**Physiology of smooth muscle contraction pdf**

Involuntary unstorted muscles Smooth muscle tissueSSes of the esophagus wall: MucosaSubmucosamuscularisAdventitiaStriated muscleStriated and smoothSmooth muscleLamina muscularusesophageal glandsDetailsIdentifiersLatintextus muscularis levis; textus muscularis nonstriatusMeSHD009130THH2.00.05.1.00001FMA14070Anatomic terminology (edited on Wikidata) Smooth muscle tissue, emphasizing the inner circular layer (the nucleus, then the rest of the cells in pink), the outer longitudinal layer (the nucleus, then the rest of the cells), then the serous membrane in front of the lumen of the abdominal cavity Smooth muscle is an involuntary involuntary intriduous muscle. It is divided into two subgroups; single (unitary) and multi-smart smooth muscle. In single-number cells, the entire beam or sheet contracts as a syncytim. Smooth muscle cells are found in the walls of hollow organs, including the stomach, intestines, bladder and uterus, as well as in the walls of passages such as arteries and veins of the circulatory system, as well as in the respiratory, urinary and reproductive systems. In the eyes of the ciliary muscle, the type of smooth muscle expand and contract the iris and change the shape of the lens. In the skin, smooth muscle cells cause the hair to stand directly in response to cold temperature or fear. The structure of dense bodies and intermediate threads network through sarcoplasm, which cause muscle fiber to contract. A series of axon-like swellings called varicose veins or buds from vegetative neurons form motor units through a smooth muscle. Most smooth muscles are a single block variety, that is, either all muscle contracts or the entire muscle relaxes, but there are multifunctional smooth muscles in the trachea, large elastic arteries, and iris. Single block of smooth muscle, however, is the most common and lines of blood vessels (except for large elastic arteries), urinary tract and digestive tract. However, the terms of single- and multifunctional smooth muscles is a simplification. This is due to the fact that the smooth muscles are mostly controlled and are influenced by a combination of different nerve elements. It has also been noted that most of the time there will be some cells to cellular communication and activators/inhibitors are produced locally. This leads to a somewhat coordinated reaction even in multifunctional smooth muscles. Smooth muscle is fundamentally different from skeletal muscles and heart muscle in terms of structure, function, regulation of contraction and arousal-reducing communication. Smooth muscle cells, known as myocytes, have a fusiform shape and, like striped muscles, can strain and relax. However, smooth muscle tissue tends to show greater elasticity and function within a longer voltage curve than striped muscles. This ability to stretch and maintain contractuality organs such as the intestines and bladder. In a relaxed state, each cell has a spindle shape, 20-500 micrometers long. The molecular structure of a substantial part of the volume of the cytoplasm of smooth muscle cells is incorporated by molecules of myosin and actin, which together have the ability to contract, and, through a chain of tense structures, make the entire smooth muscle tissue contract with them. Miosin Miosin is primarily grade II in smooth muscle. Myosin II contains two heavy chains (MHC) that make up the head and tail area. Each of these heavy chains contains the N-terminal head domain, while C-terminal tails take on a spiral coil of morphology, holding two heavy chains together (imagine two snakes wrapped around each other, for example, in caduceus). Thus, myosin II has two heads. In smooth muscles there is one gene (MYH11) that encodes the heavy chains of myosine II, but there are variants of splicing of this gene that lead to four separate isoforms. In addition, a smooth muscle may contain MHC, which is not involved in contraction, and which may arise from multiple genes. Myosin II also contains 4 light circuits (MLC), bringing 2 per head to the national, weighing 20 (MLC20) and 17 (MLC17) kDa. They bind heavy chains in the neck area between the head and tail. MLC20 is also known as the regulatory light chain and is actively involved in muscle contraction. Two isoform MLC20s are in smooth muscles and are encoded by different genes, but only one isoform is involved in contraction. MLC17 is also known as the main light chain. Its exact function is unclear, but it is believed to contribute to the structural stability of the head of myosine along with the MLC20. Two variants of MLC17 (MLC17a/b) exist as a result of alternative splicing in the MLC17 gene. Different combinations of heavy and light chains allow for up to hundreds of different types of myosine structures, but it is unlikely that more than a few of these combinations are actually used or allowed within a particular smooth muscle bed. In the uterus, a shift in expression of myosin has been suggested to take advantage of changes in the direction of uterine contraction that are visible during the menstrual cycle. Actin Thin Threads, which are part of the contracting technique, are mostly made up of α- and γ-actin. Smooth muscle with α (alpha-actin) is the predominant isoform in a smooth muscle. There is also a lot of actin (mostly β-actin) that is not involved in the contraction, but that polymerizes just below the plasma membrane in the presence of a contracting stimulant and thus can help in mechanical stress. Alpha actin is also expressed as various genetic isoforms such as smooth muscles, heart muscle and skeletal muscles specific alpha-actin isoforms. Actin-to-myoine ratio 2:1 and 10:1 in a smooth muscle. Conversely, in terms of mass ratio (as opposed to molar ratio), myosine is the dominant protein in striped skeletal muscles with an actin-to-myosin ratio falling out in the 1:2 to 1:3 range. The typical value for healthy young people is 1:2.2. Other proteins of the Smooth muscle contracting apparatus do not contain protein troponin; Instead of calmodulin (which takes on a regulatory role in smooth muscles), caldesmone and calponin are significant proteins expressed in a smooth muscle. Tropomyosin is present in a smooth muscle covering seven actin monomers and laid out from end to end along the entire length of thin strands. In striped muscles, tropomyosine serves to block actin-myosine interaction until calcium is present, but in a smooth muscle, its function is unknown. Calponin molecules may exist in equal amounts as actin, and have been suggested to be a protein carrier. Caldesmon was invited to participate in tying actin, myosine and tropomyosine, and thereby enhancing the ability of smooth muscles to maintain tension. In addition, all three of these proteins may play a role in inhibiting the activity of the ATPase complex of myosine, which otherwise provides energy for fuel muscle contraction. Other tense structures of myosin and actin are contract parts of continuous chains of tense structures that extend both across and between smooth muscle cells. The act of threads of the contract unit are attached to dense bodies. Dense bodies are rich α-actinin, as well as attach intermediate threads (consisting mainly of vimentine and desmin) and thus serve as anchors from which thin threads can exert force. Dense bodies are also associated with β-actin, which is a type found in the cytoskeleton, suggesting that dense bodies can coordinate tensions from both the contracting machine and the cytoskeleton. Dense bodies appear darker under an electron microscope, and so they are sometimes described as electron dense. Intermediate strands are connected to other intermediate threads through dense bodies, which eventually attach to joining compounds (also called focal adhesions) in the cell membrane of a smooth muscle cell called sarcolem. Joining compounds consist of a large number of proteins, including α actinine, vinculin and cytoskeletonal actin. The joints stick scattered around the dense strips that trim the smooth muscle cell into the rib-like pattern. Areas of dense strip (or dense plaques) alternate with areas of the membrane containing numerous caves. When the complexes of actin and myosin are captured, the force is transulated into the sarcolem through intermediate threads attached to such dense stripes. During the contraction, there is a reorganizing contract equipment to optimize optimization Development. Part of this reorganization consists of phosphorylated phosphoryled phosphoryl in Ser56 using activated p21 kinase, which leads to some disassembly of vimentin polymers. In addition, the number of myosine filaments is dynamic between a relaxed and contract state in some tissues as the ratio of actin to myosin changes, as well as changes in the length and number of myosine filaments. Isolated single smooth muscle cells were observed in a spiral corkscrew fashion, and isolated permeable smooth muscle cells adhered to glass (so contracting proteins allowed internally contracted) demonstrate areas of contracting protein interaction along the long axis, like cell contracts. Smooth muscle-containing tissue should be stretched frequently, so elasticity is an important attribute of smooth muscle. Smooth muscle cells can secrete a complex extracellular matrix containing collagen (mostly types I and III), elastin, glycoproteins and proteoglycans. The smooth muscle also has specific elastin and collagen receptors to interact with these proteins in the extracellular matrix. These fibers with their extracellular matrix contribute to the viscosity of these tissues. For example, large arteries are viscolelast vessels that act as Windkessel, spread ventricular compression and smoothing pulsating flow, and the smooth muscle in the tunic media contributes to this property. Caveolae Sarcolemma also contains caveolae, which are microdomes of lipid rafts specializing in cellular event signaling and ion channels. These invaginations in the sarcoplasm contain many receptors (proscacycline, endoteline, serotonin, muscarial receptors, adrenergic receptors), generators of the second messenger (adenylate cyclase, phospholipase C), G proteins (RhoA, G alpha), kinase (rodinas-ROCK, protein kinase C, protein Kinase A), ion channels (L-channels of calcium, ATP sensitive potassium channels, calcium) Caves are often located close to sarcoplamic rithicum or mitochondrial, and have been suggested to organize signaling molecules in the membrane. Excitement-reducing compound smooth muscle is excited by external stimuli, which leads to a contraction. Here's a look at each step. The inducing stimuli and factors of the Smooth Muscle can contract spontaneously (through the dynamics of the ion channel) or how in the intestines special pacemakers of Cajal cell cells produce rhythmic contractions. In addition, contraction, as well as relaxation, can be caused by a number of physiochemical agents (e.g. hormones, drugs, neurotransmitters - especially from the autonomic nervous system). Smooth muscle in various areas of the vascular, respiratory tract and lungs, kidneys and vagina differs in the expression of ion channels, hormonal receptors, cellular signaling pathways other proteins that are Functions. External substances Such as blood vessels in the skin, gastrointestinal system, kidneys and brain react to norepinephrine and epinephrine (from sympathetic stimulation or adrenal medulla) by producing vasodilators (this response is mediated through alpha-1 adrenargic receptors). However, blood vessels in the skeletal muscles and heart muscle react to these catecholamines that produce vasodilation because they have beta-adrenergic receptors. Thus, there is a difference in the distribution of different adrenergic receptors, which explains the difference in why blood vessels from different regions respond to the same norepinephrine/epinephrine agent differently, as well as differences due to the different amounts of these catecholamines that are released and the sensitivity of different receptors to concentrations. Typically, the arterial smooth muscle reacts to carbon dioxide by producing vasodilation, and reacts to oxygen by vasoconstricting. Pulmonary blood vessels in the lungs are unique because they are vasodilated to high oxygen voltage and are vascularized in the fall. Bronchiol, a smooth muscle that sticks out the airways of the lungs, responds to high-carbon carbon dioxide and vascularity when carbon dioxide is low. These reactions to carbon dioxide and oxygen pulmonary blood vessels and airway bronchioles smooth muscle relief in matching perfusion and ventilation in the lungs. Further different smooth muscle tissues show extremes abundant to a little sarcoplasmic reticulum so the arousal-reduction connection changes with its dependence on intracellular or extracellular calcium. (quote necessary) Recent studies show that sphingosine-1-phosphate (S1P) signaling is an important regulator of vascular smooth muscle contraction. When transmural pressure increases, sphingosine kinase 1 phosphorylate sphingosine S1P, which binds to the S1P2 receptor in the plasma membrane of cells. This leads to a transient increase in intracellular calcium, and activates Rac and Rhoa signaling pathways. Taken together, they contribute to increased MLCK activity and reduced MLCP activity, contributing to muscle contraction. This allows arterioles to increase resistance in response to high blood pressure and thus maintain constant blood flow. The Rhoa and Rac part of the signaling pathway provides a calcium-independent way of regulating the tone of the artery resistance. Spreading momentum To maintain the size of the organs against the force, the cells are attached to each other by joining the joints. As a result, the cells are mechanically connected to each other in such a way that the contraction of a single cell causes some degree of contraction in the adjacent cell. Breaking the compound of a pair of adjacent cells chemically and electrically, facilitating the spread of substances (such as calcium) or the potential for action between smooth muscle cells. The single-seater block of smooth muscle displays numerous The compounds and these fabrics are often arranged in sheets or bundles that are contracted in bulk. The compression of smooth muscle contractions is caused by the sliding of myosin and actin filament (sliding filament mechanism) over each other. The energy for this is provided by ATP hydrolysis. Myozin functions as ANPase, using ATP to produce molecular conformation of the change of part of myosin and produces movement. The movement of the strands over each other occurs when the ball heads protruding from the myosine filaments are attached and interact with the actin thread to form cross bridges. The heads of myosins bend and are dragged along the actin thread for a short distance (10-12 nm). The heads then release the actin thread and then change the angle to move to another location on the actin thread for an additional distance (10-12 nm). They can then re-bind to the actin molecule and drag it further. This process is called crossbridge cycling and the same for all muscles (see muscle contraction). Unlike heart and skeletal muscles, a smooth muscle does not contain calcium binding troponin protein. The compression is triggered by calcium-regulated myosine phosphorylation, not by a troponin-activated calcium system. Crossbridge cycling causes a reduction in myosine and actin complexes, in turn causing increased tension throughout the chain of tense structures, eventually leading to a reduction in all smooth muscle tissue. Phasic or tonic This section does not cite any sources. Please help improve this section by adding links to reliable sources. Non-sources of materials can be challenged and removed. (September 2020) (Learn how and when to remove this pattern message) Smooth muscle can contract phase with rapid contraction and relaxation, or tonally with slow and steady contraction. Reproductive, digestive, respiratory and urinary tract, skin, eyes and blood vessels all contain this tonic type of muscle. This type of smooth muscle can maintain strength for a long time with only a little energy use. There are differences in muscle heavy and lung chains that also correlate with these differences in the contracting model and kinetic contractions between tonic and phase smooth muscle. The activation of the myosin head of Crossbridge cycling cannot occur until the myosine heads have been activated to make the bridges in shape. When light chains are phosphorylated, they become active and allow them to shrink. The enzyme, which phosphorylates light circuits, is called myosine light-chain kinase (MLCK), also called MLC20 kinase. In order to control the contraction, MLCK will only work when the muscles are stimulated to contract. Stimulation will increase intracellular concentration of calcium ions. They bind to a molecule called calmodulin, and form Complex. It is this complex complex communicate with MLCK to activate it, allowing the chain of reactions to reduce going. Activation consists of serin phosphorylation at position 19 (Ser19) on the MLC20 light chain, that causes a conformational change that increases the angle in the neck area of the heavy chain of myosine, which corresponds to the part of the intersmostal cycle where the head of myosine is not tied to the actine filament and moves to another area on it. to provide energy for fuel subsequent reductions. The phosphorylation of the trionin at position 18 (Thr18) on the MLC20 is also possible and may further increase the activity of the ATPase complex of myosin. Sustained maintenance of MLC20 light circuit phosphorylation correlates well with the reduction in smooth muscle speed. During this period, there was a rapid surge in energy use, measured by oxygen consumption. Within minutes of starting, calcium levels decrease markedly, MLC20 myosine light chains of phosphorylation decreases, and energy use decreases and muscles can relax. However, a smooth muscle has the ability to sustainally maintain strength in this situation as well. This sustained phase has been attributed to some myosine crossbridges, called bridge snaps, which crawl very slowly on the bike, in particular slowing down the progress towards the cycle stage in which the dephosforyled myosin separates from the actin, thus maintaining strength at low energy costs. This phenomenon is of great importance, especially for tonally active smooth muscles. Isolated vascular and visceral smooth muscle contract drugs with the depolarization of high potassium balanced saline solution generating a certain amount of contracting force. The same drug is stimulated in a normal balanced saline solution with an agonist such as endoteline or serotonin will generate more contracting force. This increase in strength is called calcium sensitization. Fosine light chain phosphate is inhibited to increase the amplification or sensitivity of myosine light chain kinase to calcium. There are a number of cell signaling pathways believed to regulate this decrease in myosine light circuit phosphate: Roa-Rock Kinase Pathway, protein kinase C Protein-kinase C potency protein inhibitor 17 (CPI-17) pathway, telcokin, and cip kinase pathway. Further Rock kinase and zip kinase were directly involved phosphorylate 20kd myosine light circuits. Other contracting mechanisms Other cellular signaling pathways and protein kinases (Protein kinase C, Rho kinase, cip kinase, focal adhesion kinase) were involved, and and actin polymerization dynamics played a role in maintaining strength. While the phosphorylation of the myosintin light chain is good with reduced speed, other other signaling paths were involved in the development of strength and the maintenance of strength. It is noteworthy that the phosphorylation of specific tyrosine residues on the focal adhesion adapter of the protein-paxillin specific tyrosine kinas has been demonstrated as necessary for power development and maintenance. For example, cyclical nucleotides can relax arterial smooth muscles without reducing cross-phosphorylation, a process called force suppression. This process is mediated by the phosphorylation of a small heat shock protein, hsp20, and can prevent phosphorylated myosin heads from interacting with the actin. Relaxing phosphorylation of THEC light circuits is altered by the myosine light of the phosphate chain, which defosforylates MLC20 myosine light circuits and thus suppresses the contraction. Other signaling pathways have also been involved in the regulation of actin and myozin dynamics. In general, the relaxation of smooth muscles by cellular signaling of pathways that increase the activity of phosphatase myosine, Reducing intracellular calcium levels, hyperpolarizing smooth muscles, and/or regulating actin and muscle myosin can be mediated by endotely-derived relaxing factor-nitric oxide, endothelial derivative hyperpolarizing factor (either endogenous cannabinoid, cytochrome P450 metabolite, or hydrogen peroxide), or citation (PGI2). Nitric oxide and PGI2 stimulate soluble guanilat cyclase and membranes linked adenylate cyclase, respectively. The cyclic nucleotides (cGMP and cAMP) produced by these cyclases activate G kinase protein and protein kinase A and phosphorylate a range of proteins. The events of phosphorylation lead to a decrease in intracellular calcium (inhibit L-channels of calcium, inhibit IP3 receptor channels, stimulate sarcolasmic rithicum of calcium pump ATPase), decrease 20kd myosine light chain phosphorylation by altering calcium sensitization and increasing myosine light chain phosphate activity, stimulation of calcium-sensitive potassium channels that hyperpolary cell, and phosphorylation of amino acid residues of serin 16 on a small heat shock protein (hsp20) by Kinases A and G protein. Phosphorylation of hsp20 appears to alter actin and coordination of adhesive dynamics and actin-myosine interactions, and recent evidence suggests that hsp20 binding to a 14-3-3 protein is involved in the process. An alternative hypothesis is that phosphorylated Hsp20 can also alter the affinity of phosphorylated myosine with actin and inhibit contracting by interfering with cross bridge formation. Endothelium, derived from hyperpolarizing factor, stimulates calcium-sensitive potassium channels and/or ATP-sensitive potassium channels and stimulates potassium effluxe, which hyperpolarizes the cell and produces relaxation. Smooth invertebrates In the invertebrates smooth muscles, the contraction is initiated with calcium directly to myosin and then quickly cycling cross bridges, generating strength. Like the mechanism of the vertebrate smooth muscle, there is a low level of calcium and a low energy use phase. This steady phase or catch phase has been attributed to a protein catch that bears similarities to myosine light chain kinase and an elastic thitin protein called twitchin. Molluscs and other bivalve molluscs use this phase of catching smooth muscles to keep their shell closed for long periods of time with little energy intake. Specific effects Although the structure and function are basically the same in smooth muscle cells in different organs, their specific effects or end functions are different. The contract function of the vascular smooth muscle regulates the lumenal diameter of small arteries-arteriolys, called resistance vessels, thus making a significant contribution to the establishment of blood pressure and blood flow to vascular beds. Smooth muscle contracts slowly and can support contraction (tonicly) for long periods in blood vessels, bronchioles, and some sphincters. Activating the arteriole smooth muscle can reduce the lumenal diameter of 1/3 of the rest, so it dramatically alters blood flow and resistance. The activation of the aortic smooth muscle does not alter the diameter of the lumen, but serves to increase the viscosity of the vascular wall. In the digestive tract, smooth muscle contracts in a rhythmic peristaltic way, rhythmically forcing food through the digestive tract as a result of phase contraction. The non-content function is seen in specialized smooth muscles in the afferent arteriole juxtaglomecular apparatus, which secretes renin in response to osmotic and pressure changes, and is also thought to secrete ATP in tubulglomerular regulation of the glomerular filtration rate. Renin, in turn, activates the renin-angiotensin system to regulate blood pressure. The growth and restructuring of the Mechanism, in which external factors stimulate growth and restructuring, is not yet fully understood. A number of growth factors and neurohumoral agents affect smooth muscle growth and differentiation. It has been proven that the Notch receptor and cellular signaling pathway are essential for vasculogenesis and the formation of arteries and veins. The spread is involved in the pathogenesis of atherosclerosis and is inhibited by nitric oxide. The embryonic origin of smooth muscle is usually mesodermal origin, after the creation of muscle fibers in a process known as myogenesis. However, the smooth muscle in the aorta and pulmonary arteries (Great Arteries of the Heart) is derived from the ectomesenchyme of the nerve-shaped comb, although the coronary artery has a smooth muscle of mesodermal origin. The associated disease Smooth muscle condition is a condition in which the body of the developing embryo does not smooth muscle for the gastrointestinal tract. It's a deadly condition. Anti-smooth muscle antibodies (ASMA) can be a symptom of an autoimmune disorder such as hepatitis, liver cirrhosis or lupus. Smooth muscle tumors are most often benign and are then called leiomyomas. They can occur in any organ, but the most common forms occur in the uterus, colon and oesophagus. Malignant smooth muscle tumors are called leiomyosarcoma. Leyomyosarcoma are one of the most common types of soft tissue sarcoma. Vascular smooth muscle tumors are very rare. They can be malignant or benign, and the incidence can be significant with any type. Intrasescular leiomyomatosis is a benign neoplasm that extends through the veins; angiolooma is a benign neoplasm of the limbs; vascular leuomyosarcoma is a malignant neoplasm that can be found in the lower veins of cava, pulmonary arteries and veins, and other peripheral vessels. Look at atherosclerosis. See also Atromentin has been shown to be a smooth muscle stimulant. Skeletal Muscles of the Heart Muscle Help : 10.8 Smooth Muscle - Anatomy and Physiology. opentextbc.ca archive from the original dated February 1, 2018. Received on April 28, 2018. Byrne and Levi. Physiology, 6th edition b. 174 in: Vascular smooth muscle cell: molecular and biological reactions to the extracellular matrix. Authors: Stephen M. Schwartz, Robert. Sword. Editors: Stephen M. Schwartz, Robert. Sword. 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