Antioxid Redox Signal. 2013 Nov 20;19(15):1855-60. doi: 10.1089/ars.2013.5346. Epub 2013 May 29.

Could mitochondrial dysfunction be a differentiating marker between chronic fatigue syndrome and fibromyalgia?

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Abstract

Chronic fatigue syndrome (CFS) and fibromyalgia (FM) are complex and serious illnesses that affect approximately 2.5% and 5% of the general population worldwide, respectively. The etiology is unknown; however, recent studies suggest that mitochondrial dysfunction has been involved in the pathophysiology of both conditions. We have investigated the possible association between mitochondrial biogenesis and oxidative stress in patients with CFS and FM. We studied 23 CFS patients, 20 FM patients, and 15 healthy controls. Peripheral blood mononuclear cell showed decreased levels of Coenzyme Q10 from CFS patients ($p<0.001$ compared with controls) and from FM subjects ($p<0.001$ compared with controls) and ATP levels for CFS patients ($p<0.001$ compared with controls) and for FM subjects ($p<0.001$ compared with controls). On the contrary, CFS/FM patients had significantly increased levels of lipid peroxidation, respectively ($p<0.001$ for both CFS and FM patients with regard to controls) that were indicative of oxidative stress-induced damage. Mitochondrial citrate synthase activity was significantly lower in FM patients ($p<0.001$) and, however, in CFS, it resulted in similar levels than controls. Mitochondrial DNA content (mtDNA/gDNA ratio) was normal in CFS and reduced in FM patients versus healthy controls, respectively ($p<0.001$). Expression levels of peroxisome proliferator-activated receptor gamma-coactivator 1-alpha and transcription factor A, mitochondrial by immunoblotting were significantly lower in FM patients ($p<0.001$) and were normal in CFS subjects compared with healthy controls. These data lead to the hypothesis that mitochondrial dysfunction-dependent events could be a marker of differentiation between CFS and FM, indicating the mitochondria as a new potential therapeutic target for these conditions.

PMID: 23600892 [PubMed - indexed for MEDLINE]