Muscle Histology Myths vs Muscle Histology Realities

Muscle Histology is a complex structure allowing multiple myths to occur. It is
also compounded by reiteration ad nauseam. 5 major myths are currently being promoted. Any one of which would render the reiteration inoperable.

In the first 14, the myth promoters seemed to be unaware of the reality. The promoters of 15 actually viewed and described the 5 realities. But they were
never mentioned again and did not show-up in their construct of the Histology. They must have been left on the cutting room floor.

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A construction of the Histology using the missing realities will show how it
should be structured and function. A comparison between the usual parenchymal Histology and the stromal Histology will also be made.

COMPARISON

The fibril Histology consists of multiple molecules linked in reiteration.

The tendon histology consists of only one type of molecule (collagen) that is
repeated but not interlinked. A fibril is made by twisting 7 collagens together.
This simpler Histology is described fully and accurately in Biochem books

The actin filaments carry the force and movement they helped to produce to the tendons. The Actin is a triple alpha helix.

The tendons carry the force and movement to the bone attachment.
The collagen is also a triple alpha helix.

The myosin cross bridge pivots on an actin globule to produce movement of the actin filament. The head of the cross bridge acts as a motion amplifier at this beginning of the muscle.

The tendons attach near the joints of the bones to make the bones act as motion amplifiers at the end of the muscle.

Ions are much involved in the function of the fibril Histology.

Ions play a role in the collagen but not as a part of muscle function. The hydroxyproline in collagen has a unique hydrogen bonding that allows collagen to biologically produce something similar to inorganic piezoelectricity. Maintaining the Greek protocol would make it tentomalelectricity because it is generated by stretching.
It is used to stimulate the fibroblasts to make more collagen.

THE FIBRIL HISTOLOGY

This Histology is inside the muscle fibers surrounded by the endomysium. It is composed of two different organs that are reiteratively intertwined. There
is a large hollow nerve which is insulated from the endomysium by a fibrous basement membrane. To differentiate the tissues, the Greek sarco will be used for tissues not directly involved with actomyosin and the Latin mayo for those that are. The outer nerve tissue is the sarcolemma and has
its own satellite nucleus to produce proteins for nerve tissue.
Transverse tubules penetrate the interior and form a cross sectional reiteration of interconnected circular patterns. These patterns are also reiterated lengthwise at a specific distance.
The circular fibrils are then fit into all of the loops. They are surrounded by myoplasm. This, in turn, is surrounded by the sarcoplasmic reticulum. This is where the Ca++ ion that was referred in the comparison section is stored and released. These structures also encircle the fibrils and span between transverse tubules. They have an enlarged terminal cistern at each end. This intermediary between the nerve and the actinomyosin had to wait for the electron microscope to finally, be found. Before the electron microscope, the action potential at

the motor end plate of the muscle fibers was measured to reach peak value in 2ms and return leaving a 4ms narrow pulse. This produced a 50 ms wide muscle twitch at the tendon. There seemed to be some kind of motion amplifier involved.

This brings us back to the sarcoplasmic reticulum. Keep an eye on the ions.

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The previous amplifications discussed were produced structurally by using a mechanical advantage. The sarcoplasmic Ca++ ion approach Increases the number of cross bridge cycle activations over that which the narrow nerve pulse could accomplish.

The cycle activation is now produced by the concentration level of the Ca++ ions.

The number of Ca++ ions and. the number of opposing Ca pumps located in the reticular membrane are controlled so that the amplification will be squelched when the basic twitch has the proper time width.

The basic fast twitch starts with a rapid rise. This requires that the myoplasmic Ca++ concentration must be an explosive transfer. The terminal cisterns each contain an intrareticular membrane that is studded with reiterated granular molecules called calsequestrin. Each molecule performs a biological fete of magic by holding 43 Ca++ ions in a highly repulsive state.

This is the "dynamite" for the "explosion".

When the motor end plate is activated (the end plate is located in the middle of the length of the fibrils so that the synchronicity of the reiteration activities will be maximized) the transverse tubules depolarize. The "explosion" occurs

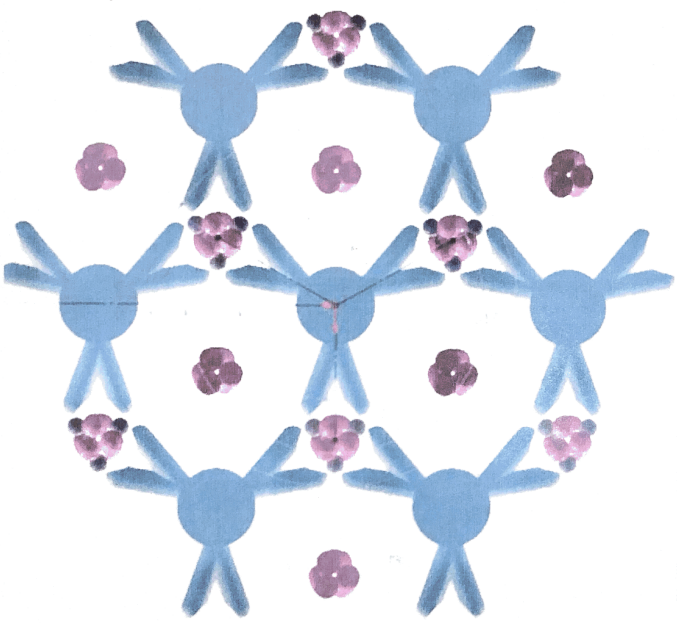
The calsequestrin has been using the negative field voltage from the t-tubules

to hold the Ca++ ions. This was accomplished by using hollow pillars which.
can carry a charge inside while insulating the outside. They touch the t-tubules
on one end and the membrane holding the calsequestrin on the other. They are
fixed to the reticular membrane to prevent it from touching the charged
membranes. The activity in the sarcoplasm casts the die for what happens in
the myoplasm in that one ion begets another, etc. There will be a panoply of ions
(Some chelates, some not).
The incoming herd of Ca** ions will be attracted to a negative charge offered
by the tropönin. The tropönin is too small to have its own ions. The tropönin
must borrow a negative charge just as the calsequestrin did. The tropönin is in
contact with two adjoining actin globules. The globules would have to carry a
negative charge, most likely free Cl- ions. Inside the globules'
The tropönin can only offer borrowed negative charges requiring tropomyosin
to carry a positive charge. (The tropomyosin has the same amino acid
sequence as the flexible tail on the cross bridge). The array of Ca**ions
stiffens it some but leaves it flexible enough to bend 17degrees as it follows
the fibrous actin. The positive field charge it emits between tropönins
floats the straddling cross bridge heads to keep them away from the negative
actin globules. The field also keeps the visiting free Ca**ions moving
toward the tropönins or back to the sarcoplasmic reticulum.

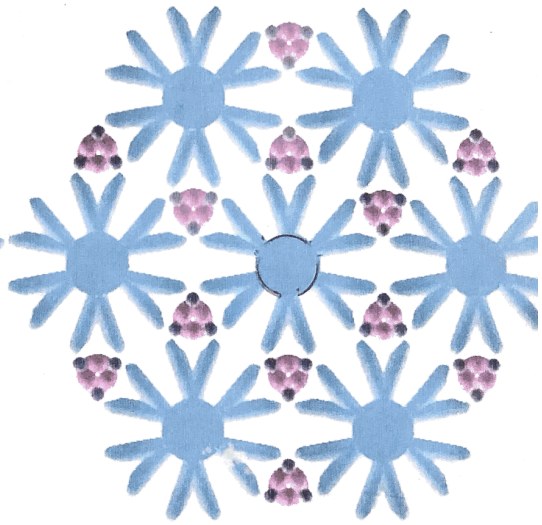
The myosin filament is also known as the thick filament. It consists mainly
of heavy meromyosin also known as heavy chain. There are 2 heavy

chains and 5 light chains associated with each cross bridge assembly. The
cross bridge tail which was mentioned earlier, is the longest of the 5 alpha helical light chains. The heavy chains are made by binding 2 light chains into an alpha helical super coil which is rigid.
The perpendicular portion of the cross bridge is 2 globular heads with a common base and a 40 degree angle between them. The heads lye in a plane that is perpendicular to the axis of the filament. Each head contains a rigid heavy chain "spine" so it can act as a motion amplifier.
The 2 very short light chains already mentioned provide 2 different functions. The longer one carries Mg++ ions. The last of the ions!
This will allow the tip of the head to bind to the actin globules and perform the power stroke by pivoting on its attachment point.
The other light chain holds 2 adenosine molecules in varying degrees of Phosphorylation. They function to break the rigor at the end of the stroke.
As the cross bridge pivots, it develops an angle in the tail resulting in a Transverse force which will also be used to break the rigor A triad of cross bridges on both the myosin and the actin filaments controls this force throughout the reiteration,
As the cross bridges translocate (no sliding, please) the actin filament a cross
bridge to actin bind must be maintained by using consecutive hand-offs. The alternating triads make that possible.
The tropönin is kidney bean shaped because it is curved to follow the curvature of the 2 actin globules it rests against.
The 2 cross bridge head tips perform opposite functions. One shortens the sarcomere while the other lengthens the sarcomere. Obviously, they would not operate simultaneously in the same muscle. The tropönin determines which will operate by offering the negative voltage to attract the free Ca**ions on one side of the tropönin only. The ion will repel the positively. charged tip further away from the actin globules thereby rotating the tip to be used onto the actin globules for an instant bind . The cross bridge head is then attracted to pivot on the contact point to begin moving the actin filament. There is a concavity next to the pivot point that has the same spherical curvature as the actin

globules. Mg **ions located from the pivot point to half way along the concavity will complete the stroke. The stroke stops in rigor precisely when the concavity is filled. (It appears as though the cross bridge is starting to swallow the actin globules).
The alternate heads have the concavity located at the opposite polarity.
The rigor is broken by using ATP energy to briefly form a triple negative orthophosphate ion. Recharging the ATP needs enough time that it requires an additional "holiday" between tropönins
The lengthening head works in tandem coordination with the shortening head of associated muscles. There is no passive dragging.



MYOSIN CROSS BRIDGE TRIAD



ADJACENT TRIADS