Skeletal muscle excitation-metabolism coupling

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Mitochondria represent the main source of ATP in skeletal muscle and mitochondria activity increases after muscle fiber depolarization. The regulation of mitochondrial function during contraction in skeletal muscle, however, is poorly understood. Skeletal muscle has a particular distribution of mitochondria where three distinct populations can be recognized. The most studied populations are the ones positioned deep into the myofibers between the myofibrils (intermyofibrillar mitochondria), and that located immediately beneath sarcolemma (subsarcolemmal mitochondria); a less studied population locates covering the myonuclei, as a continuation of the subsarcolemmal population. All mitochondria populations undergo fusion and fission events and intermyofibrillar mitochondria are interconnected; mitochondrial communication is necessary to maintain not only the energetic homeostasis of the muscle but its contractile function, as well. The mechanism supporting communication between subsarcolemmal and intermyofibrillar mitochondria is unknown. The recently described MCU complex of proteins has provided a new insight into the role of calcium as a regulator of mitochondrial function. Whether the different mitochondria populations have different calcium handling capacity and whether mitochondria Ca 2+ has a role in energy transmission along the mitochondria network are intriguing issues that emerge when studying the link between electrical stimulation of the muscle fiber and the mitochondria metabolic output.