

Improvements in aerobic performance, endurance and metabolism are benefits that come from regular endurance exercise training. These adaptations are brought about by changes that take place with the activation of molecules within each muscle cell. This includes the activation of multiple proteins which control cell signaling and communication pathways upon exposure to exercise. A better understanding of the communication system that gets turned on with an acute bout of exercise is imperative for the future of exercise as a therapeutic tool in treating conditions like obesity, type 2 diabetes, cardiovascular disease, and certain types of cancer.

p53 is a well-known protein in cancer research, because it normally serves to suppress cancer development. Typically, p53 is located inside the nucleus of the cell, where it can bind to the DNA and promote the expression of genes. We have shown previously that p53 also resides inside the cell's energy production center, the mitochondrion, where it appears to affect the very small amount of DNA found there, called mitochondrial DNA (mtDNA). In fact, exercise promotes the movement of p53 to the mitochondria to bind mtDNA.

Animals without p53 have a greater incidence of cancer, and they also have lower aerobic capacities, and cannot exercise nearly as much as animals with an intact p53 protein. This is likely due to decreased mitochondria in the muscle coupled with an impairment of function in the mitochondria that remain. Since p53 is important in maintaining mitochondrial function, and it is also involved in the response to acute exercise, in this study we sought to determine whether the presence of p53 is required for alterations in cell communication. Our data indicate that after a single bout of exercise, lack of p53 protein in the muscle reduces and/or completely abolishes the activation of information signaling vital to translating the benefits of endurance activity into beneficial muscle adaptations.

Since many different diseases, from obesity to cancer, implement exercise as part of the treatment strategy to improve health, it is important for us to understand what cellular changes are taking place with the onset of exercise. Here, we identify p53 to be a necessary protein for conveying cellular signals during endurance exercise. This finding enhances our understanding of the cellular communication system within muscle cells, and brings to light a specific protein that is necessary for promoting the positive benefits of exercise. When implementing exercise programs, consideration of these findings may assist in tailoring the exercise dosage to optimize the resulting adaptations. This information also helps us understand one potential reason for the greater fatigability and lesser endurance in cancer patients.

Reference: Saleem A, Carter HN, Hood DA. [p53 is necessary for the adaptive changes in cellular milieu subsequent to an acute bout of endurance exercise.](#) Am J Physiol Cell Physiol. 2014 Feb;306(3):C241-9.

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