell (oocyte) before fertilization and accumulate in it, stimulating and supporting the development of the embryo until it can synthesize its own proteins. The Nanog protein (homeobox protein) plays a key role in maintaining the pluripotency of cells, which is the ability of cells to differentiate into different types of tissues. At the blastula stage, the main proteins are the cadherin family proteins.[5]

The formation and dynamics of muscle protein accumulation in ontogenesis have been studied by a number of authors. Special attention has been paid to identifying the relationship between the formation of

contractile muscle protein at various stages of embryonic and postnatal development in animals and the development of contractile function. The study of actomyosin complex proteins using new research methods introduced by Saint-Dierd in muscle biochemistry in ontogenesis and phylogenesis was apparently the first attempt by I. I. Ivanov and his colleagues. In 1948, I. I. Ivanov and B. S. Kasav'ina established that the muscles of newborn mice and rats, as well as the muscles of guinea pig embryos that are still hairless, contain only very small amounts of typical actomyosin complex proteins. On the other hand, the proteins that make up the same muscles (quadriceps and m. gastrocnemius) in newborn guinea pigs are almost identical in their properties (ability to contract in response to ATP and to bind to actin) to the proteins found in the muscles of adult animals. Thus, the typical contractile protein of striated muscles, actomyosin (as well as myosin), accumulates in significant quantities in the muscles of various animal species at different stages of embryonic or postnatal development. Since during the development of the neuromuscular apparatus in the embryonic and postnatal periods of an animal's life, the tonic contractile reaction is replaced over time by a non-tonic one (see, for example, L. A. Orbeli, 1938; A. T. Khudorozheva, 1947), I. I. Ivanov and B. S. Kasavina (1948) suggested that a change in the protein composition of muscles in various animal species, the last days of embryonic or the first day

of postnatal life coincide with the development of fibers capable of a pronounced rapid contractile reaction of a tetanic nature.

When adult animal muscles are extracted with saline solutions with an ionic strength of about 0.4 for 24 hours, the peak corresponding to the albumin fraction is barely detectable on muscle protein electrophoregrams. In embryonic muscle, on the other hand, according to I. I. Ivanov, V. A. Yuriev, and others (1956), this fraction can account for up to 40-50% of the total amount of extractable protein substances.

Thus, based on the electrophoretic and biochemical study of the composition of the proteins in embryonic muscle, the above-mentioned authors conclude that there is a significant change in the ontogenesis of animals in the fractional composition of the proteins in striated muscle, which is enriched with actomyosin at certain stages of development, either embryonic or postnatal. Actomyosin is apparently preceded by the proteins of the proactomyosin complex. At the same time, it should be noted that the high content of myoalbumin in embryonic muscle is not accidental. According to Gitlin et al. (1955), this protein, which is identical or very similar to serum albumin, may be used as a source of protein nitrogen for the construction of various muscle proteins. As the embryo develops, the content of nucleoproteins and nucleic acids in muscle tissue decreases rapidly. Embryonic muscle tissue differs significantly from functionally mature skeletal muscle in terms of the fractional composition of proteins in both myofibrils and sarcoplasm. These data were first obtained by Krepax (1952). As already mentioned, the most characteristic feature of embryonic muscle tissue is a sharply elevated albumin content.[11]

Embryonic muscle tissue differs significantly in its chemical composition from functionally mature muscle tissue. Embryonic muscles contain significantly more water than similar muscles in adult animals. Consequently, the protein content in embryonic muscles, calculated based on wet weight, is lower than in postnatal muscle tissue of animals of the same species.[6]

At the microstructural level, muscle tissue in mammals has a common structure. It consists of striated cylindrical muscle fibers. Their length in cross-section reaches 10-30 cm, and the average diameter is 50 microns. A distinctive feature is the presence of specific organelles called myofibrils in the sarcoplasm. Striated skeletal muscle tissue (musculus skeleti) is the most abundant in the animal's body, with the muscles of the pelvic limbs being the most developed, as they bear the primary load of the animal's movements. The size of skeletal muscle fibers in different muscles varies depending on the animal's gender, diet, and degree of

functional load. Skeletal muscles in different animals have specific differences at the organoleptic (color, smell), physical-chemical, macro- and microstructural levels, which also depend on the animal's age, gender, and living conditions and diet.[6]

Non-coding genes, such as microRNAs and long non-coding RNAs (lncRNAs), which are widely studied, play an important role in the regulatory network of skeletal muscle development.

Recent advances in sequencing technology have contributed to the identification and characterization of more ncRNAs (mainly microRNAs (miRNAs), long non-coding RNAs (lncRNAs), and circular RNAs (cRNAs)) involved in the development of skeletal muscles in domestic chickens, where they are widely involved in the proliferation, differentiation, fusion, and apoptosis of myoblasts and satellite cells, as well as in the determination of muscle fiber type. MicroRNAs are a class of small non-coding RNAs consisting of approximately 19-25 nucleotides, which often regulate gene expression post-transcriptionally and play an important role in processes such as cell proliferation and metabolic stabilization. Long non-coding RNA (lncRNA) is a transcript that does not encode protein and is longer than 200 nucleotides. It has a lower level of sequence conservation and expression than protein-coding genes, but more and more data is confirming its key role in biological processes such as organ growth and development, cell proliferation and differentiation, glycerol lipid metabolism and diseases. [17,18,19]

The amount of muscle protein synthesis is controlled at the mRNA translation level; in turn, the mRNA pool depends on the number of nuclei. Therefore, the intensive protein synthesis in growing animals is due to the high rate of nuclear accumulation, but with age, this process slows down and the number of nuclei per unit of muscle fiber length reaches a plateau.[7]

Several sarcolemmal proteins are responsible for the repair of muscle fibers. Dysferlin, encoded by the DYSF gene, is one of these proteins. Dysferlin promotes the repair of membranes in striated muscle fibers (SMFs). Mutations in the DYSF gene lead to the loss or reduced expression of dysferlin, impairing the repair of membranes in SMFs and causing membrane damage, which is clinically manifested as dysferlinopathy.[8]

In terms of its chemical composition, the heart muscle is intermediate between skeletal muscle and smooth muscle. The content of cholesterol and phospholipids in the heart muscle is twice as high as in skeletal muscle. In addition to lecithin and cephalin, the myocardium also contains sphingomyelin among the phosphatides. The content of fats and lipids in smooth muscle can sometimes vary significantly. The concentration of these substances depends on various factors, such as the species of the animal, its habitat, the functional state of the organ being studied, such as the uterus, etc. All of the above applies to the content of glycogen in smooth muscle. The concentration of various minerals (Na,

K, Mg, Ca, Cl, P, etc.) in smooth muscle is usually similar to that in skeletal muscle. It should be noted that the chemical composition of muscle tissue is determined by the interaction of various factors.[9]

Histochemical studies are used to identify specific proteins, enzymes, and other molecules in the sarcoplasm, such as glycogen and fat. These studies involve examining the chemical and biochemical properties of muscle cells using various staining and analysis methods.[6]

The main biological factors that control the total protein supply in the mammalian body include the intake of amino acids from food and the cell growth regulation systems, which include numerous anabolic and catabolic regulators. The body uses dietary protein only to compensate for the loss of amino acids during growth, oxidation, or excretion. This is an important adaptive feature, as it helps to maintain the level of free amino acids in the blood plasma and tissue fluids within a certain range, even if the intake (and absorption in the intestines) is zero. Maintaining a certain balance between protein synthesis and breakdown in skeletal muscles is

essential for the normal growth and health of animals. It is known that proteins in animal cells are constantly being renewed, and this homeostatic mechanism is essential for the normal functioning of the organism. The formation of protein pools in the organs and tissues of the body is determined by the balance between protein synthesis and degradation. However, the quantitative characteristics of these processes, which are typically assessed using the isotopic tracer method, are currently unknown for all species of growing animals, in all tissues, and under the influence of a limited set of regulatory factors (Yerimbetov et al., 2005).

Given that the amount of free amino acids does not exceed 0.5% of their total pool in the animal's body, protein metabolism is a unique mechanism for the constant redistribution of amino acids for the synthesis of proteins necessary for normal development (Swick, 1982). The total amount of protein turnover in the body (g/day) is typically five times higher than the animal's daily requirement. For example, the typical daily intake of protein in the human diet is 50-100 g, while protein synthesis exceeds 300 g/day (Clugston, Garlick, 1982). It is known that a large number of factors, including nutritional factors (Seve, Pontier, 1997; Loble, 1998) and hormones (Davis et al., 2003), stimulate protein synthesis in muscles, and the rate of protein degradation can also be controlled. For example, rapid muscle growth in sheep carrying the muscle hypertrophy gene (callipyge gene) is partially achieved by inhibiting protein degradation (Lorenzen et al., 2000). The amount of muscle protein synthesis is controlled at the mRNA translation level; in turn, the mRNA pool depends on the number of nuclei. Therefore, the intensive synthesis of proteins in growing animals is due to the high rate of nuclear accumulation, and with age, this process decreases, and the number of nuclei per unit length of muscle fiber reaches a plateau (Cherepanov, 1994). Nutrition increases muscle protein synthesis through increased eIF4 complex formation (Kimball et al., 2000; Wilson et al., 2009), while fasting and low feed protein levels reduce muscle protein synthesis by decreasing eIF4 formation (Liu et al., 2015). The stimulation of muscle protein synthesis by nutritional factors is particularly pronounced during early growth periods, and the efficiency of the translational mechanism decreases with age (Davis et al., 2008). It has been shown that bolus feeding in newborns stimulates muscle protein synthesis more effectively than continuous feeding (Davis et al., 2015). Catabolic stimuli, such as infection, inflammation, intestinal malabsorption, and aging, blunt or block the nutritional stimulation of muscle protein synthesis at the level of translation initiation and reduction of the formation of the initiation triplet complex and/or the eIF4 complex. [7,11]

Conclusion

Maintaining a certain balance between protein synthesis and breakdown in skeletal mice is essential for normal growth and health. During periods of intensive growth, muscle mass accumulation is stimulated by increased protein synthesis at the level of mRNA translation, and the cellular mNC pool depends on the number of nuclei. Therefore, intensive protein synthesis in growing animals is due to a high rate of nuclear accumulation. With age, this process declines, and the number of nuclei per unit of muscle fiber length reaches a plateau. Genetic factors, hormones, amino acids, phytoecdysteroids, rhodanines, β -adrenergic tomatidine, ursolic acid influence protein metabolism through signaling pathways, altering the effectiveness of muscle growth. After muscle fiber formation is complete during embryonic development of skeletal muscle, myosatellite cells are located outside the multinucleated fiber and remain proliferatively inactive. Changes in the genetic program of muscle stem cells during the transition from the embryonic to the juvenile and then to the adult stage prompt caution in applying knowledge gained from embryonic research to adult stem cell biology.

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