MUSCLE PHYSIOLOGY

PART 3: MUSCLES – THE WORKING UNITS

Figure 1. Structure of the sarcomere

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Part two of this series explored the anatomy of a muscle, looked at the blood and nerve supply and examined how the arrival of a nerve stimulus spread a wave of excitation across the muscle. This part examines the structure of the smallest functional unit of muscle tissue – the sarcomere – and how these units function in order to shorten or contract a muscle.

**SARCOMERES**

A single muscle myofibril (see part two of this four-part series) contains about 10,000 tiny functional units called sarcomeres (Martini, 2005), which are laid end to end rather like sweets in a tube or the carriages of a train. Interactions within these units are responsible for muscle shortening and contraction. Sarcomeres have a striped appearance under the microscope because of their anatomical structure (Fig 1).

**A BANDS**

These bands, which look dark, are found in the centre of the sarcomere and are composed of relatively thick filaments of the protein myosin. The M line is a central sheet of protein, to which two bands of thick filaments, facing in opposite directions, are attached. It stabilises the structure. Within the A band is a zone where thick and thin filaments overlap. Two T tubules (see part two) encircle each sarcomere at these points. This is where stored calcium ions are released and enter the sarcolemma. An area known as the H zone contains only thick filaments and no thin filaments when the cell is at rest.

**THICK FILAMENT**

A single muscle may contain 15 billion thick filaments. These are twice the diameter and half as long again as thin filaments and each consists of over 100 molecules of the protein myosin (McLaren, 2005). Myosin molecules have a double head and a long tail, which is bound to other myosin molecules (Fig 2). The heads (also known as cross-bridges) are arranged in a spiral and protrude towards the nearest thin filaments. The myosin molecules are arranged with their heads pointing away from the M line. The ‘neck’ of the molecule acts as a hinge and allows the head to pivot and to move towards or away from the M line. This pivoting is the key step in muscle contraction.

**I BANDS**

These are the lighter bands in the sarcomere and are composed of thin actin filaments. They run from the A band of one sarcomere to the A band of the next (Fig 1). The Z line marks the boundaries of the sarcomeres. A protein called connectin extends from the Z line, where it is bound to the thin filament, to the M line. It stabilises the thin filaments.

Thin filaments contain three different proteins; these are actin, troponin and tropomyosin. The actin filaments have binding sites that can attach to thick filaments but, at rest, this binding is prevented by the troponin and the tropomyosin molecules.
FIG 2. MYOSIN MOLECULES

Muscle contraction occurs when the thick and thin filaments slide along one another. This is only possible when calcium is released around the sarcomere allowing calcium ions to bind to the troponin molecules. This changes the position of the troponin-tropomyosin complex and exposes the binding sites on the actin strands.

When an electrical stimulus reaches a triad (a T tubule surrounding a sarcomere and the two cisternae – see part two – associated with it), it triggers the release of calcium from the sarcoplasmic reticulum and within a millisecond, calcium levels around the sarcomere reach 100 times their resting level. Because the cisternae are situated at the zones of overlap, the effect of calcium release on the sarcomere is almost instantaneous.

Calcium binds to troponin and exposes the active sites on the thin actin filaments.

The myosin heads are able to bind to these sites. Each myosin head pivots and slides the actin filaments along (Fig 3), thus shortening the muscle. The head then detaches, recocks itself and binds to the next actin molecule, pivots and detaches again. This pivoting and recocking can occur five times per second.

When muscle fibres contract, the H zones disappear, the I bands become smaller and the zones of overlap become larger. The Z lines (the sarcomere boundaries) move closer together but the width of the A bands remains constant. Muscles often shorten by about 30–35% of their length (Marieb, 2006).

A muscle will contract as long as a nerve stimulus continues to arrive at the neuromuscular junction. Once it stops, calcium levels in the sarcoplasm fall, calcium ions detach from troponin, which returns to its original place, and the binding sites on the actin strands are covered once more. Muscles need external forces to return to their original length – they do not recoil automatically.

FIG 3. MYOSIN FILAMENTS

Muscle energy – ATP

This activity expends energy which, in muscle cells, is released by breaking down the energy source adenosine triphosphate (ATP). The demand for ATP is enormous (when a muscle is contracting, each thick filament breaks down about 2,500 ATP molecules per second and a single muscle can contain 15 billion thick filaments) and muscles quickly use up their stores of it as well as manufacturing it while they are working.

When oxygen is present in the muscle, the mitochondria absorb substances, particularly fatty acids, from the cytoplasm and break them down. This is an efficient process that releases large amounts of energy, which the cells use to make ATP. At rest, skeletal muscles produce more ATP than they need and this is used to form a high-energy compound – creatine phosphate – which is stored in the muscle cells. However, the stores are used up within a few seconds when muscles are active. To prepare for this, excess ATP in muscle cells is used to convert glucose to glycogen for storage in the muscle and liver cells.

During muscle movement, the demand for ATP rises and creatine phosphate stores are soon used up. The mitochondria are not able to make ATP fast enough and so glycogen is mobilised from its stores in the muscle fibres, which contain large reserves, and is split apart to release glucose. The glucose is further broken down and ATP is released in the process. This is a much less efficient process and far less ATP is released, but it does not require oxygen and so it is important when the mitochondria cannot meet the high demand for ATP.

The final article in this series outlines how shortening muscles cause limb movements and addresses some of the problems that occur with our skeletal muscles.

REFERENCES


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