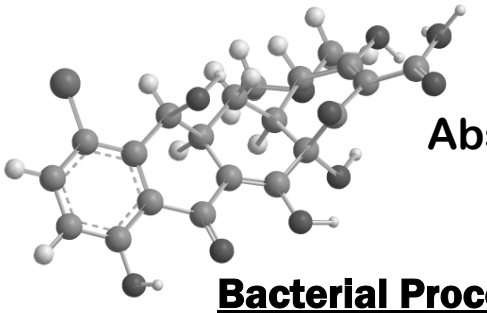

Autoimmunity Research Foundation

Abstract of Poster presented at AACFS 2004

Bacterial Processes Seem Key to CFS Remission

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Studies over the last two decades have described tiny 'pleomorphic' bacteria living within the cells of the immune system. Cantwell (1982) documented tiny, coccoid, acid-fast bacterial forms associated with several 'autoimmune' diseases. He used Fite-Faraco stains, and x1000 optical magnification. In 1989 Wirostko, et al, produced Transmission Electron Micrographs of immune phagocytes containing hundreds of tiny coccoid bacterial forms, each around 0.01 - 0.025 microns in diameter, living in colonies within the cytoplasm of the very cells which are tasked with killing such bacterial parasites. However, subsequent attempts at using conventional antibiotic therapies to kill these bacteria have proven unsuccessful.

We identified that the hormone Angiotensin II (Ang-II), and the steroid hormone 1,25-dihydroxyvitamin-D (1,25-D), were key to the Th1 inflammatory biochemistry. When these hormones were controlled (Ang-II blockaded with Olmesartan, and 1,25-D controlled with careful attention to Solar Exposure and dietary intake), combinations of the antibiotics Azithromycin, Minocycline and Sulfa-Trimeth, were capable of inducing remission in Sarcoidosis. These remissions also resolved a second issue, whether such intra-cytoplasmal bacteria were pathogenic or benign. In the case of Sarcoidosis, the coccoid bacterial forms proved pathogenic. Two recent in-vitro studies have described how a similar species of intracellular bacteria directly modulated the transfer factor Nuclear factor-kappaB (NF-kB) in the cytoplasm, initiating a release of mRNA (signaling cytokine release) from the Nucleus. This is likely to be the pathogenic mechanism, and we have described how this mechanism can fuel the granulomatous inflammation of sarcoidosis, an inflammation which is not driven by normal lymphocyte-phagocyte signaling, but by a steady release of cytokines independent of any lymphocytic intervention.

A clinician can measure the levels of 1,25-D and soluble InterLeukin-2-Receptor (sIL2R) in the bloodstream to assess the relative magnitudes of Th1 and Th2 inflammatory processes. Preliminary data from patients with CFS show levels of 1,25-D at, or beyond, the upper limit of 'normal'. This is indicative of an active Th1 inflammatory process. Initial assays have been as high as 106 pg/ml (compare with the 2-sigma population limit of 47 pg/ml).

The use of Ang-II blockade (with Olmesartan) has resulted in profound symptomatic changes, changes which cannot be explained with reference solely to any hypotensive effect of the Angiotensin Receptor Blocker (ARB). Those CFS patients who have been able to weather the consequent hormonal rebalancing have reported dramatic improvement in energy and cognitive functions, energy, together with a resolution of sleep dysfunction.

Administration of the identical antibiotic protocols proven effective in sarcoidosis, has resulted in further symptomatic improvement of CFS. The authors will present data tracking the progress of the initial group of CFS patients being treated with this ARB/Antibiotic protocol.