

# BIOLOGY

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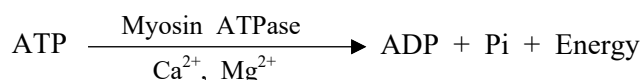
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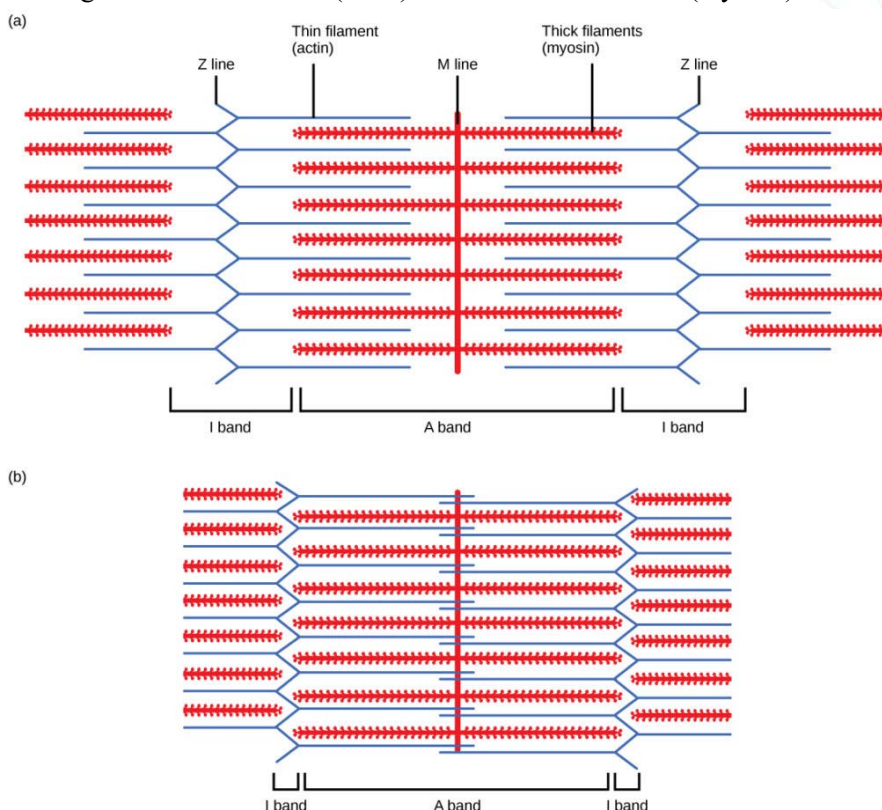
## 01. Mechanism of Muscle Contraction

The mechanism of muscle contraction is explained by sliding filament theory. This theory was given by two groups of workers namely Andrew Huxley and Ralph Niedergerke, Hugh Huxley and Jean Hanson in 1954.

- (i) The process of muscle contraction begins, when a neural signal reaches the neuromuscular junction from CNS *via* motor neurons. Neuromuscular junction is the junction between the nerve fibre and muscle fibre.
- (ii) These impulses then reach the terminal ends of axons of neuromuscular junction. Due to this, the vesicles having neurotransmitter, i.e. acetylcholine fuses with the axon membranes. Thus, acetylcholine is released. This acetylcholine travels through synaptic cleft (region between axon membrane and motor end plate) and an action potential is generated within the sarcolemma.
- (iii) This action potential is transmitted to the T-tubules and then to sarcoplasmic reticulum. This relay is aided by a protein called **dystrophin**.
- (iv) Saracoplamic reticulum upon receiving the action potential release  $\text{Ca}^{2+}$  binds to troponin C and as a result of this troponin exposes the myosin binding sites of actin.
- (v) These events are accompanied by the ATPase activity of globular head of myosin. It starts hydrolysing ATP molecule. The energy released is utilised by myosin cross-bridges to bind with actin.



After the myosin-actin cross-bridges are formed the contraction of muscle fibre begins by the sliding of thin filaments (actin) over thick filaments (myosin).

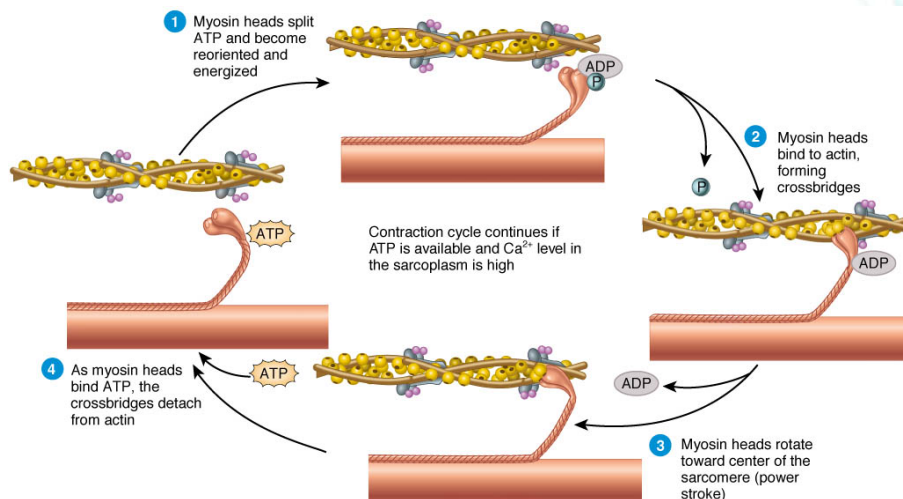


- (vi) During sliding movement, the cross-bridges of myosin undergo rotation. Due to this rotation actin is pulled towards the centre of A-band. Thus, the Z-line attached to these actins is also pulled. As a result of pulling of Z-line, shortening of sarcomere, takes place, i.e. contraction occurs.
- (vii) The myosin further releases ADP + Pi and become relaxed. Again ATP binds to myosin head and cross-bridges are broken between myosin and actin. ATP is again hydrolysed and this cycle of formation and breakage of cross-bridges is repeated causing sliding. The process continues till calcium ion is pumped back into sarcoplasmic reticulum.
- (viii) The length of the thick and thin myofilaments does not change during muscle contractions.

## 02. Muscle Relaxation

The steps of this process are as follows

- (i) It is believed that relaxation may begin due to an enzyme called **acetylcholinesterase**. This enzyme causes breakdown of acetylcholine present in the synaptic cleft into acetate and choline. As a result of this generation of action potential is retarded.
- (ii) This causes calcium ions ( $\text{Ca}^{2+}$ ) to return quickly to the sarcoplasmic reticulum by active transport, with the help of ATP. In the sarcoplasmic reticulum,  $\text{Ca}^{2+}$  binds to a calcium-binding protein called **calsequestrin**.
- (iii) this make troponin to be free thus, it again covers myosin binding site of actin.
- (iv) The myosin-actin cross-bridge formation stops. This in turn prevents sliding of filaments thus preventing the muscle from contraction.



## 03. Energy Release During Muscle Contraction

The ultimate source of energy for the process of muscle contraction is glycogen. It is stored in the muscles along with glucose.

*Following points are important regarding the energy release :*

- (i) Carbohydrate are broken down in the muscle both aerobically and anaerobically.
- (ii) In aerobic breakdown, oxygen is made available by oxymyoglobin and also from blood.
- (iii) In anaerobic breakdown, glucose is broken down through anaerobic glycolysis. This forms pyruvate which is then converted into lactic acid along with two molecules of ATP.
- (iv) ATP is an immediate source of energy for muscle contraction.
- (v) The muscle also contains reserve energy currency in the form of Creatine Phosphate (CTP).

