RESEARCH PAPER

Reverse Sliding Filament Theory

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ABSTRACT

The Sliding Filament Theory (SFT) has long served as the foundational model for explaining muscle contraction, primarily focusing on the one-way interaction between actin and myosin filaments during concentric (shortening) contractions. However, this traditional model does not fully explain the complex behaviors of muscles during eccentric (lengthening) contractions, relaxation, elastic recoil, or injury. These limitations create a conceptual gap in understanding muscle mechanics, particularly the bidirectional and reversible nature of filament movement. To address this gap, the present study introduces the Reverse Sliding Filament Theory (RSFT), which proposes a more comprehensive mechanism encompassing the full spectrum of muscle activity.

The study aims to conceptualize and describe the Reverse Sliding Filament Theory (RSFT) as an extension of existing muscle contraction models. It employs a theoretical and analytical design, integrating insights from molecular biology, biomechanics, and neurophysiology. The framework emphasizes the role of the neuromuscular junction and the transmission of neural impulses that regulate both forward (contraction) and reverse (relaxation or recovery) filament movements. The RSFT model examines muscle behaviour at the microscopic level, considering actin–myosin interactions during concentric, eccentric, and isometric contractions, as well as during stretching, recovery, and injury processes.

The RSFT framework reveals that muscle function is not unidirectional but rather bidirectional, with filaments capable of both forward and reverse motion. This theory explains how muscles return to a state of rest from a state of tension through molecular backward movement. It also elucidates the physiological basis of eccentric contractions, the mechanical aspects of elastic recoil, and the processes leading to filament-level injuries. Furthermore, RSFT accounts for the influence of neural instructions on muscle regulation and tension control, providing a holistic view of muscle behaviour across various states of activity.

The Reverse Sliding Filament Theory (RSFT) offers a more complete and dynamic understanding of muscle mechanics by explaining both contraction and relaxation mechanisms at the microscopic level. It fills critical gaps left by the traditional SFT, especially in relation to eccentric stress, injury formation, and recovery processes. This theory holds significant implications for fields such as kinesiology, biomechanics, sports medicine, and bioengineering, potentially advancing the modelling of muscle behaviour, improving injury prevention strategies, and refining rehabilitation and performance optimization approaches.

Key words: Sliding Filament Theory, Muscle, Injury, Reverse Sliding Filament Theory, Muscle Contraction, Eccentric Contraction, Muscle Relaxation, Stretching, Actin, Myosin, Titin, Golgi Tendon Organ

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INTRODUCTION

Muscles are of three types but the proposed study concentrates on skeletal muscle movement explanation only. Sliding Filament theory and other research including stretching and Three Filament Model give a wide range of movement types in a muscle. The proposed study gives an explanation about the point of injury, mechanism of muscle relaxation, energy storage and energy usage points in the process of complete muscle movement. Emerging knowledge from biomechanics, molecular imaging, and neuromuscular physiology indicates muscle recovery is more than a return to baseline, there are intricate interactions among structural proteins, proprioceptive signaling, and elastic energy control. This Perspective presents new Reverse Sliding Filament Theory (RSFT), a framework, bidirectional muscle dynamics.

METHODS

This paper is based on thematic analysis with a qualitative research design. Thematic analysis helps to uncover themes or patterns in the information by identifying and analysing them and reporting them appropriately, which is very helpful in the general exploration of deep and subjective health issues, especially when related to women police officers.

MUSCLE CONTRACTIONS

Muscle contraction is the process by which muscle fibres generate tension and shorten, leading to movement or maintaining posture. It occurs when muscle fibres respond to signals from the nervous system, specifically through a sequence of events involving the interaction of proteins, mainly actin and myosin. With the help of this protein observation regarding the concentric and isometric are explained but

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to examine the eccentric muscle contraction , titin comes into the light , providing the structural stability (Huxley, 2004b).

CONCENTRIC CONTRACTION

A concentric contraction occurs when a muscle shortens as it contracts. Myosin heads bind to actin filaments, forming "cross-bridges". These myosin heads then pivot, pulling the actin filaments toward the center of the sarcomere. ATP provides the energy for the myosin heads to release from actin, re-cock , and bind to a new site on the actin filament to continue the process. Calcium binds to the protein troponin, which moves the protein tropomyosin away from the actin binding sites, allowing myosin to attach to actin. This cycle repeats, causing the sarcomere to shorten, leading to muscle contraction (J. E. (John E. Hall et al., 2026).

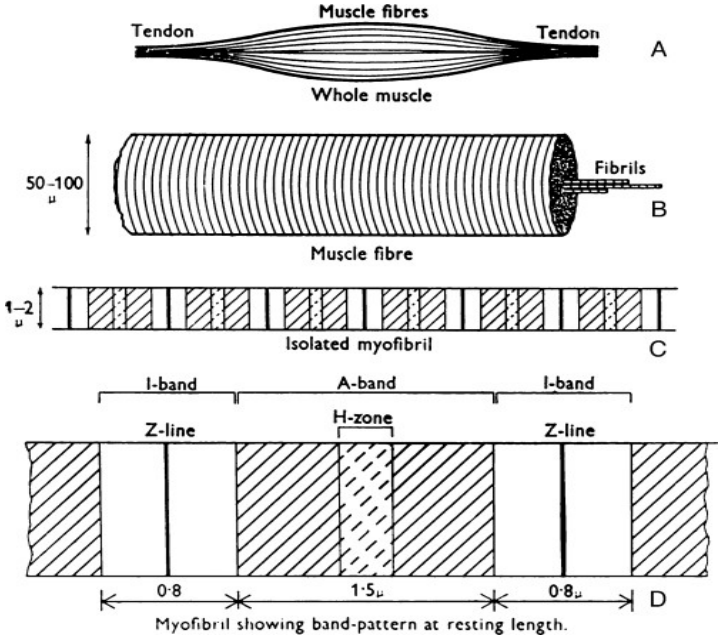

Figure 1: Skeletal Muscle (Huxley, 2004a)

ECCENTRIC CONTRACTION

The fundamental mechanism by which titin elongates during low-force stretching (eccentric contraction) of the sarcomere is the straightening of interdomain linkers in the Ig domains. However, the PEVK (Proline–Glutamate–Valine–Lysine) element plays a key function in stretching during high-force stretching (eccentric contraction), giving titin spring-like characteristics and allowing it to absorb external forces. Additionally, as a regulator of sarcomere force, the PEVK element's spring stiffness increases with increasing cytoplasmic Ca2+ concentrations. Researchers believe that titin winding around the narrow filament via cross-bridge rotation is the underlying regulating mechanism. The elastic region gets shorter as a result of this process, which raises the PEVK element's spring stiffness.

Binding to the thin filament does not cause the increase in titin stiffness that occurs when cytoplasmic Ca2+ levels are increased. Rather, it is linked to Ca2+ binding to the PEVK element, which inhibits titin elongation and encourages the distal end of titin to the central M- band of the sarcomere.

Notably, Labeit et al. examined recombinant PEVK molecules with 28-residue PEVK repeats and E-rich motifs using a skinned fibers model (mice soleus). They found that Ca2+ could bind to the E-rich motif of the PEVK element at the distal end of titin, giving titin more stiffness(Qian et al., 2024a).

ISOMETRIC CONTRACTION

Isometric contractions are contractions in which there is no change in the length of the muscle. No joint or limb motion occurs (“Therapeutic Exercise,” 2021).

RELAXATION OF MUSCLE FIBRE

Muscle force production occurs within an environment of tissues that exhibit spring-like behavior, and this elasticity is a critical determinant of muscle performance during locomotion. Muscle force and power output both depend on the speed of contraction, as described by the isotonic force-velocity curve. By influencing the speed of contractile elements, elastic structures can have a profound effect on muscle force, power and work. In very rapid movements, elastic mechanisms can amplify muscle power by storing the work of muscle contraction slowly and releasing it rapidly (Roberts, 2016). Biomechanical principle - Elastic potential energy converts to kinetic energy which brings the muscle fibre back to their resting state. While it is proven that muscles produce elastic potential energy in the movements requiring much force (such as jump) to amplify their performance in sports (explosive strength). When the

contraction is comparatively weaker less EP energy is generated.

The resting tension of the muscle while the body is in deep sleep is much lower to the resting tension of the muscle while the body is awake. Similarly, any state of the muscle activity compared to a lower activity state is mistakenly understood and interpreted as rest or relaxation of the muscle fiber. Due to reciprocal inhibition the muscle fibres work in sync. Resting skeletal muscle tone is an intrinsic viscoelastic tension exhibited within the body's kinematic chains. It functions inseparably from fascial (i.e., myofascial) tissues and ligamentous structures (Masi & Hannon, 2008)

Conventional models identify relaxation of muscle as a passive decline in tension, typically the conclusion of cross-bridge cycling. However, the Reverse Sliding Filament Theory (RSFT) perceives relaxation as an active energy-dependent mechanism. It consists of the transformation of elastic energy stored into kinetic force, under the influence of titin's viscoelasticity and forces such as fluid resistance and sarcoplasmic drag within the muscle.

TENSION

Sarcomere length and amount of myosin-actin filament overlap have a direct effect on the active tension developed by a contracting muscle fiber. The figure: 2 shows the interaction of actin myosin in the length-tension graph. Different points namely: A, B, C and D at the graph clearly show the interaction between actin and myosin diminishing. Especially between points C and D the fibre is still intact while actin and myosin have been moved long past the interaction point (J. E. (John E. Hall et al., 2026).

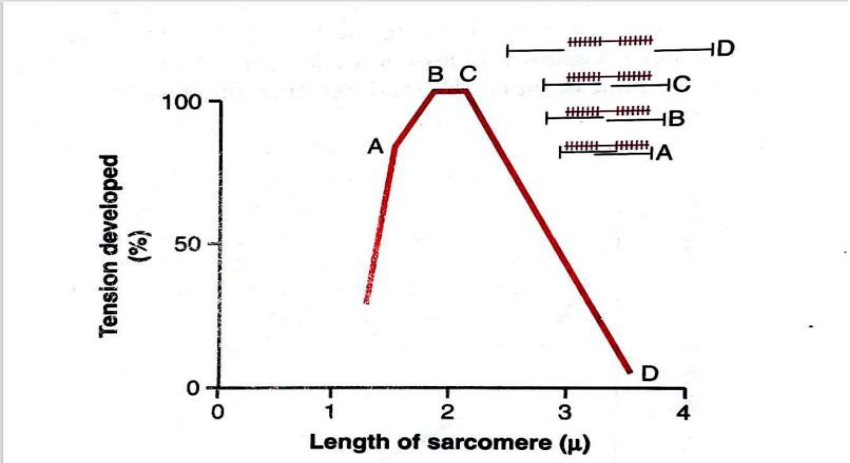

Figure 2: Length-Tension Graph (Schmidt et al., 2024)

THREE FILAMENT THEORY

The suppleness of relaxed striated muscle is attributed to the enormous muscle protein titin, additionally referred to as Connecting, which also serves as the molecular framework for the development of thick filaments (Herzog et al., 2012). Titin, which varies in size from 3.4 to 3.9 MDa1, is the biggest protein in the human body. It has crucial structural and signalling roles in contractile units and extends from the Z disk to the sarcomere's M band area (Sun et al., 2024). Titin incorporates an elastic spring-like component that joins the Z-disk and thick filament in the sarcomere's I-band. This part is essential to the formation and upkeep of the sarcomere ultrastructure because it functions as a molecular spring. The PEVK region, N2A-us region, and N2B-us region are the three intrinsically disordered areas that make up this structure, along with tandem immunoglobulin (Ig) domain repeats (Krüger & Linke, 2011). The functions of titin in the sarcomere are regulated differently by these three domains (Qian et al., 2024b).

Titin extends across the half-sarcomere, binding between the Z-disk and the M-line, and is responsible for muscle elasticity, passive stiffness, and structural integrity. Titin acts like a viscoelastic spring when it is stretched and stores elastic potential energy (EPE) that is utilized later for muscle recoiling (Herzog, 2018a). Based on the Reverse Sliding Filament Theory (RSFT), following an eccentric contraction, titin accumulates energy that is subsequently utilized in the form of kinetic energy to enable the regulated relaxation of muscle fibres (*The Role of Titin in Eccentric Muscle Contraction | Journal of Experimental Biology | The Company of Biologists*, n.d.). Specific areas of titin particularly the PEVK (*refers to Proline, Glutamate, Valine, and Lysine-rich region*) and N2A regions (*refers to the N2A region of titin, which contains immunoglobulin (Ig) domains*) are sensitive to alterations in calcium concentrations within the muscle. Upon activation of the Golgi Tendon Organ (GTO) upon eccentric loading, elevated cytoplasmic Ca^{2+} interacts with these regions, causing titin to become stiffer and assist in avoiding overstretch (Herzog, 2018b). This calcium-sensitive response differentiates titin from actin and myosin and makes it the core element in the recoil or reversal stage of contraction (Granier et al., 2014). Titin stiffness also is controlled by post-translational modifications like the phosphorylation by PKA and PKG [*the cAMP-dependent protein kinase (PKA) and the cGMP-dependent protein kinase (PKG)* are homologous enzymes with different binding and

activation specificities for cyclic nucleotides)], which vary with muscle fibre types and on the condition of the muscle (Kötter et al., 2013).

PHYSIOLOGY OF STRETCHING

Stretching and eccentric contraction are very closely related as while passive or active stretching the body resists the motion to prevent overstretching, this resistance is caused by eccentric contractions. On the contrary these contractions relax within seconds to let the muscle be able to stretch further. As mentioned in the mechanism of eccentric contractions, the delay in stiffening of the Titin is stretching irrespective of passive or active. In other words, the movement of the fibres beyond the eccentric contraction but before the extreme pain starts while the complete ROM is achieved is stretching. It is the small area between eccentric contraction and a fully stretched functional muscle fibre.

NEUROMUSCULAR JUNCTION and IMPULSES

To conduct contraction there is a specialised neuromuscular pathway and specific impulses. The neuromuscular junction (NMJ) is a chemical synapse that is anatomically and functionally differentiated for the transmission of a signal from the motor nerve terminal to a circumscribed postsynaptic region on the muscle fibre. Despite the differences, all NMJs have principal components such as a Schwann cell process, a nerve terminal, a synaptic space lined with a basement membrane, a postsynaptic membrane, and junctional sarcoplasm. In normal mature muscle, a single extrafusal muscle Fiber receives its innervation from a single motor neuron, but each motor neuron supplies more than one muscle fibber. At the lateral borders of the NMJ basement, the membrane only separates the synaptic from the extracellular space. The synaptic space is situated between the presynaptic and postsynaptic membranes ("Chapter 3 The NEUROMUSCULAR JUNCTION," 2008).

Reciprocal inhibition- inhibition of tension developed in the antagonist muscles resulting from activation of muscle spindles (S. J. Hall, 2012).

Future Directions

RSFT validation demands a multi-modal research strategy. Imaging modalities such as super-resolution microscopy and fluorescent titin tagging can monitor titin behaviour in real-time during stretch-recoil. Electrophysiology, based on GTO and Electromyography (EMG) recordings, can explain neuromechanical switching. Computational resources such as Open Sim and Any Body can model RSFT by modelling titin stiffness, recoil, and neural feedback, enhancing injury prediction. Experimental investigations of titin phosphorylation, isoforms, and calcium-binding throughout muscles can also provide insights into age-related alterations in muscle elasticity and function.

Results

1. Muscle contraction and relaxation are two opposite movements of the same muscle working in pairs such that the contraction, stretching, eccentric contraction, and isometric contraction all move to the state of tension but have the same instinct to come back to the resting state of the muscle. Hence, the reverse movement of the fiber comes into action.
2. Muscle contraction, specifically concentric contraction, is explained through SFT, which only specifies the movement of filaments during contraction.
3. As the contraction of muscle Fibers is explained by SFT, the reverse movement of the filaments is explained in the proposed study, "REVERSE SLIDING FILAMENT THEORY" which specifically focuses on the reverse movement of Actin and Myosin while different states of muscle namely; isotonic, eccentric, relaxation, stretching, and injury.
4. The study suggests that the interaction between actin and myosin does not always have to be there in every situation. The interaction only suggests the movement and direction of the filament.
5. Titin is the only filament of the three filaments that is attached medially from actin and distally from the Z Disk.
6. Injury must happen when any kind of attachment breaks cross-bridges while contraction or titin while stretching irrespective of the degree of interaction between actin and myosin filaments.
7. When SFT works Satellite cell is the "switch", but in case of stretching while RSFT is working Golgi Tendon Organ (GTO) is the "switch".
8. When GTO is switched on, calcium ions are attached to Titin to calcify it, in a similar way as to which myosin does while the satellite is switched on.
9. Muscle fibre filaments do not have the ability to push or pull, rather different filaments pull. As understood above, during SFT, myosin pulls and in RSFT Titin pulls. Therefore the filaments can only pull.
10. The delay in stiffening of the Titin is stretching irrespective of passive or active.
11. Elastic potential energy converts to kinetic energy while bringing back the muscle at resting tension.
12. Muscle fibers are never completely at rest; a baseline level of tension is always maintained, so relaxation is only relative rather than absolute.
13. What is termed "relaxation" is actually a comparative reduction in muscle tone, achieved when ATP-driven processes convert chemical energy into elastic potential energy, later released as kinetic energy during filament reversal.
14. Thus, muscle fibers transition between higher and lower tension states, but true absolute relaxation does not occur—only relative relaxation is physiologically possible.
15. Reciprocal inhibition only works when the fibre is stretched or contracted within a range. Going beyond, the elastic potential energy converted to kinetic energy, works to bring the fibre back to that range.

Discussion

The Reverse Sliding Filament Theory offers a comprehensive, bidirectional framework for understanding muscle mechanics, encompassing concentric, eccentric, and isometric actions, as well as

filament-level relaxation and injury. It has ramifications for improving injury prevention, rehabilitation techniques, and performance optimization. The theory fills in important gaps left by conventional models, redefining our biomechanical understanding of muscle function while also shedding light on the complex filament dynamics during eccentric stress and damage. The findings of the study, not all circumstances necessitate an interaction between actin and myosin to exist. Only the filament's orientation and direction of movement are suggested by the interaction. The only one of the three filaments that is connected distally from the Z Disk and medially from actin is titin. The "switch" in the case of stretching when RSFT is operating is the GTO; in the case of SFT, the "switch" is the satellite cell. Similar to how myosin calcifies when the satellite is turned on, calcium ions are linked to titin when GTO is turned on. Muscle fiber filaments are not able to push, rather different filaments just pull for different movements: myosin pulls during SFT and titin pulls during RSFT. Thus, the filaments are limited to pulling. Its implications extend to enhancing rehabilitation strategies, injury prevention and provide insights about the lesser tensed state of muscle as rest. Future experimental validation will further solidify its relevance in modern muscle physiology. This model has the potential to change muscle physiology in bioengineered movement analysis, injury prevention and athletic performance for future research.

Conclusion

With its bidirectional and energy-efficient framework, RSFT harmonizes with contemporary findings in physiology, neurology, and biomechanics. It integrates neuromechanical feedback, molecular regulation, and elastic energy dynamics to explain not only muscle contraction but also recovery and relaxation. By challenging conventional theories in muscle science, RSFT opens new possibilities for research, therapeutic applications, and interdisciplinary use. However, empirical validation and further refinement through future investigations are necessary to establish its definitive role in modern physiology.

Conflict of Interest

The authors declare no conflict of interest related to this manuscript.

Financial Disclosure

No financial support or sponsorship was received for this study.

Ethics

This research paper is based solely on previously published literature; hence, no ethical approval was required. All sources have been properly cited to ensure academic integrity and avoid plagiarism. The study maintains honesty, transparency, and objectivity in presenting and interpreting existing research.

Publication

The authors declare that this review is their original work and has not been published or submitted elsewhere. All sources are properly acknowledged, and permissions have been obtained where required. The publication rights are granted to the respective journal or institution in accordance with their guidelines and policies.

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