Regular endurance exercise training increases the oxidative capacity of skeletal muscle by producing an increase in mitochondrial content. The transcriptional coactivator protein, PGC-1α, is an important regulator of mitochondrial content in skeletal muscle. Many experiments have demonstrated that exercise increases PGC-1α expression and leads to subsequent up-regulation of mitochondrial proteins. Coordinated changes in the expression of PGC-1α and mitochondrial proteins suggest that PGC-1α may be required for exercise-induced mitochondrial biogenesis in skeletal muscle.

We performed this study to determine the role of PGC-1α in producing mitochondrial adaptations in skeletal muscle cells, particularly in response to the stress of imposed exercise. We hypothesized that there would be a reduced mitochondrial adaptive response to contractile activity in cells depleted of PGC-1α.

Our data indicate that PGC-1α is necessary for most of the mitochondrial adaptations that occur with exercise, as tested using a muscle cell culture model of chronic contractile activity. However, there are additional pathways which function with PGC-1α to mediate the elevated expression of specific proteins which are vital for mitochondrial function. These data help us to understand the cellular processes governing exercise-induced mitochondrial biogenesis.

Reference: Uguccioni G, **Hood DA**. The importance of PGC-1α in contractile activity-induced mitochondrial adaptations. Am J Physiol Endocrinol Metab. 2011 Feb;300(2):E361-71.

View this article [PDF].