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BIOPHYSICS OF SHORT SYSTEMS

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ABSTRACT

This article discusses the molecular mechanisms of contraction, muscle mechanics, muscle mechanics, and the theoretical aspects of cell mobility. The sarcomere is the main motor structure of the muscle and comprises thick and thin fibers. Fine fibers comprise actin protein and thick fibers of myosin protein. The myosin molecule has functional parts - "hinges". The heads of the transverse bridges move like a paddle, moving the actin filaments into the myosin space. The amplitude of the bridge movements is 20 nm, the frequency is 5-50 oscillations per second. The diameter of the transverse tube is 50 nm. In the muscle fibers of vertebrates, these tubes approach the myofibrils in the area of the discs.

KEYWORDS: *Cell, Organism, Mechanical, Energy, Pigment, Muscle, Contraction, Animal, Actin, Myosin.*

INTRODUCTION

Molecular mechanisms of contraction. Muscle mechanics.

Cells and organisms move and do mechanical work. We carried this work out under isothermal and isobaric conditions, in which heat and chemical energy serve as a source of energy.

Within the fibers of the transverse skeletal muscle are many myofibrils. They are 1-2 mm in diameter and comprise sarcomeres. They bound each sarcomere by a Z-sphere. The length of the sarcomeres is 2.0 μm. The inner part of the myofibrils is called the sarcoplasm, which contains mitochondria and the endoplasmic reticulum. The sarcomere is the main motor structure of the muscle and comprises thick and thin fibers. Fine fibers comprise actin protein and thick fibers of myosin protein. The myosin molecule has functional parts - "hinges". One part of this molecule is in the body of the thick fiber and the other part is on its outer side. In severe myositis, there are active and action-binding centers. When a muscle fiber is activated, Ca ++ binds to the control complex of thin fibers, and as a result, the active center of these fibers opens and the bridges of myosin connect with these active centers. The bridges of the thick fiber structure do not change and connect with the active centers of the thin fiber. At rest, the bridges perpendicular to the thick fiber bend at a certain angle during contraction. As the bridges bend, the thin fiber shifts. In contraction, the thick and thin fibers move relative to each other. **[1]**

Muscle mechanics

All sarcomeres have transverse bridges that connect the myosin filaments to the action filaments. When muscle fibers contract, the myosin and actin filaments do not shorten, the actin filaments slide between the myosin filaments, resulting in the discs shortening, and the length of the discs not changing. **[2]**

Myosin filaments branch off to form multiple heads, each comprising about 150 myosin molecules. These heads are tufts of myosin filaments that bind them to action filaments. The heads of the transverse bridges move like a paddle, moving the actin filaments into the myosin space. The amplitude of the bridge movements is 20 nm, the frequency is 5-50 oscillations per second. Although the bridges move asynchronously, the gravitational force that results from being too much is kept constant during contraction. **[3]**

At rest, we enrich the bridge with energy, but cannot bind to the actin filament, which is interrupted by the tropomyosin filament bound to the troponin protein between them. When a muscle is activated, we form free Ca ++ ions in its myoplasm. Troponin binds to calcium, changes its conformation, and pushes the tropomyosin thread, allowing the diurnal bridges to bind to the action threads. As a result of the coupling, the conformation of the bridge changes abruptly, its head is bent, and the action strip is pushed 20 nm. The energy expended for this action is released due to the macroregion phosphate bond in the phosphorylated actomyosin. Actomyosin, which has ATF-aza activity, promotes the breakdown of macroergic phosphates. **[4]**

The actin and myosin then separate from the tropomyosin troponin due to a decrease in the amount of Ca ++ around the filaments and again become a barrier between the transverse bridge and the actin filament. Myosin is phosphorylated by ATF. ATF is a substance needed not only to enrich myosin into energy but also to temporarily separate the strands from each other. This separation allows the muscle to relax and stretch. **[5]**

Ca ++ ions required for contraction are stored in the sarcoplasmic reticulum at rest in the muscle. In this case, the permeability of the reticulum membrane to calcium is low, a small amount of ions released into the myoplasm is pumped by a calcium pump into the sarcoplasmic reticulum, where it maintains a high concentration of calcium. The concentration of calcium ions in the sarcoplasmic reticulum cavity is higher than in the sarcoplasm, and the activation of the sarcoplasmic reticulum membrane leads to the opening of calcium channels in it and the release of calcium around actin and myosin filaments according to the concentration gradient. For reticular membrane activation, the excitation generated in the outer membrane of the muscle fiber must propagate through the T-system to the sarcoplasmic reticulum membrane. The T-system is the part of the outer membrane that sinks into the sarcomere. The diameter of the transverse tube is 50 nm. In the muscle fibers of vertebrates, these tubes approach the myofibrils in the area of the discs. **[6]**

Perpendicular to transverse tubes, elongated tubes are located parallel to myofibrils. The two ends of the elongated tubes widen to form cisterns. The transverse tube and the cisterns on both sides are joined in threes.

The impulse to reach the muscle fiber through the nerve fiber creates motion potential in the outer membrane, which potentially propagates through the diaphragm, activates the cistern

membrane, and causes calcium ions to exit, actin and myosin to multiply around the fibers and activate the contraction mechanism. **[7]**

The processes that lead to muscle contraction are exposure, formation of the action potential, its transfer into myofibrils, the release of calcium ions and diffusion around actin and myosin filaments, sliding of actin filaments between myosin filaments and shortening of the sarcomere, activation of calcium channels, and activation of calcium channels. **[8]**

The length of the sarcomere of a loose muscle fiber is 3.6 μm, and 2.0 - 2.2 μm when the fiber is shortened.

Muscle-free forms of cell motility

In non-cellular forms of cell motility, actin and myosin protein enter the cell structure and regulate their movement - the amoebic movement of platelets, leukocytes, fibroblasts, and similar cells; as well as intracellular movements, such as chromosome proliferation, endocytosis, exocytosis, all micro-level movements, and microvilli. **[9]** Unlike muscle cells, these cells have a relatively low amount of myosin, some contain only action protein, and inactive cells, such as platelets, actin makes up 20-30% of the total cytoplasmic protein. Some note that tubulin protein is also involved in muscle movement.

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