Vitamin B3 revitalizes energy metabolism in muscle disease

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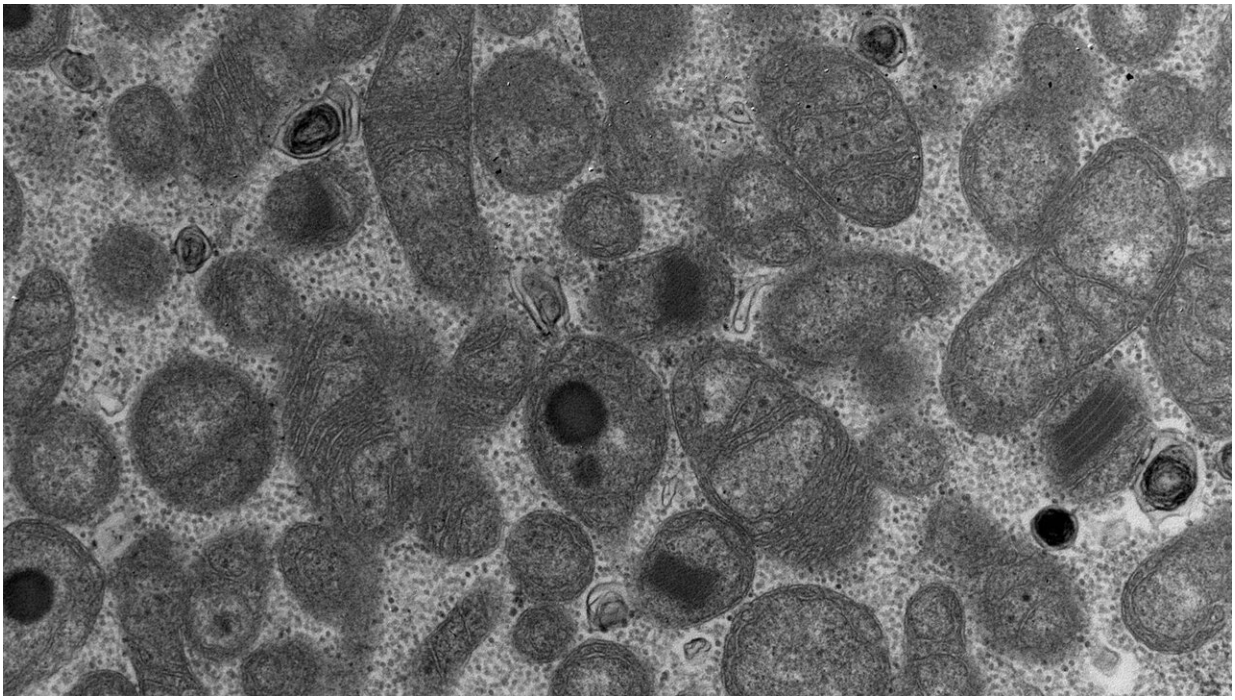

Electron micrograph of the muscle of a patient with mitochondrial disease. Credit: Eija Pirinen

An international team of scientists, led by University of Helsinki reported that vitamin B3, niacin, has therapeutic effects in progressive muscle disease. Niacin delayed disease progression in patients with mitochondrial myopathy, a progressive disease with no previous curative treatments.

Vitamin B3 forms have recently emerged as potent boosters of energy metabolism in rodents. These vitamins are precursors for NAD^+, a molecular switch of metabolism between fasting and growth modes.

As fasting has been shown promote health and longevity in for example mice, a variety of "NAD boosters" are being developed. However, whether actual NAD^+ deficiency exists in human disease, and whether NAD^+ boosters could have curative effects in patients with degenerative diseases, has remained elusive.

In the current publication, a collaborative team of investigators led by academy professor Anu Suomalainen-Wartiovaara and academy research fellow Eija Pirinen report lowered NAD^+ levels in both blood and muscle of mitochondrial myopathy patients.

"The disease is characterized by progressive muscle weakness, exercise intolerance and cramps. Currently, no treatments that would slow down disease progression exist," says Suomalainen-Wartiovaara.

Niacin—a promising treatment option

Pirinen and colleagues report that niacin treatment efficiently increased blood NAD^+ both in patients and healthy subjects. Niacin restored NAD^+ in the muscle of the patients to the normal level and improved strength of large muscles and mitochondrial oxidative capacity. Overall metabolism shifted towards that of normal subjects.

The results of this open pilot study revealed that niacin is a promising treatment option for mitochondrial myopathy. The authors emphasize, however, that niacin and NAD^+ are efficient metabolic modifiers and niacin treatment should be cautiously applied only, when NAD deficiency is detected for example in the patient's blood.

"Our results are a proof-of-principle that NAD^+ deficiency exists in humans and that NAD^+ boosters can delay progression of mitochondrial muscle disease," Suomalainen-Wartiovaara comments.

"The study is a significant leap in the development of targeted therapy options for energy metabolic diseases," Suomalainen-Wartiovaara continues.

More information: Eija Pirinen et al. Niacin Cures Systemic NAD+ Deficiency and Improves Muscle Performance in Adult-Onset Mitochondrial Myopathy, *Cell Metabolism* (2020). DOI: 10.1016/j.cmet.2020.04.008

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