Striated Muscle Contraction

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This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the Reactome Textbook.

17/11/2022
**Introduction**

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

**Literature references**


Reactome database release: 82

This document contains 1 pathway and 4 reactions (see Table of Contents)

https://reactome.org
Striated muscle contraction is a process whereby force is generated within striated muscle tissue, resulting in a change in muscle geometry, or in short, increased force being exerted on the tendons. Force generation involves a chemo-mechanical energy conversion step that is carried out by the actin/myosin complex activity, which generates force through ATP hydrolysis. Striated muscle is a type of muscle composed of myofibrils, containing repeating units called sarcomeres, in which the contractile myofibrils are arranged in parallel to the axis of the cell, resulting in transverse or oblique striations observable at the level of the light microscope.

Here striated muscle contraction is represented on the basis of calcium binding to the troponin complex, which exposes the active sites of actin. Once the active sites of actin are exposed, the myosin complex bound to ADP can bind actin and the myosin head can pivot, pulling the thin actin and thick myosin filaments past one another. Once the myosin head pivots, ADP is ejected, a fresh ATP can be bound and the energy from the hydrolysis of ATP to ADP is channeled into kinetic energy by resetting the myosin head. With repeated rounds of this cycle the sarcomere containing the thin and thick filaments effectively shortens, forming the basis of muscle contraction.

**Literature references**


**Editions**

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Calcium Binds Troponin-C

**Location:** Striated Muscle Contraction

**Stable identifier:** R-HSA-390595

**Type:** binding

**Compartments:** cytosol

Troponin (Tn) is the central regulatory protein of striated muscle contraction. Tn consists of three components: troponin I (TNNI3; the inhibitor of actomyosin ATPase), Tn-T (which contains the binding site for tropomyosin) and troponin C (TNNC1, Tn-C). The binding of calcium to TNNC1 abolishes the inhibitory action of Tn on actin filaments. At the start of the striated muscle contraction cycle, a myosin head lacking a bound nucleotide is locked tightly onto an actin filament in a rigor conformation. TNNC1 binds four calcium ions. In an actively contracting muscle this state is very short-lived, being rapidly terminated by the binding of a molecule of ATP.

**Followed by:** Myosin Binds ATP

**Literature references**


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Myosin Binds ATP

Location: Striated Muscle Contraction

Stable identifier: R-HSA-390598

Type: binding

Compartment: cytosol

A molecule of ATP binds to the large cleft on the side of the myosin head farthest from the actin filament and immediately causes a slight change in the conformation of the domains that make up the actin-binding site. This reduces the affinity of the myosin head for actin and allows it to move along the filament.

Preceded by: Calcium Binds Troponin-C, Release Of ADP From Myosin

Followed by: ATP Hydrolysis By Myosin

Literature references


The cleft closes like a clam shell around the ATP molecule, triggering a large shape change that causes the myosin head to release actin and be displaced along the actin filament by a distance of about 5 nm. Hydrolysis of ATP occurs, but the ADP remains tightly bound to the protein.

**Preceded by:** Myosin Binds ATP

**Followed by:** Release Of ADP From Myosin

**Literature references**


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Release Of ADP From Myosin

**Location:** Striated Muscle Contraction

**Stable identifier:** R-HSA-390597

**Type:** dissociation

**Compartments:** cytosol

The weak binding of the myosin head to the new site on the actin filament causes release of the inorganic phosphate produced by ATP hydrolysis, concomitantly with the tight binding of the head to actin. This release triggers the power stroke, a force-generating change in the shape during which the head regains its original conformation. In the course of the power stroke, the head loses its bound ADP, thereby returning to the start of a new cycle.

**Preceded by:** ATP Hydrolysis By Myosin

**Followed by:** Myosin Binds ATP

**Literature references**


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