Mitochondria are small structures found in almost all human cells, which produce energy through chemical reactions in order to “power” the cell. Different types of cells contain different numbers of mitochondria, depending on their energy needs. For example, liver and skeletal muscle cells may each contain hundreds or thousands of mitochondria, while red blood cells have none. Exercise is known to increase the number and activity of mitochondria in the skeletal (voluntarily controlled) muscles. Recent research has shown that exercise may also increase the number and activity of mitochondria in tissues other than skeletal muscle, such as adipose (fat), liver, brain, and kidney tissues. Since mitochondrial dysfunction is tied to several different disorders in these tissues (such as type 2 diabetes and obesity), it is important to understand not only what impact exercise has on mitochondria number and activity, but also what chemical signals tell the body to produce more mitochondria.

In this article, the authors reviewed existing research on the impacts of exercise on the production of mitochondria in skeletal muscle tissues. Since most of the research on mitochondrial production in tissues other than skeletal muscle has been done on adipose (fat) tissue, the authors considered adipose tissue as a model for other organ tissues. Finally, a brief note was made regarding research concerning liver, brain, and kidney tissues.

Keywords:
Exercise, muscle, liver, kidney, brain, fat, mitochondria, energy

Like skeletal muscle, fat tissue increases the activity and number of mitochondria (the cell’s energy producers) in response to chemical signals associated with exercise. Although more research is needed on brain, liver, and kidney tissues, exercise may be useful in treating disorders associated with mitochondrial dysfunction. This research may also explain why increased exercise leads to overall better health.
What did the researchers find?

In skeletal muscle, high calcium concentrations and low energy levels following exercise trigger the production of the protein PCG-1α, which increases the production of mitochondrial proteins. Like in skeletal muscle, adipose tissue responds to exercise by increasing the number and activity of mitochondrial proteins via the protein PCG-1α. In adipose tissue, different chemical signals related to exercise – such as the waste product lactate and the hormones epinephrine, glucagon, and IL-6 – may be responsible for this effect. Increased mitochondrial activity in response to exercise has also been observed in brain and kidney tissues, although there is mixed evidence regarding liver tissue. More research is needed to understand how exercise affects brain, kidney, and liver tissues and to identify the chemical signals that bring about these changes.

How can you use this research?

Physicians can use this research to understand how exercise may be used to help treat disorders related to mitochondrial dysfunction.

Physiologists can further this research by conducting more detailed studies of the impact of exercise on mitochondrial activity in brain, liver, and kidney tissues, and identify the chemical signals responsible for these changes.

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