Viral infections

(a) **HHV-6a virus**
(b) **Enteroviruses**
(c) **Epstein Barr virus & Herpes family viruses, including reactivation of latent herpes viruses (EBV, CMV and HHV6a)**
(d) **Retrovirus - HTLV viruses, HGRV virus, MLV's, JHK virus, HIV Negative AIDS**
(e) **Parainfluenza Virus-5 (PIV-5) & Paramyxovirus**
(f) **JHK virus**
(g) **Paryovirus B-19**
(h) **Genetic Evidence of Infections**
(i) **Borna virus**
(j) **Cryptovirus**
(k) **HERV-K18 virus**
(l) **Stealth Viruses**
(m) **H1N1 virus**
(n) **Other viruses**
(o) **ME outbreaks suggesting role of viruses and other pathogens**

(a) **HHV-6a virus**

- [Chronic Fatigue Syndrome linked to HHV6 virus.](http://example.com)
- [International Conference on Chronic Fatigue Points to Low-grade Viral Infections in Brain (2005)](http://example.com)
- Frequent HHV-6 reactivation in Multiple Sclerosis and Chronic Fatigue Syndrome patients. DV. Ablashi, Journal of Clinical Virology, 2000, 16, 3, 179-19


Levine S (2001), "Prevalence in the cerebro spinal fluid of the following infectious agents in a cohort of 12 CFS subjects: Human Herpes Virus 6 & 8; Chlamydia Species; Mycoplasma Species; EBV; CMV and Coxsackie B Virus", Journal of Chronic Fatigue Syndrome, 9:91-2:41-51


Persistent Active Human Herpesvirus Six (HHV-6) Infections In Patients With Chronic Fatigue Syndrome. AACFS Conference (1998)

Nicolson et al found a high prevalence of mycoplasmal (52%), Chlamydia pneumoniae (7.5%), and active HHV-6 (30.5%) infections in 200 CFS patients.

The clinical findings of Dr. Edward Conley of the Fatigue, Fibromyalgia and Autoimmune Clinic, Michigan, USA, show that virus infections are consistently found in ME / CFS patients and this is detailed in his book America Exhausted: Breakthrough Treatments of Fatigue and Fibromyalgia


Zorzenon, Marcella; Rukh, Gull; Botta, Giuseppe; Colle, Roberto; Barsanti, Laura; Ceccherini-Nelli, Luca (1996). "Active HHV-6 Infection in Chronic Fatigue Syndrome Patients from Italy". *Journal of Chronic Fatigue Syndrome* 2: 3–12.

"Evidence of active HHV6 infection and its correlation with RNaseL (LMW) protein in CFS patients was presented by Dharam Ablashi (Washington, USA). His team had looked at HHV6 in plasma, CSF and white blood cells. The aim was to correlate HHV6 with presence of the 37KDa protein. The 35 CFS patients studied showed that 65% had active HHV6 infection with varying HHV6 IgM antibody and HHV6 infected PBMCs. In the CSF, 26.7% had HHV6 DNA. Nested PCR showed 34% patients had HHV6 in plasma, but using TaqMan PCR, 48.5% were shown positive in plasma, and 40% in CSF. This test was therefore more sensitive in this assay. HHV6 variant A was identified by TaqMan PCR in almost all positive patients. Variant A tends to be acquired in adult life, variant B in early childhood. Ratio of LMW to HMW(80KDa) protein was detected in 70% PMBC samples. Correlation with HHV6 was significant when the ratio was greater than 4. IgM antibody and PCR correlation was less significant."

Important lecture by Dr. Martin Lerner on the role of viruses in ME / CFS. Click on links to view videos.

Lecture part 1. Dr. A Martin Lerner presenting at William Beaumont Hospital in Royal Oak, MI USA, August 2011

Lecture part 2. Dr. A Martin Lerner presenting at William Beaumont Hospital in Royal Oak, MI USA, August 2011

- Slides of Dr. Martin Lerner's lecture

Dr. Martin Lerner

The Treatment Center for Chronic Fatigue Syndrome which has successfully treated thousands of ME / CFS patients.

Dr Martin Lerner has the following credentials:

Medical doctor with 40 years experience. Certified by the American Board of Internal Medicine and is an Infectious Disease Specialist. Residency, Internal Medicine, Harvard Medical Services. Boston City Hospital and Barnes Hospital, St. Louis, MO. Washington University School of Medicine, M.D. Two Years, National Institute of Allergy and Infectious Diseases, Epidemiology Unit.

Alumni Awardee, Washington University School of Medicine.

Three years research fellow in infectious diseases at the Thorndike Memorial Laboratory, Boston City Hospital and Harvard Medical School under the direction of *Dr. Maxwell Finland, (founder of subspecialty infectious diseases). Chief of the Division of Infectious Diseases and Professor of Internal Medicine at Wayne State University School of Medicine, 1963-1982. Established a clinical virology laboratory and trained 33 physicians in the subspecialty of infectious diseases, Wayne State University, 1963-1982.

- “Two disorders of significant importance, MS and CFS, appear to be associated with HHV-6 infection… the data presented here show that both MS and CFS patients tend to carry a higher rate of HHV-6 infection or reactivation compared to normal controls. This immunological and virological data supports a role of HHV-6 in the symptomatology of these diseases… Based
on biological, immunological and molecular analysis, the data show that HHV-6 isolates from 70% of CFS patients were Variant A...Interestingly, the majority of HHV-6 isolates from MS patients were Variant B...These data demonstrate that the CFS patients exhibited HHV-6 specific immune responses...Seventy percent of the HHV-6 isolates from CFS patients were Variant A, similar to those reported in AIDS...It has already been shown that active HHV-6 infection in HIV-infected patients enhanced the AIDS disease process. We suspect that the same scenario is occurring in the pathogenesis of MS and CFS...The immunological data presented here clearly shows a significantly high frequency of HHV-6 reactivation in CFS and MS patients. We postulate that active HHV-6 infection is a major contributory factor in the aetiologies of MS and CFS" (DV Ablashi, DL Peterson et al. Journal of Clinical Virology 2000:16:179-191).

- The work of psychiatrists - Dr. Thomas Henderson and Dr. William Pridgen
  - The Role of Antiviral Therapy in Chronic Fatigue Treatment
  - Treatment Resistant Depression or Chronic Fatigue Syndrome? Child Psychiatrist Finds Success With Antivirals
  - Doctors on Missions

  "Over the last decade a wide variety of infectious agents has been associated with CFS by researchers from all over the world. Many of these agents are neurotrophic and have been linked to other diseases involving the central nervous system (CNS)...Because patients with CFS manifest a wide range of symptoms involving the CNS as shown by abnormalities on brain MRIs, SPECT scans of the brain and results of tilt-table testing, we sought to determine the prevalence of HHV-6, HHV-8, EBV, CMV, Mycoplasma species, Chlamydia species and Coxsackie virus in the spinal fluid of a group of patients with CFS. Although we intended to search mainly for evidence of actively replicating HHV-6, a virus that has been associated by several researchers with this disorder, we found evidence of HHV-8, Chlamydia species, CMV and Coxsackie virus in (50% of patient) samples...It was also surprising to obtain such a relatively high yield of infectious agents on cell free specimens of spinal fluid that had not been centrifuged" (Susan Levine. JCFS 2002:9:1/2:41-51).

- The destructive power of HHV-6a virus
  3. HHV-6a can cause other immune system cells (like CD8 cells) to express the CD4 cell surface antigen. Lusso, P. et al; "Induction of CD4 and Susceptibility to HIV-1 Infection in Human CD98-Positive T Lymphocytes by Human Herpesvirus 6"; Nature 349:533, February 7, 1991.
  4. HHV-6a destroys the B-cells of the immune system at a rapid rate. This has been found in research carried out by Robert Gallo MD of the NIH in the USA and research by virologist Berch Henry, Nevada. This research is cited in the book 'Osler's Web', by Hillary Johnson, Penguin Books 1997
  5. HHV-6a can infect a wide variety of organ tissues (besides immune system cells), including brain, spinal cord, lung, lymph node, heart, bone marrow, liver, kidney, spleen, tonsil, skeletal muscle, adrenal glands, pancreas, and thyroid. Knox, K.K. and D.R. Carrigan; "Disseminated Active HHV-6a Infections in Patients With AIDS"; The Lancet 343:577, March 5, 1994.
  6. HHV-6a has been found to be closely associated with Kaposi's sarcoma, and suggested as a possible causitive agent of this "AIDS"-related cancer. Bovenzi, P. et al.; "Human Herpesvirus 6 (Variant A) in Kaposi's Sarcoma"; The Lancet 341:1288, May 15, 1993.
  7. HHV-6a has been associated with thrombocytopenia, a blood clotting disorder common in "AIDS" patients. Kitamura, K. et al.; "Idiopathic Thrombocytopenic Purpura After Human Herpesvirus 6 Infection"; The Lancet 344:830, September 17, 1994.
  9. HHV-6a can infect other species; most notably, it has been found in 100 percent of some populations of African green monkeys. Higashi, K. et al.; "Presence of Antibody to HHV-6 In Monkeys"; J. Gen. Virol. 70:3171, 1989.
10. HHV-6a can cause fatal, disseminated infections. Knox and Carrigan, op cit.
11. HHV-6a can cause fatal pneumonia (lung infection). R.W. Cone, op cit.
13. HHV-6a can cause hepatitis, a sometimes-fatal liver infection. Y. Asano et al., ibid.
15. HHV-6a is associated with a particular type of skin rash, or dermatitis, that occurs frequently following bone marrow transplantation. Michel, D. et al.; "Human Herpesvirus 6 DNA in Exanathematous Skin in BMT Patient"; The Lancet 344:686, September 3, 1994.
18. The two variants of HHV-6, Variant A and Variant B, appear to cause very different symptoms. Variant B seems to be associated with mild, childhood infection and disease; Variant A is found in immunocompromised adults with illnesses like "AIDS," cancer, and Chronic Fatigue Syndrome. Dewhurst, S.W. et al.; "Human Herpesvirus-6 (HHV-6) Variant B Accounts for the Majority of Symptomatic Primary HHV-6 infections in a Population of U.S. Infants"; Journal of Clinical Microbiology, February 1993.
19. HHV-6a's growth is stopped by the experimental drug Ampligen. Ablashi, D.V. et al.; Ampligen Inhibits In Vitro Replication of HHV-6; Abstract from CFS conference, Albany, NY, October 2-4, 1992
20. When HHV-6a's growth is stopped by the experimental drug Ampligen in Chronic Fatigue Syndrome patients, their symptoms resolve. (In a trial published in 1987, the same appeared to be true for "AIDS" patients treated with Ampligen.) Strayer, D.R. et al.; "A Controlled Clinical Trial With a Specifically Configured RNA Drug, Poly(I):Poly(C12U), in Chronic Fatigue Syndrome"; Clinical Infectious Diseases, January 1994.
21. HHV-6a has been suggested as a "cofactor" in the development of "AIDS." P. Lusso and R.C. Gallo; "Human Herpesvirus 6 in AIDS"; The Lancet 343:555, March 5, 1994.

Effects of HHV-6a virus compiled by Neenyah Ostrom

- Association of Active Human Herpesvirus-6, -7 and Parvovirus B19 Infection with Clinical Outcomes in Patients with Myalgic Encephalomyelitis/Chronic Fatigue Syndrome. Svetlana Chapenko, Angelika Krumina, Inara Logina, Santa Rasa, Maksims Chistjakovs, Alina Sultanova, Ludmila Viksna, and Modra Murovska. Advances in Virology. Volume 2012, Article ID 205085, 7 pages

- Nicolson et al showed that multiple co-infections (Mycoplasma, Chlamydia, HHV-6) in blood of chronic fatigue syndrome patients are associated with signs and symptoms: “Differences in bacterial and/or viral infections in (ME)CFS patients compared to controls were significant…The results indicate that a large subset of (ME)CFS patients show evidence of bacterial and/or viral infection(s), and these infections may contribute to the severity of signs and symptoms found in these patients” (Nicolson GL et al. APMIS 2003:111(5):557-566).


- There was evidence for ongoing infections with herpes viruses. A subset of patients (those with onset associated with EBV and those with recurrent herpes lesions) who improved on valaciclovir. She recommends trying a course in these patients. Some patients may have ongoing EBV activation. (Invest in ME Scientific Conference, 2013 Professor Carmen Scheibenbogen, Berlin, Germany)

Infections are of interest to ME / CFS patients and research.

- In his Summary of the Viral Studies of CFS, Dr Dharam V Ablashi concluded: “The presentations and discussions at this meeting strongly supported the hypothesis that CFS may be triggered by more than one viral agent… Komaroff suggests that, once reactivated, these viruses contribute directly to the morbidity of CFS by damaging certain tissues and indirectly by eliciting an on-going immune response” (Clin Inf Dis 1994:18 (Suppl 1):S130-133). It is recommended that the entire 167-page Journal be read.

- The Putative Role of Viruses, Bacteria, and Chronic Fungal Biotoxin Exposure in the Genesis of Intractable Fatigue Accompanied by Cognitive and Physical Disability. Morris et al. 2015

- Vojdani A, Lapp CW. Interferon-induced proteins are elevated in blood samples of patients with chemically or virally induced chronic fatigue syndrome. Immunopharmacol Immunotoxicol. 1999 May;21(2):175-202. PMID: 10319275

Certain toxic chemicals and certain viruses produce the same kinds of inflammatory effects and defects in 2-5A Synthetase and Protein Kinase RNA (PKR)). Anti IFN beta inhibited the reactions.

- Ablashi and Loomis pointed out that an analysis of studies of HHV-6 in (ME)CFS differentiated between active and latent virus, with 83% being positive (Assessment and Implications of Viruses in Debilitating Fatigue in CFS and MS Patients. Dharam V Ablashi et al. HHV-6 Foundation, Santa Barbara, USA. Submission to Assembly Committee/Ways & Means, Exhibit B1-20, submitted by Annette Whittemore 1st June 2005).


- The Virus Within By Nicholas Regush explore the role of HHV-6a virus in ME / CFS and other diseases, and provides many references.

- Freeman ML, Burkum CE, Jensen MK, Woodland DL, Blackman MA(2012), "γ-Herpesvirus Reactivation Differentially Stimulates Epitope-Specific CD8 T Cell Responses", Immunology, 2012 doi: 10.4049/jimmunol.1102787,


- "CFS patients with active HHV6 infection were shown to have activation of coagulation and are hypercoagulable. This may be a significant factor in CFS contributing to many symptoms." J. Brewer, research paper presented to the AACFS 5th International Research, Clinical and Patient Conference, 2001


- Carver LA et al. Military Medicine 1994; 159: 580

- HYPOTHESIS: A UNIFIED THEORY OF THE CAUSE OF CHRONIC FATIGUE SYNDROME

A. Martin Lerner, Marcus Zervos, Howard J. Dworkin, Chung Ho Chang, and William O'Neill
Infectious Diseases in Clinical Practice, 1997;6:239-243

- Presence of Viral Protein 1 (VP1)

"There are no tests to confirm a diagnosis, although 60% of sufferers will have a specific protein in their blood called viral protein 1, (VP1)."

Susan Clark, www.whatreallyworks.co.uk

- Findings and Testimony of Burke A. Cunha, MD., chief, infectious disease division, Winthrop-University Hospital, Mineola, N.Y., USA.
"But the most consistent lab evidence that we look for are elevations of coxsackie B-titers and elevations of HHV-6 titers in combination with the decrease in the percentage of natural killer T cells," Cunha explained. "If the patient has two or three of these abnormalities in our study center, then he or she fits the laboratory criteria for chronic fatigue. Nearly all patients with crimson crescents have two out of three of these laboratory abnormalities," he said.

- "Poly(I)-Poly(C12U)" is the molecular name for Ampligen
- Barnes, Deborah; "Mystery Disease at Lake Tahoe Challenges Virologists and Clinicians"; Science 234:541, 1986.
- Relationships Between Human T-Lymphotropic Virus Type II (HTLV-2) and Human Lymphotropic Herpes viruses in Chronic Fatigue Syndrome Project funded by The National CFIDS Foundation, Inc.; Needham, Massachusetts
- New Cardiomyopathy: Pilot Study of Intravenous Ganciclovir in a Subset of the Chronic Fatigue Syndrome. Infectious Disease in Clinical Practice, 1997;6:110-117
- Dynamics of Chronic Active Herpesvirus-6 Infection in Patients with Chronic Fatigue Syndrome: Data Acquisition for Computer Modeling Authors: Krueger GR, Koch B, Hoffmann A, Rojo J, Brandt ME, Wang G, Buja LM. Affiliation: Department of Pathology & Laboratory Medicine, University of Texas-Houston Medical School, 6431 Fannin St, MSB 2.246, Houston, Texas 77030, USA. 04-02-2002 Journal: In Vivo 2001 Nov-Dec;15(6):461-5
- Moore, Patrick S. and Yuan Chang; "Detection of Herpes virus-Like DNA Sequences in Kaposi's Sarcoma in Patients With


• Boshoff, Chris et al.; "Kaposi's Sarcoma-Associated Herpes virus in HIV-Negative Kaposi's Sarcoma"; The Lancet 345:1043, April 22, 1995.


• Lerner, AM et al. A small randomised placebo-controlled trial of the use of antiviral therapy for patients with chronic fatigue syndrome. Clinical Infectious Diseases, 2001, 32, 1657-1658

• "(ME)CFS is associated with objective underlying biological abnormalities, particularly involving the nervous and immune system. Most studies have found that active infection with HHV-6 – a neurotropic, gliotropic and immunotropic virus – is present more often in patients with (ME)CFS than in healthy control subjects...Moreover, HHV-6 has been associated with many of the neurological and immunological findings in patients with (ME)CFS" Anthony L Komaroff. Journal of Clinical Virology 2006:37:S1:S39-S46.

• "Assessment of the frequency of HHV6 in CFS was undertaken by the team at Columbia. D Ablashi showed that the majority of 24 patients studied from Incline Village, Nevada had HHV6 infection. HHV6 was detected in the plasma, CSF and PBMCs. The data suggests the presence of cell free infectious virus in the CSF. It was postulated that HHV6 invading the CNS may participate in the neurological manifestations of the disease."

"A poster also presented by Ablashi et al, showed good concordance between reactivation of HHV6 and presence of RnaseL. They could therefore be used together or separately in monitoring response to treatment. 2 patients were treated with ampligen, which inhibited HHV6 replication and upregulated the 2-5a synthetase/RnaseL pathway."

D Ablashi (Columbia University) research papers submitted to the AACFS 5th International Research, Clinical and Patient Conference, 2001

• Prevalence in the cerebrospinal fluid of the following infectious agents in a cohort of 12 CFS subjects: human herpes virus 6 and 8; chlamydia species; mycoplasma species; EBV; CMV; and coxsackie virus. Journal of Chronic Fatigue Syndrome, 2001, 9, 1/2, 41-51


• Torrisi, et al, wrote "The absence of lycoproteins on the cell surface of the infected cells," showing where the virus is hiding and how it infects (Virology, 257, 1999).

• Wu et al showed how HHV6 can infect human umbilical vein endothelial cells in the J Gen Vir (1998, 79)

• Landay AL et al. Lancet 1991; 338: 707


[ Back to Listing at top of page ]
Enteroviruses

Top ME doctors A. Gilliam, Melvin Ramsay, John Richardson of Newcastle-upon-Tyne, W.H. Lyle, Elizabeth Bell, James Mowbray of St Mary’s, Peter Behan and Byron Hyde all believed that the majority of primary M.E. patients fell ill following exposure to an Enterovirus. Dr. John Richardson, a medical doctor based in Newcastle in England treated ME patients from many parts of Britain for over 40 years. He developed an expertise in diagnosing the illness, and became one of the world's foremost experts in ME. He even used autopsy results from dead patients to investigate the illness. He found that Enteroviruses and toxins played a major role in ME, and that this led to immune dysfunction, neurological abnormalities, endocrine dysfunction, and over time to increased risk of cardiac failure, cancers, carcinomas, and other organ failure. He wrote a book about his medical experiences called Enteroviral and Toxin Mediated Myalgic Encephalomyelitis/Chronic Fatigue Syndrome. This book is a classic medical book on the illness, and provides an excellent introduction to ME. Historically, Enterovirus infections mainly target the nervous system, brain, muscles and intestines, all of which abnormal in ME patients.

  “Enteroviruses are well known causes of acute respiratory and gastrointestinal infections, with tropism for the central nervous system, muscle, and heart. Initial reports of chronic enteroviral infections causing debilitating symptoms in patients with CFS were met with skepticism, and largely forgotten for the past decade … Recent evidence not only confirmed the earlier studies but also clarified the pathological role of viral RNA through antiviral treatment.”
- Dr. John Chia, is a world renowned doctor who has successfully treated ME / CFS patients. He has found that Enteroviruses are present in some subgroups of ME / CFS patients and that treating these Enterovirus infections can lead to significant improvement and recovery.

His research paper provides some important insights - Chronic fatigue syndrome is associated with chronic enterovirus infection of the stomach. Chia JK, Chia AY. J Clin Pathol. 2008 Jan;61(1):43-8. Epub 2007 Sep 1. See diagram below:

Dr. John Chia presents his research findings up to the year 2011 to the National Institutes of Health (NIH) in the USA below:

- http://www.youtube.com/watch?v=obHTCwhg3-0 Part 1
- http://www.youtube.com/watch?v=BO-yxqZuXTY Part 2

Video Lecture of medical findings by Dr. John Chia
**ME Epidemics**

Most ME epidemics mention polio-like viruses, Cocksacke viruses and Enteroviruses. Click here to view listing of research papers from ME epidemics

Chronic Pelvic Pain (CPP) in Patients with Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) is Associated with Chronic Enterovirus Infection of Ovarian Tubes

John Chia, M.D., David Wang, Rabiba El-habbel and Andrew Chia, EV Med Research, Lomita California

**IACFS/ME Conference, Translating Science into Clinical Care**, March 20-23, 2014 • San Francisco, California, USA

Pathogenesis of chronic enterovirus infection in myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) –in vitro and in vivo studies of infected stomach tissues

John Chia, M.D., Andrew Chia, David Wang, Rabiba El-Habbal. EV Med Research. Lomita, CA.
Dowsett EG, Ramsay AM, McCartney RA, Bell EJ (1990), "Myalgic Encephalomyelitis (M.E.) -- A Persistent Enteroviral Infection?", Postgraduate Medical Journal, 66:526-530


Myalgic encephalomyelitis: a review with emphasis on key findings in biomedical research M Hooper. J Clin Pathol. 2007 May; 60(5): 466–471. doi: 10.1136/jcp.2006.042408


"Enteroviral sequences were found in significantly more ME/CFS patients than in the two comparison groups....This study provides evidence for the involvement of enteroviruses in just under half of the patients presenting with ME/CFS and it confirms and extends previous studies using muscle biopsies. We provide evidence for the presence of viral sequences in serum in over 40% of ME/CFS patients" (J Med Virol 1995:45:156-161)

" Primary M.E. is always an acute onset illness. Doctors A. Gilliam, A. Melvin Ramsay and Elizabeth Dowsett (who assisted in much of his later work,) John Richardson of Newcastle-upon-Tyne, W.H. Lyle, Elizabeth Bell of Ruckhill Hospital, James Mowbray of St Mary's, and Peter Behan all believed that the majority of primary M.E. patients fell ill following exposure to an Enterovirus. (Poliovirus, ECHO, Coxsackie and the numbered viruses are the significant viruses in this group, but there are other enteroviruses that exist that have been discovered in the past few decades that do not appear in any textbook that I have perused.) I share this belief that enteroviruses are a major cause."

Source: http://www.nightingale.ca/documents/Nightingale_ME_Definition_en.pdf


Quantitative analysis of viral RNA kinetics in coxsackievirus B3-induced murine myocarditis: biphasic pattern of clearance


- Spence V A, Khan F, Kennedy G. et al Inflammation and arterial stiffness in patients with Chronic Fatigue Syndrome. 8th International IACFS Conference on Chronic Fatigue Syndrome, Fibromyalgia and other related illnesseses, Fort Lauderdale, Florida, USA, January, 2007


- Parish JG (1978), Early outbreaks of 'epidemic neuromyasthenia', Postgraduate Medical Journal, Nov;54(637):711-7, PMID: 370810. 'Epidemic Neuromyasthenia' was used to describe ME in the past.

- Medical and Scientific Books
  - Enteroviral and Toxin Mediated Myalgic Encephalomyelitis/Chronic Fatigue Syndrome
  - The Clinical and Scientific Basis of Myalgic Encephalomyelitis--Chronic Fatigue Syndrome by Dr. Jay Goldstein, Dr. Byron Hyde, P. Levine, Nightingale Research Foundation.
  - Myalgic Encephalomyelitis and Post Viral Fatigue States: the Saga of the Royal Free Disease by Dr Melvin Ramsay

- In the UK, about 60% of patients with ME / CFS have evidence of enterovirus infection, most commonly Cocksackie B. This has been demonstrated by the finding of enterovirus RNA in muscle and in blood. Many other patients have reactivated Epstein Barr virus. It has not been verified if the virus itself causes ME / CFS or it is the result of a weakened immune system. Source: Action for ME, Britain.


- The Putative Role of Viruses, Bacteria, and Chronic Fungal Biotoxin Exposure in the Genesis of Intractable Fatigue Accompanied by Cognitive and Physical Disability. Morris et al. 2015

- “Virological studies revealed that 76% of the patients with suspected myalgic encephalomyelitis had elevated Coxsackie B neutralising titres (and symptoms included) malaise, exhaustion on physical or mental effort, chest pain, palpitations, tachycardia, polyarthralgia, muscle pains, back pain, true vertigo, dizziness, tinnitus, nausea, diarrhoea, abdominal cramps, epigastric pain, headaches, paraesthesiae, dysuria)....The group described here are patients who have had this miserable illness. Most have lost many weeks of employment or the enjoyment of their family (and) marriages have been threatened...” (BD Keighley, EJ Bell. JRCP 1983:33:339-341).

- The Nightingale Definition of Myalgic Encephalomyelitis (M.E.), 2011

- The Nightingale Definition of M.E., 2007


- The case for Enteroviruses


- Enteroviral Myalgic Encephalomyelitis - EvME: A treatise on EvME by Dr Irving Spurr
The outbreak in Iceland was important, and provided some vital clues about the illness and the role of Enteroviruses. "However, children in epidemic Neuromyasthenia areas responded to poliomyelitis vaccination with higher antibody titres than in other areas not affected by the poliomyelitis epidemic, as if these children had already been exposed to an agent immunologically similar to poliomyelitis virus (Sigurdsson, Gudnadottir Petursson, 1958). Thus, the agent responsible for epidemic Neuromyasthenia would appear to be able to inhibit the pathological effects of poliomyelitis infection. When an American airman was affected in the 1955 epidemic and returned home, a similar secondary epidemic occurred in Pittsfield, Massachusetts, U.S.A. (Hart, 1969: Henderson and Shelokov, 1959)."


"an agent was repeatedly transmitted to monkeys from two patients (Pellew and Miles, 1955). When the monkeys were killed minute red spots were observed along the course of the sciatic nerves. Microscopically infiltration of nerve roots with lymphocytes and mononuclear cells was seen and some of the nerve fibres showed patchy damage to the myelin sheaths and axon swellings. Similar findings had been produced by the transmission of an agent to monkeys from a child with poliomyelitis in Boston, Massachusetts, in 1947 (Pappenheimer, Cheever and Daniels, 1951). How- ever, in these monkeys the changes were more widespread, involving the dorsal root ganglia, cervical and lumbar nerve roots and peripheral nerves. Perivascular collars of lymphocytes and plasma cells were seen in the cerebral cortex, brain stem and cerebellum, spinal cord and around blood vessels to the nerve roots. There was no evidence of damage to the nerve cells in the brain or spinal cord. The distribution and intensity of the lesions varied considerably from monkey to monkey. This pathological picture of mild diffuse changes corresponds closely to what might be expected from clinical observations of patients with neurological involvement in epidemic Neuromyasthenia." Parish JG (1978), Early outbreaks of ‘epidemic neuromyasthenia’, Postgraduate Medical Journal, Nov;54(637):711-7, PMID: 370810.

Three Babuska Clusters of Enteroviral-Associated Myalgic Encephalomyelitis
Nightingale Research Foundation
Paper Presented by Byron Marshall Hyde M.D.
New South Wales, February 1998


"Myeloadenaminate Deaminase deficiency in muscles of ME patients. It is known that the enzyme is missing after a viral attack" Professor Peter Behan, The Institute of Neurological Sciences, University of Glasgow, Scotland.

"Recently associations have been found between Coxsackie B infection and a more chronic multisystem illness. A similar illness…has been referred to as… myalgic encephalomyelitis…140 patients presenting with symptoms suggesting a postviral syndrome were entered into the study…Coxsackie B antibody levels were estimated in 100 control patients…All the Coxsackie B virus antibody tests were performed blind…Of the 140 ill patients, 46% were found to be Coxsackie B virus antibody positive…This study has confirmed our earlier finding that there is a group of symptoms with evidence of Coxsackie B infection. We have also shown that clinical improvement is slow and recovery does not correlate with a fall in Coxsackie B virus antibody titre" (BD Calder et al. JRCGP 1987:37:11-14).

Presence of Viral Protein 1 (VP1)
"There are no tests to confirm a diagnosis, although 60% of sufferers will have a specific protein in their blood called viral protein 1, (VP1)."
Susan Clark, www.whatreallyworks.co.uk


Anti-pathogen and immune system treatments. Treatment of 741 Italian patients with chronic fatigue syndrome. U. TIRELLI, A. LLESHI, M. BERRETTA, M. SPINA, R. TALAMINI, A. GIACALONE. European Review for Medical and Pharmacological Sciences 2013; 17: 2847-2852
Cunningham L, Bowles NE, Lane RJ, Dubowitz V, and Archard LC: Persistence of Enteroviral RNA in Chronic Fatigue Syndrome is Associated with the Abnormal Production of Equal Amounts of Positive and Negative Strands of Enteroviral RNA. J General Virol 1990; 71:1399--1402

Findings and Testimony of Burke A. Cunha, MD., chief, infectious disease division, Winthrop-University Hospital, Mineola, N.Y., USA.

"But the most consistent lab evidence that we look for are elevations of coxsackie B-titers and elevations of HHV-6 titers in combination with the decrease in the percentage of natural killer T cells," Cunha explained. "If the patient has two or three of these abnormalities in our study center, then he or she fits the laboratory criteria for chronic fatigue. Nearly all patients with crimson crescents have two out of three of these laboratory abnormalities," he said.

"These results show that chronic infection with enteroviruses occurs in many PVFS (post-viral fatigue syndrome, a classified synonym for ME/CFS) patients and that detection of enterovirus antigen in the serum is a sensitive and satisfactory method for investigating infection in these patients. Several studies have suggested that infection with enteroviruses is causally related to PVFS... The association of detectable IgM complexes and VP1 antigen in the serum of PVFS patients in our study was high... This suggests that enterovirus infection plays an important role in the aetiology of PVFS" (GE Yousef, EJ Bell, JF Mowbray et al. Lancet January 23rd 1988:146-150).

Prevalence in the cerebrospinal fluid of the following infectious agents in a cohort of 12 CFS subjects: human herpes virus 6 and 8; chlamydia species; mycoplasma species; EBV; CMV; and coxsackie virus. Journal of Chronic Fatigue Syndrome, 2001, 9, 1/2, 41-51


"However, children in epidemic Neuromyasthenia areas responded to poliomyelitis vaccination with higher antibody titres than in other areas not affected by the poliomyelitis epidemic, as if these children had already been exposed to an agent immunologically similar to poliomyelitis virus (Sigurdsson, Gudnad6ttir Petursson, 1958). Thus, the agent responsible for epidemic Neuromyasthenia would appear to be able to inhibit the pathological effects of poliomyelitis infection. When an American airman was affected in the 1955 epidemic and returned home, a similar secondary epidemic occurred in Pittsfield, Massachusetts, U.S.A. (Hart, 1969: Henderson and Shelokov, 1959)."


"Myalgic encephalomyelitis is a common disability but frequently misinterpreted... This illness is distinguished from a variety of other post-viral states by a unique clinical and epidemiological pattern characteristic of enteroviral infection... 33% had titres indicative and 17% suggestive of recent CBV infection... Subsequently... 31% had evidence of recent active enteroviral infection... There has been a failure to recognise the unique epidemiological pattern of ME...

Coxsackie viruses are characteristically myotropic and enteroviral genomic sequences have been detected in muscle biopsies from patients with ME. Exercise related abnormalities of function have been demonstrated by nuclear magnetic resonance and single-fibre electromyography including a failure to coordinate oxidative metabolism with anaerobic glycolysis causing abnormal early intracellular acidosis, consistent with the early fatiguability and the slow recovery from exercise in ME. Coxsackie viruses can initiate non-cytolytic persistent infection in human cells. Animal models demonstrate similar enteroviral persistence in neurological disease... and the deleterious effect of forced exercise on persistently infected muscles. These studies elucidate the exercise-related morbidity and the chronic relapsing nature of ME” (EG Dowsett, AM Ramsay et al. Postgraduate Medical Journal 1990:66:526-530).

“The findings described here provide the first evidence that postviral fatigue syndrome may be due to a mitochondrial disorder precipitated by a virus infection... Evidence of mitochondrial abnormalities was present in 80% of the cases with the commonest change (seen in 70%) being branching and fusion of cristae, producing 'compartmentalisation'. Mitochondrial pleomorphism, size variation and occasional focal vacuolation were detectable in 64%... Vacuolation of mitochondria was frequent... In some cases there was swelling of the whole mitochondrion with rupture of the outer membranes... prominent secondary lysosomes were common in some of the worst affected cases... The pleomorphism of the mitochondria in the patients' muscle biopsies was in clear contrast to the findings in normal control biopsies... Diffuse or focal atrophy of type II
Persistent enteroviral infection of muscle has been reported, and this does indicate muscle damage and not just muscle disuse” (WMH Behan et al. Acta Neuropathologica 1991:83:61-65).

- "Persistent enteroviral infection of muscle may occur in some patients with postviral fatigue syndrome and may have an aetiological role….The features of this disorder suggest that the fatigue is caused by involvement of both muscle and the central nervous system…We used the polymerase chain reaction to search for the presence of enteroviral RNA sequences in a well-characterised group of patients with the postviral fatigue syndrome...53% were positive for enteroviral RNA sequences in muscle…Statistical analysis shows that these results are highly significant…On the basis of this study…there is persistent enteroviral infection in the muscle of some patients with the postviral fatigue syndrome and this interferes with cell metabolism and is causally related to the fatigue” (JW Gow et al. BMJ 1991:302:696-696).

- "Postviral fatigue syndrome / myalgic encephalomyelitis... has attracted increasing attention during the last five years...Its distinguishing characteristic is severe muscle fatiguability made worse by exercise...The chief organ affected is skeletal muscle, and the severe fatiguability, with or without myalgia, is the main symptom. The results of biochemical, electrophysiological and pathological studies support the view that muscle metabolism is disturbed, but there is no doubt that other systems, such as nervous, cardiovascular and immune are also affected…Recognition of the large number of patients affected…indicates that a review of this intriguing disorder is merited....The true syndrome is always associated with an infection...Viral infections in muscle can indeed be associated with a variety of enzyme abnormalities...(Electrophysiological results) are important in showing the organic nature of the illness and suggesting that muscle abnormalities persist after the acute infection...there is good evidence that Coxsackie B virus is present in the affected muscle in some cases” (PO Behan, WMH Behan. CRC Crit Rev Neurobiol 1988:4:2:157-178).

- "The main features (of ME) are: prolonged fatigue following muscular exercise or mental strain, an extended relapsing course; an association with neurological, cardiac, and other characteristic enteroviral complications. Coxsackie B neutralisation tests show high titres in 41% of cases compared with 4% of normal adults…These (chronic enteroviral syndromes) affect a young, economically important age group and merit a major investment in research” (EG Dowsett. Journal of Hospital Infection 1988:11:103-115).

- The impact of persistent enteroviral infection by Dr. Betty Dowsett

- “Ten patients with post-viral fatigue syndrome and abnormal serological, viral, immunological and histological studies were examined by single fibre electromyographic technique….The findings confirm the organic nature of the disease. A muscle membrane disorder...is the likely mechanism for the fatigue and the single-fibre EMG abnormalities. This muscle membrane defect may be due to the effects of a persistent viral infection...There seems to be evidence of a persistent viral infection and/or a viral-induced disorder of the immune system...The infected cells may not be killed but become unable to carry out differentiated or specialised function” (Goran A Jamal, Stig Hansen. Euro Neurol 1989:29:273-276).

- Encephalomyelitis simulating Poliomyelitis.

- Benign myalgic encephalomyelitis

- 'Epidemic Myalgic Encephalomyelitis'


- Epidemiological aspects of an outbreak of encephalomyelitis at the Royal Free Hospital, London, in the summer of 1955


- Punta Gorda outbreak


- The Clinical Syndrome Various Called Benign Myalgic Encephalomyelitis, Iceland Disease and Epidemic Neuromyasthenia.
Molecular viral studies have recently proved to be extremely useful. They have confirmed the likely important role of enteroviral infections, particularly with Coxsackie B virus (Postviral fatigue syndrome: Current neurobiological perspective. PGE Kennedy. BMB 1991:47:4:809-814).

In his Summary of the Viral Studies of CFS, Dr Dharam V Ablashi concluded: “The presentations and discussions at this meeting strongly supported the hypothesis that CFS may be triggered by more than one viral agent…Komaroff suggests that, once reactivated, these viruses contribute directly to the morbidity of CFS by damaging certain tissues and indirectly by eliciting an on-going immune response” (Clin Inf Dis 1994:18 (Suppl 1):S130-133). It is recommended that the entire 167-page Journal be read.

“Our focus will be on the ability of certain viruses to interfere subtly with the cell’s ability to produce specific differentiated products as hormones, neurotransmitters, cytokines and immunoglobulins etc in the absence of their ability to lyse the cell they infect. By this means viruses can replicate in histologically normal appearing cells and tissues…Viruses by this means likely underlie a wide variety of clinical illnesses, currently of unknown aetiology, that affect the endocrine, immune, nervous and other systems” (JC de la Torre, P Borrow, MBA Oldstone. BMB 1991:47:4:838-851).

“We conclude that persistent enteroviral infection plays a role in the pathogenesis of PVFS…The strongest evidence implicates Coxsackie viruses…Patients with PVFS were 6.7 times more likely to have enteroviral persistence in their muscles” (JW Gow and WMH Behan. BMB 1991:47:4:872-885).

“The postviral fatigue syndrome (PVFS), with profound muscle fatigue on exertion and slow recovery from exhaustion seems to be related specifically to enteroviral infection. The form seen with chronic reactivated EBV infection is superficially similar, but without the profound muscle fatigue on exercise” (JF Mowbray, GE Yousef. BMB 1991:47:4:886-894).

A New and Simple Definition of Myalgic Encephalomyelitis and a New Simple Definition of Chronic Fatigue Syndrome & A Brief History of Myalgic Encephalomyelitis & An Irreverent History of Chronic Fatigue Syndrome by Dr Byron Hyde 2006

“Skeletal samples were obtained by needle biopsy from patients diagnosed clinically as having CFS (and) most patients fulfilled the criteria of the Centres for Disease Control for the diagnosis of CFS (Holmes et al 1988)…These data are the first demonstration of persistence of defective virus in clinical samples from patients with CFS…We are currently investigating the effects of persistence of enteroviral RNA on cellular gene expression leading to muscle dysfunction” (L Cunningham, RJM Lane, LC Archard et al. Journal of General Virology 1990:71:6:1399-1402).

“These results suggest there is persistence of enterovirus infection in some CFS patients and indicate the presence of distinct novel enterovirus sequences…Several studies have shown that a significant proportion of patients complaining of CFS have markers for enterovirus infection….From the data presented here…the CFS sequences may indicate the presence of novel enteroviruses…It is worth noting that the enteroviral sequences obtained from patients without CFS were dissimilar to the sequences obtained from the CFS patients…This may provide corroborating evidence for the presence of a novel type of enterovirus associated with CFS” (DN Galbraith, C Nairn and GB Clements. Journal of General Virology 1995:76:1701-1707).

“We will report at the First International Research Conference on Chronic Fatigue Syndrome to be held at Albany, New York, 2-4 October 1992, our new findings relating particularly to enteroviral infection…We have isolated RNA from patients and probed this with large enterovirus probes…detailed studies...showed that the material was true virus…Furthermore, this virus was shown to be replicating normally at the level of transcription. Sequence analysis of this isolated material showed that it had 80% homology with Coxsackie B viruses and 76% homology with poliomyelitis virus, demonstrating beyond any doubt that the material was enterovirus” (Press Release for the Albany Conference, Professor Peter O Behan, University of Glasgow, October 1992).

“In the CFS study group, 42% of patients were positive for enteroviral sequences by PCR, compared to only 9% of the comparison group…Enteroviral PCR does, however, if positive, provide evidence for circulating viral sequences, and has been used to show that enteroviral specific sequences are present in a significantly greater proportion of CFS patients than other
“Samples from 25.9% of the PFS (postviral fatigue syndrome) were positive for the presence of enteroviral RNA, compared with only 1.3% of the controls...We propose that in PFS patients, a mutation affecting control of viral RNA synthesis occurs during the initial phase of active virus infection and allows persistence of replication defective virus which no longer attracts a cellular immune response” (NE Bowles, RJM Lane, L Cunningham and LC Archard. Journal of Medicine 1993;24:2&3:145-180).

“These data support the view that while there may commonly be asymptomatic enterovirus infections of peripheral blood, it is the presence of persistent virus in muscle which is abnormal and this is associated with postviral fatigue syndrome...Evidence derived from epidemiological, serological, immunological, virological, molecular hybridisation and animal experiments suggests that persistent enteroviral infection may be involved in...PFS” (PO Behan et al. CFS: CIBA Foundation Symposium 173, 1993:146-159).

Seeking to detect and characterise enterovirus RNA in skeletal muscle from patients with (ME)CFS and to compare efficiency of muscle metabolism in enterovirus positive and negative (ME)CFS patients, Lane et al obtained quadriceps biopsy samples from 48 patients with (ME)CFS. Muscle biopsy samples from 20.8% of patients were positive, while 100% of the controls were negative for enterovirus sequences. Lane et al concluded: “There is an association between abnormal lactate response to exercise, reflecting impaired muscle energy metabolism, and the presence of enterovirus sequences in muscle in a proportion of (ME)CFS patients” (RJM Lane, LC Archard et al. JNNP 2003:74:1382-1386).

Kerr et al then go on to provide evidence of other triggers of (ME)CFS which include Parvovirus; C. pneumoniae, C. burnetti; toxin exposure and vaccination including MMR, pneumovax, influenza, hepatitis B, tetanus, typhoid and poliovirus (LD Devanur, JR Kerr. Journal of Clinical Virology 2006: 37(3):139-150).

“Research studies have identified various features relevant to the pathogenesis of CFS/ME such as viral infection, immune abnormalities and immune activation, exposure to toxins, chemicals and pesticides, stress, hypotension...and neuroendocrine dysfunction....Various viruses have been shown to play a triggering or perpetuating role, or both, in this complex disease....The role of enterovirus infection as a trigger and perpetuating factor in CFS/ME has been recognised for decades...The importance of gastrointestinal symptoms in CFS/ME and the known ability of enteroviruses to cause gastrointestinal infections led John and Andrew Chia to study the role of enterovirus infection in the stomach of CFS/ME patients...They describe a systematic study of enterovirus infection in the stomach of 165 CFS/ME patients, demonstrating a detection rate of enterovirus VP1 protein in 82% of patients...the possibility of an EV outbreak...seems unlikely, as these patients developed their diseases at different times over a 20 year period” (Jonathan R Kerr. Editorial. J Clin Pathol 14th September 2007. Epub ahead of print).

“Since most (ME)CFS patients have persistent or intermittent gastrointestinal (GI) symptoms, the presence of viral capsid protein 1 (VP1), enterovirus RNA and culturable virus in the stomach biopsy specimens of patients with (ME)CFS was evaluated...Our recent analysis of 200 patients suggests that...enteroviruses may be the causative agents in more than half of the patients...At the time of oesophagogastroduodenoscopy, the majority of patients had mild, focal inflammation in the antrum...95% of biopsy specimens had microscopic evidence of mild chronic inflammation...82% of biopsy specimens stained positive for VP1 within parietal cells, whereas 20% of the controls stained positive...An estimated 80-90% of our 1,400 (ME)CFS patients have recurring gastrointestinal symptoms of varying severity, and epigastric and/or lower quadrant tenderness by examination...Finding enterovirus protein in 82% of stomach biopsy samples seems to correlate with the high percentage of (ME)CFS patients with GI complaints...Interestingly, the intensity of VP1 staining of the stomach biopsy correlated inversely with functional capacity...A significant subset of (ME)CFS patients may have a chronic, disseminated, non-cytolytic form of enteroviral infection which can lead to diffuse symptomatology without true organ damage” (Chia JK, Chia AY. J Clin Pathol 13th September 2007 Epub ahead of print).

In a review of the role of enteroviruses in (ME)CFS, Chia noted that initial reports of chronic enteroviral infections causing debilitating symptoms in (ME)CFS patients were met with scepticism and largely forgotten, but observations from in vitro experiments and from animal models clearly established a state of chronic persistence through the formation of double stranded RNA, similar to findings reported in muscle biopsies of patients with (ME)CFS. Recent evidence not only confirmed the earlier studies, but also clarified the pathogenic role of viral RNA (JKS Chia. Journal of Clinical Pathology 2005:58:1126-
• Torrisi, et al, wrote "The absence of lycoproteins on the cell surface of the infected cells," showing where the virus is hiding and how it infects (Virology, 257, 1999).


• History of ME presented by Lisa Petrison to the CFSAC in 2014

• As mentioned elsewhere, researchers from the Enterovirus Research Laboratory, Department of Pathology and Microbiology, University of Nebraska Medical Centre wrote a specially-commissioned explanatory article for the UK charity Invest in ME, in which they stated that human enteroviruses were not generally thought to persist in the host after an acute infection, but they had discovered that Coxsackie B viruses can naturally delete sequence from the 5' end of the RNA genome, and that this results in long-term viral persistence, and that "This previously unknown and unsuspected aspect of enterovirus replication provides an explanation for previous reports of enteroviral RNA detected in diseased tissue in the apparent absence of infectious virus particles" (S Tracy and NM Chapman. Journal of IiME 2009:3:1). (http://www.investinme.org/Documents/Journals/Journal%20of%20IiME%20Vol%203%20Issue%201.pdf).

• “Recent developments in molecular biology…have revealed a hitherto unrecognised association between enteroviruses and some of the most disabling, chronic and disheartening neurological, cardiac and endocrine diseases…Persistent infection (by enteroviruses) is associated with ME/CFS…The difficulty of making a differential diagnosis between ME/CFS and post-polio sequelae cannot be over-emphasised…(EG Doswett. Commissioned for the BASEM meeting at the RCGP, 26th April 1998:1-10).

• “To prove formally that persistence rather than re-infection is occurring, it is necessary to identify a unique feature retained by serial viral isolates from one individual. We present here for the first time evidence for enteroviral persistence (in humans with CFS)...” (DN Galbraith et al. Journal of General Virology 1997:78:307-312).

(c) Epstein Barr virus & Herpes family viruses, including reactivation of latent herpes viruses (EBV, CMV and HHV6a)


Scientific analysis and discussion on http://simmaronresearch.com/2014/03/1591/


• The spread of EBV to ectopic lymphoid aggregates may be the final common pathway in the pathogenesis of ME/CFS


• Could the Epstein-Barr Virus – Autoimmunity Hypothesis Help Explain Chronic Fatigue Syndrome ? and

EBV I: A Deficient Immune Response, Increased Levels of Epstein-Barr Virus Opens Up EBV Question in Chronic Fatigue Syndrome Again Simmaron Research, USA

A subset of ME / CFS patients have been found to have EBV infection - "There is prolonged elevated antibody level against the encoded proteins EBV dUTPase and EBV DNA polymerase in a subset of CFS patients, suggesting that this antibody panel could be used to identify these patients." Antibody to Epstein-Barr Virus Deoxyuridine Triphosphate Nucleotidohydrolase and Deoxyribonucleotide Polymerase in a Chronic Fatigue Syndrome Subset. A. Martin Lerner, Maria E. Ariza, Marshall Williams, Leonard Jason, Safedin Beqaj, James T. Fitzgerald, Stanley Lemeshow, Ronald Glaser
Childhood 'kissing disease' linked to adult chronic illnesses, 11 Alive News, USA, December 2016.


The Putative Role of Viruses, Bacteria, and Chronic Fungal Biotoxin Exposure in the Genesis of Intractable Fatigue Accompanied by Cognitive and Physical Disability. Morris et al. 2015


"Over the last decade a wide variety of infectious agents has been associated with CFS by researchers from all over the world. Many of these agents are neurotrophic and have been linked to other diseases involving the central nervous system (CNS)...Because patients with CFS manifest a wide range of symptoms involving the CNS as shown by abnormalities on brain MRIs, SPECT scans of the brain and results of tilt-table testing, we sought to determine the prevalence of HHV-6, HHV-8, EBV, CMV, Mycoplasma species, Chlamydia species and Coxsackie virus in the spinal fluid of a group of patients with CFS. Although we intended to search mainly for evidence of actively replicating HHV-6, a virus that has been associated by several researchers with this disorder, we found evidence of HHV-8, Chlamydia species, CMV and Coxsackie virus in (50% of patient) samples...It was also surprising to obtain such a relatively high yield of infectious agents on cell free specimens of spinal fluid that had not been centrifuged" (Susan Levine. JCFS 2002:9:1/2:41-51).


Vojdani A, Lapp CW. Interferon-induced proteins are elevated in blood samples of patients with chemically or virally induced chronic fatigue syndrome. Immunopharmacol Immunotoxicol. 1999 May;21(2):175-202. PMID: 10319275

Certain toxic chemicals and certain viruses produce the same kinds of inflammatory effects and defects in 2-5A Synthetase and Protein Kinase RNA (PKR)). Anti IFN beta inhibited the reactions.

Shapiro JS (2009), "Does varicella-zoster virus infection of the peripheral ganglia cause Chronic Fatigue Syndrome?", Medical Hypotheses Volume 73, Issue 5, November 2009, Pages 728-734, PMID: 19520522.

There was evidence for ongoing infections with herpes viruses. A subset of patients (those with onset associated with EBV and those with recurrent herpes lesions) who improved on valaciclovir. She recommends trying a course in these patients. Some patients may have ongoing EBV activation. (Invest in ME Scientific Conference, 2013 Professor Carmen Scheibenbogen, Berlin,Germany)


“…from an immunological point of view, patients with chronic active EBV infection appear ‘frozen’ in a state typically found only briefly during convalescence from acute EBV infection” (G Tosato, S Straus et al. The Journal of Immunology 1985:134:5:3082-3088.)

Freeman ML, Burkum CE, Jensen MK, Woodland DL, Blackman MA(2012), "γ-Herpesvirus Reactivation Differentially Stimulates Epitope-Specific CD8 T Cell Responses", Immunology, 2012 doi: 10.4049/jimmunol.1102787,

In his Summary of the Viral Studies of CFS, Dr Dharam V Ablashi concluded: "The presentations and discussions at this meeting strongly supported the hypothesis that CFS may be triggered by more than one viral agent…Komaroff suggests that, once reactivated, these viruses contribute directly to the morbidity of CFS by damaging certain tissues and indirectly by eliciting an on-going immune response"(Clin Inf Dis 1994:18 (Suppl 1):S130-133). It is recommended that the entire 167-page Journal be read


"Ninety percent of the patients tested had antibodies to Epstein-Barr virus and 45% tested had antibodies to cytomegalovirus...if this fatigue syndrome is triggered by an infectious agent, an abnormal immune response may be involved" (TJ Marrie et al. Clinical Ecology 1987:V:1:5-10).


In the UK, about 60% of patients with ME / CFS have evidence of enterovirus infection, most commonly Cocksackie B. This has been demonstrated by the finding of enterovirus RNA in muscle and in blood. Many other patients have reactivated Epstein Barr virus. It has not been verified if the virus itself causes ME / CFS or it is the result of a weakened immune system.

Source: Action for ME, Britain.


Prevalence in the cerebrospinal fluid of the following infectious agents in a cohort of 12 CFS subjects: human herpes virus 6 and 8; chlamydia species; mycoplasma species; EBV; CMV; and coxsackie virus. Journal of Chronic Fatigue Syndrome, 2001, 9, 1/2, 41-51


"Myeloadenamate Deaminase deficiency in muscles of ME patients. It is known that the enzyme is missing after a viral attack" Professor Peter Behan, The Institute of Neurological Sciences, University of Glasgow, Scotland.

Presence of Viral Protein 1 (VP1)
"There are no tests to confirm a diagnosis, although 60% of sufferers will have a specific protein in their blood called viral protein 1, (VP1)."
(d) Retrovirus - HTLV viruses, HGRV virus, MLV"s, JHK virus, HIV Negative AIDS

- "an agent was repeatedly transmitted to monkeys from two patients (Pellew and Miles, 1955). When the monkeys were killed minute red spots were observed along the course of the sciatic nerves. Microscopically infiltration of nerve roots with lymphocytes and mononuclear cells was seen and some of the nerve fibres showed patchy damage to the myelin sheaths and axon swellings. Similar findings had been produced by the transmission of an agent to monkeys from a child with poliomyelitis in Boston, Massachusetts, in 1947 (Pappenheimer, Cheever and Daniels, 1951). How-ever, in these monkeys the changes were more widespread, involving the dorsal root ganglia, cervical and lumbar nerve roots and peripheral nerves. Perivascular collars of lymphocytes and plasma cells were seen in the cerebral cortex, brain stem and cerebellum, spinal cord and around blood vessels to the nerve roots. There was no evidence of damage to the nerve cells in the brain or spinal cord. The distribution and intensity of the lesions varied considerably from monkey to monkey. This patho-logical picture of mild diffuse changes corresponds closely to what might be expected from clinical observations of patients with neurological involvement in epidemic Neuromyasthenia Parish JG (1978), *Early outbreaks of 'epidemic neuromyasthenia*', Postgraduate Medical Journal, Nov;54(637):711-7, PMID: 370810.


- Torrisi, et al, wrote "The absence of lycoproteins on the cell surface of the infected cells," showing where the virus is hiding and how it infects (Virology, 257, 1999).


Lipkin / Hornig Chronic Fatigue Initiatitive Study in September 2013 found that 85% of patients had evidence of Retroviral infection and 65% had evidence of Annelovirus infection


The world patent entitled "Method and Compositions for Diagnosing and Treating Chronic Fatigue Immunodysfunction Syndrome" #WO9205760 issued to Elaine DeFreitas and Brendan Hilliard, inventors assigned to Wistar Institute, USA. This patent was applied for in August 1991. It concerns the discovery of a new virus the CAV virus which may lie at the root of CFS / ME

- HTLV virus found in CFS patients, infection rates range from 50-75% among CFS patients. HTLV has been found to infect macrophages, B-cells and T-cells. Research cited in *Osler's Web: Inside the Labyrinth of the Chronic Fatigue Syndrome Epidemic* by Hillary Johnson, penguin books, 1997, pages 285, 290-291, 352-353.

- Plague: One Scientist's Intrepid Search for the Truth about Human Retroviruses and Chronic Fatigue Syndrome (ME/CFS), Autism, and Other Diseases by Dr. Judy Mikovits and Kent Heckenlively


- Relationships Between Human T-Lymphotropic Virus Type II (HTLV-2) and Human Lymphotropic Herpes viruses in Chronic Fatigue Syndrome Project funded by The National CFIDS Foundation, Inc.; Needham, Massachusetts


- Dr. Michael Holmes of the Department of Microbiology of the University of Otago (New Zealand) carried out detailed studies into a CFS-like illness in New Zealand in the 1980's and 1990's. He found evidence of retrovirus infection in most samples and electron microscope pictures of cells with convoluted nuclei similar to AIDS patients. This indicated infection with a retrovirus. He also found evidence of excessive interferon levels, which are linked to retrovirus infection. His findings suggest that a retrovirus was responsible and that there is also significant immune dysfunction in CFS. Reported in the book 'Oslers Web', by Hillary Johnson, Penguin Books 1997, pages 661-663

  Chapter 33: A Retrovirus Aetiology for CFS?, Michael J. Holmes, M.D from the book The Clinical and Scientific Basis of Myalgic Encephalomyelitis--Chronic Fatigue Syndrome by Dr. Jay Goldstein, Dr. Byron Hyde, P. Levine, Nightingale Research Foundation.

  Dr Michael Holmes wrote in The Clinical and Scientific Basis for Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (1/97) that "structures consistent in size, shape and character with various stages of a Lentivirus (retrovirus) replicative cycle were observed by electron microscopy in cultures from CFS patients..."

- Some Papers by Dr. Michael Holmes

- In 1994, Dr. Anthony Komaroff of Harvard Medical School reported that the brains of those people with ME/CFS were identical to those with AIDS dementia, when viewed with SPECT imaging. They were both completely different to normal healthy brains. He believed that ME/CFS cases were the result of viral infection of the brain and nervous system, similar to AIDS.

- Dr. Seymour Grufferman and Dr. William Blattner found some evidence of HTLV infection in ME patients from the North Carolina Symphony in the early 1990's. Osler's Web, Hilary Johnson, pages 651-652

- Frequent detection of infectious xenotropic murine leukemia virus (XMLV) in human cultures established from mouse xenografts

- Xenotropic MLV envelope proteins induce tumor cells to secrete factors that promote the formation of immature blood vessels.

- Identification of Replication Competent Murine Gammaretroviruses in Commonly Used Prostate Cancer Cell Lines PLOS Published: June 17, 2011
  DOI: 10.1371/journal.pone.0020874


- Innate Immune Changes in the Peripheral Blood of Chronic Fatigue Syndrome Patients: Risk Factors for Disease Progression

Torrisi, et al, wrote "The absence of lycoproteins on the cell surface of the infected cells," showing where the virus is hiding and how it infects (Virology, 257, 1999).

In 1995, Dr. Jay Levy, a world famous virologist from University of California, wrote on the "Isolation of Infectious Agent for CFS: Innoculation of Animals," from one of his patents, that he used cultures that were "evaluated for reverse transcriptase activity" and "we have found that the CD11b+ cells (suppressor CD8+ cells) decrease during CFS."

Presence of Viral Protein 1 (VP1)
"There are no tests to confirm a diagnosis, although 60% of sufferers will have a specific protein in their blood called viral protein 1, (VP1)."
Susan Clark, www.whatreallyworks.co.uk

In his Summary of the Viral Studies of CFS, Dr Dharam V Ablashi concluded: “The presentations and discussions at this meeting strongly supported the hypothesis that CFS may be triggered by more than one viral agent… Komaroff suggests that, once reactivated, these viruses contribute directly to the morbidity of CFS by damaging certain tissues and indirectly by eliciting an on-going immune response”(Clin Inf Dis 1994:18 (Suppl 1):S130-133). It is recommended that the entire 167-page Journal be read


Vaccines and Retroviruses: A Whistleblower Reveals What the Government is Hiding


The 1995 National Academies analysis for xenotransplantation (still in use today), which includes xenografting human and animal cells

Infectious Disease Risk to Public Health Posed by Xenografting: The possibility that infections can be transmitted from animals to humans is of concern not only because of the threat to the health of the recipient, but also because such infections may be transmissible to others, creating a public health hazard. Further, such infections may be due to previously unrecognized organisms, making detection difficult if not impossible. If the time from infection to clinical symptoms is long, the risk of widespread transmission is greater, because during this time the new organism may silently spread from person to person, as happened with human immunodeficiency virus (HIV).

Emergence of a new public health risk appears to be a two-step process (Morse, 1995). First, a new infectious agent is introduced into a given human population from other human populations, animals, or environmental exposures. Frequently these new agents are zoonoses, defined as animal microbes that can infect humans as well as the animal species from which they come. The second step is establishment and dissemination of organisms that prove to be infective and transmissible from person to person. The first step, introduction of a potentially transmissible agent into a human, could be accomplished by transplanting an organ that was infected with the agent. It is the second step of establishment and dissemination, however, that raises public health concerns, particularly if the agent is viral since current therapies for viral illnesses are limited.
Basis For Public Health Concern
Historic experience with many zoonotic diseases suggests that the potential for human infection with xenogeneic pathogens has implications for the community that extend beyond the individual transplant recipient.

“examples demonstrate that some zoonotic infections have the potential to extend beyond the individual and into the community. Thus, the risk of xenotransplant-associated infection is not restricted to the xenotransplant recipient alone. The potential for xenogeneic infections to be transmitted through human populations is real and poses a public health concern. Further, the risk for health care workers in close contact with the xenograft recipient is probably higher than for the community at large.”

The potential for the introduction of a new retrovirus into human hosts via implanted xenogeneic tissue is of public health concern due to the long period of clinical latency associated with all known human retroviral infections. This long latency period provides the opportunity for silent person-to-person transmission to occur before pathogenicity is evident. There are concerns that xenogeneic viruses may recombine or reassort with viruses latent in human tissues and result in variants that possess either a broader host range or an increased pathogenic potential. What options are available for risk management of xenotransplant-associated infectious public health risk? One option is to eliminate all risk by avoiding all use of xenogeneic tissue in humans.

Conclusions and Recommendations
Xenotransplantation may also be valuable for the treatment of human diseases. However, it is well recognized that infectious agents can be transmitted from animals to humans and that organisms benign in one species can be fatal when introduced into other species. Further, it is known that the pathogenicity of infectious organisms can change under a variety of conditions and that the effects of infection by some organisms, such as the human immunodeficiency virus, are delayed for years or even decades. Because xenotransplants involve the direct insertion of potentially infected cells, tissues, or organs into humans, there is every reason to believe that the potential for transmission of infectious agents (some of which may not even now be recognized) from animals to human transplant recipients is real. If established in the recipient, the potential for transmission to caregivers, family, and the population at large must be considered a real threat. However, all members of the committee agreed that some mechanism is needed to ensure attention to and reduction of the risk of infectious disease transmission.


Lectures by Dr. Judy Mikovitts
• HIV Negative AIDS
  - Acquired immunodeficiency without evidence of infection with human immunodeficiency virus types 1 and 2 J Laurence, MD, E Schattner, MD F.P Siegal, MD, I Gelman, PhD, S Morse, PhD. The Lancet Volume 340, Issue 8814, 1 August 1992, Pages 273-274
  - Acquired immunodeficiency without evidence of HIV infection: national retrospective survey A McNulty, JM Kaldor, AM McDonald, K Baumgart. BMJ, 1994 - bmj.com
  - AIDS or Chronic Fatigue, Newsweek magazine 1992
  - America's Biggest Cover-Up: 50 More Things Everyone Should Know About The Chronic Fatigue Syndrome Epidemic And Its Link To AIDS
    by Neenyah Ostrom
  - Some research listings about HIV negative AIDS
  - Some research listings on Non HIV AIDS
  - 50 Things you should know about the Chronic Fatigue Syndrome Epidemic by Neenyah Ostrom

• JHK virus

Dr. Sidney Grossberg, a world renowned virologist from the Medical College of Wisconsin, wrote the following in his Patent number 5,827,750 on 10/98*

The human virus on which the present invention is based has not been classified as to which virus family it belongs, but it most nearly resembles a retrovirus ....The present invention relates to the detection of the presence of an NMA (neuromyasthnia) virus that is associated with CFIDS." He goes on to talk of the "protein spikes in the envelope" which are called peplomers and these spikes are characteristic of a retrovirus. He calls this retrovirus the "JHK virus." He mentions that the retrovirus that is close to the same size is called the "mouse mammary tumor virus." In his only publication on the virus, one that went unannounced by the CFIDS Association despite their funding of him, Grossberg writes ( Res Virol, 1997; 148(3): 191-206 ), "The human B-lymphoblastoid cell line, designated JHK-3, with pre-B-cell characteristics, chronically produces two viruses, Epstein-Barr virus (EBV) and JHK virus, an apparently novel retrovirus...most nearly resembling C-type retroviruses."

JHK retrovirus isolate JHK-3 5' LTR, partial sequence; and gag protein (gag) gene, partial cds

Research by Dr. Sidney Grossberg

"Myeloadenamate Deaminase deficiency in muscles of ME patients. It is known that the enzyme is missing after a viral attack"

Professor Peter Behan, The Institute of Neurological Sciences, University of Glasgow, Scotland.


Torrisi, et al, wrote "The absence of lycoproteins on the cell surface of the infected cells," showing where the virus is hiding and how it infects (Virology, 257, 1999).

(e) Parainfluenza Virus-5 (PIV-5) & Paramyxovirus

- Potential Role of Persistent Paramyxovirus Infection in Chronic Fatigue Syndrome; Knox KK, Carrigan DR; National CFIDS Foundation: Interim Progress Report and Research Proposal; January 14, 2005

- National CFIDS Foundation: Personal Communications with Robert Lamb, Ph.D., Sc.D.; Professor of Molecular and Cellular Biology, Northwestern University; 2006

- Parainfluenza Virus-5: A new paradigm and a serious host challenge

- Viral Mechanisms of Immune Evasion; Alcami A, Koszinowski UH; Trends Microbiol. 2000 Sep;8(9):410-8


- Potential Role of Persistent Paramyxovirus Infection in Chronic Fatigue Syndrome. Konstance K. Knox, PhD Donald R. Carrigan, PhD Wisconsin Viral Research Group 10437 Innovation Drive Milwaukee, Wisconsin

"an agent was repeatedly transmitted to monkeys from two patients (Pellew and Miles, 1955). When the monkeys were killed minute red spots were observed along the course of the sciatic nerves. Microscopically infiltration of nerve roots with lymphocytes and mononuclear cells was seen and some of the nerve fibres showed patchy damage to the myelin sheaths and axon swellings. Similar findings had been produced by the transmission of an agent to monkeys from a child with poliomyelitis in Boston, Massachusetts, in 1947 (Pappenheimer, Cheever and Daniels, 1951). How- ever, in these monkeys the changes were more widespread, involving the dorsal root ganglia, cervical and lumbar nerve roots and peripheral nerves. Perivascular collars of lymphocytes and plasma cells were seen in the cerebral cortex, brain stem and cerebellum, spinal cord and around blood vessels to the nerve roots. There was no evidence of damage to the nerve cells in the brain or spinal cord.
The distribution and intensity of the lesions varied considerably from monkey to monkey. This pathological picture of mild diffuse changes corresponds closely to what might be expected from clinical observations of patients with neurological involvement in epidemic Neuromyasthenia.


(f) JHK virus

- **Partial molecular cloning of the JHK retrovirus using gammaretrovirus consensus PCR primers**


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  - The human virus on which the present invention is based has not been classified as to which virus family it belongs, but it most nearly resembles a retrovirus....The present invention relates to the detection of the presence of an NMA (neuromyasthenia) virus that is associated with CFIDS.” He goes on to talk of the "protein spikes in the envelope" which are called peplomers and these spikes are characteristic of a retrovirus. He calls this retrovirus the "JHK virus." He mentions that the retrovirus that is close to the same size is called the "mouse mammary tumor virus." In his only publication on the virus, one that went unannounced by the CFIDS Association despite their funding of him, Grossberg writes (Res Virol, 1997;148(3):191-206 ), “The human B-lymphoblastoid cell line, designated JHK-3, with pre-B-cell characteristics, chronically produces two viruses, Epstein-Barr virus (EBV) and JHK virus, an apparently novel retrovirus...most nearly resembling C-type retroviruses.”

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- "Myeloadenamate Deaminase deficiency in muscles of ME patients. It is known that the enzyme is missing after a viral attack" Professor Peter Behan, The Institute of Neurological Sciences, University of Glasgow, Scotland.


- **Torrisi, et al, wrote "The absence of lycoproteins on the cell surface of the infected cells," showing where the virus is hiding and how it infects** (Virology, 257, 1999).

(g) Parvovirus B-19


• **Bacterial and Viral Co-Infections in Chronic Fatigue Syndrome (CFS/ME) Patients.** Nicolson et al., Proc. Clinical & Scientific Conference on Myalgic Encephalopathy/Chronic Fatigue Syndrome, the Practitioners Challenge, Alison Hunter Foundation, Sydney, Australia 2002

• **The Putative Role of Viruses, Bacteria, and Chronic Fungal Biotoxin Exposure in the Genesis of Intractable Fatigue Accompanied by Cognitive and Physical Disability.** Morris et al. 2015

• Anti-pathogen and immune system treatments. Treatment of 741 Italian patients with chronic fatigue syndrome. U. TIRELLI, A. LLESHI, M. BERRETTA, M. SPINA, R. TALAMINI, A. GIACALONE. European Review for Medical and Pharmacological Sciences 2013; 17: 2847-2852


• Kerr et al then go on to provide evidence of other triggers of (ME)CFS which include Parvovirus; C. pneumoniae; C. burnetti; toxin exposure and vaccination including MMR, pneumovax, influenza, hepatitis B, tetanus, typhoid and poliovirus (LD Devanur, JR Kerr. Journal of Clinical Virology 2006: 37(3):139-150).


• “Presence of B19 NS1 gene sequence detected by nPCR. IgM and IgG detected by Elisa. B19 antibodies in 85.2% of patients. 57% had IgG. Genomic sequence found in more patients than healthy individuals Occurrence of Typical Clinical Symptoms and Markers of Human Parvovirus B19 Infection in Patients with Myalgic Encephalomyelitis/Chronic Fatigue Syndrome. Santa Rasa, Svetlana Chapenko, Angelika Krumina, Ludmila Viksna, Modra Murovska. IACFS/ME Conference. Translating Science into Clinical Care. March 20-23, 2014 • San Francisco, California, USA


• Association of Active Human Herpesvirus-6, -7 and Parvovirus B19 Infection with Clinical Outcomes in Patients with Myalgic Encephalomyelitis/Chronic Fatigue Syndrome. Svetlana Chapenko, Angelika Krumina, Inara Logina, Santa Rasa, Maksims Chistjakovs, Alina Sultanova, Ludmila Viksna, and Modra Murovska. Advances in Virology. Volume 2012, Article ID 205085, 7 pages

• Presence of Viral Protein 1 (VP1) "There are no tests to confirm a diagnosis, although 60% of sufferers will have a specific protein in their blood called viral protein 1, (VP1)." Susan Clark, www.whatreallyworks.co.uk


• "Myeloadenamate Deaminase deficiency in muscles of ME patients. It is known that the enzyme is missing after a viral attack." Professor Peter Behan, The Institute of Neurological Sciences, University of Glasgow, Scotland.
- Torrisi, et al, wrote "The absence of lycoproteins on the cell surface of the infected cells," showing where the virus is hiding and how it infects (Virology, 257, 1999).


(h) Genetic Evidence of Infections


- [Genetic Findings from around the world](http://example.com)

(i) Borna virus

- [Listing of research papers](http://example.com)

- [Listing op papers on PubMed databases](http://example.com)


(j) Cryptovirus

- Potential Animal (Zoonotic) Virus Identified in Patients with Chronic Fatigue Syndrome, Multiple Sclerosis and Epilepsy

(k) HERV-K18 virus

- Dr. Huber of Tufts University in the USA is carrying out research to determine if viral infections are leading to the reactivation of latent endogenous viruses such as HERV-K18 in ME/CFS patients. This is important as HERV-K18 has a super antigen which can cause the immune system to become over-activated and dysfunctional. Over time this can lead to a weakened immune system which is both activated and dysfunctional, and not capable of combatting other infections, and this is what we see in ME/CFS. This research will determine if this is what is occurring in ME/CFS and it is due to be published in 2013.

- [Plasmacytoid Dendritic Cells in the Duodenum of Individuals Diagnosed with Myalgic Encephalomyelitis Are Uniquely Immunoreactive to Antibodies to Human Endogenous Retroviral Proteins](http://example.com), De Meirleir et al. in vivo 27: 177-188 (2013).

(l) Stealth Viruses and Chronic Fatigue Syndrome
Attempts to define the chronic fatigue syndrome (CFS) as a clinical diagnostic entity have met with difficulties mainly because of a lack of clear separation of what could be considered normal variation in human functional capacity, and what should be considered a medical illness. Patients with debilitating fatigue are inappropriately grouped along with individuals with only minimal impairment in their daily activities. Some severely affected CFS patients eventually meet criteria for neurological, psychiatric and/or immunological disease classifications. The possible connection between CFS and these other diseases is unfortunately obscured by present day terminology.

The thesis of our studies is that severe CFS is but one of many manifestations of a persistent, systemic viral infection that causes brain damage. Involvement of the brain in CFS is implied by the historical use of terms such as neurasthenia, myalgic encephalomyelitis, and limbic encephalopathy. Some investigators have argued that the disturbed brain function is a secondary phenomenon resulting, for example, from the overproduction of neuromodulatory cytokines. Immune dysregulation is also proposed to explain reactivation of normally tolerated ubiquitous microorganisms, such as Epstein-Barr virus, human herpesvirus-6, Candida albicans, Mycoplasma fermentans, Chlamydia pneumoniae, etc. Recent attention has also been given to possible brain damage from exposure to environmental neurotoxins, including gut derived bacterial products.

Minimizing the potential infectious etiology of CFS has occurred in spite of past and recent epidemic outbreaks of CFS-like illnesses. Reasons for this bias include the inability of most investigators to isolate pathogenic viruses from CFS patients, and the lack of any correlation of disease with conventional anti-viral serology. Published studies using the polymerase chain reaction (PCR) to test for evidence of retroviruses, enteroviruses, conventional herpesviruses and mycoplasma infections, were also flawed by erroneous assumptions concerning the specificity of the PCR assays when performed under low stringency conditions.

These earlier studies can now be reconciled by the finding that most severely ill CFS patients are infected with atypically structured cytopathic viruses. The viruses have been termed "stealth" since they apparently lack crucial antigenic determinants that would act as effective targets for cell mediated anti-viral immunity. The viruses can be grown in a wide range of cells of both human and animal origins, inducing a foamy, vacuolating cytopathic effect (CPE). A similar CPE can be seen in brain biopsies obtained from severely ill stealth virus infected humans and from experimentally inoculated animals. The cellular changes occur in the absence of an inflammatory reaction and are easily overlooked if not specifically sought.

Although many of the patients' symptoms are referable to the brain, virus infection is widespread and can involve multiple organs. The term multi-system stealth virus infection with encephalopathy (MSVIE) more accurately conveys the complexities of the illnesses seen in infected CFS patients. This term also helps to restore the extensive overlaps between CFS and other stealth virus associated illnesses, including aberrant behavioral and learning problems in children, fibromyalgia, Gulf War syndrome and psychiatric illnesses in adults, and progressive movement disorders and dementia in the elderly. The systemic nature of the infection can explain the varied endocrine, cardiovascular, gastrointestinal, immunological and other disease manifestations seen in many of these patients.

Stealth adaptation can presumably occur with any type of cytopathic virus. I have primarily focussed on a stealth adapted African green monkey simian cytomegalovirus (SCMV). Extensive sequencing studies on this virus have confirmed the lack of critical antigens utilized by anti-cytomegalovirus cytotoxic T lymphocytes. The virus has managed to capture, amplify and mutate various non-viral genes, including cellular genes and genes of bacterial origin. The term viteria has been introduced to describe viruses infectious for humans and animals that have acquired bacterial genetic sequences. The presence of bacterial sequences can help explain the unusual serological and PCR based assay results seen in some CFS patients. They may also contribute to the
allergic manifestations occasionally observed in these patients.

Rational therapy for severely ill stealth virus infected patients can reasonably include empirical trials with anti-viral agents. Significant improvement has been reported in some patients using valcyclovir and in a larger group of patients using ganciclovir.24,25 Antibiotics may have a role if viteria infected bacteria can be demonstrated. Additional therapy needs to be individualized according to the patient's symptoms and the extent of multi-organ damage. There is a role for neurally active medications, nutritional supplements and possibly probiotics. The vexed question of how to help minimize transmission of infection within both the workplace and the family also needs to be addressed. Additional information relating to stealth viruses and copies of key publications can be found at the web site www.c cid.org

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e-mail: ccidlab@hotmail.com

References


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**H1N1 virus**


**Other viruses**


- Peripheral blood gene expression in postinfective fatigue syndrome following from three different triggering infections.Galbraith S, Cameron B, Li H, Lau D, Vollmer-Conna U, Lloyd AR.

- The Putative Role of Viruses, Bacteria, and Chronic Fungal Biotoxin Exposure in the Genesis of Intractable Fatigue Accompanied by Cognitive and Physical Disability. Morris et al. 2015


"Individuals with CFS have characteristic clinical and laboratory findings including...evidence of viral reactivation...The object of this study was to evaluate the status of key parameters of the 2-5A synthetase/RNase L antiviral pathway in individuals with CFS who participated in a placebo-controlled, double-blind, multi-centre trial...The present work confirms the finding of elevated bioactive 2-5A and RNase L activity in CFS...RNase L, a 2-5A-dependent enzyme, is the terminal effector of an enzymatic pathway that is stimulated by either virus infection or exposure to exogenous lymphokines. Almost two-thirds of the subjects...displayed baseline RNase L activity that was elevated above the control mean" (Robert J Suhadolnik, Daniel L Peterson, Paul Cheney et al. In Vivo 1994;8:599-604).


"Although serum immunoreactivity to BDV proteins observed in Swedish CFS patients by ELISA may reflect infection with related microbial agents that induce cross-reactivity with conformational determinants on BDV proteins (Kliche et al , 1996) and b - galactosidase, the serologic findings are also consistent with non specific polyclonal B-cell activation." Absence of evidence of Borna disease virus infection in Swedish patients with Chronic Fatigue Syndrome Birgitte EvengaÈrd, Thomas Briese , Gudrun Lindh , Shaun Lee and W Ian Lipkin

Vojdani A , Lapp CW . Interferon-induced proteins are elevated in blood samples of patients with chemically or virally induced chronic fatigue syndrome. Immunopharmacol Immunotoxicol. 1999 May;21(2):175-202. PMID: 10319275

Certain toxic chemicals and certain viruses produce the same kinds of inflammatory effects and defects in 2-5A Synthetase and Protein Kinase RNA (PKR)). Anti IFN beta inhibited the reactions.


Infectious agents, tranfusion or Hepatitis B vaccination may play an important role in the onset of CFS. Associated with these a number of stressors and consequent immunological and neuroendocrinological changes can contribute to the onset of the illness.

P. De Becker (Brussels), research paper presented to the AACFS 5th International Research, Clinical and Patient Conference, 2001


Michael Holmes of the Department of Microbiology of the University of Otago (New Zealand) carried out detailed studies into a CFS-like illness in New Zealand in the 1980's and 1990's. His findings suggest that a virus, possibly a retrovirus was responsible and that there is also significant immune dysfunction in CFS. Reported in the book 'Osler's Web', by Hillary Johnson, Penguin Books 1997, pages 662-663


The world patent entitled "Method and Compositions for Diagnosing and Treating Chronic Fatigue Immunodysfunction Syndrome" #WO9205760 issued to Elaine DeFreitas and Brendan Hilliard, inventors assigned to Wistar Institute, USA. This patent was applied for in August 1991. It concerns the discovery of a new virus the CAV virus which may lie at the root of CFS / ME.

"Myoadenamate Deaminase deficiency in muscles of ME patients. It is known that the enzyme is missing after a viral attack" Professor Peter Behan, The Institute of Neurological Sciences, University of Glasgow, Scotland.
Presence of Viral Protein 1 (VP1)

"There are no tests to confirm a diagnosis, although 60% of sufferers will have a specific protein in their blood called viral protein 1, (VP1)."

Susan Clark, www.whatreallyworks.co.uk


“The clinical, pathological, electrophysiological, immunological and virological abnormalities in 50 patients with the postviral fatigue syndrome are recorded. These findings confirm the organic nature of the disease (and) suggest that it is associated with disordered regulation of the immune system and persistent viral infection” (PO Behan, WMH Behan, EJ Bell. Journal of Infection 1985:10:211-222.)


In his Summary of the Viral Studies of CFS, Dr Dharam V Ablashi concluded: “The presentations and discussions at this meeting strongly supported the hypothesis that CFS may be triggered by more than one viral agent…Komaroff suggests that, once reactivated, these viruses contribute directly to the morbidity of CFS by damaging certain tissues and indirectly by eliciting an on-going immune response”(Clin Inf Dis 1994:18 (Suppl 1):S130-133). It is recommended that the entire 167-page Journal be read

“Our focus will be on the ability of certain viruses to interfere subtly with the cell’s ability to produce specific differentiated products as hormones, neurotransmitters, cytokines and immunoglobulins etc in the absence of their ability to lyse the cell they infect. By this means viruses can replicate in histologically normal appearing cells and tissues…Viruses by this means likely underlie a wide variety of clinical illnesses, currently of unknown aetiology, that affect the endocrine, immune, nervous and other …systems” (JC de la Torre, P Borrow, MBA Oldstone. BMB 1991:47:4:838-851).

“The illness has an acute onset after a variety of infections and then enters a chronic phase characterised by fatigue and numerous other symptoms….Other findings include a sleep disorder, mild immunodeficiency, slightly low complement, anti-DNA antibodies and elevated synthetase which is an interferon-associated enzyme commonly increased in viral infections” (Irving E Salit. Clinical Ecology 1987:V:3:103-107).

Torrisi, et al, wrote "The absence of lycoproteins on the cell surface of the infected cells," showing where the virus is hiding and how it infects (Virology, 257, 1999).


Acheson ED (1956), "A New Clinical Entity?", Leading Article, Lancet, 26 mei, pp. 789-90


Lerner, AM et al. A small randomised placebo-controlled trial of the use of antiviral therapy for patients with chronic fatigue syndrome. Clinical Infectious Diseases, 2001, 32, 1657-1658


Suhadolnik, Daniel L Peterson, Dharam V Ablashi, Fred Friedberg, Jay A Levy, Peter O Behan, Wilhelmina MH Behan and Mark O Loveless.

Torrissi, et al, wrote "The absence of lycoproteins on the cell surface of the infected cells," showing where the virus is hiding and how it infects (Virology, 257, 1999).


(o) ME outbreaks suggesting role of viruses and other pathogens

Viruses have been isolated from the muscles of ME patients during epidemics. Using PCR methods, 50% or more of patients had evidence of such infection.


- Altered Distribution of Lymphocyte Populations in Chronic Fatigue Syndrome Patients Isabel Barao, Ph.D., Daniel Peterson, M.D., Dorothy Hudig, Ph.D. Mountain West Clinical Translational Research. 2014.


http://www.meresearch.org.uk/information/keypubs/Acheson_AmJMed.pdf


Acheson ED. Benign myalgic encephalomyelitis. Lancet. 1957 Apr 20;272(6973):834-5. PMID: 13417614


A new Clinical Entity? Lancet 1956


Years of Epidemics
1917 Van Economo reports an illness involving brain and neurological inflammation and great fatigue and some deaths. See paper 'New Clinical Entity' published in the Lancet in 1956.
1918 - 1924, several outbreaks of an illness involving brain and neurological inflammation and fatigue reported throughout Europe. See paper 'New Clinical Entity' published in the Lancet in 1956.
1934 Los Angeles County Hospital. Called 'Atypical Poliomyelitis'
1936 Fond Du Lac, Wisconsin - St. Agnes Convent - Encephalitis
1937 Erstfeld, Switzerland - Abortive Poliomyelitis
1937 St. Gallen, Switzerland - Frohburg Hospital – Abortive Poliomyelitis
1939 Middlesex, England - Harefield Sanatorium
1939 Degersheim, Switzerland - Abortive Poliomyelitis
1945 Pennsylvania. Hospital of the University of Pennsylvania - epidemic Pleurodynia
1946 Iceland disease resembling Poliomyelitis with the character of Akureyri disease
1948 Iceland, North Coast towns - epidemic simulating Poliomyelitis
1949 Adelaide, South Australia - a disease resembling Poliomyelitis
1949 Cambridgeshire, England - aberrant poliomyelitis. Involvement of other Enteroviruses suspected.
1950 Louisville, Kentucky -- St. Joseph 's Infirmary - epidemic Neuromyasthenia
1950 Upper State New York -- outbreak resembling the Iceland disease, simulating " acute Anterior Poliomyelitis
1952 Copenhagen, Denmark - epidemic Myositis
1952 Lakeland, Florida - epidemic Neuromyasthenia
1953 Coventry and District, England - an illness resembling Poliomyelitis observed in nurses
1953 Rockville, Maryland - Chestnut Lodge Hospital - Poliomyelitis-like epidemic Neuromyasthenia
1953 Jutland, Denmark - epidemic Encephalitis with vertigo
1954 Seward, Alaska - benign Myalgic Encephalomyelitis (Iceland Disease)
1954 Berlin, Germany - British army - further outbreak of a disease resembling Poliomyelitis
1954 Liverpool, England - outbreak among medical and nursing staff in a local hospital
1955 Dalston, Cumbria, England - epidemic and sporadic outbreak of an unusual disease
1955 London, England - Royal Free Hospital - outbreak in staff and patients of Benign Myalgic Encephalomyelitis
1955 Hampstead, London
1955 Perth, Australia - virus epidemic in waves
1955 Gilfac Goch, Wales - outbreak of benign Myalgic Encephalomyelitis
1955 Durban City, South Africa - Addington Hospital - outbreak among nurses of Durban Mystery Disease
1955 Segbwema, Sierra Leone - outbreak of Encephalomyelitis
1955 Patreskfljorour and Porshofn, Iceland - unusual response to polio vaccine
1955 Northwest London, England - nurses ' residential home - acute Infective Encephalomyelitis simulating poliomyelitis
1956 Ridgefield, Connecticut - epidemic Neuromyasthenia
1956 Punta Gorda Florida - outbreak of epidemic Neuromyasthenia
1956 Newton-le-Willows, Lancashire, England - Lymphocytic Meningoencephalitis with myalgia and rash
1956 Pittsfield and Williamstown, Massachusetts - benign Myalgic Encephalomyelitis
1956 Coventry, England - epidemic malaise, benign Myalgic Encephalomyelitis
1957 Brighton, South Australia - Cocksakie Echo virus Meningitis, epidemic Myalgic Encephalomyelitis
1958 Athens, Greece - nurses ' school - outbreak of benign Myalgic Encephalomyelitis with periostitis and arthopathy noted.
1958 Southwest London, England - reports of sporadic cases of Myalgic Encephalomyelitis
1959 Newcastle Upon Tyne, England - outbreak of benign Myalgic Encephalomyelitis
1961 Basel, Switzerland - sporadic cases of benign Myalgic Encephalomyelitis
1961 New York State - outbreak of epidemic Neuromyasthenia in a convent
1964 Franklin, Kentucky - outbreak of Neuromyasthenia in a factory
1967 Edinburgh, Scotland - sporadic cases resembling benign Myalgic Encephalomyelitis
1968 " Fraidek, Lebanon - benign Myalgic Encephalomyelitis
1969 Brooklyn, New York - State University of New York Downstate Medical Center - epidemic Neuromyasthenia, unidentified symptom complex
1970 Lackland Air Force Base, Texas - epidemic Neuromyasthenia
1970 London, England - Great Ormond Street Hospital for Children - outbreak of Neuromyasthenia among nurses
1975 Sacramento, California - Mercy San Juan Hospital - Infectious Venulitis, epidemic " Phelobodynia
1976 Southwest Ireland - epidemic Neuromyasthenia, benign Myalgic Encephalomyelitis
1977 Dallas – Fort Worth, Texas - epidemic Neuromyasthenia
1979 Southampton, England - Myalgic Encephalomyelitis
1980 West Kilbridge, Ayrshire, Scotland - epidemic Myalgic Encephalomyelitis
A viral disease (or viral infection or infectious disease), occurs when an organism's body is invaded by pathogenic viruses, and infectious virus particles (virions) attach to and enter susceptible cells. Basic structural characteristics, such as genome type, virion shape and replication site, generally share the same features among virus species within the same family. Viral infections occur when viruses enter cells in the body and begin reproducing, often causing illness. Viruses are tiny germs that can reproduce only by invading a living cell. KEYWORDS: Infection, Polymerase chain reaction, Virology. For instance, rhinoviruses cause colds, influenza viruses cause flu, adenoviruses cause various respiratory problems, and rotaviruses cause gastroenteritis. Polioviruses can cause severe illnesses. Learn more about viral infections and their symptoms. National Institutes of Health. The primary NIH organization for research on Viral Infections is the National Institute of Allergy and Infectious Diseases.