NLRP3 inflammasome is activated in fibromyalgia: the effect of coenzyme Q10.

Cordero MD, Alcocer-Gómez E, Culic O, Cañón AM, de Miguel M, Díaz-Parrado E, Pérez-Villegas EM, Bullón P, Battino M, Sánchez-Alcazar JA.

Abstract

AIMS: Fibromyalgia (FM) is a prevalent chronic pain syndrome characterized by generalized hyperalgesia associated with a wide spectrum of symptoms such as fatigue and joint stiffness. Diagnosis of FM is difficult due to the lack of reliable diagnostic biomarkers, while treatment is largely inadequate. We have investigated the role of coenzyme Q10 (CoQ10) deficiency and mitochondrial dysfunction in inflammasome activation in blood cells from FM patients, and in vitro and in vivo CoQ10 deficiency models.

RESULTS: Mitochondrial dysfunction was accompanied by increased protein expression of interleukin (IL)-1β, NLRP3 (NOD-like receptor family, pyrin domain containing 3) and caspase-1 activation, and an increase of serum levels of proinflammatory cytokines (IL-1β and IL-18). CoQ10 deficiency induced by p-aminobenzoate treatment in blood mononuclear cells and mice showed NLRP3 inflammasome activation with marked algesia. A placebo-controlled trial of CoQ10 in FM patients has shown a reduced NLRP3 inflammasome activation and IL-1β and IL-18 serum levels.

INNOVATION: These results show an important role for the NLRP3 inflammasome in the pathogenesis of FM, and the capacity of CoQ10 in the control of inflammasome.

CONCLUSION: These findings provide new insights into the pathogenesis of FM and suggest that NLRP3 inflammasome inhibition represents a new therapeutic intervention for the disease.
