

# **EDUCATIONAL AND METHODOLOGICAL COMPLEX OF DISCIPLINE**

## **MiF1202 «Morphology and human physiology»**

**Course – 1 Semester – 2**

**Number of credits – 11**

**Almaty 2022**

### **Lecture 9 The muscular system II**

Physiology of Skeletal Muscle, Cardiac muscle and smooth muscle

#### **Outcomes:**

1. Describe the physiological properties that all muscle types have in common
2. Explain the mechanisms of muscle contraction and relaxation
3. Describe similarities and differences in the structure and function of the three types of muscle tissue, and indicate where they are found in the body.

Muscle contraction is described by the sliding filament model of contraction. ACh is the neurotransmitter that binds at the neuromuscular junction (NMJ) to trigger depolarization, and an action potential travels along the sarcolemma to trigger calcium release from SR. The actin sites are exposed after  $Ca^{++}$  enters the sarcoplasm from its SR storage to activate the troponin-tropomyosin complex so that the tropomyosin shifts away from the sites. The cross-bridging of myosin heads docking into actin-binding sites is followed by the “power stroke”—the sliding of the thin filaments by thick filaments. The power strokes are powered by ATP. Ultimately, the sarcomeres, myofibrils, and muscle fibers shorten to produce movement.

Smooth muscle is found throughout the body around various organs and tracts. Smooth muscle cells have a single nucleus, and are spindle-shaped. Smooth muscle cells can undergo hyperplasia, mitotically dividing to produce new cells. The smooth cells are nonstriated, but their sarcoplasm is filled with actin and myosin, along with dense bodies in the sarcolemma to anchor the thin filaments and a network of intermediate filaments involved in pulling the sarcolemma toward the fiber’s middle, shortening it in the process.  $Ca$  ions trigger contraction when they are released from SR and enter through opened voltage-gated calcium channels. Smooth muscle contraction is initiated when the  $Ca$  binds to intracellular calmodulin, which then activates an enzyme called myosin kinase that phosphorylates myosin heads so they can form the cross-bridges with actin and then pull on the thin filaments. Smooth muscle can be stimulated by pacesetter cells, by the autonomic nervous system, by hormones, spontaneously, or by stretching. The fibers in some smooth muscle have latch-bridges, cross-bridges that cycle slowly without the need for ATP; these muscles can maintain low-level contractions for long periods. Single-unit smooth muscle tissue contains gap junctions to synchronize membrane depolarization and contractions so that the muscle contracts as a single unit. Single-unit smooth muscle in the walls of the viscera, called visceral muscle, has a stress-relaxation response that

permits muscle to stretch, contract, and relax as the organ expands. Multiunit smooth muscle cells do not possess gap junctions, and contraction does not spread from one cell to the next.

Electrical synapses allow current to flow from one excitable cell to the next via low resistance pathways between the cells called gap junctions. Gap junctions are found in cardiac muscle and in some types of smooth muscle and account for the very fast conduction in these tissues. For example, rapid cell-to-cell conduction occurs in cardiac ventricular muscle, in the uterus, and in the bladder, allowing cells in these tissues to be activated simultaneously and ensuring that contraction occurs in a coordinated manner.

The heart consists of two kinds of muscle cells: contractile cells and conducting cells. Contractile cells constitute the majority of atrial and ventricular tissues and are the working cells of the heart. Action potentials in contractile cells lead to contraction and generation of force or pressure. Conducting cells constitute the tissues of the SA node, the atrial internodal tracts, the AV node, the bundle of His, and the Purkinje system. Conducting cells are specialized muscle cells that do not contribute significantly to generation of force; instead, they function to rapidly spread action potentials over the entire myocardium. Another feature of the specialized conducting tissues is their capacity to generate action potentials spontaneously. Except for the SA node, however, this capacity normally is suppressed.

There are several morphologic and functional differences between cardiac muscle and skeletal muscle, but the basic contractile machinery in the two cell types is similar. As in skeletal muscle, the cardiac muscle cell is composed of sarcomeres. The sarcomeres, which run from Z line to Z line, are composed of thick and thin filaments. The thick filaments are composed of myosin, whose globular heads have actin-binding sites and ATPase activity. The thin filaments are composed of three proteins: actin, tropomyosin, and troponin. Actin is a globular protein with a myosin-binding site, which, when polymerized, forms two twisted strands. Tropomyosin runs along the groove of the twisted actin strands and functions to block the myosin-binding site. Troponin is a globular protein composed of a complex of three subunits; the troponin C subunit binds  $\text{Ca}^{2+}$ . When  $\text{Ca}^{2+}$  is bound to troponin C, a conformational change occurs, which removes the tropomyosin inhibition of actin-myosin interaction. As in skeletal muscle, contraction occurs according to the sliding filament model, which states that when cross-bridges form between myosin and actin and then break, the thick and thin filaments move past each other. As a result of this cross-bridge cycling, the muscle fiber produces tension. The transverse (T) tubules invaginate cardiac muscle cells at the Z lines, are continuous with the cell membranes, and function to carry action potentials to the cell interior. The T tubules form dyads with the sarcoplasmic reticulum, which is the site of storage and release of  $\text{Ca}^{2+}$  for excitation-contraction coupling.

### **Review questions**

1. How do gap junctions and intercalated disks aid contraction of the heart?
2. Why do the cardiac muscle cells demonstrate autorhythmicity?

3. Chemical A binds and blocks acetylcholine receptors of muscle cells. Chemical B floods the cytoplasm of muscle cells with calcium ions. Which chemical would make the best muscle relaxant, and why?
4. Why can smooth muscles contract over a wider range of resting lengths than skeletal and cardiac muscle?
5. What are the opposite roles of voltage-gated sodium channels and voltage-gated potassium channels?

**Basic literature:**

1. Saladin, Kenneth S: Essentials of Anatomy & Physiology. (2018, McGraw-Hill Education)
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5. Andersson D, Medical Terminology: The Best and Most Effective Way to Memorize, Pronounce and Understand Medical Terms: Second Edition, ISBN-13 : 978-1519066626, 2016