Skeletal muscle allows our body to move and perform everyday activities. For movement to occur, our muscles need energy (ATP) and this is provided by special organelles called mitochondria. When we exercise on a consistent basis, our muscles adapt by generating more mitochondria. The production of mitochondria requires a coordinated series of signalling events by many proteins. Precisely what proteins are involved in the signalling sequences to create mitochondria remains as an incomplete picture. Our study examined the role of a protein complex called mTORC1 during the production of mitochondria by exercise. Using an isolated cell culture model of muscle we pharmacologically inhibited mTORC1 to ascertain if mitochondrial content would still increase with exercise when this protein complex was not active. Multiple indices of mitochondrial content indicated that exercise-induced increases in mitochondrial content were not affected with inhibition of mTORC1. However, in muscle that was not exposed to exercise, mTORC1 inhibition caused decrements in the ability of the mitochondria to function, likely impacting ATP production. With the addition of exercise, this functional deficit of the mitochondria was attenuated despite the drug-induced inactivity of mTORC1. Our results suggest that mTORC1 may not be integral to the synthesis of mitochondria with exercise, but that it is required for maintaining mitochondrial function in resting muscle.

Reference: Carter HN, Hood DA. Contractile activity-induced mitochondrial biogenesis and mTORC1. Am J Physiol Cell Physiol. 2012 Jun 13.

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