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Structure of skeletal muscle and its relationship to exercise : a review of the literature

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THE STRUCTURE OF SKELETAL MUSCLE AND ITS RELATIONSHIP TO EXERCISE, A REVIEW OF THE LITERATURE

by

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INTRODUCTION

Much has been written in the past decade concerning the various forms of exercise. After reviewing the literature on this subject, the only established facts are that: 1) performance can be improved by exercise[1], and 2) there presently is no accepted way to exercise[2]. These articles have one common finding: There is no correlation between the different types of exercise and what occurs in the skeletal muscle cell. Helander[3] concludes the only way to measure the results of exercise on the muscle as a whole is to measure the cross-sectional area of the myofilament rather than the cross-sectional area of the entire muscle or the number of fibrils present. Recent work by Gorden and his workers[4] supports the concept that microscopic and biochemical data are the significant criteria of exercise and proof of the results of exercise rests on the size of the muscle fiber itself.

This paper has been written with this as the basic premise. The paper is divided into two parts: 1) a review of the literature on the structure of skeletal muscle, and 2) a review of the literature on the various forms of exercise. The anatomy of the muscle cell has been stressed with lesser emphasis being placed on the biochemical and physiological aspects since this approach seems to offer the most logical sequence for a discussion of exercise. The scope of the paper will be limited to skeletal muscle only.

PART I: The Structure of Skeletal Muscle, a Review of the Literature

HISTORY

Hippocrates, Aristotle, and other Greek philosophers thought bones gave the body carriage and poise, the tendons and nerves gave it the power to bend the joints, and the flesh (muscle) was the supporting tissue or packing between skin and bone. Galen seems to have originated the idea that muscle was contractile, but he maintained the tendons were also a part of this contractile process. In 1664, Stene showed only the fleshy body of the muscle participated in contraction. Prior to this, the idea was that a nerve juice or nerve spirit (pneuma) emanated from a central source and when it reached the muscles, caused contraction by distending them. Van Leeuwenhoeck in 1820 observed the transverse striations of muscle fiber. Fletcher and Hopkins began the era of muscle biochemistry in 1907[5].

GENERAL STRUCTURE

Skeletal muscle is constructed of many cylindrical multinucleated fibers attached to each other by fine collagen fibrils.[6] Connective tissues, which contain nerves, vessels, and fat (also called stroma),[7] form both the framework and the means of attachment of the muscle fiber.[8] Surrounding the muscle there is a connective tissue sheath (epimysium) from the deep surface of which septa pass into the muscle at irregular intervals. These septa (perimysium) invest bundles of muscle fibers. Delicate extensions of fine connective tissue come from the perimysium and pass to surround each muscle fiber (endomysium).[9] These individual muscle fibers have a length of 1 mm. to 5 cm.[8] and a diameter of 10 to 100 microns.[4,6,8] They are composed of myofilaments aligned and

embedded within the sarcoplasm.[7,8] Each fiber is surrounded by a electrically polarized membrane, the inside of which is generally a tenth of a volt negative with respect to the outside.[4] The nuclei lie at the surface of the fiber immediately under the sarcolemma surrounded by a zone of protoplasm with their long axis parallel to the adjacent sarcolemma.[6] They have an ovoid shape and are 8-10 microns long.[9]

One can divide muscle into 5 major compartments:[10]

Compartment	Biochemistry constituents	Function
1) Sarcoplasm	myogen; numerous enzymes	glycolysis
A) mitochondria	enzymes of oxidation and phosphorylation	steady-state aerobic activity or recovery from O_2 debt
B) sarcoplasmic reticulum	active concentration of calcium; possible production of relaxing substance	off and on control of active state
2) Membrane	lipoprotein structure with variable selective permiability for ions	excitation and impulse conduction
3) Fibrils	actin; myosin; tropomyosin	contraction

1) Sarcoplasm: A heterogenous substance[10], which is one of the least defined, consisting of the contents of the sarcolemma exclusive of the contractile material.[11] It occupies the space between the myofibrils and can be extracted with water, producing a low viscosity solution[12], various enzymes and the sarcoplasmic proteins: myogen, myoalbumin, globulin x, and myoglobin.

Sarcoplasmic Proteins	Myofibril Proteins
Globular	Fibrous
Low viscosity	High viscosity
Low molecular weight ($\sim$ 80,000)	High molecular weight ($\sim$ 400,000-800,000)
Soluble in H_2O or low-salt solution	Insoluble in H_2O or low-salt solution [7]

The sarcoplasm appears to be the storehouse of muscle cell nutrients,[7] thus being related to the metabolic activity of the cell. It does not seem to be involved in the structural organization which results in contraction.[12] It is made up of 5 components: A) mitochondria, B) sarcoplasmic reticulum, C) sarcoplasmic matrix, D) lipid bodies, and E) Golgi apparatus.[11]

1A. mitochondria (also called sarcosomes): The sarcosomes are an organelle of vital importance as bearers of metabolic enzymes.[13] The structure and functional characteristics appear similar to those of other cells.[6,11] Their distribution is fairly specific: clusters beneath the sarcolemma at the periphery of the fiber; in abundance near the poles of the nuclei; and at the motor end-plate regions.[6,14] They always lie outside the contractile fibrils.[11] Various shapes have been identified in human muscle: spheroids and rodlets peripherally; short, small paired rodlets at the Z band; and long rods between the myofibrils.[14] Their number in the cell determines the capacity to perform the steady state generation of metabolic energy at a high rate.[10,13] That is, muscles with constant contractile activity may have them as numerous as the fibrils. In muscles with only occasional burst of intense activity, which

are not dependent on constant respiratory metabolism, they may be almost absent.[10,14]

1B. sarcoplasmic reticulum (also called sarcotubular system): It is a submicroscopic plexiform system of membrane-bounded tubules that occupy the interfibrilar spaces throughout the muscle fiber.[15] Grossly it appears as two alternating lace-like sets of anastomosing tubule-like channels, resembling bracelets, which surround the myofilaments.[11] With the election microscope, it appears as two systems of tubules between fibrils: one, called the intermediate (central or T) element, is located near the A-I band junction in human muscle[16] and is oriented transversly; (Porter thought the middle element was a row of vesicles, but it now appears as a continuous tubule.)[16,21,22,23]; the other is oriented longitudenally and is called the terminal cisternae (or lateral elements).[17] A pair of these terminal cisternae flanking an intermediate element is called a triad. This is located near the A-I band junction, and thus there are two triads per sarcomere,[16,17] (it is located near the Z disc in other types of muscle[18].) The two outer tranverse channels are each confluent with separate longitudinal systems of anastomosing tubules. Thus a sarcotubular lacework is formed around the myofibril and links up with neighboring myofilaments to form a continuous network within the muscle fiber.[17] The association between these two suggests a connection.[5,16,19] Walker[17] feels this connection is non-tubular. Many feel the function of the sarcoplasmic reticulum is the inward transfer of excitation from the cell membrane to the fibrils.[10,20] It has been proposed that the action potential causes some influence to spread inward, probably along the middle element of the triad, which in turn causes some other component of the reticulum to diffuse a distance of 1 micron or so to reach myosin.[16]

In addition, Muscatello[18] claims the sarcoplasmic reticulum is at least partially responsible for protein synthesis in the muscle cell.

Relaxing Factor: It was described by Marsh[24] as a substance in skeletal muscle that inhibits contraction. It resides at least in part in a particulate fashion, and may be associated with the sarcoplasmic reticulum.[10] Besides a granular form, a co-factor seems to be needed.[25] The released substance appears to require magnesium, is inhibited by calcium, and functions in the presence of ATPase. It s exact mechanism is unknown. Weber[25] suggests it causes dissociation of the actomyosin system, primarily by inhibiting the ATPase system. Others feel it works by it s ability to lower calcium concentration through an active transport system.[10,26] Norris[24] claims it is premature to attribute an active relaxation process to muscle.

1C. sarcoplasmic matrix: a continuous aqueous phase surrounded by the sarcolemma. It seems to contain many small granules.[11]

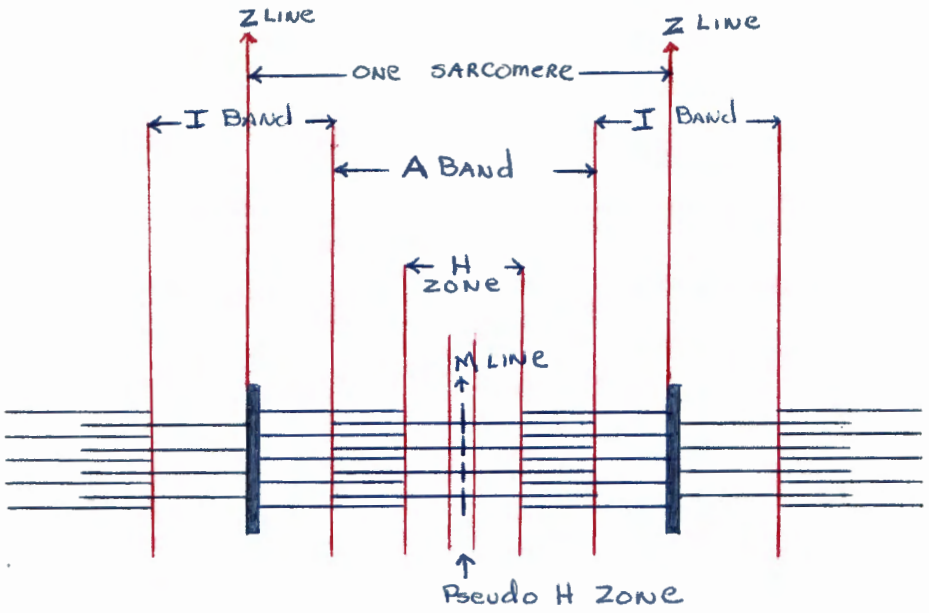
2) Cell Membrane (sarcolemma): It was first described as a thin, structureless membrane investing the muscle fiber.[11] Using the electron microscope, it appears as a transparent, tubular casing with two components:

a) plasma membrane: it appears as a thin dark line about 100 Å thick.[6,11] It seems to have a trilaminate structure, having two peaks of density, each 25 Å wide.[11] Thus, this closely resembles the plasma membrane. It is not perfectly smooth but has vesicles usually 600 Å in diameter which may be the peripheral compenents of the sarcoplasmic reticulum[6] and thus has a function of transfer in and out of the cell.[11]

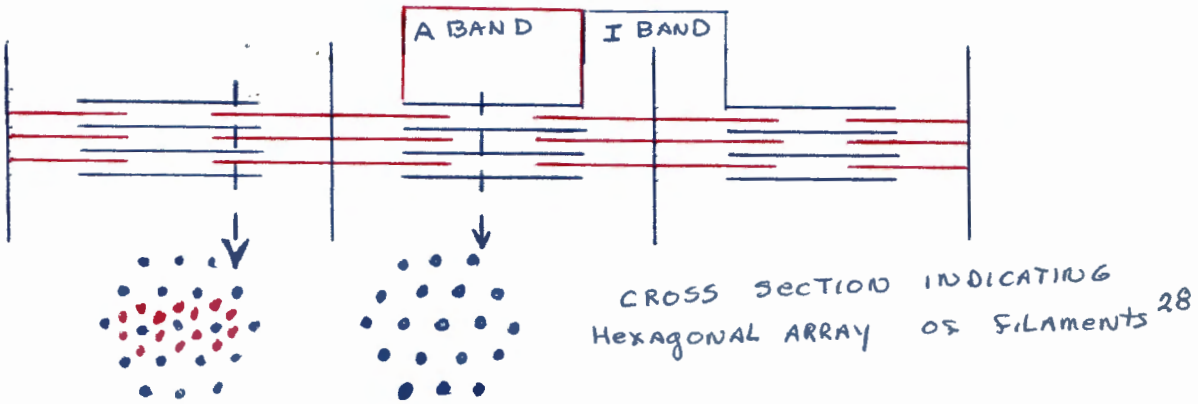
b) basement membrane: it appears as a uniform, moderately dense, structureless lamella, 300-500 Å wide,[6,11,14] gradually decreasing in density as it extends further away from the cell.[11,14] It appears to

be resistant to trauma and remains as the sarcolemmal tube that guides regenerating fibers.[6]

3) Myofilaments: An individual muscle fiber is made up of a number of elements called myofilaments[27], which are composed of protein and lie parallel to its long axis and are arranged in a series of overlapping arrays.[28] This overlap gives rise to a characteristic pattern of light and dark bands which lie in phase with similar bands of other myofilaments to give the striated pattern seen with the light microscope.[6] There are two types of protein filament: thick and thin. It has been well established that the thick filament is composed of myosin.[29] It is also well established that the thin filament is composed primarily of actin[29], but many claim tropomyosin B makes up some of its structure.[6] This thin filament composes the light (I, isotropic) band. The interdigitation of the thick and thin filaments forms the dark (A, anisotropic) band.[6,20,27,28] The I band is bisected by the Z line, a narrow dense structure 0.05 microns wide.[30] The distance between two Z lines is called the sarcomere and is about 2-3 microns long.[6,30] This distance is such that one-half the length of a thin filament and two-thirds the length of an adjacent thick filament overlap.[27] The thin filaments terminate at the edge of the Hzone, a region of low density in the center of the A band,[27] 0.3 microns wide.[31] In the center of the H zone lies a region of even lower density, the pseudo H zone, which maintains its width no matter how the length of muscle changes. This light zone surrounds a thin dark strip (M line) which is thought to be caused by a slight bulge in the center of each thick filament.[27] The gap between ends of the thin filaments appears to be bridged by fine filaments, thought to be the S filaments, running from



The major features of the sarcomere. See text for explanation.[27]



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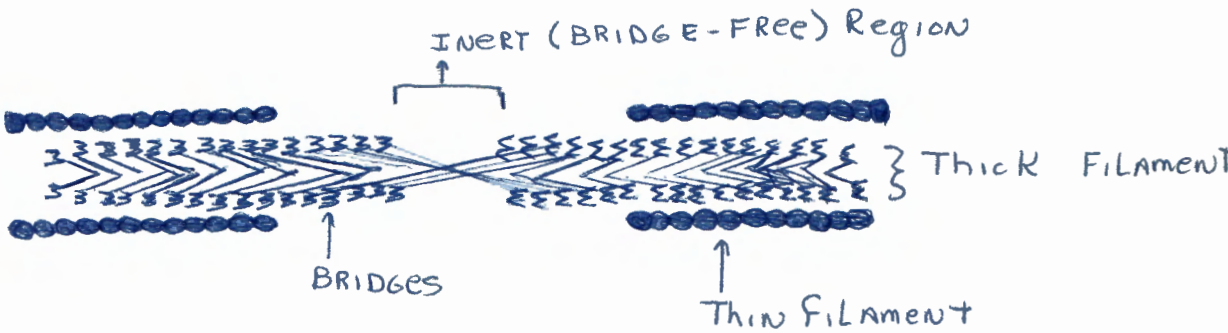


Schematic model of contact of thin and thick filaments. See text for explanation.[27]

the end of each thin filament through the H zone to the end of the corresponding thin filament at the other end of the sarcomere.[31]

On cross-section, the thick filaments lie in a hexagonal array about 450 Å apart. Where the thick and thin filaments interdigitate, the thin filaments lie in the trigonal positions within the hexagonal array. Thus, each thick filament is encircled by 6 thin ones, and each thin one is shared by 3 thick ones.[28] Each thin filament is connected to each of its neighboring thick filaments by a cross-bridge every 400 Å along the length of the region of overlap, giving about 54 bridges at resting length.[16,17] The thick filaments have a diameter of about 100 Å, and the thin filaments about 50 Å.[30] In both resting and excited muscle, the fibril lengths remain the same; thick = 1.6 microns; thin - 2.05 microns at all sarcomere lengths above 2.1 microns and all deviations from these values can be accounted for by preparation procedures.[30,32,33] Of the total dry mass of the sarcomere: 54% = A substance, 36% = I substance, and 6% = Z line; or myosin = 54% of the total protein of the sarcomere, actin = 20-25% and tropomyosin = 11%.[28]

Recently, at short sarcomere lengths, a dense zone has appeared in the center of the A band, which increases in width as the muscle shortens. This probably corresponds to a region where the thin filaments from each end of the sarcomere overlap.[27]

A regular feature of the thick filament is a region 0.15-0.20 microns wide midway along its length, where the cross-bridges appear to be absent.[27,30] This is responsible for the pseudo H zone. This zone maintains its uniform size because it is a structural feature of the filaments and is not created by their pattern of overlap.[27]

The Ultrastructure of the Individual Myofilaments: myosin, actin, and tropomyosin B comprise almost all of the fraction of soluble, fibrous, contractile proteins isolated from muscle. Certain proteins are present only in small amounts: Delta protein,[34] metamyosin,[35] and extraprotein (EP),[7,36] but there is no evidence that they play a significant role in contraction.[29]

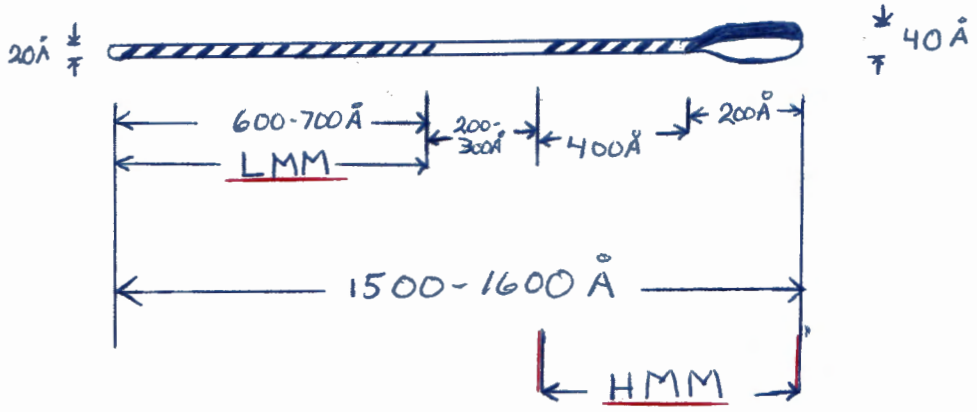
Myosin

The myosin filament is about 110 Å in diameter and 1.6 microns long.[21] It appears to have small lateral protrusions (cross-bridges) that project out at short intervals (six every 400 Å along the myosin filament[38]). It is the only fibrous protein with the properties of an enzyme.[39] There are probably about 400 myosin molecules per filament.[28,40]

The myosin molecule appears as an elongated structure that looks like a rod with a bump on its end.[29] It has a large effective volume 2.5 ml/gm,[29] a length of 1500-1700 Å,[27,29,40] width of 20-40 Å,[27,29] and a molecular weight of 450,000[41]-500,000.[29,42] It is split by Trypsin into two well-defined fragments: heavy meromyosin (HMM) and light meromyosin (LMM).

Heavy meromyosin consists of a large globular head with an alpha-helical rodlike tail.[39] The sites responsible for myosin's enzymatic activity and affinity for actin are located in the head.[27,39] The head represents 55-60% of the HMM mass,[39] is 40 Å in diameter, and accounts for one-sixth of the length of the HMM.[27] Its tail accounts for the rest and is 20 Å in diameter. The globular portion probably also serves as the cross-bridge .[27,40,43] HMM's length is about one-half and its mass about two-thirds of the whole molecule.[39] Its molecular weight is about 320,000.[28,41,42]

Schematic model of myosin indicating the inherent directionality of the filaments.[27]



One representation of myosin molecule, here pictured as with 2 stranded helical configuration.

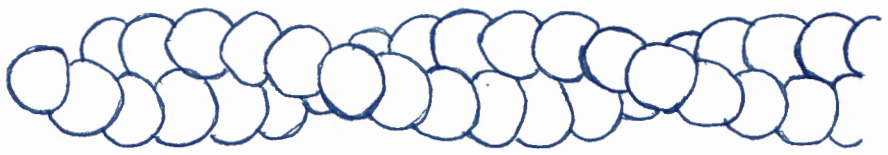
Light meromyosin has neither enzymatic activity nor an affinity for actin but it retains the solubility properties that enable it to form the same kind of structure that intact myosin does;[27] it appears to be the backbone of the molecule.[44] It appears as a simple linear strand with a molecular weight of about 120,000.[28,41,42]

Thus the entire molecule appears asymmetrical with one LMM and one HMM subunit per molecule in an end-to-end linear arrangement.[27,42] It appears to definitely have an helical arrangement of 2[42,44] or 3[45] alpha-helices twisted together. The molecule is oriented in one of two opposite directions depending on which end of the filament a given molecule is joining. The molecules aggregate with their heads pointed in one direction along half of the filament and in the opposite direction along the other half. Thus, they have an inherent directionality.[27] This also accounts for the central zone of 0.15-0.20 microns in the myosin filament without cross-bridges.[44]

Actin

The actin filament is 50-70 Å in diameter and 2.06 microns long (1 micron on either side of the Z line). The molecular arrangement resembles 2 strings of beads twisted around each other.[27] That is, it appears to be a double helix of 2 chains of actin monomers wound around each other.[28,40] There are 13 subunits per turn of helix and the strands cross over each other at intervals of 350 Å.[46,47] These subunits have a molecular weight of 60,000-70,000, a large effective volume of 2.7 ml/gm[51], and are about 55 Å in diameter; there are 600 molecules per filament.[28]

One of the striking properties of actin is the formation of fibrous aggregates, the G-F transformation, induced by the addition of salts.[52] This is said to be similar to a gas-liquid condensation, dispersed G-actin



Structure of actin = 2 chains of beads twisted into a double helix.[27]

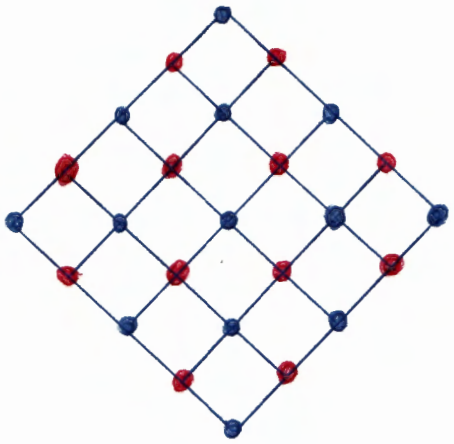
molecules corresponding to the gas, and F-actin fibers corresponding to the liquid.[53]

Hanson[47] feels actin forms only 60% of the I band and tropomyosin, much, if not all of the rest. Others agree[49,50] but do not state an amount; tropomyosin may form the backbone of the filament.[46] The transverse striations of the I band are 406 Å apart and are due to material located between the filaments.[47]

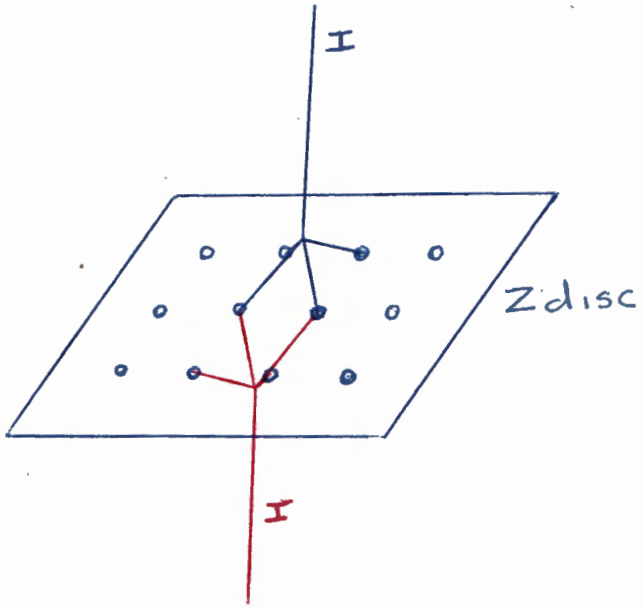
All the molecules are oriented in the same sense and they can all interact with a given myosin cross-bridge in identical fashion. The actin filaments always point away from the Z line, the filaments forming the I band on one side are all similarly oriented and reversed on the other side.[27,40]

Tropomyosin B

The position of tropomyosin is uncertain; even though evidence points to the Z-line, there is much more present than can be accounted for.[20,54] It does appear to have a structure similar to that present in the Z-line.[40,44] It is felt that part of tropomyosin forms the filament lattice in the Z-line. It appears that each thin filament branches into four subunits at the junction with the Z-line.[55] This suggests that 2 strands of tropomyosin accompany each actin filament.[47] Each rod-like projection to which a thin filament is attached on one side of the Z-line lies somewhere between 2 rod-like projections on the other side. Thus, the thin filaments appear to be arranged in a tetragonal pattern in the Z-line region while they are in a hexagonal pattern in the zone of overlap. This is less difficult to accept if it is considered that a displacement of each thin filament by 90-110 Å suffices to change a tetragonal pattern to a hexagonal one.[55]



Cross-section through the Z-disc. Black dots are I filaments from one side. Red dots are I filaments from the other side. The lines represent the Z filaments which form a tetragonal network.[47,55]



Representation of Z-disc. Black line is I filament from one sarcomere. Red line is I filament from an adjacent sarcomere.[47]

Tropomyosin itself is the most physically stable of the contractile proteins. It has a molecular weight of 53,000. It appears to be a supercoiled 2-fold alpha-helix, 341 Å long with a mean diameter of 14 Å.[29]

Contraction

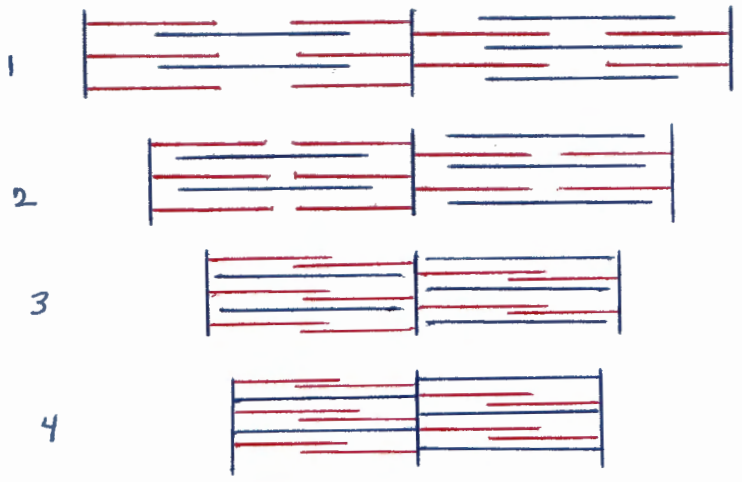
Several theories of contraction have been presented:

One filament theory of Meyer: The contractibility unit resides in a single filament. Contraction occurs by configurational changes (folding) within this single filament.[43]

Contralateral Filament theory of Szent-Gyorgyi: There are connections between the thick and thin filaments across the H zone. Contraction is due to a shift of thick filaments toward the lateral edges of the A band, drawing in contralaterally attached thin filaments.[56]

Folding theory of Podolsky: The ends of the thin filaments are fixed relative to the thick filaments on activation and the contractile force is generated by the tendency of the thin filaments to shorten by folding. The central feature of this theory is that the thin filaments have a series of sites to which substrate can bind and thus the force is proportional to the number of full sites.[56,57,58,59]

Sliding Filament theory of Huxley, et.al.: It appears that the arrival of an impulse depolarizes the cell membrane causing the release throughout the fiber of an activating substance, probably calcium, enabling one of the proteins (probably myosin) to act as an enzyme and split a phosphate group from ATP.[27] ATP appears to be the primary energy source.[60] It is produced in the sarcosomes by oxidative phosphorylation and in the sarcoplasm by glycolysis. The velocity of oxidative metabolism is limited by the availability of ADP, formed in the utilization ATP and rephosphorylated to ATP again.[10,61] As the sarcomere length changes, the

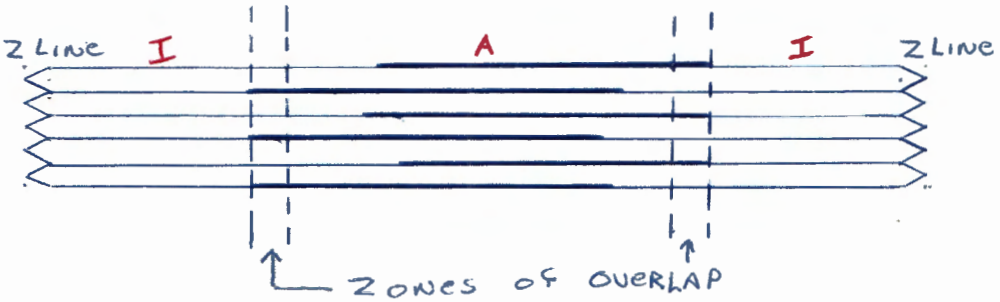


The sliding filament theory of contraction. First the H zone closes (1), then a new dense zone develops in the center of A band (2,3,4) as the thin filaments from each end of the sarcomere overlap.[27]

filaments do not perceptably change in length but slide past one another. The myosin cross-bridges are the only mechanical linkage between the filaments and they are responsible for the structural and mechanical continuity along the whole length of the muscle. These bridges probably are attached to one site on the filament for part of the contraction, then detach and re-attach themselves at a new site further along.[27] A fixed amount of relative force between the filaments is generated at each of these sites. The total tension on a thin filament is the sum of these forces exerted on it at each of the bridges which it overlaps.[57] For a given load on this system, the number of cross-bridges is just sufficient to bear the load. The rate limiting factor is the rate at which unattached cross-bridges can become attached again and develop tension.[36]

Since the thin filaments slide toward each other in the center of the A band, it is required that all the elements of force generated by the cross-bridges in one-half of the A band be oriented in the same direction and this direction be reversed in the other half. Electron microscopy has shown myosin is arrayed so that it points in the same direction in one-half of the A band and opposite in the other.[27,56]

Maximum tension occurs at sarcomere length 2.2 microns,[62] where the ends of the thin filaments are in the inert region (0.15-0.2 microns) at the center of the thick filament, thus giving the maximum number of overlapping sites.[57] As the sarcomere length is stretched beyond a certain point, according to this theory, the rise of tension should decrease since total tension is proportional to the zone of overlap. Thus tension should start to decrease beyond 2.0 microns and reach zero at 3.5[63] 3.65 microns[20,64] (the sum of the thick (about 1.5 microns) and thin



One possible explanation for shortening of highly stretched fibers. As the fibril is stretched, the ends of the I filaments are pulled into positions where they may overlap the ends of the A filaments, creating new zones of overlap at each A-I border.[65]

(about 2.0 Microns) is about equal to 3.5 microns). Some investigators have found a weak contraction at sarcomere length 4 microns.[65] This could be due to a nonuniformity of striation spacing so that overlap, while absent for most of the length of the fiber, is present at some places, especially near the fiber ends.[30] It could also be that the ends of the thin filaments are pulled into positions where they approach and actually overlap the thick filament, thus creating new zones of overlap at each A-I junction.[65]

It has been shown also that tension decreases below sarcomere length 2 microns. This may be due to an increase in passive resistence to the sliding movement caused by tighter packing of the filaments and by compression of the thick filaments when they come up to the Z line.[57] As the sarcomere length becomes less than 50% of the resting length, the thick filaments actually crumple as they hit the Z disc and contraction bands form as the material piles up.[41] The thin filaments are not so impeded and are free to interdigitate when they meet in the middle of the sarcomere.[37]

In the resting state, the cross-bridges are not attached to the thin filaments so that the latter are free to slide, explaining the high extensibility and relative plasticity of resting muscle. With rigor, the cross-bridges become permanently attached to the thin filaments; thus the muscle becomes rigid since sliding can no longer occur.[30,38]

It now appears that this sliding filament theory is the most acceptable.[6,25,43,56,57,64,66]

PART II: Exercise

Exercise may be classified according to the quality developed in the

exercised muscle: power, endurance, speed, and coordination.[67] Exercise may also be classified according to the results of the muscle contraction. If a muscle is allowed to pass at least through part of its useful arc, an isotonic contraction occurs. If no shortening is incurred, an isometric contraction occurs. In the laboratory, two ends of an isolated muscle can be fixed so that with a tetanic stimulus, the contraction develops tension without shortening (isometric). If one end is fixed and the other loaded, the entire muscle shortens (isotonic). At the level of the single motor unit, all fibers seem to contract nearly isometrically. In natural motor acts, whole muscles fail to show isotonic features. On the other hand, tension is only approximately isometric because of the elasticity of the soft tissue attachments of muscle to bone. It may be that isometric and isotonic exercise are only special features of a laboratory system. It thus seems better to classify muscle that works against an immovable resistance as static, implying that no angular motion of the lever arm has occurred; muscle that moves a weight through a distance is dynamic, implying movement of the lever arm around its joint axis.[4]

Several variations of static and dynamic exercises have been recorded in the literature.

Static

1) Hettinger and Muller claim one static exercise per day lasting 6 seconds with two-thirds maximum strength gives maximum training.[68]

2) Full static exercise is better than two-thirds.[69]

3) A 15 second exercise is better than a 6 second one.[1]

Dynamic

1) Hypertrophy program: designed to create muscle hypertrophy and secondarily increase strength. All exercise is done in 2-3 sets of 8-10 repetitions using the maximum weight that can be lifted. It is done on alternate days.

2) Power Program: the starting weight is never less than can be lifted 10 times. The weight is increased with each set and the number of repetitions decreased until only one repetition can be done. It is done 4-5 days per week.[70]

3) Low resistance with high repetitions: said to increase endurance but not power.

4) High resistance with low repetitions: said to increase power but not endurance.[67]

5) Overload Principle: muscles made to contract repetitively at levels of performance which strain the limits of capacity, respond by hypertrophy. The amount of work done per unit time can be either increased by increasing the cadence (speed) at which the exercise is performed, or by increasing the resistance against which the muscle shortens. Speed and load are mutually dependent variables. There is an optimal rhythm of working of exercises of every severity. Thus the overload principle may occur by holding either one constant and increasing the other or by increasing both concurrently.[71]

The results of all these programs are many and varied. Many authors feel static exercise is the better form of exercise.[72,73,74,75,76,77,78] Others claim dynamic is better.[2,68,70,79] Many feel the results are about the same.[69,76,79,80,81]

There have been numerous attempts to explain the inconsistencies listed above. One train of thought is to explain this by mechanisms involving things other than the muscle itself. A great deal of increased strength is due to improved neuromuscular adaptations, including the spread of neuronal activity from the initial focus to the anterior horn cells innervating functionally related muscles and a reduction of inhibiting impulses.[72] Some feel the muscle and peripheral nerves are the most stable links in the neuromuscular sequence that produces voluntary contraction and the causes of differences of strength must be sought in central mechanisms. Strength appears to be limited by psychologically induced inhibitions. (It was found that a shot, a shout, and hypnosis could increase strength by 7.4%, 12.2%, and 26.5% respectively.)[89] The capacity

with which performance can be augmented when stress is imposed suggests something other than hypertrophy of contractile tissues is responsible for changes. A significant proportion of voluntary exercise is due to motor learning.[71,80]

Another school of thought attempts to explain these differences by changes in the muscle itself: The results of muscle training are directly related to the function of that muscle and as such, exercises should be based on muscle function.[72] Failure to discriminate the various classes of exercises leads to the use of the wrong type of exercise to develop the quality needed in the muscle.[67] Tension depends on the number of fibers contracting and the frequency of their response. No voluntary effort can recruit more motor units than the number required by the magnitude of the task.[71]

Others have broken exercise down into three variables: frequency, duration, and intensity.[61] Some stress the importance of the number of contractions per unit time.[73,82] Others feel the duration of each contraction is the important factor.[73] Still others claim muscle tension is the effective factor in developing strength.[2,67,69,71,73,80]

The capacity of muscle to increase in size with exercise is common knowledge.[9] It has been generally concluded that hypertrophy is not due to an increase in the number of fibers present but from an enlargement of the individual fibers. Some investigators have found an increase in the number of fibers along with an increase in the size of the fibers.[83,84]

Goldspenk[85] feels there are two types of fibers present, a large phase fiber (40 microns in diameter) and a small phase fiber (20 microns in diameter). With exercise the small phase fibers enlarge to become large

fibers, developing at the expense of the extracellular components which decrease. Thus the initial effect of exercise may be not to produce hypertrophy of the whole muscle, but to consolidate the tissue. Further exercise leads almost to the exclusion of extracellular components, and then there will be an increase in the girth of the whole muscle with exercise. He feels muscle responds to work by the conversion of these small fibers to large fibers, producing a 4-fold increase in the contractile machinery.

Recent evidence[4] has suggested that dynamic exercise is associated with an increase in the myofibrils along with hypertrophy of the muscle fibers, but not necessarily muscle mass; that is, forceful exercise appears to increase myofilamental proteins and decrease sarcoplasmic proteins.

It can thus be concluded that muscle reacts to exercise by some change in form and composition of its fibers.

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