

Health & Physiology

Exercise helps restore aged muscles

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Much like parts on an older car become rusted and need to be replaced, muscle cells can accumulate damage which may contribute to declines in function with age. But instead of going to a mechanic, a trip to the gym may stimulate the replacement of damaged parts. Proteins are the functional components of cells and are continually made and degraded, collectively termed protein turnover. The aging process appears to decrease the turnover rate of proteins, possibly leading to the accumulation of damaged proteins. In particular, the mitochondria are the "power plants" of cells, being also the primary energy-producing source in muscle cells. Their ability to produce energy is decreased with aging and may contribute to decreases in muscle function with age that affects so many adults.

We studied how exercise training improves mitochondria and determined if the processes are different between younger and older people. Our approach included three types of exercise training programs to determine potential similarities between different types of training. Younger (~25 years old) and older adults (~70 years old) were randomized to perform 3 months of either aerobic, resistance or combined training. A sedentary group did not include any structured exercise. We collected muscle samples before and after training and performed functional measurements of exercise and mitochondrial capacity. Regulatory pathways were identified from muscle samples including identifying the specific types of genes and proteins that were activated.

The aerobic training plan was 3 days per week of

high-intensity intervals (termed HIIT), a form of stationary biking that involves repeating short bursts of hard pedaling followed by rest, and 2 days per week of walking on a treadmill. The resistance training group lifted weights for 4 days per week. The combined group performed a combination of 30 minutes of biking at a moderate pace and 30 minutes of weight lifting each day.

The primary finding was that exercise training, particularly aerobic training with HIIT, increased mitochondria in both age groups, particularly in the pathways regulating the making of new mitochondria. These pathways included increases in the ribosomes, which are the protein machinery involved with making new proteins, and the rate at which proteins were being made. The no-exercise control group had an increase in damaged proteins while the aerobic and resistance training groups did not. Such findings emphasize that exercise can remodel muscle cells and may help protect against the accumulation of damaged proteins.

An important public health consideration is that older adults responded very well to aerobic training with HIIT with robust gains in aerobic capacity and mitochondria. Exercising at higher intensities is an important consideration and helps increase the number of people who have benefits from exercise.

An intriguing finding was that older adults had similar absolute improvements to the younger group, which resulted in some gains being greater relative to the lower starting capacity of older adults. Such results support that older people retain the ability to adapt to exercise.



It has been known since the 1960's that exercise is a powerful stimulus to increase the mitochondrial capacity. Much research has helped determine the pathways that regulate how exercise stimulates adaptations to mitochondria including identifying specific genes that are turned on with exercise. But there are still gaps in knowledge about why certain genes are turned on and understanding if those genes will eventually be used to make new proteins. Our approach included multiple approaches to compare if the changes in proteins had much overlap with changes to genes. The results indicate that exercise adaptations to the mitochondria appear to be regulated primarily in the processes involved with making new proteins. The new proteins appear to have less damage and may help improve muscle function with age, much like a few replacement parts can help an older car run like new again.

