**Anatomy**

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Skeletal excitation and contraction are complex physiological processes that the journey starts when a motor neuron generates action potentials that reach the muscle fiber. This electrical signal follows the neuron's path down to its terminal where acetylcholine (ACh), a neurotransmitter, is released into the synaptic cleft, a, small gap, between the neuron and the muscle fiber. ACh attaches to sarcolemma receptors which is the muscle fibers external membrane causing a series of events until the muscle starts to contract. This relates to the transmission of the action potential first along the sarcolemma and finally to within the muscle fiber with the use of tubules called T-tubules. This electrical activity causes the influx of calcium ions from the sarcoplasmic reticulum, a specific endoplasmic reticulum present in muscle cells, into the cytosol of the muscle fiber.

The spike of calcium ions in the cytoplasm is the key to muscle contraction. Calcium binds to the troponin, a regulatory protein specifically attached to the thin filaments of the muscle fiber. This binding results in a conformational change in another protein, tropomyosin, which under normal circumstances, occupies the active sites on actin molecules in the thin filaments. Therefore, the displacement of tropomyosin creates new binding sites for myosin heads that can bind to them, resulting in the formation of cross-bridges. Powering with Adenosine Triphosphate (ATP), each myosin head swings, thereby bringing the thin filament farther past the thick filament; shortening the muscle fiber—this is the contraction. At the end of the process, calcium is pumped back into the sarcoplasmic reticulum, tropomyosin covers the binding sites on actin, and muscle fiber relaxes. Acetylcholine release stoppage and ACh metabolism by acetylcholinesterase are also instrumental factors in muscle relaxation.