REVIEW OF THE ONLINE PUBLICATION FROM THE HOURNAL OF EXPERIMENTAL BIOLOGY AND MEDICINE --June 5, 2008

High plasma levels of MCP-1 and eotaxin provide evidence for an immunological basis of fibromyalgia. Exp Biol Med (Maywood). 2008 Jun 5. Zhang Z, Cherryholmes G, Mao A, Marek C, Longmate J, Kalos M, St Amand RP, Shively JE. City of Hope. PMID: 18535166

The report of the research study from the City of Hope (Duarte, California) described new findings in some of our patients with Fibromyalgia. This very technical paper can be read on line (www.ebmonline.org) but I will try to simplify it. My summary will omit some fascinating and pertinent material in its simplification.

The function of our genes is to dictate the formation of proteins throughout the body. You know these proteins as enzymes, hormones, antibodies, cell structures and so on. Our study examined twenty-five circulating proteins known as cytokines or chemokines. We found that several were abnormally elevated in the blood of patients as well as in some of their family members (with and without fibromyalgia) when compared with normal controls.

Two proteins were most prominently elevated. They are known as eotaxin and MCP-1 (monocyte chemotactic protein-1). When tested in tandem, the elevations correctly identified fibromyalgia in up to 50% of the patients. But, when two other less-prominently abnormal cytokines (four total) were factored into the testing, the diagnostic probability rose to somewhere between 70-80%. These two additional cytochines are: TNF-alpha and IFN-gamma. (tumor necrosis factor alpha and Interferon-gamma)

Interestingly, eotaxin was even higher in patients treated with guaifenesin. MCP-1, on the other hand, showed the same elevated levels in treated or untreated fibromyalgics. The other two cytokines actually dropped down to normal in patients treated with guaifenesin. Obviously, this raises the question whether more eotaxin is needed by fibromyalgics to protect them from something else---such as inflammatory effects of other cytokines. It certainly reflects that guaifenesin has distinct effects on cytochines that have been previously unknown.

This is a very technical research paper that addressed only laboratory findings. It does not describe the feasibility of testing cytokines by an average clinical laboratory. However, the study is ongoing and will certainly produce more data and hone in on what could become mainstream testing for fibromyalgia. Such facts will surely become more relevant as results from the current genetic arm of the study are published. Genetic findings point the way for future biochemical analyses, and this paper did reinforce our stance that the disease is inherited in the vast majority of patients.

Obviously, we are closer to a solution for the complicated aberrant biochemistry and physiology that so thoroughly disrupts the life of fibromyalgics and their families. As Churchill would say, "this is not the end, but it is the beginning of the beginning." It gives credence to our protocol even though its benefits to patients were not part of this project.