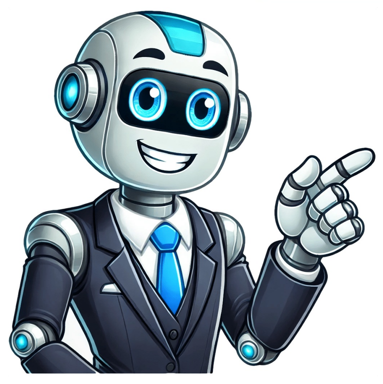

Clark, M. Milestone 3 (1954): Sliding filament model for muscle contraction. Muscle sliding filaments. Nature Reviews Molecular Cell Biology 9, s6–s7 (2008) doi:10.1038/nrm2581. Goody, R. S. The missing link in the muscle cross-bridge cycle. Nature Structural Molecular Biology 10, 773–775 (2003) doi:10.1038/nsb1003-773. Hoyle, G. Comparative aspects of muscle. Annual Review of Physiology 31, 43–82 (1969) doi:10.1146/annurev.ph.31.030169.000355. Huxley, H. E. & Hanson, J. Changes in the cross-striations of muscle during contraction and stretch and their structural interpretation. Nature 173, 973–976 (1954) doi:10.1038/173973a0. Huxley, A. F. & Niedergerke, R. Structural changes in muscle during contraction: Interference microscopy of living muscle fibres. Nature 173, 971–973 (1954) doi:10.1038/173971a0. Hynes, T. R. et al. Movement of myosin fragments in vitro: Domains involved in force production. Cell 48, 953–963 (1987) Doi:10.1016/0092-8674(87)90704-5. Lehman, W., Craig, R. & Vibert, P. Ca2+-induced tropomyosin movement in Limulus thin filaments revealed by three-dimensional reconstruction. Nature 368, 65–67 (1994) doi:10.1038/368065a0. Lorand, L. "Adenosine triphosphate-creatine transphosphorylase" as relaxing factor of muscle. Nature 172, 1181–1183 (1953) doi:10.1038/1721181a0. Spudich, J. A. The myosin swinging cross-bridge model. Nature Reviews Molecular Cell Biology 2, 387–392 (2001) doi:10.1038/35073086. The sliding filament theory explains how muscle fibres contract. The sliding filament theory can be best explained as how muscles contract by the interaction of actin and myosin filaments sliding past each other within muscle cells. The process requires ATP for energy. The sliding filament theory was proposed in 1954 by Andrew Huxley and Rolf Niedergerke. In this article, we will study the sliding filament theory of muscle contraction notes in detail. What is Sliding Filament Theory?The sliding filament theory states that the two main types of muscle filaments slide past each other during contraction, causing the muscle to shorten. The actin filaments are thin and have a double helix structure, while the myosin filaments are thick and have a globular head. The myosin heads bind to the actin filaments and pull them towards the center of the sarcomere. This causes the muscle fiber to shorten and the muscle to contract. The sliding filament theory is important because it helps us to understand how muscles work. Also Read: Mechanism of Muscle Contraction What is Sarcomere in Muscle?A sarcomere is the fundamental unit of muscle contraction and consists of bundle of thick and thin filaments. It has the following key features: Sarcomeres are present in series to form a myofibril and span from Z-line to Z-line. It is only a few micrometers long. Z-lines mark the boundaries of a sarcomere and anchor the thin filaments.It consists of overlapping actin and myosin filaments.It is present in a repeating pattern.Actin filaments are thin and extend from the Z-line towards the center. Myosin filaments on the other hand, are thicker and are located in the center.H- Zone is the central region of a sarcomere where only myosin filaments are present. It shortens during muscle contraction.I-band is the region containing only actin filaments, extending from the Z-line towards the center, and shortening during muscle contraction.A-band is the central region of the sarcomere where both actin and myosin filaments overlap.M-line is present at the center of the A-band that anchors the myosin filaments.Muscle contraction occurs as actin and myosin filaments slide past each other, causing the sarcomere to shorten.Sarcomeres contract when stimulated by a nerve impulse, leading to the shortening of the muscle fiber and the generation of force.Sarcomere DiagramAlso Read: Major Difference Between Actin and Myosin Sliding Filament Theory of Muscle Contraction Sliding Filament Theory The sliding filament theory describes the molecular changes that occur during muscle contraction at the sarcomere level, which is the basic functional unit of a muscle fiber. In the resting state, myosin heads are in a low-energy position, and the actin and myosin filaments do not overlap.When a motor neuron signals a muscle fiber to contract, an action potential is generated. It travels along the muscle cell membrane and into the muscle fiber through the transverse tubules.The action potential triggers the release of calcium ions from the sarcoplasmic reticulum.Calcium binds to troponin that results in change in shape of troponin. It allows tropomyosin to move away from the myosin-binding sites on actin.With the myosin-binding sites exposed, myosin heads can bind to actin, forming cross-bridges.The myosin heads undergo forceful contraction, pulling the thin actin filaments towards the center of the sarcomere.As myosin heads continue to cycle through binding, pulling, and releasing, the actin filaments slide past the myosin filaments, causing the sarcomere to shorten.When the action potential ceases and calcium ions are actively pumped back into the sarcoplasmic reticulum, the troponin-tropomyosin complex returns to its original position, blocking the myosin-binding sites on actin. This leads to muscle relaxation.Also Read: Muscular Tissue - Structure, Functions, Types and Characteristics Sliding Filament Theory DiagramThe following is a well-labeled diagram of sliding filament theory: The sliding filament theory of muscle contraction involves the steps: Resting State: Actin and myosin filaments overlap only slightly and muscle fibers are relaxed.Excitation of the nerve: A nerve impulse stimulates the muscle fiber. It causes the release of calcium ions from the sarcoplasmic reticulum into the sarcoplasm.Cross-Bridge Formation: Calcium ions bind to troponin, causing tropomyosin to move. It expose the myosin-binding sites on actin. Myosin heads then bind to these sites and forms the cross-bridges.Role of ATP: The ATP molecule is hydrolyzed and causes the myosin head to pivot. It pull actin filaments towards the center of the sarcomere.Repeat: The cycle continues as long as calcium ions are present and ATP is available, resulting in the shortening of sarcomeres and muscle contraction.How Does Muscle Contraction Occur?Muscle contraction is a physiological process where muscle fibers generate tension and exert a force, resulting in movement or the stabilization of body parts. Muscle contraction begins with a signal from the central nervous system through a motor neuron.The neuromuscular junction connects the motor neuron to the sarcolemma.Acetylcholine is released at the neuromuscular junction. It results in the action potential in the sarcolemma.An action potential triggers the release of calcium ions from the sarcoplasmic reticulum.Calcium ion binds to troponin on actin filaments. It exposes the myosin-binding sites.Myosin binds to the exposed active sites on actin and forms the cross bridges.The hydrolysis of ATP at the myosin head causes sliding of thin filaments over thick filaments.As thin filaments slide, the Z lines are pulled closer together. It leads to muscle contraction.The cycle of cross-bridge formation, contraction, and sliding repeats until calcium ions are actively pumped back into the sarcoplasmic reticulum.With decreasing calcium levels, troponin covers the myosin-binding sites, allowing for muscle relaxation.Recurrent muscle activation may lead to the accumulation of lactic acid, contributing to muscular fatigue.Myoglobin, a pigment in muscles, contributes to their red color. Muscles rich in myoglobin are adapted for sustained, aerobic activities.Red fibers with myoglobin-rich content also have a large number of mitochondria, supporting energy production during prolonged activities.Muscles lacking myoglobin appear white and are associated with anaerobic, short cycle of activity.As calcium is pumped back, the Z lines return to their initial positions, and the muscle returns to a relaxed state.Also Read: Difference Between Cardiac Muscle And Skeletal Muscle Importance of Sliding Filament TheoryThe sliding filament theory is the most widely accepted theory for explaining how muscle fibers contract. It describes how the interaction between actin and myosin filaments produces contractile force. Explains the molecular mechanism behind muscle contraction.Forms the basis for understanding different body movements.It help in diagnosing and treating muscle-related disorders.Forms the basis for studying muscle physiology and related disorders.Also Read: Difference between Origin and Insertion Muscles Conclusion: Sliding Filament TheoryThe sliding filament theory is the most accepted theory that explains how muscle fibers contract. It states that when a muscle contracts, the actin and myosin filaments slide past each other, causing the sarcomere to shorten. The filaments themselves do not change in length. The number of fibers that contract determines the strength of the muscular force. The theory help us to understand various muscle functions and different body movements. Also Read: scorevideos The sliding filament theory is a complex process, especially when it's explained in an intricate way. In this article, I will break down the basics of this theory to help you understand the process of how it happens and what some key words mean. The sliding filament theory was proposed by Andrew Huxley in 1954 and has helped scientists understand how muscle contractions work at cellular level with proteins sliding against each other causing cross-bridges which then leads to muscle contractions – thought this may sound complex, this is how movement appears which is unique to the traits of an individual such as the flexibility or ballerinas or the strength of a powerlifter. In this article, we will explain the sliding filament theory in a more simple nature, but first, to help our understanding on the sliding filament theory, it's useful to have a quick recap on muscle structures and the three types of muscle contractions… Muscle fibre structure – When muscle fibres (i.e. a muscle) are put under a microscope, we can see they contain smaller fibres called myofibrils. 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This happens across every sarcomere in the muscle! From our example of our bicep curl, this step would result in the dumbbell being lifted upwards. Jacob Krans from Central Connecticut State University provides a great analogy for the sliding filaments when sarcomere shortening (i.e. steps 1-3) occurs, which we'll include below: Jacob uses a bookcase for his analogy, he says, "imagine you are standing between two bookcases, that are a couple of meters apart and each filled with books. You must bring the two book cases together, by only using your arms and two ropes, which you have one end in each hand and the other end tied to each end of the bookcases. You repeatedly pull each rope towards you, re-grip it, and then pull again. Eventually, as you progress through the length of the rope, the bookcases move together and approach you. In this example, your arms are similar to the myosin molecules, the ropes are the actin filaments, and the bookcases are the z discs to which the actin is secured, which make up the lateral ends of a sarcomere. Similar to the way you would remain centered between the bookcases, the myosin filaments remain centered during normal muscle contraction." For the dumbbell to be lowered, myosin lets go of actin with the cross-bridge being broken. The stages are then reversed as tropomyosin returns to it's original place. As long as the human body has enough energy (and calcium) available, then this process can occur over and over – without it, we would not be able to function as humans. Now that we've explained muscle contraction from a concentric and eccentric portion of movement, we must think about the sliding filament theory during an isometric contraction. During an isometric contraction, cross bridges are still formed (stage 1-2), however, force is equally distributed between filaments. Though it must be noted that force production is reduced during isometric contraction in comparison to the potential force production of eccentric and concentric contraction. Though the sliding filament theory was proposed in the 1950s, it has been proven to be applicable to all muscle fibre types throughout the body. This process occurs over and over throughout the muscle during everyday life from performing bicep curls in the gym to simply standing up from a chair. Now that you understand the 5 step process of muscle contraction, we must begin to think about applying this knowledge to different movements such as a squat or pull-up. Though the process is the same for every single muscle fibre, think about how the different muscles work that are involved. Below I have referenced a few important sliding filament theory papers that will help give you an even better understanding as well as provide a reference point for your understanding. 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The Basics of Muscle Structure To comprehend the Sliding Filament Theory, it's essential to first understand the basic structure of muscles. Skeletal muscles, responsible for voluntary movements, are composed of bundles of muscle fibers. Each muscle fiber is a single, long cell containing numerous myofibrils, which are the contractile elements of the cell. Myofibrils are made up of repeating units called sarcomeres, the fundamental units of muscle contraction. Sarcomeres are delineated by Z-lines and contain two primary types of protein filaments: thick filaments (myosin) and thin filaments (actin). The regular arrangement of these filaments within the sarcomere gives muscles their striated appearance under a microscope. The Mechanism of Sliding Filament Theory The Sliding Filament Theory posits that muscle contraction occurs through the sliding of actin filaments over myosin filaments within the sarcomere. This sliding action shortens the sarcomere, leading to overall muscle contraction. Here's a step-by-step breakdown of this process: Activation and Calcium Release: The process begins with a nerve impulse triggering the release of calcium ions (Ca^{2+}) from the sarcoplasmic reticulum into the cytoplasm of the muscle cell. Binding of Calcium to Troponin: Calcium ions bind to the troponin complex on the actin filaments. This binding causes a conformational change in the troponin-tropomyosin complex, exposing the myosin-binding sites on the actin filaments. Cross-Bridge Formation: Myosin heads, which are equipped with ATP (adenosine triphosphate), bind to the now-exposed binding sites on actin, forming cross-bridges. Power Stroke: The myosin heads pivot, pulling the actin filaments towards the center of the sarcomere. This action is known as the power stroke and is powered by the hydrolysis of ATP to ADP (adenosine diphosphate) and inorganic phosphate. Detachment and Resetting: After the power stroke, a new ATP molecule binds to the myosin head, causing it to detach from the actin filament. The myosin head then resets to its original position, ready to bind to another actin site. Repetition and Sarcomere Shortening: This cycle repeats multiple times, causing the actin filaments to slide past the myosin filaments progressively. As this occurs across many sarcomeres, the muscle fiber contracts. Relaxation: When the nerve impulse ceases, calcium ions are pumped back into the sarcoplasmic reticulum, and the troponin-tropomyosin complex returns to its original state, blocking the myosin-binding sites on actin. This leads to muscle relaxation. The Importance of ATP ATP plays a crucial role in muscle contraction. It is not only necessary for the power stroke of myosin heads but also for their detachment from actin after the stroke. Without sufficient ATP, muscles would remain in a contracted state, leading to conditions such as rigor mortis after death. Applications and Implications Understanding the Sliding Filament Theory is essential for various fields, including medicine, sports science, and rehabilitation. For instance, in conditions such as muscular dystrophy or during muscle injuries, the normal sliding filament mechanism is disrupted, leading to impaired muscle function. In sports, knowledge of this theory helps in designing effective training and rehabilitation programs that enhance muscle performance and recovery. Furthermore, understanding muscle contraction at the molecular level aids in the development of treatments for muscle-related diseases and conditions. Frequently Asked Questions (FAQs) 1. What is the Sliding Filament Theory? The Sliding Filament Theory explains how muscles contract by the sliding action of actin and myosin filaments within the sarcomere, leading to the shortening of muscle fibers. 2. Who developed the Sliding Filament Theory? The theory was independently developed by Hugh Huxley and Andrew Huxley in the 1950s. 3. What role does calcium play in muscle contraction? Calcium ions bind to troponin on the actin filaments, causing a conformational change that exposes myosin-binding sites, allowing muscle contraction to occur. 4. Why is ATP important for muscle contraction? ATP provides the energy needed for the power stroke of myosin heads and their detachment from actin after the stroke. Without ATP, muscles would remain contracted. 5. How does muscle relaxation occur? Muscle relaxation occurs when calcium ions are pumped back into the sarcoplasmic reticulum, and the troponin-tropomyosin complex blocks the myosin-binding sites on actin. 6. What happens if there is insufficient ATP in the muscle? Insufficient ATP can lead to muscle stiffness and the inability to relax, as seen in rigor mortis. 7. How does the Sliding Filament Theory apply to sports and rehabilitation? The theory helps in understanding muscle function, which is essential for designing effective training and rehabilitation programs to enhance muscle performance and recovery. 8. Can muscle diseases affect the Sliding Filament Mechanism? Yes, diseases like muscular dystrophy can disrupt the normal sliding filament mechanism, leading to impaired muscle function and weakness. Understanding the Sliding Filament Theory provides valuable insights into how our muscles work, paving the way for advancements in medical treatments, sports science, and overall health and wellness. This comprehensive overview of the Sliding Filament Theory offers a detailed look at the molecular mechanisms driving muscle contraction and its broad applications. Fact Checked Content Last Updated: 08.12.2022 8 min reading time Content creation process designed by Content cross-checked by Content quality checked by Save Article Save Article Before diving into the sliding filament theory, let's review the skeletal muscle structure. Skeletal muscle cells are long and cylindrical. Due to their appearance, they are referred to as muscle fibres or myofibers. Skeletal muscle fibres are multinucleated cells, meaning that they consist of multiple nuclei (singular nucleus) because of the fusion of hundreds of precursor muscle cells (embryonic myoblasts) during early development. Moreover, these muscles can be pretty large in humans.Muscle fibres are highly differentiated. They have acquired particular adaptations, making them efficient for contraction. Muscle fibres consist of the plasma membrane in muscle fibres is called the sarcolemma, and the cytoplasm is called the sarcoplasm. As well as, myofibers which possess a specialised smooth endoplasmic reticulum called the sarcoplasmic reticulum (SR), adapted for storing, releasing, and reabsorbing calcium ions.Myofibers contain many contractile protein bundles called myofibrils, which extend along with the skeletal muscle fibre. These myofibrils are composed of thick myosin and thin actin myofilaments, which are the critical proteins for muscle contraction, and their arrangement gives the muscle fibre its striped appearance. It is important not to confuse myofibers with myofibrils.Fig. 1 - The ultrastructure of a microfibreAnother specialised structure seen in skeletal muscle fibre is T tubules (transverse tubules), protruding off the sarcoplasm into the centre of the myofibers (Figure 1). T tubules play a crucial role in coupling muscle excitation with contraction. We will elaborate further on their roles further on in this article.Skeletal muscle fibres contain many mitochondria to supply a large amount of ATP needed for muscle contraction. Furthermore, having multiple nuclei allows muscle fibres to produce large amounts of proteins and enzymes required for muscle contraction.Skeletal myofibers have a striated appearance due to the sequential arrangement of thick and thin myofilaments in myofibrils. Each group of these myofilaments is called sarcomere, and it is the contractile unit of a myofiber.The sarcomere is approximately 2 µm (micrometres) in length and has a 3D cylindrical arrangement. Z-lines (also called Z-discs) to which the thin actin and myofilaments are attached border each sarcomere. In addition to actin and myosin, there are two other proteins found in sarcomeres that play a critical role in regulating the function of actin filaments in muscle contraction. These proteins are tropomyosin and troponin. During muscle relaxation, tropomyosin binds along actin filaments blocking the actin-myosin interactions.Troponin is composed of three subunits:Troponin T: bind to tropomyosin.Troponin I: bind to actin filaments.Troponin C: binds to calcium ions. Since actin and its associated proteins form filaments thinner in size than the myosin, it is referred to as the thin filament.On the other hand, the myosin strands are thicker due to their larger size and multiple heads that protrude outwards. For this reason, myosin strands are called thick filaments.The organisation of thick and thin filaments in sarcomeres gives rise to bands, lines, and zones within sarcomeres. Fig. 2 - Arrangement of filaments in sarcomeresThe sarcomere is split into the A and I bands, H zones, M lines, and Z discs. A band: Darker coloured band where thick myosin filaments and thin actin filaments overlap.I band: Lighter coloured band with no thick filaments, only thin actin filaments.H zone: Area at the centre of A band with only myosin filaments.M line: Disc in the middle of the H zone that the myosin filaments are anchored to. Z-disc: Disc where the thin actin filaments are anchored to. The Z-disc Marks the border of adjacent sarcomeres.Energy in the form of ATP is needed for the movement of myosin heads and the active transportation of Ca ions into the sarcoplasmic reticulum. This energy is generated in three ways:Aerobic respiration of glucose and oxidative phosphorylation in the mitofhchondria.Anaerobic respiration of glucose.Regeneration of ATP using Phosphocreatine. (Phosphocreatine acts like a reserve of phosphate.)The sliding filament theory suggests that striated muscles contract through the overlapping of actin and myosin filaments, resulting in a shortening of the muscle fibre length. Cellular movement is controlled by actin (thin filaments) and myosin (thick filaments).In other words, for a skeletal muscle to contract, its sarcomeres must shorten in length. The thick and thin filaments do not change; instead, they slide past one another, causing the sarcomere to shorten.The sliding filament theory involves different steps. The step by step of the sliding filament theory is: Step 1: An action potential signal arrives at the axon terminal of the presynaptic neuron, simultaneously reaching many neuromuscular junctions. Then, the action potential causes voltage-gated calcium ion channels on the presynaptic knob to open, driving an influx of calcium ions (Ca2+).Step 2: The calcium ions cause the synaptic vesicles to fuse with the presynaptic membrane, releasing acetylcholine (ACh) into the synaptic cleft. Acetylcholine is a neurotransmitter that tells the muscle to contract. ACh diffuses across the synaptic cleft and binds to ACh receptors on the muscle fibre, resulting in depolarisation (more negative charge) of the sarcolemma (cell membrane of the muscle cell). Step 3: The action potential then spreads along the T tubules made by the sarcolemma. These T tubules connect to the sarcoplasmic reticulum. Calcium channels on the sarcoplasmic reticulum open in response to the action potential they receive, resulting in the influx of calcium ions (Ca2+) into the sarcoplasm. Step 4: Calcium ions bind to troponin C, causing a conformational change that leads to the movement of tropomyosin away from actin-binding sites. Step 5: High-energy ADP-myosin molecules can now interact with actin filaments and form cross-bridges. The energy is released in a power stroke, pulling actin towards the M line. Also, ADP and the phosphate ion dissociate from the myosin head. Step 6: As new ATP binds to the myosin head, the cross-bridge between myosin and actin is broken. Myosin head hydrolyses ATP to ADP and phosphate ion. The energy released returns the myosin head to its original position. Step 7: Myosin head hydrolyses ATP to ADP and phosphate ion. The energy released returns the myosin head to its original position. Steps 4 to 7 are repeated as long as calcium ions are present in the sarcoplasm (Figure 4). Step 8: Continued pulling of actin filaments towards the M line causes the sarcomeres to shorten. Step 9: As the nerve impulse stops, calcium ions pump back into the sarcoplasmic reticulum using the energy from ATP. Step 10: In response to the decrease in calcium ion concentration within the sarcoplasm, tropomyosin moves and blocks the actin-binding sites. This response prevents any further cross bridges from forming between actin and myosin filaments, resulting in muscle relaxation. Fig 4. Actin-myosin cross-bridge formation cycle.As the sarcomere shortens, some zones and bands contract while others stay the same. Here are some of the main observations during contraction (Figure 3):The distance between Z-discs is reduced, which confirms the shortening of sarcomeres during muscle contraction. The H zone (region at the centre of A bands containing only myosin filaments) shortens. The A band (the region where actin and myosin filaments overlap) remains the same. The I band (the region containing only actin filaments) shortens too. Fig. 3 - Changes in the length of sarcomere bands and zones during muscle contractionMyofibers contain many contractile protein bundles called myofibrils which extend along with the skeletal muscle fibre. These myofibrils are composed of thick myosin and thin actin myofilaments.These actin and myosin filaments are arranged in a sequential order in contractile units called sarcomeres. The sarcomere is split into the A band, I band, H zone, M line and Z disc: A band: Darker coloured band where thick myosin filaments and thin actin filaments overlap.I band: Lighter coloured band with no thick filaments, only thin actin filaments.H zone: Area at the centre of A bands with only myosin filaments.M line: Disc in the middle of the H zone that the myosin filaments are anchored to. Z disc: Disc where the thin actin filaments are anchored to. Z-disc marks the border of the adjacent sarcomeres.In muscle stimulation, action potential impulses are received by the muscles and cause a surge in intracellular calcium levels. During this process, the sarcomeres are shortened, causing the muscle to contract.The sources of energy for muscle contraction is supplied via three ways:Aerobic respirationAnaerobic respirationPhosphocreatine How do muscles contract according to sliding filament theory? According to the sliding filament theory, a myofiber contracts when myosin filaments pull actin filaments closer towards the M line and shorten sarcomeres within a fibre. When all the sarcomeres in a myofiber shorten, the myofiber contracts. Does the sliding filament theory apply to cardiac muscle? Yes, the sliding filament theory applies to striated muscles. What is the sliding filament theory of muscle contraction? The sliding filament theory explains the mechanism of muscle contraction based on actin and myosin filaments that slide past each other and cause sarcomere shortening. This translates to muscle contraction and muscle fibre shortening. What are the sliding filament theory steps? Step 1: Calcium ions are released from the sarcoplasmic reticulum into the sarcoplasm. Myosin head does not move. Step 2: Calcium ions cause tropomyosin to unblock actin-binding sites and permit cross bridges to form between actin filament and myosin head. Step 3: Myosin head utilises ATP to pull on actin filament toward the line. Step 4: Sliding of actin filaments past myosin strands results in shortening of sarcomeres. This translates to contraction of the muscle. Step 5: When calcium ions are removed from the sarcoplasm, tropomyosin moves back to block calcium-binding sites. Step 6: Cross bridges between actin and myosin are broken. Hence, the thin and thick filaments slide away from each other and the sarcomere returns to its original length. How does sliding filament theory work together? According to the sliding filament theory, myosin binds to actin. The myosin then alters its configuration using ATP, resulting in a power stroke that pulls on the actin filament and causes it to slide across the myosin filament towards the M line. This causes the sarcomeres to shorten. Save Article At StudySmarter, we have created a learning platform that serves millions of students. Meet the people who work hard to deliver fact based content as well as making sure it is verified. Lily Hulatt is a Digital Content Specialist with over three years of experience in content strategy and curriculum design. She gained her PhD in English Literature from Durham University in 2022, taught in Durham University's English Studies Department, and has contributed to a number of publications. Lily specialises in English Literature, English Language, History, and Philosophy. Get to know Lily Gabriel Freitas is an AI Engineer with a solid experience in software development, machine learning algorithms, and generative AI, including large language models' (LLMs) applications. Graduated in Electrical Engineering at the University of São Paulo, he is currently pursuing an MSc in Computer Engineering at the University of Campinas, specializing in machine learning topics. Gabriel has a strong background in software engineering and has worked on projects involving computer vision, embedded AI, and LLM applications. Get to know Gabriel StudySmarter is a globally recognized educational technology company, offering a holistic learning platform designed for students of all ages and educational levels. 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By understanding this theory, we gain insight into how muscles function at a molecular level to produce movement and force.Skeletal muscle fibers, also known as myofibers, are long, cylindrical cells that are multinucleated due to the fusion of precursor muscle cells during development. These fibers are specialized for contraction and contain several key structures:Sarcoplasmic Reticulum (SR): A specialized type of smooth endoplasmic reticulum that stores and releases calcium ions (Ca^{2+}), which are crucial for muscle contraction.Myofibrils: These are the contractile elements of the muscle fiber, consisting of thick myosin filaments and thin actin filaments arranged in a repeating pattern along the length of the muscle fiber.T-Tubules (Transverse Tubules): Extensions of the sarcolemma (muscle cell membrane) that penetrate into the muscle fiber, allowing the action potential to reach the SR.Mitochondria: Numerous in muscle fibers to provide the ATP needed for contraction.A sarcomere is characterized by its highly organized structure, which can be observed under a light microscope as a repeating unit of alternating dark and light bands. The key structural elements of the sarcomere include:SarcomereZ Discs (Z Lines): The Z discs define the boundaries of each sarcomere and anchor the thin filaments of actin. They are located at both ends of the sarcomere and play a crucial role in maintaining the structural integrity of the sarcomere. The Z disc also serves as an attachment site for titin, a large protein that contributes to sarcomere elasticity.A Band: The A band is the region of the sarcomere that contains the entire length of the thick filaments (myosin). It appears darker under the microscope due to the overlap of myosin with actin filaments. The A band does not change in length during muscle contraction, as it represents the length of the myosin filaments, which do not shorten but rather slide past the actin filaments.I Band: The I band is the lighter region of the sarcomere that contains only thin filaments (actin) and extends from the end of one A band to the beginning of the next A band. The I band shortens during muscle contraction as the actin filaments slide inward, overlapping more with the myosin filaments.H Zone: The H zone is a central region within the A band where only thick filaments are present, with no overlap from thin filaments. It appears lighter than the rest of the A band. During contraction, the H zone narrows as the actin filaments slide into the region, reducing the distance between adjacent myosin filaments.M Line: The M line is a structural protein assembly located in the center of the sarcomere, within the A band. It helps anchor the thick filaments and maintains their alignment during contraction. The M line consists of myomesin and other proteins that stabilize the myosin filaments.Myosin: Structure, Synthesis, Classification, and Functions – The Science NotesActin: Structure, Function, and Dynamics – The Science NotesThe sliding filament theory, initially proposed by A. F. Huxley and R. Niedergerke and later expanded by H. E. Huxley and J. Hanson, explains how muscle contraction occurs. According to the theory:Sarcomere Structure During Contraction: As a muscle contracts, the A band, which contains myosin filaments, remains the same length, while the I band, which contains actin filaments, shortens. This shortening is due to the sliding of actin filaments past myosin filaments.Actin-Myosin Interaction: Myosin heads bind to actin filaments, forming cross-bridges. These cross-bridges undergo a power stroke powered by ATP hydrolysis, pulling the actin filaments towards the center of the sarcomere and causing contraction.Sliding Filament Theory of muscle contractionAccording to the sliding filament theory, muscle contraction occurs through the sliding of actin and myosin filaments past one another, resulting in the shortening of the sarcomere without changing the length of the filaments themselves. The process involves several key steps:An action potential from a motor neuron arrives at the neuromuscular junction, causing the release of acetylcholine (ACh).ACh binds to receptors on the sarcolemma, leading to depolarization and the initiation of an action potential that travels along the T-tubules.The action potential stimulates the SR to release Ca^{2+} into the sarcoplasm.Calcium ions bind to troponin C, a component of the troponin complex associated with the thin filaments.This binding causes a conformational change in the troponin-tropomyosin complex, moving tropomyosin away from the actin-binding sites.With the binding sites exposed, the high-energy myosin heads attach to actin, forming cross-bridges.ATP bound to myosin is hydrolyzed to ADP and inorganic phosphate, providing energy for the power stroke.During the power stroke, the myosin head pivots, pulling the actin filaments toward the M line and shortening the sarcomere.The release of ADP and phosphate from the myosin head is followed by the binding of a new ATP molecule, which causes the detachment of the myosin head from actin.ATP is hydrolyzed to ADP and phosphate, which re-energizes the myosin head, preparing it for the next cycle of binding and pulling.The cycle repeats as long as calcium ions remain present in the sarcoplasm.When the action potential ceases, calcium ions are actively transported back into the SR.The decrease in calcium concentration causes tropomyosin to cover the actin-binding sites again, preventing further cross-bridge formation and resulting in muscle relaxation.Muscle contraction is tightly regulated by several factors, including calcium ions and ATP:Calcium's Role: Calcium ions play a pivotal role in muscle contraction by binding to troponin, a regulatory protein associated with actin filaments. In the absence of calcium, tropomyosin covers the myosin-binding sites on actin, preventing cross-bridge formation. When calcium levels increase, troponin undergoes a conformational change that moves tropomyosin away from the binding sites, allowing myosin heads to attach to actin.ATP's Role: ATP is essential for muscle contraction as it provides the energy needed for the power stroke and the detachment of myosin from actin. ATP hydrolysis also allows the myosin head to return to its cocked position. Without sufficient ATP, muscles cannot relax and remain in a state of contraction, leading to conditions such as rigor mortis.Muscle contractionMolecular Mechanisms: The detailed molecular mechanisms of the sliding filament theory have been elucidated through various studies. For example, high-resolution microscopy and X-ray diffraction studies have provided insights into the structural changes that occur during muscle contraction. These studies have confirmed that the sliding of actin filaments past myosin is responsible for sarcomere shortening.Protein Structures: The structural details of the myosin and actin filaments have been further explored through techniques such as cryo-electron tomography and atomic force microscopy. These studies have revealed the precise arrangements of actin and myosin, as well as the conformational changes that occur during the cross-bridge cycle.Several observations support the sliding filament theory:Sarcomere Shortening: During muscle contraction, the distance between Z-discs decreases, the H zone narrows, and the I band shortens, while the A band remains unchanged.Microscopic Studies: Electron microscopy reveals that the actin and myosin filaments do not change length but slide past each other.The sliding filament theory has profoundly shaped our understanding of muscle contraction, revealing the intricate molecular interactions that enable muscle fibers to shorten and generate force. By elucidating the roles of actin, myosin, ATP, and calcium, scientists have gained valuable insights into muscle function and regulation. Despite its comprehensive nature, ongoing research continues to refine our understanding and address unresolved questions. The continued exploration of muscle physiology promises to uncover new insights and applications, advancing our knowledge of this fundamental physiological process.Huxley, A. F., & Niedergerke, R. (1954). Structural changes in muscle during contraction: Interference microscopy of living muscle fibres. Nature, 173, 971-973.Huxley, H. E., & Hanson, J. (1954). Changes in the cross-striations of muscle during contraction and stretch and their structural interpretation. Nature, 173, 973-976.Spudich, J. A. (2001). The myosin swinging cross-bridge model. Nature Reviews Molecular Cell Biology, 2, 387-392.Lehman, W., Craig, R., & Vibert, P. (1994). Ca2+-induced tropomyosin movement in Limulus thin filaments revealed by three-dimensional reconstruction. Nature, 368, 65-67.Goody, R. S. (2003). The missing link in the muscle cross-bridge cycle. Nature Structural Biology, 10, 773-775.