Mitochondria are the organelles which provide energy for cell survival. Numerous studies have indicated that mitochondrial content in muscle of aging individuals is either reduced or dysfunctional. We undertook this study to investigate some of the mechanisms associated with mitochondrial assembly in muscle from old and young animals. We hypothesized that steps in the process of mitochondrial assembly would be impaired in muscle from old animals, compared to young animals, and that exercise in the form of chronic stimulation-induced contractile activity would attenuate the deficit found in old muscle.

Our results did not confirm a reduced mitochondrial assembly in old muscle. To our surprise, we found that the assembly of a large multi-subunit protein complex in the outer membrane was actually elevated in muscle of old, compared to young, animals. This may represent some form of cellular compensation in an attempt to maintain mitochondrial content in old animals, but the mechanism of this remains unknown. Chronic exercise elevated the rate of assembly in muscle of both old and young animals, but the effect of exercise was attenuated in the muscle of the old animals. Thus, exercise is effective as a treatment to improve the processes of mitochondrial assembly under conditions where mitochondrial content is reduced. However, the assembly process does not appear to be the cause of the reduced mitochondrial function and/or content evident in muscle of old animals.

Joseph AM, Ljubicic V, Adhihetty PJ, Hood DA. **Biogenesis of the mitochondrial Tom40 channel in skeletal muscle from aged animals and its adaptability to chronic contractile activity.** Am J Physiol Cell Physiol. 2010 Jun;298(6):C1308-14.

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