

# MORPHOFUNCTIONAL, STRUCTURAL AND BIOCHEMICAL FOUNDATIONS OF HUMAN MUSCLE TISSUE

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## Abstract

This article provides a detailed analysis of the morphological and functional properties of human muscle tissue, their structural levels and biochemical basis. Muscle tissue - smooth, cardiac and skeletal muscles - each has its own unique structure and function, and their cellular and tissue-level structures and physiological processes carried out through them are studied. Also, the energy sources of muscles, metabolic pathways (anaerobic and aerobic), ATP synthesis, creatine phosphate system and glycogenesis processes are considered from a biochemical point of view. The article covers the movement of muscle tissue in the human body, the biochemistry of the muscle contraction mechanism, clinical conditions and biochemical changes.

**Keywords:** Muscle tissue, endomysium, myofibril, sarcomere, muscle types, energy metabolism, ATP, contraction and relaxation, skeletal muscle, cardiac muscle, myosin, actin, myoglobin, muscular dystrophy, Atrophy, Creatinuria.

## Introduction

Motility is a characteristic feature of various life forms; the precise alignment and distribution of chromosomes in the mitotic apparatus, the jumping of a flea, as well as the amazing movements of human hands and the hard work of leg muscles can be demonstrated. However, not many chemical mechanisms are involved in the implementation of these diverse functions. The contractile apparatus of vertebrate skeletal muscles is the best studied system.

Functions and tasks of muscles:

- Provides movement - moves the body and its parts.
- Maintains body position - maintains upright posture and balance.
- Controls the activity of internal organs - heartbeat, breathing, digestion, etc.
- Generates heat - maintains body temperature at a normal level.
- Protects internal organs - protects against shock

Biochemistry of muscle types

- White muscle fibers (fast-twitch, Type II)



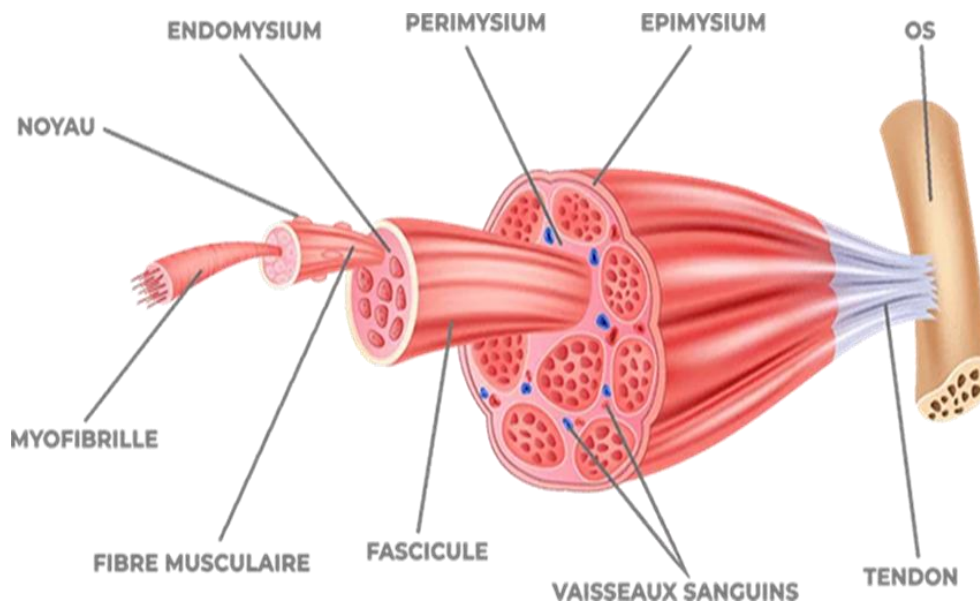


Uses anaerobic energy sources. Active in fast, powerful movements.

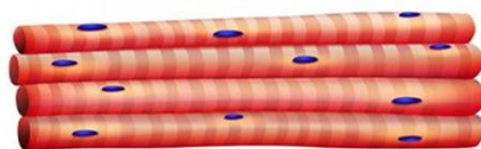
-Red muscle fibers (slow-twitch, Type I)

Many mitochondria, developed aerobic metabolism. Adapted for long-term movements

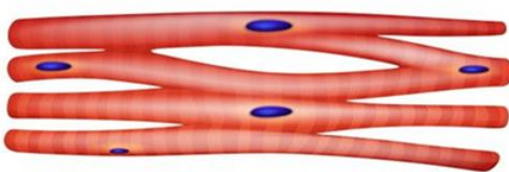
Muscle layers: epimysium, perimysium, endomysium.



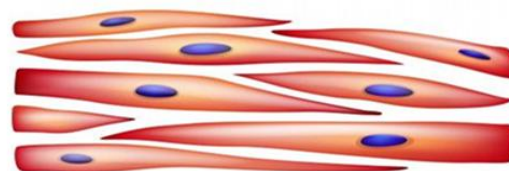
## Types of Muscle Cells



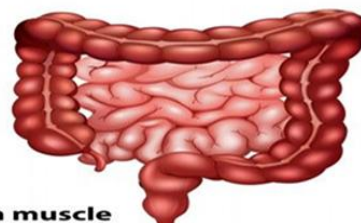
skeletal muscle



cardiac muscle



smooth muscle



**In the human body:**

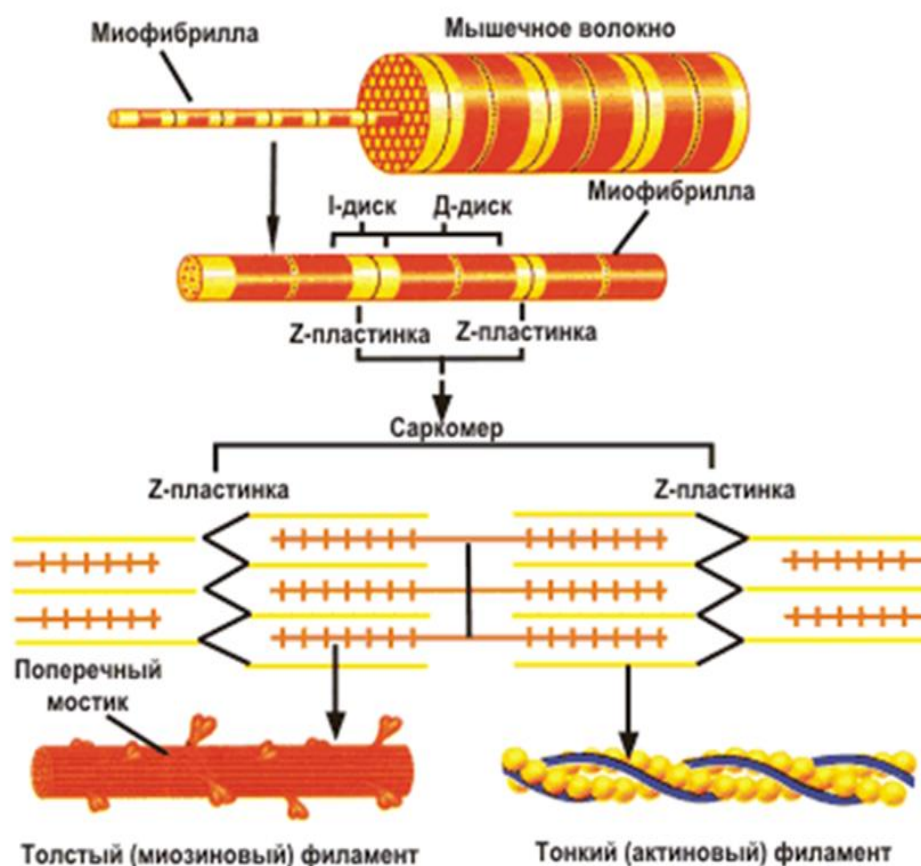
There are 3 types of muscles, which differ from each other as follows:

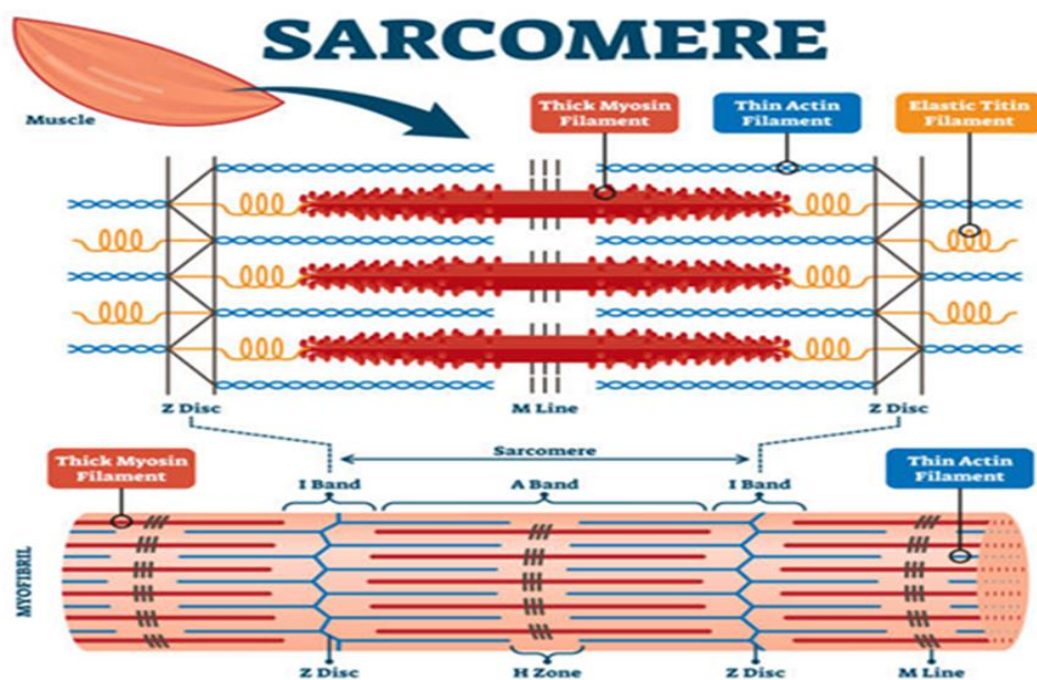
- Skeletal muscle;
- Cardiac muscle;
- Smooth muscle.

Muscle fibers - are made up of transversely arranged fibrils with a diameter of 1  $\mu\text{m}$ , in which successive black and white discs are visible. Black discs have the ability to refract light and are called A-(anisotropic) discs; white discs do not have the ability to refract light. They are called I-(isotropic) discs. In the middle of the I disc is a dense Z line with a width of about 80 nm. This line crosses the fiber along the entire cross section, holds the fibrils in one bundle, and at the same time regulates the location of many fibrils A- and I-discs.

The presence of myofibril structures as aggregates was determined under an electron microscope. They consist of thick filaments with a diameter of 14 nm, a length of 1500 nm and a distance of 20-30 nm from each other, and thin filaments with a diameter of 7-8 nm between them.

In a resting state, there are no thin filaments in the H zone, and no thick filaments in the I disk. Thick filaments consist of myosin, thin ones - actin. During contraction, the sarcomere shortens by 25-30% compared to its initial length. At maximum contraction, the opposing thin filaments slide past each other and partially cover each other. At the same time, the ends of the thick filaments are connected to both Z-lines of the sarcomere.





### Contraction:

Main ingredients: ATP,  $\text{Ca}^{2+}$  (calcium), actin, myosin.

Process:

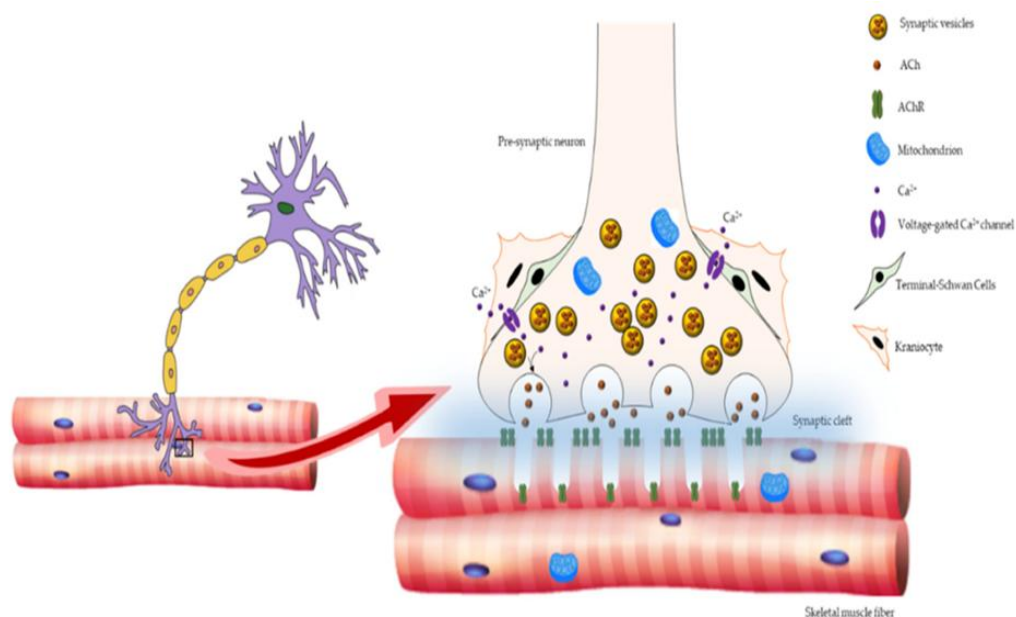
Nerve impulse arrives at the muscle.

$\text{Ca}^{2+}$  is released from the sarcoplasmic reticulum.

$\text{Ca}^{2+}$  binds to troponin → tropomyosin leaves the actin pathway.

The myosin head breaks down ATP, receives energy, and binds to actin.

Myosin moves → muscle contracts.





After receiving a nerve impulse, the permeability of the sarcoplasmic reticulum membrane changes rapidly and  $\text{Ca}^{+2}$  ions are released into the sarcoplasm. The muscle contraction that occurs in this case depends on the ability of myofibrils to bind to ATP when the  $\text{Ca}^{+2}$  concentration is  $10^{-5}$  –  $10^{-6}\text{M}$ . The sensitivity of the actomyosin system to  $\text{Ca}^{+2}$  ions depends on the presence of the troponin protein in the actin filaments. Calcium binds to troponin and causes conformational changes in its molecule. This leads to the movement of the troponin-tropomyosin complex in the F-actin network, and the active centers of actin are unblocked and become capable of binding to myosin.

Relaxation: The nerve impulse stops.

$\text{Ca}^{2+}$  returns to the sarcoplasmic reticulum (with the help of ATP).

Troponin and tropomyosin return to their previous state.

Actin and myosin separate, and the muscle relaxes.

When the calcium concentration decreases, free tropomyosin blocks the active site of actin, preventing the formation of actomyosin. This is the main reason for muscle relaxation.

### Important:

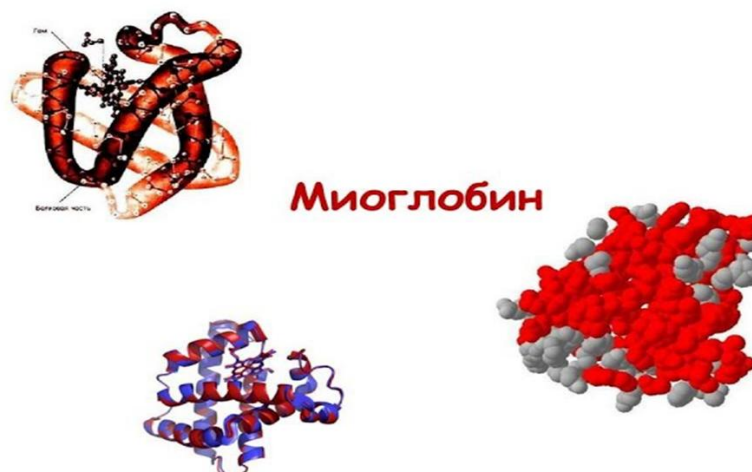
ATP is necessary for both processes (contraction and relaxation).

If ATP is absent (for example, in death) → muscles do not relax.

Specific aspects of energy metabolism in muscles Energy for muscle contraction and relaxation is provided as ATP. Reserve energy is in the form of small amounts of ATP and creatine phosphate. This reserve lasts for 10-12 seconds. When the muscle works continuously, after 40-50 seconds, anaerobic breakdown of glycogen reaches its peak, and after 60-70 seconds, aerobic processes predominate due to increased  $\text{O}_2$  transport to the working muscle. ATP is formed as a result of aerobic breakdown in mitochondria, which surround muscle fibers in large numbers. ATP resynthesis occurs due to the transphosphorylation of ADF with creatine phosphate. These reactions are catalyzed by the enzyme creatine kinase:  $\text{Creatine phosphate} + \text{ADF} \rightarrow \text{creatine} + \text{ATP}$  This ATP resynthesis pathway is very fast and efficient. The total ATP content in the muscle is approximately  $5 \mu\text{mol}$  per 1 g of muscle mass. When ATP synthesis stops, this amount is sufficient for approximately 445 1-second work. This suggests that about  $5 \mu\text{mol}$  of ATP should be synthesized per second per 1 g of muscle. Based on this, if 1/3 of the muscles in the body (about 10 kg of muscle) are used and the work lasts 10 minutes, it can be calculated that about 1.5 kg of ATP will be synthesized (and the same amount will be converted to ADP) during this time. This figure is an approximate value and depends largely on the intensity of the work.

The amount of ATP and creatine phosphate in the heart muscle is lower than in skeletal muscle, and ATP consumption is higher. Therefore, ATP resynthesis in the myocardium is faster than in skeletal muscle. The pathway for the formation of energy-rich phosphorus compounds for the heart muscle is oxidative phosphorylation, which is associated with oxygen consumption. Therefore, the heart muscle is very sensitive to oxygen deficiency. A specific aspect of the metabolism in the heart muscle compared to skeletal muscle is the oxidation of fatty acids. In the heart muscle, 65-70% of oxygen is consumed for the oxidation of fatty acids, and 30-35% for the oxidation of carbohydrates. Oleic acid is very well oxidized in the myocardium.





The main function of the myoglobin protein is to transport oxygen in the muscles. The more work the muscle does, the more myoglobin it contains, which is why it is colored red. 14% of the oxygen entering the body is stored in myoglobin. The ability of this protein to actively bind oxygen (its oxygen binding capacity is 5 times higher than that of hemoglobin) allows it to create an oxygen reserve in muscle tissue.

Biochemical changes in muscular dystrophy and denervation In muscular dystrophy and denervation, the amount of myofibrillar and some sarcoplasmic proteins, as well as myoalbumin, is sharply reduced. The concentration of ATP and creatine phosphate, the amount of carnosine and anserine decrease. In progressive muscular dystrophies associated with the breakdown of muscle tissue, changes in the composition of muscle phospholipids are observed: the concentration of phosphatidylcholine and phosphatidylethanolamine decreases sharply, and sphingomyelin and lysophosphatidylcholine increase. During vitamin E deficiency, when muscles are denervated, their movements are limited (plaster is applied), and tendons are cut, muscle fibers are torn with great force. Muscle atrophy in vitamin E deficiency is due to damage to the membranes of muscle lysosomes by lipid oxidation products in the presence of peroxide, since in the absence of an antioxidant (vitamin E), such oxidation proceeds much more actively.

### Muscle Atrophy Can Be Reversed



Atrophy is a decrease in the size of organs and a change in the quality of cells due to impaired tissue nutrition. Physiological and pathological atrophy are distinguished.

Physiological atrophy occurs as a result of the cessation of organ function. For example, when the body stops growing, the pituitary gland atrophies and adipose tissue increases. As a person ages, as a result of the weakening of organ function, the size of the glands and internal organs decreases, the skin loses elasticity, and wrinkles appear.

Pathological atrophy is caused by various diseases (tuberculosis, tumors, poisoning, etc.). In this case, the size of the organs decreases. If the causes of atrophy are eliminated, the organs can return to their original state, including after a fracture heals, atrophied muscles recover and movement normalizes. If atrophy is not prevented, it can be life-threatening. For example, optic nerve atrophy leads to blindness, muscle atrophy leads to paralysis, and gonadal atrophy leads to infertility.

**Creatinuria** - a change in creatine metabolism and its large excretion in the urine (creatinuria) is characteristic of pathologies of muscle tissue. Its amount can reach 2 g per day. In patients with myopathy, creatinuria is the result of impaired retention and phosphorylation of creatine in skeletal muscle. If creatine phosphate synthesis is impaired, creatinine is not formed and its amount in the urine decreases sharply. As a result of creatinuria and impaired creatine synthesis, the urinary creatine index keratin/keratin increases sharply.

The importance of studying morphofunctional and structural foundations

The study of the types of muscle tissue (skeletal, cardiac, smooth muscles) and their microscopic structure helps in diagnosing various diseases. The functional significance of structures such as the cell nucleus, myofibrils, sarcolemma, sarcoplasmic reticulum, and mitochondria is studied. The processes of muscle contraction, energy production, and recovery are studied in depth. The physiological processes related to movement, strength, endurance, and muscle tone are analyzed.

The importance of studying biochemical foundations

Analysis of energy metabolism mechanisms such as ATP, creatine phosphate, glycolysis, oxidative phosphorylation, and lactic acid production are among the main areas of muscle activity. Study of the synthesis of proteins (actin, myosin, troponin, tropomyosin) in muscle tissue and their functional balance. Biochemical factors affecting muscle growth, regeneration, and atrophy - for example, the role of hormones (testosterone, IGF-1), cytokines, and nutrients.

Application in sports and physical training: By studying the mechanism of muscle function in depth, it is possible to create optimal loading and recovery strategies for athletes. Specific, scientifically based programs are created to increase muscle mass and strength.

In medicine and rehabilitation: The causes and treatment of muscle diseases such as muscle atrophy, myopathies, and dystrophies are determined. The possibilities for studying regeneration and prosthetic-muscle interfaces in neuromuscular diseases are expanded.

In the direction of gerontology: Individual prevention and therapy are developed to prevent age-related muscle weakness (sarcopenia).

Future prospects

Artificial muscles: Artificial muscles developed using biotechnological methods are important for prostheses and robotics.



Muscle biosensors: Sensors that measure muscle activity in real time are used in rehabilitation and sports.

### Summary

Muscle biochemistry studies the chemical processes that ensure muscle movement, energy production, contraction and recovery. Human muscle tissue, through its complex morphological and biochemical structure, provides movement and vital functions of the organism. The structure and energy metabolism processes of muscles are the basis for their effective functioning and are a guarantee of health and mobility. To maintain normal muscle function, it is necessary to study their morphofunctional and biochemical properties in depth. The main energy sources are ATP, creatine phosphate, glycolysis and fatty acids. Disorders in these processes lead to muscle weakness, pain and genetic diseases (dystrophies, myopathies). Muscle biochemistry plays an important role in clinical diagnosis and treatment.

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