EXCITATION –CONTRACTION COUPLING.

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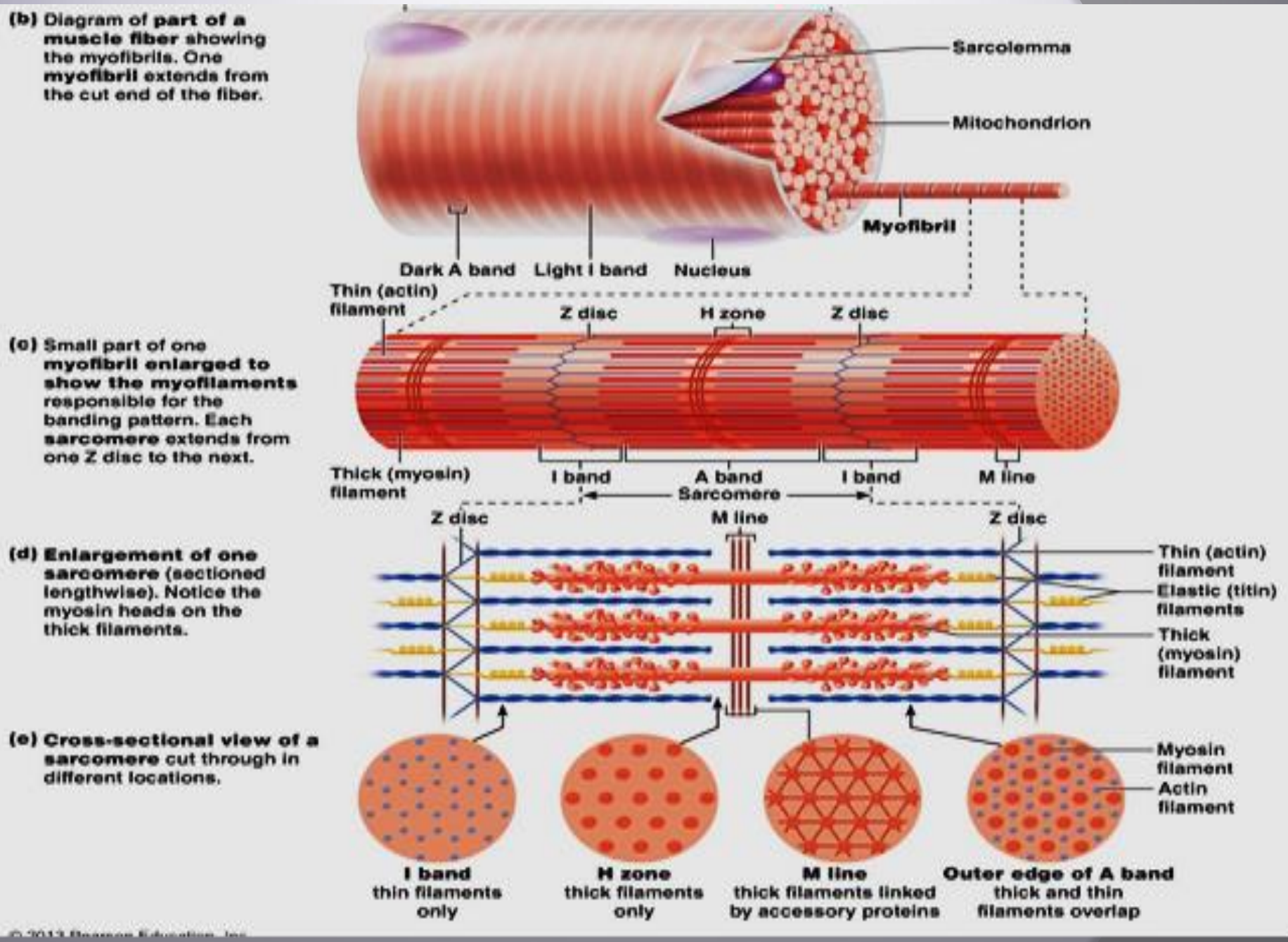


Muscle proteins

[A] Contractile proteins:

1- Myosin: -Myosin is complex protein with M.W. 480,000.

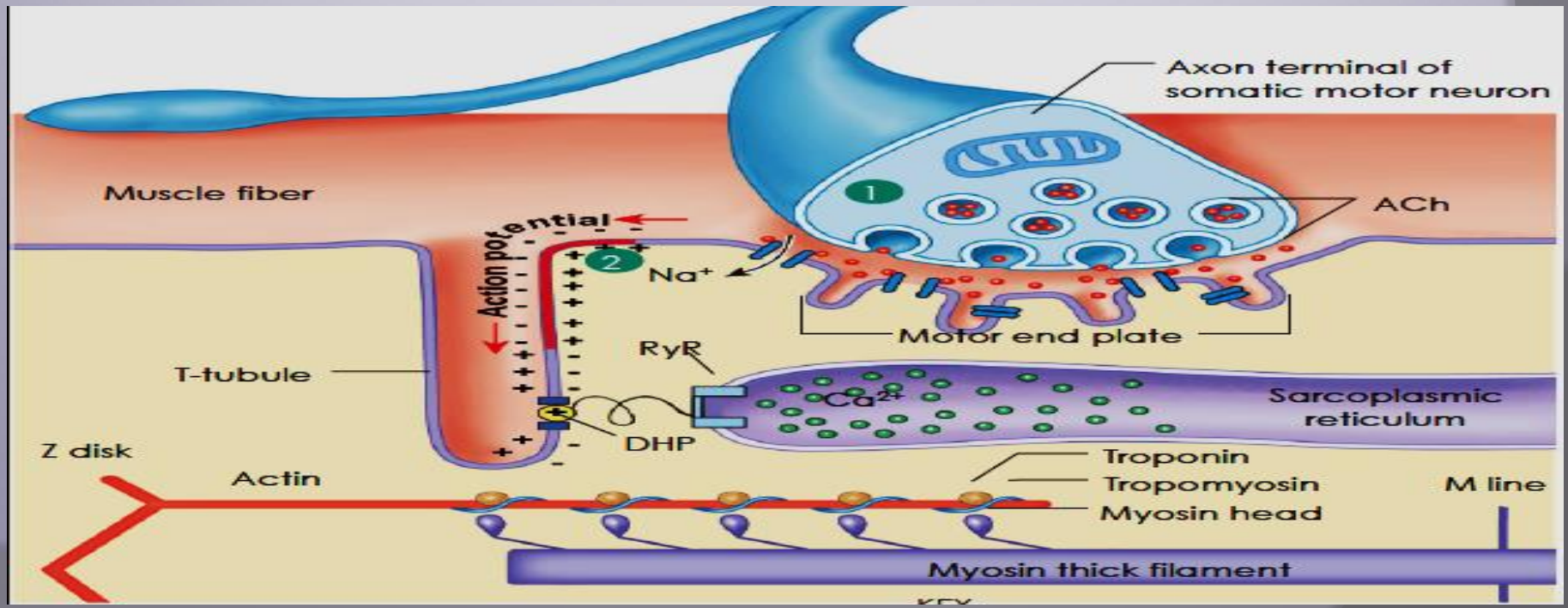
-Composed of **6** polypeptide chains (**2** heavy chains and **4** light chains).

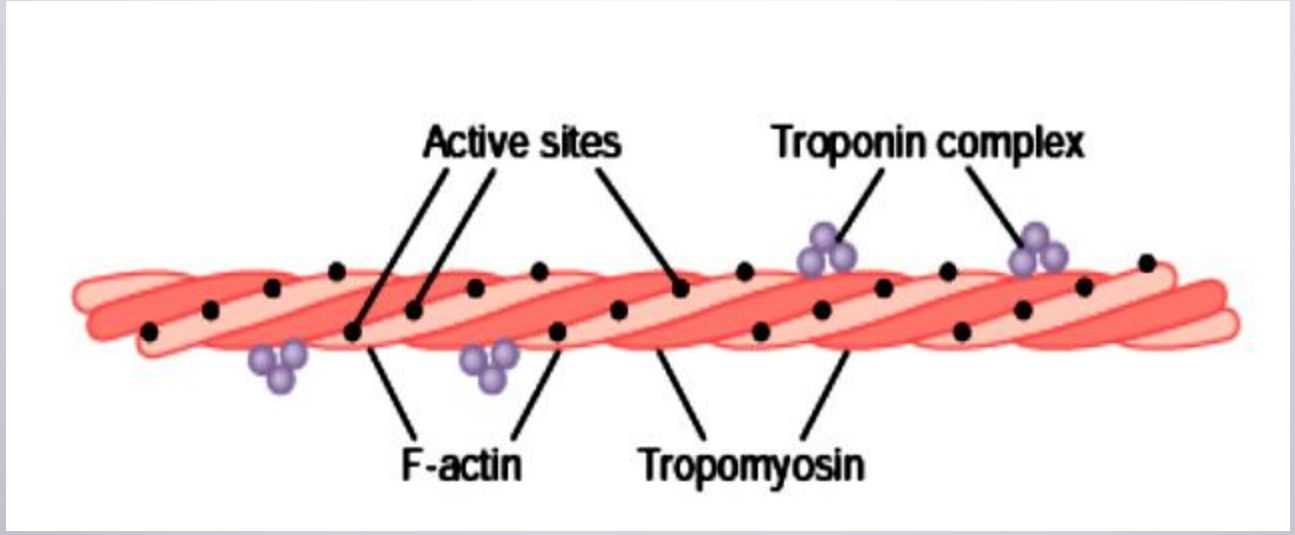
-The 2 heavy chains wrap spirally around each other as double helix forming long tail (light meromyosin) and arm (heavy meromyosin) while the terminal part combine with the 4 light chains forming 2 globular heads ,one head contains actin-binding sites and the other contain sites of ATP hydrolysis.

-Cross bridges arise from the head with arm of 2 flexible points called hinges (one between arm and tail and the other between the arm and heads) to bind to the actin.

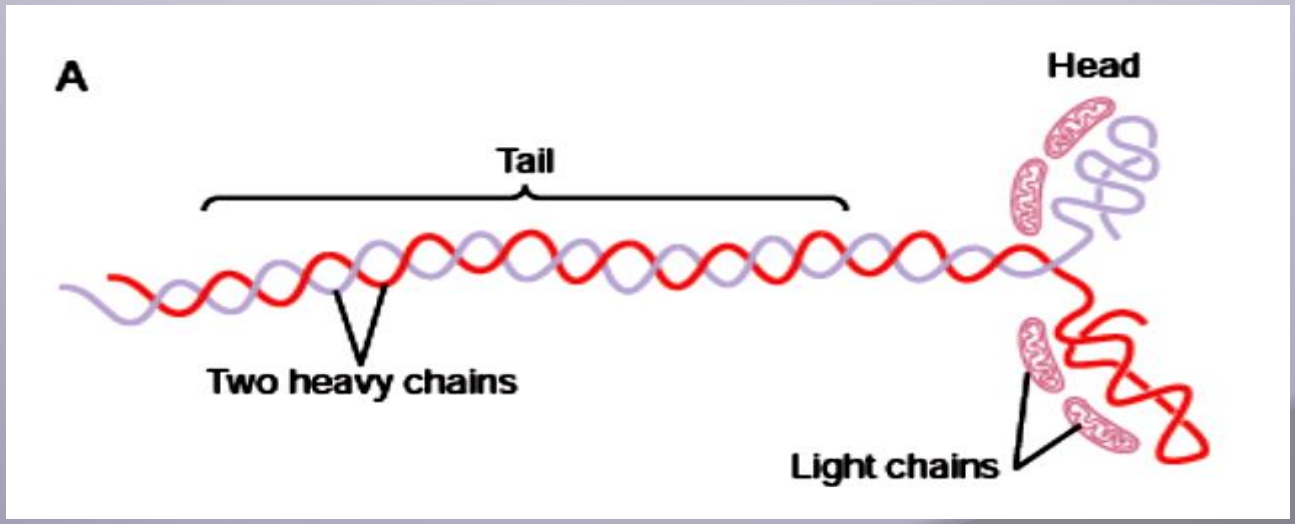
2- Actin: - It is small **globular** protein with M.W. 42,000.

- The globules attached to each other to form filamentous structure arranged in two chains as long double helix.





Actin Filaments



Myosin Filaments

[B] Regulatory protein:

1- Tropomyosin:

-It is long filament of two polypeptide chains twisting on each other and located between the 2 chains of actin covering its active sites which combine to myosin and keeps the actin structure.

2- Troponin:

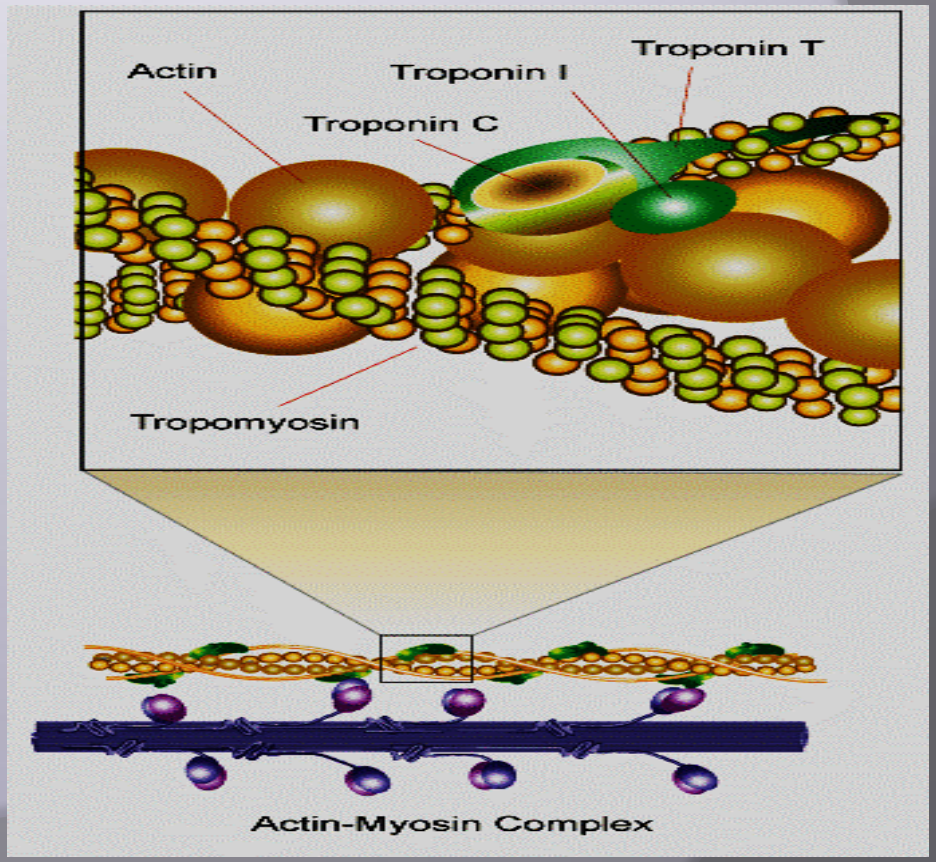
-Small globules located at intervals along tropomyosin.

-Of 3 subunits with MW 18,000-25,000.

1-Troponin T: binds troponin to tropomyosin.

2-Troponin I: inhibit binding of actin & myosin.

3-Troponin C: bind Ca+2 ions → contraction.



Mechanism of muscle contraction (Excitation - contraction coupling)

It is the process by which depolarization of the muscle fiber initiate contraction.

1-When a nerve impulse reach the MEP, it leads to Ach release from the nerve terminals.

2-Ach combines with the **cholinergic receptors** on the muscle membrane →↑ Na+ permeability → depolarization of the membrane (End plate potential).

3-When the **EPP** reaches the firing level → action potential that propagates along the muscle membrane and transmitted to all fibers via the T tubules.

4-This action potential triggers the release of Ca++ ions from the terminal cisternae of sarcoplasmic R.

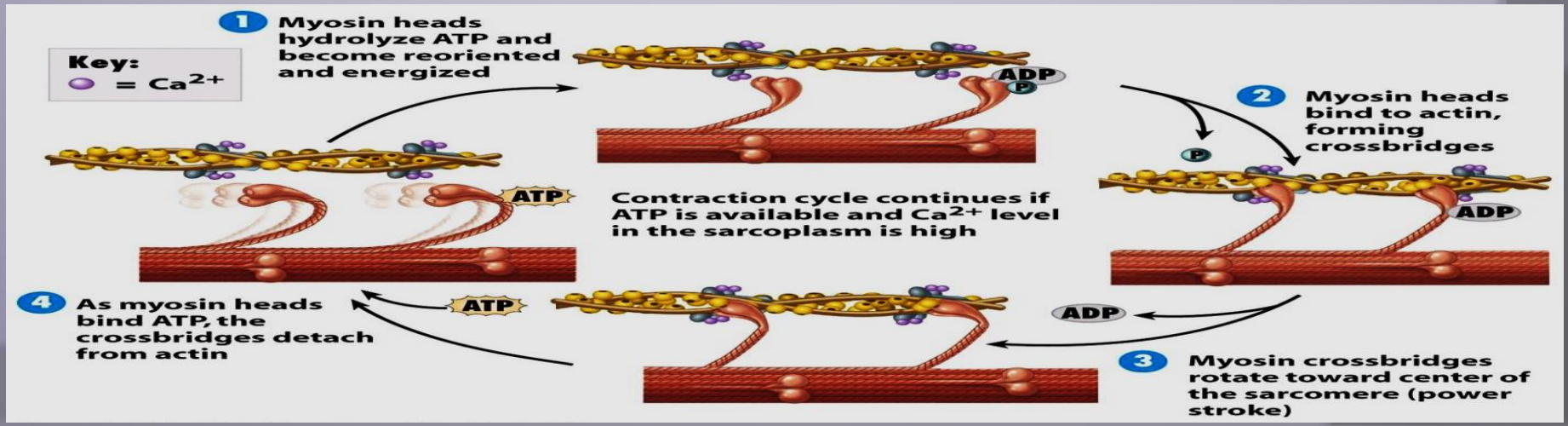
5-The concentration of Ca+2 increases and initiates contraction by binding to troponin-c leading to:

a- Weakness of the binding of troponin I to actin.

b- Movement of tropomyosin laterally into the groove between the thin filaments → uncovering the binding sites of actin for the myosin heads.

6-The interaction between actin & myosin heads leads to sliding of actin filaments between myosin filaments → muscle contraction.

6-The energy required for this mechanism is provided by breakdown of ATP to ADP by ATPase activity of myosin heads in the presence of Ca++ ions.



Results of contraction

-The **sarcomere** becomes **short**.

-The width of **I** band is **decreased**.

-The width of **A** band is remained **constant**.

-H-zone becomes **narrow**.

-M-line Constant.

Mechanism of muscle relaxation

-The **Ca+2** is actively reuptake back to the SR by **active Ca+2 pump** to be stored in the cisterns.

-Decrease the intracellular Ca+2 ions to 10-7 mol/L, the troponin-tropomyosin complex return to its original position separating myosin head from actin that is covered and inhibited by Tropomyosin resulting in muscle relaxation.

-Breakdown of ATP is necessary to Ca+2 pump.

So, ATP hydrolysis is required for **contraction and relaxation** and ↓ ATP → **no relaxation**.

Thank You