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Metformin and caloric restriction induce an AMPK-dependent restoration of mitochondrial dysfunction in fibroblasts from Fibromyalgia patients.

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Abstract

Impaired AMPK is associated with a wide spectrum of clinical and pathological conditions, ranging from obesity, altered responses to exercise or metabolic syndrome, to inflammation, disturbed mitochondrial biogenesis and defective response to energy **stress**. **Fibromyalgia** (FM) is a world-wide diffused musculoskeletal chronic pain condition that affects up to 5% of the general population and comprises all the above mentioned pathophysiological states. Here, we tested the involvement of AMPK activation in fibroblasts derived from FM patients. AMPK was not phosphorylated in fibroblasts from FM patients and was associated with decreased mitochondrial biogenesis, reduced oxygen consumption, decreased antioxidant enzymes expression levels and mitochondrial dysfunction. However, mtDNA sequencing analysis did not show any important alterations which could justify the mitochondrial defects. AMPK activation in FM fibroblast was impaired in response to moderate **oxidative stress**. In contrast, AMPK activation by metformin or incubation with serum from caloric restricted mice improved the response to moderate **oxidative stress** and mitochondrial metabolism in FM fibroblasts. These results suggest that AMPK plays an essential role in FM pathophysiology and could represent the basis for a valuable new therapeutic target/strategy. Furthermore, both metformin and caloric restriction could be an interesting therapeutic approach in FM.

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KEYWORDS: AMPK; Caloric restriction; **Fibromyalgia**; Metformin; Mitochondria; **Oxidative stress**

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