



HOLTORF MEDICAL GROUP, INC.

CENTER FOR HORMONE IMBALANCE, HYPOTHYROIDISM AND FATIGUE

23456 Hawthorne Blvd. Suite160, Torrance, CA 90505 Tel: 310-375-2705 Fax: 310-375-2701

Hypercoagulable State and Chronic Fatigue Syndrome and Fibromyalgia

NEW RESEARCH IS INDICATING that many chronic fatigue syndrome (CFS) and fibromyalgia (FMS) patients are in a state of low level activation of the coagulation system and treatment of this coagulation activation can result in a complete or partial resolution of symptoms. Studies have found that approximately 80% of CFS and FM patients have this low level activation of the clotting system. This low level activation does not produce a blood clot, but rather an intermediate substance called a soluble fibrin monomer (SFM). This coats the inside of the blood vessel and limits oxygen and nutrient flow into the cells, resulting in the symptoms of CFS and FMS including fatigue, muscle pain, brain fog and sleep disturbances. It has been found that 40% of CFS/FMS patients have a genetic predisposition for the production of too much SFM, while 50% have a genetic predisposition that limits the breakdown of the SFM. Both conditions result in excessive SFM coating of the blood vessels.

This genetic predisposition can be set into action by a number of factors, including trauma, exposure to heavy metals, toxins pesticides and molds and viral, yeast and bacterial infections, including, Epstein Bar Virus (EBV), Cytomegalovirus (CMV), HHV6, Parvovirus, Enterovirus, Mycoplasma, Chlamydia Pneumonia and Lyme's disease. The SFM coating not only limits the oxygen and nutrient flow, but it also provides a place for the virus, yeast and bacterial to "hide" and escape destruction by the immune system. Thus, it is very difficult for CFS/FMS patients to rid the body of these infections when compared to healthy individuals.

Diagnosis is made by the use of a specialized test called an ISAC (Immune System Activation of Clotting) panel, which measures platelet activation, soluble fibrin monomer, fibrinogen, prothrombin fragment 1 +2 and thrombin/antithrombin complexes. Treatment includes low dose heparin and substances to break up the fibrin as well as elimination of the initiating agent, whether it is a virus, bacteria, yeast or toxin. Intervention can be from several weeks to a number of months. We utilize this treatment at the Hormone and Longevity Medical Center, Inc and have seen some dramatic improvements and sometimes complete resolution of symptoms.