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Oxidative stress and fibromyalgia: A biomechanical/metabolic approach

Paul C Breeding

Blue Hole Chiropractic & Wellness, USA

The majority of patients with fibromyalgia have dyspepsia, irritable bowel Syndrome and other gastrointestinal problems. Chronic infections of the gut have been shown to trigger the production of reactive oxygen species and reactive nitrogen species to eliminate the reservoir of pathogens in the gut. Dysfunction of the gut- brain axis has been shown to trigger neuropathic pain and other symptoms including insomnia, poor memory and concentration, mood swings, chronic fatigue syndrome and fibromyalgia. Other sources of infection include the mouth, sinus cavities and urogenital system. The activation of reactive oxygen species and reactive nitrogen species in these systems activates immune and inflammatory pathways by initially switching on the cytokine, interleukin 1 beta and inhibiting its receptor antagonist. Interleukin 1 beta activation converts lysophosphatidylcholine (LPC) to lysophosphatidic acid (LPA) which is the critical initiator of neuropathic pain, demyelination and pain related protein expression. Other powerful neurotoxins include quinolinic acid and kyurenic acid. To assist in the management of fibromyalgia, tests may be conducted to identify metabolic imbalances. Hair and blood may be analyzed for the presence of heavy metals. Urine and blood may be tested for total antioxidant capacity, superoxide dismutase and glutathione peroxidase and the levels of lipid peroxides. A separate organic acids test measures 76 different urine metabolites including yeast markers, bacterial markers, oxalate metabolites, glycolytic cycle metabolites, mitochondrial markers, neurotransmitter metabolites and nutritional markers. Two additional tests involve the pentose-phosphate pathway which is the energy source for myelin repair. When this pathway is activated, it is associated with elevation of levels of Heat Shock Protein 27 and ATM Kinase. Treatment initially involves a biomechanical approach focusing on spinal biomechanics that include the identification of spinal joint fixations and areas of facet hypermobility. Manual adjustment is used to correct these spinal joint fixations followed by exercises to strengthen the spinal muscular stabilizers to reduce facet hypermobility. Nutritional therapies are also implemented to address underlying metabolic errors identified through testing.

Drpaulcbreeding@yahoo.com

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