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Review Article

Protein Composition of Striated Muscle Tissue

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Abstract:

The study of the chemical composition and metabolism of muscle tissue is a fundamental task of biochemistry and physiology, since it is proteins that provide the unique ability of muscles to contract and convert chemical energy into mechanical work.

Key words: muscle tissue; striated muscle; myosin; actin; actomyosin; ATPase activity; adenosine triphosphate (ATP)

Introduction

The proteins of muscle tissue represent a complex, hierarchically organized system, which, based on solubility and localization, is traditionally divided into three main groups: sarcoplasmic, myofibrillar, and stromal proteins [1, 2]. Sarcoplasmic proteins mainly include enzymes of energy metabolism, supplying the muscle with ATP, as well as myoglobin, involved in oxygen transport. Myofibrillar proteins—myosin, actin, actomyosin, tropomyosin, and regulatory proteins—form the contractile apparatus of the cell and directly carry out the process of muscle contraction [3]. Stromal proteins perform supporting and structural functions, ensuring the integrity of the muscle fiber and the transmission of developed force [2, 4].The aim of this work is to systematize data on the protein composition of striated musculature, to examine the structural and functional characteristics of the main muscle proteins and their role in contraction. The article analyzes representatives of the sarcoplasmic and myofibrillar fractions, their properties, and isolation methods. Special attention is paid to the relationship between protein structure and their functional activity—ATPase activity, ability for polymerization and reversible interaction—as well as modern concepts of the molecular mechanisms of muscle contraction, which are based on the interaction of actin and myosin in the presence of ATP [5, 6].

General Characteristics of Muscle Proteins

Muscle tissue proteins are divided into fractions that differ in their solubility in water and salt solutions of varying ionic strength [7].

Upon repeated extraction of a muscle homogenate with 0.6 M (or more concentrated) KCl solution, almost all proteinspass into the solution, except for the insoluble stromal proteins. Subsequent dialysis of the obtained extract at pH 7.0, reducing the ionic strength to 0.03 M KCl, leads to the precipitation of myofibrillar proteins that are insoluble at low ionic strength. Readily soluble sarcoplasmic proteins remain in the solution, along with some recently discovered myofibrillar proteins that are soluble in salt media with low ionic strength.

In addition to precipitation, other methods, such as electrophoresis, are also successfully used for the separation of muscle proteins extracted with 0.6 M KCl.

Using electrophoresis, muscle proteins are separated into the following main fractions:

- Proteins of the actomyosin complex (actomyosin, myosin, actin), as well as closely related, poorly studied proteins (contractin, Y-protein, etc.), present in minor amounts.
- Proteins of the heterogeneous myogen group.
- Myoalbumin, whose content in the muscles of adult individuals is low but significantly higher in embryonic tissue [8].
- Water-soluble myofibrillar proteins (L-protein, S-protein, D-protein, etc.), isolated by more complex methods.

Individual fractions, such as sarcoplasmic proteins, can be further separated into narrower subfractions.

Sarcoplasmic Proteins

Sarcoplasmic proteins are soluble in salt media with low ionic strength (e.g., 0.03 M KCl) [9].

When obtaining sarcoplasmic proteins by various methods (pressing out muscle juice under pressure of 50–60 atm, extraction with 0.03 M KCl, or dialysis against 0.03–0.04 M KCl—the so-called Weber extract) and subsequent dialysis against distilled water (when the KCl concentration drops below 0.005 M), precipitation of a protein known as globulin X occurs. Proteins of the myogen group and a small amount of myoalbumin remain in the solution [4, 10].

It is now established that myogen represents a heterogeneous system. It has been found to possess enzymatic (aldolase) activity. Fractionation with ammonium sulfate allows the separation of myogen into myogen A (crystallizing in the form of hexagonal prisms) and myogen B (in the form of asymmetric plates) [3]. Myogen A includes aldolase, α-

glycerophosphatase, dehydrogenases, triosephosphate isomerase, and other enzymes. Myogen B is much less studied. Thus, the terms "myogen," "myogen A," and "myogen B" are collective concepts for a whole range of proteins.

Electrophoresis proved to be the most effective method for separating sarcoplasmic proteins. In muscle extracts with an ionic strength of 0.15, free electrophoresis reveals three groups of water-soluble proteins, each consisting of several components (first group: m, n, I; second: k_1, k_2 and j; third: I, sp, h). Components m, n, and I possess aldolase and triosephosphate dehydrogenase activity; phosphorylase activity is associated with fractions K_1 and K_2; h corresponds to myoalbumin [6].

The correspondence between the components identified by free electrophoresis and the spots in paper electrophoresis has not been definitively established. Usually, three spots are detected on paper: one identified as myoalbumin (h) (particularly intense in embryos and newborns), and the other two correspond to the groups of fractions (m, n, I) and (K_1, K_2), but without further detail [7, 11].

The term "globulin X" was proposed for the fraction of muscle proteins that precipitates upon lowering the ionic strength of the solution as a result of prolonged dialysis to $\mu = 0.005$. The precipitate is only partially soluble when the ionic strength is increased (e.g., at pH 5.8 and $\mu = 0.3$, only about 40% dissolves), which is likely related to partial protein denaturation [12].

Globulin X accounts for about 20% of the total protein nitrogen of muscle tissue. Pressed juice contains about 12–13% water-insoluble globulins. Despite the long-standing description of this protein, its nature remained poorly understood for a long time. It is known that globulin X remains soluble at ionic strengths not lower than 0.005, has insignificant viscosity, shows no flow birefringence, and does not form threads when blown into water. Its isoelectric point is near pH 5, and its molecular weight is about 140,000–180,000. In electrophoresis, globulin X is not detected as a separate component but is part of the myogen fraction.

Further research showed that globulin X is not an individual protein but represents a mixture of various globulins. This is confirmed by data from fractional salting-out with ammonium sulfate: proteins possessing globulin X properties are distributed across the entire saturation range (30–70%) without a clear salting-out zone, although the maximum amount precipitates at 40–50% saturation. Electrophoretic analysis also confirms heterogeneity: removal of the water-insoluble precipitate does not lead to the disappearance of any peaks on the electrophoretogram, and the precipitate itself, upon re-dissolution, gives a broad, diffuse peak. Thus, it can be concluded that globulin X does not exist as an individual protein but represents a mixture of globulins with different physicochemical properties [13, 14]. Myoalbumin, soluble in distilled water, is also insufficiently studied. There are indications of its identity with serum albumin. Its content in the muscles of adult animals is low (about 8.5% of sarcoplasmic proteins), but it is sharply increased in the early stages of ontogenesis, as well as in smooth tonic musculature [8].

Summarizing the data, it can be concluded that sarcoplasmic proteins account for no more than 25–30% of the total muscle protein mass in the striated musculature of homeotherms, although data from different authors vary (from 24 to 38%). These discrepancies are undoubtedly due to methodological reasons, particularly the identification of sarcoplasmic proteins with water-soluble proteins. It is now established that water-soluble proteins are also present in myofibrils [1, 15]. The former division of sarcoplasmic proteins into myogen, globulin X, myoalbumin, and pigments has lost its meaning, as the existence of globulin X as an individual protein was not confirmed, and the term "myogen" is collective. In the modern literature, it is common to refer to the content of sarcoplasmic and myofibrillar proteins, which in turn can be separated into subfractions (e.g., by electrophoresis) and characterized by their enzymatic activity.

Myofibrillar Proteins

This group includes well-studied proteins: myosin, actin, actomyosin, tropomyosin, as well as recently discovered L-protein, A-protein, water-soluble myofibrillar protein, and others [16]. Myosin

Myosin is extracted from minced muscles with 0.5–0.6 M KCl (or NaCl, NH_4Cl) solution. Such extracts, containing, in addition to myosin, an admixture of actomyosin, are called myosin A. To obtain pure myosin (L-myosin), the myosin A solution is diluted to an ionic strength of 0.3, whereby actomyosin (S-myosin) precipitates. The myosin remaining in solution is precipitated by further dilution (minimum solubility at 0.025 M KCl) [5].

Myosin is localized in the A-bands of myofibrils. It is a fibrous protein; its solutions exhibit flow birefringence, which is not reduced in the presence of ATP. The myosin content in muscles, according to various data, ranges from 25–30 to 38% of total protein. Discrepancies in data are associated with the lack of precise isolation methods and the possible admixture of other proteins [10, 17]. The isoelectric point of myosin is around pH 5.4. The molecular weight, determined by ultracentrifugation, varies from 420,000 to 840,000 depending on conditions (protein concentration, temperature, presence of disaggregating agents). Under the action of urea or guanidine, myosin macromolecules break down into subunits with a molecular weight of about 165,000.

Myosin belongs to the SH-enzymes. Blockade of SH-groups with thiol poisons (Cu, Hg, Cd, salyrgan, etc.) suppresses its ATPase activity, ability to bind to actin, and contractility of actomyosin threads. These effects are reversible upon the action of antidotes (cysteine, dimercaptopropanol). It is assumed that different SH-groups are responsible for different functions: interaction with actin and ATPase activity. A peptide composing part of the ATPase center has been isolated, with the amino acid sequence: aspartic acid — cysteine (SH) — tyrosine — arginine — lysine — valine — glycine — glutamic acid [6]. Tryptic digestion of myosin yields two fragments: heavy meromyosin (H-meromyosin, ~57%, M.w. ~232,000–324,000) and light meromyosin (L-meromyosin, ~43%, M.w. ~96,000–126,000). ATPase activity and the ability to bind to actin are inherent in H-meromyosin, while contractile properties are associated with L-meromyosin.

Myosin is an ATPase, catalyzing the hydrolysis of ATP to ADP and inorganic phosphate. Activity is manifested only in the presence of salts: myosin ATPase is activated by Ca^{2+} ions and inhibited by Mg^{2+} (unlike actomyosin ATPase, which is stimulated by Mg^{2+}). In a salt-free medium, ATP hydrolysis does not occur. The rate of ATP hydrolysis is not constant: in the first seconds, it is 2–5 times higher than in the stationary period. The ATPase activity of myosin in 0.6 M KCl sharply increases in the presence of EDTA, probably due to the binding of inhibitory metal ions [14, 17].

Attempts to separate ATPase activity from myosin have been unsuccessful. High pressure (4000 atm) inactivates myosin (ATPase activity and the ability to bind actin are lost), which is associated with inactivation of functional groups, not with the separation of the enzyme.

Actin

Upon prolonged extraction of a muscle homogenate with 0.5–1.0 M KCl, actomyosin—a complex of myosin with actin (ratio in actomyosin approximately 3:1)—passes into solution. Actin is extracted with water

from muscles previously treated with 0.6 M KCl (to remove myosin) and acetone (to denature myosin residues). Actin accounts for about 20% of myofibrillar proteins [9]. Actin exists in two forms: globular (G-actin) and fibrous (F-actin). Polymerization of G-actin into F-actin (filamentous form) is induced by Mg^{2+} ions and KCl. The molecular weight of actin is about 56,000–70,000; a dimeric form (140,000) is possible. G-actin is bound to ATP, which is dephosphorylated to ADP during polymerization. Actin itself does not possess ATPase activity [13]. Actin is capable of combining with myosin in vitro to form viscous actomyosin. This reaction lacks species specificity. High pressure (1000 atm) inactivates actin, depriving it of its ability to bind to myosin [14, 15].

Actomyosin

Actomyosin ("natural" or "synthetic"—obtained from myosin and actin) possesses unique properties. When the ionic strength is lowered, its solutions form gels, including in the form of threads. Such threads retain ATPase activity. Upon addition of ATP under conditions of low ionic strength (0.05 M KCl, 0.001 M $MgCl_2$), a sharp contraction (syneresis) of the gel occurs, with water being squeezed out. At high ionic strength (0.6 M KCl), ATP causes the dissociation of actomyosin into actin and myosin, which is accompanied by a decrease in viscosity. After ATP is hydrolyzed, the viscosity is restored, and the process can be repeated many times. Syneresis is induced only by nucleoside polyphosphates with high-energy bonds, whereas dissociation can be caused by other compounds as well (e.g., pyrophosphate) [5]. The syneresis of actomyosin gel under the influence of ATP was for a long time considered a model of muscle contraction. Critics pointed to the isodimensional nature of gel contraction (volume decrease) in contrast to the anisodimensional contraction of muscle (length decrease at constant volume). However, the contraction of glycerinated muscle fibers (a model as close as possible to living muscle) under the influence of ATP occurs anisodimensionally and is accompanied by the performance of work [6].

The differences in the nature of contraction are explained by the different organization of the proteins. In an actomyosin gel, micelles are randomly arranged, and their dehydration upon interaction with ATP leads to uniform shrinkage. In a myofibril, the actin and myosin filaments are strictly oriented, and interaction with ATP leads to their sliding relative to each other (according to modern concepts) or to a change in conformation without loss of water from intermicellar spaces [2, 3]. Attempts to obtain structured threads that contract anisodimensionally (Portzehl method, film threads) have shown that their contraction is either an artifact related to partial denaturation and elasticity of stretched structures, or is also fundamentally based on a syneresis mechanism. More precise measurements have shown that film threads also lose water during contraction (their specific gravity increases), and the absence of a diameter decrease is explained by water retention in structural folds [16]. Thus, there is no fundamental difference between the mechanisms underlying the syneresis of actomyosin gel and the contraction of myofibrils. Dehydration is a concomitant effect of a more fundamental process—the transition of the actomyosin complex to an "excited" state upon interaction with ATP, which underlies muscle contraction. This conclusion is confirmed by the direct parallelism between the contractile ability of muscles and the presence in them of actomyosin capable of syneresis in the presence of ATP [1, 12].

Conclusion

Data on the chemical composition of striated musculature confirm that muscle tissue proteins represent a highly integrated system, structurally and functionally adapted to perform the contractile function [1, 4]. Sarcoplasmic proteins, constituting about a third of all muscle proteins, form a complex enzyme system providing the energy supply for muscle activity [3, 9]. In contrast, myofibrillar proteins—myosin, actin, actomyosin—form the structurally ordered contractile apparatus of the cell [5, 6]. The key functional properties of these proteins are ATPase activity, the ability for reversible interaction with each other, and for conformational changes in the presence of ATP [10, 14].

The central element of the molecular mechanism of muscle contraction is the cyclic interaction of actin and myosin, coupled with ATP hydrolysis [2, 17]. Analysis of various model systems—from actomyosin gels to glycerinated muscle fibers—shows that the basis of contraction is the transition of the actomyosin complex to a qualitatively different state upon ATP hydrolysis, which ensures the generation of mechanical force [11, 13]. The observed physicochemical changes, such as syneresis and dehydration, are concomitant manifestations of this fundamental process, whereas in the structurally organized sarcomere system, these conformational changes are transformed into directed filament sliding [7, 16]. Thus, the contractile function of muscle is determined by its specific protein composition, where each component has a strictly defined role, and their coordinated interaction ensures the efficient conversion of the chemical energy of ATP into mechanical work [8, 15].

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