

GH Increases Mitochondria Activity

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in GH is associated with increased risk of death and enhanced fat mass. There is some evidence that GH action may be important for mitochondrial regulation in skeletal muscle. Mitochondria are sometimes described as "cellular power plants" because they generate most of the cell's supply of adenosine triphosphate (ATP), used as a source of chemical energy. In addition to supplying cellular energy, mitochondria are involved in a range of other processes, such as signaling, cellular differentiation, cell death, as well as the control of the cell cycle and cell growth. Loss of mitochondria has been implicated in several human diseases and may play a role in the aging process. Exercise is a potent stimulus of GH release, and thus it is possible that GH action contributes too many exercise induced mitochondrial adaptations, including increased mitochondrial creation and fat utilization.

Researchers from the Rochester, MN Mayo Clinic School of Medicine investigated the effects of GH on mitochondrial activity, protein synthesis, and fat utilization in young adults. They infused GH (150 microg/h) for 14 h on separate days, and muscle biopsies were obtained. They found a 14-h infusion of GH causing physiological elevation of GH

resulted in increased skeletal muscle mitochondrial oxidative capacity, as shown by increased mitochondrial ATP production rate. Plasma fatty acid concentration was nearly doubled during GH infusion compared with saline and was accompanied by a shift in fuel metabolism toward greater reliance on fat. GH had no effect on muscle protein synthesis. Interestingly, the lack of effect of GH on muscle protein synthesis rate occurred despite an increased activation (phosphorylation) of mTOR, a key energy sensing anabolic signal molecule. The age related decrease in GH which causes reduced mitochondrial activity may be the reason why low GH levels are associated with increased risk of dying.

Short KR, Moller N, Bigelow ML, Coenen-Schimke J, Nair KS. Enhancement of muscle mitochondrial function by growth hormone. *J Clin Endocrinol Metab.* 2008 Feb;93(2):597-604.