Mitochondria are responsible for the energy supply to muscle cells during exercise. Despite this importance, they occupy a surprisingly low fractional volume within the cell. However, mitochondrial content is known to increase as an adaptation to repeated bouts of exercise. This adaptation has multiple health benefits for the cell, which include more efficient fat metabolism, reduced provocation of cell death signals, and lower reactive oxygen species production. Many of these beneficial changes have been well studied for many years, and this knowledge is now commonly found in undergraduate exercise physiology textbooks. Less well established are the cellular mechanisms involved in expanding the mitochondrial network within muscle cells in response to exercise. Many studies have examined the steps involved in mitochondrial synthesis at the level of gene transcription, mRNA and protein levels. In contrast, in this study we sought to investigate whether the "assembly" of mitochondrial components was accelerated by exercise. We evaluated the import and accumulation of Tom40, a protein which resides in the outer membrane, and followed the time course of its localization. Mitochondria obtained from muscles that had undergone a period of chronic contractile activity (i.e. "exercise") for seven days displayed a faster rate of incorporation of Tom40 into its large, outer membrane protein complex, where it normally resides and functions. This was accompanied by exercise-induced increases in the levels of proteins which help to transport Tom40 into the organelle. Thus, we conclude that one of the ways in which regular exercise leads to an increase in mitochondrial volume within muscle cells is via the accelerated assembly of protein complexes. In our next study, we will try to determine what signals and proteins are involved in this assembly process.

Reference: Joseph AM, **Hood DA**. Plasticity of TOM complex assembly in skeletal muscle mitochondria in response to chronic contractile activity. Mitochondrion. 2012 Mar;12(2):305-12.

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