



# UNIT 14

## MUSCLE CONTRACTION |

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### 14.1 INTRODUCTION

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In the previous unit, you have learned the structural organization and key features of muscles at molecular level. Recall that sarcomere is the functional structure in skeletal, smooth and cardiac muscles. Muscle cells contain contractile proteins, specifically myosin and actin, which are responsible for muscle contractions. The muscles in our body undergo cyclic contractions and relaxations to facilitate bodily movements. Physical activities such as running and playing games like cricket and football rely on muscle contraction. To understand the process of muscular contraction and relaxation during bodily movement, it is important to study muscle contraction in detail. The skeletal

system, surrounded by muscle tissue, allows the body to move. The muscle system has ability to contract in order to generate sufficient forces that require for any type of body movement. You know our body has three kinds of muscle cells. Skeletal muscle is responsible for bodily movement, cardiac muscle is responsible for generating force to pump blood to the rest of the body, and smooth muscle makes the wall of skin, gastrointestinal tract, kidneys, and other organs. We do many physical activities like dancing, running playing game (football, cricket etc.), or weightlifting etc. All of these tasks associated with the contraction of muscle cells. Have you ever thought how muscles contract and relax? The human body contains specialised contractile cells known as muscle cells. These cells generate motile forces by contraction, which is the direct result of the interaction between the contractile proteins actin and myosin and other proteins.

Therefore, in this unit, we will be discussing about the mechanism of muscles contraction (skeletal and smooth muscles), role of key proteins in muscle contraction, phosphogens and muscle disorders. The calcium ions play crucial role in regulation of muscle contraction and relaxation about which discussed in the unit.

### Expected Learning Outcomes

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After studying this unit, you should be able to:

- ❖ define skeletal muscle contraction;
- ❖ distinguish between skeletal and smooth muscle contraction;
- ❖ discuss the excitation–contraction coupling;
- ❖ explain the role of calcium in muscle contraction;
- ❖ explain sliding filament theory;
- ❖ describe regulation of muscle contraction;
- ❖ discuss the phosphogens and their role;
- ❖ enlist muscular dystrophies.

## 14.2 PHYSIOLOGY OF MUSCLE CONTRACTION

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To understand the mechanism of striated, cardiac and smooth muscle contraction, it is first essential to understand their structure which are described in unit 13. In this section, we will study the mechanism of muscular contraction. Muscle contraction is the basis of the body movement. We first study molecular mechanism of skeletal muscle contraction followed by other two muscle – cardiac and smooth muscles. Recall the structural organization of skeletal muscles from the previous unit section 13.3.1. The muscle cell contains thousands of myofibrils- Each myofibril consists of sarcomere “which

is the functional unit” of muscles cells. Sarcomere contains light band (I) and dark bands (A). There is a H band between A bands. You know that muscle fiber has two types of contractile proteins – myosin and actin and two regulatory proteins- troponin and tropomyosin. Now we discuss here the mechanism of muscle contraction.

### 14.2.1 Excitation-Contraction (EC) Coupling

The molecular mechanism of skeletal muscle contraction is based on the excitation-contraction coupling. The excitation-contraction (EC) coupling is a physiological mechanism by which an electrical signal (excitation) causes a mechanical contraction in muscle cells.

This process involves a series of events at the molecular level. This process is often referred to as the sliding filament theory.

Excitation–contraction coupling includes the following steps (14.1):

#### 1. Excitation of striated muscles:

When an action potential generated in the central nervous system, it reaches to the motor neuron that is located near to the muscle fiber. The action potential further reaches to an axon terminal of nerve by the voltage gated sodium channel and send signals to synaptic vesicle to release neurotransmitter at the neuromuscular junction.

**The neuromuscular junction (NMJ)** is a connecting point where the motor neurons join with the sarcolemma of a skeletal muscle fiber.

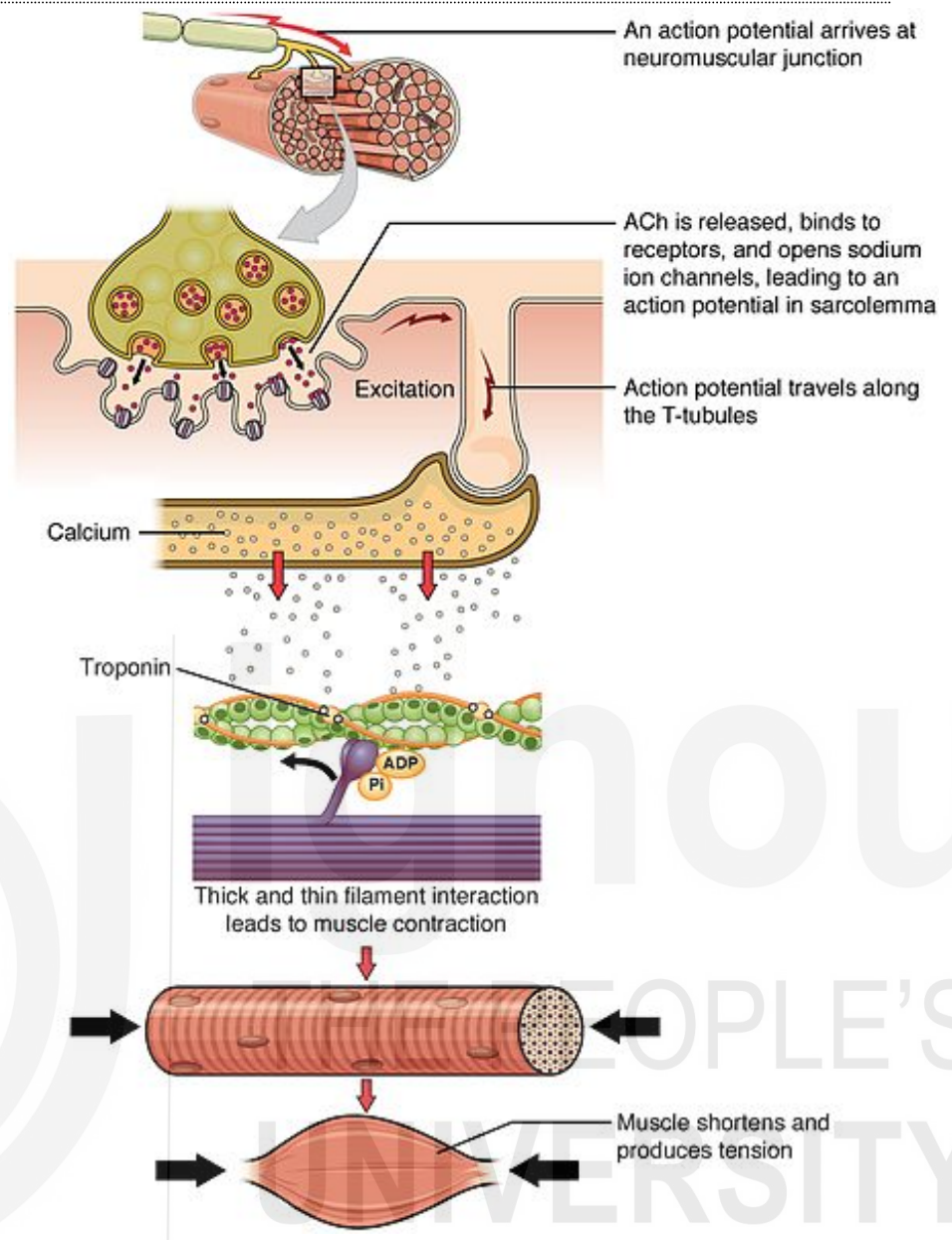
**2. Release and binding of Acetyl choline neurotransmitter (Ach):** The propagated action potential triggers synaptic vesicle to release acetylcholine (Ach) neurotransmitter (in synaptic cleft). Ach binds to the nicotinic acetylcholine receptors (nAChRs) located on the sarcolemma and further transmit active potential along the sarcolemma causing a change in membrane permeability (open sodium channel).

#### 3. Release of calcium ions:

Action potential on the sarcolemma further depolarizes and spreads to the inner side of muscle fiber through the network of transverse T tubules. This process triggers the sarcoplasmic reticulum (SR) to release calcium ions (via voltage calcium ion channel) at higher concentration into the cytoplasm. This process initiates synchronous activation of the whole skeletal muscle cell.

As the calcium ions released, they bind to the troponin, changing its shape and shifting tropomyosin on the F-actin and expose site on the actin for binding of myosin head. As a result, cross bridge is formed between actin thin filament and myosin head thick filament and leads to muscle contraction.

Recalled unit 7  
Cardiovascular system  
to understand EC  
coupling process in  
cardiac muscles which  
is similar the  
mechanism of the  
skeletal muscle.



**Fig. 14.1: Excitation-contraction coupling in a skeletal muscle contraction.**

(Image source: <https://cnx.org/contents/FPtK1zmh@8.25:fE13C8Ot@10/Preface> & <https://commons.wikimedia.org/>)

Let us understand the how skeletal muscle contraction occur.

### **14.2.2 Sliding filament theory**

Sliding filament theory describes how myosin heads bind and move along the thin filaments. Myosin is a unique contractile protein which is able to pull upon actin filament to shorten the length of sarcomere bands. Fig. 14.2 shows the mechanism of skeletal muscle contraction:

#### **a. Calcium binding:**

Calcium ions form a bond with troponin, a regulatory protein located on the thin filaments made of actin. This binding interaction induces a conformational alteration that displaces tropomyosin from the myosin-actin binding site.

**b. Cross bridge formation**

Recall that myosin head has two binding sites, one for actin binding site and another is ATPase site which are crucial for the muscle contraction.

Muscles contraction begins when ATP binds to the myosin head which hydrolyses ATP into ADP and Pi. Myosin head undergoes conformation changes (bend shape) into a coked position during ATP hydrolysis and The ADP and Pi remains bound to the myosin head. At this step, actin binds to the myosin binding site on myosin filament and forms the **cross bridge between actin thin filament and thick filament**. There are many cross bridges formed when the thick and thin filaments overlap each other.

**c. ATP powered power strock**

The power stroke occurs when the myosin heads move approximately 10 nanometres by pulling the thin filaments towards the centre (M line) of sarcomere. The movements of myosin filament slide over the thin filament is called the power strock which resulting in muscle contraction. Myosin head bends and releases the attached ADP and Pi during the power strock. As a result, the myofibril length shortens as both actin a myosin filaments move past each other and overlapping. This mechanism is known as '**Sliding filament Mechanism**'. The total lengths of the thick and thin filaments remain constant during the striated muscle contraction.

However, muscles contraction is a repetitive cycle of contraction and relaxation, occurring between the thin and thick filaments of the sarcomere. ATP plays a crucial role in preparing myosin for binding and replenishing its energy. In a nut shell, this process includes **binding, bending and releasing actin, and straightening the myosin** head again to bind to actin in a new cycle of contraction which is called myosin-actin cycling.

A force is generated by the shorting the length of sarcomere in muscle fibre is called **muscle tension**.

**d.) Cross-Bridge detachment:** This happens when a new ATP molecule attaches to the myosin head, causing the myosin thick filament to separate from the actin thin filament.

**e). Cocking of myosin head-** The myosin head hydrolyzes ATP into ADP and Pi, enabling it to reset to myosin in **cocked position** and prepare for another cycle of attachment to initiate a new power stroke.

**Relaxation:** When the nerve impulse stops, the acetylcholine at the neuromuscular junction decreases and any residual acetylcholine is hydrolyzed by an acetylcholinesterase enzyme. The excessive calcium levels are returned to the sarcoplasmic reticulum. In the absence of calcium, cross bridge formation does not occur, leading to muscle relaxation when the thin and thick filaments passively slide back in their resting state.

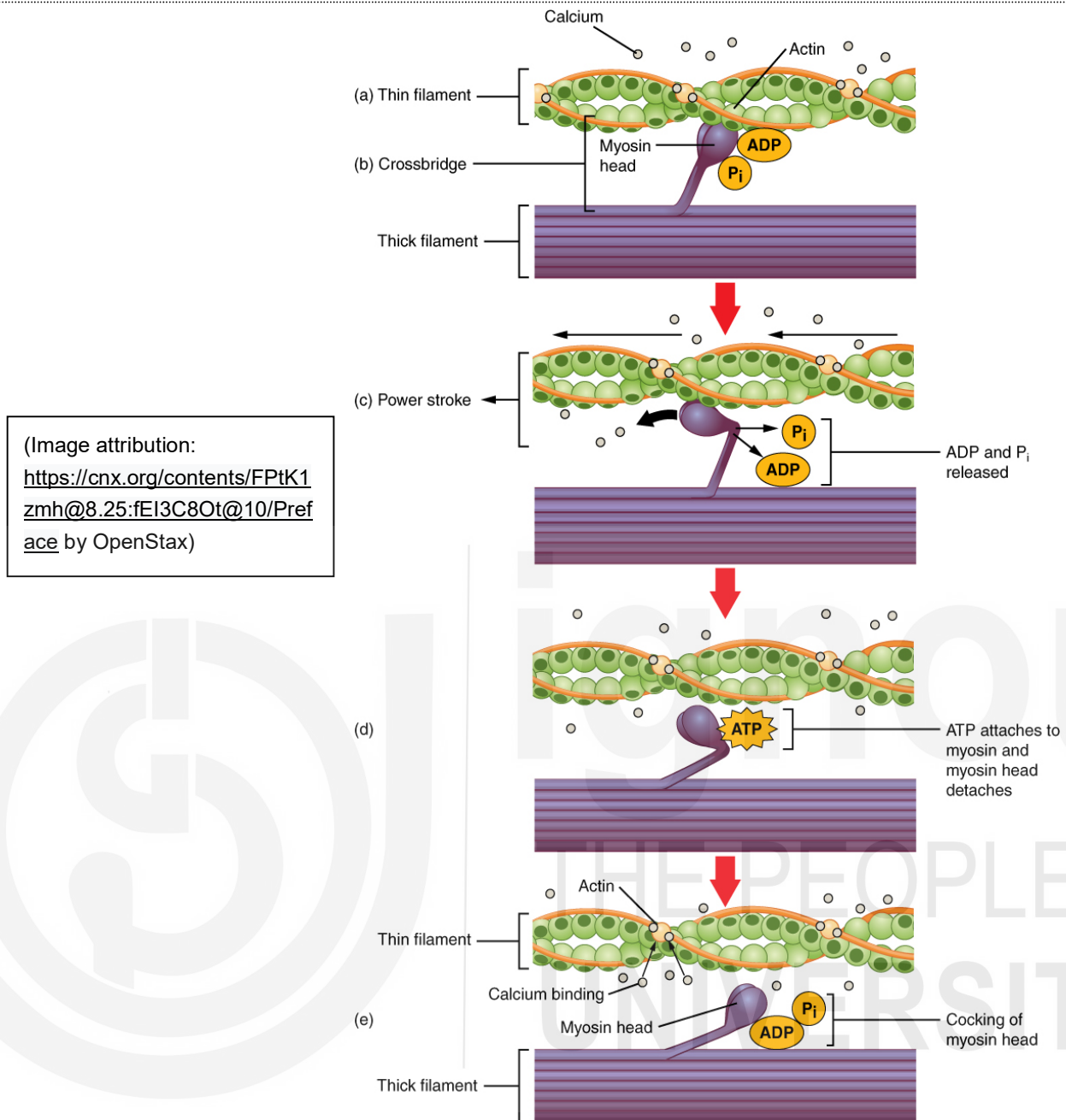


Fig. 14.2: Mechanism of skeletal muscle Contraction.

### SAQ 1

Indicate whether the following sentences are true or false

- The mechanism basis of muscle contraction underlies on the excitation-contraction coupling mechanism and cross bridge formation.
- Acetylcholine release and binding to nAChRs on the sarcolemma initiate muscle excitation.
- Hyperpolarisation of the sarcolemma leads to calcium release from the sarcoplasmic reticulum.

- iv) Calcium binding to actin initiates the contraction cycle by exposing myosin-binding sites on actin.
  - v) Cross-bridge formation occurs when myosin heads bind to actin, initiating muscle contraction.
  - vi) The power stroke is driven by the hydrolysis of ATP by myosin heads, causing filaments to slide past each other.
  - vii) Cross-bridge detachment occurs when ATP binds to myosin heads, allowing them to release from actin.
  - viii) Muscle contraction continues even after calcium is pumped back into the sarcoplasmic reticulum.
  - ix) During relaxation, tropomyosin covers myosin-binding sites on actin, preventing cross-bridge formation.
  - x) Acetylcholine breakdown by acetylcholinesterase stops the signal at the neuromuscular junction, contributing to relaxation.
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### **14.3.2 Contraction of Smooth Muscles**

Recall the structural organization of smooth muscles from the previous unit. Smooth muscles are spindle and non-striated cells unlike striated and cylindrical shape in skeletal muscle cells. They have less uniformly arranged myosin thick filament and actin thin filaments compared to skeletal muscles. They lack troponin but possess tropomyosin, caldesmon, and calponin as regulatory proteins. The diameter of smooth muscle cells is notably less than that of skeletal muscle cells. Furthermore, the action potential can be transmitted to the interior regions of the cell without the assistance of T-tubules.

#### **1. Nerve impulse**

Smooth muscle contraction is triggered by different stimuli that generate a nerve impulse along the sarcolemma, leading to the release of intracellular calcium influx.

#### **2. Release of calcium ions.**

The source of calcium ions in smooth muscle is the both extracellular fluid and sarcoplasmic reticulum (SR). The voltage gated calcium channel in the sarcolemma releases the calcium into the sarcoplasm. SR has limited amount of calcium ions compared to the SR in skeletal muscles. Recall the subsection 13.4.2 sarcoplasmic reticulum to study the process of calcium release. The level of calcium ions is sensed by calmodulin in smooth muscle cells while it is sensed by the **troponin** in skeletal muscles. The **calmodulin** is a regulatory calcium binding protein in smooth muscle contraction (Fig. 14.3).

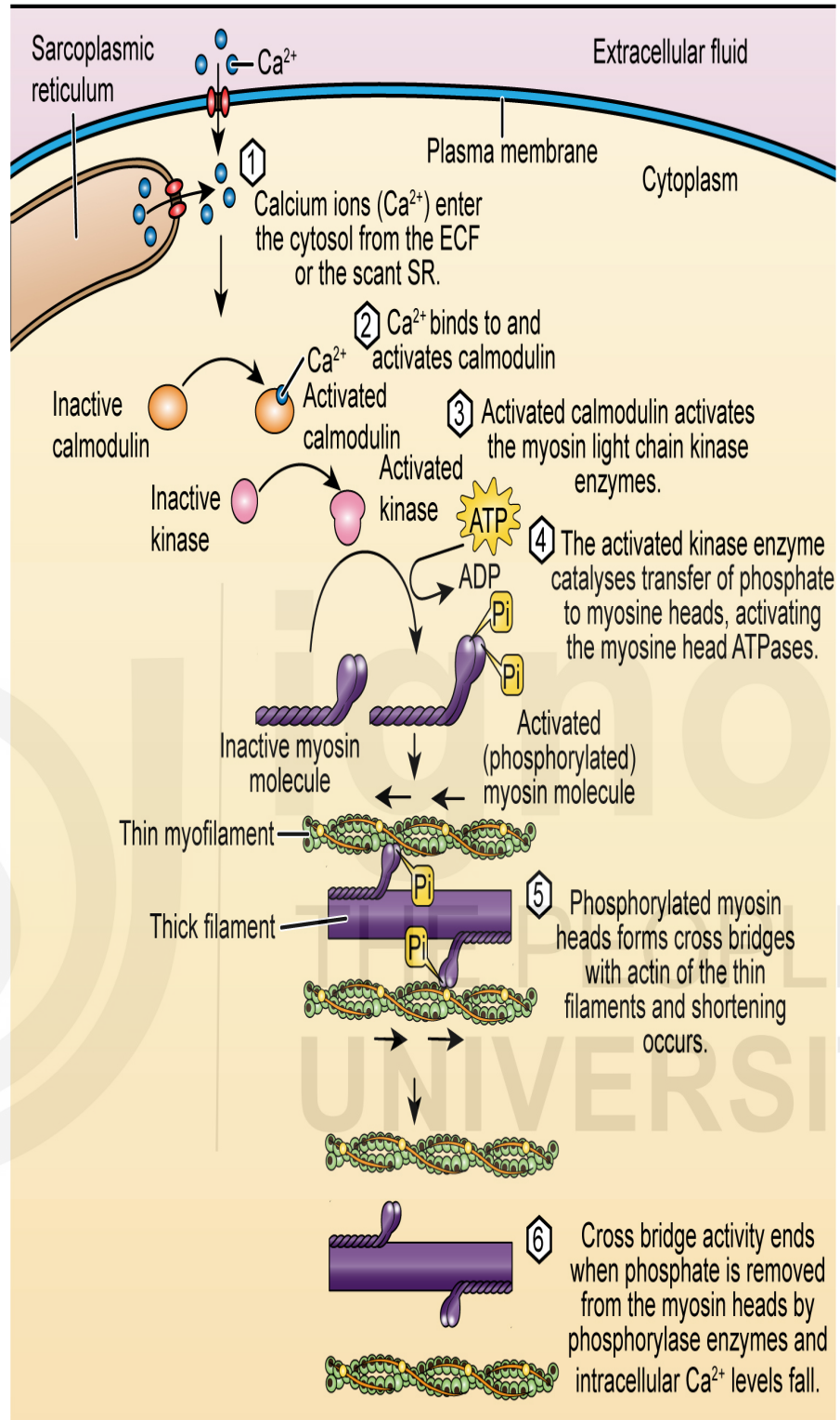


Fig. 14.3: Mechanism of smooth muscle contraction.

### 3. Activation and formation of calcium calmodulin complex

$\text{Ca}^{2+}$  ions bind and activate to calmodulin (a calcium binding protein) inside the cells and form the ' $\text{Ca}^{2+}$ -calmodulin complex' which activates 'myosin light chain kinase'.

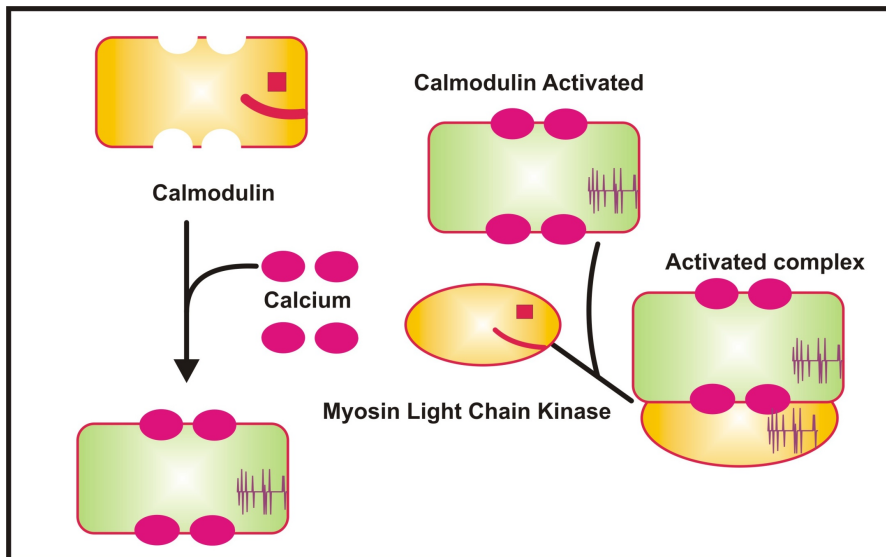


Fig. 14.4 Calcium-calmodulin complexes and activated MLCK.

#### 4. Activation of myosin light chain kinase (MLCK) and phosphorylation of myosin

The activated 'Myosin light chain kinase' uses ATP to add phosphate group at serine 19 position on myosin light chain. This phosphorylation enhances the ATPase activity of myosin. This process is driven by ATP hydrolysis into ADP and Pi. The inorganic phosphate group (Pi) remains attached to the myosin heads.

#### 5. Cross bridge formation

The cross-bridge is a sequence of molecular events that serve as the basis for the sliding filament theory using the same process as in skeletal muscle.

The phosphorylated myosin heads (Pi-myosin) undergo a conformational change that allows the actin to engage to the myosin binding site. This step initiates the **cross bridge formation** between the myosin thick filament and the actin thin filament. The cross-bridge triggers a powerful stroke, in which the myosin filament exerts a pulling force on the actin microfilament, causing the contraction of muscle fibers (sarcomere). This process is derived by ATP. Therefore, the progression of cross bridge events can persist as long as there is a sufficient amount of ATP and intracellular  $\text{Ca}^{2+}$  ions available for muscular contraction. The process of smooth muscle contraction is slower compared to the skeletal muscle contraction.

However, there is another mechanism utilized in smooth muscle for its prolonged contraction called the **latch-bridge mechanism**. The latch-bridge between thick and thin filaments extends the ability of contraction of smooth after declining the level of calcium ions and even without ATP.

#### 6. Relaxation:

The relaxation of smooth muscle filament occurs when calcium-calmodulin complex dissociates and the muscle fibers remain back in their resting position.

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## SAQ 2

### Fill in the blanks:

- i) Various stimuli can cause smooth muscle contraction by sending a nerve impulse down the \_\_\_\_\_, which releases intracellular calcium influx.
- ii) \_\_\_\_\_ detects the concentration of calcium ions in smooth muscle cells, whereas \_\_\_\_\_ detects it in skeletal muscles.
- iii) \_\_\_\_\_ serves as a regulatory protein that binds to calcium and has a role in the contraction of smooth muscle.
- iv) When  $\text{Ca}^{2+}$  ions attach to the calcium-binding protein calmodulin within the cells, they create \_\_\_\_\_. This compound activates the enzyme called \_\_\_\_\_.
- v) The enzyme that catalyzes the phosphorylation of the myosin light chain at serine 19 by adding a phosphate group is called \_\_\_\_\_.
- vi) Phosphorylated myosin head undergoes the conformational change to binds to \_\_\_\_\_ and form a \_\_\_\_\_ between thick and thin filaments.
- vii) The \_\_\_\_\_ is a mechanism that allows a specific connection to between the thick and thin filaments, without requiring the hydrolysis of ATP shortage of calcium ions.
- viii) The relaxation of smooth muscle filament takes place when the calcium-calmodulin combination \_\_\_\_\_ and the muscle fibers revert to their resting state.

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## 14.4 REGULATION OF CONTRACTION IN STRIATED AND SMOOTH MUSCLES

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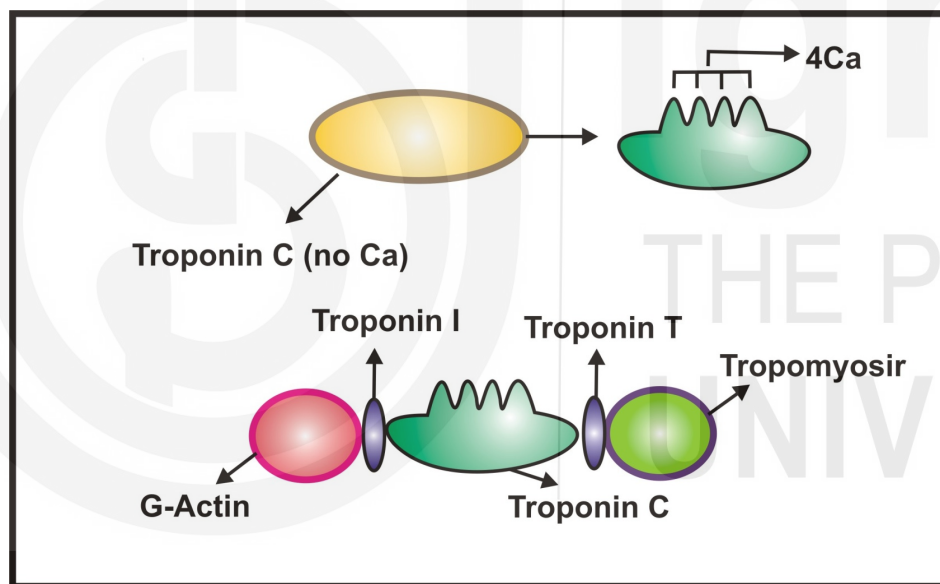
### 14.4.1 Regulation of striated skeletal muscle contraction:

Troponin and tropomyosin are essential regulatory proteins that control muscle contraction. These two proteins function together in response to calcium ions during muscle contraction and relaxation. Troponin controls the positioning of tropomyosin on the actin filament based on the concentration of calcium ions. Troponin is a complex of three subunits namely troponin C (TnC), troponin I (TnI), and troponin T (TnT).

- a) Troponin C (TnC) is a protein that is sensitive to calcium and contains four binding sites for  $\text{Ca}^{2+}$  ions. It attaches to the calcium ions and induces a change in shape to create a connection between the thin

and thick filaments during muscular contraction. Each subunit consists of a calcium coordinating loop that is abundant in acidic residues and is surrounded by two  $\alpha$ -helical segments. The formation of a complex known as troponin calcium complex induces a conformational change, which in turn leads to the displacement of tropomyosin from the actin binding site on the myosin filament.

- b) Troponin I function as an inhibitory element inside the troponin complex. It inhibits the ATPase activity of actinomyosin, which binds to actin filaments in the absence of calcium, hence preventing the binding of actin and myosin.
- c) Troponin T serves as the subunit of troponin that binds to tropomyosin. During muscle contraction, calcium influx triggers the binding of actin to the myosin binding head site. This binding causes tropomyosin to shift from the myosin side. Tropomyosin inhibits the interaction between actin thin filaments and myosin thick filaments by blocking the myosin binding site on actin. This regulation mechanism limits the creation of cross bridges and controls skeletal muscle contraction.



**Fig. 14.5 Subunits of troponin and its calcium (Ca) binding sites.**

[Credit: [https://en.wikipedia.org/wiki/Troponin\\_C#/media/File:Troponino.svg](https://en.wikipedia.org/wiki/Troponin_C#/media/File:Troponino.svg)]

#### Availability of Calcium ions;

The regulatory function of troponin and tropomyosin protein in skeletal muscles contraction depends on the increase or decrease concentration of calcium ions in response to sufficient active potential. Muscle contraction occurs when the sarcoplasmic reticulum releases calcium ions at a greater concentration, stimulating the development of cross bridges between actin and myosin filaments. As the concentration of calcium ions falls, they no longer bind to troponin C and prevent additional cross bridge formation, leading to muscular relaxation.

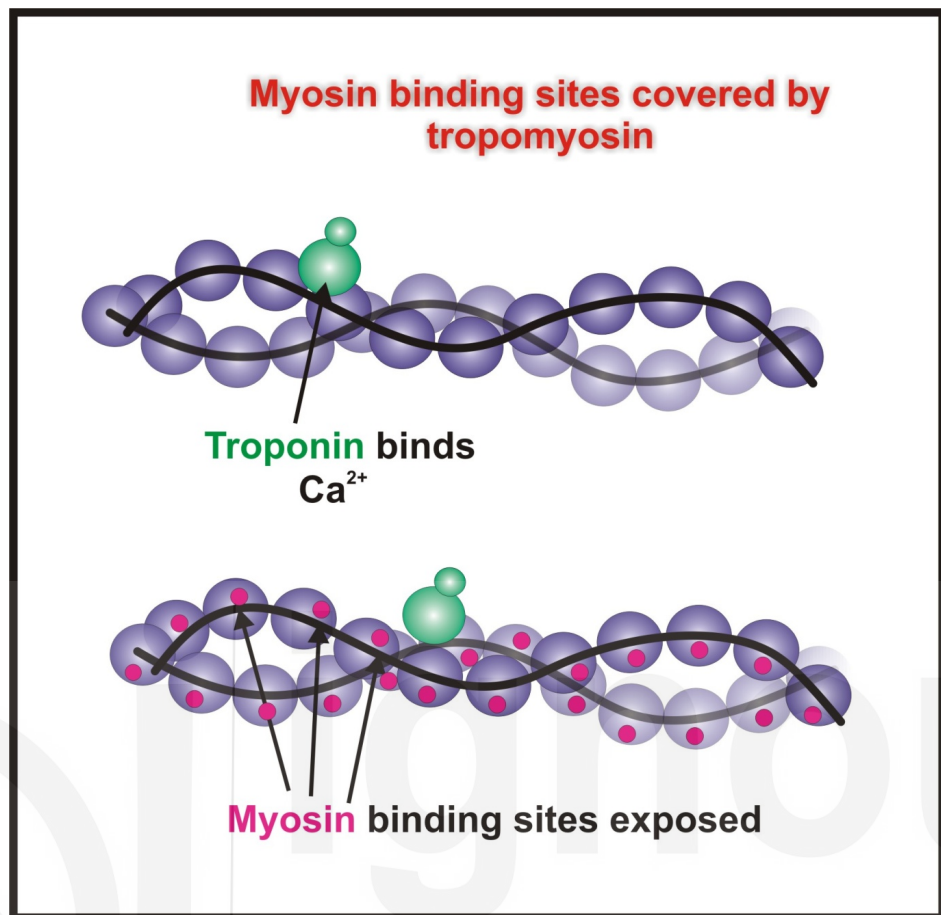


Fig. 14.6: Troponin calcium interaction and confirmation shift.

Actin's myosin-binding sites are blocked by tropomyosin during muscle relaxation, and during contraction, the protein travels away from these sites, enabling myosin to bind to the actin to produce force. This fine regulation allows for controlled and coordinated motions by limiting the occurrence of muscle contraction to relevant inputs.

#### **14.4.2 Regulation of smooth muscle contraction**

The regulation of smooth muscles contraction is basically relying on the availability of calcium ions, calmodulin activation and phosphorylation of Myosin light chain (MLC).

##### **Role of calmodulin:**

The calmodulin is key regulatory protein of the smooth muscle contraction. It is main calcium binding protein in smooth muscle. As  $Ca^{2+}$  ions release from the extracellular or SR. it first interacts with the calmodulin protein and form the calcium-calmodulin complex. This complex further activates myosin light chain kinase (MLCK)

As intracellular calcium levels decrease, calcium dissociates from calmodulin, reducing the activation of MLCK and thus smooth muscles undergo relaxation state.

## 14.5 PHOSPHAGENS

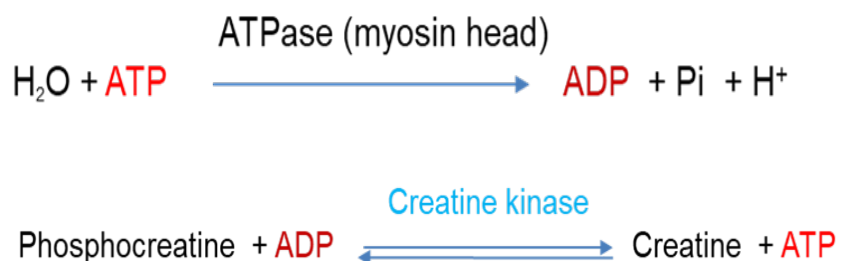
From the discussion in the preceding section of muscles contraction, you may notice that muscles require ATP as their primary source of energy to do contraction work. They utilize the chemical energy stored in ATP to generate mechanical force through the execution of several bodily functions. For muscles to undergo a continuous contraction cycle, they require a constant supply of adenosine triphosphate (ATP), which serves as the main energy source for cells. Decreased ATP levels have a direct impact on muscular contraction. In order to maintain a stable ATP level, human body muscles possess a unique type of energy storage molecules that are mostly present in skeletal muscle, cardiac cells, and other tissues. Molecules containing high-energy phosphate groups are referred to as phosphagens. Their main function is to rapidly replenish the chemical energy (ATP) needed by muscles, especially during strong muscular exercise. Phosphagens are efficient energy storing phosphate compounds that are mainly found in muscle cells and nervous system. Therefore, they play a vital role in the prompt restoration of adenosine triphosphate (ATP).

Phosphagens are stored within the sarcoplasm of muscle cells. During resting periods of muscles, ATP produced by oxidative phosphorylation or glycolysis is utilized to phosphorylate creatine or arginine, resulting in the formation of a reserve of phosphagens.

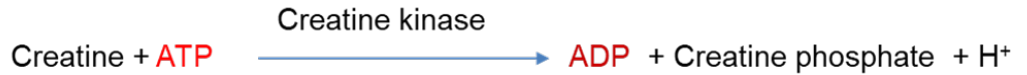
**Creatine phosphate** is a type of phosphogen compounds that is mostly present in vertebrate muscles, specifically in skeletal and cardiac muscles. It is also known as phosphocreatine (PCr) or macroergic compounds. Remember that the previous section discussed how the creation of phosphate is an energy system used in muscle contraction. Phosphocreatine is predominantly synthesized in the liver, kidney, and pancreas and subsequently transported to the muscle cells. It consists of two molecules: creatine and a phosphate group. The main function of phosphocreatine is to transfer its phosphate group to ADP and rapidly replenish ATP.

The creatine kinase enzyme facilitates the reversible chemical reaction between creatine phosphate and ADP, as depicted below: ADP released by ATP hydrolysis is reutilised for ATP production during muscle contraction.  $Mg^{2+}$  ion is required for this reaction.

**During muscle contraction:**



### Muscles at resting period



Therefore, phosphagens are essential high-energy storage phosphate molecules that supply immediate ATP during vigorous muscular exercise. They function as a homeostatic buffer system to ensure the maintenance of energy levels (ATP) for muscular contraction.

### SAQ 3

*Answer the followings:*

- What is the primary source of energy for muscle contraction?  
\_\_\_\_\_
- What do muscles use ATP for during contraction? \_\_\_\_\_
- The main function of phosphagens is \_\_\_\_\_
- Which enzyme facilitates the reaction between creatine phosphate and ADP? \_\_\_\_\_
- What happens to ATP during resting periods of muscles? \_\_\_\_\_

## 14.6 MUSCULAR DYSTROPHIES

Muscular dystrophies are related to dysfunction of muscles or abnormal neuromuscular activity. They are characterized by symptoms like muscle weakness impaired mobility, diminished reflexes, muscle atrophy and other muscle related health problems. They are generally hereditary disorders that often appear during childhood due to genetic changes. Hence, they are genetic disorders that lead to progressive muscular degeneration and muscles weakness. These disorders vary in terms of the specific muscles that are impacted, the age at which symptoms begin, and the speed at which the condition worsens over time. There are many muscular dystrophies. However, some of the typical muscular dystrophies and their inherited reason and characteristic features/symptoms are given in Table 14.1

**Table 14.1 common Muscular Dystrophies**

| Muscle dystrophies                | Inherited causes   | Occurrence      | Features   |
|-----------------------------------|--------------------|-----------------|--|
| Duchenne Muscular Dystrophy (DMD) | X-linked recessive | Early childhood | Rapid progression of muscle weakness, particularly in the legs |

|                                       |                                      |   |   |
|---------------------------------------|--------------------------------------|---|---|
| (a most common genetic disorder)      |                                      |   | and pelvis, eventually affecting all voluntary muscles and the heart.   |
| Becker Muscular Dystrophy (BMD)       | X-linked recessive                   | Adolescence or early adulthood                    | Similar to Duchenne but with a slower progression and later onset.  |
| Myotonic Dystrophy (DM)               | Autosomal dominant                   | Can occur from birth to adulthood                 | Muscle weakness, prolonged muscle contractions (myotonia), cataracts, cardiac abnormalities   |
| Limb-Girdle Muscular Dystrophy (LGMD) | Both autosomal recessive or dominant | Childhood to adulthood                            | Develop progressive weakness and wasting of the muscles around the hips and shoulders.  |
| Congenital muscular dystrophies       | Autosomal recessive or dominant both | present at birth or appear before the age of two. | develop joint problems, scoliosis, respiratory and swallowing difficulties, seizures, or vision problem   |
| Myotonic muscular dystrophy           | Autosomal dominant                   | From childhood to adults                          | difficulty with muscle relaxation; weakness in the distal extremities, such as the hands and wrists; prolonged muscle contraction, affects men and women equally. |

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## 14.4 SUMMARY

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In this chapter, we have discussed and learned that:

- Myosin and actin proteins play a crucial role in muscular contraction. The sarcomere of muscle fibers is the main functional unit of muscle contraction.
- Muscle contraction is directed by active potential mediated orderly events. Excitation-contraction coupling refers to a sequence of events that leads to the contraction of skeletal muscle.

- Motor neuron stimulation is responsible for muscle contraction. When muscle excites, it generates depolarization which stimulates synaptic vesicles to release an ACh neurotransmitter and its chemical information transmits to the muscles via neuromuscular junction (NMJ) and Transverse T tubules which further lead to EC coupling and the muscle contraction process.
- Troponin is a calcium-binding protein which plays a crucial role in the regulation of muscle contraction.
- Tropomyosin is a protein that extends throughout the entire length of the actin filament and blocks the myosin-binding sites on actin molecules during muscular relaxation.
- As the calcium-troponin C complex attaches to the myosin heads, it forms a cross-bridge and walks along the thin filaments, thereby the myofilament pulls the actin filament towards the center of the sarcomere. This process involves ATP hydrolysis which provides the power stroke for muscle contraction.
- ATP hydrolysis is the main source of energy in muscle contraction.
- Upon formation of a cross-bridge, the actin and myosin filaments overlap and slide past one another. The myosin heads attach and walk along the thin filaments, a mechanism known as 'Sliding filament mechanism'.
- Troponin plays a key role in the regulation of muscle contraction by interacting with tropomyosin and actin filaments in response to alterations in intracellular calcium ion levels.
- The contraction of smooth muscle is initiated when calcium binds to the calmodulin protein. The formation of the calmodulin-calcium complex activates myosin light chain kinase, which subsequently initiates the phosphorylation of myosin. This leads to the sliding of actin and myosin filaments over each other, facilitated by the formation of cross-bridges. ATP is involved in the mechanism that is analogous to the sliding filament theory in the contraction of skeletal muscles.
- Muscle relaxation occurs when motor neurons stop releasing their chemical signal, ACh, into the synapse at the NMJ, which in turn causes the skeletal muscle fibers and the muscle itself to relax.
- Phosphagens are high-energy phosphate groups present in muscle tissues. They are characterized by generating muscle ATP during intense exercise. Creatine phosphate is an energy storage phosphagen and maintains ATP levels in the muscles.

## 14.5 TERMINAL QUESTIONS

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1. What is excitation contraction coupling? Discuss its process.
2. Explain how nerve impulse travel to the muscle fiber.
3. Describe the sliding filament theory for muscle contraction.
4. Differentiate the muscle contraction process between skeletal muscles and smooth muscles.
5. Explain how cross bridge is formed with the stable diagram.
6. describe how muscle contraction regulate
7. What are phosphogens and discuss their role in muscle contraction.
8. Enlist the muscular disorders with key symptoms.

## 14.6 ANSWERS

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### Self assessment Questions

1.
  - i) True
  - ii) True
  - iii) False
  - iv) False
  - v) True
  - vi) True
  - vii) True
  - viii) False
  - ix) True
  - x) True
2.
  - i) sarcolemma,
  - ii) Calmodulin troponin detects it in skeletal muscles.
  - iii) Calmodulin
  - iv) 'Ca<sup>2+</sup>-calmodulin complexes'. 'myosin light chain kinase'.
  - v) 'Myosin light chain kinase'.
  - vi) actin a cross-bridge
  - vii) The latch-bridge
  - viii) separates

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3.
  - a) ATP (adenosine triphosphate).
  - b) To generate mechanical force for various bodily functions.
  - c) To rapidly replenish ATP needed by muscles, especially during strong exercise.
  - d) Creatine kinase.
  - e) It phosphorylates creatine or arginine, forming a reserve of phosphagens.

### Terminal Questions

1. Refer to subsection 14.2.1
2. Refer to subsection 14.2.1
3. Refer to subsection 14.2.2
- 4.

| Skeletal muscles contraction  | Smooth muscles contraction  |
|---|---|
| i) Striated Skeletal muscles  | i) Non-Striated Smooth muscle   |
| ii) Contraction is initiated by the occurrence of an action potential, which leads to the release of acetylcholine (ACh) at the <b>neuromuscular junction</b> . | ii) Stimulates by stimuli   |
| iii) Calcium ions bind to the troponin  | iii) Calcium ions bind to calmodulin, a regulatory protein  |
| iv) Troponin interaction with myosin promotes conformation changes to establish a cross bridge between myosin and actin filaments.                              | iv) Myosin Light Chain Kinase (MLCK) is activated by the calcium-calmodulin complex and phosphorylates myosin light chains. |
| v) Cross bridge occurs but no latch bridge mechanism.   | v) In addition to cross bridge, latch-bridge mechanism allows muscle to prolong contraction                                 |
| vi) Skeletal muscle relaxation occurs when a motor neuron stops the action potential, causing calcium levels to drop.   | vi) Dephosphorylated myosin and low intracellular calcium levels cause smooth muscle relaxation.                            |

5. Refer to subsection 14.2.2
6. Refer to section 14.4
7. Refer to section 14.5
8. Refer to section 14.6

