Abstract

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The role of mitochondrial dysfunctions due to oxidative and nitrosative stress in the chronic pain or chronic fatigue syndromes and fibromyalgia patients: peripheral and central mechanisms as therapeutic targets?

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Abstract

INTRODUCTION: Chronic fatigue syndrome (CFS) and **fibromyalgia** (FM) are characterized by persistent pain and fatigue. It is hypothesized that reactive oxygen species (ROS), caused by **oxidative** and nitrosative **stress**, by inhibiting mitochondrial function can be involved in muscle pain and central sensitization as typically seen in these patients.

AREAS COVERED: The current evidence regarding **oxidative** and nitrosative **stress** and mitochondrial dysfunction in CFS and FM is presented in relation to chronic widespread pain. Mitochondrial dysfunction has been shown in leukocytes of CFS patients and in muscle cells of FM patients, which could explain the muscle pain. Additionally, if mitochondrial dysfunction is also present in central neural cells, this could result in lowered ATP pools in neural cells, leading to generalized hypersensitivity and chronic widespread pain.

EXPERT OPINION: Increased ROS in CFS and FM, resulting in impaired mitochondrial function and reduced ATP in muscle and neural cells, might lead to chronic widespread pain in these patients. Therefore, targeting increased ROS by antioxidants and targeting the mitochondrial biogenesis could offer a solution for the chronic pain in these patients. The role of exercise therapy in restoring mitochondrial dysfunction remains to be explored, and provides important avenues for future research in this area.

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