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BIOPHYSICAL MECHANISMS OF MUSCLE CONTRACTION

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Abstract

In this article, muscle contraction is considered from a biophysical perspective as a complex process of converting chemical energy into mechanical work, carried out through the coordinated interaction of protein structures within muscle fibers. This process is of fundamental importance for understanding the mechanisms of motor activity, regulation of physiological functions, and adaptation to physical нагрузкам. The analysis of biophysical mechanisms of muscle contraction makes it possible to reveal the features of molecular interactions that determine the strength, speed, and stability of contractions in different types of muscle tissue.

Keywords: Muscle contraction; biophysics; actin–myosin interaction; contractile proteins; mechanochemical energy conversion; calcium regulation; sarcomere; action potential; neuromuscular transmission; cross-bridge cycle; contraction force; tetanic contraction; muscle biomechanics; molecular mechanisms; energy metabolism.

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Introduction

The biophysics of muscle contraction studies the molecular and mechanical mechanisms by which chemical energy, primarily from ATP hydrolysis, is converted into mechanical work, forming the basis of body movements. Muscle contraction is mediated by the coordinated interaction of contractile proteins, actin and myosin, the cyclic formation and detachment of cross-bridges, and the sliding of filaments within sarcomeres. The regulation of calcium ion concentration, controlled by the sarcoplasmic reticulum and troponin–tropomyosin complex, plays a critical role in initiating and modulating contraction. These biophysical processes determine the strength, velocity, and endurance of contraction in different types of muscle tissue, including skeletal, cardiac, and smooth muscles.

At the cellular level, contraction involves electrochemical coupling, where action potentials propagate along the muscle membrane, triggering calcium release and activating molecular motors. The mechanochemical cycle of myosin heads generates force and movement, while energy metabolism ensures a continuous supply of ATP. Factors such as temperature, ionic concentration, and load affect the kinetics and efficiency of contraction, illustrating the intricate connection between physical laws and biological function.

Understanding the biophysical principles of muscle contraction is essential in medicine, as it informs the diagnosis and treatment of disorders such as cardiomyopathies, muscular dystrophies, and spasticity. Beyond medicine, this knowledge is applied in sports science to optimize performance, in bioengineering for the design of prosthetics and artificial muscles, and in rehabilitation technologies to restore mobility and function after injury.

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Types of Muscles and Their Morphology

1.Skeletal Muscle

Skeletal muscle is striated and consists of long, multinucleated fibers with a highly ordered arrangement of sarcomeres, the basic contractile units. Each sarcomere contains interdigitating actin (thin) and myosin (thick) filaments, whose cyclic interaction forms the basis of the cross-bridge cycle, generating force and shortening the muscle fiber. Contraction is triggered by neuromuscular excitation and mediated by excitation–contraction coupling, where calcium ions released from the sarcoplasmic reticulum bind to troponin, allowing cross-bridge formation. The biophysical parameters such as contraction velocity, force generation, and fatigue resistance are determined by fiber type, ATP availability, and the efficiency of actomyosin interactions.

2.Smooth Muscle

Smooth muscle consists of spindle-shaped cells without the regular sarcomere arrangement seen in striated muscle. The contractile apparatus is organized in a network of actin and myosin filaments anchored to dense bodies, allowing cells to contract in a coordinated but slower and more sustained manner. Contraction is regulated by intracellular calcium, which activates myosin light-chain kinase, modulating cross-bridge cycling. Smooth muscle biophysics includes mechanotransduction, enabling cells to respond to stretch, pressure, and other mechanical signals, and maintaining tonic tension essential for organ function.

3.Cardiac Muscle

Cardiac muscle is striated and consists of cardiomyocytes connected by desmosomes (mechanical stability) and gap junctions (electrical coupling). These features allow synchronous contraction of the heart. The biophysical mechanisms include excitation–contraction coupling, calcium-induced calcium release, and coordinated cross-bridge cycling. Cardiac muscle exhibits intrinsic rhythmicity,

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and force generation is finely tuned to accommodate variations in preload and afterload, ensuring efficient pumping. Mechanical properties like elasticity, contractile force, and conduction velocity are critical for the hemodynamic performance of the heart.

The Sarcomere as an Elementary Contractile Machine

A sarcomere is the fundamental structural and functional unit of striated muscle, extending from one Z-disc to the M-line. It serves as an elementary contractile machine, where the conversion of chemical energy (ATP) into mechanical work occurs via the sliding filament mechanism.

Main Components:

Actin (thin filaments): provide the track along which myosin heads slide during contraction;

Myosin (thick filaments): molecular motors that generate force through cross-bridge cycling, powered by ATP hydrolysis;

Troponin–tropomyosin complex: regulates actin–myosin interaction in a calcium-dependent manner, controlling the initiation and termination of contraction.

Titin: a giant elastic protein that spans from the Z-disc to the M-line, providing passive elasticity, restoring forces, and contributing to the centering of thick filaments within the sarcomere.

Role of Titin and the Extracellular Matrix:

Titin, along with the extracellular matrix (ECM), plays a critical role in passive elasticity, mechanical stability, and force transmission in muscle fibers. The ECM provides structural support, shear resistance, and load distribution, ensuring and minimizing damage under mechanical stress.

From a biophysical perspective, the sarcomere functions as a highly coordinated molecular machine, where elastic elements, contractile proteins, and regulatory complexes integrate to produce controlled, repeatable, and efficient mechanical

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work. Understanding sarcomere mechanics is fundamental for analyzing muscle force generation, elasticity, and response to physiological and pathological conditions.

Molecular Mechanisms of Contraction

Cross-Bridge Cycle (Actin–Myosin Model)

Muscle contraction at the molecular level is driven by the **cyclical interaction between actin and myosin filaments**, known as the **cross-bridge cycle**. This process represents the **elementary mechanochemical event** that converts chemical energy from ATP into mechanical work.

Stages of the Cross-Bridge Cycle:

1. Attachment: The myosin head binds to an available binding site on actin, forming a cross-bridge.

2. Power Stroke: Conformational changes in the myosin head cause it to pivot, pulling the actin filament toward the center of the sarcomere and generating **mechanical force**.

3. Detachment: Binding of ATP to myosin reduces its affinity for actin, causing the actin–myosin complex to dissociate.

4. ATP Hydrolysis and Re-Cocking: Hydrolysis of ATP to ADP and inorganic phosphate energizes the myosin head, restoring it to a “cocked” position ready for the next cycle.

5. Rebinding: The myosin head reattaches to a new position on actin, and the cycle repeats.

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Mechanochemical Coupling:

The **hydrolysis of ATP** induces structural changes in the myosin head that generate force and movement. The **kinetic constants** of attachment, power stroke, detachment, and re-cocking determine the **speed, efficiency, and strength of contraction**. The number of cross-bridges formed simultaneously influences **tension development,** while filament overlap within the sarcomere affects **force-length relationships.**

From a **biophysical standpoint,** the cross-bridge cycle integrates **chemical energy transduction, molecular mechanics, and filament dynamics,** ensuring **reproducible, coordinated contraction** across diverse muscle types. Understanding these mechanisms is critical for interpreting **muscle performance, fatigue, and pathological alterations** such as cardiomyopathies or myopathies.

Calcium Regulation (Excitation–Contraction Coupling)

In skeletal and cardiac muscle:

action potential → ACh release (skeletal muscle) → membrane depolarization → activation of DHP receptors (L-type Ca^{2+} channels) → Ca^{2+} release from SR via ryanodine receptors (RyR) → increased intracellular Ca^{2+} → binding to troponin C → tropomyosin shift → exposure of actin binding sites → start of cross-bridge cycling.

Calcium removal mechanisms: SERCA pump, Na^{+}/Ca^{2+} exchanger (especially in heart), mitochondrial buffering.

Electrophysiology and Neuromuscular Transmission

Local potentials, conduction velocity, presynaptic vs. postsynaptic mechanisms. Differences in excitation mechanisms in smooth muscle (pharmacomechanical coupling).

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Energy and Metabolism During Contraction

Sources of ATP

Immediate: phosphocreatine via creatine kinase — instant ATP resynthesis.
Anaerobic: glycolysis (produces lactate) — fast but low efficiency.
Aerobic: mitochondrial oxidative phosphorylation — slow but highly efficient, major source in slow-twitch fibers.
Regulation: oxygen supply, substrate availability, enzyme activity, metabolic fatigue (H^+, Pi accumulation, reduced Ca^{2+} sensitivity).

Thermodynamics and Efficiency

Mechanical efficiency ~20–30%; the rest of the energy is released as heat.
Optimal contraction velocity provides maximal power output.
Mechanical Properties and Laws of Active Contraction

Length–Tension Relationship

The **length–tension relationship** describes how the **force generated by a muscle depends on the sarcomere length**. At the **optimal sarcomere length (L_0)**, the overlap between **actin and myosin filaments** is maximal, allowing the formation of the greatest number of cross-bridges. This configuration generates the **maximum isometric force**.

At lengths shorter than L_0, excessive filament overlap causes **steric hindrance**, reducing the number of effective cross-bridges and thus **decreasing force production**. Conversely, at lengths longer than L_0, the overlap between actin and myosin diminishes, leading to **fewer cross-bridges and lower force output**.

This relationship is fundamental in biophysics and muscle physiology, as it explains the mechanical constraints on force generation and informs models of muscle performance, sarcomere mechanics, and contractile efficiency across skeletal, cardiac, and smooth muscles.

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Force–Velocity Relationship

Hill's equation: $(F + a)(v + b) = (F_0 + a)b$

Power is maximal at ~1/3 of Vmax.

Viscoelastic Properties

Determined by titin, ECM, intracellular viscosity.

Models: Maxwell, Kelvin–Voigt.

Mathematical and Physical Models of Contraction

Huxley Model (1957)

Kinetic model describing the distribution and transitions of cross-bridges; predicts force–velocity and force–time curves.

Hill Model

Empirical model widely used in biomechanical simulations.

Multiscale Models. Integration of molecular kinetics, calcium signaling, and whole-organ mechanics (e.g., cardiac modeling).

Research Methods. Laboratory Techniques

Skinned fibers, micro-tensometry, Ca^{2+} fluorescence (Fura-2, Fluo-4), respirometry, patch-clamp.

Imaging and Molecular Methods

Confocal microscopy, cryo-EM, X-ray crystallography, fluorescent protein tagging.

Biomechanical and Clinical Measurements

EMG, dynamometry, MRI elastography.

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Pathological Aspects

Cardiomyopathies. Ca^{2+} dysregulation (SERCA, RyR defects), fibrosis, titin mutations → impaired systolic/diastolic function.

Myopathies

Mutations in actin/myosin/titin/dystrophin → impaired force transmission, fragility; mitochondrial diseases → reduced endurance.

Neuromuscular Disorders

Myasthenia — reduced ACh receptor function → decreased excitation and contraction.

Applied Directions and Perspectives

Rehabilitation and Sports Science

Personalized training, EMG-based feedback, ATP recovery kinetics.

Pharmacology and Therapy

Targeting SERCA, RyR, ion channels, metabolic pathways; genetic therapy of sarcomeric protein mutations.

Future Research

Multilevel modeling, real-time cellular energetics, mechanotransduction, omics integration.

Practical Recommendations

Consider fiber type, tissue source, temperature, and metabolic conditions.

Combine skinned fiber experiments with Ca^{2+} imaging and protein analysis.

Use empirical data (force curves, Vmax) for model calibration

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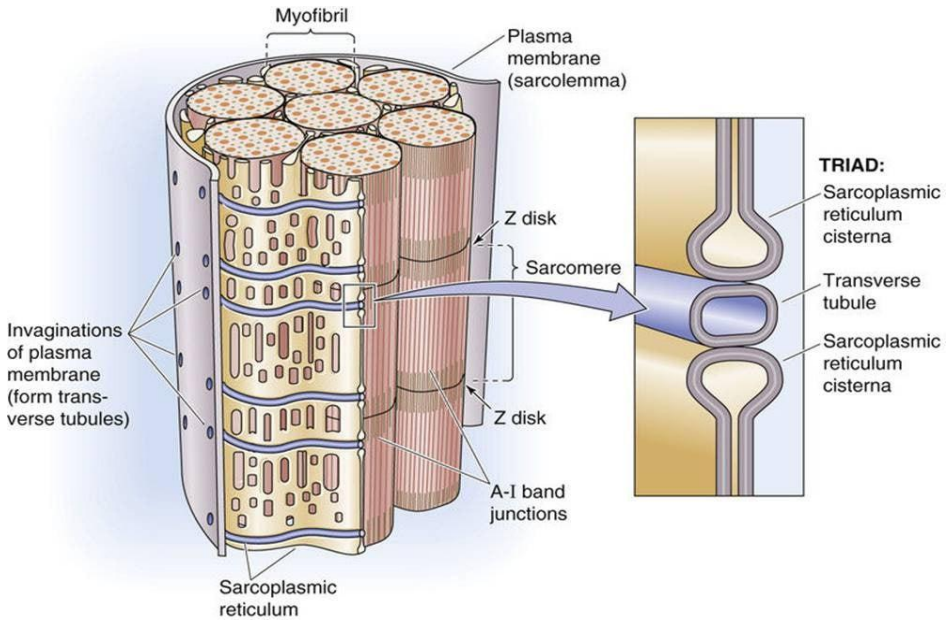
1. Molecular Mechanism of Contraction

One gram of skeletal muscle tissue contains about 100 mg of contractile proteins — actin and myosin. Their interaction during the elementary act of muscle contraction is explained by the sliding filament theory developed by Huxley and Hanson.



Function of Cross-Bridges

A. Model of contraction: myosin filament with cross-bridges attached to neighboring actin filaments (top — before the stroke, bottom — after).

B. Model of force generation by cross-bridges (left — before, right — after the power stroke).

Cross-bridges correspond to the heavy meromyosin portion of myosin, which consists of subfragment I (head) and subfragment II (neck).

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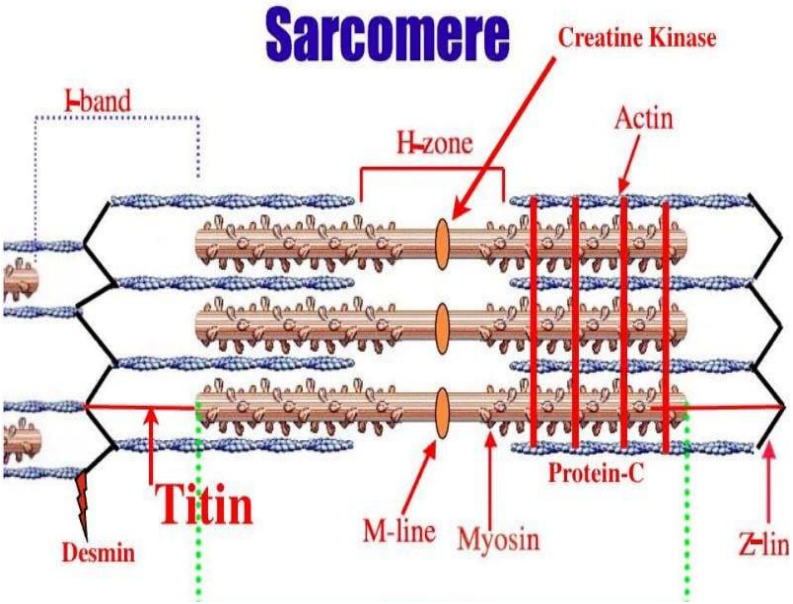

Conclusion

The biophysics of muscle contraction is a critical field that integrates molecular, cellular, and organ-level mechanisms underlying muscle function. Understanding the actin–myosin interaction, calcium regulation, cross-bridge cycling, sarcomere mechanics, and energy metabolism provides deep insight into how muscles generate force, perform work, and adapt to varying physiological and mechanical loads.

From a biophysical perspective, muscle contraction is a coordinated process in which chemical energy from ATP is transduced into mechanical work through conformational changes in myosin heads, regulated by intracellular calcium dynamics and modulated by passive elastic elements such as titin and the extracellular matrix. The kinetics of cross-bridge formation, detachment, and re-cocking determine contraction velocity, strength, and efficiency, while sarcomere architecture ensures optimal force transmission.

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This knowledge is essential for fundamental biology, medicine, sports science, and bioengineering. Biophysical data are applied in diagnosing and treating cardiomyopathies, myopathies, and neuromuscular disorders, as well as in the design of artificial muscles, prosthetics, rehabilitation devices, and performance optimization.

Thus, research in the biophysics of muscle contraction provides a foundation for integrating molecular structure, physiological regulation, and mechanical function of muscle tissue, opening new avenues for clinical applications, applied biomechanics, and bioengineering innovations.

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