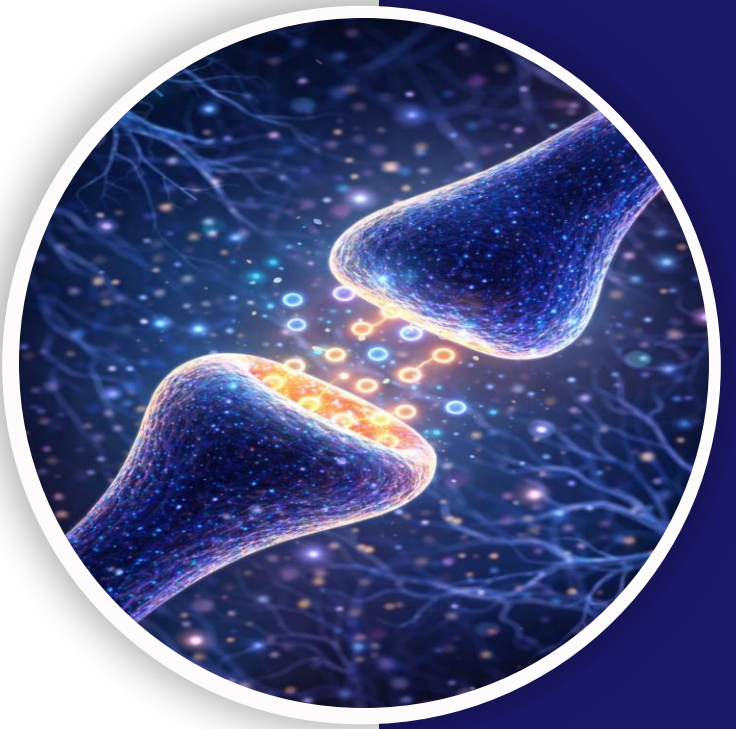
بسم الله الرحمٰن الرحيم

(وَفَوْقَ كُلِّ ذِي عِلْمٍ عَلِيمٌ)

Physiology | MID 2

Muscle Physiology pt.1



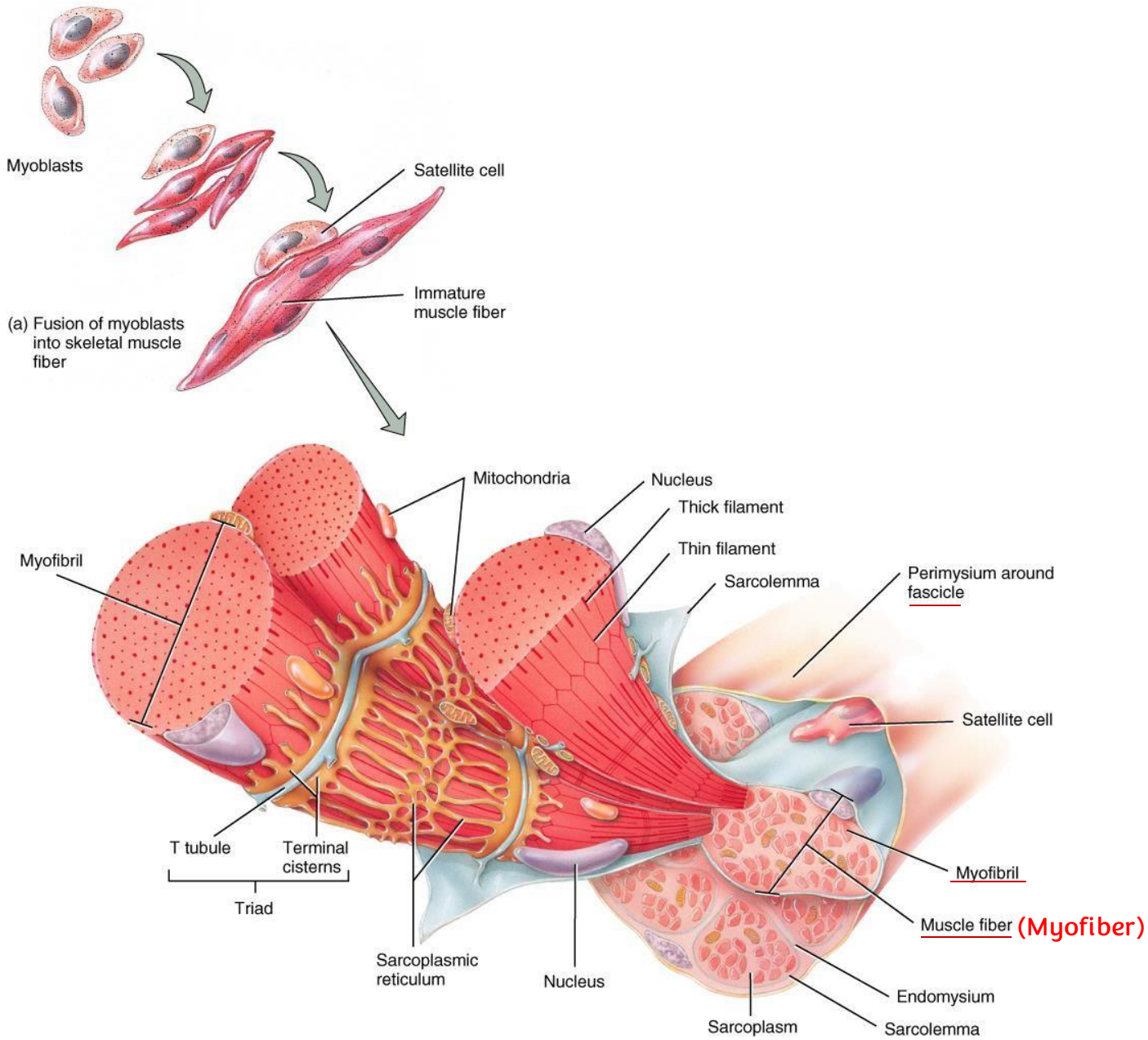
Written by : Noor Saif

Reviewed by : Maram Darweesh

Muscle Physiology Ref: Guyton, chapters: 6,7,8

Types of Muscle

Fig. 10.03


Myoblasts
Satellite cell
Immature muscle fiber
(a) Fusion of myoblasts into skeletal muscle fiber
Mitochondria
Nucleus
Thick filament
Thin filament
Sarcolemma
Perimysium around fascicle
Myofibril
Satellite cell
T tubule
Terminal cisterns
Triad
Myofibril
Muscle fiber (Myofiber)
Sarcoplasmic reticulum
Nucleus
Endomysium
Sarcoplasm
Sarcolemma
(b) Details of several myofibrils
10.03

*regarding slides 3 & 5

Muscle cells are also called muscle fibers Because they have an elongated shape, This is important for its function to allow shortening.
Each muscle is composed of fascicles, and in each fascicle there are muscle fibers.
In slide 5, we can see one of the muscle fibers and the type of skeletal muscles are striated muscles, why striated?
Because under the microscope using a specific dye which can color specific structures, we have this striated appearance of the muscle fiber.
This is one fiber also it is called Myofiber remember that anything related to muscles we add the prefix "myo"
There is also some variations in the components of the muscle fibers
Plasma membrane of the muscle is called sarcolemma, the cytoplasm of muscle is sarcoplasm, endoplasmic reticulum of muscle is sarcoplasmic reticulum.

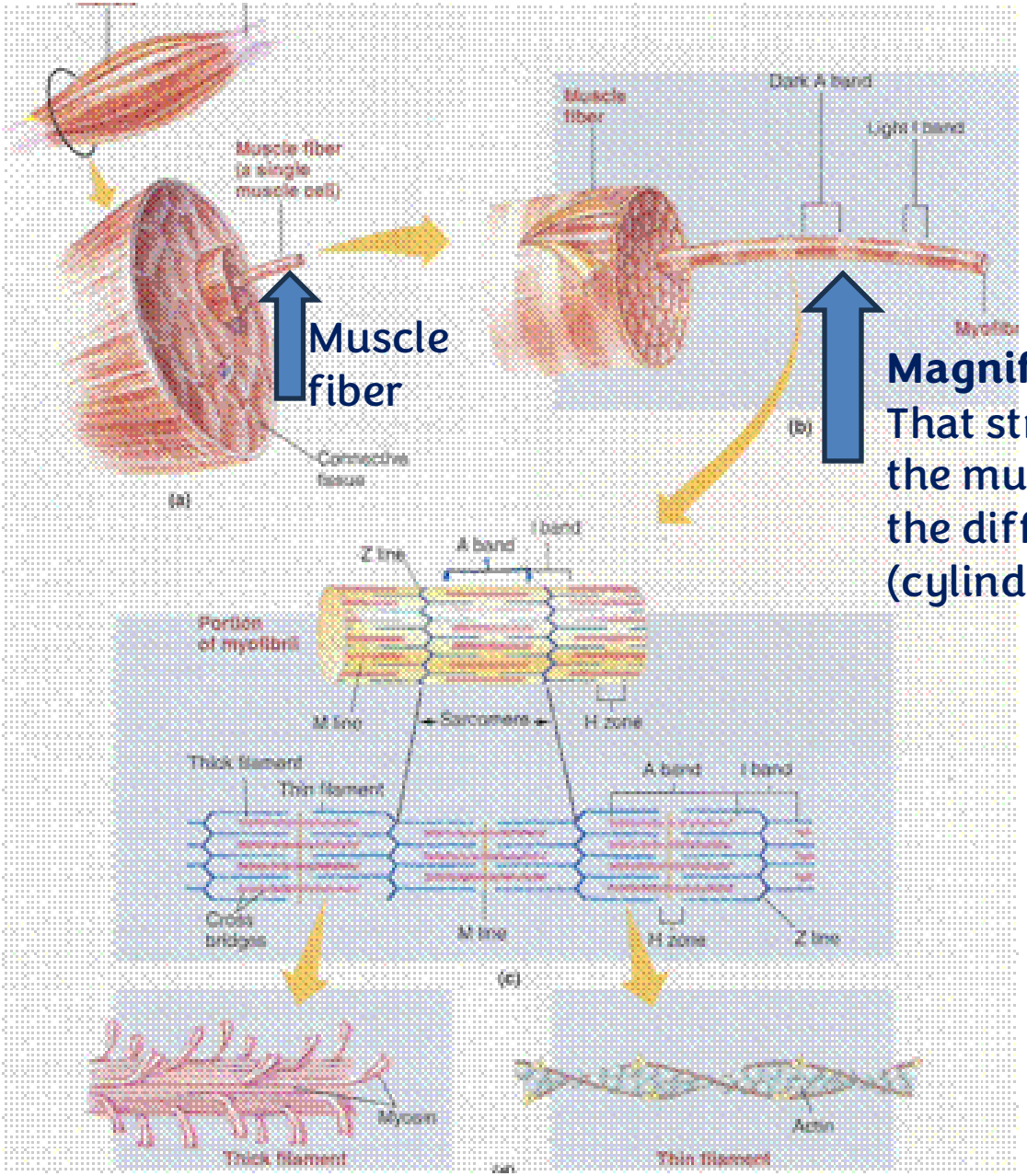
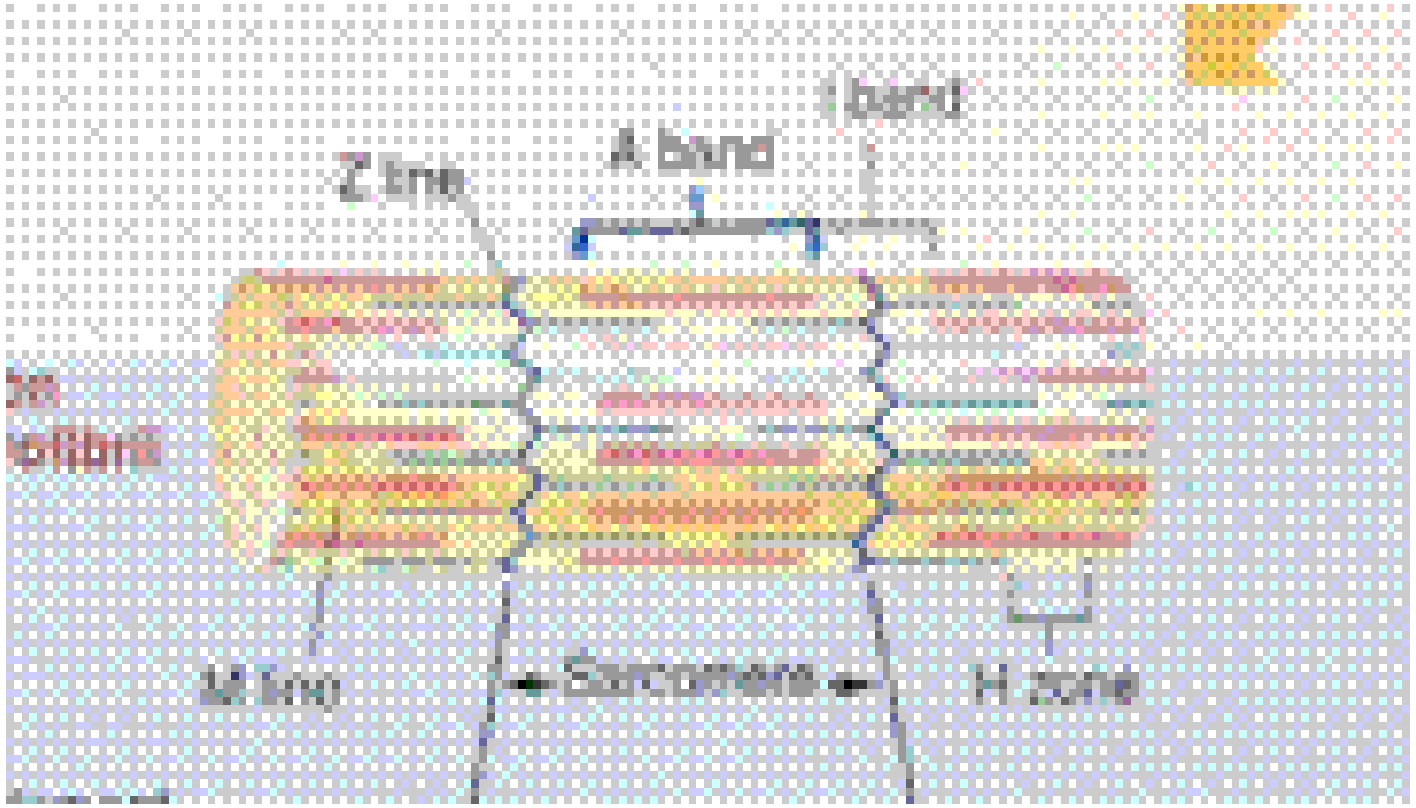


Figure 2. The components of a muscle.
Taken from Sherwood, 2004.

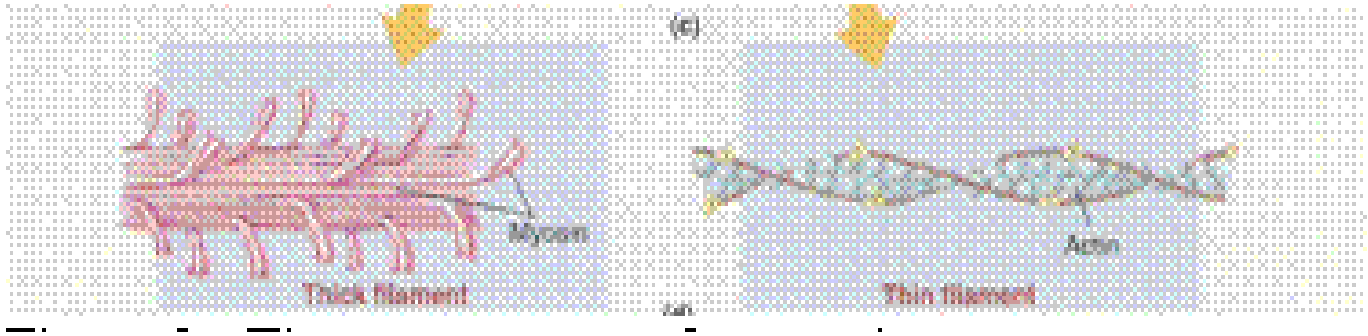
Magnified striated appearance.
That striated shape comes from the cylindrical Structures inside the muscle fibers which are called myofibrils. Make sure to know the difference between myofiber (muscle cell) and myofibril (cylindrical structures found in muscle cells) Both are striated

- Where does that striation come from?
- Simply because of the arrangement of thick and thin filaments in these myofibrils, two types of filaments arranged.
- We can get darker regions which are showing the thick filaments and lighter regions which are showing thin filaments only

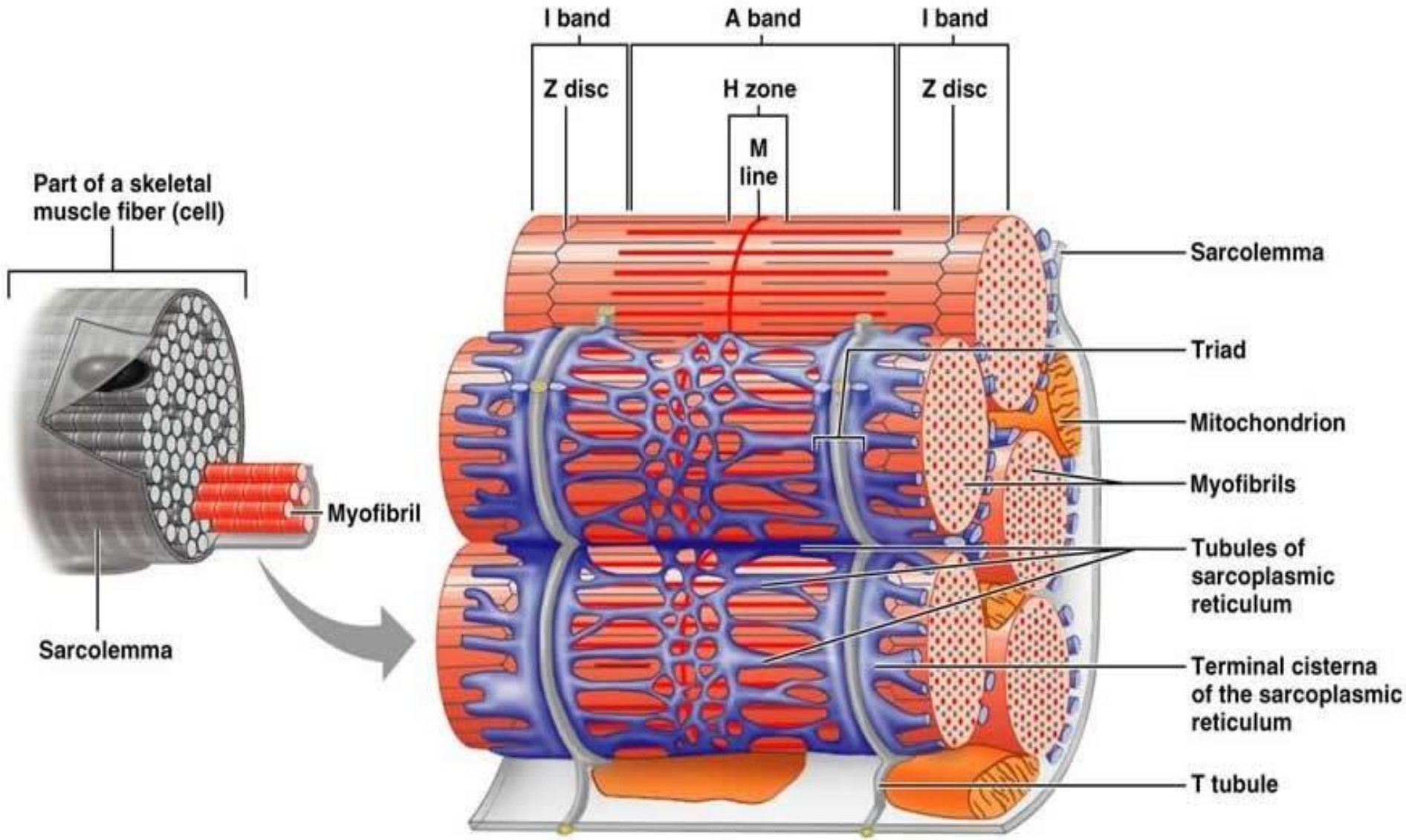


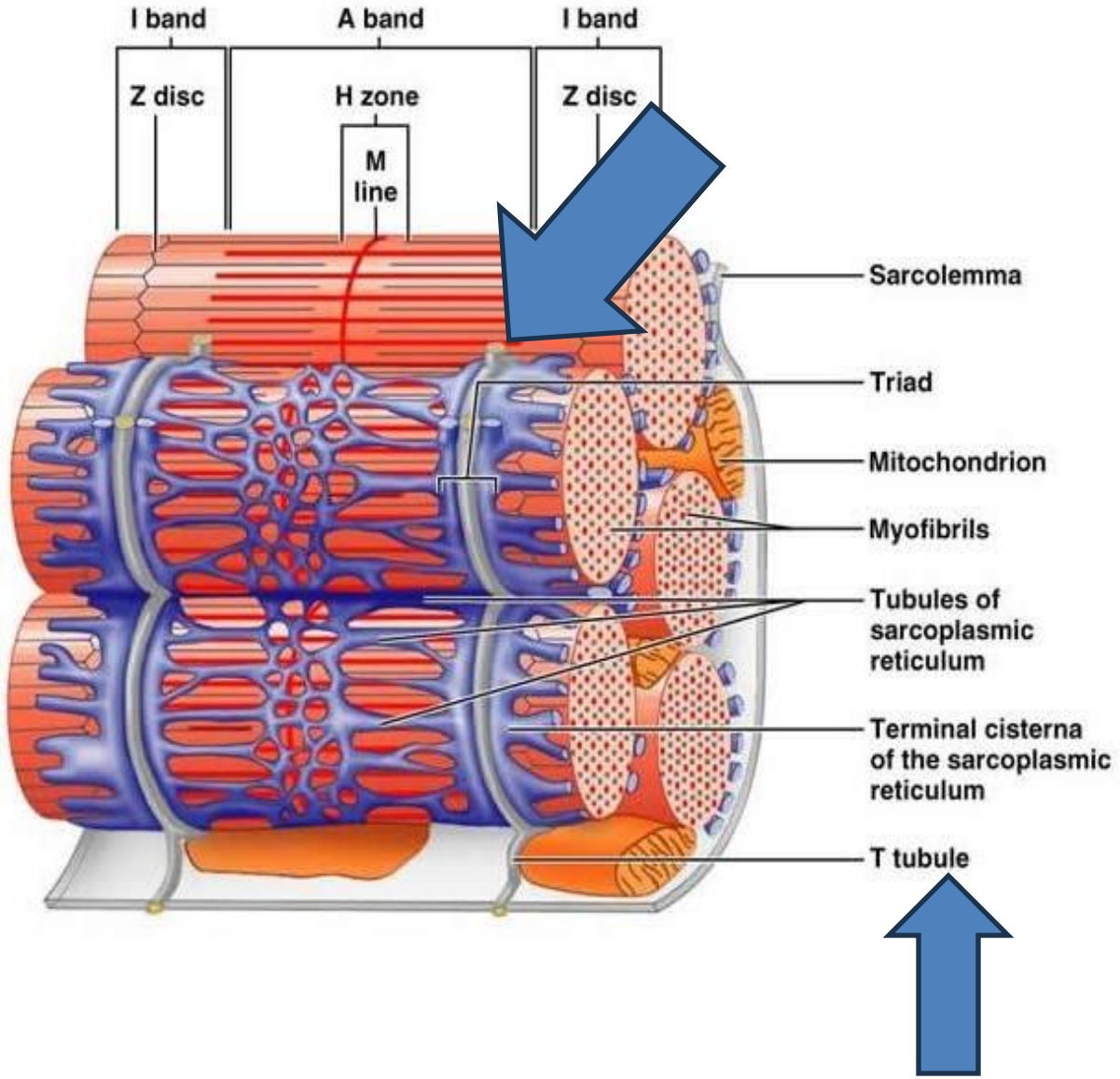
The Z line represented in the image in 2D it's called line, in 3D it's called disc
The function of the structure is holding the thin filaments in both directions

- The distance between the two Z lines is called the "sarcomere" ,,**sarcomere is the functional unit of a muscle fiber.**
- The region corresponding to thick filaments is called "A" band
- The region where we have the lighter part of that band it is called the "I" band.



Everything is arranged based on these the thick and thin filaments <--(magnified image)


I band
A band
I band
Z disc
H zone
Z disc
M line
Part of a skeletal muscle fiber (cell)
Myofibril
Sarcolemma
Sarcolemma
Triad
Mitochondrion
Myofibrils
Tubules of sarcoplasmic reticulum
Terminal cisterna of the sarcoplasmic reticulum
T tubule

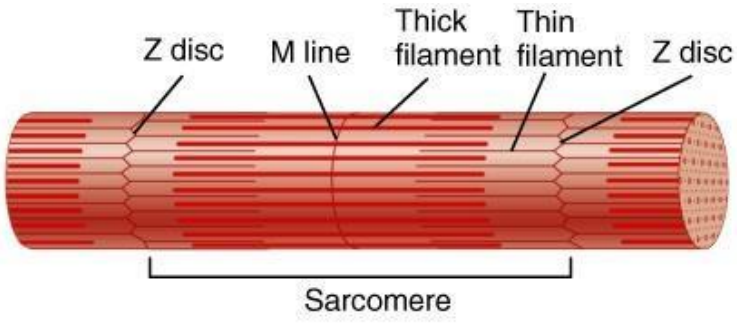


The sarcoplasmic reticulum (labeled in the image) of a striated **skeletal** muscle, is located between “A band” & “I band”. There is variation between different types of muscle cells for example this structure (sarcoplasmic reticulum) is found near the Z disc in **cardiac** muscles
In addition in skeletal muscles there are no junctions no contact between muscle fibers. while there are junctions between cardiac muscles

This is transverse tubule

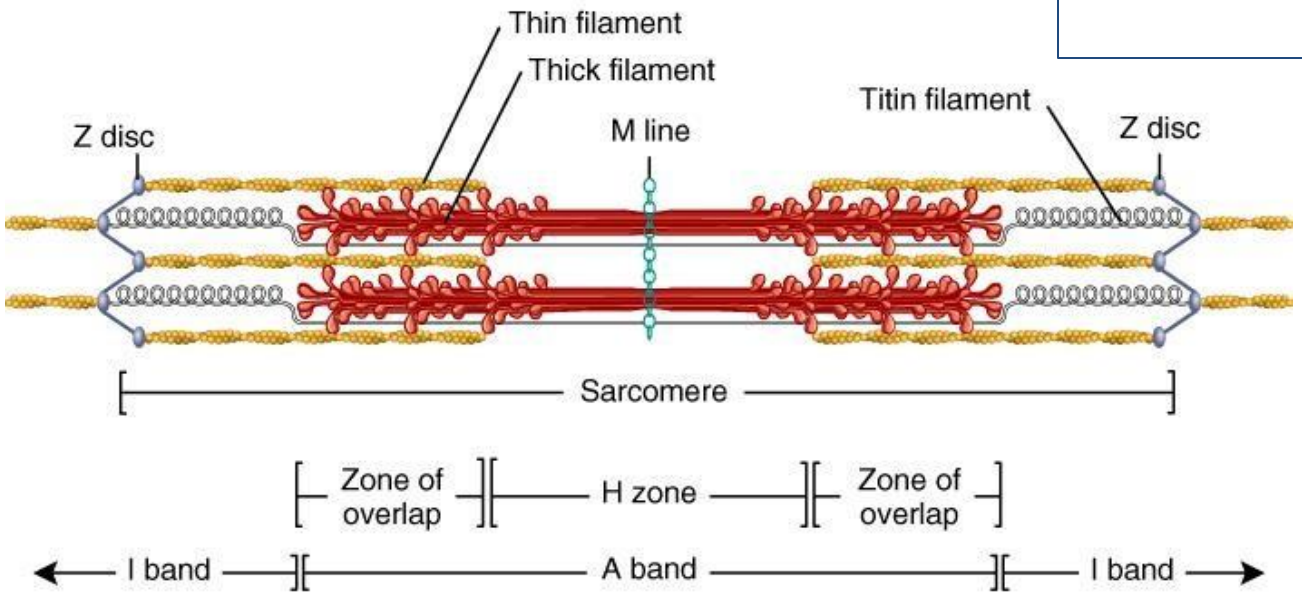
Fig. 10.04

Now, let's analyze the ultrastructure of myofibrils
To understand how we can get the interaction between thick and thin filaments to get the contractile property of the muscle



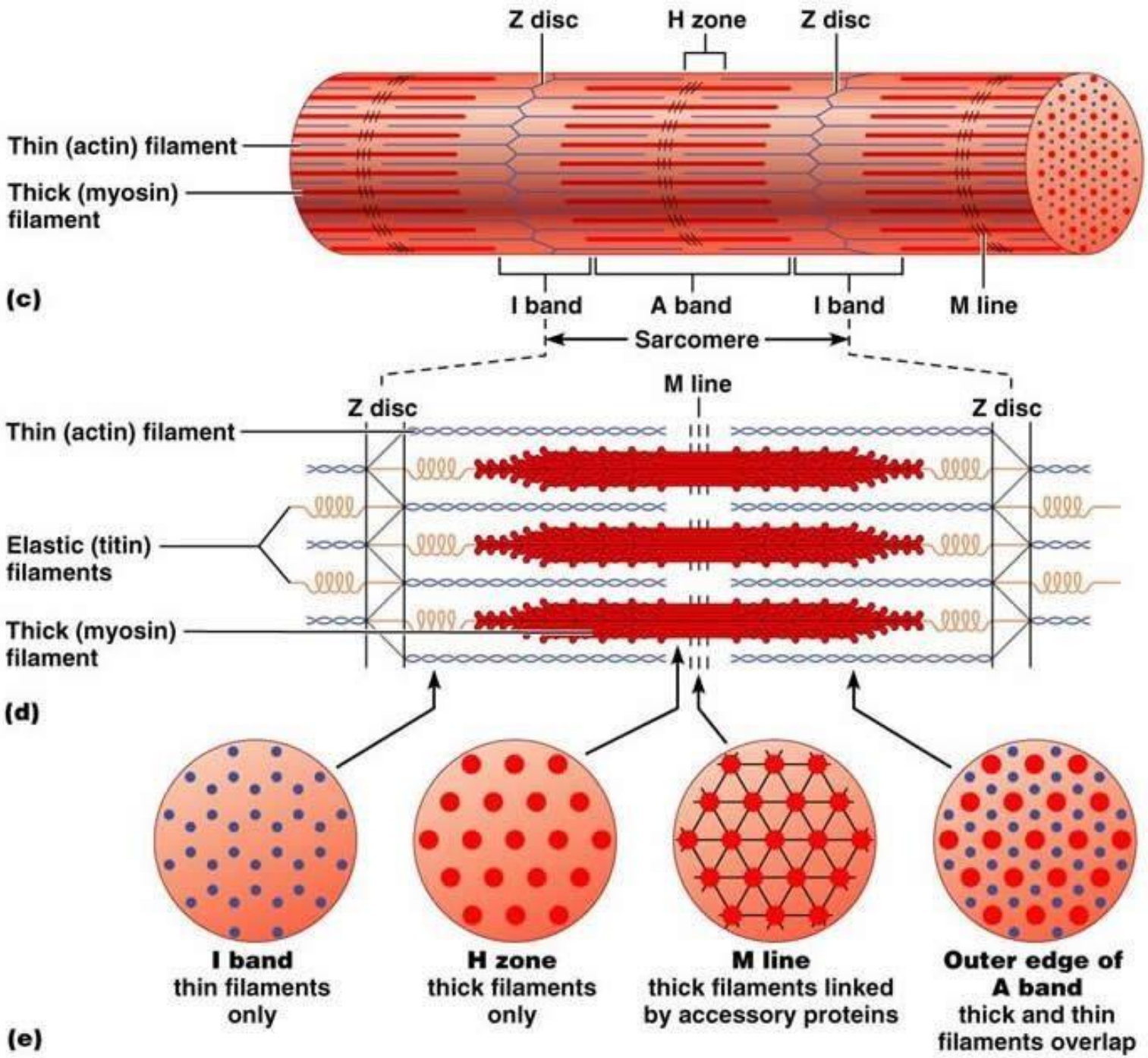
(a) Myofibril

We can find regions with only thick or only thin filaments
"H" zone has only thick filaments
"I" band has only thin filaments
Overlap in thick and thin filaments is also found

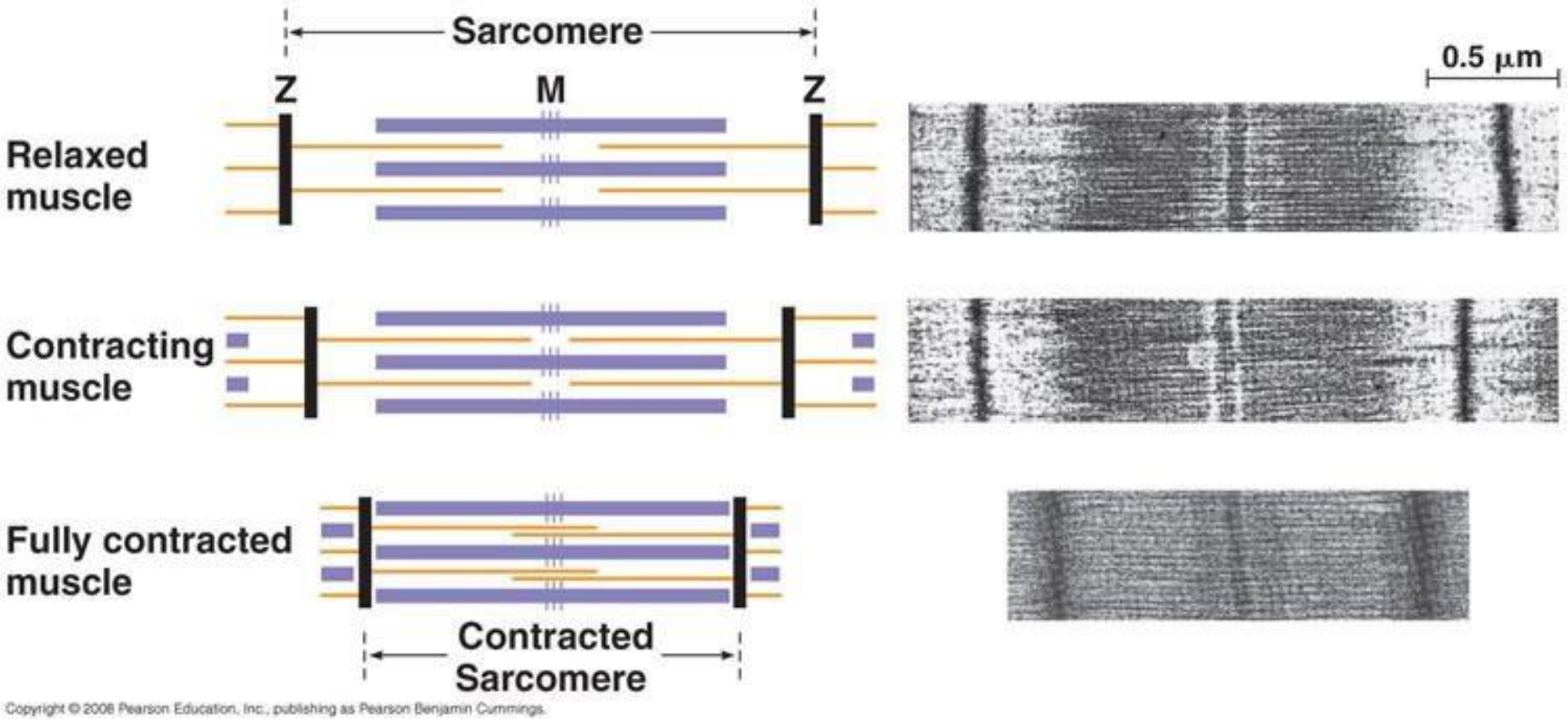


(b) Filaments

10.04


Z disc
H zone
Z disc
Thin (actin) filament
Thick (myosin) filament
(c)
I band
A band
I band
M line
Sarcomere
M line
Z disc
Z disc
Thin (actin) filament
Elastic (titin) filaments
Thick (myosin) filament
(d)
I band
thin filaments only
H zone
thick filaments only
M line
thick filaments linked by accessory proteins
Outer edge of A band
thick and thin filaments overlap
(e)

The picture above is a transverse section
You can find only thick or only thin filaments out of the overlap zone
At the overlap zone , each thick filament surrounded by six thin filaments and each thin filament is surrounded by three thick filaments so the ratio of thin to thick filaments is 2:1
In the middle of the "A" band we have a network structure of protein which is holding the thick filaments → this is the "M" line in the picture above, this is same as the "Z" disc in the "I" band holding the thin filaments

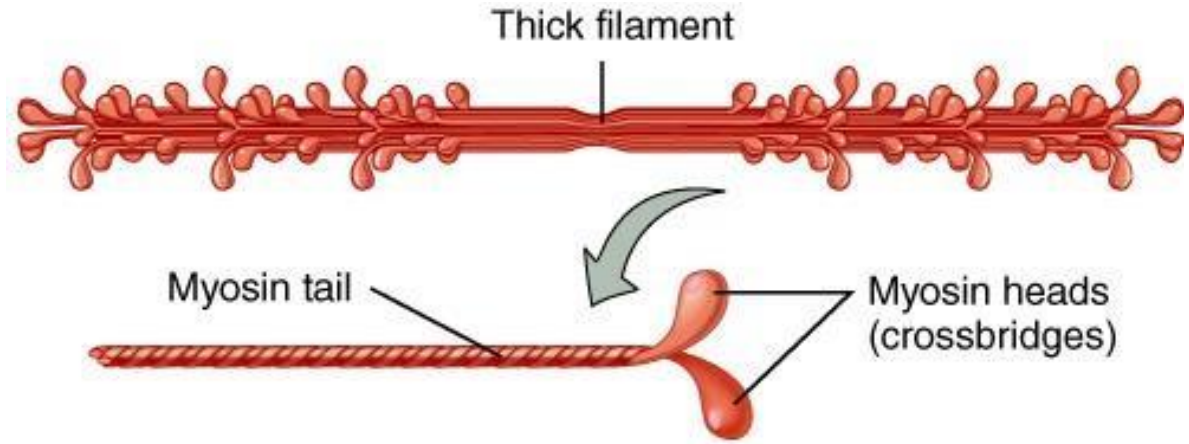


When shortening occurs (contraction) the whole sarcomere becomes shorter along with the "H" zone

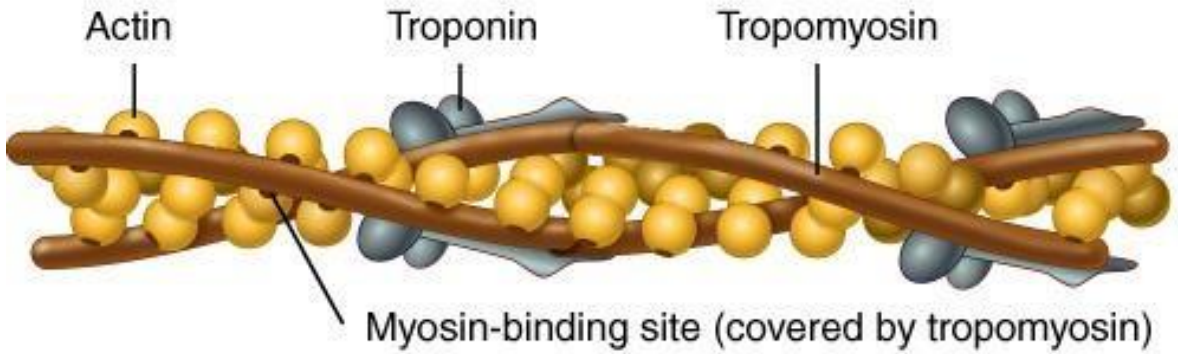
There is no change in the "A" band but there is change in the "I" band because of sliding , so the shortening happens by sliding of thin filaments over the thick filaments

We will see later on that we can get contraction without shortening by the interaction process between thin and thick filaments

Fig. 10.06

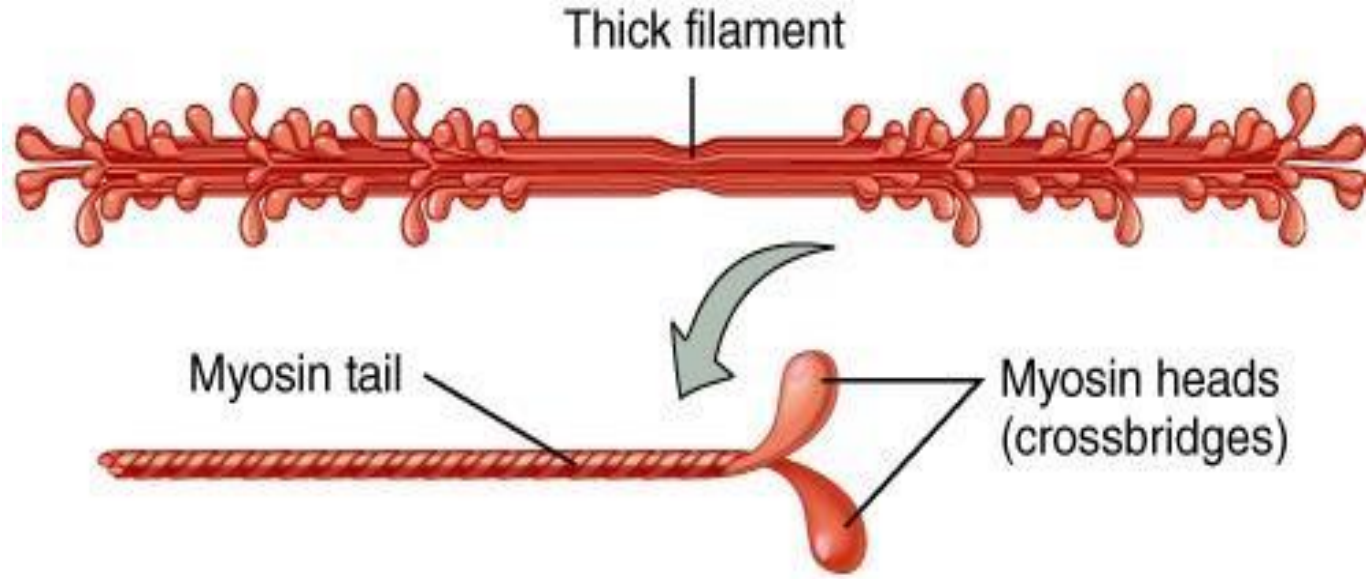


(a) One thick filament (above) and a myosin molecule (below)



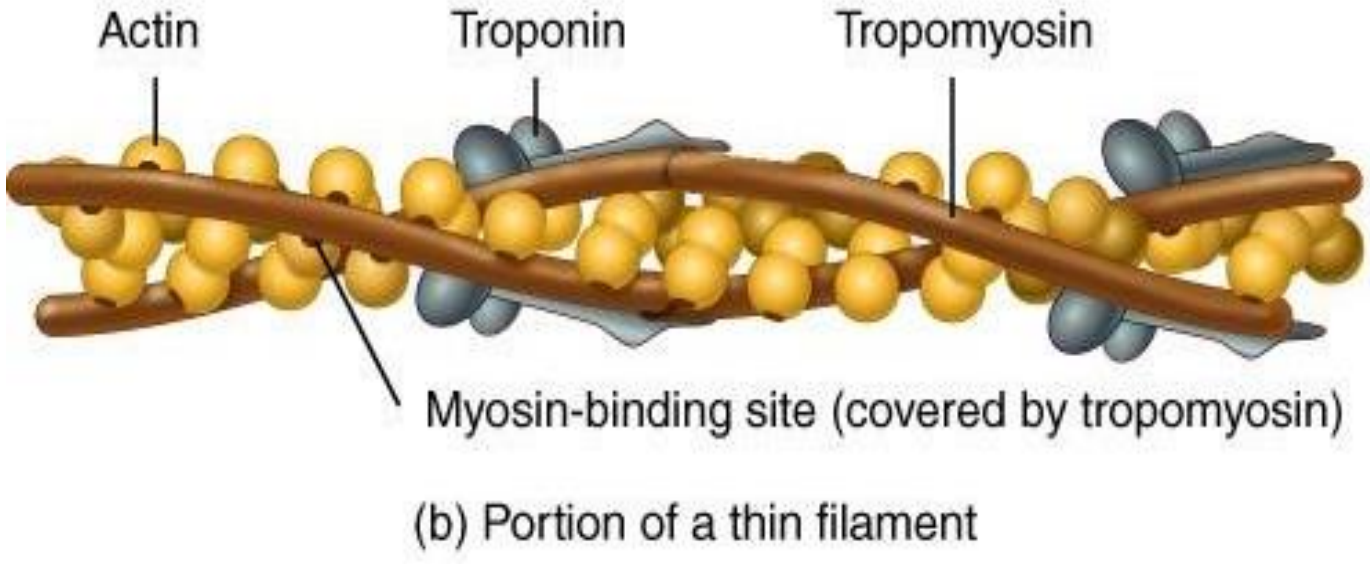
(b) Portion of a thin filament

10.06



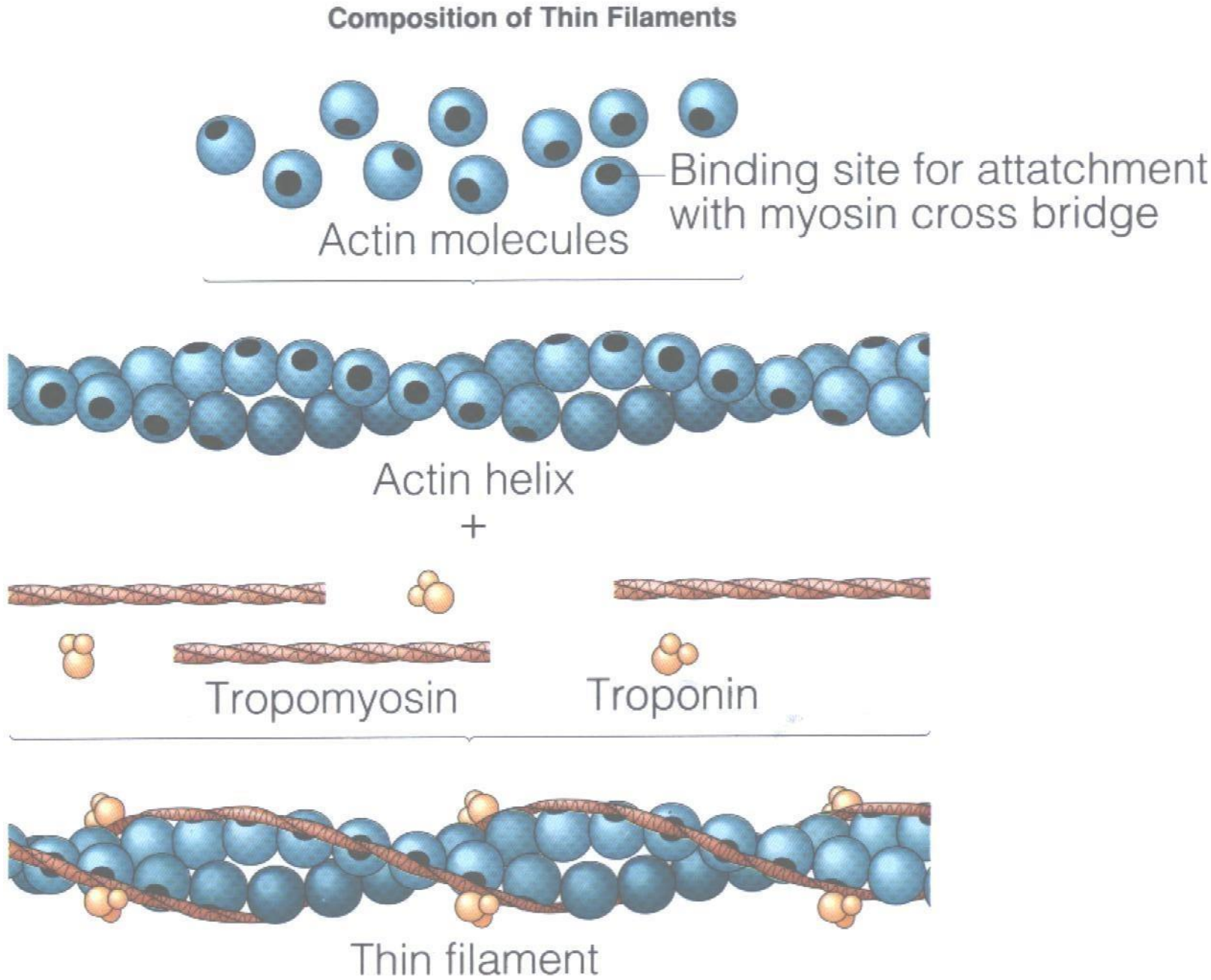
(a) One thick filament (above) and a myosin molecule (below)

the picture above represents thick filament with protein structure molecules called myosin, myosin has **1)** a tail, alpha helix structure, **2)** & two heads.
The tails form the backbone of the thick filaments while the heads are protruding outside forming cross bridges

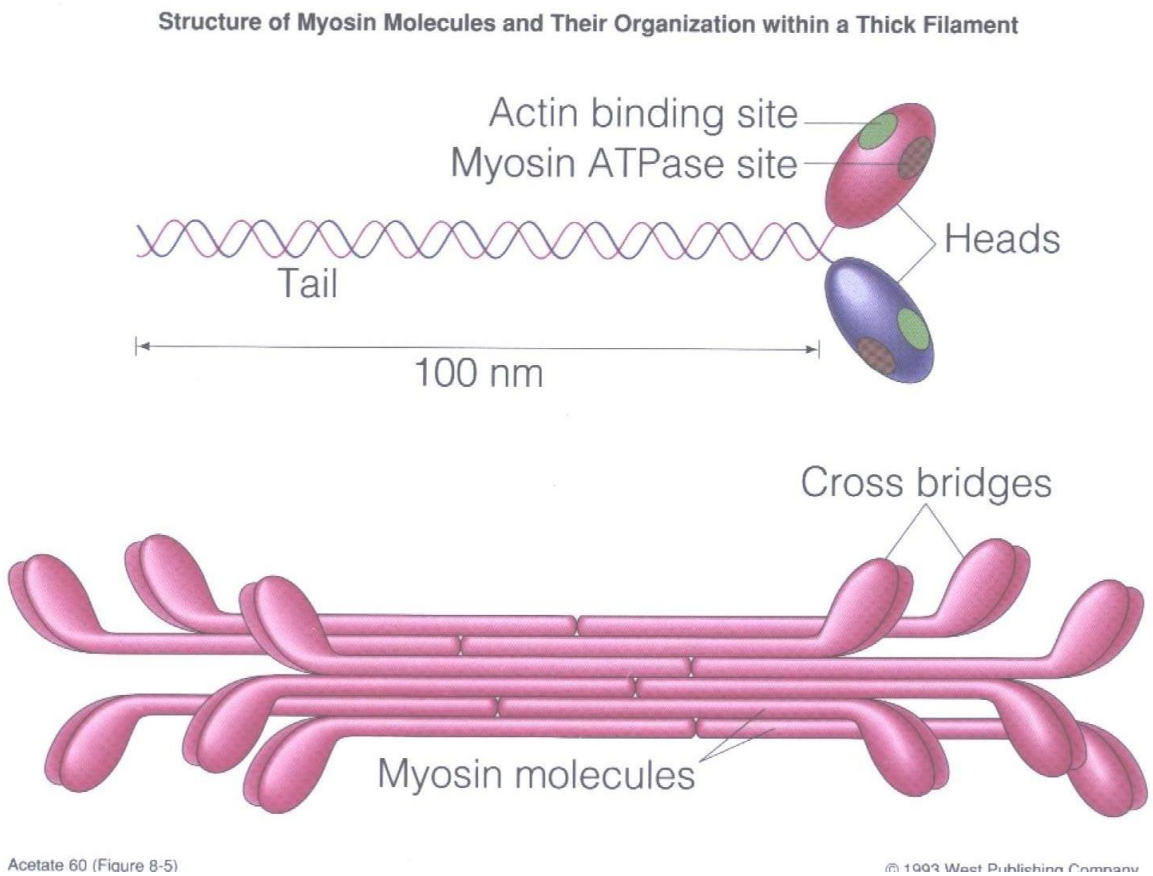


(b) Portion of a thin filament

The thin filaments are much thinner than thick filaments
The thin filaments are composed of actin molecules polymerized to form back-bone of thin filaments


Composition of Thin Filaments
Binding site for attatchment with myosin cross bridge
Actin molecules
Actin helix
+
Tropomyosin
Troponin
Thin filament

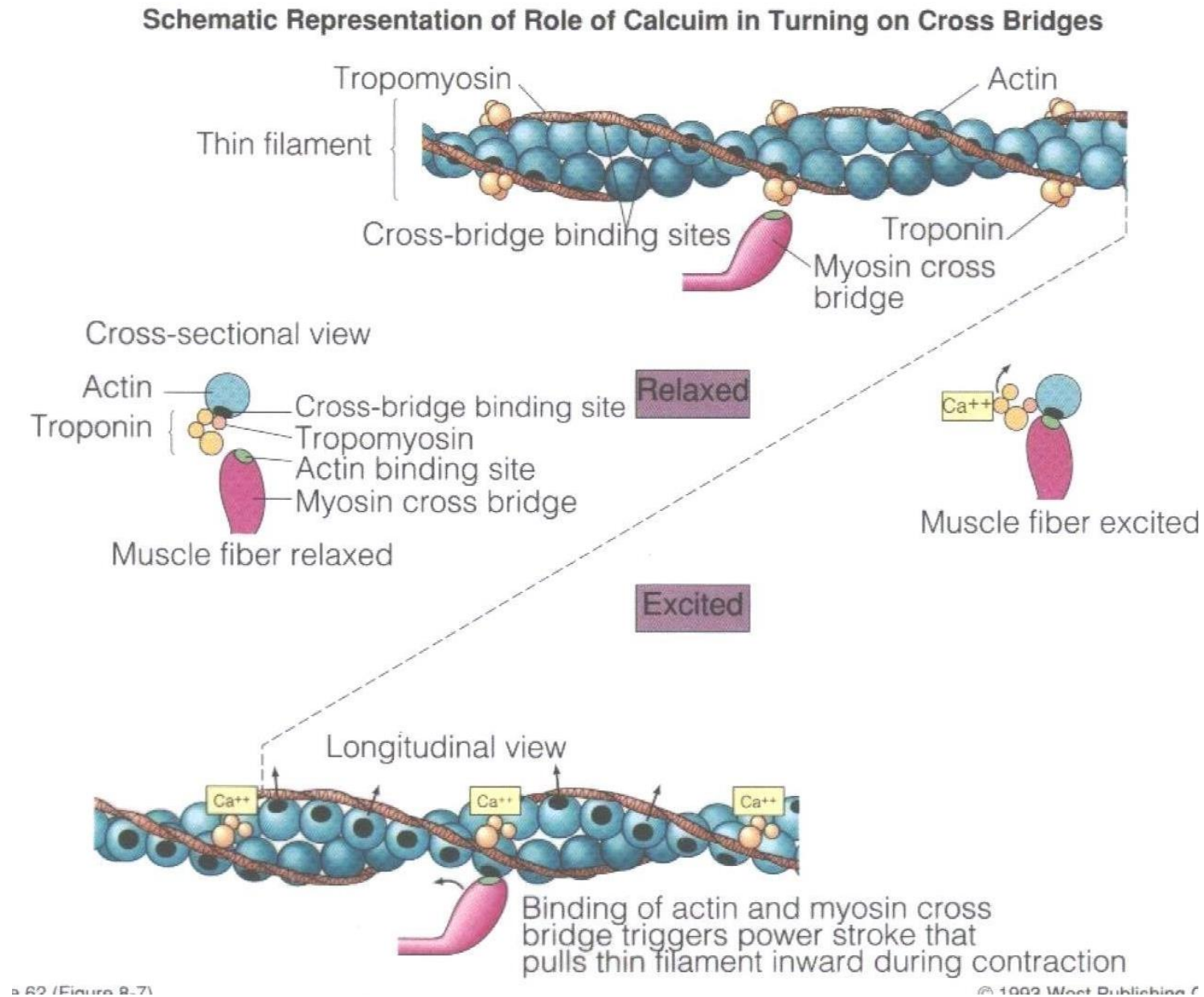
→Describing the image above in slide 16 you can see actin molecules having black dots representing the site of interaction with the heads of myosin forming cross bridges
→We also have tropomyosin which is covering all the sites of binding sites
→Another regulatory protein "troponin" there are three subunits forming troponin. One of the subunits is called "troponin C" Which is responsible for Calcium binding. The other subunit is called "troponin T" linked to tropomyosin. The last is called "troponin I" "intermediate" linking the other two subunits



Let's describe this image representing the heads of the myosin, there are two sites at the head:

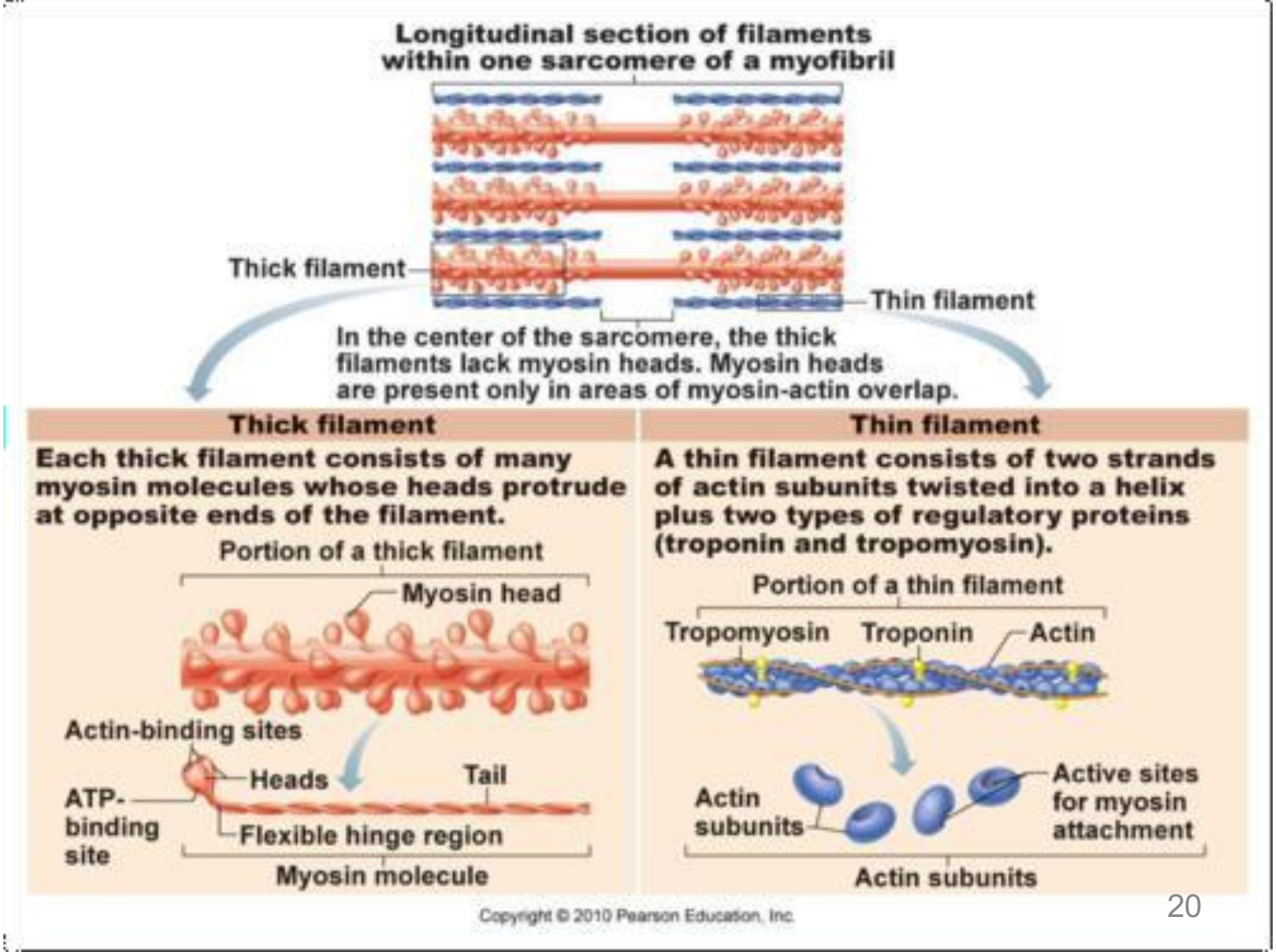
1.Actin binding site responsible for the interactions with the actin binding site

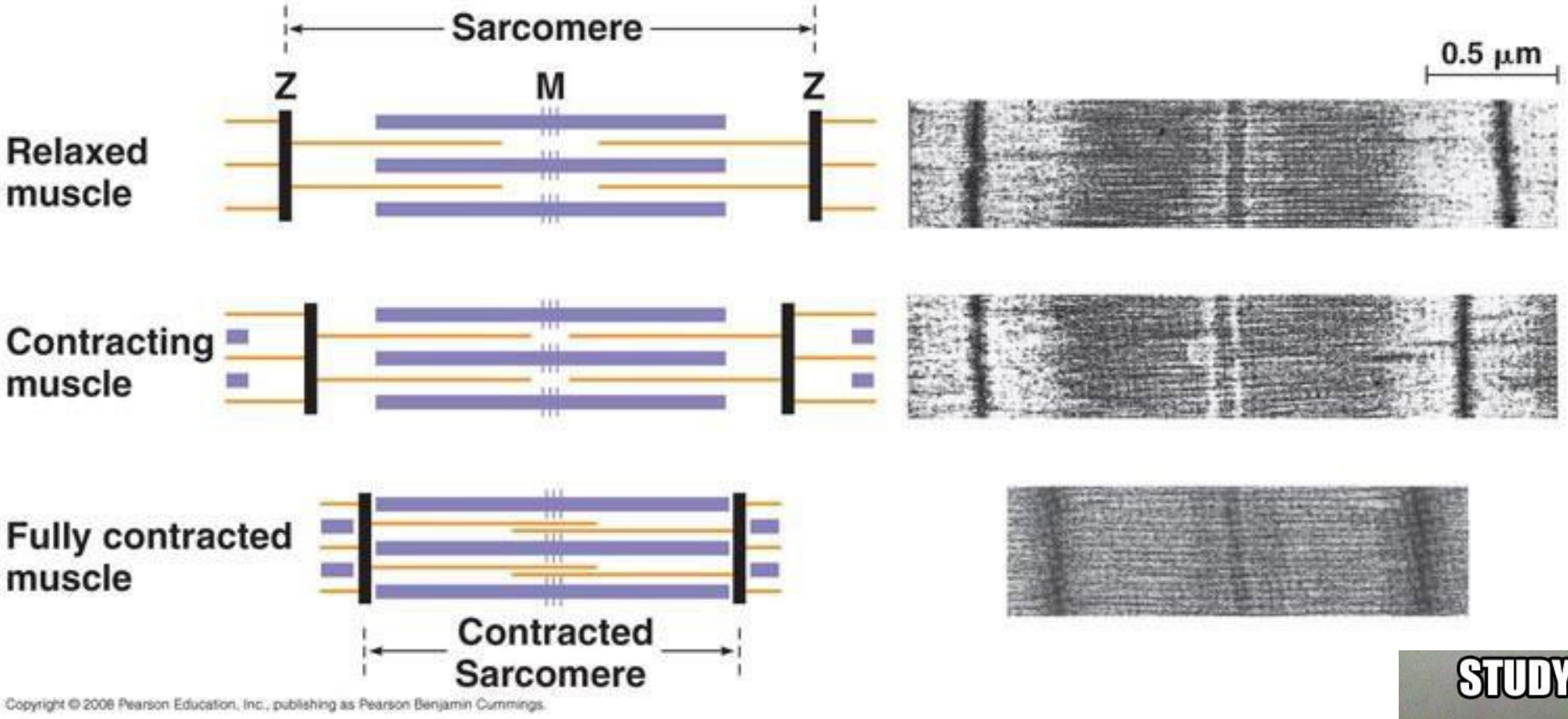
2.Myosin ATPase site responsible for energy regulation and phosphorylation of heads



To get this interaction we need Calcium, which will bind to "troponin C" causing conformational change leading to exposure resulting in interaction between acting binding site on myosin head and binding sited on actin filament.

Images showing ultrastructure of thin and thick filaments





After the interaction you **may** get shortening of the sarcomere leading to the changes we have discussed above (shortening of "H" zone, no change in the "A" band, change in the "I" band because of sliding) check the image in the following slide

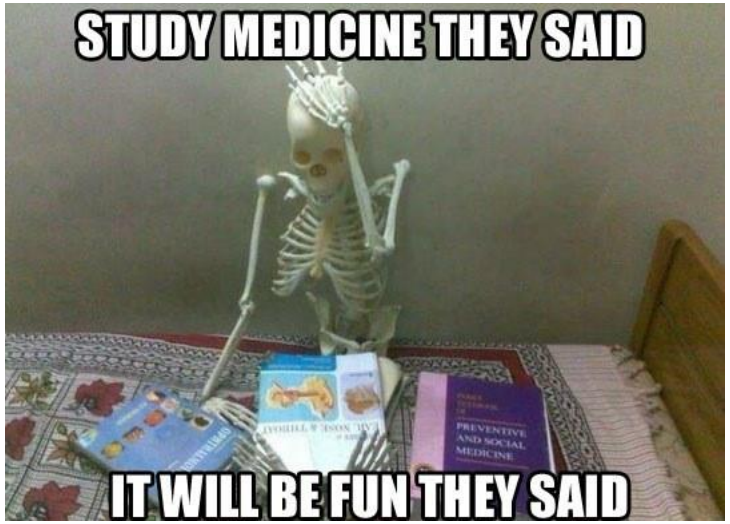
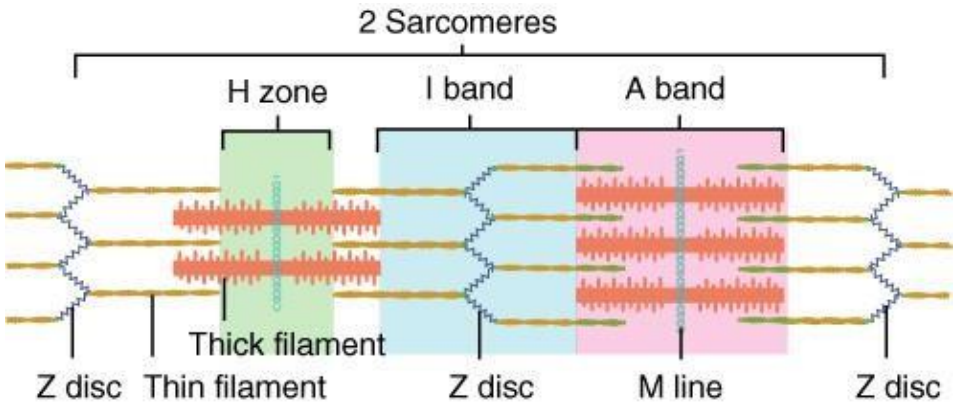
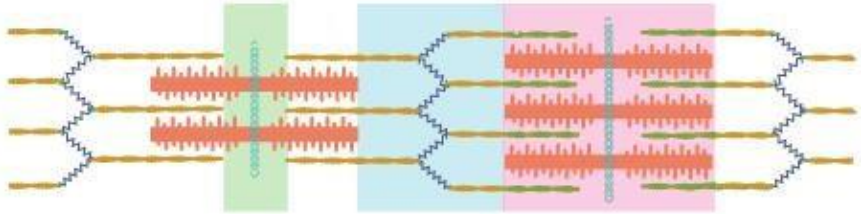


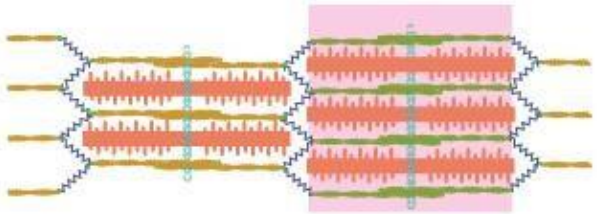
Fig. 10.07



(a) Relaxed muscle



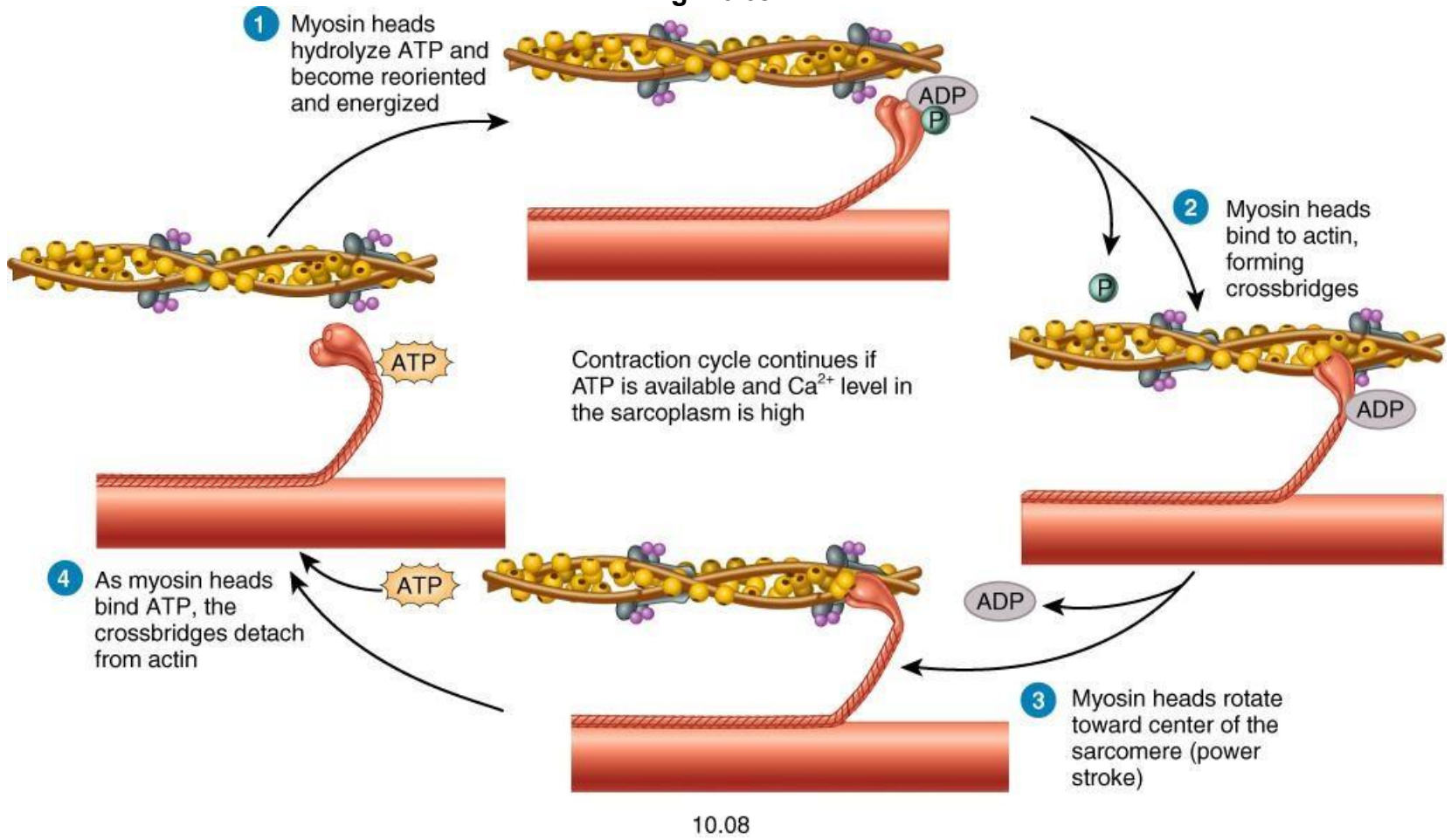
(b) Partially contracted muscle



(c) Maximally contracted muscle

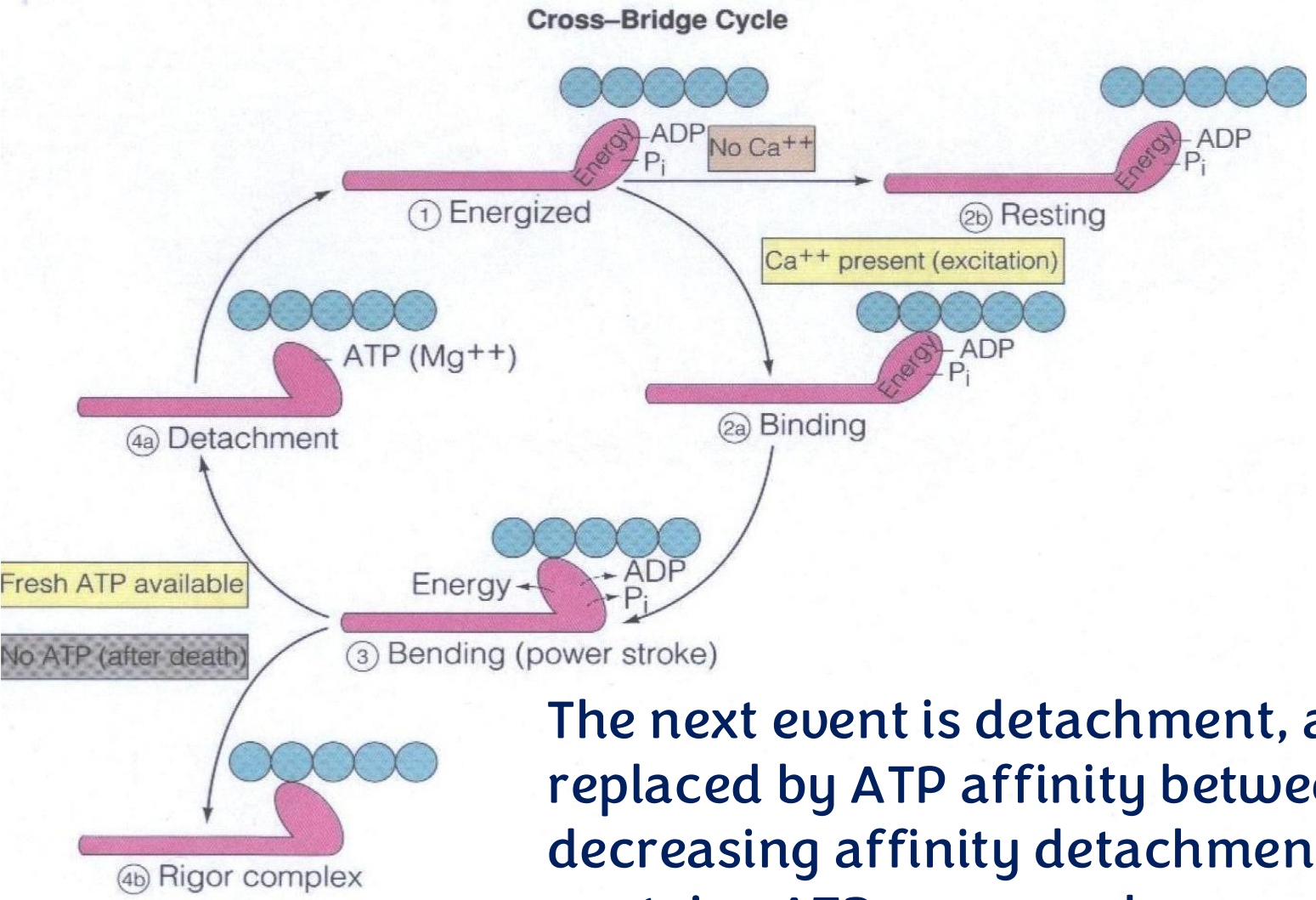
10.07

Fig. 10.08



10.08

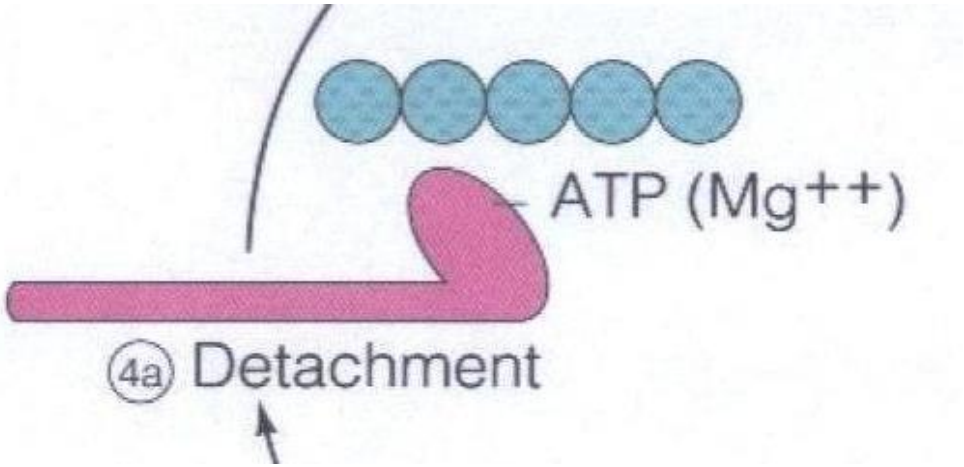
By the interaction (docking) of the two binding sites we are getting bending. Represented as well in the image below



The first event happening after the increase of Calcium concentration we have binding
Followed by bending of the head toward the center of sarcomere, also called "power stroke".

The next event is detachment, at first you have ADP, once ADP is replaced by ATP affinity between the two sides decreases, by decreasing affinity detachment occurs. The head of myosin contains ATPase as we know so it hydrolyzes ATP to ADP **energizing** the head again, if Ca is present the myosin binds again and power stroke can occur again.
If no ATP, it can't occur again and remains stuck bound to actin.

Explanation: If myosin head does not have the ATPase enzyme ATP won't be hydrolyzed into ADP as a result it will remain stuck in the same position (because of lower affinity) and won't be able to become in its energized form again; ATP binds to myosin so that detachment from actin occurs, however ATP isn't hydrolyzed to ADP+Pi without hydrolysis, no energy is released, therefore myosin head won't enter the energized position, so it won't be able to perform another "power stroke" again and unable to cycle.
We have discussed lack of ATPase now let's talk about lack of ATP; no ATP to replace ADP and to phosphorylate the head so again it remains stuck at that position. This phenomenon is called "Rigor Mortis" which happens two hours after death, when ATP is depleted in the muscle stiffness occurs, they can define time of death through measuring the stiffness.
-To summarize the steps of interaction:
1.Binding of active sites
2.Bending of head (automatic process)
3.Detachment (to get detachment we need ATP)
4.Hydrolysis of ATP to ADP by ATPase to get energized form again & continue cycle, Ca is needed to bind the sites again and form interaction (power stroke)



What is the importance of magnesium here?
It's needed for affinity of the ATPase
What happens if a patient has hypomagnesium? He will have muscle stiffness, contractile problems.

Summary of Muscle Contraction

https://www.youtube.com/watch?v=6YvdLWgT5mg

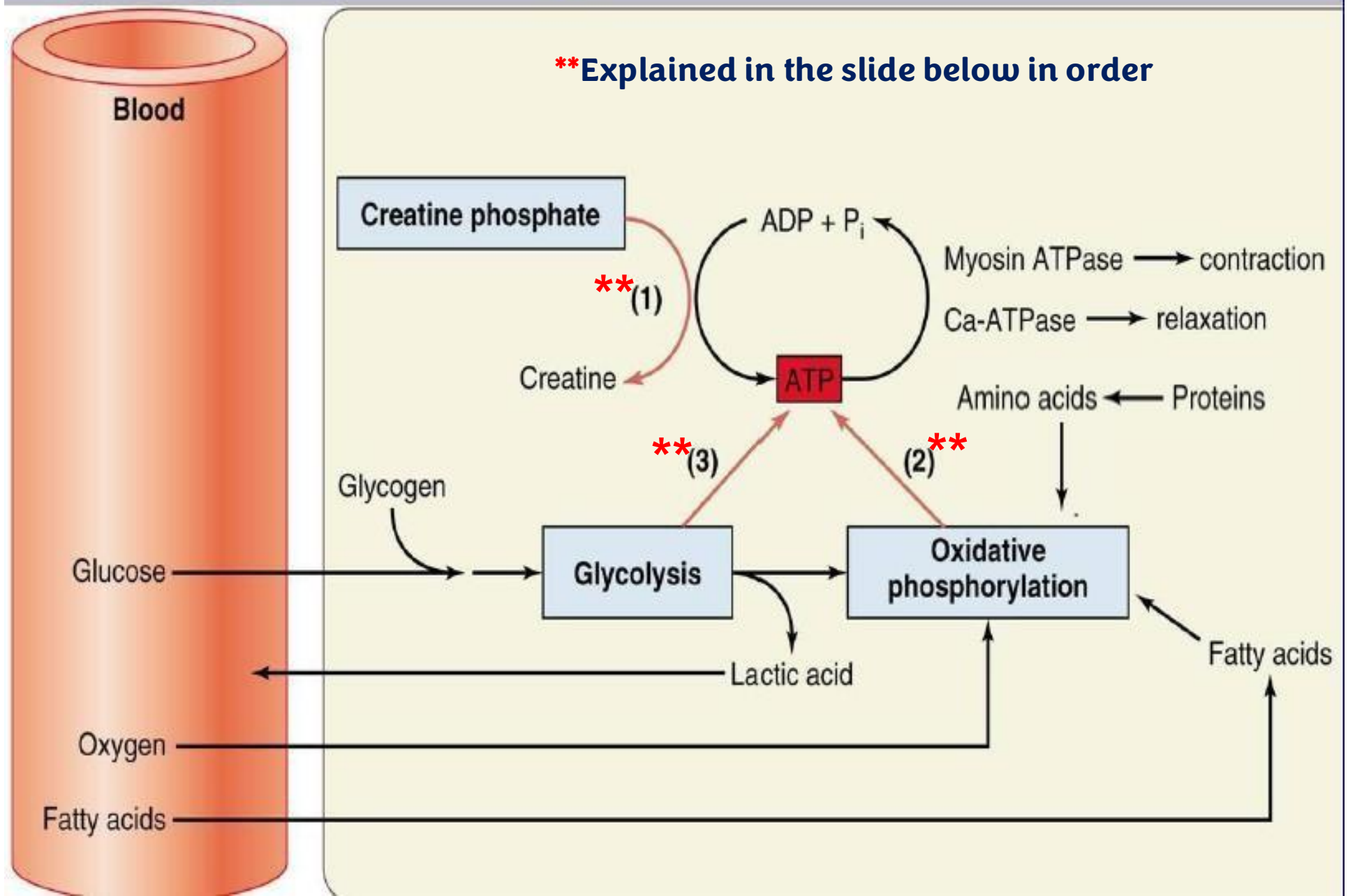
The doctor said that this link has a summary for muscle physiology it has a lecture for muscle physiology, but it's an hour long :) use this one if the one in purple doesn't work

https://www.youtube.com/watch?v=6YvdLWgT5mg

Muscle Energy

From where are we getting the ATP required for contraction?

Sources of energy for muscle contraction



There are reservoirs of ATP in muscles which are enough for a few seconds, almost 20-30 seconds, we can consume all the ATP in the muscle
So if you have consumed all the ATP will it not work? No, we have another (1)immediate source --> Creatine Phosphate which can be enough for "minutes“,,we have enzyme that transfer phosphate from creatine phosphate to an ADP to get regeneration of ATP
If we consumed all reservoires and Creatinine Phosphate will the muscle stop working? No, we still have other sources --> (2)Process of glycolysis which if you remember (ofc you do) we took in metabolism it takes place in cytoplasm generating little amount of ATP (3) but luckily! We have the OxPhos process taking place in mitochondria
What are we consuming in OxPhos? Oxygen, huge amounts
Do we store oxygen in the muscle? Yes, in Myoglobin similar to hemoglobin in blood.
For more activity we are requiring more oxygen to compensate for the "oxygen debt".

The color of myoglobin is red that's why muscles are colored Red. But we do have muscles that are white, why? White muscles do have myoglobin as well but in fewer amounts.
What are the differences between fast muscles & slow muscles?

Fast twitch muscles	Slow twitch muscles
White/pale	Red/dark
Depend on fast process to get energy	Depend on slow process to get energy
Myoglobin content lower	Myoglobin content higher

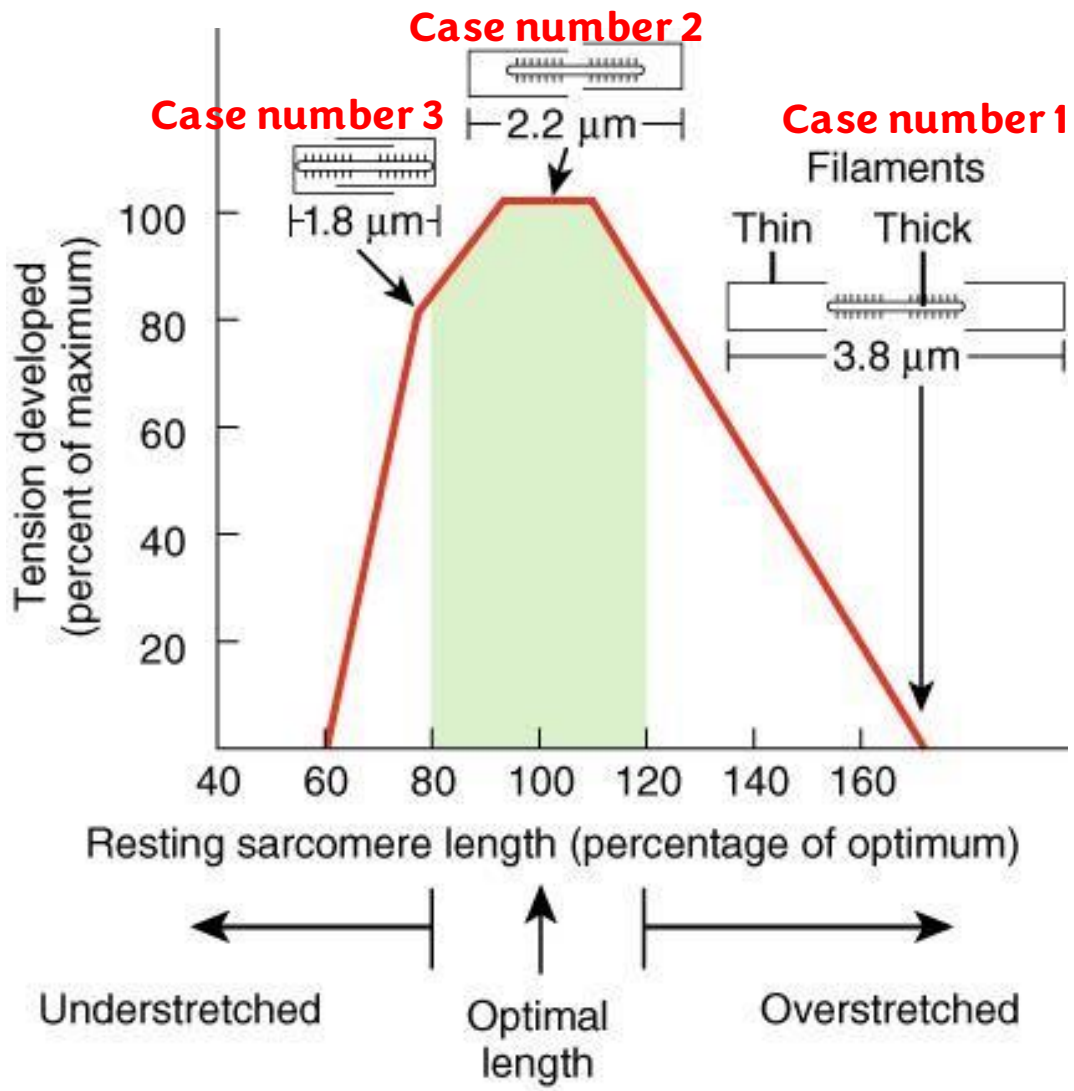
→Some sports require slow fibers (ex: marathon race) and other types of sports require fast fibers (ex: racing for 100m swimming)

→If we get someone who has higher number of fast twitch muscles he won't do good in sports requiring slow twitch and vise versa .

→How to get higher number of slow fibers in muscles?
Training since childhood, same goes for fast twitch. So proportion of fast twitch to slow twitch depends on person's activity since childhood and can change by training.

Muscle Mechanics

Fig. 10.10



10.10

Remember that myofibril is found inside myofiber. **Case number 1:** Here we take a myofibril and stretch it until we get NO overlap between thin & thick filaments, and then we add some Calcium, do u expect interaction between the thick & thin filaments? No, there will be no interaction because we have stretched and fixed the heads **so** no shortening will occur in this case no interaction.

Case number 2: If you stretched to a **lower** length but this time there is overlap between thick and thin filaments but **the heads are fixed that means no shortening will be** , then when we add Ca we get interaction between thin & thick filaments. By this interaction we can record **tension** developed by the interaction between the thin & thick filaments.

As a result ,we record whether we are having interaction or not,,, remember: No shortening occurs (cuz the heads are fixed). If a muscle is contracting without shortening we call its method of contracting **isometric contraction** (no change in length)

Can muscles in our body contract isometrically? Yes, simply no change in length and it's stimulated to contract, the only thing changing is the tension in that muscle, which is recorded by special transducer placed inside the muscle at the level of the myofibrils then the tension can be recorded.

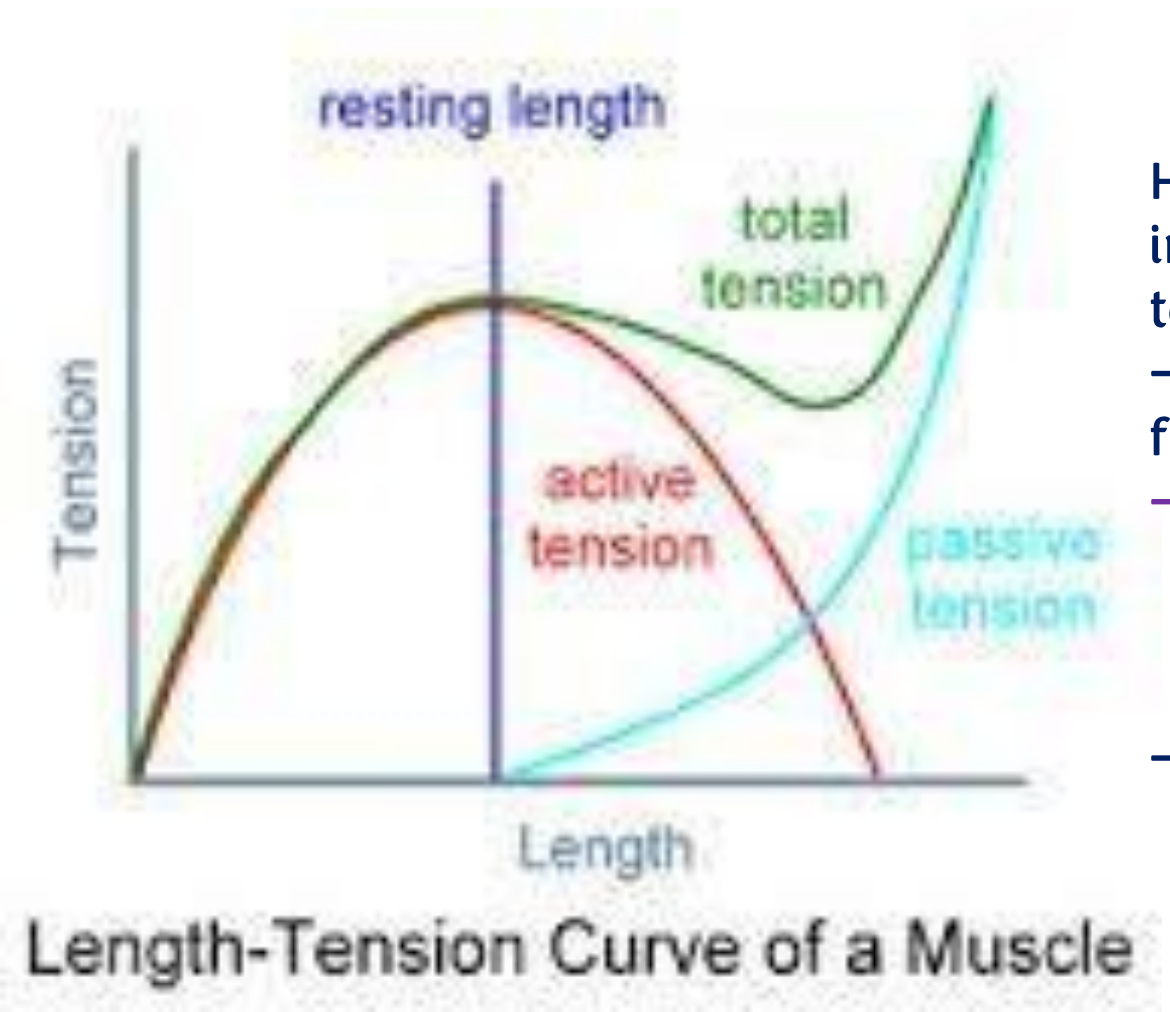
→ The more the overlap the more the tension.

Let's discuss more cases

Taking the fiber out without any stretch and fixing the head, and stimulating the muscle to contract we get the maximal tension that could be developed at 2.2microM (check the graph on slide 33) maximal tension is achieved by maximal interaction between thin & thick filaments at the resting length. Remember that the relation between tension and interaction between thin & thick is direct.

Case number 3: If we have shortening first then fixing the head and stimulating contraction the tension is less, represented by 1.8 microM on slide 33. Why less? Because we have some part of thin filament overlapping and the other half of thick filament, and each is pulling in opposite directions, this gets us to lower tension developed.

Summary: we have relationship between the sacromere and the tension that can be developed,,, and the maximal tension is at the resting level.

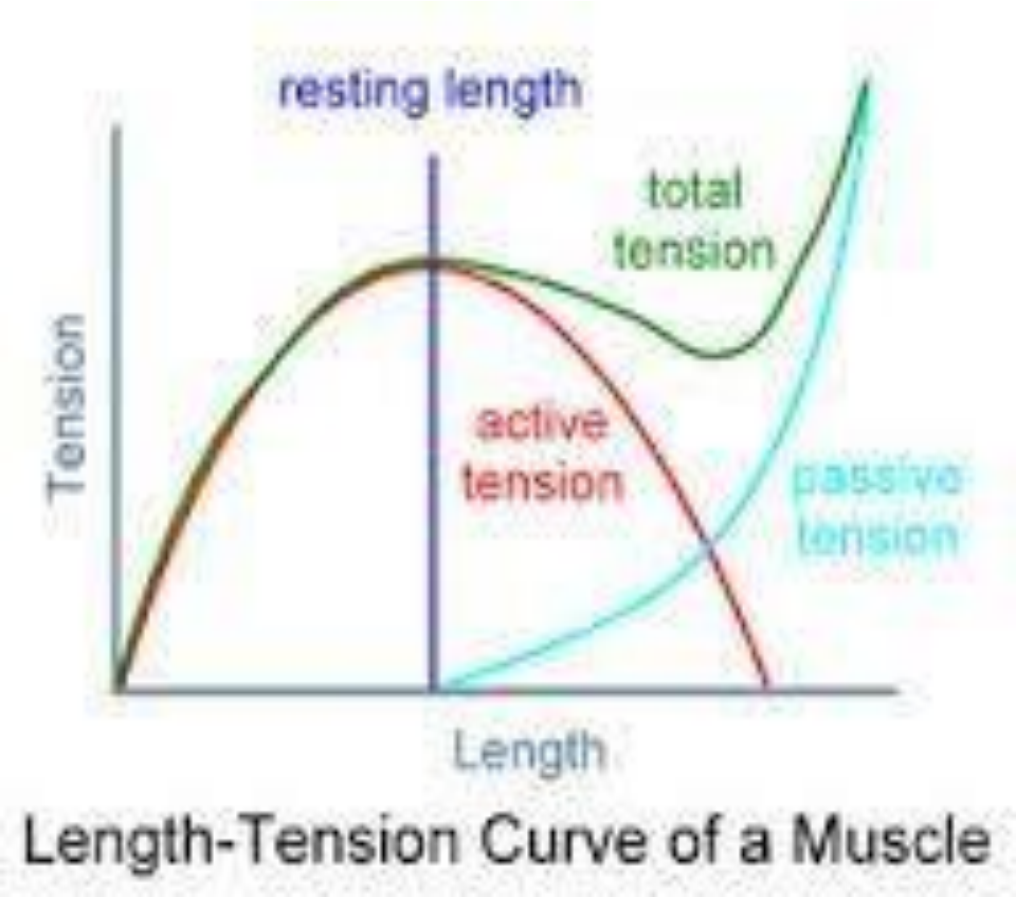


Length-Tension Curve of a Muscle

More stretch less overlap & less tension —> indirect relation (this is for active tension only not total)

Here we are measuring the tension of a whole muscle instead of a myofibril. So we are developing different tension by stretching

- when we fix the head we get the red line seen on the following graph.
- when we stretch the muscle we are creating another tension in the muscle represented on the blue line called passive tension. Explanation about passive tension → click here.
- But What happens to active tension by stimulation(when we stretch the muscle)? Decreases because of less overlap between thin & thick. **More stretch less overlap and tension** (seen on red line) while passive increases, muscles have elastic properties, imaging stretching elastic rubber you develop tension, hence the tissue is elastic.
- If we do shortening of muscle & then fixing the heads, there will be more overlap so less tension.



Length-Tension Curve of a Muscle

Isometric → same length but the tension is changing
Isotonic →changing the length without changing the tension

Where do we have the highest active tension?
At the resting length of the muscle (the vertical blue line)
Sum of active and passive is the total tension (green line)
We can't record active tension during stretch, we can measure the total tension so what we do is record passive tension & record the total tension, we subtract passive from total & get active tension.

If heads are free→ we get shortening
if they're fixed →we are getting only the change in tension
Once you have shortening & both heads are free we have **isotonic contraction**
(don't confuse it with isometric contraction)

University of Jordan Faculty of Medicine
Department of Physiology & Biochemistry

Medical students,
****

Muscle physiology and the basis of contraction

Ref: Textbook of Medical Physiology, by Guyton, 13th and Jordan Edition: Chapts. 6, 7, 8,

Introduction:

Three types of muscle are found in our body. Skeletal, cardiac, and smooth muscle cells. These cells are found where mechanical activity is needed. Movements of the whole body or parts of it need contraction of skeletal muscles. Pumping of blood in vessels need contraction of cardiac muscle. Emptying the content of hollow organs requires contraction of smooth muscle in that particular organ.

Muscle cells have been classified according to their characteristics, first (according to their appearance under the microscope) in **striated** (cardiac and skeletal muscle) and **unstriated** (smooth muscle) fibers. Second, (according to their innervation): **voluntary** (have somatic innervation), an example: skeletal muscle, and **involuntary** (have autonomic innervation), example: cardiac and smooth muscle.

Structure of skeletal muscle:

One muscle is composed of many **muscle fibers** that are lying parallel to each other and bundled together by a connective tissue. The most dominant structure in muscle fibers is the presence of **myofibrils**. Each myofibril consists of a regular arrangement of cytoskeletal elements known as **thick and thin filaments.** Which give the striated appearance in skeletal muscle.

The special arrangement of thick and thin filaments in lighter and darker (I and A) bands, gives the striated appearance in skeletal muscle. The **I** band is formed only from thin filaments. While the **A** band is formed from thick filaments with the portion of thin filaments that

overlap on both ends on thick filaments. The area of thick filaments that is not overlapped by thin filaments is known as **H zone**.

In the middle of I band, there is a dense vertical structure (flattened disc-like structure that hold thin filaments) known as **Z disc**. The area between 2 Z discs is known as a **sarcomere**, which represents the functional unit in skeletal muscle contraction. In the A band, a similar system holds thick filaments known as **M line**.

The cross-sectional arrangement in the area where is an overlap between thin and thick filaments shows 6 thin filaments around one thick filament and 3 thick filaments around one thin filament.

The thick filament (1.6 μm length) is composed of several hundreds of **myosin** molecules that are held together in a specific arrangement. A myosin molecule is composed of 2 identical subunits. Each has a globular head that projects out to one end and a tail that is intertwined with the tail of the other molecule. Each myosin head has 2 binding sites. One can interact with thin filaments and the other is myosin ATP-ase site. The heads and the portions of the tail that are protruding from thick filaments are known as **cross bridges**.

Thin filament (1.0 μm length) is composed of three proteins, actin, tropomyosin and troponin. **F-Actin** helix forms the backbone of a double-stranded structure of the thin filaments. Each strand is formed of polymerized G-actin. On actin molecules, there is a site that can interact with myosin head (**myosin binding site**). It is believed that this site is an ADP molecule bound to G-actin. The bases are inserted to Z disc. The ends lie in the space between thick filaments.

Tropomyosin is protein molecules that wrap around the F-actin helix. In resting state, this protein covers the active site (myosin binding site) on actin and prevents interaction of actin with myosin head.

Troponin is a complex structure of 3 subunits, which plays a role in controlling muscle contraction. One subunit has affinity for actin (troponin I), the other has affinity for tropomyosin (troponin T), and the third has affinity for Ca++ (troponin C).

Interaction of thick and thin filaments to induce contraction, and the role of Ca++:

The myosin binding site on actin is the place where myosin heads bind to actin. In the absence of troponin –tropomyosin complex, myosin can bind strongly to actin in the presence of ATP and Mg++. When troponin-tropomyosin complex is added, the binding is inhibited. From

these it was suggested that in relaxed muscle, troponin-tropomyosin complex inhibits or physically covers the binding site on actin and prevents the interaction between myosin heads and actin.

In the presence of high Ca++ concentration, the inhibitory effect of tropomyosin-troponin complex on myosin and actin binding was inhibited (so, binding was induced). From this it was suggested that during muscle contraction Ca++ binds to troponin C (up to 4 Ca++ bind to one molecule of troponin C), this produce conformational changes that results in the displacement of tropomyosin away from the active sites on thin filaments. The uncovered active sites can interact with myosin and induce contraction in the muscle. This theory shows the relation between contractile and regulatory proteins (troponin and tropomyosin), and explains the role of Ca++ on muscle contraction.

During contraction, the two Z lines become closer. This results by pulling thin filaments inward toward the center of the sarcomere. This will result in a decrease in the H zone, I band and the whole sarcomere length. This happens after binding of myosin heads to the active site on actin. After this binding, myosin bends between the head and the arm of cross bridges, which pulling the thin filament toward the center of the sarcomere. Bending (tilting) of myosin head is known as *power stroke*. Then the head detach from the actin and bind to another active site on actin, which located closer to the base of the thin filament, and the cycle is repeated many times. The result of this mechanism is more overlap will be obtained between thick and thin filaments by pulling thin filaments inside. This theory is known as “sliding theory” or “walk-along” theory.

According to this theory, after many cycles of (binding, power stroke, detachment, then binding again) that are taking place between cross bridges and actin, a shortening of the sarcomere will be induced in the muscle by sliding thin filaments toward the sarcomere center.

https://www.youtube.com/watch?v=GneonFlcZG8

Requirement of energy for contraction:

We have mentioned that myosin head has an ATP-ase site. At this site, ATP binds, where it splits into ADP and Pi. This needs Mg++ to attach the ATP before ATP-ase can split the ATP molecule. This breakdown of ATP occurs before the head links to actin. The resulted ADP and Pi remain bound to myosin (becomes phosphorylated), and the generated energy from splitting is stored within the cross bridge. During

relaxation of the muscle, the head is energized. When the muscle fiber is excited, the increase in Ca++ concentration in the sarcoplasm, pulls tropmyosin-troponin complex out of their blocking position. This will enable myosin head to attach to actin. When attached, the myosin head undergoes conformational changes and bends. After this power stroke, the head releases ADP and the site is dephosphorylated. At this point, the detachment of myosin head will take place ONLY when another ATP molecule binds to myosin head. After detachment the new molecule is cleaved, the head returns to its position and energized by splitting ATP. The cycle continues as long as we have high Ca++ concentration inside the sarcoplasm (cytosol) to keep active sites on actin ready for interaction with myosin.

ATP is necessary for the detachment of cross bridges from actin. Not enough ATP will cause muscle to stiff because of the inability cross bridges to detach from actin. This phenomenon is called **rigor mortis** (a stiffness of skeletal muscle after 3-4 hours of death).

Source of energy for muscle contraction:

During muscle activity, ATP is needed to provide energy for the power stroke. In addition to that, Ca++ is pumped into the sarcoplasmic reticulum by the activity of the Ca++ pump. This pump needs ATP for its operation. Pumping of Na+ and K+ through sarcolema maintains the ionic composition of cytosol and permits optimal activity of muscle cells. All these activities need a direct use of ATP. In muscle, the amount of ATP is sufficient for only few seconds.

3 ways by which muscle cells supply additional ATP as needed:

1. Transfer of high energy phosphate from **creatine phosphate** to ADP:
   Creatine phosphate contains a high-energy phosphate bond. This bond can be transferred to an ADP molecule to form an ATP by the activity of an enzyme known as creatine kinase. The amount of creatine phosphate in muscle is 5 times that of ATP. For that, the muscle needs more efficient supply for longer activities of muscle.

2. Oxidative phosphorylation: This takes place in the muscle when a sufficient supply of O2 is present. This pathway provides a rich supply of ATP (from one glucose molecule processed by oxidative phosphorylation, 36 ATP molecules are yielded). This source is slow and needs constant supply of

O2. This way can be sufficient for ATP supply when there is a moderate demand for ATP, such as during light and moderate exercise (walking, jogging, or swimming).

3. Glycolysis: high amount of glycogen are stored in muscle cells. The breakdown of glycogen to glucose, which can be broken down by glycolysis into two pyruvic acid molecules to yield 2 ATP molecules. Pyruvic acid can undergo further degradation by oxidative phosphorylation. Glycolytic pathway is much faster than oxidative phosphorylation in generating ATP molecules. And it is operating anaerobically (there is no need for O2). On this process, fast muscles are depending on for energy supply.

Although it is very useful during intense exercise when the O2 supply is reduced, but it can lead to a muscle **fatigue** because of accumulation of lactic acid in muscle which results in inhibition of enzymes (involved in energy-producing pathways or excitation- contraction coupling) and depletion of energy reserves.

Muscle mechanics:

We have seen that muscle contraction induces shortening in the sarcomere, which results by pulling thin filaments toward the center of the sarcomere. This contraction is seen in the whole muscle as a change in length. When a muscle contracts by changing its length without changing its tension, the contraction is said to be *isotonic*. If a muscle develops tension without changing its length, the contraction is said to be *isometric* (which can be recorded by using an electronic force transducer to measure tension).

Tension and sarcomere length relation:

The tension that can develop on muscle depends on the length of the sarcomere and the length of the muscle. When sarcomere length is more than 3.6 μm (length of one thick filaments and 2 thin filaments), the tension that can develop is almost zero. When the sarcomere length decreases, the tension increases as the overlap increases and cross bridges that can be recruited for muscle contraction increases. This increase reaches a maximum, after which more overlap will reduce developed tension. The *maximum tension* that can develop is at the sarcomere length of 2.0-2.2 μm (this known as optimal length). Below this length (from

2.0-1.6 μm) an interaction between thin filaments and cross bridges from the other half of the sarcomere is suggested, which may result in a decrease in tension.

From this, we can conclude that more overlap between thin and thick filaments located in the same half of sarcomere will induce more tension. This tension is reduced by decreasing the overlap in the same side, or increase in the interaction of thin filaments with cross bridges from the other side of thick filaments (increasing overlap with the other side).

Tension and whole muscle length relation:

We have seen that maximum tension develops at a sarcomere length of 2.0 –2.2 μm. This corresponds with the resting length of the muscle. At its normal length, the muscle also responded with the maximum *active tension* (tension induced by stimulation). By stretching muscle (increasing its length), before stimulation we increase the inactive (passive) tension (due to elastic property) in the muscle. When the muscle stimulated at this new length will develop less active tension. That corresponds to the increase in sarcomere length beyond 2.2μm.

Velocity of contraction and load:

Skeletal muscle contracts with maximum velocity when it is not loaded. By loading the muscle, the velocity of contraction decreases as the load increases.

Muscle twitches and characteristics:

Once a nerve of a nerve and muscle preparation is electrically stimulated, the muscle will respond by a contraction then followed by relaxation. The whole recordings from the beginning of stimulation until the end of muscle relaxation is known as *simple muscle twitch*. The simple muscle twitch can take less time in muscles composed of fast fibers such as ocular muscle, or longer time in muscles composed of slow fibers such a soleus muscle. These muscles not only differ in their speed of contraction but also in their color and composition. Fast fibers are large fibers, have extensive sarcoplasmic reticulum, contain large amount of glycolytic enzymes, and fewer mitochondria. These fibers also have less extensive blood supply. Slow fibers are smaller, have more extensive blood supply, and contain more mitochondria. These fibers also contain a larger amount of myoglobin (an iron containing molecule similar to

رسالة من الفريق العلمي:

I hope you enjoyed :)
(I didn't)

أن يصبح همك إصلاح نفسك ، والصلاة في وقتها
وذكر الله كل لحظة ، واستشعار معنى وجودك في هذه
الحياة ، وإمساك لسانك عن كل ما لا يعنيك ولا فائدة
منه ، أن تُطهر لسانك من كل ذنب يقترفه واعتداء
أن تعيش وتأنس بالقرب من الله والبحث عن ما يرضيه
أن لا يهمك إلا رضا الله ثم قربك من عائلتك

X:@Pii3i_

لحظة تفكُّرية في قوله تعالى

﴿ وَأَن لَّيْسَ لِلْإِنسَانِ إِلَّا مَا سَعَىٰ ﴾

فالإنسان لا يملك إلا سعيه ، لا سعي غيره !
اسعى ولا تفكِّر في النتيجة ، تيقّن بأن لا شيء يضيع
عند ربك

﴿ وَأَنَّ سَعْيَهُ سَوْفَ يُرَى ﴾

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