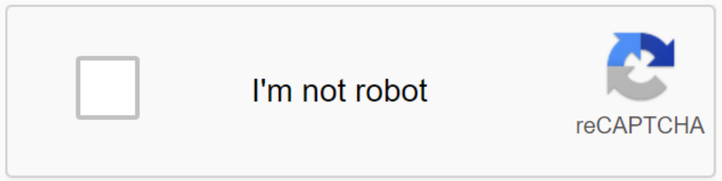

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Terminal cisternae of the sr

There are several mechanisms directly related to the terminal tank that facilitate the merging of excitation and contraction, which include ryanodine (RYR) receptors and a voltage-sealed calcium channel that helps transfer action potential to the calcium efflux of the sarcopsmical reticule. Wikipedia Cartoon part of the skeletal muscle, which shows the t-tubules leading deep into the center of the cell between two terminal tanks/connecting SR. Thinner projections, working horizontally between two terminal tanks are longitudinal parts of the SR. Sarcoplasmic reticulum (SR) is a membrane-bound structure found within muscle cells that is similar to endoplasmic reticulum in other cells. The main function of SR is the storage of calcium ions (Ca2+). Calcium ion levels are kept relatively constant, and calcium ion concentrations inside the cell are 10,000 times lower than calcium ions concentration outside the cell. [1] This means that small increases in calcium ions inside the cell are easily detected and can lead to important cellular changes (calcium is said to be another messenger; see calcium in biology for more details). Calcium is used to make calcium carbonate (found in chalk) and calcium phosphate, two compounds that the body uses to make teeth and bones. This means that too much calcium inside cells can lead to hardening (calcification) of certain intracellmental structures, including mitochondria,[2] leading to cell death. Therefore, it is vital that calcium ion levels are tightly controlled and can be dropped into the cell if necessary, and then removed from the cell. The structure of sarcoplasm reticulum is a network of tubules that stretch through muscle cells, wrapping (but not in direct contact with) myofibrili (contractile unit cells). Cardiac and skeletal muscle cells contain structures called transverse tubules (T-tubules), which are extensions of cell membranes that travel to the center of the cell. T-tubules are closely related to a particular region of SR, known as the terminal tanker in skeletal muscles, with a distance of approximately 12 nanometers, separating them. This is the primary place of calcium discharge. [3] Longitudinal SR are thinner projects, which are run between the terminal tank/connecting SR, and are where the ion channels needed to absorb calcium ions are most unstable. [4] These processes are explained in more detail below and are fundamental to the process of excitation-contraction of the coupling in skeletal, cardiac and smooth muscles. Calcium absorption of SR contains ion channel pumps, inside its membranes that are responsible for pumping Ca2+ into SR. Since the calcium iion concentration within SR is higher than in the rest of the cell, calcium ions will not flow freely into SR, so the pumps energy, obtained from a molecule called adenosine triphosphate (ATP). These calcium pumps are called Sarco(endo)plasma reticulum ATPases (SERCA). There are different forms of SERCA, with SERCA 2a located primarily in heart and skeletal muscles. [5] SERCA consists of 13 subunits (marked M1-M10, N, P and A). Calcium esoni bind to subunits M1-M10 (located within the membrane), while ATP binds to N, P and A subunits (located outside the SR). When 2 calcium ions, together with the ATP molecule, bind to the cytosolic side of the pump (that is, the pump area outside the SR), the pump opens. This occurs because ATP (containing three groups of phosphates) releases one group of phosphates (becoming adenosine diphosphate). The released group of phosphates then binds to the pump, causing the pump to change shape. This change in shape causes the cytosolic side of the pump to open, allowing two Ca2+s to enter. The cytosolic side of the pump then closes and the sarcoplasmal reticulum side opens, excluding Ca2+ to SR. [6] A protein found in the heart muscle, called phospholamban (PLB), has been shown to prevent SERCA from working. It does this by binding to SERCA and reducing its attractiveness (affinity) to calcium, therefore preventing calcium intake in SR. Failure to remove Ca2+ from cytosol, prevents muscle relaxation and therefore means that muscle contraction also occurs. However, molecules such as adrenaline and norepinephanine, can prevent PLB from inhibiting SERCA. When these hormones bind to a receptor, called beta 1 adrenoceptor, which is found on the cell membrane, they produce a series of reactions (known as the cyclic AMP pathway) that produces an enzyme called kinase protein A (PKA). PKA can add phosphate to PLB (this is known as phosphorusilation), preventing it from inhibiting SERCA and allowing muscle relaxation. [7] Calcium storage Located within SR is a protein called kalequestrin. This protein can be tied to about 50 Ca2+, which reduces the amount of free Ca2+ within SR (because it is more related to kalequestrin). [8] Therefore, more calcium can be stored (kalsequestrin is said to be a buffer). It is primarily located within the connecting SR/terminal tank, in close connection with the calcium release channel (described below). [9] Calcium ion release from SR occurs in connecting SR/terminal cisternae via ryanodin receptors (RyR) and is known as calcium spark. [10] There are three types of ryanodin receptors, RyR1 (in the skeletal muscle), RyR2 (in the heart muscle) and RyR3 (in the brain). [11] Calcium release via ryanodin receptors in SR is triggered differently in different muscles. In the heart and smooth muscle, the electrical impulse (action potential) triggers calcium ions to enter the cell through calcium channel found in the cell membrane (smooth muscle) or T-tubule membrane (heart muscle). These calcium ies bind to and activate RyR, creating a greater increase in intracellular calcium. In skeletal muscles, however, the L-type calcium channel is tied to RyR. Therefore, the activation of calcium channel type L, through action potential, directly activates RyR, causing calcium release (see calcium sparks for more details). [12] Also, caffeine (found in coffee) can bind and stimulate RyR. Caffeine makes RyR more sensitive to either action potential (skeletal muscle) or calcium (cardiac or smooth muscle), thereby producing calcium sparks more often (this is partly responsible for the effect of caffeine on heart rate). [13] Triad and Junctin are proteins found within the SR membrane, which are bound to RyR. The main role of these proteins is to anchor kalsequestrin (see above) to ryanodin receptors. At normal (physiological) calcium levels, kalsequestrin binds to RyR, Triadin and Junctin, which prevents RyR from opening. [14] If the calcium concentration within SR falls too low, there will be less calcium related to caleleestrine. This means that there is more space on kalsequestrin, bind to junctin, triadine and ryanodin receptors, therefore it binds tighter. However, if the calcium inside the SR grows too high, the more calcium binds to the kalsequestrin and therefore binds to the junctin-triadin-RyR complex less firmly. RyR can therefore open and release calcium into the cell. [15] In addition to the effects PKA had on phosphorusamban (see above) that resulted in increased relaxation of the heart muscle, PKA (as well as another enzyme called calmodulin kinase II) can also phospholyze ryanodine receptors. When phosphorus, RyRs are more sensitive to calcium, therefore they open more often and for a long time. This increases calcium release from SR, increasing the rate of contraction. [16] Therefore, in the heart muscles, activation of PKA, via cyclic AMP times, results in increased muscle contraction (via phosphorusation of RyR2) and increased relaxation (via phosphorus phosphorylation), which increases the heart rate. The mechanism behind the cessation of calcium release through RyR has not yet been fully ified. Some researchers believe this is due to the accidental

closure of ryanodin receptors (known as stohastic attrition), or ryanodin receptors becoming inactive after calcium sparks,[17] while others believe that by reducing SR calcium, it triggers receptors to close (see calcium sparks for more detail). The role in rigor mortis The breakdown of sarcoplasmical reticulaum, together with the resulting release of calcium, is an important contribution to rigor mortis, stiffening muscles after death. If calcium concentration increases in sarcoplasm can then also cause muscle stiffness. References ^ Bronner, F. (2003) Extracelltial and intracellential regulation of calcium homeostasis, TheScientificWorldJournal., 1, p. 919–25. ^ Trump, B., Berezesky, I., Laiho, K., Osornio, A., Mergner, W. and Smith, M. (1980) 'The role of calcium in cell injury. Examination, Electron Microscopy Scan., p. 437-62. ^ Anatomy of sarcoplasm reticule in vertebrate skeletal muscle: Its implications for fusing excitation contractions', Zeitschrift fur Naturforschung. Section C, Biosciences., 37, p. 665–78. ^Arai, M.; Matsui, H.; Periasamy, M. (1994-1904-1901). Sarcoplasmal reticulum gene expression in cardiac hypertrophy and heart failure. Circulatory research. 74 (4): 555–564. doi:10.1161/01.OI.74.4.555. ISSN 0009-7330. PMID 8137493. ^ Periasamy, M. and Kalyanasundaram, A. (2007) SERCA pump isoforms: Their role in transport and calcium ion disease, Muscle & Nerve, 35(4), p. 430–42. ^ Kekenes-Huskey, P.M., Metzger, V.T., Grant, B.J. and McCammon, A.J. (2012b) Calcium binding and allosteric signaling mechanisms for sarcoplasmal reticulum Ca2+ ATPase, 21(10). ^ Akin, B., Hurley, T., Chen, Z. and Jones, L. (2013) Structural basis for phosphorus inhibition of calcium pumps in sarcoplasmic reticulum, Journal of Biological Chemistry., 288(42), p. 30181–91. ^ Beard, N.A.; Laver, D.R.; Dulhunty, A. F. (2004-2005-2001). Kalequestrin and calcium channel for releasing skeletal and cardiac muscles. Advances in biophysics and molecular biology. 85 (1): 33–69. doi:10.1016/j.pbiomolbio.2003.07.001. ISSN 0079-6107. PMID 15050380. ^MacLennan, DH; Wong, PT (film). Isolation of proteins that sequence calcium from the sarcoplasm reticule. Proc Natl Acad Sci USA. 68 (6): 1231–1235. doi:10.1073/pnas.68.6.1231. PMC 389160. PMID 4256614. ^Cheng, H.; Lederer, W.J.; Cannell, M.B. (1993-10-29). Calcium sparkles: elementary events of the underlying excitation-contraction of the coupling in the heart muscle. Science. 262 (5134): 740–744. doi:10.1126/science.8235594. ISSN 0036-8075. PMID 8235594. ^ Lanner, J.T., Georgiou, D.K., Joshi, A.D. and Hamilton, S.L. (2010b) Ryanodine receptors: Structure, expression, molecular details and function in calcium release, 2(11). ^ Cheng, H. and Lederer, W. (2008) Calcium Sparkles, Physiological Reviews., 88(4), pp. 1491–545. ^ Sitsapesan R, Williams AJ. Mechanisms of activation of caffeine of individual channels for the release of calcium of the sheep's cardiac sarcolasm reticuum. J Fiziol (Lond) 1990;423:425-439] ^ Zhang, L; Kelley, J. Schmeisser, G; Kobayashi, YM; Jones, LR (film). Complex formation between junktin, triadine, kalsequestrin and ryanodin receptors: cardiac compounding sarcoplasmic reticulum membrane protein. J Biol Chem. 272 (37): 23389–23397. doi:10.1074/jbc.272.37.23389. PMID 9287354. Györke, I., Hester, N., Jones, L.R. and Györke, S. (2004) Role of Calsequestrin, Triad and Junctin in the given response of cardiac Ryanodin receptors to luminal calcium, 86(4). ^ Bers, D.M. (2006) Phosphorilation of cardiac ryanodin receptors: target sites and functional consequences, 396(1). ^ Sham, J.S.K.; Dr. (1998); Discontinuation of ca2+ release by local inactivation of ryanodin receptors in heart myocytes. Proc. It's Natl. It's Acad. Sci. Now. 95 (25): 15096–15101. doi:10.1073/pnas.95.25.15096. PMC 24581. PMID 9844021. Retrieved from