

At this point, with Th1 being suppressed, the only defense against being “eaten alive” is the enzyme, Rnase-L. (See below.) Rnase-L cannot kill any of these things. It only stops them from reproducing. According to Dr. Cheney, “It’s a line in the sand saying ‘no more replication,’ and it waits for the Th1 to come and kill them. But Th1 never comes. Rnase-L sits there and grinds away, possibly going up and down as the pathogens activate and reactivate. But they never get wiped out. Rnase-L holds the line, waiting for the cavalry that never arrives.”

In Summary...

The body’s energy is taxed by this constant demand on the immune system. Added to this are other influential factors involving the nervous and endocrine systems (which we do not get into here) that contribute to the energy drain that is so notable in ME/CFS. Tests that measure the levels and activity of Th1 and Th2 cells can help determine the state of a ME/CFIDS patient’s immune function.

Dr. Cheney talks about Rnase-L:
(from *The Three Phases of CFIDS*, by Carol Sieverling)

Let’s start with Phase I illness. A virus usually triggers CFIDS. (A viral-like onset is reported by 70% of all patients.) The specific virus may not really matter. What matters is that RNase-L activity is initiated as a consequence of the viral assault. All viruses stimulate Rnase-L, but some more so than others. The mono viruses [herpes viruses such as EBV, CMV, HHV6] most easily stimulate Rnase-L.

However, the trigger may not be a virus. RNase L is a defense against intracellular organisms, and a few unusual bacteria are intracellular. Therefore some bacteria, like mycoplasma and chlamydia pneumoniae, are possible triggers. Any intracellular organism that stimulates Rnase-L is a possible trigger for CFIDS, which is why the disease can look different among various patients in the beginning.

What does RNase-L do? It is designed to destroy messenger RNA at the cellular level. The good news is that it hits viral messenger RNA, stopping the virus from reproducing. But it also chews up human messenger RNA, inhibiting every enzyme in the body. Hence, CFIDS patients end up on their backs. It is not the virus that makes them sick, so much as it is their body fighting the virus, specifically the Rnase-L activity.

Dr. Cheney had suspicions that this illness did change over time, but it was the discovery of a unique lower weight form of Rnase-L in many CFIDS patients, especially in the first five years of illness, that confirmed his suspicions. (For more on Rnase-L see *The CFIDS Chronicle*, Winter 2000, pg. 5, 1-800-442-3437)

Research indicated that this aberrant form of Rnase-L was up to six times more destructive than the typical form. When Dr. Cheney saw these results, (based in part on blood samples from some of his own patients), the hair on the back of his neck stood up. He had always known that CFIDS patients were very sick, but he didn’t really understand why. The burning question for him had been “Why are these people so sick?” With the discovery of the low weight Rnase-L, the question became “How are these people surviving?”

What is supposed to happen when a healthy person encounters an intracellular organism? The regular form of Rnase-L inhibits viral replication, the immune system revs up and wipes out the virus, then everything down-regulates, and they recover.

But unfortunately that is not what happens in CFIDS. In this illness, the Rnase-L activity shifts to the more destructive lower weight form and does not shut off. It stays activated much longer, resulting in pronounced cellular metabolic dysfunction, which ultimately affects the liver. Liver function declines because the enzymes used by the liver are being creamed by the activity of the aberrant Rnase-L.

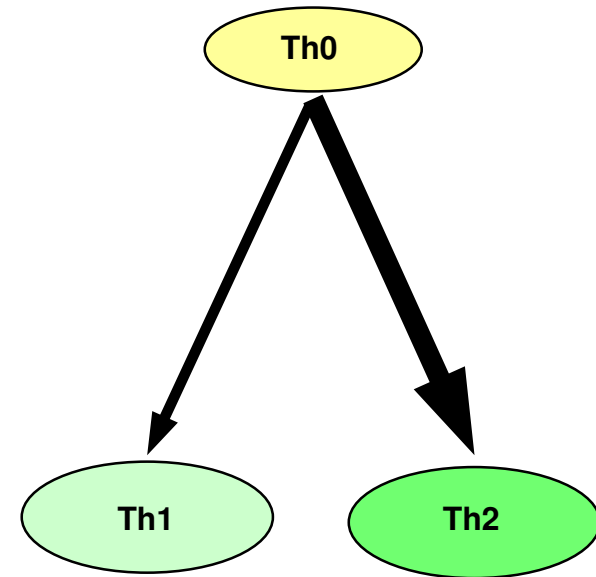
(For more on *The Three Phases of CFIDS*, see <http://virtualhometown.com/dfwcfids/menu.html>)

©2003 Illustrations by Lois Ventura. Based on articles by Carol Sieverling, which in turn are based on a her visit with Dr. Paul Cheney in October, 2000, transcripts of a presentation Dr. Cheney gave at a conference in Orlando in October 1999, and the tape of his seminar in Irving, Texas in May 1999. Dr. Cheney gave her permission to share this information but has not reviewed or edited it. Other articles based on Dr. Cheney’s experiences and data gathered from his practice with CFS patients can be found at <http://virtualhometown.com/dfwcfids/medical/ch Cheney.html>

Over 3000 articles and abstracts can be found at <http://www.immunesupport.com/library/>, including those by Dr. Paul Cheney and other practitioners specializing in ME/CFS & FM (Myalgic Encephalomyelitis/Chronic Fatigue Syndrome & Fibromyalgia).

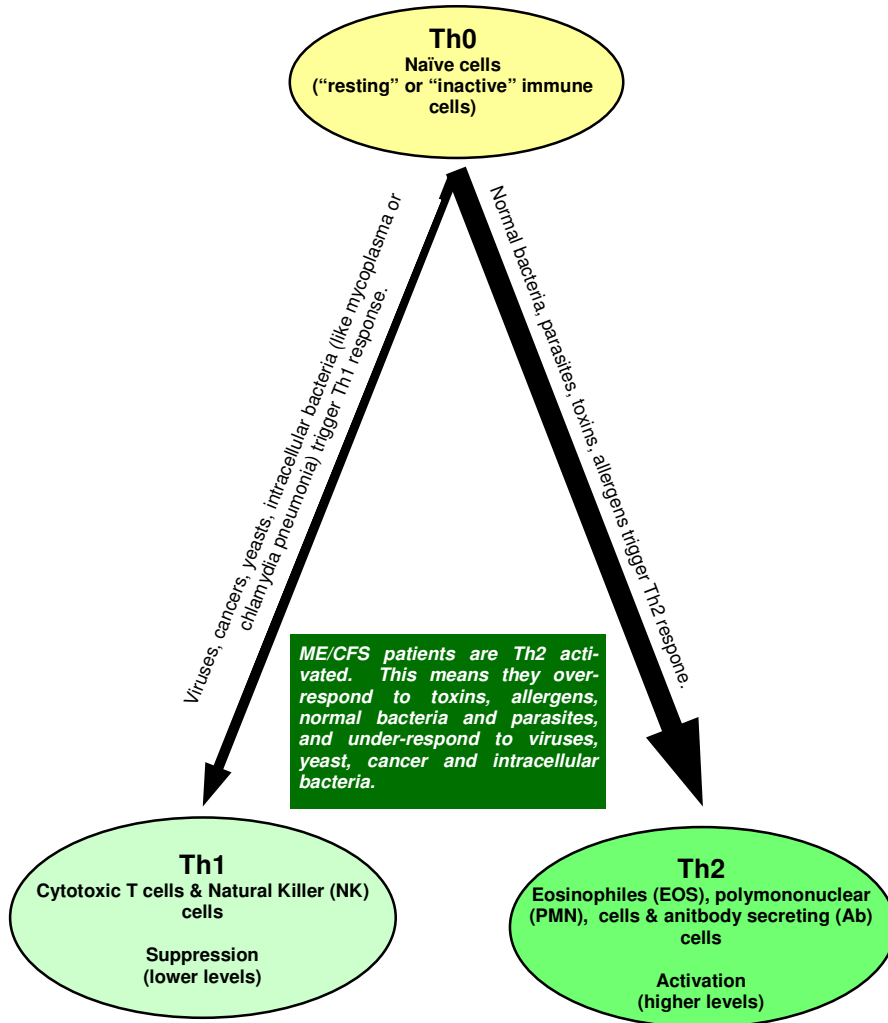
Immune Dysfunction In Myalgic Encephalomyelitis/ Chronic Fatigue Syndrome

A brief overview for patients and families



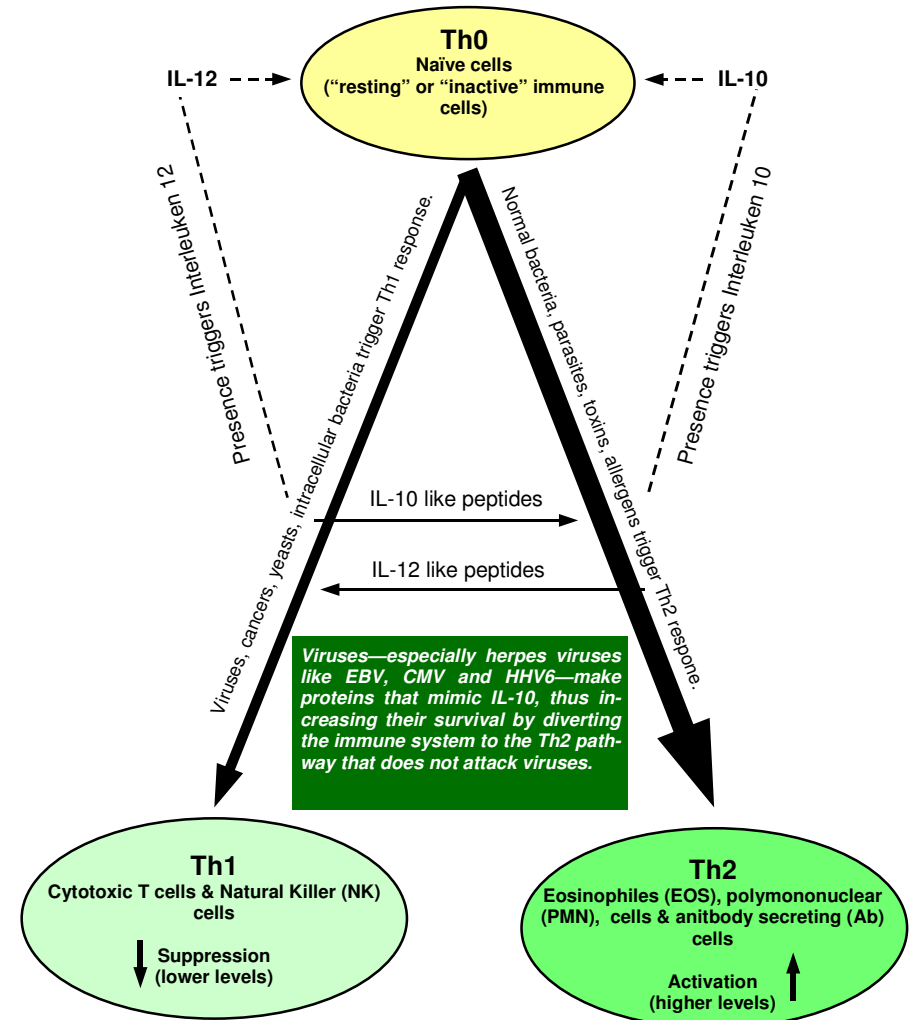
Based on interviews and information presented at seminars by ME/CFS specialist, Paul Cheney, M.D.

Figure 1: Th0 are the naïve, or unformed, cells of the immune system. They are resting, waiting for an invader. When infection occurs, they convert to either Th1 or Th2, depending on the type of threat.



Glossary
 CFIDS—Chronic Fatigue Immune Dysfunction Syndrome (pronounced SEE-fids; synonymous with ME/CFS)
 CMV—cytomegalovirus
 EBV—Epstein-Barr virus
 HHV6—human herpesvirus 6
 Cytokines—small proteins released by cells and have specific effects on other cells
 Enzyme—a protein molecule that catalyzes a chemical reaction
 Peptides—small pieces of proteins
 Pathogen—any disease-producing organism

Figure 2: The naïve Th0 cell chooses its path (Th1 or Th2) depending on the cytokine information it receives. Organisms on the left trigger the cytokine Interleukin 12; organisms and substances on the right trigger the cytokine Interleukin 10.



Researchers have demonstrated that most ME/CFS patients end up stuck in Th2 mode, due to pathogens' ability to deceive and divert the immune system. When the Th2 system activates, it blocks the Th1 system, thus suppressing the Th1 weapons, particularly NK cell function. Accordingly, there is also an increase in Th2 weapons—white cells and antibodies. When a patient is Th2 activated, they no longer have the defense mechanisms to keep dormant, or suppress, things like EBV, Chlamydia pneumonia, CMV, etc. These viruses reactivate, and yeast also begins to appear.

(Continued...)