

“Good Enough to Take It to the Bedside”
American Association for Chronic Fatigue Syndrome
October 8-10, 2004, Madison, Wisconsin
Summary by Paula M. Carnes

Dharam Ablashi, DVM, MS, Dip.Bact., and current president of the American Association for Chronic Fatigue Syndrome stood before us with a small, pleased smile on his face. “This conference is what we wanted to have when we first started the AACFS.” The 2004 conference held in Madison, Wisconsin October 8 -10 included research and clinical presentations. Ablashi stated, “It [research] should be good enough to take it to the bedside.”

Anthony Komoroff, MD, Harvard School of Medicine, began the **conference** with an **overview of current CFS/FM research**. This summary alone was worth the trip to Madison.

- ◆ Reduction of function in CFS patients is substantial as demonstrated by a study using SF 36 scores. The CDC has determined that the total cost to the US each year from productivity losses due to cfs is \$9.1 billion. Cases of CFS followed three years show a relapsing remitting course with slight improvement in symptoms but not in employment over time. There is only a 10% rate of total remission.
- ◆ Studies of the brain in CFS indicate evidence of CNS involvement and hypothalamic-pituitary axis abnormalities. Cognition studies reveal slowed processing of information.
- ◆ Sleep disorders involve non-restorative sleep, sleep apnea, and restlessness. Treatment is of only modest benefit.
- ◆ Studies of immune activation indicate that “activated lymphocytes can pass through the blood-brain barrier in small numbers...and thereby activate lymphocytic and dendritic cells that reside in the brain, particularly microglia and perivascular cells, and this state of low-level activation can last decades. Activated microglia, like macrophages, secrete pro-inflammatory cytokines. (e.g., TNF alpha, IL-1 beta) and NO (nitric oxide).” There is increased neutrophil apoptosis in CFS.
- ◆ The following infections have been found in CFS patients: enterovirus (coxsackie B-like virus), post-Q fever (coxiella burnetii, a rickettsia), post-parvovirus syndrome with elevated IFN gamma, TNF alpha, mycoplasmas (68% of 261 patients in one study).
- ◆ Gene expression studies were done by the CDC. A rheumatology study indicated low vitamin D levels in FMS patients. Omega 3 fatty acid treatment may reduce viral activity and inflammation.

William C. Reeves, MD from the CDC began his presentation of their **new epidemiology studies** by stating clearly that the “fatigue” in chronic FATIGUE syndrome is not just “tired.” It is a fatigue not relieved by rest and includes hurting all over. He then moved on to some startling new information. The CDC study found 235 cases of CFS per 100,000 in their Wichita study. Women are more likely than men to have it, but CFS is more common than breast cancer among women. These details are familiar, but here is a list of surprises.

- ◆ Rural rates of CFS are twice those of urban areas
- ◆ Minorities have the highest risk
- ◆ Lower socioeconomic groups have a much greater risk
- ◆ Eighty percent have a GRADUAL onset
- ◆ No regional differences in the disease are manifest
- ◆ Median duration of the illness is 2-7 years
- ◆ Only 16% of the cases uncovered in the survey are diagnosed
- ◆ Unemployed or cases on disability total 25%
- ◆ Level of disability equals that of patients with major health problems such as COPD

- ◆ The cost in lost productivity (\$9 billion) is equal to WalMart's annual profit margin, or the cost of the hurricane in Miami in 2004. The cost to a family with CFS for one year is \$20,000. This includes the cost of treatment. In the UK it is costing the government \$4 billion to treat CFS patients.

The CDC is working to develop a network system to create more clinical studies of CFS. The next epidemiological study will be in the Atlanta, Georgia area, and will include both urban and rural counties. The location in the CDC area will enable them to do the complex studies efficiently.

“Let me come clean here. I hate tender points,” spoke Dr. Daniel J. Clauw, MD in his **overview of fibromyalgia**. His point was that in FMS pain is everywhere, and there are many other symptoms in addition to pain. The level of pain seems to be related to the level of stress, but stress is not the cause. Patients' brains have become more sensitive to signals of all sorts, sensory, affective, and cognitive. Using functional MRIs, pain levels have been studied indicating that areas of the FMS patient's brain activate at levels far lower than normal healthy individuals. This means that FMS patients are indeed feeling more pain than normal individuals.

These constant levels of pain and stress tend to reveal three groups of patients. One group has low pain and is emotionally stable. One group has high pain and is out of emotional control. The third group is experiencing a high level of pain but is able to remain stable.

Cerebrospinal fluid of FMS patients has high levels of corticotropin releasing hormone (CRH) even after controlling for melancholic depression. Increasing serotonin and norepinephrine levels reduces pain signaling, thus using antidepressants that raise both levels may be useful. Examples of this type of combination drug would be milnacipram and imipramine.

The following **microbiology/immunology papers** indicated possible infection and immune dysregulation in CFS:

- ◆ Robert Suhadolnik, Ph.D. reviewed the evidence and effect of upregulated 2-5A synthetase RNase L antiviral defense pathway. There is a 500 fold excess of bioactive 2-5A. This was determined in a study of 53 CFS patients and 37 controls. The higher the RNase L activity, the lower the patient's ability to function. These patients also have a low molecular weight 37 kDa RNase L which is not found in healthy controls, patients with only depression, or fibromyalgia patients. What are the effects of elevated RNase L activity and low molecular weight RNase L? The patient has lowered signal transduction, lowered cell proliferation, lowered ATP production, lowered cellular metabolism, lowered protein synthesis, impaired natural killer cell function, abnormal exercise response, loss of potassium from muscle, abnormal sodium retention, hyperventilation, central fatigue, sleep disturbances, and muscle cramps and weakness. This probably sounds familiar to many patients.
- ◆ Charles L. Raison, MD from the CDC presented information on the fatiguing effects of interferon alpha on hepatitis C patients. IFN alpha is released by the body early in a viral infection. It causes fatigue and activation of inflammatory TNF alpha, IL 1 and IL 6. When 154 hepatitis C patients were given IFN alpha (ribavirin) for 24 weeks they developed a CFS-like syndrome. On ribavirin 45% of them looked exactly like CFS patients. It was difficult to predict which patients would develop these symptoms, but it became apparent that the ones with the highest fatigue score were the ones unable to clear the virus. Either these patients were not able to clear the virus or they had an increased pro-inflammatory response to IFN gamma. These data suggest a role for viruses and/or antiviral immune responses in fatiguing illnesses including CFS.
- ◆ James F. Jones, MD presented a study of prolonged fatigue following acute infection with mononucleosis, Ross River Virus, and Q fever in Dubbo, Australia. A post infection syndrome occurred in 10% of these patients and was predicted by the severity of the acute infection.

- ◆ Konstance Knox, PhD, presented evidence that CFS patients have reduced signal transducers and activators of transcription, specifically STAT1. This would seem to follow Suhadolnik's study showing lowered signal transduction due to the cleaved low molecular weight RNase L. The conclusion drawn by Knox and Carrigan was "a subpopulation of CFS patients may exist who suffer from an abnormally low STAT1 response to interferons. This immunodeficiency may underlie the increased susceptibility to infections seen in many CFS patients." [However, if one looks at the RNase L research it is just as likely that the STAT1 deficiency is **caused** by infections, especially when one considers that some CFS patients never seem to get other infections such as colds or flu.]
- ◆ Kevin J. Maher, PhD, from the University of Miami, presented evidence that "key proteins associated with the cytolytic process are present at lower cellular concentrations in NK cells from individuals with CFS."
- ◆ Delia Racciatti, MD, PhD summarized a study in Italy of 130 cfs patients. In 66 of the total patients 37.9% had chlamydia trachomatis, 50% had ureaplasma urealyticum, and 12.1% mycoplasma species. The same urogenital infections were detected in 6 sexual partners of the patients. There was often a lack of serological evidence due to a low immune system response. When patients were treated with antibiotics 22 of the 66 patients who were infected had a recovery from "fatigue." "The authors recommended that the diagnostic protocol for rheumatologic diseases and CFS should include investigations for infectious agents of sexually transmitted diseases."
- ◆ Marc Freemont, PhD, from RED Laboratories, Belgium, reported that patients show a genetic susceptibility to immune dysfunction. He commented that Dr. Vojdani is finding the same RNase L dysfunction as Suhadolnik. Some patients have a reduced capacity to mount a normal immune and inflammatory response, while other patients have an increased PKR expression and activation leading to induction of nitric oxide (NO) which furthers the inflammatory response. Symptoms of this would include neuronal reactivity, muscle function, and abnormal vasodilatory response. The thyroid is producing thyroid hormones, but these hormones are not able to interact with cells. PKR is activated by certain infections and chemicals. They are finding mycoplasma infection is high in patients with RNase L dysfunction at 69.4%.

Epidemiology is the sphere of the Center for Disease Control. Dieter Wagner, PhD presented data on the relationship between the Multidimensional Fatigue Inventory (MFI) and the Short-Form 36 (SF36) scales in CFS patients. Both the MFI and the SF36 correlate with fatigue levels in CFS patients.

Ashley Morris, a professor in computer science, has devised a computer list of 26 survey questions to accurately diagnose CFS.

Hemex research on hypercoagulability due to genetic factors was presented by Harold Harrison, MD, PhD. The study compared the ISAC panel of tests for low-level activation of coagulation with the Hereditary Thrombotic Risk Panel data. These data support the general hypothesis of concerted genetic contribution(s) of coagulation protein abnormalities to CFS/FMS and are consistent with family histories.

The next study presented by Rosemary Underhill, MB, presented a pilot study of the role of heredity and environment in CFS. A study involving 219 CFS patients revealed that 20.5% of the patients had family members with CFS. About twice as many were blood relatives. Secondary cases of CFS occurring in the genetically unrelated household members of CFS patients indicates that an infectious agent which can cause CFS may persist in at least some CFS patients and can be shed into the environment. The increased prevalence of CFS in non-household genetic relatives indicates that genetic factors may also be involved in a subgroup of patients.

The **neurophysiology** of CFS is characterized by chronic orthostatic intolerance. Julian M. Steward, MD, PhD, presented evidence of three variants of POTS in CFS patients. In all three there was enhanced thoracic hypovolemia related to inadequate cardiac venous return during orthostasis. One group had a higher than normal resting heart rate, one had lower blood volume, and one had a high blood flow. This typically follows a viral infection.

A study from Japan presented by Hirohiko Kuratsune, MD, D. Med. Sci. revealed that CFS patients had cerebral hypoperfusion in a variety of brain regions. These results suggested that CNS dysfunction might be related to the neuropsychiatric symptoms found in CFS. This cannot be explained by a psychiatric diagnosis.

Jo Nijs, PhD, presented a study from Belgium in which 16 CFS patients performed a bicycle exercise stress test. The level of elastase was the only factor related to the reduction in oxygen uptake, the protein kinase R activity was the principle factor related to the reduction in workload, and the elastase level was the principle factor related to the reduction in % of target heart rate achieved. This is evidence for an association between intracellular immune deregulation and impairments on cardiorespiratory fitness in CFS patients. This study indicates subtle underlying lung damage. Further study is needed. A question was asked following this presentation: How does one reduce elastase? The reply was, "Antibiotics."

The cerebral blood flow (CBF) of healthy sedentary controls was compared to CBF of CFS patients in a study done in Japan and presented by Kazuhiro Yoshiuchi, MD, PhD. Blood flow was shown to be reduced in the temporal regions and the right inferior frontal cortex. Psychiatric status and illness severity did not play a role in this reduced blood flow.

Five **physiology** studies were presented. The first, presented by Susan Levine, MD, was an analysis of metabolic features of CFS. There was a detection of elevated lactate production in 20% of the patients studied. It is thought that some of the symptoms observed among these patients are caused by an accumulation of the potent oxidant, peroxynitrite which produces both nitric oxide and superoxide.

Ulf Hannestad, MSc, described a small study that demonstrated that CFS patients secreted a significantly increased amount of beta-alanine in the urine. Other reports suggest that there is an active transport of beta-alanine and GABA from CNS over the blood-brain barrier. This means that it could be possible that an excretion of abnormal amounts of beta-alanine or GABA in the urine reflects abnormally high concentrations of GABA in CNS. Many symptoms of CFS are seen in patients being treated with antiepileptic drugs. These symptoms increase GABA activity in CNS giving side effects like fatigue, headache, impaired concentration, and muscle weakness.

In an ongoing study presented by Barry Hurwitz, PhD, it was determined that CFS patients have low red blood cell volume. This was more common in women (77%) than men (33%). These patients will be treated with Epoetin Alpha.

An in vitro study from Belgium, presented by Marc Fremont, PhD, showed that cells with the low molecular weight forms of RNase L demonstrate a much higher sensitivity to mercury exposure. This causes an initial membrane depolarization followed by cell death. Alteration of MRP-1 activity by interactions with the ankyrin fragment of RNase L could be a mechanism explaining the high sensitivity of patients to different chemicals, including heavy metals. MRP-1 is also involved in the maintenance of the Th1/Th2 balance.

Martin L. Pall, PhD presented a hypothesis for the disease state found in CFS, MCS and FM. All are reported to produce increases in nitric oxide. The consequent elevation of nitric oxide and its oxidant product, peroxynitrite, is proposed to initiate a biochemical/physiological vicious cycle mechanism that is responsible for the chronic nature of these illnesses. The basic mechanism is local, because mechanisms act at the level of the individual cell and because nitric oxide, superoxide and peroxynitrite have limited diffusion in tissues. This provides an explanation for the symptom variation from one case to another. The variation is largely due to variation in tissue distribution from one case to another.

Angela Lyden, MS, began the **Clinical Program** presentations with a study on 27 FMS patients. Their response to pain, thumbnail pressure and thermal pain was compared to controls. The FMS patients experienced 11 times more pain than controls. The two groups also were evaluated for perceived exertion on exercise bikes. The FMS patients had an increased sense of work level compared to controls. Patient response to pain and exertion correlated well.

A Spanish study on CFS and aerobic exercise, presented by Anna Garcia-Quintana, MD, demonstrated that CFS patients compared to sedentary, healthy controls are 24% lower functioning than the sedentary group. The physically active group of controls reached maximal heart rate values similar to those of the sedentary individuals, while the CFS patients reached much lower values. The average maximal oxygen uptake of the CFS patients on a cycle-ergometer was only 15.2, whereas the sedentary but healthy controls were 25.9, and the physically active controls were 66.6.

A second study from the same clinic in Barcelona, presented by Jose Alegre-Martin, PhD, evaluated 350 subjects for CFS definitional symptoms. Some of these results were startling and fit in with the CDC information from Kansas.

- ◆ Patients were predominantly women 85% and the mean age was 40
- ◆ Predominant symptoms were weakness, muscle pain, malaise >24 hrs, unrefreshing sleep, joint pain, new, headache, impaired memory concentration
- ◆ Onset was gradual in 50% of patients
- ◆ Only 10% of patients improved, worsening was seen in 53%
- ◆ Only 33% of patients were able to work

Do support groups help CFS patients? This was a study presented by Fred Friedberg, PhD. The conclusion was that support groups helped patients to legitimize their illness, but were not used to locate physicians.

Jo Nijs, PhD, presented information on how to evaluate a CFS patient for disability. His conclusion was that the associations between either exercise capacity parameters, or self-reported disability (i.e. the CFS-APQ total scores or the SF-36 subscale scores), and the current employment status are too weak to predict occupational disability.

Ampligen is the only drug currently being developed especially for CFS. David R. Strayer, MD presented a study of 40 weeks of Ampligen treatment in 234 severely ill CFS patients. Results showed a medically and statistically significant increase in the primary endpoint, exercise treadmill duration, (two-fold improvement) compared to placebo. There were no adverse events related to Ampligen. Hemispherx Biopharma will be applying for FDA drug approval in the near future. No date was given.

Leonard Jason, PhD discussed the case definitions of CFS and ME. His comments can be viewed at <http://condor.depaul.edu/~ljason/cfs>. In conclusion Jason said that we need an empirically derived case definition. It must be objective and based on standardized interviews.

Further information on defining CFS fatigue was presented by James Jones, MD, CDC. Jones covered the causes of fatigue in fatiguing illness. He questioned whether “memory of fatigue/illness alters behavior?” He compared this to PTSD in which flashbacks or memories of an old trauma create the same symptoms of anxiety and fear in a person. He wondered if the immune system might be doing this in CFS. He also questioned CFS patient surveys asking, “What is the patient remembering?” [This was an overview of articles and not a new study. I found it difficult to grasp the significance of this other than to comment that it is difficult for patients to objectively define their fatigue to themselves. Was the point that we may have permanent damage to our memory of fatigue, so that just thinking about how fatigued we were makes us feel tired right now? I may have missed the point.]

Kenny DeMeirleir, MD, PhD, presented “Diagnosis and Management of CFS As an Immuno Vigilance Disorder.” CFS is a host response to initiating multiple factors. Evidence based medicine does not apply to the heterogeneous nature of CFS. Infection and innate immune defects activate the OAS and PKR pathways. DeMeirleir mentioned finding endogenous retroviruses as DeFrias and Urnovitz have found in the past. He compared PKR in MS (down) and CFS (up). DeMeirleir mentioned that the digestive tract is altered by the disease. Bacterial gut infections need to be treated with antibiotics and probiotics. He also mentioned that resistance to the thyroid hormone causes hypothyroidism not detectable in the usual tests, severe fatigue and weight gain. He suggested that heavy metals such as mercury may need to be chelated. His list of therapies include the following: decrease microorganisms, restore intestinal flora, balance hormones, decrease PKR activity, treat metal allergy.

Autonomic dysfunction was reviewed by Dr. Charles Lapp, MD. He stated that a TILT test should be done with no medication, including no vitamins for 24 hours. The person should not be anemic, have diabetes, dehydration as these would cause fainting apart from autonomic dysfunction and invalidate the results. The room should be warm, dark and quiet with no talking. Lapp prefers a passive test without the use of drugs. Heart and blood pressure should be continuously monitored. Most patients will faint within 30 minutes if they have autonomic dysfunction. Treatments consist of extra salt and water, fludrocortisone, Midodrine, or Atenolol. SSRIs may be helpful as may IV erythropoetin.

Dr. David Bell, MD, presented a case study of a 24 year old with acute onset of polyuria and polydipsia, “a compulsive water drinking diagnosis.” This patient’s plasma blood volume was normal but the red blood cell mass was 60-70% below normal. Patients having their blood volume measured may appear to be normal if they have been drinking a lot of water. Also IV saline may have a placebo effect in which the blood volume is increased but the red blood cell mass is still abnormally low.

This information led to Dr. Barry Hurwitz’ presentation on an ongoing study of patients taking Erythropoetin to stimulate development of red blood cells. These patients were also given iron and salt tablets as this is needed in order for the Erythropoetin to work. It is of note that a greater sedimentation rate and lower red blood cell volume is an indication of chronic inflammatory conditions. This study has one more year to go.

In the **Doctor to Doctor** session Nancy Klimas, MD presented the CDC hope to develop a network for CFS research among clinical doctors. For example doctors such as Teitelbaum and Lapp claim an 80% success rate. Several say that 80% of their patients improve, but we must do testing and evaluation to determine which treatments are working. Some expressed concern that a network might further limit individual researchers from getting government funding.

This was followed by a question and answer time with a panel. Here are several comments of note:

- ◆ Adult ADD and FMS seem to have similarities. CNS stimulants such as Provigil may give some improvement. (Lapp is seeing 60% improvement. DeMeirleir stated that CFS moms have more children with ADD and autism. He suggested that this may be caused by strep infections and can be treated with penicillin. He said that amphetamines seem to work because penicillin interacts with the same brain receptors when used to treat strep. Natelson spoke up to say that the Provigil trial was negative. Bateman said that in her practice some responded to Provigil, but she prefers Aderol. If the patient had an onset of a flu-like illness it does not work.
- ◆ Klimas mentioned the need for hrt in menopausal women, and another doctor discussed using GHB to treat sleep disorder.
- ◆ Natelson discussed giving a complete neuro exam, in particular a tandem Romberg balance with eyes closed. He commented that peripheral neuropathy can be diagnosed by touching the patient’s toe with piece of cotton. The patient will be unable to feel it.

- ◆ Teitelbaum mentioned that low testosterone can lead to low red blood cell mass.
- ◆ Lapp said that exercise tolerance testing measuring VO2 max is the best way to measure physical ability to do work, and is also a good follow-up to measure improvement. The Ampligen studies indicated that the VO2 max will drop before FX36.
- ◆ Erhlich commented that she is seeing an interaction of strep and Lyme toxins in her Madison, WS patients.
- ◆ Opioids seem to give some patients a wonderful response. This may be explained because the inflammatory cytokines cross the blood/brain barrier. When the opioids block the opioid channel the cytokines cannot get in. Brigita Evengard, MD from Sweden spoke up that Swedish patients are not keen on the meds. They prefer therapy instead. She also commented that borreliosis is endemic in Sweden, and it is seronegative. Pat Fennell stated that this is also a huge problem in upstate New York. This led to some discussion of possible CFS subtypes.
- ◆ Klimas pointed out that we may have no clue how to group subtypes since the immune system tends to shift over time in the course of the disease. Also it is not even clear what rapid onset and slow onset really mean. Natelson seemed to think that illness severity is the key to grouping. Rich VanKonynenberg stated that we do not have a well-posed problem yet. We must look at all the input before we attempt to subgroup patients. The discussion of subgrouping in terms of Lyme was continued in the next presentation.

“Are CFS and Chronic Lyme One and the Same?” asked Dr. Stanley N. Schwartz, MD, director of The Warren Clinic in Tulsa, Oklahoma and current vice-president of the AACFS. Schwartz hastened to state that Oklahoma did not have many cases of Lyme, but he had been asked to overview the existing literature to see how likely there was to be an overlap of Lyme and cfs. In reviewing the literature Schwartz set four limiting guidelines.

- ◆ Only randomized controlled trials
- ◆ Only observational studies
- ◆ Only expert published opinion
- ◆ Only available in Medline

He added the caveat, “The plural of anecdote is not data.” – Kotsonis
 “Experience without measurement is not data.” – Schwartz

[This did not bode well for my later comment that three of us in my family were now positive for borrelia in a state, South Carolina, where supposedly Lyme is not prevalent. I was to be both anecdotal and from a state not considered a Lyme state. One of the CDC doctors told the conference that Lyme in the southeast is a different strain of borrelia and is a mild disease lasting only three weeks. I did make the point that my particular southeastern version of borrelia was a severe illness that had already lasted at least years.]

Schwartz drew a distinction between late Lyme and chronic Lyme. This distinction was that late Lyme had objective signs and serologic confirmation. Chronic Lyme only had serologic confirmation in some cases and no reliable objective signs. His comments on which serology was reliable are as follows:

- ◆ Reliable: ELISA, western blot, skin culture, synovial fluid PCR
- ◆ Unreliable: urinary antigen assay, borreliacidal antibody, VisE, PCR, immune complex disruption, t-cell proliferative response to borrelia antigens. If the patient had a classic Lyme rash that would be helpful, but one would still be diagnosed with CFS if there was no positive serology.

Schwartz presented four studies. The first, Klempner (N Engl J Med, 2001, Jul 12;345(2):85-92) <http://content.nejm.org/cgi/content/abstract/345/2/85> may indicate that 3 month treatment with antibiotics will not cure Lyme disease, but it does not indicate whether there is such a thing as late Lyme or chronic Lyme. Without a clear transition his presentation moved

from considering if there were such a thing as ongoing chronic borrelia infection to whether longterm antibiotics would treat whatever these patients actually had. The conclusion of the presentation was “Basically an unresolved issue” It was not clear which issue or both were unresolved. A study from Germany by D. Hassler showed that patients did recover with longterm antibiotics, but one of Schwartz’s conclusions was that European borreliosis may be different from US borreliosis.

The final day of presentations involved three speakers on the use of therapy to help patients deal with their illness. The most encouraging presentation was by Elke Van Hoof, PhD, Clinical Psychologist working with Dr. DeMeirleir in Belgium. She takes patients through four steps: 1. Defining the crisis of their illness 2. Stabilization 3. Meaning and restructuring 4 Integration and rehabilitation including graded exercise. She explains what is going on with their illness and always includes the patient’s partner or a friend. She makes sure in follow-up that the patient is following the treatment. The partner is critical to implementing and following treatment. She uses EMDR to treat the patient’s sense of victimization. Her final statement was a joy. Because she works with Dr. DeMeirleir and his patients are recovering she stated, “I don’t have to worry about patients who do not improve.”

There were a large number of **poster presentations**. I will briefly mention only those which suggest treatment options for CFS.

- ◆ Roundworm structures of *c. pulmonis* have been found in CFS patients. These roundworms are endemic to Southeast Asia and in the post Vietnam era with increased world travel these roundworms may have spread to other parts of the world. Patients who wish to get tested for this can contact Lawrence Klapow, PhD, Bioscience, 2841 Creekside Rd. Santa Rosa, CA 95405, 707-546-4101 klapow123@sbcglobal.net
- ◆ Dr. Berg of Hemex presented further research on blood coagulation problems in CFS. This coagulation problem can be caused by various infections and/or genetic predisposition. Treatment involves low dose heparin. For further information contact <http://www.hemex.com>
- ◆ Richard Van Konynenburg, PhD presented a study which indicates glutathione levels are depleted in CFS. It is possible to increase glutathione levels using IV glutathione, sublingual glutathione, and by taking undenatured whey protein.
- ◆ Dr. Ritchie Shoemaker, MD runs the Chronic Fatigue Center in Maryland. He is testing and treating patients for chronic neurotoxins. Information on his treatment can be found at <http://www.chronicneurotoxins.com>
- ◆ T. Wijnhuizen has done studies on B 12 deficiency in CFS. He has developed a sublingual B 12 which delivers the same amount of B 12 as injections. He can be contacted at <http://www.vaakmoe.nl>
- ◆ Trevor Marshall, PhD, developed a treatment protocol for sarcoidosis patients. His research indicates that sarcoidosis and CFS are caused by intracellular pathogens such as borrelia and mycoplasmas among others. These pathogens increase the secosteroid hormone 1,25 D levels in the body in order to avoid detection by the immune system. By using an angiotensin-II blockade and temporarily avoiding vitamin D sources one can lower the D hormone level. Then a low dose, pulsed, appropriate antibiotic is added to aid the immune system in killing the pathogens. For more information visit <http://www.marshallprotocol.com>
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